

# A Control Model for Dengue Fever in a Disease-Endemic Region

Emmanuel Chidiebere Duru \* and Michael Chimezie Anyanwu

Department of Mathematics, Michael Okpara University of Agriculture, Umudike 440101, Abia, Nigeria

\* Correspondence: [duru.emmanuel@mouau.edu.ng](mailto:duru.emmanuel@mouau.edu.ng)

**How To Cite:** Duru, E.C.; Anyanwu, M.C. A Control Model for Dengue Fever in a Disease-Endemic Region. *Applied Mathematics and Statistics* 2026, 3(2), 12. <https://doi.org/10.53941/ams.2026.100012>

Received: 6 June 2026

Revised: 17 June 2026

Accepted: 22 June 2026

Published: 8 July 2026

**Abstract:** Dengue fever is one of the life-threatening diseases transmitted by mosquitoes, specifically, the *Aedes aegypti* mosquitoes. It is mostly seen in the tropical regions of Africa and Asia, contributing to significant health burdens globally. Despite several efforts to control the disease and several studies in this area, the disease remains endemic in some places of the world. The increase in the endemicity of the disease is due to the increasing rate of mosquito-resistance to insecticide spray and absence of drugs for its treatment. These challenges underscore the need for researches to suggest better ways of controlling the disease. In this work, a system of nonlinear ordinary differential equations is used to model Dengue fever in a disease-endemic region. This new model incorporates vaccination, preventive measures such as use of mosquito treated bed-nets and mosquito repellent, use of insecticide spray and fogging as well as encouraging regular testing for the disease since a large proportion of infected humans are usually asymptomatic. The model is first shown to be epidemiologically and mathematically well-posed before obtaining the equilibrium points of the system as well as the control reproduction number. The conditions for local and global stability analysis of the equilibrium points of the system are established. Sensitivity analysis is also carried out to show the parameters that affect the endemicity of the disease. Numerical simulations are employed to show the effects of the proposed controls. The controls were seen to be very effective in reducing the spread of the disease and it is recommended that in any region where Dengue is endemic, employing the proposed controls can help reduce or eliminate the spread of the disease.

**Keywords:** dengue; vaccination; sensitivity analysis; bifurcation; stability analysis

## 1. Introduction

Dengue fever (break-bone fever) is a viral infection that is predominantly spread from mosquitoes to human [1,2]. It is more common in tropical and subtropical regions than in temperate climates [1]. Most people who get dengue do not have symptoms. Some develop severe dengue and need care in a hospital. In severe cases, dengue can be fatal [1]. The dengue virus (DENV) is transmitted to humans through the bites of infected female mosquitoes, primarily the *Aedes aegypti* mosquito [3,4]. Mosquitoes can become infected by people who are viremic with Dengue fever. This can be someone who has a symptomatic dengue infection, someone who is yet to have a symptomatic infection (those who are pre-symptomatic), and also someone who shows no signs of illness (those who are asymptomatic). Dengue fever has four serotype viruses responsible for the disease transmission and they are; DENV-1, DENV-2, DENV-3 and DENV-4 [1,5]. It is reported that about 400 million new cases occur annually in the world with over 3.9 billion humans at risk [2,6]. There were 6 million cases across 92 countries in 2023 alone [7].



Most people with dengue have mild or no symptoms and will get better within 1–2 weeks. Dengue can rarely be severe and lead to death. Symptoms usually begin 4–12 days after infection and last for 2–7 days when they occur [8,9]. Symptoms may include: high fever (40 °C/104 °F), severe headache, pain behind the eyes, muscle and joint pains, nausea, vomiting, swollen glands, rash [1,10]. Severity of the disease is usually associated with reinfection with symptoms such as severe abdominal pain, persistent vomiting, rapid breathing, bleeding gums or nose, fatigue, restlessness, blood in vomit or stool, being very thirsty, pale and cold skin, feeling weak [10]. People with these severe symptoms should seek care immediately. After recovery, people who have had dengue may experience fatigue for several weeks. There is evidence, however, of the possibility of maternal transmission (i.e., from a pregnant mother to her baby). At the same time, vertical transmission rates appear low, with the risk of vertical transmission seemingly linked to the timing of acquiring the dengue infection during pregnancy. When a mother does have a dengue infection when she is pregnant, babies may suffer from pre-term birth, low birthweight and fetal distress [2]. Currently, one vaccine (QDenga) is available and licensed in some countries. However, it is recommended only for those aged 6–16 years in high transmission settings. Several additional vaccines are under evaluation (11).

There are several literatures on modelling the control of Dengue fever such as [1] which proposed a Dengue fever model with data from West Java Province, Indonesia where optimal control analysis was employed to show the effects of fumigation and prevention on the dynamics of the disease. The authors in [11] proposed the use of Wolbachia-infected mosquitoes to control the population of *Aedes aegypti* mosquito responsible for Dengue fever. In [12], the authors worked on a model for Dengue fever which classified sex in mosquitoes. [13] discussed a model with two different serotype infections and vaccination as control. The authors in [14] proposed a model for the control of the disease in Jakarta, Indonesia. In [15], the authors incorporated isolation, asymptomatic occurrence and vigilance in an optimal control model of Dengue fever. In this work, a new mathematical model for the disease is proposed. The model incorporates vaccination, use of insecticides spray and fogging, preventive measures against mosquito bites such as use of mosquito repellent and mosquito treated bed-nets as well as periodic testing of possible infection and isolation as control measures. The authors in [5] explored on the effects of vector, sexual and vertical transmission on the dynamics of a Dengue fever model. Similar approach was adopted in the works of [16–18]. The authors in [6,19,20] however concentrated on climate variability, vector control and human behaviour. Several works on mathematical modelling of Dengue fever disease include the works in [4,21–24].

In this work, a new mathematical model for the disease is proposed. The model incorporates vaccination, use of insecticides spray and fogging, preventive measures against mosquito bites such as use of mosquito repellent and mosquito treated bed-nets as well as periodic testing of possible infection and isolation as control measures. Regular testing was employed to help detect possible asymptomatic occurrence and manage it. The model is set up to control the spread of the disease in a region where the disease is endemic, hence the suggested controls. All the works considered in literature did not incorporate regular testing of susceptible humans for possible infection and isolation neither did they focus mainly on endemic regions of the disease. It is expected that as humans in an endemic region undergoes regular testing for possible infection, some asymptomatic cases will be identified and managed to reduce the spread of the disease. The rest of this work is arranged thus; in section two, the model is introduced and shown to be well-posed. Also, the disease-free equilibrium point and control reproduction number are obtained. In section three, stability analyses are carried out while in section four, sensitivity analysis is done. Numerical simulation is carried out in section five while the work is concluded in section six.

## 2. Model Formulation

The new mathematical model proposed in this work for the control of Dengue fever is developed using a system of ordinary differential equation made up of seven (7) compartments; five (5) compartments for human population and two compartments (2) for vector population. The human compartments are; susceptible humans,  $S_h$ , traced and tested humans,  $T_h$ ; vaccinated humans,  $V_h$ ; infectious humans,  $I_h$  and recovered humans,  $R_h$ . The mosquito population is divided into the susceptible mosquitoes,  $S_D$  and infectious mosquitoes,  $I_D$ . The system variables and parameters are described in Table 1 and Figure 1.

### 2.1. Model Assumptions

To set up the mathematical model, the following assumptions were made;

- A proportion of susceptible humans go for regular testing and those detected with Dengue fever are isolated for treatment.
- A proportion of susceptible humans are vaccinated at the rate,  $\theta$  and loses their immunity as the vaccine wanes with time at the rate,  $\phi$ .

- Vaccination does not grant permanent immunity to the disease; hence the vaccinated humans can get infected though at a lower rate than unvaccinated humans.
- Four controls are incorporated into the system which are; vaccination, use of mosquito repellent, use of mosquito treated bed-nets and regular testing for possible infection.

Regular testing was introduced since there is more occurrence of asymptomatic infections than symptomatic infections. It is expected that the controls will reduce or eliminate the spread of Dengue fever in the system.

The model equation is given by;

$$\begin{aligned}\frac{dS_h}{dt} &= \pi_h + \phi V_h - (\lambda_1 + \theta + \rho + \mu_h)S_h, \\ \frac{dT_h}{dt} &= \rho S_h + \kappa I_h - (v_1 + \mu_h)T_h, \\ \frac{dV_h}{dt} &= \theta S_h - (\phi + \lambda_2 + \mu_h)V_h, \\ \frac{dI_h}{dt} &= \lambda_1 S_h + \lambda_2 V_h - (\mu_h + \mu_D + v_2 + \kappa)I_h, \\ \frac{dR_h}{dt} &= v_1 T_h + v_2 I_h - \mu_h R_h, \\ \frac{dS_D}{dt} &= \pi_m - (\lambda_3 + \mu_m + \mu_c)S_D, \\ \frac{dI_D}{dt} &= \lambda_3 S_D - (\mu_m + \mu_c)I_D,\end{aligned}\tag{1}$$

where  $S_h^0, T_h^0, V_h^0, I_h^0, R_h^0, S_D^0, I_D^0$  are the initial conditions of the Equation (1). The total human population is given by

$$N_h = S_h + T_h + V_h + I_h + R_h\tag{2}$$

and the total mosquito population is given by

$$N_D = S_D + I_D.\tag{3}$$

The forces of infection in the Equation (1) are

$$\begin{aligned}\lambda_1 &= \frac{\alpha\beta_1(1-c_1)(1-c_2)I_D}{N_h}, \\ \lambda_2 &= \frac{\alpha\beta_2(1-c_1)(1-c_2)I_D}{N_h}, \\ \lambda_3 &= \frac{\alpha\beta_3(1-c_1)(1-c_2)I_h}{N_h}.\end{aligned}$$

The state variables  $(S_h, T_h, V_h, I_h, R_h)$  lies in the region  $\Omega_h$  while  $(S_D, I_D)$  lies in  $\Omega_D$  with the entire solution space defined by  $\Omega = \Omega_h \times \Omega_D$  where

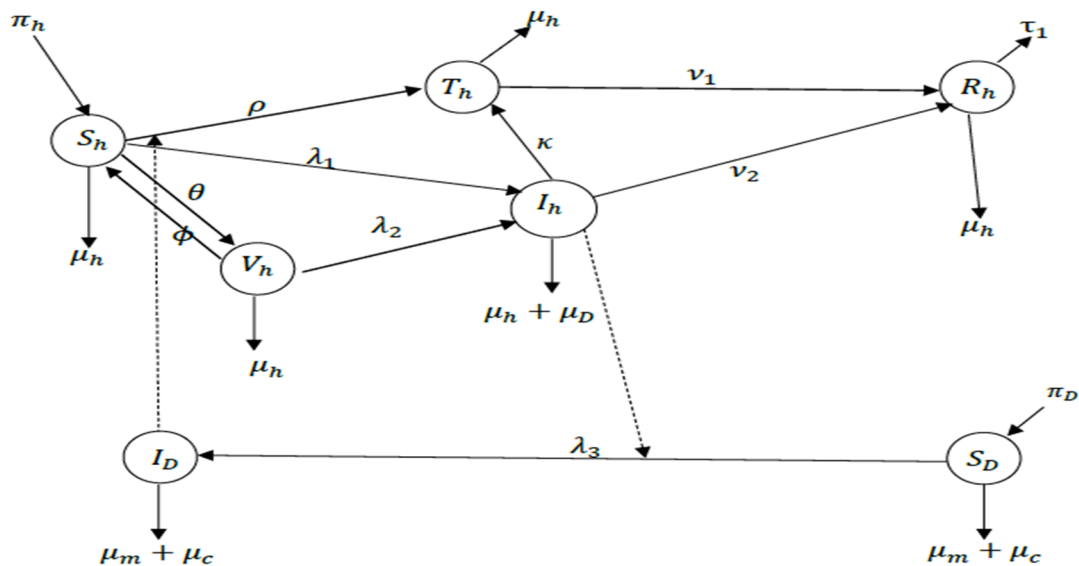
$$\Omega_h = \left\{ (S_h, T_h, V_h, I_h, R_h) \in \mathcal{R}_+^5 : N_h \leq \frac{\pi_h}{\mu_h} \right\}$$

and

$$\Omega_D = \left\{ (S_D, I_D) \in \mathcal{R}_+^2 : N_D \leq \frac{\pi_m}{\mu_m + \mu_c} \right\}.$$

**Table 1.** Description of Variables and Parameters in the model.

Variables	Descriptions	Values	Source
$S_h$	Susceptible humans	1000	Assumed
$T_h$	Tested and isolated humans	80	Assumed
$V_h$	Vaccinated humans	120	Assumed
$I_h$	Infectious humans	280	Assumed
$R_h$	Recovered humans	60	Assumed
$S_D$	Susceptible mosquitoes	10000	Assumed
$I_D$	Infectious mosquitoes	370	Assumed
Parameters	Descriptions		
$\pi_h$	Rate of human recruitment	100	Assumed
$\pi_m$	Rate of vector recruitment	500	Assumed
$\phi$	Rate of waning of vaccine	0.01	Assumed
$\theta$	Rate of vaccination	0.05	Assumed
$\mu_h$	Natural mortality rate in humans	0.00004	[25]
$\mu_D$	Disease-induced death rate	0.002	Assumed
$\mu_m$	Natural mortality rate in vectors	0.0556	[26]
$\mu_c$	Mosquito death rate due to insecticide	0.2	Assumed
$\rho$	Proportion of $S_h$ tested, infected and isolated for treatment	0.01	Assumed
$\nu_1$	Rate of recovery of isolated infectious humans	0.25	Assumed
$\nu_2$	Rate of recovery of non-isolated infectious humans	0.1	Assumed
$\kappa$	Proportion of $I_h$ hospitalized for treatment	0.15	Assumed
$\beta_1$	Probability of transmission from $I_D$ to $S_h$	0.3632	[27]
$\beta_2$	Probability of transmission from $I_D$ to $V_h$	0.1312	Assumed
$\beta_3$	Probability of transmission from $I_h$ to $S_D$	0.3252	[27]
$c_1$	Rate of use of mosquito treated bed-nets	0.1	Assumed
$c_2$	Rate of use of mosquito repellent	0.05	Assumed
$\alpha$	Biting rate of mosquitoes	0.4	[25]



**Figure 1.** Disease Transmission diagram.

2.2. Positivity of Solutions and Invariant Region

In this subsection, the model is shown to be well-posed mathematically and epidemiologically to guarantee that the system is well-defined and can be analyzed.

**Theorem 1.** Given that the initial conditions of the system  $(S_h^0, T_h^0, V_h^0, I_h^0, R_h^0, S_D^0, I_D^0) > 0$  at  $t = 0$  lie in the interval  $\Omega$ , then the solution set  $(S_h(t), T_h(t), V_h(t), I_h(t), R_h(t), S_D(t), I_D(t))$  to the system remains positive, for all  $t > 0$ .

**Proof.** From Equation (1);

$$\frac{dS_h}{dt} = \pi_h + \phi V_h - (\lambda_1 + \theta + \rho + \mu_h)S_h, \quad \Rightarrow \quad \frac{dS_h}{dt} \geq -(\lambda_1 + \theta + \rho + \mu_h)S_h,$$

$$\Rightarrow \quad \frac{dS_h}{S_h} \geq -(\lambda_1 + \theta + \rho + \mu_h)dt, \quad \Rightarrow \quad \int \frac{dS_h}{S_h} \geq -\int (\lambda_1 + \theta + \rho + \mu_h)dt,$$

$$\Rightarrow \quad \ln S_h \geq -\int (\lambda_1 + \theta + \rho + \mu_h)dt + c,$$

$$S_h(t) \geq e^{-\int (\lambda_1 + \theta + \rho + \mu_h)dt} \times e^c = Ae^{-\int (\lambda_1 + \theta + \rho + \mu_h)dt}.$$

At  $t = 0$ ,  $S_h(0) = S_h^0$ , then  $A = S_h^0$ . This implies that

$$S_h(t) \geq S_h^0 e^{-\int (\lambda_1 + \theta + \rho + \mu_h)dt} > 0, \quad \forall t > 0.$$

Similarly,

$$T_h(t) \geq T_h^0 e^{(v_1 + \mu_h)t} > 0, \quad \forall t > 0.$$

$$V_h(t) \geq V_h^0 e^{-\int (\phi + \lambda_2 + \mu_h)dt} > 0, \quad \forall t > 0.$$

$$I_h(t) \geq I_h^0 e^{-\int (\mu_h + \mu_D + v_2 + \kappa)dt} > 0, \quad \forall t > 0.$$

$$R_h(t) \geq R_h e^{-\mu_h t} > 0, \quad \forall t > 0.$$

$$S_D(t) \geq S_D^0 e^{-\int (\lambda_3 + \mu_m + \mu_c)dt} > 0, \quad \forall t > 0.$$

$$I_D(t) \geq I_D^0 e^{-(\mu_m + \mu_c)t} > 0, \quad \forall t > 0.$$

Hence, all the solutions will remain positive for all  $t > 0$ .  $\square$

**Theorem 2.** Every solution to the Equation (1) lies within the region  $\Omega$ . That is, the region  $\Omega$  is bounded.

**Proof.** In this section, the region,  $\Omega$  is shown to be positively invariant by showing that all the solutions to the model system will enter and remain in the region  $\Omega$ . The total human population is given by

$$N_h = S_h + T_h + V_h + I_h + R_h,$$

and satisfies the differential equation

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dT_h}{dt} + \frac{dV_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt}.$$

Thus,

$$\frac{dN_h}{dt} = \pi_h - \mu_h N_h - \mu_D I_D.$$

But,  $\frac{dN_h}{dt} \leq \pi_h - \mu_h N_h$  and  $\frac{dN_h}{dt} + \mu_h N_h \leq \pi_h$ .

By integration,

$$N_h(t) \leq \frac{\pi_h}{\mu_h} + ce^{-\mu_h t}.$$

As  $t \rightarrow \infty$ , we have  $N_h(t) \leq \frac{\pi_h}{\mu_h}$ , showing that the human population is bounded and the set,  $\Omega$  is positively invariant and is an attractor of all positive solutions to the system described by the human population.

Similarly, the total vector population is given by

$$N_D = S_D + I_D,$$

which satisfies the differential equation

$$\frac{dN_D}{dt} = \frac{dS_D}{dt} + \frac{dI_D}{dt}.$$

Thus,

$$\frac{dN_D}{dt} = \pi_m - (\mu_m + \mu_c)N_D.$$

By integration,  $N_D(t) \leq \frac{\pi_m}{\mu_m} + ce^{-(\mu_m + \mu_c)t}$ . As  $t \rightarrow \infty$ , we have  $N_D(t) \leq \frac{\pi_m}{\mu_m + \mu_c}$ , showing that the vector population is bounded and the set,  $\Omega_D$  is positively invariant and is an attractor of all positive solutions to the system described by the vector population. If  $N_h$  and  $N_D$  are bounded, then  $N = N_h + N_D$  is also bounded and  $\Omega = \Omega_h \times \Omega_D$  is positively invariant and is an attractor of all positive solutions to the system. The proofs of

Theorems 1 and 2 show that the system is well-posed mathematically and epidemiologically. This is a sufficient condition for the system to be studied and analyzed [25,26]. □

### 3. Stability Analysis of the Disease-Free Equilibrium Point

#### 3.1. Disease-Free Equilibrium Point

The model system admits only one disease-free equilibrium point,  $E_0 = (S_h^0, T_h^0, V_h^0, 0, R_h^0, S_m^0, 0)$ , which is the steady-state solution to  $F(x) = 0$ , when there is no dengue infection in the human population and no mosquito is infected with the virus. Here,  $F(x)$  is the column vector of the right-hand side of the model system,  $x$  are the state variables. Also, we have that

$$S_h^0 = \frac{\pi_h(\phi + \mu_h)}{\phi(\rho + \mu_h) + \mu_h(\theta + \rho + \mu_h)}, T_h^0 = \frac{\pi_h\rho(\phi + \mu_h)}{(v_1 + \mu_h)(\phi(\rho + \mu_h) + \mu_h(\theta + \rho + \mu_h))}, V_h^0 = \frac{\pi_h\theta}{\phi(\rho + \mu_h) + \mu_h(\theta + \rho + \mu_h)},$$

$$R_h^0 = \frac{\pi_h\rho v_1(\phi + \mu_h)}{\mu_h(v_1 + \mu_h)(\phi(\rho + \mu_h) + \mu_h(\theta + \rho + \mu_h))}, S_D^0 = \frac{\pi_m}{\mu_m + \mu_c}.$$

#### 3.2. The Control Reproduction Number

The relevant equations in the determination of the control reproduction number are the equations for the infected compartments. In this case, we have only two equations,

$$\frac{dI_h}{dt} = \lambda_1 S_h + \lambda_2 V_h - (v_2 + \mu_h + \mu_D + \kappa)I_h,$$

$$\frac{dI_D}{dt} = \lambda_3 S_m - (\mu_m + \mu_c)I_D.$$

Application of the Next-Generation Matrix method gives the control reproduction number,  $\mathcal{R}_0$  as the spectral radius of the of the Next-Generation Matrix  $FV^{-1}$ . In this work, the matrices  $F$  and  $V$  are the Jacobian matrices of  $\begin{pmatrix} \lambda_1 S_h + \lambda_2 V_h \\ \lambda_3 S_D \end{pmatrix}$  and  $\begin{pmatrix} (v_2 + \mu_h + \mu_D + \kappa)I_h \\ (\mu_m + \mu_c)I_D \end{pmatrix}$ , respectively, evaluated at the dengue-free equilibrium point. Hence, we have

$$F = \begin{pmatrix} 0 & a_1 \\ a_2 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} v_2 + \mu_h + \mu_D + \kappa & 0 \\ 0 & \mu_m + \mu_c \end{pmatrix},$$

where  $a_1 = \frac{\alpha\beta_1(1-c_1)(1-c_2)S_h^0}{N_h^0} + \frac{\alpha\beta_2(1-c_1)(1-c_2)V_h^0}{N_h^0}$ ,  $a_2 = \frac{\alpha\beta_3(1-c_1)(1-c_2)S_D^0}{N_h^0}$ .

The eigenvalues of  $FV^{-1}$  are  $\pm \sqrt{\frac{a_1 a_2}{(\mu_m + \mu_c)(v_2 + \mu_h + \mu_D + \kappa)}}$ . Hence, the control reproduction number of dengue fever in this model is  $\mathcal{R}_0 = \sqrt{\frac{a_1 a_2}{(\mu_m + \mu_c)(v_2 + \mu_h + \mu_D + \kappa)}}$ . This represents the average number of persons that can be infected with dengue by a single infectious person throughout their infectious lifetime when present in a purely susceptible population.

In the control reproduction number,  $\mathcal{R}_0$ , if there are no controls in the system, that is,  $c_1 = c_2 = \rho = \kappa = \theta = \mu_c = v_1 = v_2 = 0$ , then  $\mathcal{R}_0$  becomes

$$\mathcal{R}_0^* = \sqrt{\frac{a_1^* a_2^*}{\mu_m(\mu_h + \mu_D)}}$$

where  $a_1^* = \frac{\alpha\beta_1 S_h^0}{N_h^0}$  and  $a_2^* = \frac{\alpha\beta_3 S_D^0}{N_h^0}$ .

The value of  $\mathcal{R}_0$  using the parameter values in Table 1 is 0.05798145 while the value of  $\mathcal{R}_0^*$  is 0.7741646 using the same parameter values. This shows that  $\mathcal{R}_0 < \mathcal{R}_0^*$ , a proof that the proposed controls significantly reduced the average number of persons that can be infected by one Dengue infectious human in an entirely susceptible population.

#### 3.3. Local Stability Analysis of the Disease-Free Equilibrium Point

Establishing local stability of the disease-free equilibrium point is essential to show whether the disease will die out or not when the control reproduction number is less than one, depending on the initial sizes of the infected population.

**Theorem 3.** *The disease-free equilibrium is locally asymptotically stable if  $\mathcal{R}_0 < 1$ , and unstable, otherwise.*

**Proof:** At the disease-free equilibrium, the right-hand side of the model system admits a Jacobian matrix of the form

$$J(E_0) = \begin{pmatrix} -(\theta + \rho + \mu_h) & 0 & \phi & 0 & 0 & 0 & -b_1 \\ \rho & -(v_1 + \mu_h) & 0 & 0 & 0 & 0 & 0 \\ \theta & 0 & -(\phi + \mu_h) & 0 & 0 & 0 & -b_2 \\ 0 & 0 & 0 & -(v_2 + \mu_h + \mu_D + \kappa) & 0 & 0 & b_3 \\ 0 & v_1 & 0 & v_2 & -\mu_h & 0 & 0 \\ 0 & 0 & 0 & -b_4 & 0 & -(\mu_m + \mu_c) & 0 \\ 0 & 0 & 0 & b_4 & 0 & 0 & -(\mu_m + \mu_c) \end{pmatrix},$$

where  $b_1 = \frac{\alpha_1 \beta_1 (1-c_1)(1-c_2) S_h^0}{N_h^0}$ ,  $b_2 = \frac{\alpha_1 \beta_2 (1-c_1)(1-c_2) V_h^0}{N_h^0}$ ,  $b_3 = \frac{\alpha_1 \beta_1 (1-c_1)(1-c_2) S_h^0}{N_h^0} + \frac{\alpha_1 \beta_2 (1-c_1)(1-c_2) V_h^0}{N_h^0}$ ,  $b_4 = \frac{\alpha_1 \beta_3 (1-c_1)(1-c_2) S_D^0}{N_h^0}$ . By considering that the matrix  $J$  has known eigenvalues, we obtain the submatrix

$$J_1 = \begin{pmatrix} -(\theta + \rho + \mu_h) & \phi & 0 & -b_1 \\ \theta & -(\phi + \mu_h) & 0 & -b_2 \\ 0 & 0 & -(v_2 + \mu_h + \mu_D + \kappa) & b_3 \\ 0 & 0 & b_4 & -(\mu_m + \mu_c) \end{pmatrix},$$

which can be written in the block-triangular form  $J_1^B = \begin{pmatrix} A & C \\ \mathbf{0} & B \end{pmatrix}$ , where

$A = \begin{pmatrix} -(\theta + \rho + \mu_h) & \phi \\ \theta & -(\phi + \mu_h) \end{pmatrix}$ ,  $\mathbf{0} = \begin{pmatrix} 0 & 0 \\ 0 & 0 \end{pmatrix}$ ,  $B = \begin{pmatrix} -(v_2 + \mu_h + \mu_D + \kappa) & b_3 \\ b_4 & -(\mu_m + \mu_c) \end{pmatrix}$  and  $C = \begin{pmatrix} 0 & -b_1 \\ 0 & -b_2 \end{pmatrix}$ . Since the matrix  $J_1^B$  is in upper-triangular form, its eigenvalues are the eigenvalues of the diagonal block matrices,  $A$  and  $B$ . The eigenvalues,  $\lambda$  of  $A$  satisfies the characteristic equation

$$\lambda^2 + ((\theta + \rho + \mu_h) + (\phi + \mu_h))\lambda + (\theta\mu_h + \rho\phi + \rho\mu_h + \phi\mu_h + \mu_h^2) = 0. \tag{4}$$

The solution to Equation (4) are all negative since there is no change of sign in the coefficients. Hence, all the eigenvalues of  $A$  are negative. For the matrix,  $B$ , its eigenvalues satisfy the characteristic equation

$$\lambda^2 + (v_2 + \mu_h + \mu_D + \kappa + \mu_m + \mu_c)\lambda + (v_2 + \mu_h + \mu_D + \kappa)(\mu_m + \mu_c)(1 - \mathcal{R}_0^2) = 0, \tag{5}$$

whose solution will all be negative if  $\mathcal{R}_0 < 1$ . This shows that the disease-free equilibrium will be locally asymptotically stable if  $\mathcal{R}_0 < 1$ . □

### 3.4. Bifurcation Analysis

Here, we use the Center Manifold Theorem to determine the possibility of bifurcation in the model at the critical points. That is the investigate the existence and stability of equilibrium points bifurcating from the disease-free equilibrium at  $\mathcal{R}_0 = 1$ . If we choose  $\alpha_1$  to be the bifurcation parameter in the model, then we must have  $\alpha_1^* = \frac{N_h^0}{(1-c_1)(1-c_2)} \sqrt{\frac{(\mu_m + \mu_c)(v_2 + \mu_h + \mu_D + \kappa)}{\beta_1 \beta_3 S_h^0 S_D^0 + \beta_2 \beta_3 V_h^0 S_D^0}}$  so that  $\mathcal{R}_0 = 1$ . Let  $J(\alpha_1^*; E_0)$  denote Jacobian matrix evaluated at  $E_0$  with  $\alpha_1 = \alpha_1^*$ . With this, the model, the model system has a hyperbolic equilibrium point. This means that the Jacobian matrix  $J(\alpha_1^*; E_0)$  has a simple eigenvalue with zero real part, and the remaining six eigenvalues have negative real part. This is due to the loss of stability of  $E_0$  through transcritical bifurcation. Hence, the Center Manifold Theorem can be applied to analyse the dynamics of the model (1) near the bifurcation parameter,  $\alpha_1 = \alpha_1^*$ . Using the method of Castillo Chavez and Song [28] requires finding the right and left eigenvectors of the Jacobian matrix  $J(\alpha_1^*; E_0)$ .

$$J(\alpha_1^*; E_0) = \begin{pmatrix} -k_1 & 0 & \phi & 0 & 0 & 0 & -b_1 \\ \rho & -k_2 & 0 & 0 & 0 & 0 & 0 \\ \theta & 0 & -k_3 & 0 & 0 & 0 & -b_2 \\ 0 & 0 & 0 & -k_4 & 0 & 0 & b_3^* \\ 0 & v_1 & 0 & v_2 & -\mu_h & 0 & 0 \\ 0 & 0 & 0 & -b_4 & 0 & -(\mu_m + \mu_c) & 0 \\ 0 & 0 & 0 & b_4 & 0 & 0 & -(\mu_m + \mu_c) \end{pmatrix},$$

where in this case,  $b_1 = \frac{\alpha_1^* \beta_1 (1-c_1)(1-c_2) S_h^0}{N_h^0}$ ,  $b_2 = \frac{\alpha_1^* \beta_2 (1-c_1)(1-c_2) V_h^0}{N_h^0}$ ,  $b_3 = \frac{\alpha_1^* \beta_1 (1-c_1)(1-c_2) S_h^0}{N_h^0} + \frac{\alpha_1^* \beta_2 (1-c_1)(1-c_2) V_h^0}{N_h^0}$ ,

$k_1 = \theta + \rho + \mu_h, k_2 = v_1 + \mu_h, k_3 = \phi + \mu_h, k_4 = v_2 + \mu_h + \mu_D + \kappa$ .

The right eigenvector

$\tilde{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$  of  $J(\alpha_1^*; E_0)$  satisfies the system

$$\begin{aligned}
 -w_1 + \phi w_3 - b_1 w_7 &= 0, \\
 \rho w_1 - k_2 w_2 &= 0, \\
 \theta w_1 - k_3 w_3 - b_2 w_7 &= 0, \\
 -k_4 w_4 + b_3 w_7 &= 0, \\
 v_1 w_2 + v_2 w_2 - \mu_h w_5 &= 0, \\
 -b_4 w_4 - (\mu_m + \mu_c) w_6 &= 0, \\
 b_4 w_4 - (\mu_m + \mu_c) w_7 &= 0.
 \end{aligned}$$

So that in terms of the component,  $w_7$ , we obtain other components as

$$\begin{aligned}
 w_1 &= \frac{(\phi b_2 - b_1 k_3)}{k_1 k_3 + \phi \theta} w_7, \quad w_2 = \frac{\rho(\phi b_2 - b_1 k_3)}{k_2(k_1 k_3 + \phi \theta)} w_7, \quad w_3 = \left( \frac{k_1 b_2 + \theta b_1}{k_1 k_2 + \phi \theta} \right) w_7, \quad w_4 = \frac{b_3}{k_4} w_7, \\
 w_5 &= \left( \frac{v_1 \rho(\phi b_2 - b_1 k_3)}{\mu_h k_2(k_1 k_3 + \phi \theta)} + \frac{v_2 b_2}{\mu_h a_4} \right) w_7, \quad w_6 = -\frac{b_3 b_4}{(\mu_m + \mu_c) k_4} w_7, \quad w_7 > 0.
 \end{aligned}$$

On the other hand, the left eigenvector  $\tilde{v} = (v_2, v_3, v_4, v_5, v_6, v_7)^T$  satisfies the system

$$\begin{aligned}
 -k_1 v_1 + \rho v_2 &= 0, \\
 -k_2 v_2 + v_1 v_5 &= 0, \\
 \phi v_1 - k_3 v_3 &= 0, \\
 -k_4 v_4 + v_2 v_5 - b_4 v_6 + b_4 v_7 &= 0, \\
 -\mu_h v_5 &= 0, \\
 -(\mu_m + \mu_c) v_6 &= 0, \\
 -b_1 v_1 - b_2 v_3 + b_3 v_4 - (\mu_m + \mu_c) v_7 &= 0.
 \end{aligned}$$

The only non-zero components of the left eigenvector, are  $v_4$  and  $v_7$ , since they are the components that corresponds to the infected states in the model, where  $v_4$  and  $v_7$  are related by  $v_7 = \frac{b_3}{(\mu_m + \mu_c)} v_4$ , with  $v_4 > 0$ . Therefore, the bifurcation variables  $a, b$  in [1] are

$$\begin{aligned}
 a &= v_4 \sum_{i,j=1}^7 w_i w_j \frac{\partial^2 f_4(0,0)}{\partial x_i \partial x_j} + v_7 \sum_{i,j=1}^7 w_i w_j \frac{\partial^2 f_7(0,0)}{\partial x_i \partial x_j}, \\
 b &= v_4 \sum_{i=1}^7 w_i \frac{\partial^2 f_4(0,0)}{\partial x_i \partial \alpha_1^*} + v_7 \sum_{i=1}^7 w_i \frac{\partial^2 f_7(0,0)}{\partial x_i \partial \alpha_1^*},
 \end{aligned}$$

where  $f_4 = \lambda_1 S_h + \lambda_2 V_h - (v_1 + \mu_h + \mu_D + \kappa) I_h$ ,  $f_7 = \lambda_3 S_m - (\mu_m + \mu_c) I_m$ ,  $x_1 = S_h, x_2 = T_h, x_3 = V_h, x_4 = I_h, x_5 = R_h, x_6 = S_m, x_7 = I_m$ .

For the variable,  $a$ , the nonzero second order partial derivatives of  $f_4$  are  $\frac{\partial^2 f_4(0,0)}{\partial x_i \partial x_j}, i = 1, 2, \dots, 5$ , with

$$\begin{aligned}
 \frac{\partial^2 f_4(0,0)}{\partial x_7 \partial x_1} &= \frac{\partial^2 f_4(0,0)}{\partial x_1 \partial x_7} = \frac{\alpha_1 \beta_1 (1 - c_1)(1 - c_2)(N_h^0 - S_h^0)}{(N_h^0)^2} - \frac{\alpha_1 \beta_2 (1 - c_1)(1 - c_2) V_h^0}{(N_h^0)^2}, \\
 \frac{\partial^2 f_4(0,0)}{\partial x_7 \partial x_2} &= \frac{\partial^2 f_4(0,0)}{\partial x_2 \partial x_7} = -\frac{\alpha_1 \beta_1 (1 - c_1)(1 - c_2) S_h^0}{(N_h^0)^2} - \frac{\alpha_1 \beta_2 (1 - c_1)(1 - c_2) V_h^0}{(N_h^0)^2}, \\
 \frac{\partial^2 f_4(0,0)}{\partial x_7 \partial x_3} &= \frac{\partial^2 f_4(0,0)}{\partial x_3 \partial x_7} = \frac{\alpha_1 \beta_2 (1 - c_1)(1 - c_2)(N_h^0 - V_h^0)}{(N_h^0)^2} - \frac{\alpha_1 \beta_1 (1 - c_1)(1 - c_2) S_h^0}{(N_h^0)^2}, \\
 \frac{\partial^2 f_4(0,0)}{\partial x_7 \partial x_4} &= \frac{\partial^2 f_4(0,0)}{\partial x_4 \partial x_7} = -\frac{\alpha_1 \beta_1 (1 - c_1)(1 - c_2) S_h^0}{(N_h^0)^2} - \frac{\alpha_1 \beta_2 (1 - c_1)(1 - c_2) V_h^0}{(N_h^0)^2}, \\
 \frac{\partial^2 f_4(0,0)}{\partial x_7 \partial x_5} &= \frac{\partial^2 f_4(0,0)}{\partial x_5 \partial x_7} = -\frac{\alpha_1 \beta_1 (1 - c_1)(1 - c_2) S_h^0}{(N_h^0)^2} - \frac{\alpha_1 \beta_2 (1 - c_1)(1 - c_2) V_h^0}{(N_h^0)^2},
 \end{aligned}$$

whereas for  $f_7$ , we have  $\frac{\partial^2 f_7(0,0)}{\partial x_i \partial x_4}, i = 1, 2, \dots, 6$  as

$$\begin{aligned} \frac{\partial^2 f_7(0,0)}{\partial x_4 \partial x_1} &= \frac{\partial^2 f_7(0,0)}{\partial x_1 \partial x_4} = -\frac{\alpha_1 \beta_3 (1 - c_1)(1 - c_2) S_D^0}{(N_h^0)^2}, \\ \frac{\partial^2 f_7(0,0)}{\partial x_4 \partial x_2} &= \frac{\partial^2 f_7(0,0)}{\partial x_2 \partial x_4} = -\frac{\alpha_1 \beta_3 (1 - c_1)(1 - c_2) S_D^0}{(N_h^0)^2}, \\ \frac{\partial^2 f_7(0,0)}{\partial x_4 \partial x_3} &= \frac{\partial^2 f_7(0,0)}{\partial x_3 \partial x_4} = -\frac{\alpha_1 \beta_3 (1 - c_1)(1 - c_2) S_D^0}{(N_h^0)^2}, \\ \frac{\partial^2 f_7(0,0)}{\partial x_4^2} &= -2 \frac{\alpha_1 \beta_3 (1 - c_1)(1 - c_2) S_D^0}{(N_h^0)^2}, \\ \frac{\partial^2 f_7(0,0)}{\partial x_4 \partial x_5} &= \frac{\partial^2 f_7(0,0)}{\partial x_5 \partial x_4} = -\frac{\alpha_1 \beta_3 (1 - c_1)(1 - c_2) S_D^0}{(N_h^0)^2}, \\ \frac{\partial^2 f_7(0,0)}{\partial x_4 \partial x_6} &= \frac{\partial^2 f_7(0,0)}{\partial x_6 \partial x_4} = -\frac{\alpha_1 \beta_3 (1 - c_1)(1 - c_2) S_D^0}{N_h^0}. \end{aligned}$$

On the other hand, for the variable  $b$ , the nonzero second order partial derivatives of  $f_4$  and  $f_7$  are  $\frac{\partial^2 f_4(0,0)}{\partial x_7 \partial \alpha_1^*} = \frac{\beta_1(1-c_1)(1-c_2)S_h^0}{N_h^0} + \frac{\beta_2(1-c_1)(1-c_2)V_h^0}{N_h^0} > 0$ ,  $\frac{\partial^2 f_7(0,0)}{\partial x_4 \partial \alpha_1^*} = \frac{\beta_3(1-c_1)(1-c_2)S_D^0}{N_h^0} > 0$ . So that the bifurcation variables become

$$\begin{aligned} a &= 2v_4w_7 \left[ w_1 \frac{\partial^2 f_4(0,0)}{\partial x_1 \partial x_7} + w_2 \frac{\partial^2 f_4(0,0)}{\partial x_2 \partial x_7} + w_3 \frac{\partial^2 f_4(0,0)}{\partial x_3 \partial x_7} + w_4 \frac{\partial^2 f_4(0,0)}{\partial x_4 \partial x_7} + w_5 \frac{\partial^2 f_4(0,0)}{\partial x_5 \partial x_7} \right] \\ &\quad + v_7w_4 \left[ 2w_1 \frac{\partial^2 f_7(0,0)}{\partial x_1 \partial x_4} + 2w_2 \frac{\partial^2 f_7(0,0)}{\partial x_2 \partial x_4} + 2w_3 \frac{\partial^2 f_7(0,0)}{\partial x_3 \partial x_4} + w_4 \frac{\partial^2 f_7(0,0)}{\partial x_4^2} + 2w_5 \frac{\partial^2 f_7(0,0)}{\partial x_5 \partial x_4} \right. \\ &\quad \left. + 2w_6 \frac{\partial^2 f_7(0,0)}{\partial x_6 \partial x_4} \right] \\ b &= v_4w_7 \frac{\partial^2 f_4(0,0)}{\partial x_7 \partial \alpha_1^*} + v_7w_4 \frac{\partial^2 f_7(0,0)}{\partial x_4 \partial \alpha_1^*} \end{aligned}$$

All the partial derivatives  $\frac{\partial^2 f_4(0,0)}{\partial x_i \partial x_4}$ ,  $i = 1, 2, \dots, 6$  are negative except  $\frac{\partial^2 f_4(0,0)}{\partial x_1 \partial x_7}$  and  $\frac{\partial^2 f_4(0,0)}{\partial x_3 \partial x_7}$  which may be positive or negative so that the quantities  $w_1 \frac{\partial^2 f_4(0,0)}{\partial x_1 \partial x_7}$  and  $w_3 \frac{\partial^2 f_4(0,0)}{\partial x_3 \partial x_7}$  are either positive or negative, whereas other quantities in the bifurcation parameter are negative. Hence, we can conclude that  $a < 0$ . On the other  $b > 0$  since  $\frac{\partial^2 f_4(0,0)}{\partial x_7 \partial \alpha_1^*} > 0$ , and  $\frac{\partial^2 f_7(0,0)}{\partial x_4 \partial \alpha_1^*} > 0$ . This shows that the model exhibits forward transcritical bifurcation, indicating that the model has a unique stable endemic equilibrium when  $\mathcal{R}_0 > 1$ . Since no endemic equilibrium exists when  $\mathcal{R}_0 < 1$ , we conclude that the disease-free equilibrium is globally stable if  $\mathcal{R}_0 < 1$ .

#### 4. Sensitivity Analysis

The sensitivity analysis is carried out by means of the normalized forward sensitivity index approach [25]. Given that the control reproduction number of the Equation (1) is denoted by  $\mathcal{R}_0$  then, the sensitivity index of any parameter  $q$  in  $\mathcal{R}_0$  is given by

$$S_q^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial q} \times \frac{q}{\mathcal{R}_0}.$$

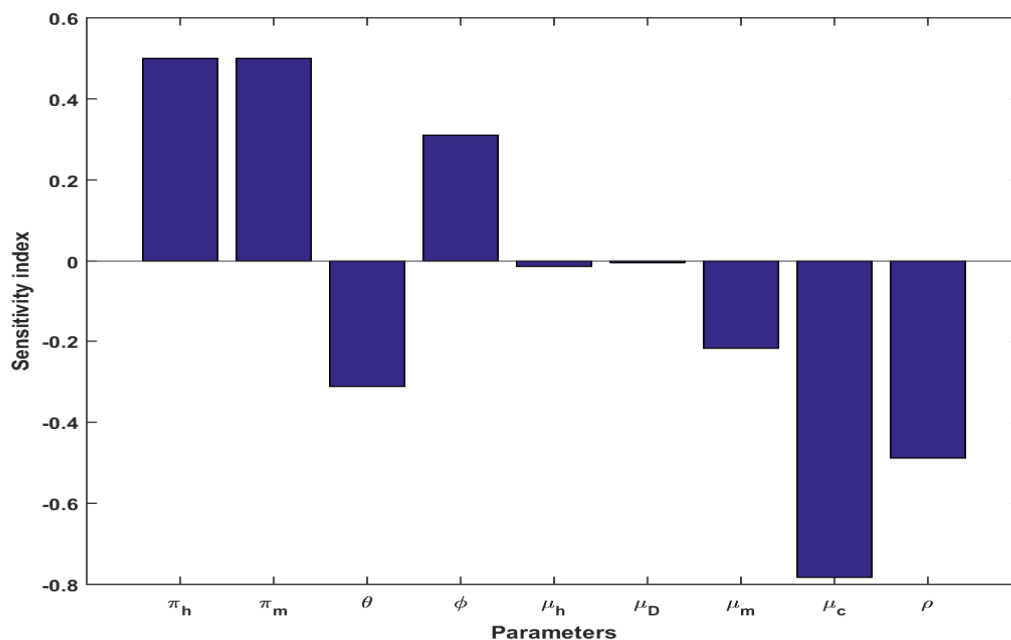
Sensitivity analysis helps to understand the parameters that causes the disease to be endemic so as to inform intervention measures. Parameters with positive sensitivity index increase the endemicity of the disease and should be reduced while those with negative sensitivity index reduce the endemicity of the disease and should be increased. The sensitivity analyses are presented in Figures 2–7 and Table 2. The sensitivity analysis shown in Table 2 and Figure 2 showed that the probabilities of infection,  $\beta_1, \beta_2$  and  $\beta_3$ , mosquito biting rate,  $\alpha$  as well as rate of recruitment of humans and mosquitoes,  $\pi_h$  and  $\pi_D$  into the same system increases the endemicity of Dengue fever. This is further highlighted by the 3-D surface plots in Figures 4 and 5. This shows that the values of these parameters must be reduced to effectively control the spread of the disease. epidemiologically, it means that for Dengue fever to be controlled effectively in the system, efforts should be made to ensure that humans and mosquitoes are not recruited into the system to co-exist. This informs the use of insecticide spray and fogging to

control the population of mosquitoes. Reducing the population of mosquitoes will also cause the biting rate of mosquitoes and probability of infection to consequently reduce too.

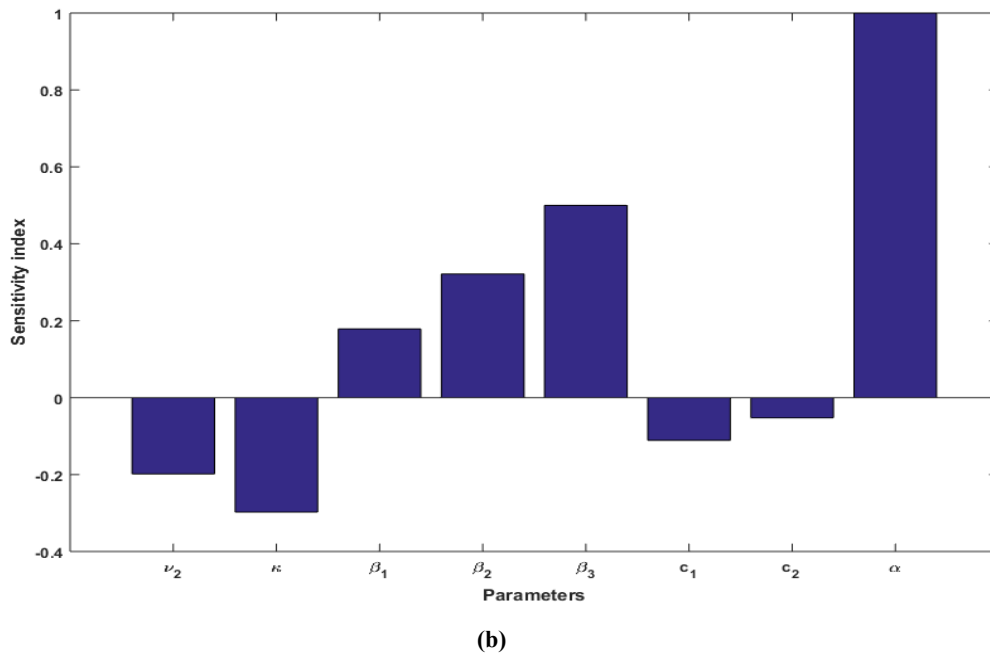
The sensitivities indices for the control parameters introduced into the system such as rate of vaccination,  $\theta$ , preventive measures by use of mosquito treated bed-nets and repellent,  $c_1$  and  $c_2$ , rate of testing and isolating infectious humans,  $\rho$  and  $\kappa$  are all negative showing that these parameters does not increase the endemicity of the disease but rather reduce it. Hence, they must be increased in order to reduce the endemicity of the disease. This means that improving the rate of vaccination, use of preventive measures, testing and isolation of infectious humans will definitely help reduce the spread of Dengue fever in the system. This is also further highlighted by the 3D surface plots in Figures 3, 6 and 7.

**Table 2.** Sensitivity indices for parameters of  $\mathcal{R}_0$ .

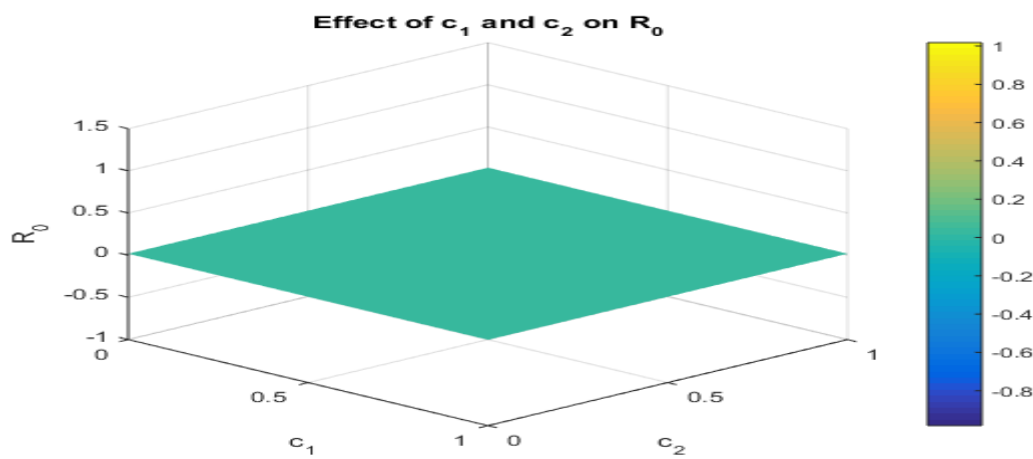
Parameters	Values	Sensitivity Indices
$\pi_h$	100	0.5
$\pi_m$	500	0.5
$\phi$	0.01	0.3104
$\theta$	0.05	-0.3116
$\mu_h$	0.00004	-0.0130
$\mu_D$	0.002	-0.0040
$\mu_m$	0.0556	-0.2175
$\mu_c$	0.2	-0.7825
$\rho$	0.01	-0.4883
$\nu_2$	0.1	-0.1983
$\kappa$	0.15	-0.2976
$\beta_1$	0.3632	0.1786
$\beta_2$	0.1312	0.3214
$\beta_3$	0.3252	0.5
$c_1$	0.1	-0.1111
$c_2$	0.05	-0.0526
$\alpha$	0.4	1



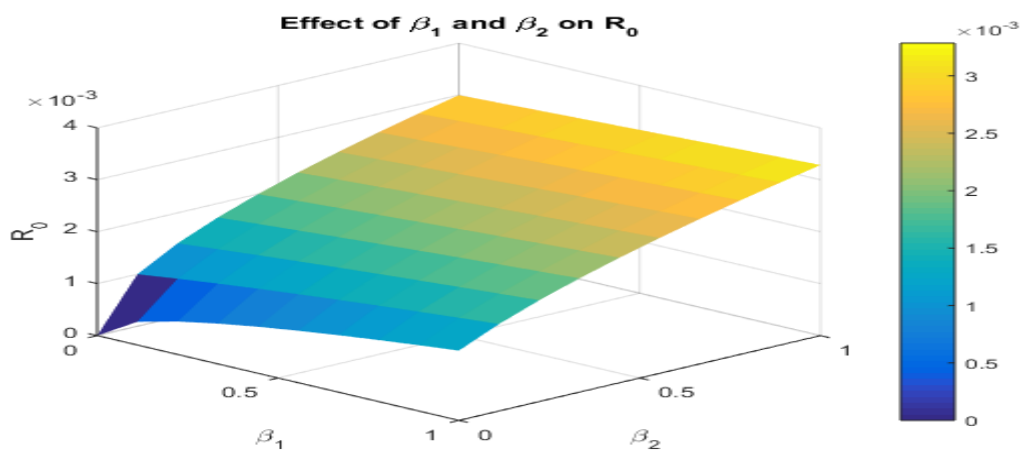
(a)



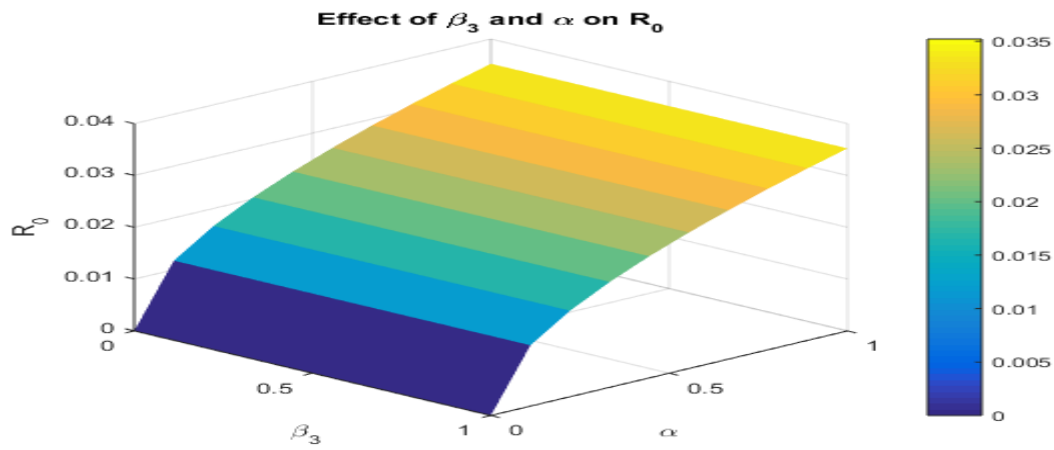
**Figure 2.** (a) Sensitivity plots for parameters of  $\mathcal{R}_0$ . (b) Sensitivity plots for parameters of  $\mathcal{R}_0$ .



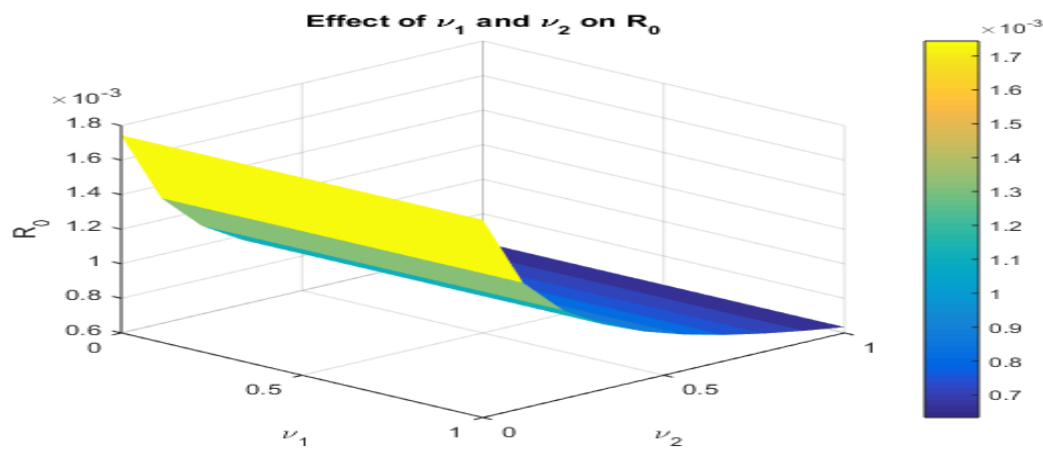
**Figure 3.** Effect of  $c_1$  and  $c_2$  on  $\mathcal{R}_0$ .



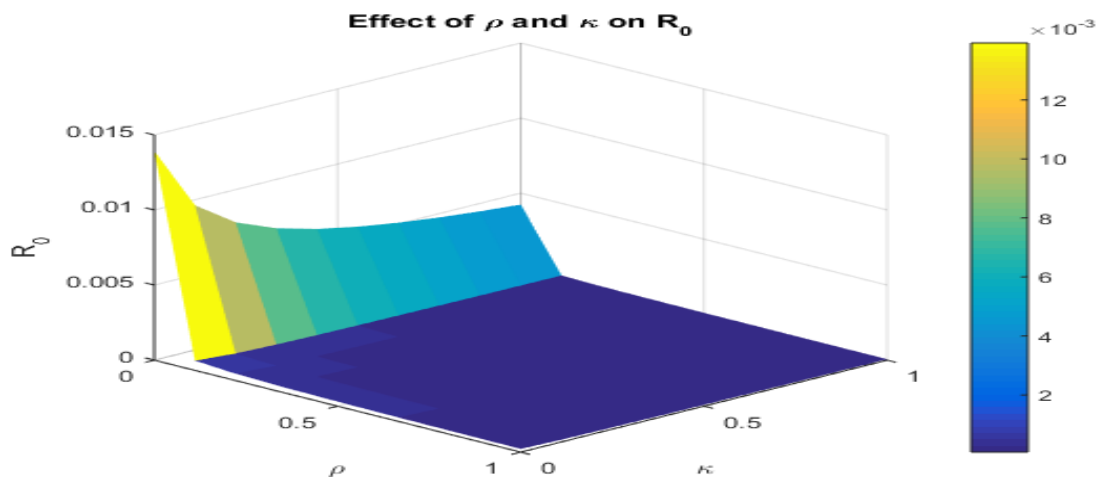
**Figure 4.** Effect of  $\beta_1$  and  $\beta_2$  on  $\mathcal{R}_0$ .



**Figure 5.** Effect of  $\beta_3$  and  $\alpha$  on  $\mathcal{R}_0$ .



**Figure 6.** Effect of  $\nu_1$  and  $\nu_2$  on  $\mathcal{R}_0$ .



**Figure 7.** Effect of  $\rho$  and  $\kappa$  on  $\mathcal{R}_0$ .

### 5. Numerical Simulations

The effects of the various controls incorporated into the system are investigated in this section using the parameter values in Table 1. The numerical simulations are carried out using the in-built ODE programme in MATLAB. The controls whose effects are investigated here include: preventive measures against mosquito bites such as use of mosquito treated ben-nets and mosquito repellent; vaccination; testing and isolating infected humans; use of insecticide spray; and improving rate of recovery of humans.

5.1. *Effect of Preventive Measures on the System*

The preventive measures suggested in this work against mosquito bites are use of mosquito treated bed-nets and mosquito repellent. The result of the simulation is shown in Figures 8–10. In Figure 8, the infectious human population is seen to reduce as the rate at which the preventive measures employed are increased. The reduction in the susceptible population causes a corresponding decrease in the recovered human population and infectious mosquito population as seen in Figures 9 and 10. The recovered human population reduced because as more humans are protected from getting bitten and infected, the number of infectious human population are reduced. The number of infectious mosquito population also reduced since there are fewer infectious humans that can infect the mosquitoes.

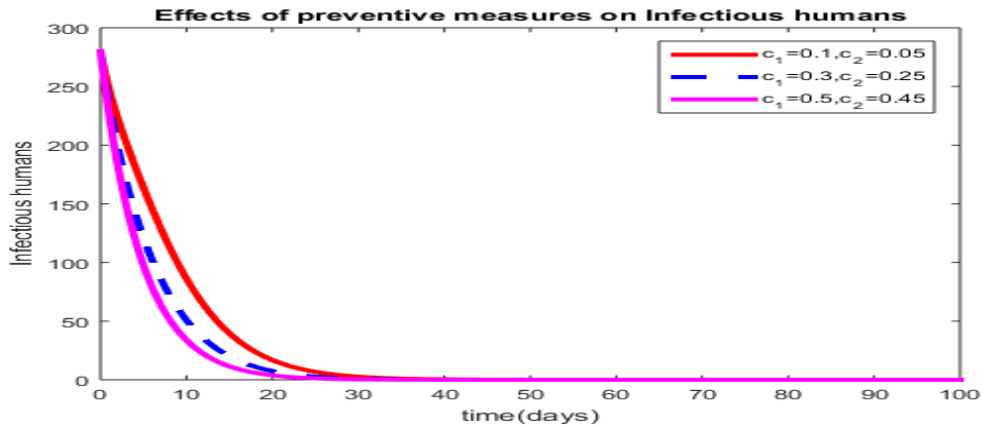


Figure 8. Infectious humans under preventive measures.

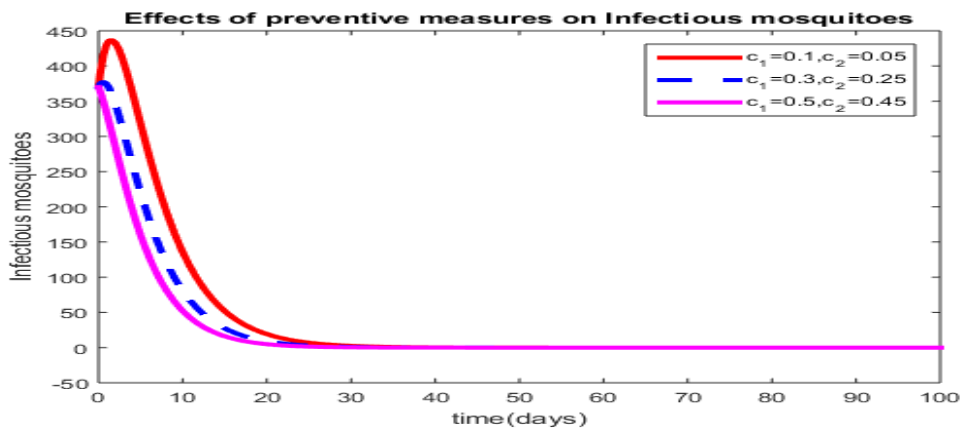


Figure 9. Infectious mosquitoes under preventive measures.

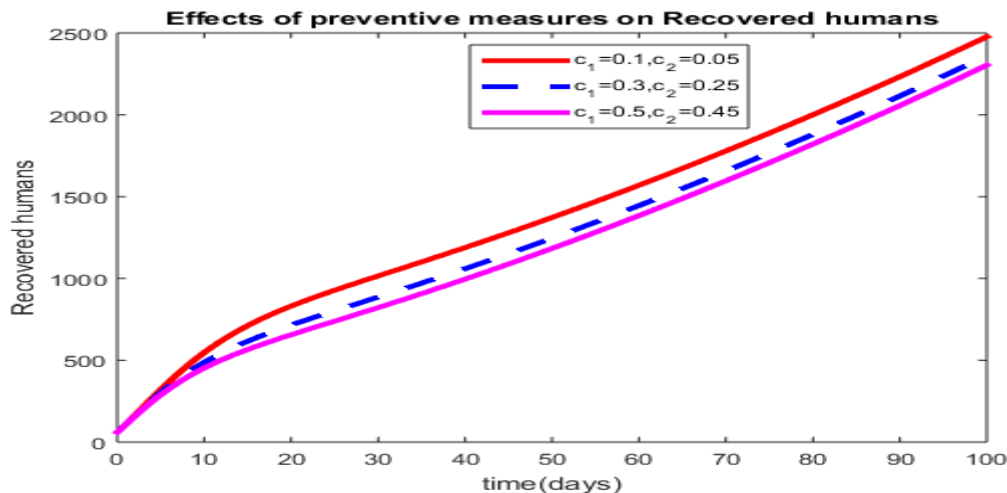
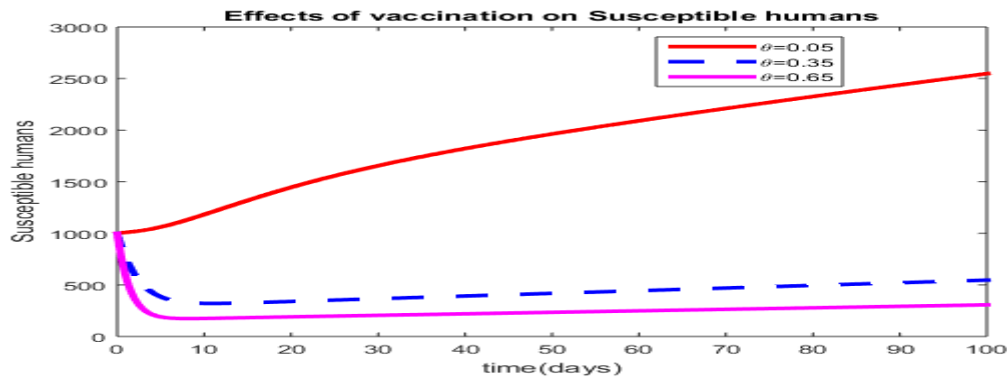


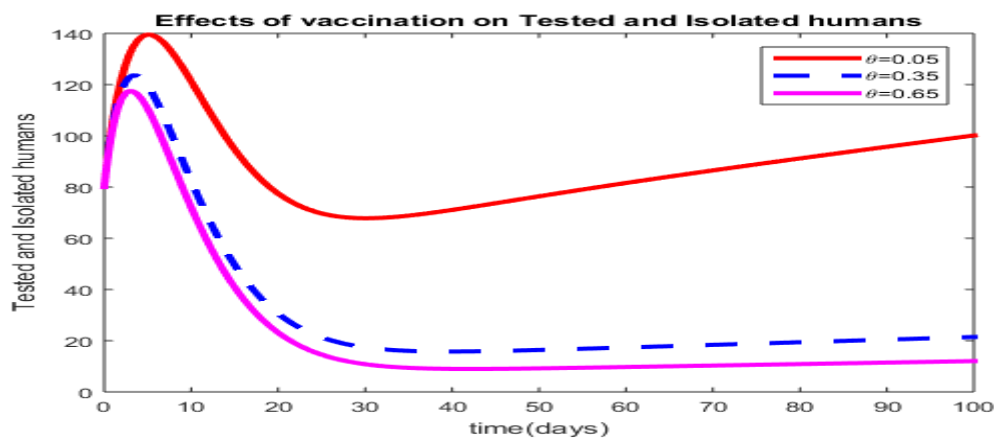
Figure 10. Recovered humans under preventive measures.

### 5.2. Effects of Vaccination on the System

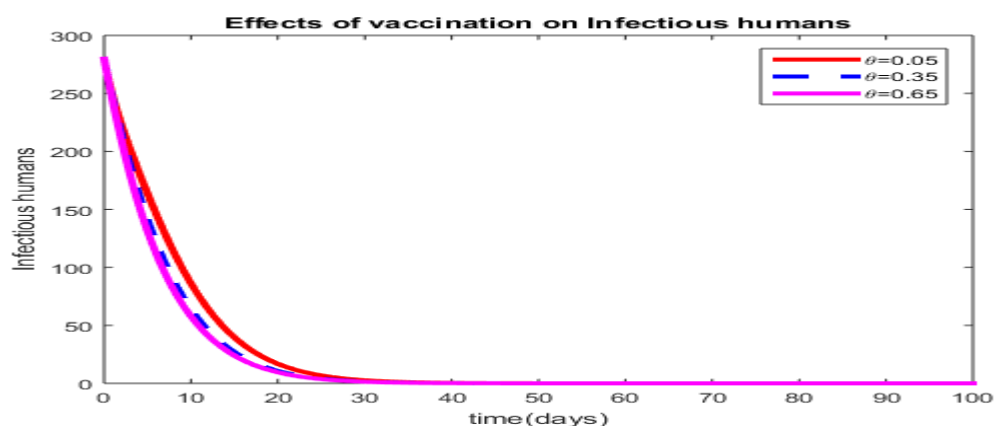
In Figures 11–13, the effect of improving the rate of vaccination in the system is shown. The susceptible human population is reduced as seen in Figure 11 as more humans are protected from the disease. Also, the tested and isolated human population as well as the infectious human population (Figures 12 and 13) reduced as well as there are few humans that can be infected. Vaccination reduces the number of humans who can be infected by providing immunity against the disease in them. The result of the simulation has shown that vaccination plays a vital role in controlling the spread of the disease.



**Figure 11.** Susceptible humans under vaccination.



**Figure 12.** Tested and Isolated humans under vaccination.

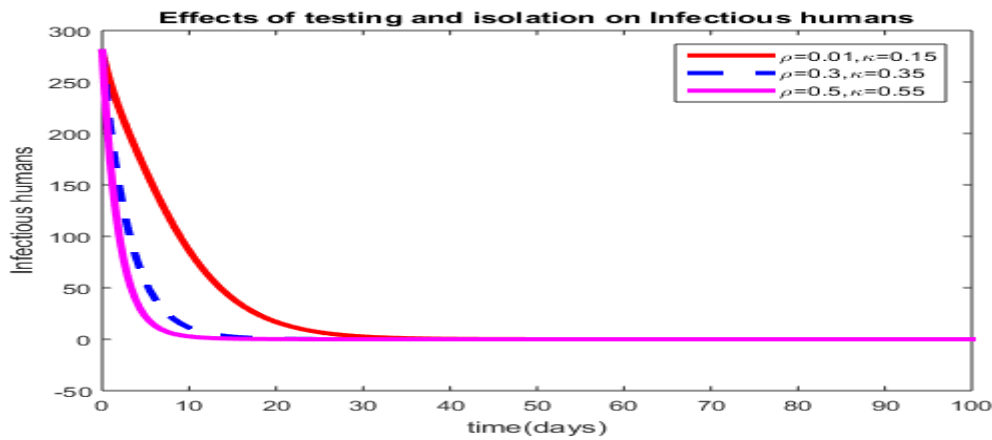


**Figure 13.** Infectious humans under vaccination.

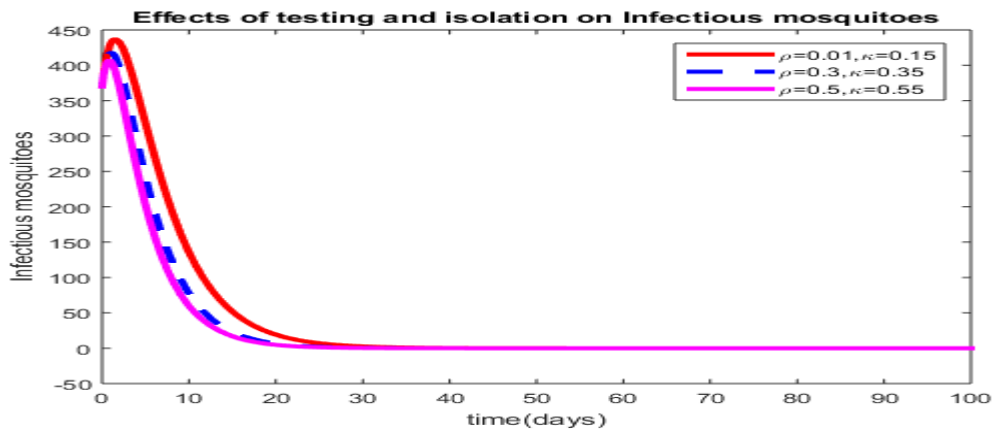
### 5.3. Effects of Testing and Isolation on the System

This work proposed that in a dengue-endemic region, humans should voluntarily go for testing to check their status since most cases are usually asymptomatic. This will ensure early detection of infection, isolation and treatment. The effect of this control is seen in Figures 14–16. In Figure 14, the infectious human population is

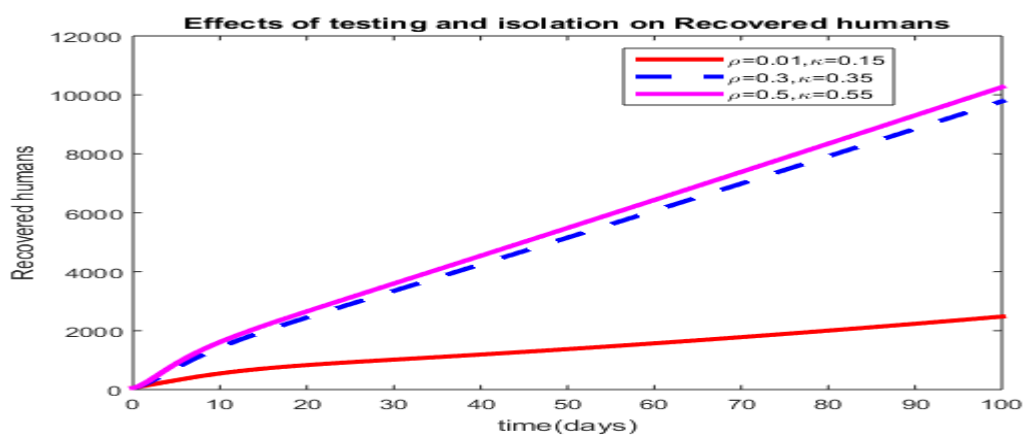
reduced as the rate of the testing and isolating infected humans increases. By isolating this infected group, the number of humans who can contribute to the circulation of the disease is reduced thus ensuring that the disease will be controlled within a short period of time. This also causes a corresponding decrease in the number of infectious mosquitoes as seen in Figure 15. In Figure 16 however, the recovered human population increases as isolation and treatment improves the rate of recovery from the disease.



**Figure 14.** Infectious humans under testing and isolation.



**Figure 15.** Infectious mosquitoes under testing and isolation.



**Figure 16.** Recovered humans under testing and isolation.

#### 5.4. Effects of Use of Insecticide Spray on the System

In Figures 17–19, the effect of using insecticide spray to control the spread of mosquitoes in the system is investigated. The simulation results showed that as more mosquitoes are eradicated due to increasing the rate of application of insecticide spray, the infectious human and mosquito populations reduce too. The reduction in the infectious populations is because there are small number of mosquitoes that can carry on the cycle of infection.

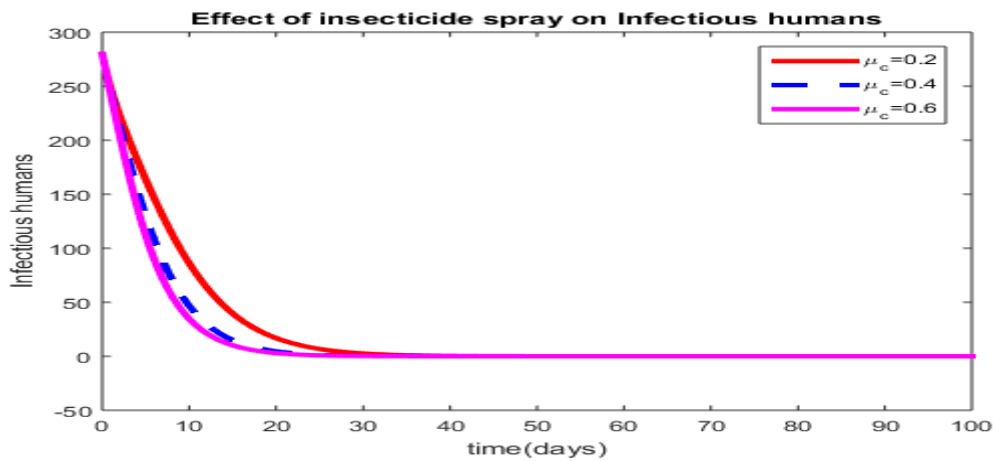


Figure 17. Infectious humans under use of insecticide spray.

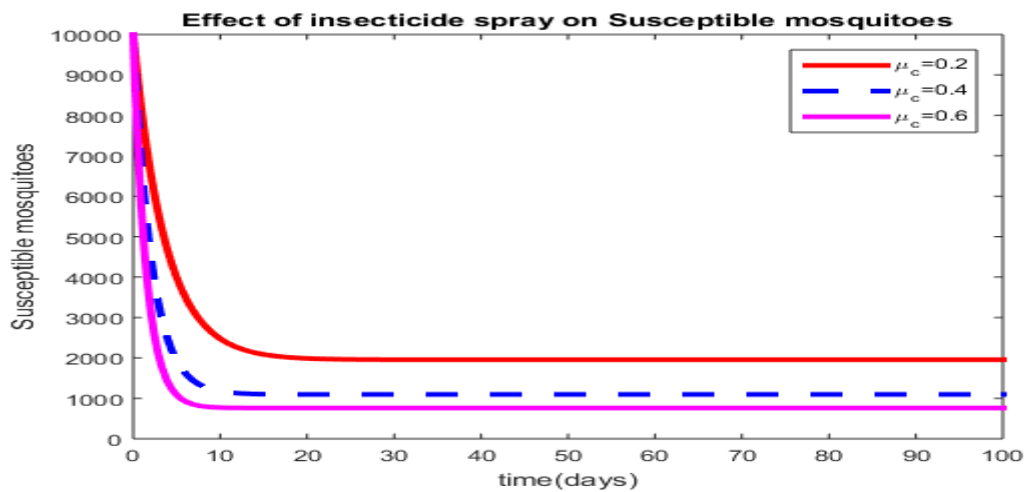


Figure 18. Susceptible mosquitoes under use of insecticide spray.

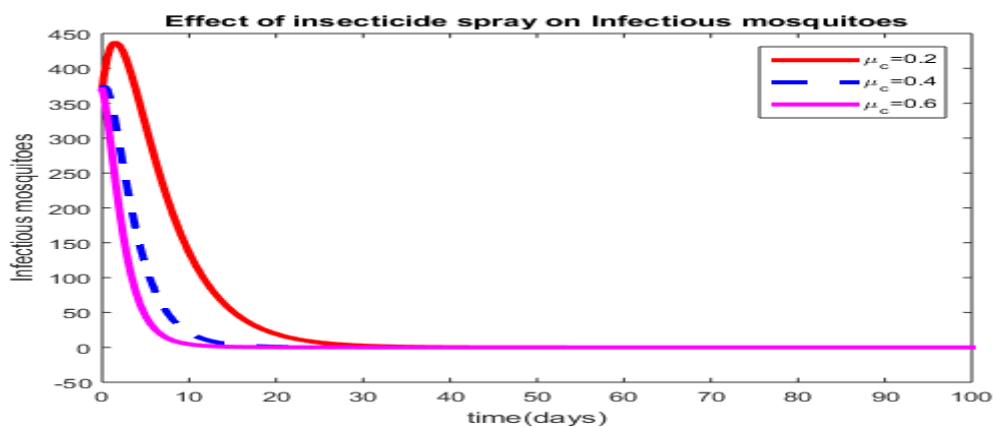


Figure 19. Infectious mosquitoes under use of insecticide spray.

5.5. Effects of Improving Recovery Rate on the System

Implementing steps that will help improve the recovery rate of humans such as treatment also ensured that the infectious population is reduced with time. As shown in Figures 20 and 21, improving the rate of recovery helped to reduce the infectious human and mosquito populations. This means that the faster humans recover from the disease, it reduces the period of infectiousness and the possibility of infecting mosquitoes is reduced. Though there are currently no treatment for Dengue fever, but efforts that will help infectious humans recover faster are encouraged.

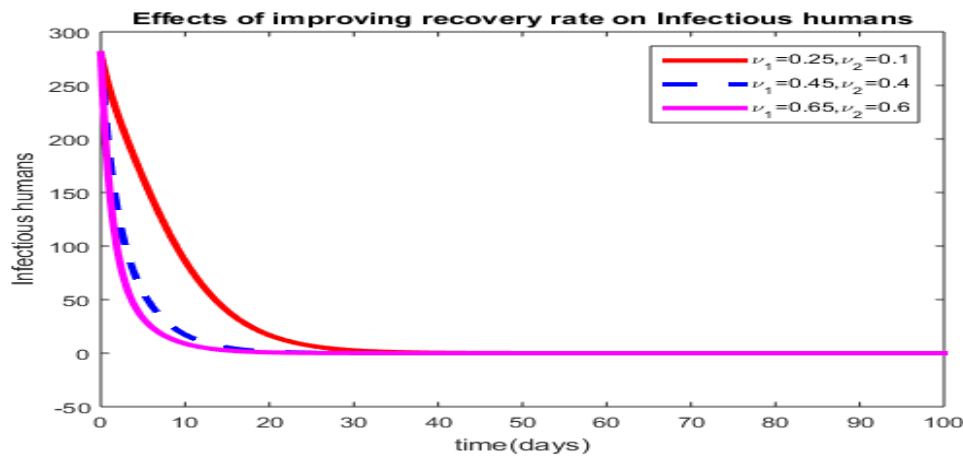


Figure 20. Infectious humans under improved recovery rate.

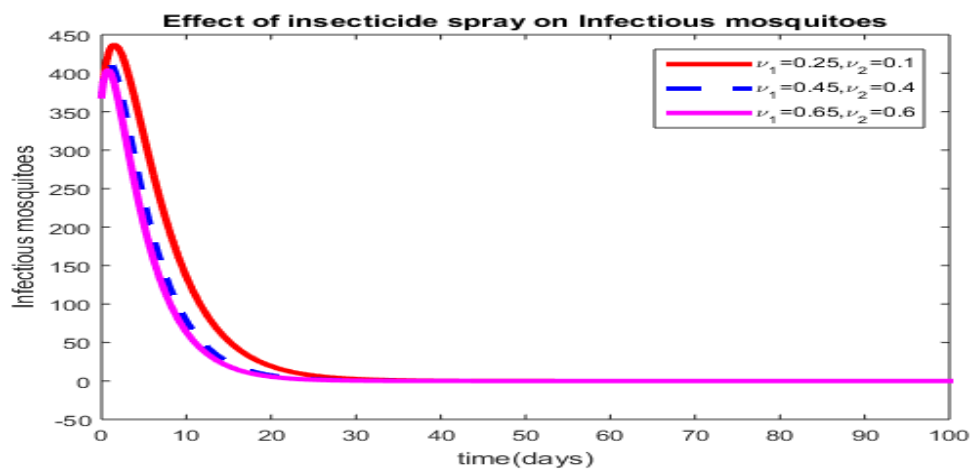


Figure 21. Infectious mosquitoes under improved recovery rate.

## 6. Conclusions

This work presents a new model for the control of Dengue fever in a disease-endemic region. The model incorporates vaccination, preventive measures such as use of mosquito treated bed-nets and mosquito repellent, use of insecticide spray and fogging as control measures. The model also incorporates periodic testing by humans to know their status since most infectious humans with Dengue fever are asymptomatic which is the novelty in this work. The model was shown to be well-posed mathematically and epidemiologically by establishing that the solutions to the system will always remain positive and in the invariant region. The disease-free equilibrium was shown to be both locally and globally asymptotically stable when the control reproduction number is less than one and unstable otherwise. The parameters that affect the endemicity of the disease or not were identified through the normalized sensitivity index approach. It was shown that to control the disease, the biting rate of mosquitoes and probabilities of infection must be reduced by ensuring that the number of mosquitoes which co-exist with humans are reduced drastically. This informs the choice of using insecticide spray and fogging. The sensitivity analysis also showed that improving the rate of vaccination, periodic testing and isolation of infectious humans as well as increasing the preventive steps against mosquito bites will help reduce the spread of the disease. This result is further validated by the numerical simulations carried out which are presented in Figures for easy understanding. All the controls employed were seen to be effective in collectively reducing the spread of the disease in the system. Further works in this regard will consider performing optimal control analysis, data fitting and parameter estimation as well as further possible extensions of the model to incorporate other control measures and fractional derivatives.

## Author Contributions

E.C.D.: conceptualization, methodology, software; writing—original draft preparation; visualization, investigation; M.C.A.: supervision; software, validation; writing—reviewing and editing. All authors have read and agreed to the published version of the manuscript.

## Funding

This research received no external funding.

## Institutional Review Board Statement

Not applicable.

## Informed Consent Statement

Not applicable.

## Data Availability Statement

The study is supported by data derived from published literature, with appropriate citations provided throughout.

## Conflicts of Interest

The author declares no conflict of interest.

## Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

## References

1. Herdicho, F.F.; Fatmawati, F.; Alfiniyah, C.; et al. Optimal control of dengue haemorrhagic fever model by classifying sex in West Java Province, Indonesia. *Sci. Rep.* **2025**, *15*, 17127. <https://doi.org/10.1038/s41598-025-01742-4>.
2. World Health Organization. Fact Sheets Dengue and Severe Dengue. 2022. Available online: <https://www.who.int/news-room/fact-sheets/detail/dengue-and-severe-dengue> (accessed on 19 December 2025).
3. Thongrunkiat, S.; Wasinpiyamongkol, L.; Maneekan, P.; et al. Natural transovarial dengue virus infection rate in both sexes of dark and pale forms of *Aedes aegypti* from an urban area of Bangkok, Thailand. *Southeast Asian J. Trop. Med. Public Health* **2012**, *43*, 1146–1151.
4. Xu, Z.; Zhang, H.; Yang, D.; et al. The Mathematical modelling of the host–virus Interaction in dengue virus infection: A quantitative study. *Viruses* **2024**, *16*, 216.
5. Ahman, Q.O.; Aja, R.O.; Omale, D.; et al. Mathematical modelling of dengue virus transmission: Exploring vector, vertical, and sexual pathways with sensitivity and bifurcation analysis. *BMC Infect. Dis.* **2025**, *25*, 999. <https://doi.org/10.1186/s12879-025-11435-y>.
6. Yagan, A.J.C.; Jasmine, D. Mathematical modelling and its stability analysis of an SEIR model to control dengue by segregating the infective: An approach for efficient resource allocation. *Indian J. Sci. Technol.* **2024**, *17*, 1800–1812. <https://doi.org/10.17485/ijst/v17i17.247>.
7. Islam, N.; Borhan, J.R.M.; Prodhon, R. Application of Mathematical Modelling: A Mathematical Model for Dengue Disease in Bangladesh. *Int. J. Math. Sci. Comput.* **2024**, *10*, 19–30.
8. Herpa Awasthi, M. Reproductive factors of dengue and chlamydia. *Glob. J. Reprod. Med.* **2019**, *6*, 91–94. <https://doi.org/10.19080/gjorm.2019.06.555695>.
9. Naaly, B.Z.; Marijani, T.; Isdory, A.; et al. Mathematical modelling of the effects of vector control, treatment and mass awareness on the transmission dynamics of dengue fever. *Comput. Methods Programmes Biomed. Update* **2024**, *6*, 100159.
10. Dave, R.D.; Yeolekar, B.M.; Khirsariya, S.R.; et al. Fractional-Order modelling of Dengue Transmission Dynamics Using the Atangana-Baleanu Fractional Derivative. *New Math. Nat. Comput.* **2025**, *9*, 1–29.
11. Zhang, H.; Lui, R. Releasing wolbachia-infected aedes aegypti to prevent the spread of dengue virus: A mathematical study. *Infect. Dis. Model.* **2020**, *5*, 142–160.
12. Taghikhani, R.; Sharomi, O.; Gumel, A.B. Dynamical of a two-sex model for the population ecology of dengue mosquitoes in the presence of Wolbachia. *Math. Biosci.* **2020**, *328*, 108426.
13. Ndi, M.Z. The effects of vaccination, vector controls and media on dengue transmission dynamics with a seasonally varying mosquito population. *Results Phys.* **2022**, *34*, 105298.
14. Aldila, D.; Ndi, M.Z.; Anggriani, N.; et al. Impact of social awareness, case detection, and hospital capacity on dengue eradication in Jakarta: A mathematical model approach. *Alex. Eng. J.* **2023**, *64*, 691–707.
15. Abidemi, A.; Fatmawati, O.; Peter, O.J. An optimal control model for dengue dynamics with asymptomatic, isolation, and vigilant compartments. *Decis. Anal.* **2024**, *10*, 100413.

16. Aguiar, M.; Anam, V.; Blyuss, K.B.; et al. Mathematical models for dengue fever epidemiology: A 10-year systematic review. *Phys. Life Rev.* **2022**, *40*, 65–92.
17. Ogunlade, S.T.; Meehan, M.T.; Adekunle, A.I.; et al. A systematic review of mathematical models of dengue transmission and vector control: 2010–2020. *Viruses* **2023**, *15*, 254.
18. Alhaj, M.S. Mathematical model for dengue fever with vertical transmission and control measures: Dengue fever model. *J. Math. Anal. Model.* **2023**, *4*, 44–58.
19. Hasan, M.R.; Alshehri, A.H.A. Dynamic vector-host dengue epidemic model with vector control and sensitivity analysis. *Adv. Dyn. Syst. Appl.* **2023**, *18*, 1–21. <http://www.ripublication.com/ijde.htm>. (accessed on 19 December 2025).
20. Defterli, O. Comparative analysis of fractional order dengue model with temperature effect via singular and non-singular operators. *Chaos, Solitons Fractals* **2021**, *144*, 110654.
21. Nyanaro, B.; Kimathi, G.; Wainaina, M. Mathematical modelling of dengue fever transmission dynamics in Kenya. *J. Appl. Math.* **2024**, *2*, 1807.
22. Oguntolu, F.A.; Peter, O.J.; Babasola, O.; et al. Mathematical modelling on the dynamics of dengue fever with vaccination and transovarial transmission with real statistical data. *Qual. Quant.* **2026**, *60*, 6327–6368.
23. Wardhani, R.; Widowati, W.; Sunarshih, S. Mathematical modelling of dengue haemorrhagic fever transmission: Analysis and numerical simulation. *AIP Conf. Proc.* **2025**, *3301*, 040002.
24. Alnoor, F.; Wahbi, H.; Saadi, F.; et al. A Mathematical Model for the Dengue Fever Epidemic with Vaccination and Treatment. *Eur. J. Pure Appl. Math.* **2025**, *18*, 5815.
25. Duru, E.C.; Anyanwu, M.C.; Mbah, G.C.E. Mathematical Analysis of a Malaria model with vaccination, treatment and vector control using Sterile-insect technique. *J. Math. Anal. Model.* **2025**, *6*, 82–106.
26. Duru, E.C.; Anyanwu, M.C.; Mbah, G.C.E. A mathematical model to investigate the effect of misdiagnosis and wrong treatment in the co-circulation and co-infection of Malaria and Zika virus disease. *Bull. Biomath.* **2025**, *3*, 79–110.
27. Kurniawati, A.T.; Fatmawati, F.; Chukwu, C.W.; et al. Optimal control of dengue fever model with a logistically growing human population. *Math. Model. Control.* **2025**, *5*, 48–60.
28. Castillo-Chavez, C.; Song, B. Dynamical models of tuberculosis and their applications. *Math. Biosci. Eng.* **2004**, *2*, 361–404.