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Research article

A biological mathematical model of vector-host disease with saturated treatment function and optimal control strategies

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Abstract: The aims of this paper to explore the dynamics of the vector-host disease with saturated treatment function. Initially, we formulate the model by considering three different classes for human and two for the vector population. The use of the treatment function in the model and their brief analysis for the case of disease-free and endemic case are briefly shown. We show that the basic reproduction number (< or >) than unity, the disease-free and endemic cases are stable locally and globally. Further, we apply the optimal control technique by choosing four control variables in order to maximize the population of susceptible and recovered human and to minimize the population of infected humans and vector. We discuss the results in details of the optimal controls model and show their existence. Furthermore, we solve the optimality system numerically in connection with the system of no control and the optimal control characterization together with adjoint system, and consider a set of different controls to simulate the models. The considerable best possible strategy that can best minimize the infection in human infected individuals is the use of all controls simultaneously. Finally, we conclude that the work with effective control strategies.

Keywords: optimal control applications; vector-host model; saturated treatment; basic reproduction number; global stability; numerical simulations **Subject classification**: 92D25, 49J15, 93D20

1. Introduction

Mathematical modeling is used often for better understanding the infectious diseases dynamics. Mathematical models not only describe the mathematical process of the infectious diseases but also give useful information about the disease possible control and spread. There are a lot of infectious diseases in our world by providing many infected cases and death around the world. In which, vector-borne diseases are regarding a major threat to the human health causes many death and infection each year.

Vectors are the biological agents, which are the considered to be the main source of infection in human society. Dengue, malaria etc are the important vector-borne diseases that provide many infections and death cases to the human world. Vector-host disease mostly targeted the children especially in the developed countries. Some of the symptoms such as joint pains, headache, muscle, fever, and a skin rash similar to the measles. It is documented in infected cases few number of cases become the life-threatening dengue hemorrhagic fever. This results to bleeding, blood platelets with low level and with leakage of blood plasma, or the dengue shock syndrome in which a low blood pressure occurs. Approximately, one million deaths occurs per year and with over all 17% in all infectious diseases, so vector-borne diseases are considered to be responsible. Due dengue only, approximately 2.5 billion people in almost 100 countries of the world are currently at risk. Similarly, each year globally, 0.4 millions deaths are reported due to malaria, in all these cases most of the children are under the age of 5 years. Besides this, the infectious diseases such as leishmaniasis, chagas disease, and schistosomiasis provide millions of cases to the human population globally. Due to dengue, malaria, human African trypanosomiasis, yellow fever, schistosomiasis, onchocerciasis, and Japanese encephalitis contributed more than one billion cases and due to these a lot of deaths recorded/discovered globally [1].

Regarding the infectious diseases, the prevention is a useful protective tool to safe the society from infection whenever there is no vaccine or treatment. Dengue is a contagious disease caused by a virus, which is still epidemic in many regions such as tropical and sub-tropical areas of the world [2]. The disease is common in South Asia, Africa, USA and Western Pacific regions. There were 9 countries before 1970, which faced this problems but the increment were four times larger after 1995 [3]. The report of World Health Organization (WHO) suggested that dengue fever cases per year vary 50 to 100 million cases with approximately 10000 children death due to bleeding caused by dengue [4].

In order to understand the mechanism of vector-host diseases, the researchers developed numerous mathematical models in literature. For example, a mathematical model suggested by Ross [5] and then it is extended by the authors in [5] suggested the modeling and its analysis with optimal control analysis. The vector-borne disease transmission can be horizontally or vertically. A vector-borne disease model with time delay has been analyzed in [6]. The dynamics of vector-host model is studied in [7]. The analysis of vector-host disease with demographic structure is considered in [8]. The analysis of dengue dynamics with different mode of transmission is studied in [9]. The phenomenon of backward bifurcation analysis in dengue dynamics is considered in [10]. A vector-host disease with direct transmission is considered in [11]. Computer simulations and modeling formulation of dengue

fever is analyzed in [12]. The dynamics of dengue infection in Pakistan with optimal control strategies has been proposed in [13]. Dengue dynamics with variable population is discussed in [14]. The authors considered in [15], the dynamics of malaria disease and presented the optimal control analysis with different control strategies. The dynamics of vector-host model with delay differential equation is studied in [16]. A dynamical model of vector-host disease with analysis of backward bifurcation and optimal control is considered in [17].

We aim here to formulate a mathematical model for vector-host dynamics through saturated treatment function. The use of the treatment function in mathematical models have been used by many authors, see [19, 20, 21, 22]. The authors in [19] used the saturated treatment function in SIR model related to the network and presented the bifurcation analysis. The saturated treatment function for the age structured viral infection is analyzed in [20]. The rumors spread dynamics in social network is studied in [21]. The dynamics of SIR model with age dependent susceptibility with nonlinear incidence rate is investigated in [22]. We first develop the model and present mathematical results briefly. Then, we formulate a control problem and suggest a set of control combinations for possible control of infection. The rest of work is as follows: brief model formulation is given in Section 2. Model equilibria and its stability has been discussed in Section 3. Optimal control problem and its related results have been discussed in details in Section 4. The results are discussed briefly in Section 5 while the work is summarized in Section 6.

2. Model formulation

We present here briefly the dynamics of vector-host disease by denoting the total population of human by N_h , subdividing further into three different classes, namely, the susceptible humans $S_h(t)$, infected humans $I_h(t)$ and the recovered humans $R_h(t)$ at any time t, thus $N_h(t) = S_h(t) + I_h(t) + R_h(t)$. The population of susceptible human is increased by the recruitment of the individuals at a rate of Λ_h . It is decreased by the effective contact with $\beta_1 S_h I_v / (1 + \alpha_1 I_v)$, where the disease contact rate between susceptible human and infected vector is represented by β_1 and α_1 is the saturation constant. It is further decreased by the natural death rate μ_h . This rate of change can be represented through the following differential equation:

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_1 S_h I_v}{1 + \alpha_1 I_v} - \mu_h S_h.$$
(2.1)

The population of infected humans is generated by the effective contact rate $\beta_1 S_h I_v / (1 + \alpha_1 I_v)$ and decreased by the natural death rate μ_h , the disease related death rate δ_h and $\gamma u I_h / (1 + b u I_h)$. It can be seen when *I* or *u* is considered to be very small, then the treatment function converges to a near-zero value and whenever if the value of *I* is considered to be very large, then it approaches to a limit of finite value. The using of such type of function (treatment) will naturally reflect the epidemic system and thus, we consider it in our considered model. The term γ/b is defined to be the the maximal supply of medical resource per unit time while $1/(1 + buI_h)$ shows the reverse effect of infected people that are delayed for treatment and have an important effect on the disease spread, for details see [18]. We mathematically write the discussion in the form below:

$$\frac{dI_h}{dt} = \frac{\beta_1 S_h I_v}{1 + \alpha_1 I_v} - (\mu_h + \delta_h) I_h - \frac{\gamma u I_h}{1 + b u I_h}.$$
(2.2)

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Figure 1. Flow chart.

The individuals in the recovered class are generated by the treatment function $\gamma uI_h/(1 + buI_h)$ while due to the natural death μ_h it becomes decreasing. We mathematically obtain the following form:

$$\frac{dR_h}{dt} = \frac{\gamma u I_h}{1 + b u I_h} - \mu_h R_h.$$
(2.3)

We denote the vector population by N_v and distribute it into two subclasses, namely, S_v susceptible vector and I_v infected vector. Thus, we can write $N_v = S_v + I_v$. The susceptible vector population is generated through the birth rate Λ_v while decreased by the contact rate $\beta_2 S_v I_h / (1 + \alpha_2 I_h)$ and the natural death rate μ_v . This discussion leads to the differential equation given below:

$$\frac{dS_v}{dt} = \Lambda_v - \frac{\beta_2 S_v I_h}{1 + \alpha_2 I_h} - \mu_v S_v.$$
(2.4)

The infected vector population is generated through the contact rate $\beta_2 S_v I_h / (1 + \alpha_2 I_h)$ while decreased by the natural death rate μ_v . This dynamics of the infected vector can be represented by the following differential equation:

$$\frac{dI_v}{dt} = \frac{\beta_2 S_v I_h}{1 + \alpha_2 I_h} - \mu_v I_v. \tag{2.5}$$

The equations (2.1-2.5) above can be written as a single system as follows:

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_1 S_h I_v}{1 + \alpha_1 I_v} - \mu_h S_h,$$

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$$\frac{dI_h}{dt} = \frac{\beta_1 S_h I_v}{1 + \alpha_1 I_v} - (\mu_h + \delta_h) I_h - \frac{\gamma u I_h}{1 + b u I_h},$$

$$\frac{dR_h}{dt} = \frac{\gamma u I_h}{1 + b u I_h} - \mu_h R_h,$$

$$\frac{dS_v}{dt} = \Lambda_v - \frac{\beta_2 S_v I_h}{1 + \alpha_2 I_h} - \mu_v S_v,$$

$$\frac{dI_v}{dt} = \frac{\beta_2 S_v I_h}{1 + \alpha_2 I_h} - \mu_v I_v,$$
(2.6)

subject to the initial conditions (ICs)

$$S_h(0) \ge 0, \ I_h(0) \ge 0, \ R_h(0) \ge 0, \ S_v(0) \ge 0, \ I_v(0) \ge 0.$$
 (2.7)

Let $N_h = S_h + I_h + R_h$, describes the dynamics of human population at time t and then it is given by

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h, \qquad (2.8)$$

i.e.,

$$\frac{dN_h}{dt} + \mu_h N_h \le \Lambda_h. \tag{2.9}$$

According to the results that are given in Birkhoff and Rota [23], we have the following result:

$$0 \le (S_h, I_h, R_h) \le \frac{\Lambda_h}{\mu_h} (1 - e^{-\mu_h t}) + N_h \Big(S_h(0) + I_h(0) + R_h(0) \Big) e^{-\mu_h t}.$$

Now, taking $t \longrightarrow \infty$, we obtain $0 \le N_h \le \frac{\Lambda_h}{\mu_h}$. Let $N_v = S_v + I_v$, describes the total dynamics of vector at time *t* and then it is given by

$$\frac{dN_{\nu}}{dt} = \Lambda_{\nu} - \mu_{\nu}N_h. \tag{2.10}$$

The exact solution of (2.10) is $N_{\nu} = \frac{\Lambda_{\nu}}{\mu_{\nu}}$. The feasible region for the proposed model is

$$\Xi = \left\{ (S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^5, \ N_h \le \frac{\Lambda_h}{\mu_h}, \ N_v \le \frac{\Lambda_v}{\mu_v} \right\}.$$
(2.11)

Proposition 2.1. The set

$$\Xi = \left\{ (S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^5, \ N_h \le \frac{\Lambda_h}{\mu_h}, \ N_v \le \frac{\Lambda_v}{\mu_v} \right\}$$

is positively invariant.

Proof. To show the above result that is Ξ is positively invariant, we use standard comparison theorem

$$0 \le N_h \le N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}), \quad 0 \le N_\nu \le N_\nu(0)e^{-\mu_\nu t} + \frac{\Lambda_\nu}{\mu_\nu}(1 - e^{-\mu_\nu t}).$$

As $t \to \infty$, $(0 \le N_h \le \frac{N_h}{\mu_h}, \le N_v \le \frac{N_v}{\mu_v})$.

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3. Equilibria and local stability

In the present section, we examine the dynamics of the model (2.6) by the available fixed points. There exists two fixed points namely, the disease-free and the endemic equilibrium. We denote the disease-free equilibrium by P_0 and obtained the following:

$$P_0 = \left(S_h^0, 0, 0, S_v^0, 0\right) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0\right).$$

In order to find the stability analysis of the model (2.6), we need to calculate the basic reproduction number \mathcal{R}_0 of the model (2.6) by considering the next generation method [24]. The desired matrices are computed as follows:

$$F = \begin{bmatrix} 0 & \beta_1 \frac{\Lambda_h}{\mu_h} \\ \beta_2 \frac{\Lambda_v}{\mu_v} & 0 \end{bmatrix},$$

and

$$V = \begin{bmatrix} (\mu_h + \delta_h + \gamma u) & 0 \\ 0 & \mu_v \end{bmatrix}.$$

The spectral radius $\mathcal{R}_0 = \rho(FV^{-1})$, that represents the basic reproduction number of (2.6), shown by

$$\mathcal{R}_0 = \sqrt{\frac{\beta_1 \beta_2 \Lambda_h \Lambda_v}{\mu_v^2 \mu_h (\mu_h + \delta_h + \gamma u)}}$$

Based on \mathcal{R}_0 , the following are suggested:

Theorem 3.1. If $\mathcal{R}_0 < 1$, the disease-free equilibrium P_0 of the system (2.6) is locally asymptotically stable.

This result based on Theorem 2 in Van den Driessche and Watmough (2002) [24].

Endemic Equilibria

We obtain the endemic equilibria of the system (2.6) denoted by $P_1^* = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$, and get,

$$S_{h}^{*} = \frac{\left(\delta_{h}\left(buI_{h}^{*}+1\right)+\mu_{h}\left(buI_{h}^{*}+1\right)+\gamma u\right)\left(I_{h}^{*}\left[\alpha_{1}\beta_{2}\Lambda_{v}+\mu_{v}\left(\beta_{2}+\alpha_{2}\mu_{v}\right)\right]+\mu_{v}^{2}\right)}{\beta_{1}\beta_{2}\Lambda_{v}\left(buI_{h}^{*}+1\right)},$$

$$R_{h}^{*} = \frac{\gamma uI_{h}^{*}}{\mu_{h}\left(buI_{h}^{*}+1\right)},$$

$$S_{v}^{*} = \frac{\Lambda_{v}\left(\alpha_{2}I_{h}^{*}+1\right)}{\beta_{2}I_{h}^{*}+\alpha_{2}I_{h}^{*}\mu_{v}+\mu_{v}},$$

$$I_{v}^{*} = \frac{\beta_{2}I_{h}^{*}\Lambda_{v}}{\mu_{v}\left(\beta_{2}I_{h}^{*}+\alpha_{2}I_{h}^{*}\mu_{v}+\mu_{v}\right)}.$$

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We have the following solution by using the above in first equation of the model (2.6):

$$C_0 I_h^{*2} + C_1 I_h^* + C_2 = 0, (3.1)$$

where

$$C_{0} = bu (\delta_{h} + \mu_{h}) \left(\mu_{h} (\alpha_{1}\beta_{2}\Lambda_{v} + \mu_{v} (\beta_{2} + \alpha_{2}\mu_{v})) + \beta_{1}\beta_{2}\Lambda_{v} \right) (> 0),$$

$$C_{1} = \mu_{h} \left[\mu_{v} \left(\mu_{v} (bu (\delta_{h} + \mu_{h}) + \alpha_{2} (\delta_{h} + \mu_{h} + \gamma u)) + \beta_{2} (\delta_{h} + \mu_{h} + \gamma u) \right) + \alpha_{1}\beta_{2}\Lambda_{v} (\delta_{h} + \mu_{h} + \gamma u) \right]$$

$$+ \beta_{1}\beta_{2}\Lambda_{v} (-bu\Lambda_{h} + \delta_{h} + \mu_{h} + \gamma u),$$

$$C_2 = \mu_h \mu_v^2 (\delta_h + \mu_h + \gamma u) (1 - \mathcal{R}_0^2).$$

Lemma 3.1. Endemic equilibrium(s) and their existence criteria

- Consider if b or u is zero then equation (3.1) represents a linear equation in I_h and thus the existence of a unique endemic equilibrium, feasible if and only if $\mathcal{R}_0 > 1$.
- If b or u are non-zero, then equation (3.1) becomes a quadratic equation with two roots for I_h if $C_1 < 0$ and $\mathcal{R}_0 < 1$. Also, if $C_1^2 \ge 4C_0C_2$ then there exists two positive roots, and namely the two positive equilibria $E_1 = (S_h^1, I_h^1, R_h^1, S_v^1, I_v^1)$ and $E_2 = (S_h^2, I_h^2, R_h^2, S_v^2, I_v^2)$
- If b and u are both non-zero and $\mathcal{R}_0 < 1$, then equation (3.1) has only one change of sign and so by the Descartes rule of sign it can be claimed that the system has a unique feasible equilibrium $E_2 = (S_h^2, I_h^2, R_h^2, S_v^2, I_v^2).$

Now, we have in the following the results for the local asymptotic stability of the model at P_1^* . Consider the theorem given below:

Theorem 3.2. For $\mathcal{R}_0 > 1$, then the vector-host system (2.6) at P_1^* is locally asymptotically stable.

Proof. We obtain the Jacobian matrix below at P_1^* :

$$J^{*} = \begin{bmatrix} -\mu_{h} - \frac{\beta_{1}I_{v}^{*}}{1+\alpha_{1}I_{v}^{*}} & 0 & 0 & -\frac{\beta_{1}S_{h}^{*}}{(1+\alpha_{1}I_{v}^{*})^{2}} \\ \frac{\beta_{1}I_{v}^{*}}{1+\alpha_{1}I_{v}^{*}} & -\mu_{h} - \delta_{h} - \frac{\gamma u}{(1+buI_{h}^{*})^{2}} & 0 & \frac{\beta_{1}S_{h}^{*}}{(1+\alpha_{1}I_{v}^{*})^{2}} \\ 0 & -\frac{\beta_{2}S_{v}^{*}}{(1+\alpha_{2}I_{h}^{*})^{2}} & -\frac{\beta_{2}I_{h}^{*}}{1+\alpha_{2}I_{h}^{*}} & 0 \\ 0 & \frac{\beta_{2}S_{v}^{*}}{(1+\alpha_{2}I_{h}^{*})^{2}} & \frac{\beta_{2}I_{h}^{*}}{1+\alpha_{2}I_{h}^{*}} & -\mu_{v} \end{bmatrix}$$
(3.2)

 $det[J^* - \lambda I] = 0$, gives

$$\lambda^{4} + k_{1}\lambda^{3} + k_{2}\lambda^{2} + k_{3}\lambda + k_{4} = 0, \qquad (3.3)$$

where

$$k_1 = \mu_h + m_1 + m_4 + m_5 + Q_1 + 2\mu_\nu > 0,$$

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$$k_{2} = \mu_{h} (m_{5} + Q_{1}) + 2\mu_{v} (\mu_{h} + m_{5} + Q_{1}) + m_{4} (\mu_{h} + Q_{1} + \mu_{v}) + m_{1} (m_{4} + m_{5} + Q_{1} + 2\mu_{v}) + \mu_{v}^{2} + (\underline{m_{4}m_{5} - m_{2}m_{3}}),$$

$$k_{3} = m_{4} (Q_{1}\mu_{h} + \mu_{v} (\mu_{h} + Q_{1})) + \mu_{v} (2\mu_{h} (m_{5} + Q_{1}) + \mu_{v} (\mu_{h} + m_{5} + Q_{1})) + (\underline{m_{4}m_{5} - m_{2}m_{3}}) (\mu_{h} + \mu_{v}),$$

$$+ m_{1} (m_{4} (m_{5} + Q_{1} + \mu_{v}) + \mu_{v} (2 (m_{5} + Q_{1}) + \mu_{v})) + (\underline{m_{4}m_{5} - m_{2}m_{3}}) (\mu_{h} + \mu_{v}),$$

$$k_{4} = \mu_{v} (\mu_{h} ((m_{5} + Q_{1})\mu_{v} + m_{4}Q_{1}) + m_{1} (m_{5} + Q_{1}) (m_{4} + \mu_{v})) + (\underline{m_{4}m_{5} - m_{2}m_{3}}) \mu_{h}\mu_{v}.$$
(3.4)

The coefficients involved in (3.4) are

$$m_{1} = \frac{\beta_{1}I_{\nu}^{*}}{1 + \alpha_{1}I_{\nu}^{*}}, m_{2} = \frac{\beta_{2}S_{\nu}^{*}}{\left(1 + \alpha_{2}I_{h}^{*}\right)^{2}}, m_{3} = \frac{\beta_{1}S_{h}^{*}}{\left(1 + \alpha_{1}I_{\nu}^{*}\right)^{2}}, m_{4} = \frac{\beta_{2}I_{h}^{*}}{1 + \alpha_{2}I_{h}^{*}}, m_{5} = \frac{\gamma u}{\left(1 + buI_{h}^{*}\right)^{2}}, Q_{1} = \delta_{h} + \mu_{h}.$$

If the term $(\underline{m_4m_5 - m_2m_3}) > 0$, then all coefficients $k_1, ..., k_4$ become positive and then the Routh-Hurwitz condition $k_1 > 0, k_3 > 0, k_4 > 0$, and $k_1k_2k_3 > k_3^2 + k_1^2k_4$ can be satisfied easily. The eigenvalues of the characteristics equation (3.3) then will have negative real parts if $k_i > 0$ for i = 1, 2, 3, 4 > 0 and $\mathcal{R}_0 > 1$ and the term under braces is positive. So, the result follows from Routh-Hurwitz criteria that the system (2.6) is locally asymptotically stable, if $\mathcal{R}_0 > 1$ and the terms under braces is positive. \Box

3.1. The existence of backward bifurcation

Here, we investigate the phenomenon of backward bifurcation for the system (2.6) using the center manifold theory described in [25]. Consider β_1 to be the bifurcation parameter and at $\mathcal{R}_0 = 1$, we have

$$\beta_1 = \frac{\mu_h \mu_v^2 \left(\delta_h + \mu_h + \gamma u\right)}{\beta_2 \Lambda_h \Lambda_v}.$$

Further, we make changes to the model variables by $S_h = y_1$, $I_h = y_2$, $R_h = y_3$, $S_v = y_4$, and $I_v = y_5$. Using the vector notation $y = (y_1, y_2, y_3, y_4, y_5)^T$, then, we write the model (2.6) in the form dy/dt = f, where $f = (f_1, ..., f_5)$ is given by

$$\frac{dy_1}{dt} = \Lambda_h - \frac{\beta_1 y_1 y_5}{1 + \alpha_1 y_5} - \mu_h y_1,
\frac{dy_2}{dt} = \frac{\beta_1 y_1 y_5}{1 + \alpha_1 y_5} - (\mu_h + \delta_h) y_2 - \frac{\gamma u y_2}{1 + b u y_2},
\frac{dy_3}{dt} = \frac{\gamma u y_2}{1 + b u y_2} - \mu_h y_3,
\frac{dy_4}{dt} = \Lambda_v - \frac{\beta_2 y_4 y_2}{1 + \alpha_2 y_2} - \mu_v y_4,
\frac{dy_5}{dt} = \frac{\beta_2 y_4 y_2}{1 + \alpha_2 I_y} - \mu_v y_5.$$
(3.5)

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Evaluating the Jacobian matrix at P_0 with $\beta_1 = \beta_1^*$, we have

$$J = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & -\frac{(u\gamma + \delta_h + \mu_h)\mu_v^2}{\beta_2 \Lambda_v} \\ 0 & -u\gamma - \delta_h - \mu_h & 0 & 0 & \frac{(u\gamma + \delta_h + \mu_h)\mu_v^2}{\beta_2 \Lambda_v} \\ 0 & u\gamma & -\mu_h & 0 & 0 \\ 0 & -\frac{\beta_2 \Lambda_v}{\mu_v} & 0 & -\mu_v & 0 \\ 0 & \frac{\beta_2 \Lambda_v}{\mu_v} & 0 & 0 & -\mu_v \end{pmatrix}$$

It is obvious that a simple zero eigenvalues exists for the matrix J while the remaining have negative real part, so, it is possible now to apply the center manifold theory to the model (2.6). Next, we compute the left and right eigenvectors denoted by $V = (v_1, ..., v_5)$ and $W = (w_1, ..., w_5)$ and is given by

$$v_1 = 0, v_3 = 0, v_4 = 0, v_5 = \frac{v_2 \mu_v (\delta_h + \mu_h + \gamma u)}{\beta_2 \Lambda_v}, v_2 = v_2 > 0,$$

and

$$w_1 = -\frac{w_2 \left(\delta_h + \mu_h + \gamma u\right)}{\mu_h}, w_3 = \frac{\gamma u w_2}{\mu_h}, w_4 = -\frac{\beta_2 w_2 \Lambda_v}{\mu_v^2}, w_5 = \frac{\beta_2 w_2 \Lambda_v}{\mu_v^2}, w_2 = w_4 > 0.$$

Now, computing the values of a_1 and b_1 given by

$$a_{1} = -\frac{2\nu_{2}w_{2}^{2}\left(\mu_{\nu}^{2}L_{1} + \alpha_{1}\beta_{1}\beta_{2}^{2}\Lambda_{h}\Lambda_{\nu}^{2}\right)}{\mu_{h}\mu_{\nu}^{4}}$$

where $L_1 = \left(\mu_h \mu_v \left(\mu_v \left(\alpha_2 \left(\delta_h + \mu_h + \gamma u\right) - b\gamma u^2\right) + \beta_2 \left(\delta_h + \mu_h + \gamma u\right)\right) + \beta_1 \beta_2 \Lambda_v \left(\delta_h + \mu_h + \gamma u\right)\right)$ and

$$b_1 = \frac{\beta_2 v_2 w_2 \Lambda_h \Lambda_v}{\mu_h \mu_v^2} > 0.$$

It can be seen that a_1 is negative while for the backward bifurcation a_1 and b_1 should be positive.

3.2. Global stability of DFE by Lyapunov method

We now consider the model (2.6) by obtaining its global stability at the disease-free and endemic case. First, defining the Lyapunov function for the model (2.6) at the disease-free case and present the result in the following theorem:

Theorem 3.3. The disease-free equilibrium of the model (2.6) is globally asymptotically stable, if $\mathcal{R}_0 < 1$ and otherwise unstable.

Proof. In order to have the proof for the above result, we define the following Lyapunov function.

$$L(t) = \beta_2 S_{\nu}^{0} \Big(S_h - S_h^{0} - S_h^{0} ln \frac{S_h}{S_h^{0}} \Big) + \beta_2 S_{\nu}^{0} I_h + (\mu_h + \delta_h + \gamma u) \Big(S_{\nu} - S_{\nu}^{0} - S_{\nu}^{0} ln \frac{S_{\nu}}{S_{\nu}^{0}} \Big) + (\mu_h + \delta_h + \gamma u) I_{\nu},$$
(3.6)

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Now, by taking the time derivative of (3.6) and using the equations of the system (2.6), then we get

$$L'(t) = \beta_{2}S_{\nu}^{0} \Big(\frac{S_{h} - S_{h}^{0}}{S_{h}}\Big) \Big[\Lambda_{h} - \mu_{h}S_{h}\Big] - \beta_{2}S_{\nu}^{0} \Big(\frac{S_{h} - S_{h}^{0}}{S_{h}}\Big) \frac{\beta_{1}S_{h}I_{\nu}}{1 + \alpha_{1}I_{\nu}} + \beta_{2}S_{\nu}^{0} \frac{\beta_{1}S_{h}I_{\nu}}{1 + \alpha_{1}I_{\nu}} + (\mu_{h} + \delta_{h} + \gamma u) \frac{\beta_{2}S_{\nu}I_{h}}{1 + \alpha_{2}I_{h}} - (\mu_{h} + \delta_{h} + \gamma u)\mu_{\nu}I_{\nu} - \beta_{2}S_{\nu}^{0}(\mu_{h} + \delta_{h} + \gamma u)I_{h} - \beta_{2}S_{\nu}^{0} \frac{bu(\mu_{h} + \delta_{h})I_{h}}{1 + buI_{h}} + (\mu_{h} + \delta_{h} + ru)\Big(\frac{S_{\nu} - S_{\nu}^{0}}{S_{\nu}}\Big)\Big[\Lambda_{\nu} - \mu_{\nu}S_{\nu}\Big] - (\mu_{h} + \delta_{h} + \gamma u)\Big(\frac{S_{\nu} - S_{\nu}^{0}}{S_{\nu}}\Big) \frac{\beta_{2}S_{\nu}I_{h}}{1 + \alpha_{2}I_{h}}.$$
(3.7)

Use $S_h^0 = \frac{\Lambda_h}{\mu_h}$ and $S_v^0 = \frac{\Lambda_v}{\mu_v}$ in (3.7) and taking some arrangements of the terms, then we get

$$L'(t) = -\frac{\beta_2 \Lambda_{\nu} \mu_h}{\mu_{\nu}} \frac{(S_h - S_h^0)^2}{S_h} - \mu_{\nu} (\mu_h + \delta_h + \gamma u) \frac{(S_\nu - S_\nu^0)^2}{S_\nu} - \beta_2 S_\nu^0 b u (\mu_h + \delta_h) \frac{I_h^2}{1 + b u I_h} - (\mu_h + \delta_h + \gamma u) \mu_{\nu} \alpha_1 \frac{I_\nu^2}{(1 + \alpha_1 I_\nu)} - \frac{I_\nu}{(1 + \alpha_1 I_\nu)} (\mu_h + \delta_h + \gamma u) \mu_{\nu} (1 - \mathcal{R}_0^2).$$
(3.8)

L'(t) is negative if $\mathcal{R}_0 < 1$ and L'(t) = 0 if $S_h = S_h^0$, $S_v = S_v^0$, $I_h = I_v = 0$. Hence, the largest compact invariant set $(S_h, I_h, R_h, S_v, I_v) \in \Xi$: L'(t) = 0, is the singleton set E_0 , where E_0 is the disease-free equilibrium. Thus, by Principle [26], P_0 is globally asymptotically stable in Ξ .

3.3. Global stability of endemic equilibrium

We determine the global asymptotical stability of the model (2.6) by applying the geometric approach at P_1^* . To do this, we reduce the system (2.6) by using $S_v = \frac{\Lambda_v - \mu_v I_v}{\mu_v}$ in the last equation of the model (2.6), and have the reduced system given by a new endemic equilibrium point P_2^* :

$$\frac{dS_{h}}{dt} = \Lambda_{h} - \frac{\beta_{1}S_{h}I_{v}}{1 + \alpha_{1}I_{v}} - \mu_{h}S_{h},$$

$$\frac{dI_{h}}{dt} = \frac{\beta_{1}S_{h}I_{v}}{1 + \alpha_{1}I_{v}} - \mu_{h}I_{h} - \delta_{h}I_{h} - \frac{\gamma uI_{h}}{1 + buI_{h}},$$

$$\frac{dI_{v}}{dt} = \frac{\beta_{2}I_{h}(\Lambda_{v} - \mu_{v}I_{v})}{\mu_{v}(1 + \alpha_{2}I_{h})} - \mu_{v}I_{v},$$
(3.9)

subject to the non-negative initial conditions

$$S_h = S_h(0) \ge (0), I_h = I_h(0) \ge (0), I_v = I_v \ge (0).$$

Lemma 3.2. If the model $\frac{dx}{dt} = g(x)$ where $g(x) : D \to \mathbb{R}^n$ possesses a unique equilibrium x^* and also a compact absorbing set exists for x^* . Then, x^* is globally asymptotically stable given that a function P(x) and a Lozinskii measure ℓ exists such that $q = \lim_{t \to \infty} \sup \frac{1}{t} \int_0^t \ell(H(x(s, x))) ds < 0$ [27, 28], where the symbols P, ℓ and H shall be defined in the result below.

Theorem 3.4. The reduced vector-host model (3.9) is globally asymptotically stable at P_2^* whenever $\mathcal{R}_0 > 1$.

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Proof. The Jacobian matrix evaluated at P_2^* of the model (3.9) is given by

$$J = \begin{bmatrix} -\mu_h - \frac{\beta_1 I_v}{1 + \alpha_1 I_v} & 0 & -\frac{\beta_1 S_h}{(1 + \alpha_1 I_v)^2} \\ \frac{\beta_1 I_v}{1 + \alpha_1 I_v} & -\mu_h - \delta_h - \frac{\gamma u}{(1 + b u I_h)^2} & \frac{\beta_1 S_h}{(1 + \alpha_1 I_v)^2} \\ 0 & \frac{\beta_2 (\Delta_v - \mu_v I_v)}{\mu_v (1 + \alpha_2 I_v)^2} & -\frac{\beta_2 I_h}{1 + \alpha_2 I_h} - \mu_v \end{bmatrix}.$$

Related to the matrix J, we define the following second additive compound matrix:

$$J^{[2]} = \begin{bmatrix} Q_{11} & \frac{\beta_1 S_h}{(1+\alpha_1 I_v)^2} & \frac{\beta_1 S_h}{(1+\alpha_1 I_v)^2} \\ \frac{\beta_2 (\Lambda_v - \mu_v I_v)}{(1+\alpha_2 I_h)^2 \mu_v} & Q_{22} & 0 \\ 0 & \frac{\beta_1 I_v}{1+\alpha_1 I_v} & Q_{33} \end{bmatrix},$$

where

$$Q_{11} = -\frac{\gamma u}{(1+buI_h)^2} - \frac{\beta_1 I_v}{1+\alpha_1 I_v} - 2\mu_h - \delta_h,$$

$$Q_{22} = -\frac{\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_2 I_h}{1+\alpha_2 I_h} - \mu_h - \mu_v,$$

$$Q_{33} = -\frac{\gamma u}{(1+buI_h)^2} - \frac{\beta_2 I_h}{1+\alpha_2 I_h} - \delta_h - \mu_h - \mu_v.$$
(3.10)

Consider a matrix P

$$P = \left[\begin{array}{ccc} 1 & 0 & 0 \\ 0 & \frac{I_h}{I_v} & 0 \\ 0 & 0 & \frac{I_h}{I_v} \end{array} \right],$$

with

$$P^{-1} = \begin{bmatrix} 1 & 0 & 0 \\ 0 & \frac{I_{\nu}}{I_{h}} & 0 \\ 0 & 0 & \frac{I_{\nu}}{I_{h}} \end{bmatrix},$$

where P_f in the direction of vector field f shows the derivative of P. More precisely, we have:

$$P_f = \left[\begin{array}{ccc} 0 & 0 & 0 \\ 0 & \frac{I_v I_h' - I_v' I_h}{I_v^2} & 0 \\ 0 & 0 & \frac{I_v I_h' - I_v' I_h}{I_v^2} \end{array} \right],$$

and

$$P_f P^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{I'_h}{I_h} - \frac{I'_v}{I_v} & 0 \\ 0 & 0 & \frac{I'_h}{I_h} - \frac{I'_v}{I_v} \end{bmatrix},$$

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We obtain the matrix as follows:

$$P_{f}J^{[2]}P^{-1} = \begin{bmatrix} Q_{11} & \frac{\beta_{1}S_{h}I_{v}}{(1+\alpha_{1}I_{v})^{2}I_{h}} & \frac{\beta_{1}S_{h}I_{v}}{(1+\alpha_{1}I_{v})^{2}I_{h}} \\ \frac{I_{h}\beta_{2}(\Lambda_{v}-\mu_{v}I_{v})}{I_{v}(1+\alpha_{2}I_{v})^{2}\mu_{v}} & Q_{22} & 0 \\ 0 & \frac{\beta_{1}I_{v}}{1+\alpha_{1}I_{v}} & Q_{33} \end{bmatrix},$$

where

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

where

$$\begin{aligned} H_{11} &= -2\mu_{h} - \delta_{h} - \frac{\gamma u}{(1+buI_{h})^{2}} - \frac{\beta_{1}I_{v}}{1+\alpha_{1}I_{v}}, \\ H_{12} &= max \bigg\{ \frac{\beta_{1}S_{h}I_{v}}{I_{h}(1+\alpha_{1}I_{v})^{2}}, \frac{\beta_{1}S_{h}I_{v}}{I_{h}(1+\alpha_{1}I_{v})^{2}} \bigg\}, \\ H_{21} &= \bigg(\frac{I_{h}\beta_{2}(\Lambda_{v} - \mu_{v}I_{v})}{I_{v}\mu_{v}(1+\alpha_{2}I_{h})^{2}}, 0 \bigg), \\ H_{22} &= \bigg[-\frac{\beta_{2}I_{h}}{I+\alpha_{2}I_{h}} - \mu_{h} - \frac{\beta_{1}I_{v}}{1+\alpha_{1}I_{v}} - \mu_{v} + \frac{I_{h}'}{I_{h}} - \frac{I_{v}'}{I_{v}} \qquad 0 \\ -\frac{\gamma u}{(1+buI_{h})^{2}} - \delta_{h} - \frac{\beta_{2}I_{h}}{1+\alpha_{2}I_{h}} + \frac{I_{h}'}{I_{h}} - \frac{I_{v}'}{I_{v}} \bigg]. \end{aligned}$$

Let the vector $(\hat{u}, \hat{v}, \hat{w})$ in \mathbb{R}^3 and its norm $\|.\|$ is defined as

$$\|(\hat{u}, \hat{v}, \hat{w})\| = max\{|\hat{u}|, |\hat{v}|, |\hat{w}|\}.$$

Let μH denote the Lozinski measure with the norm defined above. It follows from [27, 28], we have

$$\mu(H) \le \sup(f_1, f_2),$$

where

$$f_1 = \mu(H_{11}) + |H_{12}|, f_2 = |H_{21}| + \mu(H_{22}),$$

 $|H_{21}|$ and $|H_{12}|$ show the matrix norm related to the vector ℓ and μ , denote the Lozinski measure with respect to ℓ norm, then

$$\mu(H_{11}) = -2\mu_h - \delta_h - \frac{\gamma u}{(1 + b u I_h)^2} - \frac{\beta_1 I_v}{1 + \alpha_1 I_v},$$

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$$|H_{12}| = max\{\frac{\beta_1 S_h I_v}{I_h (1 + \alpha_1 I_v)^2}, \frac{\beta_1 S_h I_v}{I_h (1 + \alpha_1 I_v^2)}\}.$$
(3.11)

Therefore,

$$f_1 = \mu(H_{11}) + |H_{12}|,$$

$$= -2\mu_h - \delta_h - \frac{\gamma u}{(1+buI_h)^2} - \frac{\beta_1 I_v}{1+\alpha_1 I_v} + \frac{\beta_1 S_h I_v}{I_h (1+\alpha_1 I_v)^2}$$

$$\leq -\mu_h - \frac{\beta_1 I_v}{1 + \alpha_1 I_v} - \mu_h - \delta_h - \frac{\gamma u}{(1 + b u I_h)} + \frac{\beta_1 S_h I_v}{I_h (1 + \alpha_1 I_v)}.$$

Now using system (3.9),

$$\frac{I'_h}{I_h} = \frac{I_v}{I_h} \frac{\beta_1 S_h}{1 + \alpha_1 I_v} - \mu_h - \delta_h - \frac{\gamma u}{1 + b u I_h}.$$

Then, we get

$$f_1 \leq \frac{I'_h}{I_h} - \mu_h - \frac{\beta_1 I_v}{1 + \alpha_1 I_v}.$$

Also,

$$H_{21} = \frac{I_h \beta_2 \left(\Lambda_v - \mu_v I_v \right)}{I_v \mu_v \left(1 + \alpha_2 I_h \right)^2},$$

$$\mu(H_{22}) = Sup \left\{ \frac{I'_h}{I_h} - \frac{I'_v}{I_v} - \frac{\beta_1 I_v}{1 + \alpha_1 I_v} - \frac{\beta_2 I_h}{1 + \alpha_2 I_h} - \mu_h - \mu_v + \frac{\beta_1 I_v}{1 + \alpha_1 I_v}, \frac{I'_h}{I_h} - \frac{I'_v}{I_v} - K_1 \right\}$$
$$= \frac{I'_h}{I_h} - \frac{I'_v}{I_v} - \mu_h - \mu_v - \frac{\beta_2 I_h}{1 + \alpha_2 I_h} - \frac{\gamma u}{1 + b u I_h} - (\mu_h + \delta_h + \mu_v),$$

where $K_1 = \frac{\gamma u}{(1+buI_h)^2} - \frac{\beta_2 I_h}{1+\alpha_2 I_h} - \delta_h - \mu_h - \mu_v$. Now,

$$f_2 = |H_{21}| + \mu(H_{22}),$$

$$=\frac{I_{h}\beta_{2}(\Lambda_{v}-\mu_{v}I_{v})}{I_{v}\mu_{v}(1+\alpha_{2}I_{h})}+\frac{I_{h}'}{I_{h}}-\frac{I_{v}'}{I_{v}}-\frac{\beta_{2}I_{h}}{1+\alpha_{2}I_{h}}-\frac{\gamma u}{1+buI_{h}}-(2\mu_{h}+\delta_{h}+2\mu_{v}),$$

$$\leq \frac{I'_{h}}{I_{h}} - \frac{I'_{v}}{I_{v}} - \frac{\beta_{2}I_{h}}{1 + \alpha_{2}I_{h}} - \frac{\gamma u}{1 + buI_{h}} - (2\mu_{h} + \delta_{h} + 2\mu_{v}) + \frac{I_{h}\beta_{2}(\Lambda_{v} - \mu_{v}I_{v})}{I_{v}\mu_{v}(1 + buI_{h})}.$$

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In f_2 above, we used the third equation of the system (3.9).

$$\frac{I'_{\nu}}{I_{\nu}} = \frac{I_h}{I_{\nu}} \frac{\beta_2(\Lambda_{\nu} - \mu_{\nu}I_{\nu})}{\mu_{\nu}(1 + \alpha_2I_h)} - \mu_{\nu}$$

Then, we can get

$$f_2 \leq \frac{I'_h}{I_h} - \frac{\beta_2 I_h}{1 + \alpha_2 I_h} - \frac{\gamma u}{1 + b u I_h} - (2\mu_h + \delta_h + 2\mu_\nu).$$

So,

$$\mu(H) \leq Sup(f_1, f_2) \leq \frac{I'_h}{I_h} - \mu.$$

Then,

$$q = \frac{1}{t} \int_0^t \mu H ds \le \frac{1}{t} \int_0^t (\frac{I'_h}{I_h} - \mu) ds = \frac{1}{t} \ln \frac{I_h(t)}{I_h(0)} - \mu$$

This implies that $q \le -\frac{\mu}{2} < 0$. So, it follows from [27] that considered system is globally asymptotically stable.

4. Optimal control problem

This section investigates the application of the optimal control technique to the system (2.6) by modifying the birth rate of susceptible human and vector by the assumptions of the density effects $\Lambda_h \rightarrow \Lambda_h + cN_h$ and $\Lambda_\nu \rightarrow \Lambda_\nu N_\nu$, while the constant *c* shows the density impact on the birth rate. Our main purpose is to formulate an optimal control problem and provide the best possible strategies of control for minimization of infection in human population. The use of optimal controls to the biological models with brief analysis are used by many researchers, see [29, 30, 31, 32, 33]. Here, in the optimal control system, we consider four controls, u_i for i = 1, 2, 3, 4, which are defined as follows: u_1 is defined to be drugs or vaccine which can decrease the human and mosquitoes contacts such as insect repellents, the second control u_2 shows the level of larvicide and adulticide utilized in order to control mosquitoes breading places, the third control u_3 shows the minimization of human and mosquitoes contacts by the use of bed nets as a preventions and the control variable u_4 represents the control (through some specific prevention or treatment). The term $(1 - u_1)$, is considered for the reduction of the force of infections in human population and b_o is a positive rate constant. The factor $(1 - u_2)$ is used for the reduction of the reproduction rate of mosquito population. The discussion above leads to the following control system:

$$\begin{aligned} \frac{dS_h}{dt} &= \Lambda_h + cN_h - \frac{\beta_1 S_h I_v}{1 + \alpha_1 I_v} (1 - u_1) - \mu_h S_h, \\ \frac{dI_h}{dt} &= \frac{\beta_1 S_h I_v}{1 + \alpha_1 I_v} (1 - u_1) - (\mu_h + \delta_h) I_h - \frac{\gamma u_4 I_h}{1 + b u_4 I_h}, \\ \frac{dR_h}{dt} &= \frac{\gamma u_4 I_h}{1 + b u_4 I_h} - \mu_h R_h, \end{aligned}$$

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$$\frac{dS_{\nu}}{dt} = \Lambda_{\nu}N_{\nu}(1-u_{2}) - \frac{\beta_{2}S_{\nu}I_{h}}{1+\alpha_{2}I_{h}}(1-u_{3}) - \mu_{\nu}S_{\nu} - b_{0}u_{2}S_{\nu},$$

$$\frac{dI_{\nu}}{dt} = \frac{\beta_{2}S_{\nu}I_{h}}{1+\alpha_{2}I_{h}}(1-u_{3}) - \mu_{\nu}I_{\nu} - b_{0}u_{2}I_{\nu}$$
(4.1)

with the ICs (2.7).

In optimal control system (4.1), we considered the controls $u(t) = (u_1, u_2, u_3, u_4) \in \mathcal{U}$ with a brief discussion. The control variables $u(t) = (u_1, u, u_2, u_3) \in \mathcal{U}$ are subjected to the state variables S_h, E_h, I_h, S_v and I_v which are measured and bounded with

$$\mathcal{U} = \{(u_1, u_2, u_3, u_4) | u_i \text{ is Lebsegue measurable on } [0, 1], \ 0 \le u_i(t) \le 1, \\ t \in [0, T], \ i = 1, 2, 3, 4\}.$$

$$(4.2)$$

We have the objective function for the vector-host control problem, given by,

$$J(u_1, u_2, u_3, u_4) = \int_0^T \left[D_1 I_h + D_2 N_v + \frac{1}{2} (D_3 u_1^2 + D_4 u_2^2 + D_5 u_3^2 + D_6 u_4^2) \right] dt.$$
(4.3)

The constants in (4.3), D_1 , D_2 , D_3 , D_4 , D_5 and D_6 denote the weight or balancing constants. The constants D_1 and D_2 are used respectively for infected human and for vector population. The weight constant D_3 for drug or vaccine, D_4 for larvicide of mosquitos control, D_5 is for minimizing the mosquitoes-humans contacts by using the repellents and D_6 for control through specific prevention or treatment. Further, these constants D_1 , D_2 and D_3 , D_4 , D_5 , D_6 show the cost relative measurement of the interventions in the interval [0, T].

To determine the control problem for u_i^* where i = 1, ..., 4, such that

$$J(u_i^*) = \min_{\mathcal{U}} J(u_i), \tag{4.4}$$

where \mathcal{U} is defined in equation (4.2) and subjected to the system (4.1) with non-negative initial conditions. Consider the technique Pontryagin's Maximum Principle, to get the desired solution of the optimality system mathematically.

4.1. Existence of the optimality system

We use the results given in [34] for the control problem existence. The equations of the control (4.1) are bounded, which enable us to apply the result in [34] to our problem, if the following conditions are met:

- 1. O_1 : The state and control variables are nonempty.
- 2. O_2 : The control \mathcal{U} is closed and convex.
- 3. O_3 : In system (4.1), the equations on right side are bounded and continuous and can be shown as a linear function of u, where the coefficients depend on time and state.
- 4. O_4 : The constants $l_1, l_2 > 0$ and m > 1 exist such that the integrand L(y, u, t) of the objective functional J is convex and satisfies

To show these conditions $(C_1 - C_4)$, we follow the results of [35] to find the result for the existence of (4.1). The controls and the state variables are clearly bounded, which confirms O_1 . The claim O_2 is confirmed because of bounded solution and convex. In order to fulfill C_3 , the model is bilinear in control variables. The last claim O_4 and their verification is given below,

$$D_1I_h + D_2N_v + \frac{1}{2}\left(D_3u_1^2 + D_4u_2^2 + D_5u_3^2 + D_6u_4^2\right) \ge l_1(|u_1|^2 + |u_2|^2 + |u_3|^2 + |u_4|^2)^{\frac{m}{2}} - l_2.$$

where $D_i, l_1, l_2 > 0$ and m > 1 for i = 1, ..., 6. Thus, we have

Theorem 4.1. The objective functional (4.3) with the control set (4.2) subject to the optimality (4.1), then there exists an optimal control $u^* = (u_i^*)$ such that $J(u_i^*) = \min_{\mathcal{U}} J(u_i)$ for i = 1, ..., 4.

For the solution of an optimal control problem, the construction of the Lagrangian L and the Hamiltonian H is required, which are defined below:

$$L(I_h, N_v, u_1, u_2, u_3, u_4) = D_1 I_h + D_2 N_v + \frac{1}{2} \left(D_3 u_1^2 + D_4 u_2^2 + D_5 u_3^2 + D_6 u_4^2 \right), \tag{4.5}$$

and $\underline{X} = (S_h, I_h, R_h, S_v, I_v), \mathcal{U} = (u_1, u_2, u_3, u_4)$ and $\lambda = (\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5)$, to get:

$$H(\underline{X}, \mathcal{U}, \lambda) = L(I_{H}, N_{V}, u_{1}, u_{2}, u_{3}) + \lambda_{1} \Big[\Lambda_{h} + cN_{h} - \frac{\beta_{1}S_{h}I_{v}}{1 + \alpha_{1}I_{v}} (1 - u_{1}) - \mu_{h}S_{h} \Big] + \lambda_{2} \Big[\frac{\beta_{1}S_{h}I_{v}}{1 + \alpha_{1}I_{v}} (1 - u_{1}) - (\mu_{h} + \delta_{h})I_{h} - \frac{\gamma u_{4}I_{h}}{1 + bu_{4}I_{h}} \Big] + \lambda_{3} \Big[\frac{\gamma u_{4}I_{h}}{1 + bu_{4}I_{h}} - \mu_{h}R_{h} \Big] + \lambda_{4} \Big[\Lambda_{v}N_{v}(1 - u_{2}) - \frac{\beta_{2}S_{v}I_{h}}{1 + \alpha_{2}I_{h}} (1 - u_{3}) - \mu_{v}S_{v} - b_{0}u_{2}S_{v} \Big] + \lambda_{5} \Big[\frac{\beta_{2}S_{v}I_{h}}{1 + \alpha_{2}I_{h}} (1 - u_{3}) - \mu_{v}I_{v} - b_{0}u_{2}I_{v} \Big].$$

$$(4.6)$$

4.2. Optimal control problem solution

Using Pontryagin's Maximum Principle [36] for the solution of the optimality system as follows: suppose u_i^* for i = 1, ..., 4 denote the optimal solution of the optimality system (4.1), then the adjoint variables say, λ_i for i = 1, ..., 5 exists which satisfy the conditions below,

$$\frac{dx}{dt} = \frac{\partial H(t, u_1^*, u_2^*, u_3^*, u_4^*, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5)}{\partial \lambda},$$

$$0 = \frac{\partial H(t, u_1^*, u_2^*, u_3^*, u_4^*, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5)}{\partial u},$$

$$\frac{d\lambda}{dt} = -\frac{\partial H(t, u_1^*, u_2^*, u_3^*, u_4^*, \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5)}{\partial x}.$$
(4.7)

Using these conditions to *H*, the following are obtained:

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Theorem 4.2. For the controls u_i^* for i = 1, ..., 4 and $S_h^*, I_h^*, R_h^*, S_v^*, I_v^*$ represent the solution of system of state, then there exists adjoint variables, say, λ_i for i = 1, ..., 5,

$$\begin{aligned} \lambda_{1}' &= \lambda_{1}(\mu_{h} - c) + (\lambda_{1} - \lambda_{2}) \frac{\beta_{1}I_{\nu}(1 - u_{1})}{1 + \alpha_{1}I_{\nu}}, \\ \lambda_{2}' &= -\lambda_{1}c + \lambda_{2}(\mu_{h} + \delta_{h}) + (\lambda_{2} - \lambda_{3}) \frac{\gamma u_{4}}{(1 + bu_{4}I_{h})^{2}} + (\lambda_{4} - \lambda_{5}) \frac{\beta_{2}S_{\nu}(1 - u_{3})}{(1 + \alpha_{2}I_{h})^{2}} - D_{1}, \\ \lambda_{3}' &= -\lambda_{1}c + \lambda_{3}\mu_{h}, \\ \lambda_{4}' &= (\lambda_{4} - \lambda_{5}) \frac{\beta_{2}I_{h}(1 - u_{3})}{1 + \alpha_{2}I_{h}} + \lambda_{4}(b_{0}u_{2} - \Lambda_{\nu}(1 - u_{2})) - D_{2}, \\ \lambda_{5}' &= (\lambda_{1} - \lambda_{2})(1 - u_{1}) \frac{\beta_{1}S_{h}}{(1 + \alpha_{1}I_{\nu})^{2}} + \lambda_{4}(\mu_{\nu} - \Lambda_{\nu}(1 - u_{2})) + \lambda_{5}b_{0}u_{2} - D_{2}, \end{aligned}$$

$$(4.8)$$

with transversality conditions

$$\lambda_1(T_f) = \lambda_2(T_f) = \lambda_3(T_f) = \lambda_4(T_f) = \lambda_5(T_f) = 0.$$
(4.9)

Further, the control u_i^* for i = 1, ..., 4 are

$$u_{1}^{*} = \max \left\{ \min\{1, \frac{(\lambda_{2} - \lambda_{1})\beta_{1}S_{h}^{*}I_{v}^{*}}{(1 + \alpha_{1}I_{v}^{*})D_{3}}\}, 0 \right\}, \\ u_{2}^{*} = \max \left\{ \min\{1, \frac{\lambda_{4}\Lambda_{v}N_{v}^{*} + b_{0}S_{v}^{*} + \lambda_{5}b_{0}I_{v}^{*}}{D_{4}}\}, 0 \right\}, \\ u_{3}^{*} = \max \left\{ \min\{\frac{(\lambda_{5} - \lambda_{4})(\beta_{2}S_{v}^{*}I_{h}^{*})}{(1 + \alpha_{2}I_{h}^{*})D_{5}}\}, 0 \right\}, \\ u_{4}^{*} = \max \left\{ \min\{\frac{(\lambda_{3} - \lambda_{2})\gamma I_{h}^{*}}{(1 + bu_{4}I_{h}^{*})^{2}D_{6}}\}, 0 \right\},$$

$$(4.10)$$

Proof. To obtain the results stated in above theorem, we solve the control system together with the Hamiltonian H (4.6) to have the results for the adjoint system (4.8) and the transversality conditions (4.9), with setting $S_h = S_h^*$, $I_h = I_h^*$, $R_h = R_h^*$, $S_v = S_v^*$, and $I_v = I_v^*$ and the derivative of H with respect to S_h , I_h , R_h , S_v , I_v , we have (4.8). To get the equations of optimal control characterization in (4.10), we use $\frac{\partial H}{\partial u_i} = 0$, for i = 1, 2, 3, 4.

5. Discussion

This section describes the numerical results of the proposed model (2.6) and the optimal control problem (4.1), which are solved numerically. The optimal control solution is obtained through backward Runge-Kutta order four scheme. We denote the solution of the control system via dashed line and those without control by a bold line. The time unit considered in the numerical solution is per day. The numerical values for the parameters are presented in Table 1. The weight and balancing constants with their proposed values are $D_1 = D_2 = 1000$, $D_3 = 10$, $D_4 = 0.005$, $D_5 = 0.03$ and $D_6 = 3$. We choose different cases to investigate the optimal control solutions. We present the

following cases:

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Case (i): In this case, we consider the control variable $u_1 = 0$ and make the rest of the controls $u_2 = u_3 = u_4 \neq 0$ and simulating the model. The resulting graphical results are depicted in Figure 2 with subfigures (a-f). In this case, the population of infected humans decreases and the recovered human increased. Also, the vector populations decrease sharply. This case effective for the infected population as it decreases very fast after day 14 and becomes steady.

Case (ii) In this case, we set $u_2 = 0$ and $u_1 = u_3 = u_4 \neq 0$. The resulting graphical results are presented through Figure 3 with subfigures (a-f). In this case, the population of susceptible human less increased compared to Case (i), but no decrease in the population of infected vector and susceptible vector. Although the population of the recovered and infected human the same as in Case case (ii). Thus, the strategy is not a good one.

Case (iii): In this case, we set $u_3 = 0$ and $u_1 = u_2 = u_4 = 0$. The resulting graphical results are presented through Figure 4 with subfigures (a-f). In this case, the population of susceptible humans increases sharply compared to Case (i) and (ii). The population of infected humans, infected vector and susceptible vector is increasing more compared to previous strategies. Also, the population of recovered human increases.

Case (iv): In this case, we choose to set $u_4 = 0$ and $u_1 = u_2 = u_3 \neq 0$ and simulate the model and obtain the results graphically given in Figure 5 with subfigures (a-f). Comparing the control system with and without controls system, the population of susceptible individuals increases and decreases the population of infected but it can be seen that there is no increase in the population of recovered individuals. It can also be observed that this strategy minimizes the infection in the vector population.

Case (v): In the above combinations of the controls and their simulations, which suggest the increase or decrease in different compartments of the humans and vector populations. In all these strategies from (i-iv) no one get the desired results for the humans and vectors population to be minimized as desired. So, we utilize all the controls active and simulate the model of control in connection with the model having no controls. We observe that this set of controls provide that the population of susceptible and recovered human increase sharply while the population of infected humans, susceptible vector and infected vector are decreasing better, see Figure 6. Comparing to the above cases, this strategy is comparatively better.



Figure 2. Simulation results for the Case (i).



Figure 3. Simulation results for the Case (ii).



Figure 4. Simulation results for the Case (iii).



Figure 5. Simulation results for the Case (iv).



Figure 6. Simulation results for the Case (v).

Table1. Parameters used in simulation.	
Notation	Value
Λ_h	0.0002/day
с	0.0002/day
β_1	0.000044 /day
α_1	0.003 /day
μ_h	0.00020 /day
b	0.4/day
δ_h	0.002 /day
γ	0.1/day
Λ_{v}	0.08/day
β_2	0.007 /day
α_2	0.02/day
μ_{v}	0.2/day
b_o	0.01/day

6. Conclusions

We presented a mathematical model for vector-host disease with saturated treatment function and presented its dynamical results with optimal controls analysis. Stability analysis of the model for the disease-free and endemic cases are obtained and discussed. The disease-free equilibrium is stable when the basic reproduction number $\mathcal{R}_0 < 1$. When the basic reproduction number $\mathcal{R}_0 > 1$, then the endemic equilibrium found to be stable both locally and globally. The optimal control problem with controls variables are formulated and the desired results are obtained and discussed briefly. The optimal control problem together with controls function, and with adjoint equations are simulated and the results of both the models with and without controls are showed. A set of different controls were used to obtain the graphical results and we found that the Case (v) is considered to be the best strategy to control the infection in humans. The use of saturated treatment function in the modeling of vector-host disease is a novel practice and could be useful for the mathematicians and scientists working on vector-host disease research.

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Conflict of interest

No conflict of interest exists regarding the publications of this work.

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