Mathematical modeling of zika virus disease with nonlinear incidence and optimal control

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Abstract. The Zika virus was first discovered in a rhesus monkey in the Zika Forest of Uganda in 1947, and it was isolated from humans in Nigeria in 1952. Zika virus disease is primarily a mosquito-borne disease, which is transmitted to human primarily through the bite of an infected Aedes species mosquito. However, there is documented evidence of sexual transmission of this disease too. In this paper, a nonlinear mathematical model for Zika virus by considering nonlinear incidence is formulated and analyzed. The equilibria and the basic reproduction number ($R_0$) of the model are found. The stability of the different equilibria of the model is discussed in detail. When the basic reproduction number $R_0 < 1$, the disease-free equilibrium is locally and globally stable i.e. in this case disease dies out. For $R_0 > 1$, we have endemic equilibrium which is locally stable under some restriction on parameters. Further this model is extended to optimal control model and is analyzed by using Pontryagin’s Maximum Principle. It has been observed that optimal control plays a significant role in reducing the number of zika infectives. Finally, numerical simulation is performed to illustrate the analytical findings.

1. Introduction

The Zika virus is a mosquito-borne disease. The infection is transmitted to human population through the bite of infected Aedes mosquitoes. In 1947 in the Zika Forest of Uganda first Zika virus was identified in a rhesus monkey and later in 1952 it was isolated from human population in Nigeria. The general symptoms of Zika virus are conjunctivitis, headache, joint pain, mild fever, muscle and, skin rash, etc. The symptom of the virus is exhibited within two to seven days, [1]. During the infected period of Zika, people should drink plenty of fluids and rest sufficiently. Since 2015, the sexual transmission of the disease and blood transfusion of the infected individual are being investigated in the following nations - Argentina, Canada, Chile, France, Italy, New Zealand, Peru, Portugal, and the United States of America. The first sexually transmitted case of Zika virus disease was diagnosed in France in February 2016.

In April 2007, in the island of Yap, the first outbreak Zika virus was identified which was outside of Africa and Asia. During 2013-14 the largest outbreak of Zika cases estimated to be around 30,000 was reported in the French Polynesia. In 2015, rapid spread of Zika virus was recorded among South
American countries, especially Brazil and Colombia notified PAHO/WHO [2]. In Brazil the Public Health authority detected Zika virus in 14 states and in Colombia out of 98 samples 9 samples were confirmed. In Brazil, between October 2015 to February 2016, more than 6000 Zika virus infected cases were recorded including 139 cases of congenital microcephaly. In Colombia, during December 2015 to February 2016, nearly 200 GBS cases were identified with suspected link with Zika infection and in El Salvador nearly 118 GBS cases confirmed as Zika where 5 died [3]. On 1st February 2016, the World Health Organizing (WHO) declared that the Zika virus epidemic as a Public Health Emergency of International Concern (PHEIC) [4].

The transmission dynamics of Zika Virus has been studied and analyzed recently by various researchers [5, 6, 7, 8, 9] using mathematical models as a tool. In [5], authors proposed a mathematical model for transmission dynamics of Zika virus disease and they have shown the control measure of Zika virus through prevention, treatment and insecticides. In [6], authors incorporated the sexual transmission of this disease in the proposed model and computed basic reproduction number and using real data the numerical simulation illustrated the analysis. In [7] authors proposed a new deterministic model introducing vertical transmission of Zika virus and incorporating the cases of new born babies with microcephaly and asymptomatic infected populations. In [8], authors proposed a two patch model for the transmission dynamics of Zika virus to see the effect of short-term dispersal. In [9], authors proposed a mathematical model of Zika virus of the 2013 –14 French Polynesia outbreaks and analyzed using real data. Authors estimated the reproduction number using the real data performed sensitivity analysis.

At present Zika virus is endemic and hence its study is most essential. Here we present a mathematical model for Zika Virus disease by considering standard incidence type interaction for human to human transmission of the disease. Here we assume that the total human population is variable. In this model we introduce the incidence rate \( \frac{c_{lh}}{1 + I_h} \), which was also proposed in [10, 11], where \( c_{lh} \) measures infected force of the virus and \( \frac{1}{1 + I_h} \) measures susceptible individuals when the number of infected individuals increases. Further our proposed mathematical model is extended to optimal control problem. This helps in finding the optimal control strategies to reduce the number of Zika infected cases.

This paper is organized as follows: in Section 2 we formulation our mathematical model; in Section 3 we compute the basic reproduction number and find the existence of equilibria; in Section 4 we present the stability analysis of the model; in Section 5 we illustrate the numerical simulation and results of the model; in Section 6, we study the optimal control model and its analysis; in Section 7 we demonstrate the numerical simulation results of the optimal control model and finally in the Section 8 we conclude our paper.

2. Model formulation:

Here we have proposed a mathematical model for Zika virus disease by considering nonlinear incidence. The model was formulated by considering two populations human and vector (mosquito). In the proposed model the human population has been divided into three independent compartments such as Susceptible individuals (\( S_h \)), Infected individuals (\( I_h \)) and Recovered individuals (\( R_h \)). Also the vector population has been divided into two compartments Susceptible vector (\( S_v \)) and Infected vector (\( I_v \)). The transmission of the disease is possible between human to human, human to vector and vector to human. In this model we incorporate a nonlinear incidence term for human to human transmission, which is of saturated incidence type. Several authors have considered this type of nonlinear incidence term in their respective model [10, 11, 12].
Figure 1: Flow chart for the transmission of Zika virus disease

The proposed mathematical model of Zika virus is as follows:

\[
\frac{dS_h}{dt} = \Lambda_h - \beta_{h1} \left( \frac{cI_h}{1 + I_h} \right) S_h - \beta_{h2} \frac{I_v}{N_h} S_h - \mu_h S_h \\
\frac{dI_h}{dt} = \beta_{h1} \left( \frac{cI_h}{1 + I_h} \right) S_h + \beta_{h2} \frac{I_v}{N_h} S_h - (\gamma_h + \mu_h + \mu_1) I_h \\
\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h \\
\frac{dS_v}{dt} = \Lambda_v - \beta_v \frac{I_h}{N_h} S_v - \mu_v S_v \\
\frac{dI_v}{dt} = \beta_v \frac{I_h}{N_h} S_v - \mu_v I_v
\]  

(1)

where

\( \Lambda_h \): Recruitment rate of human individuals  
\( \Lambda_v \): Recruitment rate of vector individuals (mosquito)  
\( \mu_h \): Natural death rate of human individuals  
\( \mu_v \): Natural death rate of vector individuals (mosquito)  
\( \mu_1 \): Death rate of human due to infection  
\( \beta_{h1} \): Contact rate between \( S_h \) and \( I_h \)  
\( \beta_{h2} \): Contact rate between \( S_h \) and \( I_v \)  
\( \beta_v \): Contact rate between \( S_v \) and \( I_h \)  
\( \gamma_h \): Recovery rate of infectives (human)

Consider \( (S_h, I_h, R_h, S_v, I_v) \) be any solution with positive initial conditions and \( N_h(t) \) and \( N_v(t) \) be the total human and vector population respectively. Then we have \( N_h = S_h + I_h + R_h \) and \( N_v = S_v + I_v \). The time derivative of \( N \) in the above equation is given by

\[
\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \mu_1 I_h
\]
Hence we can rewrite the model in the following form:

\[
\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v
\]

\[
\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \mu_I I_h
\]

\[
\frac{dI_h}{dt} = \left( \beta_{h1} \left( \frac{cI_h}{1 + I_h} \right) + \beta_{h2} \frac{I_v}{N_h} \right) (N_h - I_h - R_h) - (\gamma_h + \mu_h + \mu_I) I_h
\]

\[
\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h
\]

\[
\frac{dN_v}{dt} = \Lambda_h - \mu_v N_v
\]

\[
\frac{dI_v}{dt} = \beta_v \frac{I_h}{N_h} (N_v - I_v) - \mu_v I_v
\]

(2)

3. Existence of equilibrium and the basic reproduction number

3.1 Disease free equilibrium point \( E_0 \) and the basic reproduction number \( R_0 \)

We consider the system (2) and find the disease free equilibrium point. The disease free equilibrium for this model we have \( E_0 = \left( N_h^0, I_h^0, R_h^0, N_v^0, I_v^0 \right) = \left( \frac{\Delta_h}{\mu_h}, 0, 0, \frac{\Delta_v}{\mu_v}, 0 \right) \)

Now we find the basic reproduction number \( R_0 \) by following the next generation matrix methods as described in [13 - 14]. We find the matrix \( F \) and \( V \) as follows:

\[
F = \begin{pmatrix} \beta_{h1} \left( \frac{cI_h}{1 + I_h} \right) + \beta_{h2} \frac{I_v}{N_h} & (N_h - I_h - R_h) \\ \beta_v \frac{I_h}{N_h} (N_v - I_v) \\ \end{pmatrix}
\]

\[
V = \begin{pmatrix} \gamma_h + \mu_h + \mu_I \end{pmatrix}
\]

\[
F = \text{Jacobian of } F \text{ at } E_0 = \begin{pmatrix} \beta_{h1} c N_h^0 & \beta_{h2} \\ \beta_v N_v^0 & 0 \end{pmatrix}
\]

\[
V = \text{Jacobian of } V \text{ at } E_0 = \begin{pmatrix} \gamma_h + \mu_h + \mu_I \end{pmatrix}
\]

And it follows that

\[
FV^{-1} = \begin{pmatrix} \beta_{h1} c N_h^0 D_1 & \beta_{h2} D_2 \\ \beta_v N_v^0 D_2 & 0 \end{pmatrix}
\]

where \( D_1 = \frac{1}{\gamma_h + \mu_h + \mu_I} \), \( D_2 = \frac{1}{\mu_v} \)

The largest eigenvalue of \( FV^{-1} \) is called the basic reproduction number \( R_0 \) and is obtained as follows:
\[ R_0 = \frac{\beta_{hh} c N_h^0 D_1 + \sqrt{(\beta_{hh} c N_h^0 D_1)^2 + 4 \beta_{hh} \beta_{hv} N_h^0 D_2}}{2} = \frac{R_{hh} + \sqrt{(R_{hh})^2 + 4 R_{hv}}}{2} \]

where

\[ R_{hh} = \frac{\beta_{hh} c N_h^0}{\gamma_h + \mu_h + \mu_1} \quad \text{and} \quad R_{hv} = \beta_{hv} N_h^0 D_1 D_2. \]

Here \( R_{hh} \) denotes the basic reproduction due to human to human transmission by ignoring the transmission due to vectors. Similarly, \( R_{hv} \) denotes the basic reproduction due to interactions with vectors in the absence of human to human transmission. The reproduction number \( R_0 \) gives the average number of infected individuals generated by the one infected in a fully susceptible population and for our model it is given by above expression of \( R_0 \).

### 3.2 Existence of endemic equilibrium

The endemic equilibrium point of the model we have \( E_1 = (N_h^*, I_h^*, R_h^*, N_v^*, I_v^*) \), where,

\[ N_h^* = \frac{\Lambda_h - \mu_1 I_h^*}{\mu_h} \]

\[ R_h^* = \frac{\gamma_h I_h^*}{\mu_h} \]

\[ N_v^* = \frac{\Lambda_v}{\mu_v} \]

\[ I_v^* = \frac{\Lambda_v \mu_h \beta_{hv} I_h^*}{\mu_v \mu_h \beta_{hv} I_v^* + \mu_v^2 (\Lambda_v - \mu_1 I_h^*)} \]

And there is a positive root of \( I_h^* \) of the following non-linear equation

\[ g(I_h) = [A_h - (\mu_1 + \mu_h + \gamma_h) I_h] \left[ \frac{c \beta_{hh} (A_h - \mu_1 I_h)}{(1 + I_h) \mu_h} + \frac{\beta_{hv} \Lambda_v \beta_{hv} \mu_h}{\mu_h \mu_v \beta_{hv} I_h + (A_h - \mu_1 I_h) \mu_v^2} \right] \]

\[-(A_h - \mu_1 I_h)(\mu_1 + \mu_h + \gamma_h) = 0. \]

Here, we note the following:

\[ g(0) = A_h \left[ \frac{c \beta_{hh} A_h}{\mu_h} + \frac{\beta_{hv} \Lambda_v \beta_{hv} \mu_h}{\mu_h \mu_v^2} \right] - A_h (\mu_1 + \mu_h + \gamma_h) > 0 \]

\[ g \left( \frac{A_h}{\mu_1 + \mu_h + \gamma_h} \right) = -A_h (\mu_h + \gamma_h) < 0 \]

\[ g \left( \frac{\Lambda_v}{\mu_2} \right) = -A_h \frac{\mu_h + \gamma_h}{\mu_1} \left[ \frac{\beta_{hv} \Lambda_v \beta_{hv} \mu_1}{\mu_v \mu_h \beta_{hv}} \right] < 0 \]

It is easy to observe that \( \frac{A_h}{\mu_1 + \mu_h + \gamma_h} < I_h < \frac{\Lambda_v}{\mu_1} \) \( g(I_h) \) is always negative, i.e. there is no change of sign in \( g(I_h) \), so there is no root of \( g(I_h) \) in the interval \( \frac{A_h}{\mu_1 + \mu_h + \gamma_h} < I_h < \frac{\Lambda_v}{\mu_1} \). Hence we can conclude that there is at least one root of \( g(I_h) = 0 \) in the interval \( 0 < I_h < \frac{\Lambda_v}{\mu_1 + \mu_h + \gamma_h} \). Thus under the
following condition, there exists unique positive root of \( g(I_h) = 0 \) and we name that root as \( I_h^* \). Hence we get our endemic equilibrium point as \( E_1 = (N_h^*, I_h^*, R_h^*, N_v^*, I_v^*) \).

\[
\frac{d g(I_h)}{dt} = -[A_h - (\mu_h + \mu + \gamma_h)I_h]\left[\frac{c\beta_h\mu_h(1+I_h) + c\beta_v\mu_v}{(1+I_h)\mu_h} + \frac{\beta_h^2\mu^2_h}{(\mu_h\mu_v(\lambda_h - \mu_v)\mu_v^2)}\right] - (\mu_1 + \mu_h + \gamma_h)\left[\frac{c\beta_h(\lambda_h - \mu_v)I_h}{\mu_h(1+I_h)\mu_h} + \frac{\beta_h^2\mu^2_h}{(\mu_h\mu_v(\lambda_h - \mu_v)\mu_v^2)}\right] + (\mu_1 + \mu_h + \gamma_h) < 0.
\]

4. Stability analysis

4.1 Local stability of disease-free equilibrium

**Theorem 4.1.1** When \( R_0 < 1 \), the disease free equilibrium \( E_0 \) is locally asymptotically stable otherwise it is unstable.

The Jacobian matrix of the system (2) at disease free equilibrium point \( E_0 \) is obtained as follows:

\[
J_0 = \begin{pmatrix}
-\mu_h & -\mu_1 & 0 & 0 & 0 \\
0 & m_{22} & 0 & 0 & \beta_h^2 \\
0 & \gamma_h & -\mu_h & 0 & 0 \\
0 & 0 & 0 & -\mu_v & 0 \\
0 & \beta_v \frac{N_v^0}{N_h} & 0 & 0 & -\mu_v \\
\end{pmatrix}
\]

Where

\[
m_{22} = \beta_h^1 \frac{\lambda_h}{\mu_h} - (\gamma_h + \mu_h + \mu_1)
\]

Clearly three eigenvalues of the matrix \( J_0 \) are \( -\mu_h, -\mu_h \) and \( -\mu_v \) and the remaining two roots are obtained by solving the following equation.

\[
\lambda^2 + (\mu_v - m_{22})\lambda - (m_{22}\mu_v + \beta_h^2 \beta_v \frac{N_v^0}{N_h}) = 0,
\]

Where

\[
m_{22} = \beta_h^1 \frac{\lambda_h}{\mu_h} - (\gamma_h + \mu_h + \mu_1) = \frac{\beta_h^1 \frac{\lambda_h}{\mu_h}}{\gamma_h + \mu_h + \mu_1} - 1 = R_{hh} - 1 < 0 \text{ for } R_{hh} < 1.
\]

Hence \( (\mu_v - m_{22}) > 0 \)

Now.

\[-(m_{22}\mu_v + \beta_h^2 \beta_v \frac{N_v^0}{N_h^0}) = \mu_v (\gamma_h + \mu_h + \mu_1)[1 - (R_{hh} + R_{hv})] > 0 \text{ for } R_{hh} + R_{hv} < 1.
\]

It is observed that \( R_{hh} + R_{hv} < 1 \), whenever \( R_0 < 1 \). Hence the disease free equilibrium is locally asymptotically stable.

4.2 Global stability of disease-free equilibrium

To prove the global stability of disease free equilibrium, we are using the theorem described in [15,16]

**Theorem 4.2.1** If the given mathematical model can be written in the form:

\[
\frac{dX}{dt} = F(X,Y), \text{ and } \frac{dY}{dt} = G(X,Y), G(X,0) = 0 \quad (*)
\]
where, \( X = (N_h, N_v)^T \) and \( Y = (I_h, I_v)^T \), represent the classes of uninfected and the class of infected individuals respectively. Then the DFE is represented here by \( E_0 = (X_0, 0) = \left( \frac{\lambda_h}{\mu_h}, \frac{\lambda_v}{\mu_v}, 0, 0 \right) \).

For the global asymptotic stability of \( E_0 \), the condition (H1) and (H2) given below must be satisfied.

\( H_1: \frac{dX}{dt} = F(X_0, 0), \) \( X_0 \) is global asymptotically stable,

\( H_2: G(X, Y) = AY - \hat{G}(X, Y), \) \( \hat{G}(X, Y) \geq 0, \) here \( A = D_Y G(X_0, 0) \) is \( M \)-matrix (In \( M \)-matrix, all the off diagonal element of matrix are non-negative). If the system (2) satisfies the given condition in (*) then the equilibrium point \( E_0 = (X_0, 0) \) is a global asymptotically stable equilibrium provided \( R_0 < 1 \).

And for our mathematical model, the result is shown in the next theorem, as given below.

**Theorem 4.2.2** The equilibrium point \( E_0 = (X_0, 0) \) is GAS (Global Asymptotically Stable), provided \( R_0 < 1 \) and the conditions given in (*) are satisfied.

Proof: By using Theorem (4.2) to mathematical model system, we consider

\[ F(X_0, 0) = \Lambda - \mu S, \quad G(X, Y) = AY - \hat{G}(X, Y) \]

where,

\[ A = \begin{bmatrix}
\beta_{h1}c\left(\frac{N_v}{N_h}\right) - (\gamma_h + \mu_h + \mu_1) & \beta_{h2} \\
\beta_{v}\frac{N_v}{N_h} & -\mu_v
\end{bmatrix} \]

And

\[ \hat{G}(X, Y) = \begin{bmatrix} \hat{G}_1(X, Y) \\ \hat{G}_2(X, Y) \end{bmatrix} = \begin{bmatrix} \left(\beta_{h1}\frac{cI_h}{1 + I_h} + \beta_{h2}\frac{I_v}{N_h}\right)(N_h - I_h - R_h) \\ \beta_{v}\frac{I_v}{N_h}(N_v - I_v) \end{bmatrix} \]

Here we can easily observe that \( \hat{G}_1(X, Y) > 0, \hat{G}_2(X, Y) > 0 \), hence \( \hat{G}(X, Y) > 0 \) for all \( (X, Y) \). Also by the definition of \( M \) matrix we can say that the matrix \( A \) is \( M \) matrix. Hence the DFE \( (E_0) \) is globally stability.

### 4.3 Local stability of endemic equilibrium point

**Theorem 4.3.1** When \( R_0 > 1 \) then endemic equilibrium \( E_1 \) is locally asymptotically stable under the condition \( \left( \frac{N_v - I_v^*}{N_h} \right) > \frac{\mu_1}{\beta_v} \).

Proof: The jacobian matrix \( J_1 \) at the endemic equilibrium point \( E_1 \) is given by

\[ J_1 = \begin{bmatrix}
-\mu_h & -\mu_1 & 0 & 0 & 0 \\
m_{21} & m_{22} & m_{23} & 0 & m_{25} \\
0 & \gamma_h & -\mu_h & 0 & 0 \\
0 & 0 & 0 & -\mu_v & 0 \\
m_{51} & m_{52} & 0 & m_{54} & m_{55}
\end{bmatrix} \]

where,

\[ m_{21} = \beta_{h1}\frac{cI_h^*}{1 + I_h} + \beta_{h2}\frac{I_v^*(I_h + R_h)}{N_h^2} \]
Clearly, the two roots of the matrix \( J_1 \) are \(-\mu_h, -\mu_v\) and the remaining roots are obtained by solving the following equation.

\[
\lambda^3 + A\lambda^2 + B\lambda + C = 0
\]

where,

\[
A = \mu_h - m_{22} - m_{55} > 0
\]

\[
B = - (\mu_h m_{22} + \mu_h m_{55} - m_{22} m_{55} + \gamma_h m_{23} + m_{23} m_{52} - \mu_1 m_{21}) > 0
\]

\[
C = (\mu_h m_{22} m_{55} + \gamma_h m_{23} m_{55} - \mu_h m_{25} m_{52} - \mu_1 m_{21} m_{55} + \mu_1 m_{51} m_{25}) > 0
\]

Therefore, by Routh Hurwitz’s theorem all the three roots of the cubic equation are negative or have negative real parts, provided \( AB - C > 0 \), we get

\[
AB - C = \mu_1 \mu_h m_{21} - \mu_1 \mu_h (m_{21} + m_{55}) + \mu_h (4m_{22} m_{55} - \gamma_h m_{23} - m_{23} m_{52} + m_{22}^2 + m_{55}^2 - m_{25} m_{52}) + \gamma_h (m_{22} m_{23} + 2m_{23} m_{55}) + \mu_1 (-m_{21} m_{22} - 2m_{21} m_{55} + m_{51} m_{25}) + m_{23} m_{52}(m_{22} + m_{55}) - m_{22} m_{55}(m_{22} + m_{55})
\]

which is positive under the condition \( \beta_v \left( \frac{N^*_h - I^*_h}{N^*_h} \right) > \mu_1 \). Hence, the endemic equilibrium is locally asymptotically stable.

**5. Numerical simulation:**

To support our analytical results, we perform numerical simulation. Here most of the parameters values are taken from the reference [6, 9] and some parameter values are assumed. In the proposed model, all the parameter are in per day. The values of the parameters are listed in the following Table 1.
Table 1. Parameters and their values

| Parameter | Value (Day) |
|-----------|-------------|
| $\Lambda_h$ | 20 |
| $\Lambda_v$ | 20(DFE), 40 (EE) |
| $\beta_{h1}$ | 0.005 |
| $\beta_{h2}$ | 0.4 |
| $\mu_h$ | $1/ (60\times365)$ |
| $\mu_v$ | $1/14$ |
| $\gamma_h$ | $1/20$ |
| $\beta_v$ | 0.5 |
| $\mu_1$ | 0.04227 |

To demonstrate the stability of disease-free equilibrium point, we consider $\Lambda_v = 20$ and all other parameters as listed in the Table 1. For this set of parameters $R_0 = 0.8802$ and disease free equilibrium point $E_0$ is $(2100, 0, 0, 600, 0)$. In the Fig. 2 is demonstrating the stability of disease free equilibrium point $E_0$. The stability of the endemic equilibrium $E_1$ is demonstrated by changing the value of $\Lambda_v$ from 20 to 30. For this set of parameters, the basic reproduction number $R_0 = 2.1347$ and equilibrium point $E_1$ is $(97, 200, 68, 100, 250)$. This is demonstrated in Figure 3. In Figure 4, variation of $I_h$ with time is shown for different values of $\gamma_h$.

Figure 2: Variation of $S_h, I_h, R_h, S_v, I_v$ with time showing the stability of disease-free equilibrium point.
Figure 3: Variation of $S_h, I_h, R_h, S_v, I_v$ with time showing the stability of endemic equilibrium point.

Figure 4: Variation of $I_h$ with time for different values of $\gamma_h$.

6. Optimal control model

In this section we have extended the mathematical model (2) to optimal control problem by including two optimal control parameters $u_1$ and $u_2$. If $u_1$ and $u_2$ are equal to zero, then there is no effort being placed in these controls at time $t$ and if they are equal to one then maximum effort is applied. The control variable $u_1$ represents the use of insecticide-treated bed nets and the use of mosquito repulsive lotions and electronic devices, to reduce mosquito biting rate. The control variable $u_2$ corresponds to additional death rate of mosquitoes due to control efforts. Based on the above assumptions, the optimal control model as follows:
The optimal control problem

Here, with the help of optimal control theory we analyze the behavior of our system. The objective functional for fixed time $t_f$ is given below:

$$J = \int_0^{t_f} \left[ (A_1 I_h + A_2 S_v + I_v) + \frac{1}{2} A_3 u_1^2 + \frac{1}{2} A_4 u_2^2 \right] dt$$

Here the parameter $A_1 \geq 0, A_2 \geq 0, A_3 \geq 0, A_4 \geq 0$, and they represent the weight constants.

Here we aim to find the optimal controls parameters $u_1^*$ and $u_2^*$ such that

$$f(u_1^*, u_2^*) = \min_{u_1, u_2 \in \Omega} f(u_1, u_2),$$

Where, $\Omega = \{u_1, u_2 : measurable \ and \ 0 \leq u_1, u_2 \leq 1 \ and \ t \in [0, t_f]\}$

The Lagrangian of this problem is defined as:

$$L(I_h, S_v, I_v, u_1, u_2) = A_1 I_h + A_2 S_v + I_v + \frac{1}{2} A_3 u_1^2 + \frac{1}{2} A_4 u_2^2$$

The Hamiltonian of this problem defined as:

$$H = L(I_h, S_v, I_v, u_1, u_2) + \lambda_1 \frac{dS_h}{dt} + \lambda_2 \frac{dI_h}{dt} + \lambda_3 \frac{dR_h}{dt} + \lambda_4 \frac{dS_v}{dt} + \lambda_5 \frac{dI_v}{dt}$$

where $\lambda_i$ are adjoint variables and $i = 1 \ to \ 5$ and the adjoint variables in the form of differential equations are given as follows:

$$\frac{d\lambda_1}{dt} = \lambda_1 \mu_h + \beta_h \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \frac{d\lambda_2}{dt} = -A_1 \beta_h S_h \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \lambda_3 \frac{dS_h}{dt} + \lambda_4 \frac{dI_h}{dt} + \lambda_5 \frac{dR_h}{dt} + \lambda_6 \frac{dS_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

$$+ (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$

$$+ \gamma (\lambda_2 - \lambda_3) + (\mu_h + \mu_4) \lambda_2 + (1 - u_1) \beta_h S_v \left( \frac{c I_h}{1 + I_h} \right) (\lambda_1 - \lambda_2) + (1 - u_1) \beta_h I_v (\lambda_1 - \lambda_2)$$
Let \( \tilde{\lambda} \), be the optimum values reprehensively, and the solution of the system (3) be \( \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5 \).

**Theorem 6.1.1** The given optimal controls \( u_1^*, u_2^* \in \Omega \) such that \( J(u_1^*, u_2^*) = \min J(u_1, u_2) \) subject to system (3).

**Proof:** To prove this theorem we follow the method described in Lenhart et al.[17] and Pontryagin [18]. Here the state variables and the controls are positive. For this minimizing problem, the necessary convexity of the objective functional in \((u_1, u_2)\) is satisfied. The control variable set \( \Omega \) is also convex and closed by the definition. The integrand of the functional \( A_1 l_h + A_2 (S_v + I_v) + \frac{1}{2} A_3 u_1^2 + \frac{1}{2} A_4 u_2^2 \) is convex on the control set \( \Omega \) and the state variables are bounded.

Since there exist optimal controls for minimizing the functional subject to equations (3), we use Pontryagin’s maximum principle [18] to derive the necessary conditions to find the optimal solutions as follows:

If \((x,u)\) is an optimal solution of an optimal control problem, then there exist a non-trivial vector function \( \lambda = \lambda_1, \lambda_2, \lambda_3, \ldots, \lambda_n \) satisfying the following equalities

\[
\frac{d\lambda_3}{dt} = \lambda_3 \mu_h + (1 - u_1) \beta h^2 S_v \frac{I_v}{N_h} \frac{S_h (\lambda_5 - \lambda_4)}{N_h} + (1 - u_3) \beta h^2 S_v \frac{I_v}{N_h} \frac{S_h (\lambda_5 - \lambda_4)}{N_h}
\]

\[
\frac{d\lambda_4}{dt} = -A_2 + \lambda_4 (\mu_h + u_2) + (1 - u_4) \beta \frac{I_v}{N_h} (\lambda_4 - \lambda_5)
\]

\[
\frac{d\lambda_5}{dt} = -A_2 + \lambda_4 (\mu_h + u_2) + (1 - u_4) \beta h^2 S_v \frac{S_h}{N_h} (\lambda_1 - \lambda_2)
\]

Let \( \tilde{S}_h, \tilde{l}_h, \tilde{R}_h, \tilde{S}_v, \tilde{I}_v \), be the optimum values of \( S_h, l_h, R_h, S_v, l_v \) reprehensively, and the solution of the system (3) be \( \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5 \).

**Theorem 6.1.2** The given controls \((u_1^*, u_2^*)\) which minimizes \( J \) over the region \( \Omega \) given by

\[
u_1^* = \min \{1, \max (0, \tilde{u}_1)\}
\]

\[
u_2^* = \min \{1, \max (0, \tilde{u}_2)\}
\]

**Proof:** To prove the theorem we use Pontryagin’s maximum principle [18]

Using optimally condition:

\[
\frac{\partial H}{\partial u_1} = 0, \quad \frac{\partial H}{\partial u_2} = 0
\]

we get,

\[
\frac{\partial H}{\partial u_1} = A_3 u_1 + \beta h^2 S_v \frac{I_v}{N_h} (\lambda_1 - \lambda_2) + \beta \frac{S_v}{N_h} \frac{I_v}{N_h} (\lambda_4 - \lambda_5) = 0
\]
This implies
\[ u_1 = \frac{\beta h_2 S_h N_h (\lambda_1 - \lambda_2) + \beta v S_v N_v (\lambda_4 - \lambda_5)}{A_3} \]

And
\[ \frac{\delta H}{\delta u_2} = A_4 u_2 - S_h^* \lambda_4 - I_h^* \lambda_5 = 0 \]

This implies
\[ u_2 = \frac{S_h^* \lambda_4 + I_v^* \lambda_5}{A_4} \]

Again lower and upper bounds for these controls are 1 and 0 respectively. i.e. \( u_1 = u_2 = 0 \) if \( u_1 < 0 \) and \( u_2 < 0 \), and \( u_1 = u_2 = 1 \) if \( \bar{u}_1 > 1 \) and \( \bar{u}_2 > 1 \), otherwise \( u_1 = \bar{u}_1 \) and \( u_2 = \bar{u}_2 \). Hence for these controls \( u_1^*, u_2^* \) we get optimum value of the function \( J \).

7. Simulation of optimal control:
The optimal control model is simulated using MATLAB by considering the set of parameters which corresponds to the stability of endemic equilibrium point of the model (2). The weight constants are taken as follows:
\[ A_1 = 1, A_2 = 1, A_3 = 55, A_4 = 75. \]

The time interval is considered as \([0,150]\). The system (3) is solved by iterative method with the help of forward and backward difference approximation. Here Figures 5-6 are showing the control profiles of the controls \( u_1 \) and \( u_2 \) respectively. Finally, to see the effects of optimal controls, in ‘figure 7’ and ‘figure 8’, the infected human and infected vectors \( I_h \) and \( I_v \) are plotted against time with and without control. It is easy to observe that optimal control is very much effective in reducing the number of infectives in the desired interval of time.

![Figure 5: The control profile of \( u_1(t) \).](image)
Figure 6: The control profile of $u_2(t)$.

Figure 7: The variation of infected human against time with and without control.
Figure 8: The variation of infected mosquito against time with and without control.

8. Conclusion
In this paper, we have proposed and analyzed a mathematical model for Zika virus disease. The existence of disease free equilibrium and endemic equilibrium are discussed in details. The basic reproduction number $R_0$ of the proposed model is computed. The disease free equilibrium is globally asymptotically stable whenever the basic reproduction number $R_0 < 1$. The endemic equilibrium point is locally asymptotically stable under some restriction on parameters. Further we extend our model to optimal control problem. Numerical simulations of original model and the optimal control problem suggest that optimal control strategies are the best in reducing the number of infectives in desired interval of time.

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