IOSR Journal of Mathematics (IOSR-JM)
e-ISSN: 2278-5728, p-ISSN: 2319-765X. Volume 12, Issue 6 Ver. IV (Nov. - Dec.2016), PP 21-33
www.iosrjournals.org

Mathematical Analysis of Zika Epidemic Model

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Abstract: *Zika fever has, in recent times, become a scourge to human. This is so especially because there is no specific treatment or vaccine currently available. However, we present a five compartmental mathematical model, which studies the dynamical spread of Zika fever within humans (host) and between humans and mosquitoes (vector).The mathematical analysis shows that the disease free and the endemic equilibrium point of the model exist. The model has disease free equilibrium point which is locally asymptotically stable (LAS) whenever the basic reproduction number is less than unity i.e.* $(R_0 < 1)$ *and unstable when* $R_0 > 1$. *On the other hand, the endemic equilibrium, which was computed numerically, is unstable. Numerical simulation was carried out by maple software using differential transformation method to show the effects of recovery (as a result of symptoms treatment) (γ), isolation (ρ), and vector elimination (δ_2) on the spread of Zika fever. Our numerical results showed that increasing the recovery to a very high rate has significant effect in reducing infection; and isolation of infected individuals also reduces the transmission of the ZikV infection. Also, the rate of human-induced deaths of mosquito, if increased, will result in the elimination of the vector (mosquitoes) and hence prevention in transmission of ZikV infection is ensured.*

Keywords: *Zika fever, ZikV, Isolation, Critical points, Basic reproduction number, Stability*

I. Introduction

Zika fever (also known as Zika virus disease) is an illness caused by the Zika virus. The disease is spread through the bite of daytime-active Aedes mosquitoes such as the *A. aegypti* and *A. albopictus* (these are the same mosquitoes that spread dengue and chikungunya viruses) [4] [16]. Its name comes from Zika forest in Uganda, where the virus was first isolated from a rhesus monkey in 1947[3] [11]. The first human cases were reported in Nigeria in 1954[4]. The first documented outbreak among people occurred in 2007, in the Federated State of Micronesia [4] [16]. As of January 2016, the disease was occurring in 20 regions of the Americas [9]. It is estimated that about 1.5 million people have been infected by Zika in Brazil with over 3500 cases of microcephaly reported between October 2015 and January 2016 [5] [9]. It is also known to occur in Africa, Asia and the Pacific[7]. Due to the outbreak which started in Brazil in 2015, the World health Organization has declared Zika fever a public health emergency of international concern [11] [16].

The disease of Zika virus is transmitted from infected Aedes mosquitoes to humans through mosquito bites [1] [16]. It can also be transmitted from human to human through the blood and semen of an infected human [14], and through an infected pregnant woman to the foetus [9]. Zika is a cause of microcephaly and other severe brain defects [10]. The incubation period (the time from exposure to symptoms) of Zika virus disease is not clear, but is likely to be a few days to a week. The symptoms are similar to other arbovirus infection such as dengue, and include fever, skin rashes, conjunctivitis (red eyes), muscle and joint pain, malaise and headache. These symptoms are usually mild and usually last from 2 – 7 days [1] [4] [16].

There is no specific treatment or vaccine currently available for Zika virus disease [1]. Prevention and control relies on reducing mosquitoes through source reduction (removal and modification of breeding sites), and reducing contacts between mosquitoes and people. Since there is no specific treatment currently, the symptoms can be treated [1]. Most people fully recover without severe complications, and hospitalization rates are low [1] [4]. Once a person is infected and has recovered, he or she is likely to be protected from future infections [1]. Till date, disease related deaths are rare. However, there is scientific consensus that Zika virus is a cause of complications such as microcephaly (a birth defect where a baby's head and/or brain is smaller than expected) and Guillain-Barre syndrome (a neurological disorder that could lead to paralysis and death) [10] [15].

Mathematical models for transmission dynamics of mosquito-borne diseases can be useful in providing better insights into the behaviour of this disease. The models have played great roles in influencing the decision making processes regarding intervention strategies for preventing and controlling the insurgence of mosquito-borne diseases [6].Kucharski, *et al.*[5] suggested that ZikV may exhibit similar dynamics to dengue virus in Island population, with transmission characterized by large sporadic outbreaks with a high proportion of asymptomatic or unreported cases. Perkins and his colleagues developed and applied a method that leverages highly spatially

resolved data about drivers of Zika virus transmission to project that millions of infections in childbearing women and all demographic strata could occur before the first wave of the epidemic concludes and that tens of thousands of pregnancies would be affected [9].The present study looks into how we can best slow or prevent the outbreak of Zika virus disease and protect vulnerable populations.

The rest of this work is organized as follows: we give a full description of the model and show a domain where the model is epidemiologically well posed in Section 2. Section 3 provides the existence of equilibria including a derivation of the basic reproduction number and stability analysis of the equilibria. In Section 4, we perform numerical simulations of the model with graphical illustrations and their discussion, and give concluding remark in Section 5.

II. Model Formulation

To study the transmission and spread of malaria in two interacting population of humans (the host) and mosquitoes (the vector), we formulate a model which subdivides the total human population size at time t, denoted by $N_h(t)$, into susceptible humans $S_h(t)$, infectious humans $I_h(t)$ and isolated humans $Q(t)$. Hence, we have:

$$N_h(t) = S_h(t) + I_h(t) + Q(t).$$

The mosquito population is divided into two subclasses: susceptible mosquitoes $S_m(t)$ and infectious mosquitoes $I_m(t)$. Thus, the total size of the mosquito population at any time t is denoted by:

$$N_m(t) = S_m(t) + I_m(t).$$

The transmission of ZikV between human and mosquito is governed by some basic epidemiological parameters. Susceptible individuals are recruited into the human population either by birth or immigration at a rate β_1, and that of mosquito population at a rate β_2. When a mosquito carrying ZikV bite a susceptible human, the virus is passed unto the human and the person moves to the infected class $I_h(t)$ at a rate τ_1 (the model did not include the exposed classes for both human and mosquitoes. It is assumed that the time from infection and onset of the symptomatic disease is small and negligible). Similarly, when an uninfected mosquito bites an infectious human, it carries the virus and so moves to the infected class $I_m(t)$ at a rate τ_3.

ZikV can also be transmitted from human to human through the blood and semen of an infected human. The rate of this transmission is denoted as τ_2. The fraction of births that are infected is denoted as θ. The human natural and disease-induced death rates are denoted respectively as μ_1 and δ_1. The average life of mosquitoes is $1/\mu_2$, where μ_2 is the mosquito death rate and it is assumed that there is no mosquito mortality due to presence of virus. However there is an additional human-induced death for mosquitoes, denoted as δ_2. Other parameters are as given in table 2.1.

The figure 2.1 below shows the dynamics of the model with the inflow and outflow in each compartment of both populations.

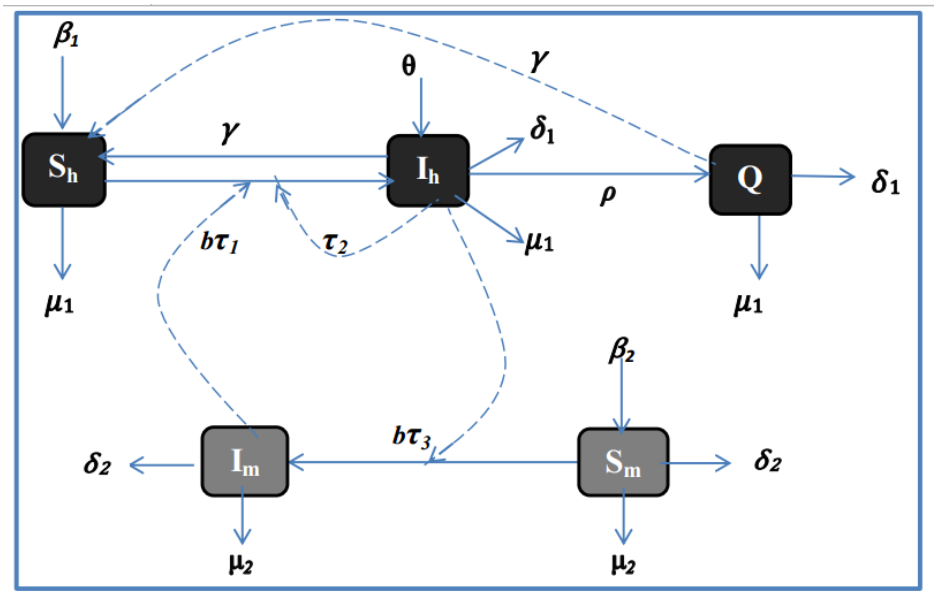

Figure2.1: The compartmental model of Zika virus transmission between humansand mosquitoes

The model is formulated as a system of coupled ordinary differential equation as:

$$\frac{dS_h}{dt} = \beta_1(1-\theta I_h) - \mu_1 S_h - (b\tau_1 I_m + \tau_2 I_h)S_h + \gamma(I_h + Q)$$

$$\frac{dI_h}{dt} = (b\tau_1 I_m + \tau_2 I_h)S_h + \theta\beta_1 I_h - (\mu_1 + \delta_1 + \gamma)I_h - \rho I_h$$

$$\frac{dQ}{dt} = \rho I_h - (\mu_1 + \delta_1 + \gamma)Q \qquad (2.1)$$

$$\frac{dS_m}{dt} = \beta_2 - (\mu_2 + \delta_2)S_m - b\tau_3 I_h S_m$$

$$\frac{dI_m}{dt} = b\tau_3 I_h S_m - (\mu_2 + \delta_2)I_m;$$

together with the initial conditions:

$$S_h(t_0) = S_{h0},\ I_h(t_0) = I_{h0},\ Q(t_0) = Q_0,\ S_m(t_0) = S_{m0},\ I_m(t_0) = I_{m0};$$

The state variables and parameters used for the transmission model are described in the following table:

Table 2.1: Description of Variables and parameters used in the model

State Variables and Parameters	Description
$S_h(t)$	Number of humans susceptible to ZikVinfection at time t
$I_h(t)$	Number of infected humans at time t
$Q(t)$	Number of isolated humans at time t
$S_m(t)$	Number of susceptible mosquitoes at time t
$I_m(t)$	Number of infected mosquitoes at time t
N_h	Total human population
N_m	Total mosquitoes population
β_1, β_2	Recruitment term of the susceptible humans and mosquitoes
b	Biting rate per human per mosquito
τ_1	Probability that a bite by an infectious mosquito results in transmission of disease to human
τ_2	Transmission coefficient from contact between susceptible and infected humans
τ_3	Probability that a bite results in transmission of parasite to a susceptible mosquito
θ	Fraction of births that are infected
ρ	Per capital rate of progression of humans from the infected state to the isolated state
γ	Per capital recovery rate for humans from the infectious and isolated states to the susceptible state
μ_1, μ_2	Human and mosquito natural death rates
δ_1	Disease-induced death rate for humans
δ_2	Extra human-induced death for mosquitoes

Existence and Uniqueness of Solution

THEOREM 2.1 **[2]:** Let

$$\begin{aligned} x_1' &= f_1(x_1, x_2, \ldots x_n, t), x_1(t_0) = x_{10} \\ x_2' &= f_2(x_1, x_2, \ldots x_n, t), x_2(t_0) = x_{20} \\ &\vdots \\ x_n' &= f_n(x_1, x_2, \ldots x_n, t), x_n(t_0) = x_{n0}. \end{aligned} \qquad (2.2)$$

Suppose D is the region in (n+1)-dimensional space (one dimension for t and n dimensions for the vector $\underline{x}$). If the partial derivatives $\frac{\partial f_i}{\partial x_j}, i, j = 1,2,\ldots,n$ are continuous in

$$D = \{(x,t) : |t - t_0| \le a, |x - x_0| \le b\},$$

then there is a constant $\delta > 0$ such that there exists a unique continuous vector solution

$\underline{x} = [x_1(t), x_2(t), ..., x_n(t)]$ in the interval $|t - t_0| \le \delta$.

***THEOREM 2.2*:** Let

$$
\begin{aligned}
f_1 &= \frac{dS_h}{dt} = \beta_1(1-\theta I_h) - \mu_1 S_h - (b\tau_1 I_m + \tau_2 I_h)S_h + \gamma(I_h + Q); && S_h(t_0) = S_{h0} \\
f_2 &= \frac{dI_h}{dt} = (b\tau_1 I_m + \tau_2 I_h)S_h + \theta\beta_1 I_h - (\mu_1 + \delta_1 + \gamma)I_h - \rho I_h; && I_h(t_0) = I_{h0} \\
f_3 &= \frac{dQ}{dt} = \rho I_h - (\mu_1 + \delta_1 + \gamma)Q; && Q(t_0) = Q_0 \\
f_4 &= \frac{dS_m}{dt} = \beta_2 - (\mu_2 + \delta_2)S_m - b\tau_3 I_h S_m; && S_m(t_0) = S_{m0} \\
f_5 &= \frac{dI_m}{dt} = b\tau_3 I_h S_m - (\mu_2 + \delta_2)I_m; && I_m(t_0) = I_{m0};
\end{aligned}
\qquad (2.3)
$$

$$
D = \{(S_h, I_h, Q, S_m, I_m) : |S_h - S_{h0}| \le a, |I_h - I_{h0}| \le b, |Q - Q_0| / \le c, |S_m - S_{m0}| \le d, |I_m - I_{m0}| \le e, |t - t_0| \le f\}.
$$

Then equation (2.3) has a unique solution.

***Proof*:**

We find the partial derivatives, evaluated at the origin thus:

$$
\left.\frac{\partial f_1}{\partial S_h}\right|_{(0,0,0,0,0)} = -\mu \qquad \left.\frac{\partial f_2}{\partial S_h}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial S_h}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_1}{\partial I_h}\right|_{(0,0,0,0,0)} = -\theta\beta_1 + \gamma \quad \left.\frac{\partial f_2}{\partial I_h}\right|_{(0,0,0,0,0)} = \theta\beta_1 - (\mu_1 + \delta_1 + \gamma - \rho) \quad \left.\frac{\partial f_3}{\partial I_h}\right|_{(0,0,0,0,0)} = \rho
$$

$$
\left.\frac{\partial f_1}{\partial Q}\right|_{(0,0,0,0,0)} = \gamma \qquad \left.\frac{\partial f_2}{\partial Q}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial Q}\right|_{(0,0,0,0,0)} = -(\mu_1 + \delta_1 + \gamma)
$$

$$
\left.\frac{\partial f_1}{\partial S_m}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial S_m}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial S_m}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_1}{\partial I_m}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial I_m}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial I_m}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_4}{\partial S_h}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial S_h}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_4}{\partial I_h}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial I_h}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_4}{\partial Q}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial Q}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_4}{\partial S_m}\right|_{(0,0,0,0,0)} = -(\mu_2 + \delta_2) \qquad \left.\frac{\partial f_5}{\partial S_m}\right|_{(0,0,0,0,0)} = 0
$$

$$
\left.\frac{\partial f_4}{\partial I_m}\right|_{(0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial I_m}\right|_{(0,0,0,0,0)} = -(\mu_2 + \delta_2).
$$

Therefore $\left|\frac{\partial f_i}{\partial x_j}\right|, i, j = 1,2,...,5$ are continuous and bounded. Hence, following Derrick and Grossman [2] of theorem 2.1 above, the problem (2.3) has a unique solution and so the model (2.1) is both epidemiologically feasible and mathematically well posed.

III. Mathematical Analysis of The Model

In this section we carry out qualitative analysis of the model (2.1) to investigate existence and stability of the steady states.

3.1 Existence of Disease-free Equilibrium Point, E_0

Disease-free equilibrium points are steady-state solutions where there is no ZikV infection (i.e. $I_h = I_m = 0$), while the steady-state solution of the system (2.1) is obtained by setting

$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dQ}{dt} = \frac{dS_m}{dt} = \frac{dI_m}{dt} = 0$ in (2.1).

Thus, the disease-free equilibrium point, E_0, for the ZikV model (2.1) yields

$$E_0 = \left(\frac{\beta_1}{\mu_1}, 0, 0, \frac{\beta_2}{\mu_2 + \delta_2}, 0\right) \qquad (2.4)$$

3.2 Derivation of Basic Reproduction Number, R_0

An important notion in epidemiological models is the basic reproduction number, usually denoted by R_0. It is a threshold value that is often used to measure the spread of a disease. It is defined as the number of secondary infections in humans that arise as a result of a single infected individual being introduced in a fully susceptible population. When $R_0<1$, it implies that on average an infectious individual infects less than one person throughout his/her infectious period and in this case the disease is wiped out. On the other hand, when $R_0>1$, then on average every infectious individual infects more than one individual during his/her infectious period and the disease persists in the population.

The derivation of basic reproduction number is essential in order to assess the local stability of the system (2.1). To do this, we employ the method of next generation matrix described by Driessche and Watmough[13].

On the assumption that all blood are screened prior to transfusion and protected sex are practiced in ZikV endemic areas, we consider the reduced system for the infectious states, with neither human–human transmissionnor infection from birth (i.e. $\tau_2 = \theta = 0$). i.e.:

$$\begin{aligned}
\frac{dI_h}{dt} &= b\tau_1 I_m S_h - (\mu_1 + \delta_1 + \gamma) I_h - \rho I_h \\
\frac{dQ}{dt} &= \rho I_h - (\mu_1 + \delta_1 + \gamma) Q \qquad (2.5) \\
\frac{dI_m}{dt} &= b\tau_3 I_h S_m - (\mu_2 + \delta_2) I_m;
\end{aligned}$$

Then we have the transmission and transition matrices to be given respectively as

$$\mathcal{F} = \begin{pmatrix} b\tau_1 I_m S_h \\ 0 \\ b\tau_3 I_h S_m \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} (\mu_1 + \delta_1 + \gamma) I_h + \rho I_h \\ (\mu_1 + \delta_1 + \gamma) Q - \rho I_h \\ (\mu_2 + \delta_2) I_m \end{pmatrix}.$$

The Jacobian matrices for $\mathcal{F}$ and $\mathcal{V}$ at DFE (E_0) are evaluated as follows:

$$F = D\mathcal{F}\big|_{E_0} = \begin{pmatrix} 0 & 0 & \frac{b\beta_1\tau_1}{\mu_1} \\ 0 & 0 & 0 \\ \frac{b\beta_2\tau_3}{\mu_2 + \delta_2} & 0 & 0 \end{pmatrix}, \quad \text{and}$$

$$V = D\mathcal{V}|_{E_0} = \begin{pmatrix} \mu_1+\delta_1+\gamma+\rho & 0 & 0 \\ -\rho & \mu_1+\delta_1+\gamma & 0 \\ 0 & 0 & \mu_2+\delta_2 \end{pmatrix}.$$

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu_1+\delta_1+\gamma+\rho} & 0 & 0 \\ \frac{\rho}{(\mu_1+\delta_1+\gamma+\rho)(\mu_1+\delta_1+\gamma)} & \frac{1}{\mu_1+\delta_1+\gamma} & 0 \\ 0 & 0 & \frac{1}{\mu_2+\delta_2} \end{pmatrix}$$

$$F.V^{-1} = \begin{pmatrix} 0 & 0 & \frac{b\beta_1\tau_1}{\mu_1(\mu_2+\delta_2)} \\ 0 & 0 & 0 \\ \frac{b\beta_2\tau_3}{\mu_2(\mu_1+\delta_1+\gamma+\rho)} & 0 & 0 \end{pmatrix}$$

Now, the basic reproduction number,which equals $\boldsymbol{\rho}(F.V^{-1})$, is obtained as the spectra radius (i.e. the dominant eigenvalue) of the product $F.V^{-1}$ thus:

$$\rho\left(F.V^{-1}\right) = \sqrt{\frac{b^2\beta_1\beta_2\tau_1\tau_3}{\mu_1\mu_2(\mu_1+\delta_1+\gamma+\rho)(\mu_2+\delta_2)}} = \tilde{R}_0.$$

This quantity gives the basic reproduction number. However, the notation can be simplified by setting $R_0 = \tilde{R}_0^2$.

Thus

$$R_0 = \frac{b^2\beta_1\beta_2\tau_1\tau_3}{\mu_1\mu_2(\mu_1+\delta_1+\gamma+\rho)(\mu_2+\delta_2)} \tag{2.6}$$

$$\Rightarrow R_0 = \frac{b\beta_1\tau_1}{\mu_1(\mu_1+\delta_1+\gamma+\rho)}.\frac{b\beta_2\tau_3}{\mu_2(\mu_2+\delta_2)} = R_1.R_2$$

where

$$R_1 = \frac{b\beta_1\tau_1}{\mu_1(\mu_1+\delta_1+\gamma+\rho)} \quad \text{and} \quad R_2 = \frac{b\beta_2\tau_3}{\mu_2(\mu_2+\delta_2)}$$

are the reproduction numbers associated with the transmission coefficients τ_1 and τ_3 respectively.

3.3 Stability of Disease-free Equilibrium Point

***THEOREM 3.1*:**

If $R_0 < 1$,then the disease-free equilibrium is locally asymptotically stable. Otherwise, it is unstable.

***Proof*:** The stability of the disease-free equilibrium is determined by the eigenvalues of the Jacobian matrix of the full system (2.1)(with $\tau_2 = \theta = 0$), evaluated at the disease-free equilibrium point, given by

$$J\big|_{E_0} = \begin{pmatrix} -\mu_1 & \gamma & \gamma & 0 & -\frac{b\beta_1\tau_1}{\mu_1} \\ 0 & -(\mu_1+\delta_1+\gamma+\rho) & 0 & 0 & \frac{b\beta_1\tau_1}{\mu_1} \\ 0 & \rho & -(\mu_1+\delta_1+\gamma) & 0 & 0 \\ 0 & -\frac{b\beta_2\tau_3}{\mu_2+\delta_2} & 0 & -(\mu_2+\delta_2) & 0 \\ 0 & \frac{b\beta_2\tau_3}{\mu_2+\delta_2} & 0 & 0 & -(\mu_2+\delta_2) \end{pmatrix}.$$

This matrix has negative eigenvalues

$$\lambda_1 = -\mu_1, \quad \lambda_2 = -(\mu_1 + \delta_1 + \gamma), \quad \lambda_3 = -(\mu_2 + \delta_2).$$

The remaining two eigenvalues can be obtained from the 2x2 block matrix [12], given by

$$A = \begin{pmatrix} -(\mu_1 + \delta_1 + \gamma + \rho) & \dfrac{b\beta_1\tau_1}{\mu_1} \\ \dfrac{b\beta_2\tau_3}{\mu_2 + \delta_2} & -(\mu_2 + \delta_2) \end{pmatrix},$$

whose trace and determinant are given by

$$Tr(A) = -\left[(\mu_1 + \delta_1) + (\mu_2 + \delta_2) + (\gamma + \rho)\right] < 0; \qquad (2.7)$$

$$Det(A) = (\mu_1 + \delta_1 + \gamma + \rho)(\mu_2 + \delta_2) - \frac{b^2\beta_1\beta_2\tau_1\tau_3}{\mu_1\mu_2}$$

$$= (\mu_1 + \delta_1 + \gamma + \rho)(\mu_2 + \delta_2)[1 - R_0] \qquad (2.8)$$

where $$R_0 = \frac{b^2\beta_1\beta_2\tau_1\tau_3}{\mu_1\mu_2(\mu_1 + \delta_1 + \gamma + \rho)(\mu_2 + \delta_2)}. \qquad (2.9)$$

Now, if: (i) $R_0 < 1$, then $Det.(A) > 1$ (i.e. A is non-singular), and so the disease-free equilibrium is locally asymptotically stable.
(ii) $R_0 > 1$, then $Det.(A) < 1$ (i.e. A is singular), and so the disease-free equilibrium is unstable.
This completes the proof.

3.4 Existence of Endemic Equilibrium Point, *E**

The Endemic equilibrium points are steady-state solutions where there is ZikV infection (i.e. $I_h \neq I_m \neq 0$), while the steady-state solution of the system (2.1) is obtained by setting

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dQ}{dt} = \frac{dS_m}{dt} = \frac{dI_m}{dt} = 0 \text{ in (2.1).}$$

Thus, the endemic equilibrium point, E^*, for the ZikV model (2.1), computed numerically by substituting the parameter values of table 4.1 into the steady state solution of system (2.1), yields:

$$E^* = (0.54313486, 17.68534025, 16.49518890, 0.48239830, 4.60602299). \qquad (2.10)$$

3.5 Stability of Endemic Equilibrium Point

***THEOREM 3.2*:**

The endemic equilibrium E^* is unstable.

***Proof*:** The stability of the endemic equilibrium is determined by the eigenvalues of the Jacobian matrix of the full system (2.1), evaluated at the endemic equilibrium point, given by

$$J\big|_{E^*} = \begin{pmatrix} -(\mu_1 + b\tau_1 I_m) & \gamma & \gamma & 0 & -b\tau_1 S_h \\ 0 & -(\mu_1 + \delta_1 + \gamma + \rho) & 0 & 0 & b\tau_1 S_h \\ 0 & \rho & -(\mu_1 + \delta_1 + \gamma) & 0 & 0 \\ 0 & -b\tau_3 S_m & 0 & -(\mu_2 + \delta_2 + b\tau_3 I_h) & 0 \\ 0 & b\tau_3 S_m & 0 & b\tau_3 I_h & -(\mu_2 + \delta_2) \end{pmatrix}$$

This is computed numerically by substituting the parameter values given in table 4.1. i.e.

$$J\big|_{E^*} = \begin{pmatrix} -0.00493 & -0.5882088871 & 0.19642857143 & 0 & -0.1506206896 \\ 0 & -0.3831788871 & 0 & 0 & 0.1506206896 \\ 0 & 0.2 & -0.2014585714 & 0 & 0 \\ 0 & -0.01367441860 & 0 & -0.215 & 0 \\ 0 & 0.01367441860 & 0 & 0 & -0.215 \end{pmatrix}.$$

The eigenvalues are evaluated to be

$$\begin{pmatrix}\lambda_1\\ \lambda_2\\ \lambda_3\\ \lambda_4\\ \lambda_5\end{pmatrix}=\begin{pmatrix}-0.00493000000\\ 0.38660249385\\ -0.20145857140\\ -0.21842360675\\ -0.21500000000\end{pmatrix}.$$

Hence, the endemic equilibrium is unstable, since there exists a positive eigenvalue.

IV. Numerical Results And Discussion

The numerical simulation for the model was carried out by maple software using differential transformation method to show the effects of isolation, recovery and vector elimination on the spread of Zikavirus disease. We usedsome of the parameter values compatible with mosquito-borne diseases as given in the table 4.1 below, and by considering the initial conditions:

$$S_h(0)=500,\ I_h(0)=100,\ Q(0)=50,\ S_m(0)=750,\ I_m(0)=150$$

Table 4.1: *Parameter values used for the model*

Parameters	Description	Values	Source
β_1	Recruitment term of the susceptible humans	0.01547	[5]
β_2	Recruitment term of the susceptible mosquitoes	0.07	[8]
b	Biting rate per human per mosquito	0.12	[8]
τ_1	Probability that a bite by an infectious mosquito results in transmission of disease to human	0.40	[9]
τ_2	Transmission coefficient from contact between susceptible and infected humans	0.25	Assumed
τ_3	Probability that a bite results in transmission of parasite to a susceptible mosquito	0.35	[9]
θ	Fraction of births that are infected	0.001	[9]
ρ	Per capital rate of progression of humans from the infected state to the isolated state	0.20	Assumed
γ	Per capital recovery rate for humans from the infectious and isolated states to the susceptible state	$\left(\frac{1}{4}+\frac{1}{7}\right)\div 2$	[5]
μ_1	Human natural death rate	0.00493	[5]
μ_2	Mosquito natural death rate	0.115	[9]
δ_1	Disease-induced death rate for humans	0.0001	Assumed
δ_2	Extra human-induced death for mosquitoes	0.10	Assumed

4.1. Presentation of Results

The results are given in Figures 4.1 – 4.8 to illustrate the system's behaviour for different values of the model's parameters.

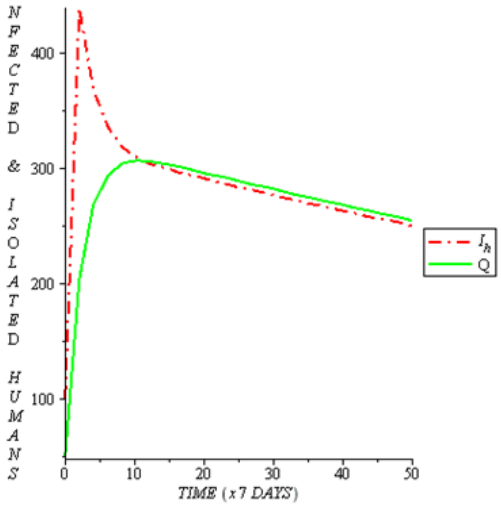

Fig.4.1: *The behaviour of human population when* $R_0<1$(β_1 = 0.01547, β_2 = 0.05, θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, δ_2 = 0.1, γ=11/56, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, ρ = 0.2, b = 0.12).

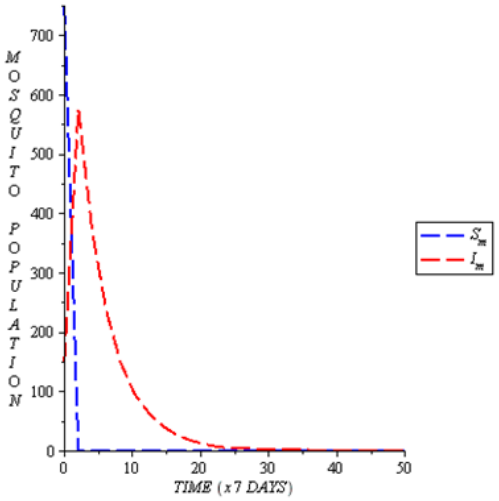

Fig.4.2: *The behaviour of mosquito population when* $R_0<1$($\beta_1 = 0.01547$, $\beta_2 = 0.05$, $\theta = 0.001$, $\mu_1=0.00493$, $\mu_2 = 0.115$, $\delta_1 = 0.0001$, $\delta_2 = 0.1$,$\gamma = 11/56$, $\tau_1 = 0.4$, $\tau_2 = 0.25$, $\tau_3 = 0.35$, $\rho = 0.2$, $b = 0.12$).

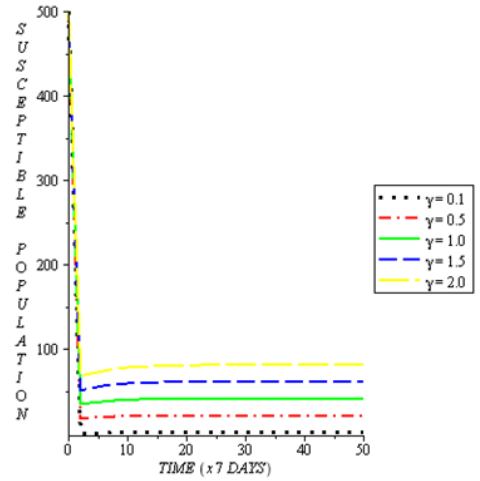

Fig. 4.3: *The behaviour of Susceptible human population for varied values of recovery rate,* γ($\beta_1 = 0.01547$, $\beta_2 = 0.05$, $\theta = 0.001$, $\mu_1=0.00493$, $\mu_2 = 0.115$, $\delta_1 = 0.0001$, $\delta_2= 0.1$, $\tau_1 = 0.4$, $\tau_2 = 0.25$, $\tau_3 = 0.35$, $\rho = 0.2$, $b = 0.12$).

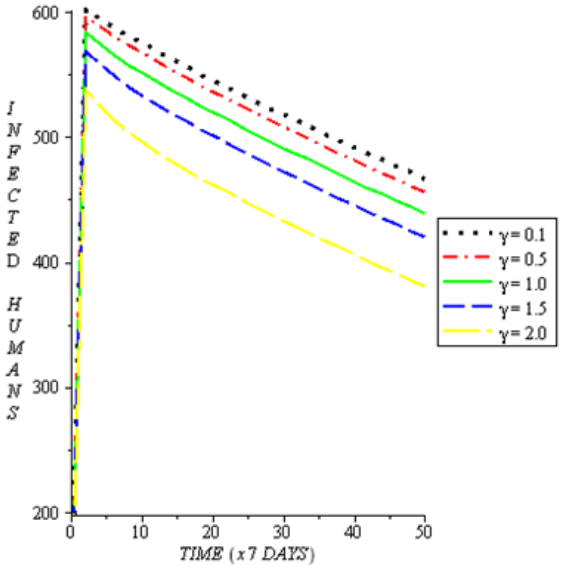

Fig. 4.4: *The behaviour of Infected human population for small values of recovery rate,* γ($\beta_1 = 0.01547$, $\beta_2 = 0.05$, $\theta = 0.001$, $\mu_1=0.00493$, $\mu_2 = 0.115$, $\delta_1 = 0.0001$, $\delta_2 = 0.1$, $\tau_1 = 0.4$, $\tau_2 = 0.25$, $\tau_3 = 0.35$, $\rho = 0.2$, $b = 0.12$).

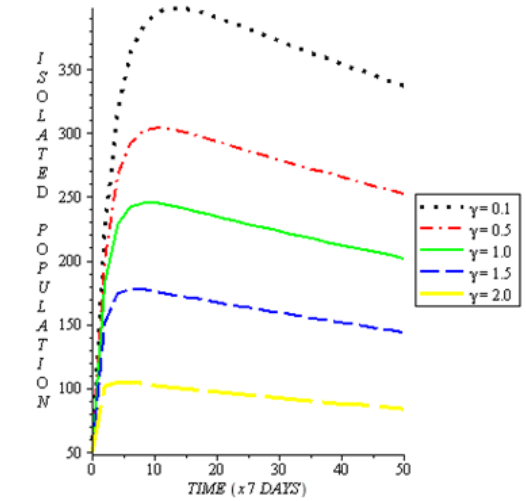

Fig. 4.5: *The behaviour of Isolated human population for varied values of recovery rate, γ(β_1 = 0.01547, β_2 = 0.05,θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, δ_2 = 0.1, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, ρ = 0.2, b = 0.12).*

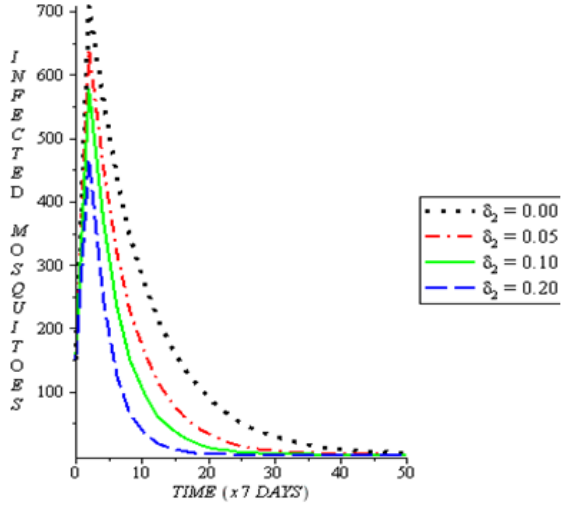

Fig. 4.6: *The behaviour of Infected mosquito population for varied values of human-induced death rate, δ_2(β_1 = 0.01547, β_2 = 0.05, θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, γ=11/56, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, ρ = 0.2, b = 0.12).*

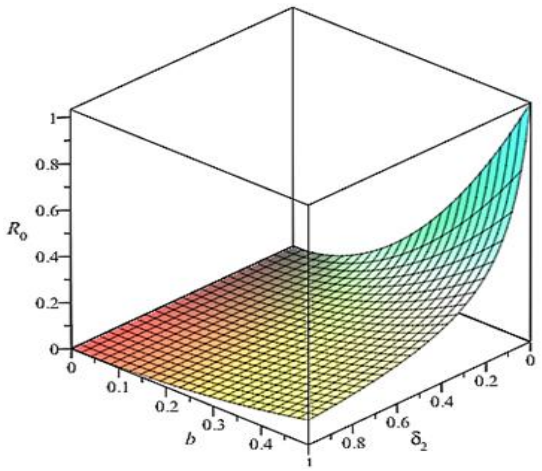

Fig. 4.7: *3-D Plot of R_0 against δ_2 and mosquito biting rate, b:* (β_1 = 0.01547, β_2 = 0.05, θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, γ=11/56, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, ρ = 0.2).

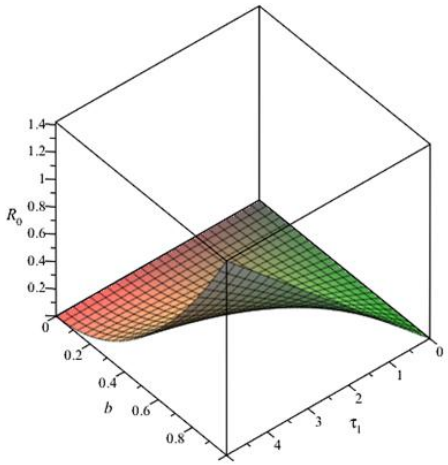

Fig. 4.8: *3-D Plot of R_0 against b and mosquito-human transmission rate, τ_1:* (β_1 = 0.01547, β_2 = 0.05, θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, γ=11/56, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, ρ = 0.2).

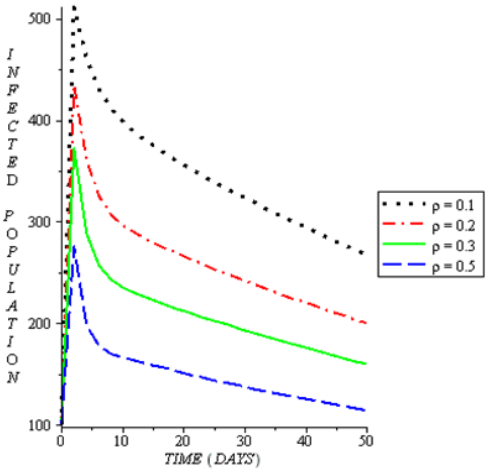

Fig. 4.9: *The behaviour of Infected human population for varied values of isolation rate, ρ*(β_1 = 0.01547, β_2 = 0.05, θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, δ_2 = 0.1, γ=11/56, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, b = 0.12).

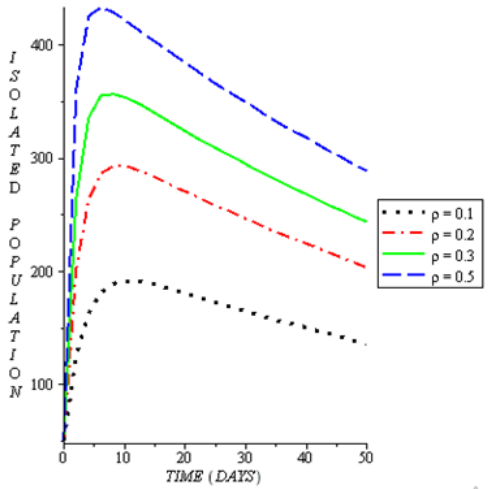

Fig. 4.10: *The behaviour of Isolated human population for varied values of isolation rate, ρ*(β_1 = 0.01547, β_2 = 0.05, θ = 0.001, μ_1=0.00493, μ_2 = 0.115, δ_1 = 0.0001, δ_2 = 0.1, γ=11/56, τ_1 = 0.4, τ_2 = 0.25, τ_3 = 0.35, b = 0.12)

4.2 Discussion on Results

Figures 4.1 and 4.2 shows the behaviour of the solution for the selected parameter values when $R_0 < 1$. Figure 4.1 shows the behaviour of the human population, and it is observed from the plot that there is decline in both the infected and isolated population. This implies that the disease will, at a point in time, be wiped out from the population. From figure 4.2 which shows the behaviour of the mosquito population, it can be seen that the population goes to zero. So, if we can work on reducing mosquitoes through source reduction (removal and clearance of breeding sites), then the entire mosquito population can be eliminated, which will lead to total elimination of the Zika virus disease.Figures 4.3 – 4.5 are the plots that show the effect of recovery rate, γ. From figure 4.3, it is observed that the susceptible population increases as recovery rate increases. Figure 4.4 shows that there is decline in infection as recovery increases. From figure 4.5, the isolated population decreases as recovery increases. It should be noted that individuals of the isolated population are still infectious, and they differs from the infected individual in that they are isolated to prevent their contact both with susceptible humans and mosquitoes. This control measure, among others, is recommended in this research paper to prevent outbreak of Zika virus disease.

Figure 4.6 is the plot of infected mosquito population against time for different values of human-induced death rate of mosquitoes (δ_2). As the value of δ_2 increases, a decrease in the infected mosquito population is noticed. Figures 4.7 and 4.8 are the 3-D plots of R_0 against biting rate (b) and δ_2 andR_0 against b and mosquito-human transmission rate (τ_1)respectively. It was shown from figure 4.7 that R_0 (ZikV prevalence) increasesas biting rate increases but decreases as$\delta_2$increases. And from figure 4.8, it was shown that increase in biting rate of mosquitoes results in increased transmission rate, and which consequently leads, to increase in R_0 (ZikV prevalence). We also observed that, from figures 4.9 and 4.10 which show the effect of isolation rate, there is decline in infection with increase in the rate of isolation, but an increase is seen in the isolated population as the isolation rate increases. Individuals in isolation can therefore be taken care of by treating their symptoms accordingly.

V. Conclusion

In this paper, we have formulated and analysed a compartmental model for ZikV transmission in human and mosquito populations with isolation and recovery parameters. The human population was divided into three compartments: susceptible, infected and isolated, while the mosquito population was divided into two compartments: susceptible, and infected. We established a region where the model is epidemiologically feasible and mathematically well-posed. The existence and stability of a disease-free equilibrium point as well as the endemic equilibrium point were determined.The numerical simulations were performed to see the effects of recovery (as a result of symptoms treatment), isolation and other key parameters on the spread of the disease. Our results showed that increasing the recovery to a very high rate has significant effect of reducing infection and isolation of infected individuals also reduces the transmission of the ZikV infection. To aid recovery, individuals in both infected and isolated classes should get plenty of rest, drink enough fluid to prevent dehydration, and treat fever and pain with common medicines such as acetaminophen (Tylenol®) and paracetamol[1]. Also, the rate of human-induced deaths of mosquito should be increased as shown by our result in figure 4.6. This can be achieved by reducing mosquito population through source reduction (removal and clearance of breeding sites) via the use of insect repellent containing DEET, picaridin, oil of lemon eucalyptus (OLE) or IR3535 [14].These control measures will greatly reduce the transmission of the ZikV infection.However, efforts should be intensified in developing vaccine for ZikV disease as this would facilitate the stimulation of the immune system in producing antibodies against ZikV infection.

Acknowledgement

The authors thank the anonymous referees whose comments led to an improved paper.

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