

Global Stability Analysis on the Transmission Dynamics of Zika Virus

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Abstract

We formulate a mathematical model on Zika virus by considering two population sizes of human beings and mosquitoes. The vertical transmission of Zika virus in human population is also considered. Basic reproductive number R_0 and equilibrium points for our model are defined. The disease-free equilibrium is proved to be globally stable when $R_0 < 1$, which means the disease, will die out and when $R_0 > 1$, the endemic equilibrium is globally stable. Extensive numerical simulations are carried out to establish the analytical results. The efficacy of personal protection from mosquito to human and from pregnant women to newborn child are also carried out by numerical simulations.

Keywords: Zika virus, Basic Reproduction Rate, Stability Analysis, Simulation.

INTRODUCTION

Zika virus was first recognized in Uganda in 1947. From 1951 to 1981, the confirmation of human infection with Zika virus was also identified from other African countries including the Central African Republic, Sierra Leone, Tanzania, Egypt, Gabon, and Uganda, as well as in parts of Asia including India, Indonesia, the Philippines, Thailand, Malaysia, and Vietnam [1, 2].

In 2007, the first documented outbreak of Zika virus disease was reported in Yap State, Federated States of Micronesia; 73% of the population within the age 3 years was estimated to have been infected [2]. Subsequent outbreaks occurred in Southeast Asia and the Western Pacific [3]. In May 2015, the World Health Organization reported the first local transmission of Zika virus in the Region of the Americas, with autochthonous cases identified in Brazil [4]. In December, the Ministry of Health estimated that 440,000–1,300,000 suspected cases of Zika virus disease had occurred in Brazil in 2015 [5].

Zika virus is transmitted to human beings through the bite of an infected *Aedes* species mosquito (*A. aegypti* and *A. albopictus*). The most common symptoms of Zika virus disease are fever, rash, joint pain, and conjunctivitis (red eyes). The illness is usually mild with symptoms lasting from several days to a week. Mosquitoes become infected when they feed on a person already infected with the virus. Infected mosquitoes can then spread the virus to other people through

bites. Mosquitoes that spread Zika are aggressive daytime biters. They can also bite at night. These mosquitoes typically lay eggs in and near standing water in things like buckets, bowls, animal dishes, flower pots and vases. They prefer to bite people, and live both indoors and outdoors near people. In human population, Zika virus is transmitted from an infected mother to her baby during pregnancy and may also be transmitted through blood transfusion and/or sexual contact with infected human. An estimated 80% of infected human with Zika virus are asymptomatic [6, 7, 8].

In past two and half decades, several epidemic models have been formulated for predicting the transmission of epidemic diseases such as HIV/AIDS [9,10], which are helpful for developing the mathematical model on the transmission dynamics of Zika virus in human population. Agosto et al. [11] formulated a model on ZIKV transmission in adult population in adult population with vertical transmission. Daihai He et al. [12] established a comparison study of Zika virus outbreaks in French Polynesia, Colombia and the State of Bahia in Brazil. Usman et al. [13] developed a mathematical model for the Transmission Dynamics of Zika Virus Infection with Combined Vaccination and Treatment Interventions. Bonyah et al. [14] formulated a theoretical model for Zika virus transmission. Gao et al. [15] developed a mathematical model on prevention and control of Zika as a Mosquito-Borne and Sexually Transmitted Disease. Eskild Petersen et al. [16] had formulated a review paper on unexpected and rapid spread of Zika Virus in the Americas - implications for public health preparedness for mass gatherings at the 2016 Brazil Olympic Games.

In this paper, we have formulated a mathematical model of Zika virus transmission for both human and mosquito population. We have considered the Susceptible class, Infected class and Recovered class in human population with vertical transmission. In mosquito population, we have considered two classes (Susceptible and Infected) for Zika virus transmission. We find basic reproduction number R_0 gives global dynamical behavior of the model. If $R_0 < 1$, the disease-free equilibrium is globally stable, which means the disease will die out and if $R_0 > 1$, the endemic equilibrium is globally stable. The basic reproduction number R_0 is also used in numerical simulations to discuss the effectiveness of control strategies.

The paper is organized as follows: Introduction is given in Section 1; the basic assumptions and parameters of the model

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and the epidemic model is developed in Section 2; Section 3 establishes the stability of the system developed; numerical simulations is given in Section 4; and finally conclusion in Section 5.

MODEL DESCRIPTION AND FORMULATION:

We divide the human population into three classes $S_H I_H R_H$ (Susceptible-Infected-Recovered) and the mosquito population into two classes $S_M I_M$ (Susceptible-Infected). Schematic flow of this model is shown in figure 2 and the state variables and associated parameters of this model are given in Table 1.

Table 1: The state variables and associated parameters

$S_H(t)$:	Susceptible proportions of human in time t
$I_H(t)$:	Infectious proportions of human in time t
$R_H(t)$:	Recovered proportions of human in time t
$S_M(t)$:	Susceptible proportions of mosquito in time t
$I_M(t)$:	Infectious proportions of mosquito in time t
$\bar{S}_H(t)$:	Susceptible humans in time t
$\bar{I}_H(t)$:	Infectious humans in time t
$\bar{R}_H(t)$:	Recovered humans in time t
$\bar{S}_M(t)$:	Susceptible mosquito in time t
$\bar{I}_M(t)$:	Infectious mosquito in time t
$N_H(t)$:	Total human population in time t
$N_M(t)$:	Total mosquito population in time t
B_H :	Birth rate and immigration rate of humans
B_M :	Birth rate and immigration rate of mosquitoes
β_H :	Transmission probability of Zika virus from human to human and from mosquito to human
β_M :	Transmission probability of Zika virus from human to mosquito
η :	Rate of transmission from Infected humans to Recovered humans
δ :	Rate of transmission from Recovered humans to Susceptible humans
μ_h :	Natural death rate of humans
μ_z :	Death rate of humans due to Zika virus
μ_m :	Natural death rate of mosquito
θ :	Rate of vertical transmission in human population.

Basic assumptions of model:

We assume that humans enter the susceptible class through a constant birth rate and immigration rate B_H . When an infected

with Zika virus mosquito bites a Susceptible human, he/she moves to the infected class with the transmission probability β_H , where β_H is the sum of the transmission probability of Zika virus from human to human and from mosquito to human. After some time, the infectious humans recover and move to the Recovered class with a constant rate η . The recovered humans have some immunity to the disease and do not get clinically ill, but they can pass the infection to mosquitoes. After some period of time, they lose their immunity and return to the Susceptible class with constant rate δ . Humans leave the population through a natural death rate μ_h , and through a per capita disease-induced death rate μ_z . In human population, the vertical transmission rate θ is assumed, that is the rate of transmission of Zika virus from infected pregnant women to new born children.

In mosquito population, we assumed that the mosquito enters the Susceptible class through a constant birth rate and immigration rate B_M . When a susceptible mosquito bites an infected human being with Zika virus, then the mosquito gets infected with Zika virus and it goes to infected class. The constant transmission probability of Zika virus from human to mosquito β_M is considered. Mosquitoes leave the population through a constant natural death rate μ_m only is assumed. We do not assume the vertical transmission in mosquito population.

Model Equations for humans population:

Based on our assumptions and the flow of transmission of zika virus in human population as depicted in figure 1, we have the following system of equations:

$$\begin{aligned} \frac{d\bar{S}_H}{dt} &= B_H N_H - \beta_H \bar{S}_H (\bar{I}_H + \bar{I}_M) - \mu_h \bar{S}_H + \delta \bar{R}_H \\ \frac{d\bar{I}_H}{dt} &= \beta_H \bar{S}_H (\bar{I}_H + \bar{I}_M) - (\mu_h + \mu_z + \eta - \theta) \bar{I}_H \\ \frac{d\bar{R}_H}{dt} &= \eta \bar{I}_H - (\mu_h + \delta) \bar{R}_H \end{aligned} \quad (1)$$

Figure 1: Schematic diagram of Zika virus transmission

Similarly, for the flow of transmission of Zika virus in mosquito population, we have the system of equations as:

$$\begin{aligned} \frac{d\bar{S}_M}{dt} &= B_M N_M - \beta_M \bar{S}_M \bar{I}_M - \mu_m \bar{S}_M \\ \frac{d\bar{I}_M}{dt} &= \beta_M \bar{S}_M \bar{I}_M - \mu_m \bar{I}_M. \end{aligned} \quad (2)$$

With these two conditions $\bar{S}_H + \bar{I}_H + \bar{R}_H = N_H$ and $\bar{S}_M + \bar{I}_M = N_M$.

Without loss of generality, we can write with the proportions

$$S_H = \frac{\bar{S}_H}{N_H}, I_H = \frac{\bar{I}_H}{N_H}, R_H = \frac{\bar{R}_H}{N_H}, S_M = \frac{\bar{S}_M}{N_M}, I_M = \frac{\bar{I}_M}{N_M}.$$

Since $R_H = 1 - S_H - I_H$ and $S_M = 1 - I_M$, the above two systems (1) and (2) can be reduced to the following equivalent system:

$$\begin{aligned} \frac{dS_H}{dt} &= B_H - \beta_H S_H (I_H + I_M) - \mu_h S_H + \delta(1 - S_H - I_H) \\ \frac{dI_H}{dt} &= \beta_H S_H (I_H + I_M) - (\mu_h + \mu_z + \eta - \theta) I_H \\ \frac{dI_M}{dt} &= \beta_M (1 - I_M) I_M - \mu_m I_M. \end{aligned} \quad (3)$$

The feasible region for the system (3) is as follows:

$$\Gamma = \{(S_H, I_H, I_M) \in \mathbb{R}^3 : S_H > 0, I_H \geq 0, I_M \geq 0, S_H + I_H \leq 1, I_M \leq 1\}.$$

STABILITY OF THE MODEL

In this section, we find the equilibrium states and basic reproduction number of the model. We also prove that our model is locally and globally stable for both disease-free equilibrium and endemic equilibrium points.

Finding equilibrium states by setting the right hand side of all the model equations of system (3) equal to zero, we obtain two equilibrium states:

- (i) Disease free equilibrium state: $E_0 = (1, 0, 0)$
- (ii) Endemic equilibrium state : $E_1 = (S_H^*, I_H^*, I_M^*)$

The system being modeled is expected to show different kinds of behavior in the long run. The equilibrium points and the conditions for their existence are that they provide us mathematical conditions based on which the long-term behavior of the system can be predicted and classified into a finite number of possibilities represented by the equilibrium points.

Endemic Equilibrium points of the system (3):

From the third equation of the system (3) by equating to zero, we get

$$I_M = \frac{\beta_M I_H}{\mu_m + \beta_M I_H}$$

Similarly, from the first and the second of the system (3) by equating to zero and solving it, we get

$$S_H = \frac{A - B I_H}{C}, \text{ where } A = B_H + \delta, B = \mu_h + \mu_z + \eta + \delta - \theta, C = \mu + \delta.$$

Putting the values of I_M and S_H in second equation of system (3), we get

$$B\beta_H\beta_M I_H^2 + [CD\beta_M - A\beta_H\beta_M + B\beta_H(\mu_m + \beta_M)]I_H + CD\mu_m - A\beta_H(\mu_m + \beta_M) = 0$$

Since all the parametric values are positive, so we consider only positive root of above equation and endemic equilibriums

$E_1 = (S_H^*, I_H^*, I_M^*)$ are as follows:

$$I_H^* = \frac{CD\beta_M - A\beta_H\beta_M + B\beta_H(\mu_m + \beta_M) + \sqrt{(CD\beta_M - A\beta_H\beta_M + B\beta_H(\mu_m + \beta_M))^2 - 4B\beta_H\beta_M(CD\mu_m - A\beta_H(\mu_m + \beta_M))}}{2B\beta_H\beta_M}$$

$$I_M^* = \frac{\beta_M I_H^*}{\mu_m + \beta_M I_H^*} \text{ and } S_H^* = \frac{(\mu_h + \mu_z + \eta - \theta) I_H^*}{\beta_H (I_H^* + I_M^*)}$$

Basic reproduction number:

For any epidemic model, the basic reproduction number is the average number of secondary infectious cases produced by a single infection in total susceptible population. The basic reproduction number is calculated by $R_0 = \rho(FV^{-1})$, where ρ is spectral radius of the matrix FV^{-1} and F & V are the matrices of new infection terms and the remaining transmission terms respectively [17].

For the systems (1) & (2), the matrices F and V are as follows:

$$F = \begin{bmatrix} \beta_H & \beta_H \\ \beta_M & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \mu_h + \mu_z + \eta - \theta & 0 \\ 0 & \mu_m \end{bmatrix}.$$

Hence, the basic reproduction number of the above model is

$$R_0 = \frac{\beta_h \mu_h^2 + \sqrt{\mu_m \beta_H^2 + 4(\mu_h + \mu_z + \eta - \theta) \beta_M^2}}{2\mu_m^2(\mu_h + \mu_z + \eta - \theta)}.$$

Theorem 1 : The system (3) is locally asymptotically stable for disease-free equilibrium, when $R_0 < 1$.

Proof: Jacobian matrix of the system (3) is as follows:

$$J = \begin{bmatrix} -(\mu_h + \delta) & -(\beta_H + \delta) & -\beta_H \\ 0 & \beta_H - (\mu_h + \mu_z + \eta - \theta) & \beta_H \\ 0 & \beta_M & -\mu_m \end{bmatrix}$$

The eigenvalues of Jacobian matrix J are as follows:

$$\begin{aligned} \lambda_1 &= -(\mu + \delta) \\ \lambda_2 &= -\frac{(\mu_h + \mu_z + \eta - \theta + \mu_m - \beta_H)}{2} - \frac{\sqrt{(\mu_h + \mu_z + \eta - \theta - \mu_m - \beta_H)^2 + 4\beta_H\beta_M}}{2} \\ \lambda_3 &= -\frac{(\mu_h + \mu_z + \eta - \theta + \mu_m - \beta_H)}{2} + \frac{\sqrt{(\mu_h + \mu_z + \eta - \theta - \mu_m - \beta_H)^2 + 4\beta_H\beta_M}}{2} \end{aligned}$$

Eigenvalues λ_1 and λ_2 have negative real value and we can easily verify that the eigenvalue $\lambda_3 < 0$, when $R_0 < 1$.

Hence, all eigenvalues of Jacobian matrix J are negative when $R_0 < 1$.

This proves that our the system is locally asymptotically stable when $R_0 < 1$.

Theorem 2: The unique endemic equilibrium point E_1 is globally asymptotically stable if $R_0 > 1$.

Proof : We will prove the global stability of endemic equilibrium E_1 using geometric approach [17], which has been attached briefly in Appendix A. The sufficient conditions for the global stability are shown in the hypotheses (H1) and (H2) with the Bendixson criteria given in Theorem (Appendix A).

For the general solution $(S_H(t), I_H(t), I_M(t))$ of system (3), the Jacobian matrix is

$$J = \begin{bmatrix} -\beta_H(I_H + I_M) - \mu_h - \delta & -\beta_H S_H - \delta & -\beta_H S_H \\ \beta_H(I_H + I_M) & \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) & \beta_H S_H \\ 0 & \beta_M(1 - I_M) & -\beta_M I_M - \mu_m \end{bmatrix}.$$

The matrix $J^{[2]}$, the second additive compound matrix of the Jacobian for $n=3$, is defined as

$$J^{[2]} = \begin{bmatrix} j_{11} + j_{22} & j_{23} & -j_{13} \\ j_{32} & j_{11} + j_{33} & j_{12} \\ -j_{31} & j_{21} & j_{22} + j_{33} \end{bmatrix}.$$

So, its second additive compound matrix $J^{[2]}$ is

$$J^{[2]} = \begin{bmatrix} x & \beta_H S_H & \beta_H S_H \\ \beta_M(1 - I_M) & y & -\beta_H S_H - \delta \\ 0 & \beta_H(I_H + I_M) & \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) - \beta_M I_M - \mu_m \end{bmatrix},$$

where $x = -\beta_H(I_H + I_M) - \mu_h - \delta + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta)$ and

$$y = -\beta_H(I_H + I_M) - \mu_h - \delta - \beta_M I_M - \mu_m.$$

Let the function $P = P(S_H, I_H, I_M)$ be defined as

$$P = P(S_H, I_H, I_M) = \begin{bmatrix} 1 & 0 & 0 \\ 0 & \frac{I_H}{I_M} & 0 \\ 0 & 0 & \frac{I_H}{I_M} \end{bmatrix} = \text{diag} \left\{ 1, \frac{I_H}{I_M}, \frac{I_H}{I_M} \right\}$$

$$\text{Then, } P_f P^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{I'_H}{I_H} - \frac{I'_M}{I_M} & 0 \\ 0 & 0 & \frac{I'_H}{I_H} - \frac{I'_M}{I_M} \end{bmatrix} \quad (4)$$

where P_f is the matrix obtained by replacing each elements of P by its derivative in the direction of f .

$$P_f J^{[2]} P^{-1} = \begin{bmatrix} x & \beta_H S_H \frac{I'_M}{I_H} & \beta_H S_H \frac{I'_M}{I_H} \\ \beta_M(1 - I_M) \frac{I'_H}{I_M} & y & -\beta_H S_H - \delta \\ 0 & \beta_H(I_H + I_M) & \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) - \beta_M I_M - \mu_m \end{bmatrix},$$

where $x = -\beta_H(I_H + I_M) - \mu_h - \delta + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta)$

and

$$y = -\beta_H(I_H + I_M) - \mu_h - \delta - \beta_M I_M - \mu_m.$$

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

where $B_{11} = [-\beta_H(I_H + I_M) - \mu_h - \delta + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta)]$,

$$B_{12} = \left[\beta_H S_H \frac{I'_M}{I_H} \quad \beta_H S_H \frac{I'_M}{I_H} \right], \quad B_{21} = \left[\beta_M(1 - I_M) \frac{I'_H}{I_M} \right] \text{ and}$$

$$B_{22} = \begin{bmatrix} y + \frac{I'_H}{I_H} - \frac{I'_M}{I_M} & -\beta_H S_H - \delta \\ \beta_H(I_H + I_M) & \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) - \beta_M I_M - \mu_m + \frac{I'_H}{I_H} - \frac{I'_M}{I_M} \end{bmatrix},$$

where $y = -\beta_H(I_H + I_M) - \mu_h - \delta + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) - \beta_M I_M - \mu_m$.

Now, for a vector (u, v, w) in \mathbf{R}^3 , we select a norm as $|(u, v, w)| = \max\{|u|, |v| + |w|\}$ and denote $\mu(B)$ the Lozinskii measure for this norm.

$$\text{From (4), it follows that } \mu(B) \leq \sup\{k_1, k_2\} \quad (5)$$

where k_1 and k_2 are defined as follows:

$k_1 = B_{11} + |B_{12}|$ and $k_2 = \mu_1(B_{22}) + |B_{21}|$, where $|B_{12}|$ and $|B_{21}|$ are matrix norms with respect to the vector norm L^1 and μ_1 denotes the Lozinskii measure with respect to the vector norm L^1 . So, we have

$$k_1 = B_{11} + |B_{12}|$$

$$= -\beta_H(I_H + I_M) - \mu_h - \delta + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) + \text{Sup} \left\{ \beta_H S_H \frac{I'_M}{I_H}, \beta_H S_H \frac{I'_M}{I_H} \right\}$$

$$k_1 = -\beta_H(I_H + I_M) - \mu_h - \delta + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) + \beta_H S_H \frac{I'_M}{I_H} \quad (6)$$

Similarly, $k_2 = \mu_1(B_{22}) + |B_{21}|$

$$= \beta_M(1 - I_M) \frac{I'_H}{I_M} + \beta_H S_H - (\mu_h + \mu_z + \eta - \theta) - \beta_M I_M - \mu_m + \frac{I'_H}{I_H} - \frac{I'_M}{I_M} \quad (7)$$

From second and third equations of system (3), we can rewrite as

$$\frac{I'_H}{I_H} + (\mu_h + \mu_z + \eta - \theta) = \beta_H S_H + \beta_H S_H \frac{I'_M}{I_H} \quad (8)$$

$$\frac{I'_M}{I_M} + \mu_m = \beta_M(1 - I_M) \frac{I'_H}{I_M}. \quad (9)$$

Putting (8) and (9) in (7) and (6) respectively, we get

$$k_1 = -\beta_H(I_H + I_M) + \frac{I'_H}{I_H} - (\mu_h + \delta) \leq \frac{I'_H}{I_H} - (\mu_h + \delta)$$

$$k_2 = -\beta_M I_M + \frac{I'_H}{I_H} - (\mu_h + \delta) \leq \frac{I'_H}{I_H} - (\mu_h + \delta).$$

Hence, from (5)

$$\mu(B) \leq \frac{I'_H}{I_H} - (\mu_h + \delta) \text{ and so, } \frac{1}{t} \int_0^t \mu(B) ds \leq \frac{1}{t} \log_e \frac{I'_H}{I_H} - (\mu_h + \delta).$$

So, $\bar{q}_2 < 0$, and hence the Bendixson criteria is also satisfied, which thus proves the global stability of the endemic equilibrium.

NUMERICAL SIMULATIONS AND EFFECT OF PARAMETERIC VALUES:

In this section, using Runge-kutta-Fehlberg method of order 4 and 5 in MATLAB, we numerically simulate our system with the parametric values as given in Table 2 when $R_0 < 1$ & Table 3 when $R_0 > 1$ and establish the stability of models by taking different examples.

Table 2: Parametric values for Zika virus model when $R_0 < 1$.

Parameter	Value	Parameter	Value
β_H	0.004	η	0.2
β_M	0.01	θ	0.1
δ	0.6	μ_h	0.6
B_H	0.8	μ_m	0.4
B_M	0.7	μ_z	0.3

Table 3: Parametric values for Zika virus model when $R_0 > 1$.

Parameter	Value	Parameter	Value
β_H	0.5	η	0.5
β_M	0.7	θ	0.1
δ	0.9	μ_h	0.4
B_H	0.8	μ_m	0.8
B_M	0.7	μ_z	0.9

Example 1: Consider the system (1) with initial conditions $S_H = 9999$, $I_H = 1000$, $R_H = 100$, $S_M = 4999$, $I_M = 100$ and the parametric values as shown in Table 2. The simulation results are shown in figure 2, which illustrates the behavior of Susceptible, Infected, Recovered classes for human population and Susceptible, Infected classes for mosquito population. These are initially positive in the region of admissible values and asymptotically approach to the disease free equilibrium for $R_0 = 0.0066 < 1$.

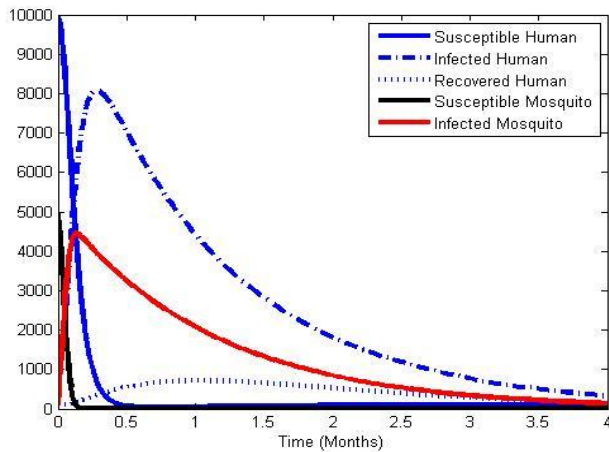


Figure 2: Comparison of all classes for human and mosquito population.

Example 2: Consider the system (1) with initial conditions $S_H = 9999$, $I_H = 100$, $R_H = 100$, $S_M = 999$, $I_M = 100$ and the parametric values as shown in Table 2. We simulate between infected humans versus recovered humans when $\eta = 0.2$, $\eta = 0.25$, $\eta = 0.3$, $\eta = 0.35$, $\eta = 0.4$, $\eta = 0.45$, $\eta = 0.5$ and $\eta = 0.55$, then the basic reproduction numbers are $R_0 = 0.019$, $R_0 = 0.020$, $R_0 = 0.021$, $R_0 = 0.022$, $R_0 = 0.024$, $R_0 = 0.025$, $R_0 = 0.027$ and $R_0 = 0.028$ respectively as shown in figure 3. From figure 3, we observe that the nature of trajectory tends to disease-free equilibrium point in infected-recovered phase plane, which shows the global stability of disease-free equilibrium point, when $R_0 < 1$.

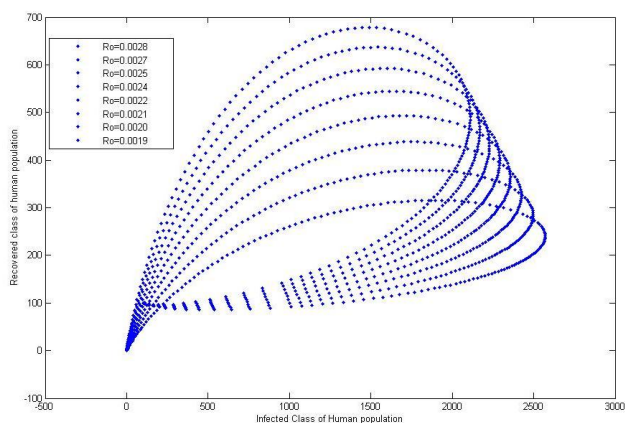


Figure 3: Infected-recovered phase plane when $R_0 < 1$.

Example 3: To show the global stability of endemic equilibrium point, when $R_0 > 1$, we consider the global dynamics of the infected human-infected mosquito plane and try to understand the nature of the trajectory towards the endemic equilibrium point. Consider the system (1) with three initial conditions of different Susceptible humans $S_H = 9899$, $S_H = 9099$, $S_H = 8099$ and $I_H = 50$, $R_H = 10$, $S_M = 999$, $I_M = 50$ and with parametric values as shown in Table 2. From figure 4, we observe that the nature of trajectory is steady spiral tends to endemic equilibrium point from any three initial point in infected human-infected mosquito plane, which shows the global stability of endemic equilibrium point, when $R_0 = 1.399 > 1$.

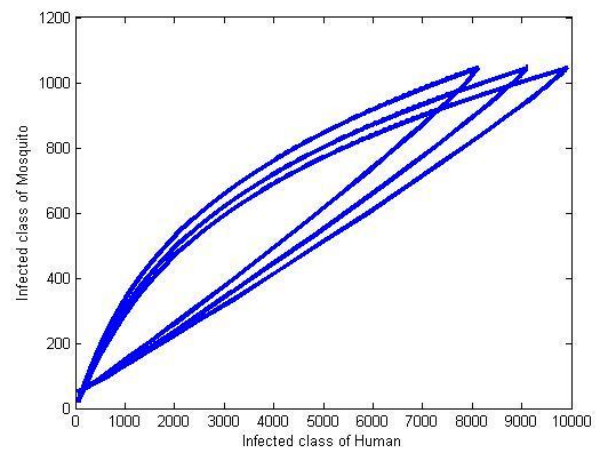


Figure 4: Infected human-infected mosquito plane when $R_0 > 1$.

Example 4: To compare the number of susceptible humans with the number of infected humans when $R_0 > 1$, we consider the system (3) with initial conditions $S_H = 9999$, $I_H = 50$, $R_H = 10$, $S_M = 999$, $I_M = 50$ and the parametric values of Table 3. The simulation result is shown in figure 5, which illustrates that total numbers of susceptible humans get infected with Zika virus and the total number of infected humans increase to $I_H = 9999$ when $R_0 = 1.399 > 1$. From the peak, the infected class decreases because there are no susceptible humans to be infected. The rank correlation coefficient between susceptible and infected humans are equal to -0.0378 . This means that when the number of infected human increases, the number of susceptible human decreases and vice-versa.

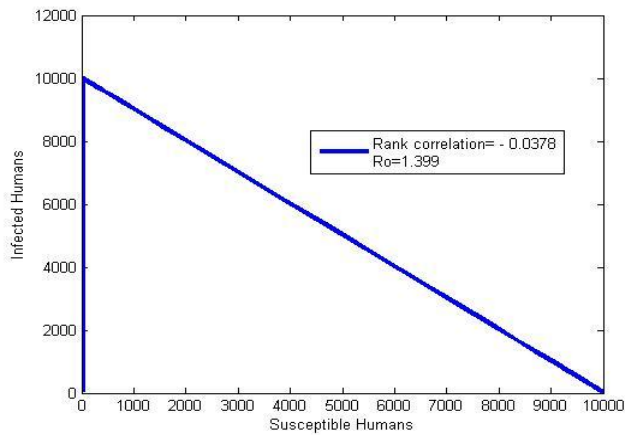


Figure 5: Comparison between susceptible and infected human when $R_0 > 1$.

Example 5: To compare the number of recovered humans with the number of infected human when $R_0 > 1$, we consider the system (1) with initial conditions $S_H = 9999$, $I_H = 50$, $R_H = 10$, $S_M = 999$, $I_M = 50$ and the parametric values of Table 3. The simulation result is shown in figure 6. From figure 6, it is clear that number of recovered human increases for short time period and again becomes susceptible human. Hence the total recovered humans become susceptible and finally total numbers of susceptible humans get infected with Zika virus and the total number of infected human increase to $I_H = 9999$ when $R_0 = 1.399 > 1$. From the peak, the infected class decreases because there are no susceptible humans to be infected. The rank correlation co-efficient between recovered and infected humans are equal to -0.1993.

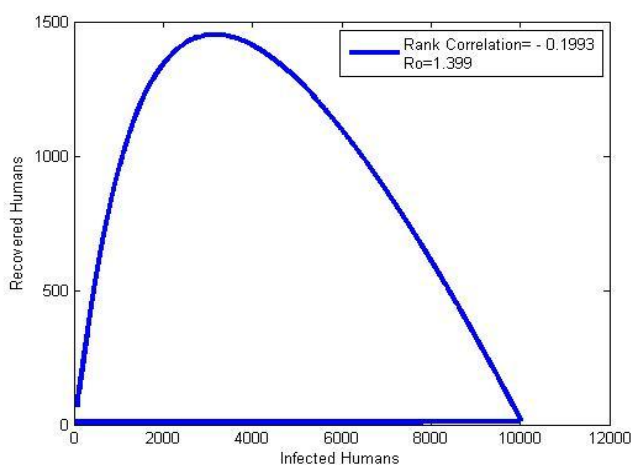


Figure 6: Comparison between infected and recovered human when $R_0 > 1$.

Example 6: Consider the system (1) with initial condition $S_H = 9999$, $I_H = 100$, $R_H = 100$, $S_M = 999$, $I_M = 100$ and the parametric values as shown in Table 2. Figure 7 shows the efficacy of personal protection from the mosquitoes, for different values of β_H .

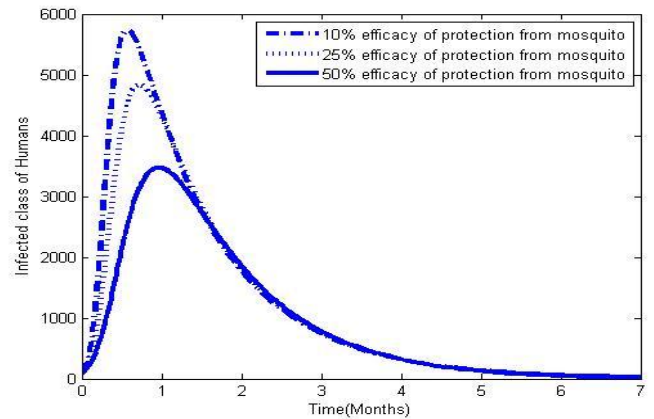


Figure 7: Efficacy of personal protection of human from mosquito

Example 7: Consider the system (1) with initial conditions $S_H = 9999$, $I_H = 100$, $R_H = 100$, $S_M = 999$, $I_M = 100$ and the parametric values as shown in Table 2. Figure 8 shows the efficacy of personal protection from the pregnant women to newborn child for different values of vertical transmission θ .

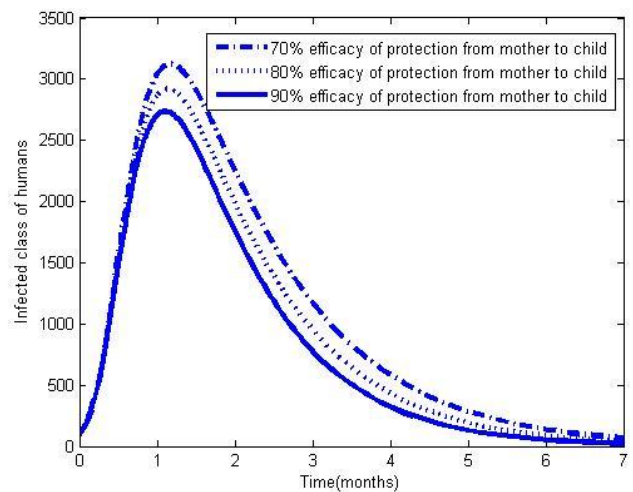


Figure 8: Efficacy of personal protection from pregnant women to child.

DISCUSSION AND CONCLUSION

The basic reproduction number of the model $R_0 = \frac{\beta_h \mu_h^2 + \sqrt{\mu_m \beta_H^2 + 4(\mu_h + \mu_z + \eta) \beta_M^2}}{2\mu_m^2(\mu_h + \mu_z + \eta)}$ is found and shown that the model is globally asymptotically stable at disease-free equilibrium when $R_0 < 1$. We proved the global stability of endemic equilibrium of the model using geometric approach with the Bendixson criteria when $R_0 > 1$. We also illustrate the analytical results numerically. If $R_0 < 1$, that means that an infective with Zika virus replaces itself by less than one

new infective with Zika virus and the disease dies out. Furthermore, the susceptible proportion approaches one because everyone is susceptible when the disease has died out and the entire removed human beings who immune have died. If $R_0 > 1$ and the initial proportion of susceptible $S = S_H + S_M$ satisfies $SR_0 > 1$, then proportion of susceptible S decreases and the infection proportion $I = I_H + I_M$ increases. The infection proportion I increases to peak, then decreases due to lack of sufficient susceptible to be infected. Due to births of new susceptible, when the susceptible proportion is larger and the secondary epidemic is smaller, then the solutions are spiral to endemic equilibrium as shown in figure 3 and figure 4. The comparisons between susceptible human class versus infected human class and infected human class versus recovered human class are shown in figure 5 and figure 6 respectively when $R_0 > 1$. The efficacy of personal protections in infected human is shown in figure 7. Figure 8 shows that the efficacy for pregnant women to newborn child by taking personal protection, which may include N,N-diethyl-m-toluamide (DEET), can be used on children aged greater than 2 months and on a precaution basis pregnant women should not travel to any area where Zika virus transmission is present.

REFERENCES

- [1] Hayes E B, 2009, "Zika virus outside Africa", *Emerg. Infect. Dis.* 15, pp. 1347–50.
- [2] Duffy M R, Chen T H, Hancock W T, Powers A M, Kool J L, Lanciotti R S, Pretrick M, Marfel M, Holzbauer S, Dubray C, Guillaumot L, Griggs A, Bel M, Lambert A J, Laven J, Kosoy O, Panella A, Biggerstaff B J, Fischer M and Hayes E B, 2009, "Zika virus outbreak on Yap Island, Federated States of Micronesia", *N. Engl. J. Med.* 360, pp. 2536–43.
- [3] Musso D, Nilles E J and Cao-Lormeau V M, 2014, "Rapid spread of emerging Zika virus in the Pacific area" *Clin. Microbiol. Infect.* 20, pp. 0595–6.
- [4] Zanluca C, de Melo V C, Mosimann A L, Dos Santos G I, Dos Santos C N and Luz K, 2015, "First report of autochthonous transmission of Zika virus in Brazil", *Mem. Inst. Oswaldo. Cruz.* 110, pp. 569–72.
- [5] European Centre for Disease Prevention and Control, 2015, "Zika virus epidemic in the Americas: potential association with microcephaly and Guillain-Barré syndrome. Stockholm, Sweden: European Centre for Disease Prevention and Control" <http://ecdc.europa.eu/en/publications/Publications/zika-virus-americas-association-with-microcephaly-rapid-risk-assessment.pdf> (accessed 17.02.2018).
- [6] Nasci R S, Wirtz R A and Brogdon W G, 2016, "Protection against mosquitoes, ticks, and other arthropods. In: CDC health information for international travel", New York, NY: Oxford University Press. <http://wwwnc.cdc.gov/travel/yellowbook/2016/the-pre-travel-consultation/protection-against-mosquitoes-ticks-other-arthropods> (accessed 17.02.2018).
- [7] Petersen E E, Staples J E, Meaney-Delman D, Fischer M, Ellington S R, Callaghan W M, Jamieson D J, 2016, "Interim guidelines for pregnant women during a Zika virus outbreak—United States", *Morb. Mortal. Wkly. Rep.* 65, pp. 30–33.
- [8] Musso D and Tu-Xuan N, 2015, "Emergence of Zika Virus", *Clin. Microbiol.* 4, pp. 1-4.
- [9] Mojaver A and Kheiri H, 2015, "Mathematical analysis of a class of HIV infection models of CD4⁺ T-cells with combined antiretroviral therapy", *Appl. Math. Comput.* 259, pp. 258-270.
- [10] Rao A S R S and Kakehashi M, 2004, "A combination of differential equations and convolution in understanding the spread of an epidemic" *Sadhana* 29, pp. 305. <https://doi.org/10.1007/BF02703780>.
- [11] Agosto F B, Bewick S, Fagan W F, 2017, "Mathematical model of Zika virus with vertical transmission", *Infectious Disease Modelling* 2, pp. 244-267.
- [12] He D, Gao D, Lou Y, Zhao S and Ruan S, 2017, "A comparison study of Zika virus outbreaks in French Polynesia, Colombia and the State of Bahia in Brazil. *Scientific Reports*", 7, pp. 273. DOI:10.1038/s41598-017-00253-1.
- [13] Usman S, Adamu I I and Babando H A, 2017, "Mathematical Model for the Transmission Dynamics of Zika Virus Infection with Combined Vaccination and Treatment Interventions", *JAMP*, 5, pp. 1964-1978. <https://doi.org/10.4236/jamp.2017.510166>
- [14] Bonyah E, Khan M A, Okosun K O and Islam S, 2017, "A theoretical model for Zika virus transmission", *PLoS ONE* 12(10): e0185540. <https://doi.org/10.1371/journal.pone.0185540>.
- [15] Gao D, Lou Y, He D, Porco T C, Kuang Y, Chowell G and Ruan S, 2016, "Prevention and Control of Zika as a Mosquito-Borne and Sexually Transmitted Disease: A Mathematical Modeling Analysis" *Scientific Reports* 6:28070. DOI: 10.1038/srep28070.
- [16] Petersen E, Wilson M E, Touch S, McCloskey B, Mwaba P, Bates M, Dar O, Mattes F, Kidd M, Ippolito G, Azhar E I and Zumla A, 2016, "Unexpected and Rapid Spread of Zika Virus in The Americas - Implications for Public Health Preparedness for Mass Gatherings at the 2016 Brazil Olympic Games", *IJID* 2529, pp. 1–5.
- [17] Li M Y and Muldowney J S, 1996, "A geometric approach to global-stability problem", *SIMS J Math Anal* 27, pp. 1070-83.

APPENDIX A:

In this appendix, we discuss in brief the geometric approach to global stability problems, developed by Li and Muldowney [18].

Consider an autonomous dynamical system $\dot{Y} = f(Y)$, where $f(Y) \in \mathbb{R}^n$ is C^1 function of Y in open subset Ω of \mathbb{R}^n .

Assumptions of two hypotheses:

H_1 : There exists a compact absorbing set K in Ω .

H_2 : Above dynamical system has unique equilibrium \bar{Y} in Ω .

Define $\bar{q}_2 = \limsup_{t \rightarrow \infty} \max_{y_0 \in \Omega} \frac{1}{t} \int_0^t \mu(B(y(s, y_0))) ds$, where the Lozinskii measure of matrix B is defined as $\mu(B) = \lim_{h \rightarrow 0} \frac{|I+hB|-1}{h}$.

Theorem: If the system satisfy the above hypothesis H_1 - H_2 and $\bar{q}_2 < 0$, then the unique equilibrium \bar{Y} is globally stable in Ω .