ANALYSIS OF THE TRANSMISSION DYNAMICS FOR ZIKA VIRUS WITH NONLINEAR FORCE OF INFECTIONS

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Abstract

This paper presents a seven-dimensional ordinary differential equation of mathematical model of zika virus between humans and mosquitoes population with non-linear forces of infection in form of saturated incidence rate. Vertical transmission is introduced into the model. These incidence rates produce antibodies in response to the presence of parasite-causing zika virus in both human and mosquito populations. The existence of region where the model is epidemiologically feasible is established (invariant set) and the positivity of the models is also established. The basic properties of the model are determined including the reproduction number of both cases, R_0 and $R_0 \mid_{p=a=0}$ respectively . Stability analysis of the disease-free equilibrium is investigated via the threshold parameter (reproduction number $R_0|_{n=a=0}$) obtained using the next generation matrix technique. The special case model results shown that the disease-free equilibrium is locally asymptotical stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. Under specific conditions on the model parameters, the global dynamics of the special case model around the equilibra are explored using Lyapunov functions. For a threshold parameter less than unity, the disease-free equilibrium is globally asymptotically stable. While the endemic equilibrium is shows to be globally asymptotically stable at threshold parameter greater than unity. Numerical simulations are carried out to confirm the analytic results and explore the possible behavior of the formulated model. The result shows that, horizontal and vertical transmission contributes a higher percentage of infected individuals in the population than only horizontal transmission.

INTRODUCTION

In 1946, scientists identified a new virus from monkey in the zika forest of Uganda – named as zikavirus. In 1948 the virus is then discovered from the mosquito Aedes, caught in the zika forest tree. The first human case of Zika virus was reported in Nigria in 1954, Macnamara (1954). In 2007, Zika virus moved out of Africa and Asia causing an outbreak in Yap Island in the Federated States of Micronesia, Duffy et.al (2009). An estimated 73% of Yap residents were infected with Zika virus. This was followed by a large outbreak in French Polynesia in 2013-2014, Cao-Lormeau and Musso (2014). And then spreading to new Caledonia, the Cook Islands and Eastern Island, Musso et.al (2014). After a very long time of silence, the Zika virus infections became a new problem in medicine, Wiwantkit et.al (2015). According to Cao-Lormeau et.al (2014), in early

2015, Zika virus was detected in Brazil. Phylogenetic analyses of the virus isolated from patients placed the Brazilian strains in the Asian lineage which has been previously detected during the French Polynesian outbreak. Since the first detection of Zika virus in Brazil, the following countries have reported ongoing substantial transmission of Zika virus in South America: Bolivia. Brazil, Colombia, Ecuador, French Guyana, Guyana, Paraguay, Suriname and Venezuela, CDC(c) (2016). Several Central America countries are also affected including Costa Rica, El Salvador, Guatemala, Honduras, Nicaragua and Panama, CDC (d) (2016). As at 10 June 2016, a total of 55 countries and territories have reported active zika virus transmission since the beginning of 2015, the majority (39, 71%) in the Americans. In the last month, two countries have reported cases of transmission of zika virus for the first time - Argentina and Indonesia. The rapid expansion of Zika virus has led the World Health Organization (WHO) to declare it a public health emergence of international concern, WHO (2016). Due to a probable association between Zika virus infection and a congenital neurological disorder called Microcephaly, Bichara et.al (2016). The epidemic trajectory of this virus infection poses a significant concern for the nearly 15 million children born in the Americas each year. WHO also reported the association between Zika virus infection in pregnant women and congenital Microcephaly in their babies, CDC (2015). Nearly 5000 cases of Microcephaly have been documented in areas experiencing Zika virus transmission and there is widespread concern that these numbers could grow rapidly as the virus sweeps across the Americas, Fauci and Morens (2016), Lucey and Gostin (2016). Further expansion to the geographic range of zika virus is anticipated in countries with active mosquito vectors, especially Aedes species. Zika virus (zikv) outbreak was initially recognized in Africa, Hayes (2009). The Zika virus problem now becomes the global public health issue. South America is currently most heavily hit with over 4200 suspected cases reported in Brazil alone, Agencia (2016) Outbreak cases in other pacific countries where also reported, Musso et.al (2015) and Roth et.al (2014). Specifically, zika virus transmission is basically vector-borne; however it can also be transmitted through sexual contact and blood transfusion process, Musso et.al (2015). In 2008, a US scientists conducting field work in Senegal fell ill with zika infection on his return home to Colorado he infected his wife which was the first documented case of sexual transmission of the Zika disease. The main vector for the zika virus is Aedes species of mosquito, which is also the vector for Chikungunya and Dengue virus, Musso et. al (2014). Zika virus is therefore likely to be capable of sustained transmission in other tropical area, Bogoch et.al (2016), as well as causing symptoms such as fever, rash, joint pain and red eyes (conjunctivitis) which normally last for 2-7 days, Zanluca (2015) and WHO (2016). Zika infection has also been linked to increased incidence of neurological sequelae, including Guillain - Barre Syndrome (GBS), Oechler et.al (2014) and Oechler et.al. (2015).

Zika virus is the focus of an ongoing pandemic and public health emergency. Previous, limited to sporadic cases in Africa and Asia, the emergency of zika virus in Brazil in 2015, heralded rapid spread throughout the Americans. Severe manifestations have been described including Guillian – Barre Syndrome in adults and mcirocephaly in babies born to infected mothers. Neither an effective treatment nor a vaccine is available for zika, therefore, the public health response

primarily focuses on preventing infection, particularly in pregnant women. Despite growing knowledge about this virus, questions remain regarding the virus, vectors and reservoirs. These questions highlight the need for research to optimize surveillance, patient management and public health intervention in the current zika virus epidemic. Inyama et.al (2017), in their model they considered three populations namely, Humans, mosquitoes and monkeys where they showed how the zika can be transmitted from monkey to human. Duong et.al (2015) studied the evidence that Aedes species of mosquito transmit both Dengue fever and Zika virus. They modeled the three compartments namely susceptible, infectives and recovered (SIR model) to examine the dynamics of transmission of Zika virus to human. Musso et.al (2013) demonstrated the potential risk for transfusion – transmission zika virus infection. They highlighted the risk of transmission through transfusion and also made reference to the region and cases affected by blood transfusion. Foy et.al (2011) studied how zika virus can be transmitted through sexual intercourse. They also described a patient who was infected with zika virus in South Eastern Senegal in 2008.Oehler (2014) and Ochler (2015) studied that there is a link between zika virus and increased incidence of neurological squealed including, Guillian - Barre system. Schuler et.al (2015) also studied microcephaly in infants born to mothers who were infected with zika virus during pregnancy.

Ebenezer and Kazeen (2016) studied the mathematical modeling of zika virus. They introduced optimal control strategies into the model, where the basic properties of the model without control strategies were determined including the reproduction number. They used pontryagin's maximum principle to characterize the necessary conditions for optimal control of zika virus. The preventive and treatment strategy without spraying the mosquitoes showed a great reduction in infection in the infected mosquitoe population. The application of preventive treatment and insecticide showed the best way of reducing the spread of zika virus. Raul and Karl (2016), studied the spread of three diseases, namely Dengue, Chikungunya and zika. They were the first people to treat the mathematical model to describe the dynamics of transmissions of these three diseases. They presented two preliminary models that consist of the SEIR model for the human population and an SEI model for the vector to describe, first the single transmission dynamics of dengue, Chikungunya or zika, and second any possible co infection between two diseases in the same population. In order word they obtained an analytical solution of the system of 17 and 30 coupled differential equations for each model respectively and later obtained the Eigen-values by analyzing the Jacobin matrix in order to begin the development of a surveillance system to prevent the spread of these three diseases.

The model presented in this work consist of four compartments in human population (that is, susceptible (S) Expose (E) Infectious (I), and recovered class (R) and also three compartment in mosquitoes, (that is, susceptible,(S) Expose (E) and Infectious (I), with inclusion of nonlinear forces of infection in form of saturated incidence rates in both the host (human) and vector (mosquitoes) populations. The disease induced death rate is also incorporated only in human population because disease may be fatal to some infectious host. We study the effects of these

inclusions on behavior of the formulated model. We also assume that the immunity is permanent and that recovered individuals do not revert to the susceptible class.

In this study we have been able to extend the works of Ebenezer and Kazeem (2016). Their model did not consider exposed class, vertical transmission and saturated incidence rate which were considered in our own model. We finally analyzed horizontal and vertical transmissions of zika virus and used compartmental deterministic model to study the dynamic of the disease. We used lyapunov function theory to obtain the global stability of both the disease free equilibrium and the endemic equilibrium.

MODEL FORMULATION

Here, we shall look at how to formulate a deterministic differential equation model from a flow diagram of zika transmission schematic to get the differential equation of the model. The Jacobin matrix of the model is gotten by differentiating the equations of the model with respect to the seven classes of model that is $S_H(t)$, $E_H(t)$, $I_H(t)$, $R_H(t)$, $S_m(t)$, $E_m(t)$ and $I_m(t)$ respectively and solving for an Eigen-value will determine if the disease free steady state is stable and we use Lyapunov theory to solve for global stability.

Symbols, Parameters and Variable of the Model

 b_H -Natural birth rate of human

 b_m - Constant recruitment rate of susceptible mosquito population

b - Biting rate of mosquitoes

 $b_{\rm H}$ - Probability that a bite by an infectious mosquito results in transmissions of disease to human

 b_m – Probability that a bite by an infectious human results in transmissions of disease to mosquito

 V_{H} – Proportion of antibody produced by human in response to the incidence of infection caused by mosquito

 V_m – Proportion of antibody produced by mosquito in response to the incidence of infection caused by human

 e_{H} – Disease induced death

- g_{H} –Recovering rate of infected human
- $m_{\rm H}$ Natural death rate of human
- m_m Natural death rate of mosquito
- $a_{\scriptscriptstyle H}$ Progression rate of exposed human into infected human population
- a_m Progression rate of exposed mosquito into infected mosquito population
- p The fraction of infected newborns from the expose class
- q The fraction of infected newborns from the infectious class
- $S_{\scriptscriptstyle H}\,$ Number of human susceptible to zika at time t
- $E_{\rm H}$ Number of human exposed to zika infections at time t
- $I_{\rm H}$ Number of infectious humans at time t
- R_{H} Number of recovered human at time t
- S_m Number of susceptible mosquitoes at time t
- I_m Number of infectious mosquitoes at time

Assumption of Model

- A fraction p and a fraction q of the offspring from the exposed and the infectious classes, respectively are born into the exposed class E
- 2. The horizontal transmissions typically occur through the bites of infected mosquitoes.
- Humans may die as a result of the disease because the disease might be fatal to some human host.

- 4. Once a person has been infected and recovered he or she is likely to be protected from future infections
- 5. The infected pregnant woman is able to transmit to her baby, either during early pregnancies or at the point of birth.
- 6. Both human and mosquitoes may die as a result of natural causes.
- The natural birth rate of human population (b_H) and mosquito population (b_m) have constant densities as 1

Model Flow Diagram



Fig 1. Model Flow Diagram

Model Formulation

The model presented by Ebenezer and Kazeem (2016) studied the mathematical modeling of zika virus with simple SIR model. Under their assumptions, individuals are born uninfected with zika.

We now extend their model by considering the exposed class and vertical transmission (motherto-child transmission) with a saturated incident rate. The total population sizes for human host and mosquito population are donated by $N_H(t)$ and $N_m(t)$, respectively. The human population $N_H(t)$ is divided into the epidemiological subclasses: Susceptible, Exposed, Infections, and permanent immune denoted by S_H, E_H , $I_{H,and}R_H$ respectively. Thus, $N_H(t)=S_H(t) + E_H(t) + I_H(t) + R_H(t)$. The mosquito vector population $N_m(t)$ has the subclasses denoted by $S_m(t), E_m(t), and I_m(t)$ for the susceptible, exposed and infected subclasses, respectively. Thus, $N_m(t)=S_m(t) + E_m(t) + I_H(t) + I_H(t) + I_H(t) + I_H(t) + I_H(t)$

 $I_m(t)$. The immune class in mosquito population does not exist since mosquitoes once infected never recover from infection that is their infection period ends with their death.

Complete interaction and transfer diagram of both human and mosquito population is showed in Fig 1. The compartmental deterministic mathematical model can be represented analytically by the following the non linear system of seven ordinary differential equation

$\frac{dS_H}{dt} = b_H - \frac{bb_H S_H I_m}{I + V_H I_m} - m_H S_H$	Ü Ï Ï Ï	
$\frac{dE_{H}}{dt} = \frac{bb_{H}S_{H}I_{m}}{I + V_{H}I_{m}} + Pb_{H}E_{H} + qb_{H}I_{H} - (a_{H} + m_{H})E_{H}$	1 1 1 1 1 1 1	
$\frac{dI_H}{dt} = a_H E_H - (g_H + m_H + e_H)I_H$		
$\frac{dR_H}{dt} = g_H I_H - m_H R_H$	 - - - - - - - - - - - - - - - - - -	(
$\frac{dS_m}{dt} = b_m - \frac{bb_m S_m I_H}{I + V_m I_H} - m_m S_m$		
$\frac{dE_m}{dt} = \frac{bb_m S_m I_H}{I + V_m I_H} - (a_m + m_m) E_m$		
$\frac{dI_m}{dt} = a_m E_m - m_m I_m$		

(2.1)

With initial conditions

$$S_{H}(0)^{3} 0, \quad E_{H}(0)^{3} 0, \quad I_{H}(0)^{3} 0, \quad R_{H}(0)^{3} 0, \quad S_{m}(0)^{3} 0, \quad E_{m}(0)^{3} 0, \quad I_{m}(0)^{3} 0$$
 (2.2)

Special Case Model

After formulation of both Horizontal and vertical transmission of zika virus, it is important to gain insight into the dynamic of Horizontal transmission (mosquito transmission). The special case model is obtained by assuming that zika virus is not vertically transmitted, that is p = q = 0 which reduces the equation of (2.1) to the following.

$$\frac{dS_{H}}{dt} = b_{H} - \frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} - \mu_{H}S_{H}$$

$$\frac{dE_{H}}{dt} = \frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} - G_{1}E_{m}$$

$$\frac{dI_{H}}{dt} = \alpha_{H}E_{H} - G_{2}I_{H}$$

$$\frac{dR_{H}}{dt}\gamma_{H}I_{h} - \mu_{H}R_{H}$$

$$\frac{dS_{m}}{dt} = b_{m} - \frac{b\beta_{m}S_{m}I_{H}}{1 + V_{H}I_{m}} - \mu_{m}S_{m}$$

$$\frac{dE_{m}}{dt} = \frac{b\beta_{m}S_{m}I_{H}}{1 + V_{H}I_{m}} - G_{3}E_{m}$$

$$\frac{dI_{m}}{dt} = \alpha_{m}E_{m} - \mu_{m}I_{m}$$
(2.3)

Where $G_1 = (\alpha_H + \mu_H)$, $G_2 = (\gamma_H + \mu_H + \varepsilon_H)$ and $G_3 = (\alpha_m + \mu_m)$

Here b_H is the natural birth rate of human, a_H is the rate at which expose human move to the infections class and human are recovered at the rate g_H , μ_H is the natural mortality rate of human also ε_H , is the disease related death rate. Similarly b_m is the constant recruitment rate of susceptible mosquitoes population by birth, α_m is the rate at which exposed mosquitoes move to infections class and the mosquitoes leave the population through natural death rate μ_m . In a

standard SEIR compartment model, the vertical transmission can be incorporated by assuming that a fraction of the offspring of infected hosts (both E and I) are infected either during early pregnancy or at the point of birth. The term $b\beta_H S_H(1)I_m(t)$, a bilinear incidence used by Lashari et.al (2012), refers to the rate at which the susceptible human $S_H(t)$ gets infected by infectious mosquitoes. In this work, we use a saturated incidence of the form $\frac{bb_H S_H(t)I_m(t)}{1+V_H I_m(t)}$ used by Olaniyi

and Obabiyi (2013). Where $\frac{1}{1+V_H I_m(t)}$ denotes a saturating feature that inhibits the force of infection from infectious mosquitoes to susceptible humans in order other words, it produces antibodies at $V_{\scriptscriptstyle H} \in [0,1]$ in response to the presence of infectious Aedes mosquitoes. Similarly, $b\beta_m(t)S_m(t)I_H(t)$ refers to the rate at which the susceptible mosquitoes $S_m(t)$ are infected by infectious human $I_H(t)$. Since mosquitoes have DNA like humans, Noutcha et.al (2011). They also develop antibodies against the zika virus. Thus, we use a saturated force of infection of the form $\frac{b\beta_m(t)S_m(t)I_H(t)}{1+V_mI_H(t)}$, where $V_m \in [0,1]$ is the rate at which antibodies are produced against the zika infection caused by infectious humans.

MODEL ANALYSIS

Invariant Property

Here, we provide the following results which guarantee that the zika virus model governed by system (2.1) is epidemiologically and mathematically well-posed in a feasible region Ω given by:

$$\Omega = \Omega_{\rm H} \times \Omega_{\rm m} \subset {\rm R}^4_+ \times {\rm R}^3_+$$

Where

$$\Omega_{\rm H} = \{ (S_{\rm H,} E_{\rm H,} I_{\rm H,} R_{\rm H}) \in R_+^4 : \le \frac{b_{\rm H}}{\mu_{\rm H}} \}$$

And

$$\Omega_{\rm m} = \{ (S_{\rm m,} E_{\rm m,} I_{\rm m}) \in R^3_+ : \le \frac{b_{\rm m}}{\mu_{\rm m}} \}$$

Theorem 1: There exists a domain Ω in which the solution set $\{S_H, E_H, I_H, R_H, S_m, E_m, I_m\}$ is contained and bounded

Proof: Given the solution set $\{S_H, E_H, I_H, R_H, S_m, E_m, I_m\}$ with positive initial condition (2.2) we let

 $N_H = S_H + E_H + I_H + R_H$ Human population

 $N_m = S_m + E_m + I_m$ Mosquito population

Then the total derivatives of human population dynamics is given by

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dR_H}{dt}$$

That is, $\frac{dN_H}{dt} = b_H - \mu_H (S_H + E_H + I_H + R_H) - \varepsilon_H I_H \le b_H - \mu_H N_H$
This imply that $\frac{dN_H}{dt} \le b_H - \mu_H N_H$

Rewriting the above we have

$$\frac{dN_H}{dt} + \mu_H N_H \le b_H \tag{3.1}$$

Solving (3.1) using integrating factor which is $e^{\mu_H t}$

We now multiply both sides of through by integrating factor $e^{\mu_H t}$

$$\frac{d}{dt}(N_H e^{\mu_H t}) \le b_H e^{\mu_H t}$$
(3.2)

Integrating (3.2), we have

$$N_H e^{\mu_H t} \le \frac{b_H}{\mu_H} e^{\mu_H t} + \mathcal{K}$$
(3.3)

Multiply (3.3) through by $e^{-\mu_H t}$

$$N_H \le \frac{b_H}{\mu_H} + \mathrm{K}e^{-\mu_H t}$$
(3.4)

As t $\rightarrow \infty$, $N_H \leq \frac{b_H}{\mu_H}$

Similarly mosquitoes' population is given by

$$\frac{dN_m}{dt} = b_m - \mu_m (S_m + E_m + I_m)$$
(3.5)
$$\Rightarrow \frac{dN_m}{dt} \le b_m - \mu_m N_m$$
(3.6)

Rewriting (3.6), we have

$$\frac{dN_m}{dt} + \mu_m N_m \le b_m \tag{3.7}$$

Solving (3.7), using integrating factor which is $e^{\mu_m t}$.

Multiply through by $e^{\mu_m t}$, we have

$$\frac{d}{dt}(N_m e^{\mu_m t}) \le b_m e^{\mu_m t}$$
(3.8)

Integrating (3.8), we have

$$N_m e^{\mu_m t} \le \frac{b_m}{\mu_m} e^{\mu_m t} + \mathbf{K}$$
(3.9)

Multiply (3.9) through by $e^{-\mu_m t}$, we have

$$N_m \le \frac{b_m}{\mu_m} + \mathrm{K}e^{-\mu_m t}$$
(3.10)

As t $\rightarrow \infty$, $N_m \leq \frac{b_m}{\mu_m}$

Thus all solutions of the humans and mosquitoes population only are confined in the feasible region Ω . Showing that the

Feasible region for the formulated model (2.1) exists and is given by

$$\Omega = \{ (S_H, E_H, I_H, R_H S_m, E_m, I_m) \in \mathbb{R}^7, N_H \le \frac{b_H}{\mu_H}, N_m \le \frac{b_m}{\mu_m} \}$$

This end proof.

Positivity of Solution

Theorem 2: The solution $\{S_H, E_H, I_H, R_H, S_m, E_m, I_m\}$ of the zika model with non-negative initial condition (2.2) in the feasible domain Ω remains non-negative in Ω for all t > 0

Proof: We will carry out the proof following ideals by Lashari et al (2012) and Chiyaka et al (2008) we assumed that for all $t \ge 0$, let there exists t > 0 such that $S_H = 0$, and $S_H, E_{H,}I_{H,}R_{H,}S_m, E_{m,}I_m > 0$ for $0 < t < t_*$

From the first equation of (2.1) we have

$$\frac{dS_H}{dt} = b_H - \frac{b\beta_H S_H I_m}{1 + V_H I_m} - \mu_H S_H$$
(3.11)
$$\frac{dS_H}{dt} > b_H - \mu_H S_H$$

$$\frac{dS_H}{dt} + \mu_H S_H - b_H > 0$$
(3.12)

Solving (3.12), we obtained

$$\frac{d}{dt} \left(S_H(t) e^{\mu_H t} \right) = \left(b_H \right) e^{\mu_H t} dt$$
(3.13)

Integrating (3.13) from 0 to t_*

$$\left(S(t)e^{\mu_{H}t} \right) \Big|_{0}^{t^{*}} > \frac{b_{H}}{\mu_{H}} e^{\mu_{H}t} t \Big|_{0}^{t^{*}}$$

$$S_{H}(t_{*})e^{\mu_{H}t_{*}} - S_{H}(0) = \int_{0}^{t_{*}} (b_{H})e^{\mu_{H}t} dt$$

$$(3.14)$$

Multiplying (3.14) by $e^{\mu_H t_*}$

$$S(t^*) \to \frac{b_H}{\mu_H} as \ t \to \infty$$
$$\therefore S(t^*) > 0 \ for \ all \ t > 0$$

Hence, $S_H(t_*) > 0$ for all t > 0

Using the second equation of (2.1) we have

$$\frac{dE_{H}}{dt} = \frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} + Pb_{H}E_{H} + qb_{H}I_{H} - (\alpha_{H} + \mu_{H})E_{H}$$
(3.15)

Rewriting (3.15) as

$$\frac{dE_H}{dt} + (\alpha_H + \mu_H - Pb_H)E_H = \frac{b\beta_H S_H I_m}{1 + V_H I_m} + qb_H I_H$$
(3.16)

Solving the first order differential equation (3.16), we obtained

$$\frac{d}{dt} \left(E_{H}(t) e^{(\alpha_{H} + \mu_{H} - Pb_{H})t} \right) = \left(\frac{b\beta_{H}S_{H}(t)I_{m}(t)}{1 + V_{H}I_{m}(t)} + qb_{H}I_{H} \right) e^{(\alpha_{H} + \mu_{H} - Pb_{H})t}$$
(3.17)

Integrating (3.17) from 0 to t_* , we have

$$E_{H}(t)e^{(\alpha_{H}+\mu_{H}-Pb_{H})t}\Big|_{0}^{t_{*}} = \int_{0}^{t_{*}}\left(\frac{b\beta_{H}S_{H}(t)I_{m}(t)}{1+V_{H}I_{m}(t)} + qb_{H}I_{H}(t)\right)e^{(\alpha_{H}+\mu_{H}-Pb_{H})t}dt$$

$$E_{H}(t_{*})e^{(\alpha_{H}+\mu_{H}-Pb_{H})t} - E_{H}(0) = \int_{0}^{t_{*}}\left(\frac{b\beta_{H}S_{H}(t)I_{m}(t)}{1+V_{H}I_{m}(t)} + qb_{H}I_{H}(t)\right)e^{(\alpha_{H}+\mu_{H}-Pb_{H})t}dt$$
(3.18)

Multiplying (3.18) by $e^{-(\alpha_H + \mu_H - Pb_H)t}$, we obtain

$$E_{H}(t_{*}) = E_{H}(0)e^{-(\alpha_{H}+\mu_{H}-Pb_{H})t_{*}} + e^{-(\alpha_{H}+\mu_{H}-Pb_{H})t_{*}} \times \int_{0}^{t_{*}} \left(\frac{b\beta_{H}S_{H}(t)I_{m}(t)}{1+V_{H}I_{m}(t)} + qb_{H}I_{H}(t)\right)e^{(\alpha_{H}+\mu_{H}-Pb_{H})t}dt > 0$$

Hence, $E_H(t_*) > 0$ for all t > 0

From the third equation of (2.1) we have.

$$\frac{dI_H}{dt} = \alpha_H E_H - (\gamma_H + \mu_H + \varepsilon_H)I_H$$
(3.19)

Solving (3.19), we obtained

$$\frac{d}{dt} (I_H(t) e^{(\gamma_H + \mu_H + \varepsilon_H)}) = \alpha_H E_H(t) e^{(\gamma_H + \mu_H + \varepsilon_H)}$$
(3.20)

Integrating (3.20) from 0 to t_* , we have

$$I_{H}(t)e^{(\gamma_{H}+\mu_{H}+\varepsilon_{H})}\Big|_{0}^{t_{*}} = \int_{0}^{t_{*}}\alpha_{H}E_{H}(t)e^{(\gamma_{H}+\mu_{H}+\varepsilon_{H})}$$
$$I_{H}(t_{*})e^{(\gamma_{H}+\mu_{H}+\varepsilon_{H})t_{*}} - I_{H}(0) = \int_{0}^{t_{*}}\alpha_{H}E_{H}(t)e^{(\gamma_{H}+\mu_{H}+\varepsilon_{H})t}dt$$
(3.21)

Multiplying (3.21) through by $e^{-(\gamma_H + \mu_H + \varepsilon_H)t_*}$

$$I_{H}(t_{*}) = I_{H}(0)e^{-(\gamma_{H}+\mu_{H}+\varepsilon_{H})t_{*}} + e^{-(\gamma_{H}+\mu_{H}+\varepsilon_{H})t_{*}} \times \int_{0}^{t_{*}} \alpha_{H}E_{H}(t)e^{(\gamma_{H}+\mu_{H}+\varepsilon_{H})t}dt > 0$$

Hence, $I_H(t_*) > 0$ for all t > 0

Considering the fourth equation of (2.1)

$$\frac{dR_H}{dt} = \gamma_H I_H - \mu_H R_H$$
(3.22)

Solving (3.22), we obtained

$$\frac{d}{dt} \left(R_H e^{\mu_H t} \right) = \gamma_H I_H(t) e^{\mu_H t}$$
(3.23)

Integrating (3.23) from 0 to t_*

$$R_{H}e^{\mu_{H}t}\Big|_{0}^{t_{*}} = \int_{0}^{t_{*}}\gamma_{H}I_{H}(t)e^{\mu_{H}t}dt$$

$$R_{H}(t_{*})e^{\mu_{H}t} - R_{H}(0) = \int_{0}^{t_{*}}\gamma_{H}I_{H}(t)e^{\mu_{H}t}dt$$
(3.24)

Multiply (3.24) through by $e^{-\mu_H t_*}$

$$R_{H}(t_{*}) = R_{H}(0)e^{-\mu_{H}t_{*}} + e^{-\mu_{H}t_{*}} \times \int_{0}^{t_{*}} \gamma_{H}I_{H}(t)e^{\mu_{H}t}dt > 0$$

Hence, $R_H(t_*) > 0$ for all t > 0

Considering the fifth equation of (2.1) we have,

$$\frac{dS_m}{dt} = b_m - \frac{b\beta_m S_m I_H}{1 + V_m I_H} - \mu_m S_m$$
(3.25)
$$\Rightarrow \frac{dS_m}{dt} > b_m - \mu_m S_m$$

Re-writing (3.25) as

$$\frac{dS_m}{dt} + \mu_m S_m > b_m$$
(3.26)

Solving (3.26), we obtained

$$\frac{d}{dt}(S_m e^{\mu_m t}) > b_m e^{\mu_m t}$$
$$\Rightarrow d(S_m e^{\mu_m t}) > b_m e^{\mu_m t} dt$$
$$(3.27)$$

Integrating (3.27) from 0 to t_{t*} we have

$$S_{m}e^{\mu_{m}t}\Big|_{0}^{t^{*}} > \frac{b_{m}e^{\mu_{m}t}}{\mu_{m}}\Big|_{0}^{t^{*}}$$
$$S_{m}e^{\mu_{m}t^{*}} > S_{m}(0) + \frac{b_{m}e^{\mu_{m}t^{*}}}{\mu_{m}}$$

Multiplying through by $e^{-\mu_m t^*}$ we have

$$S_m(t) > S_m(0)e^{-\mu_m t^*} + \frac{b_m}{\mu_m}$$
$$S_m(t) \to \frac{b_m}{\mu_H} \text{ as } t \to \infty$$
$$S_m(t) > 0 \text{ for all } t > 0$$

Considering the sixth equation of (2.1)

$$\frac{dS_m(t)}{dt} = \frac{b\beta_m S_m I_H}{1 + V_m I_H} - (\alpha_m + \mu_m) E_m$$
(3.28)

Solving (3.28), we obtained

$$\frac{d}{dt}\left(E_m(t)e^{(\alpha_m+\mu_H)t}\right) = \left(\frac{b\beta_m S_m I_H}{1+V_m I_H}\right)e^{(\alpha_m+\mu_H)t}dt$$
(3.29)

Integrating (3.29) from 0 to t_* , we have

$$E_{m}(t)e^{(\alpha_{m}+\mu_{H})t}\Big|_{0}^{t_{*}} = \int_{0}^{t_{*}} \left(\frac{b\beta_{m}S_{m}(t)I_{H}(t)}{1+V_{m}I_{H}(t)}\right)e^{(\alpha_{m}+\mu_{H})t}dt$$

$$E_{m}(t)e^{(\alpha_{m}+\mu_{H})t} - E_{m}(0) = \int_{0}^{t_{*}} \left(\frac{b\beta_{m}S_{m}(t)I_{H}(t)}{1+V_{m}I_{H}(t)}\right)e^{(\alpha_{m}+\mu_{H})t}dt$$
(3.30)

Multiplying (3.30) through by $e^{-(\alpha_m + \mu_H)t}$

$$E_{m}(t) = E_{m}(0)e^{-(\alpha_{m}+\mu_{H})t} + e^{-(\alpha_{m}+\mu_{H})t} \times \int_{0}^{t_{*}} \left(\frac{b\beta_{m}S_{m}(t)I_{H}(t)}{1+V_{m}I_{H}(t)}\right)e^{(\alpha_{m}+\mu_{H})t}dt > 0$$

Hence, $E_m(t) > 0$ for all t > 0

Finally, considering the sixth equation of (2.1)

$$\frac{dI_m}{dt} = \alpha_m E_m - \mu_m I_m$$
(3.31)

Solving (3.31), we obtained

 \Rightarrow $I_m \ge 0$, as $t \to \infty$

Hence, the solution $\{S_H, E_H, I_H, R_H, S_m, E_m, I_m\}$ of the Zika model (2.1) with non-negative initial condition (2.2) in the feasible domain Ω remains non-negative in Ω for all t > 0

Existence of Steady State (Equilibrium) Points

The steady states occur at points at which the differential equations of the model (2.1) equal to zero, that is

$$\left(\frac{dS_H}{dt} = \frac{dE_H}{dt} = \frac{dI_H}{dt} = \frac{dR_H}{dt} = \frac{dS_m}{dt} = \frac{dE_m}{dt} = \frac{dI_m}{dt}\right) = 0$$

This implies that

$$b_{H} - \frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} - \mu_{H}S_{H} = 0$$
(3.32)
$$\frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} + pb_{H}E_{H} + qb_{H}I_{H} - (\alpha_{H} + \mu_{H})E_{H} = 0$$
(3.33)
$$\alpha_{H}E_{H} - (\gamma_{H} + \mu_{H} + \varepsilon_{H})I_{H} = 0$$
(3.34)
$$\gamma_{H}I_{H} - \mu_{H}R_{H} = 0$$
(3.35)
$$b_{m} - \frac{b\beta_{m}S_{m}I_{H}}{1 + V_{m}I_{H}} - \mu_{m}S_{m} = 0$$
(3.36)
$$\frac{b\beta_{m}S_{m}I_{H}}{1 + V_{m}I_{H}} - (\alpha_{m} + \mu_{m})E_{m} = 0$$
(3.37)
$$\alpha_{m}E_{m} - \mu_{m}I_{m} = 0$$
(3.38)

Solving (3.32) to (3.38) gives the equilibrium at

$$S_{H}^{*} = \frac{b_{H} + b_{H}V_{H}I_{m} - b\beta_{H}S_{H}I_{m}}{(1 + V_{H}I_{m})\mu_{H}}$$

$$E_{H}^{*} = \frac{b\beta_{H}S_{H}I_{m} - (1 + V_{H}I_{m})qb_{H}I_{H}}{(1 + V_{H}I_{m})(\alpha_{H} + \mu_{H} - pb_{H})}$$

$$I_{H}^{*} = \frac{\alpha_{H}E_{H}}{(\gamma_{H} + \mu_{H} + \varepsilon_{H})}$$

$$R_{H}^{*} = \frac{\gamma_{H}I_{H}}{\mu_{H}}$$

$$S_{m}^{*} = \frac{b_{m} + b_{m}V_{m}I_{H} - bb_{m}S_{m}I_{H}}{(1 + V_{m}I_{H})\mu_{m}}$$

$$E_{m}^{*} = \frac{b\beta_{m}S_{H}I_{m}}{(1 + V_{m}I_{H})(\alpha_{m} + \mu_{m})}$$

$$I_{m}^{*} = \frac{\alpha_{m}E_{m}}{\mu_{m}}$$

(3.39)

It is Important to note that there is no trivial equilibrium points as long as the natural birth rate of human and mosquito b_H and b_m respectively are not zero. This means that, $(S_H^*, E_H^*, I_H^*, R_H^*, S_m^*, E_m^*, I_m^*) \neq (0,0,0,0,0,0)$

Existence of Disease-free Equilibrium Point

Disease free equilibrium points are steady-state solutions where there is no zika virus. That is where $E_H = I_H = E_m = I_m = 0$

Substituting the values of $E_H = I_H = E_m = I_m = 0$ into (3.39), we have

$$S_H^* = \frac{b_H}{\mu_H}$$
 and $S_m^* = \frac{b_m}{\mu_{mH}}$

Thus we have the disease-free steady (equilibrium) state as

$$\varepsilon_0 = \left(\frac{b_H}{\mu_H}, 0, 0, 0, \frac{b_m}{\mu_m}, 0, 0\right)$$

Basic Reproduction Number (R_0) of model (2.1)

This is an important notation in epidemiology and it is usually denoted by R_0 . This is the expected average number of new infections produced horizontal and vertically by a single infective when introduced into a completely susceptible population. Its significance in epidemiology is the fact that it determines whether a disease will spread through the population or not. To obtain R_0 for our model (2.1), we adapt the next generation matrix and method described by, van den Driessche and Watmough (2002).

Let X be the set of the entire disease compartments. That is,

$$X = \left(E_H, I_H, E_m, I_m\right)$$

Then our model can be written as

$$\frac{dX}{dt} = f(X) - v(X)$$
(3.40)

where f(X) is the rate of appearance of new infection into the disease compartments.

$$f(x) = \begin{pmatrix} \frac{b\beta_H S_H I_m}{1 + V_H I_m} + pb_H E_H + qb_H I_H \\ 0 \\ \frac{b\beta_m S_m I_H}{1 + V_m I_H} \\ 0 \end{pmatrix}$$

v(x) is the rate of transfer of individual in and out of the disease compartments, it is defined by $v(x) = v_{(x)}^- - v_{(x)}^+$

Where $v_{(x)}^{-}$ is the rate of transfer of diseases out of the disease compartments

$$v_{(x)}^{-} = \begin{pmatrix} (\alpha_{H} + \mu_{H}) E_{H} \\ (e_{H} + \gamma_{H} + \mu_{H}) I_{H} \\ (\alpha_{m} + \mu_{m}) E_{m} \\ \mu_{m} I_{m} \end{pmatrix}$$

and $v_{(x)}^{+}$ is the rate of transfer of disease into the disease compartment by other means.

$$v_{(x)}^{+} = \begin{pmatrix} 0 \\ \alpha_{H} E_{H} \\ 0 \\ \alpha_{m} E_{m} \end{pmatrix}$$

therefore

$$v(x) = v_{(x)}^{-} - v_{(x)}^{+} = \begin{pmatrix} (\alpha_{H} + \mu_{H})E_{H} \\ (e_{H} + \gamma_{H} + \mu_{H})I_{H} - \alpha_{H}E_{H} \\ (\alpha_{m} + \mu_{m})E_{m} \\ \mu_{m}I_{m} - a_{m}E_{m} \end{pmatrix}$$

F is the Jacobian of f(x) with respect to disease compartments evaluated at the point ε_0 .

$$F = \begin{pmatrix} Pb_{H} & qb_{H} & 0 & \frac{b\beta_{H}b_{H}}{\mu_{H}} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{b\beta_{m}b_{m}}{\mu_{m}} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

V is the Jacobian of v(x) with respect to disease compartments evaluated at Point ε_0 .

$$V = \begin{pmatrix} G_1 & 0 & 0 & 0 \\ -\alpha_H & G_2 & 0 & 0 \\ 0 & 0 & G_3 & 0 \\ 0 & 0 & -\alpha_m & \mu_m \end{pmatrix}$$
$$V^{-1} = \begin{pmatrix} \frac{1}{G_1} & 0 & 0 & 0 & 0 \\ \frac{\alpha_H}{G_1 G_2} & \frac{1}{G_2} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{G_3} & 0 \\ 0 & 0 & 0 & \frac{\alpha_m}{G_3 \mu_m} & \frac{1}{\mu_m} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{G_2 P b_H + q b_H \alpha_H}{G_1 G_2} & \frac{P b_H}{G_2} & \frac{b_H b \beta_H \alpha_m}{G_3 \mu_H \mu_m} & \frac{b_m b \beta_H}{\mu_H \mu_m} \\ 0 & 0 & 0 & 0 \\ \frac{b_m b \beta_m \alpha_H}{G_1 G_2 \mu_m} & \frac{b_m b \beta_m}{G_2 \mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

The basic reproduction number $R_0 = p(FV^{-1})$ is the spectral radius of the product FV^{-1} . Hence for our model (2.1) we have.

$$R_{0} = \frac{G_{3}(G_{2}pb_{H} + qb_{H}\alpha_{H})\mu_{H}\mu_{m} + \sqrt{G_{3}\mu_{H}(4b_{H}b_{m}b^{2}\beta_{H}\beta_{m}G_{1}G_{2}\alpha_{H}\alpha_{m} + G_{3}(G_{2}pb_{H} + qb_{H}\alpha_{H})^{2}\mu_{H}\mu_{m}^{2}}{2G_{1}G_{2}G_{3}\mu_{H}\mu_{m}}$$

Where
$$G_1 = (\alpha_H + \mu_H)$$
, $G_2 = (\gamma_H + \mu_H + \varepsilon_H)$ and $G_3 = (\alpha_m + \mu_m)$

Basic Reproduction Number $R_0 \mid_{p=q=0}$ of model (2.3)

This is the expected average number of new infections produce horizontally by single infective when introduced into a completely susceptible population.

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_H b_H}{\mu_H} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{b\beta_m b_m}{\mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} G_1 & 0 & 0 & 0 \\ -\alpha_H & G_2 & 0 & 0 \\ 0 & 0 & G_3 & 0 \\ 0 & 0 & -\alpha_m & \mu_m \end{pmatrix}$$
$$V^{-1} = \begin{pmatrix} \frac{1}{G_1} & 0 & 0 & 0 \\ \frac{\alpha_H}{G_1 G_2} & \frac{1}{G_2} & 0 & 0 \\ 0 & 0 & \frac{1}{G_3} & 0 \\ 0 & 0 & \frac{\alpha_m}{G_3 \mu_m} & \frac{1}{\mu_m} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{b_H b \beta_H \alpha_m}{G_3 \mu_H \mu_m} & \frac{b_H b \beta_H}{\mu_H \mu_m} \\ 0 & 0 & 0 & 0 \\ \frac{b_m b \beta_m \alpha_m}{G_1 G_2 \mu_m} & \frac{b_m b \beta_m}{G_2 \mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

The basic reproduction number $R_0|_{p=q=0} = \rho(FV^{-1})$ is the spectral radius of the product FV^{-1}

$$R_{0}|_{p=q=0} = \sqrt{\frac{b^{2}\beta_{H}b_{H}\alpha_{H}\beta_{m}b_{m}\alpha_{m}}{\mu_{H}G_{1}G_{2}G_{3}\mu_{m}^{2}}}$$

where

$$R_H \mid_{p=q=0} = \frac{b\beta_H b_H \alpha_H}{\mu_H G_1 G_2}$$
 and

$$R_m \mid_{p=q=0} = \frac{b\beta_m b_m \alpha_m}{\mu_m^2 G_3}$$

We can actually say that $R_0^2 = R_H R_m$

This implies that $R_0 = \sqrt{R_H R_m}$

Stability of the Disease-free Equilibrium Point

Theorem 3.3: The disease-free equilibrium (DFE), ε_0 is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The Jacobian matrix of the system (2.1) evaluated at the disease-free equilibrium point ε_0 , is obtained as

$$\begin{split} J(E^0) = \begin{pmatrix} -\mu_{^{_{H}}} & 0 & 0 & 0 & 0 & 0 & \frac{b\beta_{H}b_{^{_{H}}}}{\mu_{_{H}}} \\ 0 & -(\alpha_{_{H}} + \mu_{_{H}}) + pb_{_{H}} & 0 & 0 & 0 & 0 & \frac{b\beta_{H}b_{^{_{H}}}}{\mu_{_{H}}} \\ 0 & \alpha_{_{H}} & -(\gamma_{_{H}} + \mu_{_{H}} + \varepsilon_{_{H}}) & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_{_{H}} & -\mu_{_{H}} & 0 & 0 & 0 \\ 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} & 0 & -\mu_{_{H}} & 0 & 0 \\ 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} & 0 & 0 & -(\alpha_{_{H}} + \mu_{_{H}}) & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_{_{M}} & -\mu_{_{H}} \end{pmatrix} \\ \\ \left| J(E^0) - I\lambda \right| = \begin{pmatrix} -\mu_{_{H}} - \lambda & 0 & 0 & 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} \\ 0 & -(\alpha_{_{H}} + \mu_{_{H}}) + pb_{_{H}} - \lambda & 0 & 0 & 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} \\ 0 & -(\alpha_{_{H}} + \mu_{_{H}}) + pb_{_{H}} - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_{_{H}} & -\mu_{_{H}} - \lambda & 0 & 0 & 0 \\ 0 & 0 & \gamma_{_{H}} & -\mu_{_{H}} - \lambda & 0 & 0 & 0 \\ 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} & 0 & -\mu_{_{H}} - \lambda & 0 & 0 \\ 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} & 0 & 0 & -(\alpha_{_{H}} + \mu_{_{H}}) - \lambda & 0 \\ 0 & 0 & \frac{b\beta_{_{H}}b_{^{_{H}}}}{\mu_{_{H}}} & 0 & 0 & -(\alpha_{_{H}} + \mu_{_{H}}) - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_{_{H}} & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_{_{H}} & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}} - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_{_{H}}$$

$$\left|J - I\lambda\right| = 0 \Rightarrow (-\mu_m - \lambda_7)[-(\alpha_m + \mu_m) - \lambda_6](-\mu_m - \lambda_5)[-\mu_H - \lambda_4][-(\gamma_H + \mu_H + \varepsilon_H) - \lambda_3][-(\alpha_H + \mu_H) + pb_H - \lambda_2][-\mu_H - \lambda_1] = 0$$

$$\Rightarrow \lambda_1 = -\mu_H, \lambda_2 = -(\alpha_H + \mu_H) + pb_H, \lambda_3 = -(\gamma_H + \mu_H + \varepsilon_H), \lambda_4 = -\mu_H, \lambda_5 = -\mu_m, \lambda_6 = -(\alpha_m + \mu_m), \lambda_7 = -\mu_m, \lambda_8 = -(\alpha_H + \mu_H) + pb_H, \lambda_8 = -(\gamma_H + \mu_H + \varepsilon_H), \lambda_8 = -(\alpha_H + \mu_H) + pb_H, \lambda_8 = -(\gamma_H + \mu_H + \varepsilon_H), \lambda_8 = -(\alpha_H + \mu_H) + pb_H, \lambda_8 = -(\gamma_H + \mu_H + \varepsilon_H), \lambda_8 = -(\alpha_H + \mu_H) + pb_H, \lambda_8 = -(\gamma_H + \mu_H + \varepsilon_H), \lambda_8 = -(\alpha_H + \mu_H) + pb_H, \lambda_8 = -($$

Since all the real parts of the seven eigen-values are negative, the DFE is locally asymptotically stable. This ends the proof■.

Global Stability Analysis

Theorem 3:4: The disease-free equilibrium, ε_0 of the model (2.3), is globally asymptotically stable in Ω if $R_0 \mid_{p=q=0} \le 1$.

Proof: To establish the global stability of the disease-free equilibrium ε_0 , we consider the following linear Lyapunov function (L).

$$L = C_1 E_H + C_2 I_H + C_3 I_H + C_4 E_m$$
(3.42)

Where
$$C_1 = \frac{b_m b \beta_m \alpha_m \alpha_H}{G_1 G_2 G_3 \mu_m^2}$$
, $C_2 = \frac{b_m b \beta_m \alpha_m}{G_2 G_3 \mu_m^2}$, $C_3 = \frac{R_0 \alpha_m}{G_3 \mu_m}$ and $C_4 = \frac{R_0}{\mu_m}$

Then substituting the Values of C_1, C_2, C_3 and C_4 into (3.42), we have

$$L = \frac{b_{m}b\beta_{m}\alpha_{m}\alpha_{H}}{G_{1}G_{2}G_{3}\mu_{m}^{2}}E_{H} + \frac{b_{m}b\beta_{m}\alpha_{m}}{G_{2}G_{3}\mu_{m}}I_{H} + \frac{R_{0}\alpha_{m}}{G_{3}\mu_{m}}E_{m} + \frac{R_{0}}{\mu_{m}}I_{m}$$
(3.43)

Differentiating (3.43) with respect to disease compartments

$$\dot{L} = \frac{b_m b \beta_m \alpha_m \alpha_H}{G_1 G_2 G_3 \mu_m^2} \dot{E}_H + \frac{b_m b \beta_m \alpha_m}{G_2 G_3 \mu_m} \dot{I}_H + \frac{R_0 \alpha_m}{G_3 \mu_m} \dot{E}_m + \frac{R_0}{\mu_m} \dot{I}_m$$
(3.44)

Substituting the values of \dot{E}_{H} , \dot{I}_{H} , \dot{E}_{m} and \dot{I}_{m} from model (2.3) into (3.44)

$$\dot{L} = \frac{b_m b \beta_m \alpha_m \alpha_H}{G_1 G_2 G_3 \mu_m^2} \left[\frac{b \beta_H S_H I_m}{1 + V_H I_m} - G_1 E_H \right] + \frac{b_m b \beta_m \alpha_m}{G_1 G_2 G_3 \mu_m^2} \left[\alpha_H E_H - G_2 I_H \right] + \frac{R_0 \alpha_m}{G_3 \mu_m} \left[\frac{b \beta_m S_m I_H}{1 + V_m I_m} - G_3 E_m \right] + \frac{R_0 \alpha_m}{\mu_m} \left[\alpha_m E_m - \mu_m I_m \right]$$

$$= \frac{b^{2}\beta_{H}\alpha_{H}\beta_{m}b_{m}\alpha_{m}S_{H}I_{m}}{\mu_{m}^{2}G_{1}G_{2}G_{3}(1+V_{H}I_{m})} - \frac{b_{m}b\beta_{m}\alpha_{m}\alpha_{H}E_{H}}{\mu_{m}^{2}G_{2}G_{3}} + \frac{b_{m}b\beta_{m}\alpha_{m}\alpha_{H}E_{H}}{\mu_{m}^{2}G_{2}G_{3}} - \frac{b_{m}b\beta_{m}\alpha_{m}I_{H}}{G_{3}\mu_{m}^{2}} + \frac{R_{0}\alpha_{m}b\beta_{m}S_{m}I_{H}}{(1+V_{m}I_{m})\mu_{m}^{2}G_{3}} - \frac{R_{0}\alpha_{m}E_{m}}{\mu_{m}} + \frac{R_{0}\alpha_{m}E_{m}}{\mu_{m}} - R_{0}I_{m}$$

$$=\frac{b^{2}\beta_{H}\alpha_{H}\beta_{m}b_{m}\alpha_{m}S_{H}I_{m}}{\mu_{m}^{2}G_{1}G_{2}G_{3}(1+V_{H}I_{m})}-\frac{b_{m}b\beta_{m}\alpha_{m}I_{H}}{G_{3}\mu_{m}^{2}}+\frac{R_{0}\alpha_{m}b\beta_{m}S_{m}I_{H}}{(1+V_{m}I_{m})\mu_{m}^{2}G_{3}}-R_{0}I_{m}$$

Collecting like terms

$$=\left(\frac{b^2\beta_H\alpha_H\beta_m b_m \alpha_m S_H}{\mu_m^2 G_1 G_2 G_3 \left(1+V_H I_m\right)}-R_0\right)I_m + \left(\frac{R_0\alpha_m b\beta_m S_m}{\mu_m G_3 \left(1+V_m I_m\right)}-\frac{b_m b\beta_m \alpha_m}{\mu_m^2 G_3}\right)I_H$$

Substituting
$$S_H = \frac{b_H}{\mu_H}$$
 and $S_m = \frac{b_m}{\mu_m}$

$$= \left(\frac{b^2 \beta_H b_H \alpha_H \beta_m b_m \alpha_m}{\mu_H G_1 G_2 G_3 (1 + V_H I_m) \mu_m^2} - R_0\right) I_m + \left(\frac{R_0 b_m b \beta_m \alpha_m}{\mu_m^2 G_3 (1 + V_m I_m)} - \frac{b_m b \beta_m \alpha_m}{\mu_m^2 G_3}\right) I_H$$
Because $\frac{1}{1 + V_H I_m} \le 1$ and $\frac{1}{1 + V_m I_m} \le 1$

We have

$$\leq \left(\frac{b^{2}\beta_{H}b_{H}\alpha_{H}\beta_{m}b_{m}\alpha_{m}}{\mu_{H}G_{1}G_{2}G_{3}(1+V_{H}I_{m})\mu_{m}^{2}} - R_{0}\right)I_{m} + \left(\frac{R_{0}b_{m}b\beta_{m}\alpha_{m}}{\mu_{m}^{2}G_{3}(1+V_{m}I_{m})} - \frac{b_{m}b\beta_{m}\alpha_{m}}{\mu_{m}^{2}G_{3}}\right)I_{H}$$

$$\leq \left(R_0^2 - R_0\right)I_m + \left(R_0R_m - R_m\right)I_H$$

$$L \le R_0 (R_0 - 1) I_m + R_m (R_0 - 1) I_H$$

Therefore $\dot{L} \leq o$ for $R_0 |_{p=q=0} \leq 1$ and $\dot{L} = o$ if and only if $I_m = I_H = 0$ by Lasalle's invariance principle, ε_0 is globally asymptotically stable if $R_0 |_{p=q=0} \leq 1$.

Theorem 3.5: The endemic equilibrium (EE) of the model (3.3) is globally asymptotically stable whenever $R_0 \mid_{p=q=0} > 1$

To achieve this, we use the nonlinear Lyapunov function of Goh-Volterra type which has been found to be very successful. See, for instance, Geogescu and Zhang (2013) for more on the application of this Lyapunov function.

Proof: Let the endemic equilibrium of the special case model (3.3) be denoted by $\varepsilon_0 = \left(S_H^*, E_H^*, I_H^*, S_m^*, E_m^*, I_m^*\right)$ and let $R_0 \mid_{p=q=0} > 1$ so that L exists.

Consider the following nonlinear Lyapunov function.

$$L = C_{1} \left[S_{H} - S_{H}^{*} - S_{H}^{*} \ln\left(\frac{S_{H}}{S_{H}^{*}}\right) + E_{H} - E_{H}^{*} - E_{H}^{*} \ln\left(\frac{E_{H}}{E_{H}^{*}}\right) \right] + C_{2} \left[I_{H} - I_{H}^{*} - I_{H}^{*} \ln\left(\frac{I_{H}}{I_{H}^{*}}\right) \right] + C_{3} \left[S_{m} - S_{m}^{*} - S_{m}^{*} \ln\left(\frac{S_{m}}{S_{m}^{*}}\right) + E_{m} - E_{m}^{*} - E_{m}^{*} \ln\left(\frac{E_{m}}{E_{m}^{*}}\right) \right] + C_{4} \left[I_{m} - I_{m}^{*} - I_{m}^{*} \ln\left(\frac{I_{m}}{I_{m}^{*}}\right) \right]$$
(3.45)

Differentiating (3.45), we have

$$\begin{split} \dot{L} &= C_{1} \left[\dot{S}_{H} - \frac{S_{H}^{*}}{S_{H}} \dot{S}_{H} + \dot{E}_{H} - \frac{E_{H}^{*}}{E_{H}} \dot{E}_{H} \right] + C_{2} \left[\dot{I}_{H} - \frac{I_{H}^{*}}{I_{H}} \dot{I}_{H} \right] + C_{3} \left[\dot{S}_{m} - \frac{S_{m}^{*}}{S_{m}} \dot{S}_{m} + \dot{E}_{m} - \frac{E_{m}^{*}}{E_{m}} \dot{E}_{m} \right] \\ &+ C_{4} \left[\dot{I}_{m} - \frac{I_{m}^{*}}{I_{m}} \dot{I}_{m} \right] \\ &= C_{1} \left[\left(1 - \frac{S_{H}^{*}}{S_{H}} \right) \dot{S}_{H} + \left(1 - \frac{E_{H}^{*}}{E_{H}} \right) \dot{E}_{H} \right] + C_{2} \left[\left(1 - \frac{I_{H}^{*}}{I_{H}} \right) \dot{I}_{H} \right] + C_{3} \left[\left(1 - \frac{S_{m}^{*}}{S_{m}} \right) \dot{S}_{m} + \left(1 - \frac{E_{m}^{*}}{E_{m}} \right) \dot{E}_{m} \right] \\ &+ C_{4} \left[\left(1 - \frac{I_{m}^{*}}{I_{m}} \right) \dot{I}_{m} \right] \\ &\qquad (3.47) \end{split}$$

Where upper dot represents the differentiation with respect to time t. Putting the appropriate equations of model (2.3) in (3.47), we have

$$\begin{split} \dot{L} &= C_{1} \Biggl[\Biggl(1 - \frac{S_{H}^{*}}{S_{H}} \Biggr) \Biggl(b_{H} - \frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{H}} - \mu_{H}S_{H} \Biggr) + \Biggl(1 - \frac{E_{H}^{*}}{E_{H}} \Biggr) \Biggl(\frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{H}} - G_{1}E_{H} \Biggr) \Biggr] \\ &+ C_{2} \Biggl[\Biggl(1 - \frac{I_{H}^{*}}{I_{Hh}} \Biggr) (\alpha_{H}E_{H} - G_{2}I_{H} \Biggr) \Biggr] \\ &+ C_{3} \Biggl[\Biggl(1 - \frac{S_{m}^{*}}{S_{m}} \Biggr) \Biggl(b_{m} - \frac{b\beta_{m}S_{m}I_{H}}{1 + V_{m}I_{H}} - \mu_{m}S_{m} \Biggr) + \Biggl(1 - \frac{E_{m}^{*}}{E_{m}} \Biggr) \Biggl(\frac{b\beta_{m}S_{m}I_{H}}{1 + V_{m}I_{H}} - G_{3}E_{m} \Biggr) \Biggr] \\ &+ C_{4} \Biggl[\Biggl(1 - \frac{I_{m}^{*}}{I_{m}} \Biggr) (\alpha_{m}E_{m} - \mu_{m}I_{m} \Biggr) \Biggr]$$

$$(3.48)$$

At steady state of (3.48)

$$b_{H} = \frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{1 + V_{H}I_{m}} - \mu_{H}S_{H}^{*}$$

$$\frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{1 + V_{H}I_{m}} = G_{1}E_{H}^{*} \Longrightarrow G_{1} = \frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{(1 + V_{H}I_{m})E_{H}^{*}}$$

$$\alpha_{H}E_{H}^{*} = G_{2}I_{H}^{*} \Longrightarrow G_{2} = \frac{\alpha_{H}E_{H}^{*}}{I_{H}^{*}}$$

$$b_{m} = \frac{b\beta_{m}S_{m}^{*}I_{H}^{*}}{1 + V_{m}I_{H}^{*}} + \mu_{m}S_{m}^{*}$$

$$\frac{b\beta_m S_m^* I_H^*}{1 + V_m I_H} - G_3 E_m^* \Longrightarrow G_3 = \frac{b\beta_m S_m^* I_H^*}{(1 + V_m I_H) E_m^{**}}$$
$$\alpha_m E_m^* = \mu_m I_m^* \Longrightarrow \mu_m = \frac{\alpha_m E_m^*}{I_m^*}$$

Substituting the values of steady state of b_H and b_m into (3.48) we have

$$\begin{split} \dot{L} &= C_{1} \Biggl[\Biggl(1 - \frac{S_{H}^{*}}{S_{H}} \Biggr) \Biggl(\frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{1 + V_{H}I_{m}} + \mu_{H}S_{H}^{*} - \frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} + \mu_{H}S_{H} \Biggr) \\ &+ \Biggl(1 - \frac{E_{H}^{*}}{E_{H}} \Biggr) \Biggl(\frac{b\beta_{H}S_{H}I_{m}}{1 + V_{H}I_{m}} - G_{1}E_{H} \Biggr) \Biggr] + C_{2} \Biggl[\Biggl(1 - \frac{I_{H}^{*}}{I_{H}} \Biggr) (\alpha_{H}E_{H} - G_{2}I_{H}) \Biggr] \\ &+ C_{3} \Biggl[\Biggl(1 - \frac{S_{m}^{*}}{S_{m}} \Biggr) \Biggl(\frac{b\beta_{m}S_{m}^{*}I_{H}^{*}}{1 + V_{m}I_{H}} + \mu_{m}S_{m}^{*} - \frac{b\beta_{m}S_{m}I_{H}}{1 + V_{m}I_{H}} - \mu_{m}S_{m} \Biggr) \\ &+ \Biggl(1 - \frac{E_{m}^{*}}{E_{m}} \Biggr) \Biggl(\frac{b\beta_{m}S_{m}I_{H}}{(1 + V_{m}I_{H})} - G_{3}E_{m} \Biggr) \Biggr] + C_{4} \Biggl[\Biggl(1 - \frac{I_{m}^{*}}{I_{m}} \Biggr) (\alpha_{m}E_{m} - \mu_{m}I_{m}) \Biggr] \\ &- (3.49) \end{split}$$

$$C_{1} = \frac{b\beta_{m}S_{m}^{*}\alpha_{H}\alpha_{m}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}},$$

$$C_{2} = \frac{b\beta_{m}S_{m}^{*}\alpha_{m}}{G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}}$$

$$C_{3} = \frac{G_{1}G_{2}(1+V_{H}I_{m})(1+V_{M}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\beta_{H}S_{H}^{*}\alpha_{H}}$$
$$C_{4} = \frac{G_{1}G_{2}G_{3}(1+V_{H}I_{m})(1+V_{M}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\alpha_{m}\beta_{H}S_{H}^{*}\alpha_{H}}$$

Where C_1, C_2, C_3 and C_4 are the coefficients of the Lyapunov function.

Now substituting the values of C_1, C_2, C_3 and C_4 into (3.49), we have

$$\begin{split} \dot{L} &= \frac{b\beta_{m}S_{m}^{*}\alpha_{H}\alpha_{m}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\left(1 - \frac{S_{H}^{*}}{S_{H}} \right) \left(\frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{1+V_{H}I_{m}} + \mu_{H}S_{H}^{*} - \frac{b\beta_{H}S_{H}I_{m}}{1+V_{H}I_{m}} + \mu_{H}S_{H} \right) \right] \\ &+ \left(1 - \frac{E_{H}^{*}}{E_{H}} \right) \left(\frac{b\beta_{H}S_{H}I_{m}}{1+V_{H}I_{m}} - G_{1}E_{H} \right) \right] + \frac{b\beta_{m}S_{m}^{*}\alpha_{m}}{G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\left(1 - \frac{I_{H}^{*}}{I_{H}} \right) (\alpha_{H}E_{H} - G_{2}I_{H}) \right] \\ &+ \frac{G_{1}G_{2}(1+V_{H}I_{m})(1+V_{\backslash m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\beta_{H}S_{H}^{*}\alpha_{H}} \left[\left(1 - \frac{S_{m}^{*}}{S_{m}} \right) \left(\frac{b\beta_{m}S_{m}^{*}I_{H}^{*}}{1+V_{m}I_{H}} + \mu_{m}S_{m}^{*} - \frac{b\beta_{m}S_{m}I_{H}}{1+V_{m}I_{H}} - \mu_{m}S_{m} \right) \\ &+ \left(1 - \frac{E_{m}^{*}}{E_{m}} \right) \left(\frac{b\beta_{m}S_{m}I_{H}}{(1+V_{m}I_{H})} - G_{3}E_{m} \right) \right] + \frac{G_{1}G_{2}G_{3}(1+V_{H}I_{m})(1+V_{\backslash m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\alpha_{m}\beta_{H}S_{H}^{*}\alpha_{H}} \left[\left(1 - \frac{I_{m}^{*}}{I_{m}} \right) (\alpha_{m}E_{m} - \mu_{m}I_{m}) \right] \end{split}$$

$$(3.50)$$

Hence from (3.50) we collect all the terms with dot stars in the infected classes including all $\mu_H S_H, \mu_H S_H^*, \mu_m S_m$ and $\mu_m S_m^*$

$$\begin{split} \dot{L} &= \frac{b\beta_{m}S_{m}^{*}\alpha_{H}\alpha_{m}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\left(\frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{(1+V_{H}I_{m})} - \frac{b\beta_{H}S_{H}^{*}^{2}I_{m}}{(1+V_{H}I_{m})S_{H}} - \frac{b\beta_{H}S_{H}I_{m}E_{m}^{*}}{(1+V_{H}I_{m})E_{H}} - G_{1}E_{H}^{*} \right) \right] \\ &+ \frac{b\beta_{m}S_{m}^{*}\alpha_{H}\alpha_{m}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\mu_{H}S_{H}^{*} + \mu_{H}S_{H}^{*} - \frac{\mu_{H}S_{H}^{*2}}{S_{H}} - \mu_{H}S_{H} \right] + \frac{b\beta_{m}S_{m}^{*}\alpha_{m}}{G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\left(G_{2}I_{H}^{*} - \frac{\alpha_{H}E_{H}I_{H}^{*}}{I_{H}} \right) \right] \\ &+ \frac{G_{1}G_{2}(1+V_{H}I_{m})(1+V_{\vee m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\beta_{H}S_{H}^{*}\alpha_{H}} \left[\left(\frac{b\beta_{m}S_{m}^{*}I_{H}^{*}}{1+V_{m}I_{H}} - \frac{b\beta_{m}S_{m}^{*2}I_{H}}{(1+V_{m}I_{H})S_{m}} - \frac{b\beta_{m}S_{m}I_{H}E_{m}^{*}}{(1+V_{m}I_{H})E_{m}} + G_{3}E_{m} \right) \right] \\ &+ \frac{G_{1}G_{2}(1+V_{H}I_{m})(1+V_{\vee m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\beta_{H}S_{H}^{*}\alpha_{H}} \left[\mu_{m}S_{m}^{*} + \mu_{m}S_{m}^{*} - \frac{\mu_{m}S_{m}^{*2}}{S_{m}} - \mu_{m}S_{m} \right] \end{split}$$

$$+\frac{G_{1}G_{2}G_{3}(1+V_{H}I_{m})(1+V_{M}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\alpha_{m}\beta_{H}S_{H}^{*}\alpha_{H}}\left[\mu_{m}I_{m}^{*}-\frac{\alpha_{m}E_{m}I_{m}^{*}}{I_{m}}\right]$$
(3.51)

Substitute the values of steady state of G_1, G_2, G_3 inside bracket of (3.51), and factor out the common factors.

$$\begin{split} \dot{L} &= \frac{b\beta_{m}S_{m}^{*}\alpha_{H}\alpha_{m}}{G_{l}G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\left(\frac{b\beta_{H}S_{H}^{*}I_{m}^{*}}{(1+V_{H}I_{m})S_{H}} - \frac{b\beta_{H}S_{H}I_{m}E_{m}^{*}}{(1+V_{H}I_{m})S_{H}} - \frac{b\beta_{H}S_{H}I_{m}E_{m}^{*}}{(1+V_{H}I_{m})E_{H}} - \frac{b\beta_{H}S_{H}I_{m}^{*}}{(1+V_{H}I_{m})E_{H}} \right] \right] \\ &+ \frac{b\beta_{m}S_{m}^{*}\alpha_{H}\alpha_{m}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})\mu_{m}} \left[\mu_{H}S_{H}^{*} + \mu_{H}S_{H}^{*} - \frac{\mu_{H}S_{H}^{*}^{2}}{S_{H}} - \mu_{H}S_{H} \right] + \frac{b\beta_{m}S_{m}^{*}\alpha_{m}}{G_{2}G_{3}(1+V_{H}I_{H})\mu_{m}} \left[\left(\alpha_{H}E_{H}^{*} - \frac{\alpha_{H}E_{H}I_{H}^{*}}{I_{H}} \right) \right] \\ &+ \frac{G_{1}G_{2}(1+V_{H}I_{m})(1+V_{m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\beta_{H}S_{H}^{*}\alpha_{H}} \left[\left(\frac{b\beta_{m}S_{m}^{*}I_{H}^{*}}{(1+V_{m}I_{H})} - \frac{b\beta_{m}S_{m}^{*}^{2}I_{H}}{(1+V_{m}I_{H})S_{m}} - \frac{b\beta_{m}S_{m}I_{H}E_{m}^{*}}{(1+V_{m}I_{H})E_{m}} + \frac{b\beta_{m}S_{m}^{*}I_{H}}{(1+V_{m}I_{H})} \right) \right] \\ &+ \frac{G_{1}G_{2}(1+V_{H}I_{m})(1+V_{m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\beta_{H}S_{H}^{*}\alpha_{H}} \left[\mu_{m}S_{m}^{*} + \mu_{m}S_{m}^{*} - \frac{\mu_{m}S_{m}^{*}^{2}}{S_{m}} - \mu_{m}S_{m} \right] \\ &+ \frac{G_{1}G_{2}G_{3}(1+V_{H}I_{m})(1+V_{m}I_{H})}{b^{2}\beta_{m}S_{m}^{*}\alpha_{m}\beta_{H}S_{H}^{*}\alpha_{H}}} \left[\mu_{m}S_{m}^{*} + \mu_{m}S_{m}^{*} - \frac{\alpha_{m}E_{m}I_{m}^{*}}{I_{m}}} \right] \\ &= \frac{b^{2}\beta_{H}S_{m}^{*}\alpha_{m}\beta_{H}S_{m}^{*}\alpha_{H}\alpha_{H}}{b_{m}} \left[2 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H}I_{m}E_{H}^{*}}{I_{m}}} \right] \\ \\ &+ \frac{b\beta_{m}S_{m}^{*}\alpha_{m}\alpha_{H}MS_{m}^{*}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})(1+V_{m}I_{m})}\mu_{m}} \left[\left[2 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H}I_{m}E_{H}^{*}}{G_{2}G_{3}(1+V_{m}I_{H})} \mu_{m}} \right] \right] \\ &+ \frac{b\beta_{m}S_{m}^{*}\alpha_{m}\alpha_{H}MS_{m}^{*}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})}(1+V_{m}I_{H})\mu_{m}} \left[\left[2 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H}I_{m}E_{H}^{*}}{S_{H}^{*}} \right] \right] \\ &+ \frac{b(G_{1}G_{2}(1+V_{m}I_{m}))I_{H}^{*}}{G_{1}G_{2}G_{3}(1+V_{m}I_{H})}\mu_{m}} \left[\left[2 - \frac{S_{H}^{*}}{S_{m}} - \frac{S_{H}I_{m}E_{H}^{*}}{S_{m}^{*}} \right] \right] \\ &+ \frac{b(G_{1}G_{2}(1+V_{m}I_{m})M_{m}}(1+V_{m}M_{m})I_{m}}{S_{1}} \left[2 - \frac{S_{H}^{*}}{S_{m}} - \frac{S_{H}I_{H}E_{H}^{*}}{S_{1}} \right] \\ &+ \frac{b(G_{1}G_{2}(1+V_{m}I_{m})M_{m}}{b_{1}} \left[2 - \frac{S_{H}^{*}}{S_{m}} - \frac{S_{H}}{S_{m}} - \frac$$

Substituting the values of steady states of G_1, G_2, G_3 and μ_m into (3.52) we have

$$\begin{split} \dot{L} &= I_{m}^{*} \Biggl[\Biggl(2 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H}I_{m}E_{H}^{*}}{S_{H}^{*}I_{m}^{*}E_{H}} \Biggr) \Biggr] + \frac{\mu_{H}(1 + V_{m}I_{H})}{b\beta_{H}} \Biggl[2 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H}}{S_{H}^{*}} \Biggr] + I_{m}^{*} \Biggl[\Biggl(1 - \frac{E_{H}I_{H}^{*}}{E_{H}^{*}I_{H}} \Biggr) \Biggr] \\ &+ \frac{I_{m}^{*}}{(1 + V_{m}I_{H})} \Biggl[\Biggl(2 - \frac{S_{m}^{*}}{S_{m}} - \frac{S_{m}I_{H}E_{m}^{*}}{S_{m}^{*}I_{H}^{*}E_{m}} \Biggr) \Biggr] + \frac{(1 + V_{m}I_{H})\alpha_{H}E_{m}^{*}}{b\beta_{m}I_{H}^{*}} \Biggl[2 - \frac{S_{m}^{*}}{S_{m}} - \frac{S_{m}}{S_{m}^{*}} \Biggr] \\ &+ I_{m}^{*} \Biggl[1 - \frac{E_{m}I_{m}^{*}}{E_{m}^{*}I_{m}} \Biggr] \end{split}$$
(3.53)

Adding the common factors in (3.53), we have

$$\begin{split} \dot{L} &= \frac{\mu_{H} (1 + V_{H} I_{m})}{b \beta_{H}} \bigg[2 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H}}{S_{H}^{*}} \bigg] + \frac{(1 + V_{m} I_{H}) \alpha_{m} E_{m}^{*}}{b \beta_{m} I_{H}^{*}} \bigg[2 - \frac{S_{m}^{*}}{S_{m}} - \frac{S_{m}}{S_{m}^{*}} \bigg] + \frac{I_{m}^{*}}{(1 + V_{m} I_{H})} \bigg[\bigg(2 - \frac{S_{m}^{*}}{S_{m}} - \frac{S_{m} I_{H} E_{m}^{*}}{S_{m}^{*} I_{H}^{*} E_{m}} \bigg) \bigg] \\ &+ I_{m}^{*} \bigg[4 - \frac{S_{H}^{*}}{S_{H}} - \frac{S_{H} I_{m} E_{H}^{*}}{S_{H}^{*} I_{m}^{*} E_{H}} - \frac{E_{H} I_{H}^{*}}{E_{H}^{*} I_{H}} - \frac{E_{m} I_{m}^{*}}{E_{m}^{*} I_{m}} \bigg] \end{split}$$

(3.54)

Collecting all the positives and negatives term of \dot{L} in (3.54) we have

$$\dot{L} = \left\{ \frac{2\mu_{H} \left(1 + V_{H} I_{m} \right)}{b\beta_{H}} + \frac{2I_{m}^{*}}{\left(1 + V_{m} I_{H} \right)} + \frac{2\left(1 + V_{m} I_{H} \right)\alpha_{m} E_{m}^{*}}{b\beta_{m} I_{H}^{*}} + 4 I^{*}{}_{m} \right\}$$

$$\dot{L} = - \left\{ \frac{\mu_{H} \left(1 + V_{H} I_{m} \right)}{b\beta_{H}} \left(\frac{S_{H}^{*}}{S_{H}} + \frac{S_{H}}{S_{H}^{*}} \right) + \frac{2I_{m}^{*}}{\left(1 + V_{m} I_{H} \right)} \left(\frac{S_{m}^{*}}{S_{m}} \right) + \frac{\left(1 + V_{m} I_{H} \right)\alpha_{m} E_{m}^{*}}{b\beta_{m} I_{H}^{*}} \left(\frac{S_{m}^{*}}{S_{m}} + \frac{S_{m}^{*}}{S_{m}^{*}} \right) \right\}$$

$$+ I_{m}^{*} \left(\frac{S_{H}^{*}}{S_{H}} + \frac{S_{H} I_{m} E_{m}^{*}}{S_{H}^{*} I_{m}^{*} E_{m}} + \frac{E_{H} I_{H}^{*}}{E_{H}^{*} I_{H}} + \frac{E_{m} I_{m}^{*}}{E_{m}^{*} I_{m}} \right)$$

 $\Rightarrow L = A - B,$

Where

$$A = \frac{2\mu_H (1 + V_H I_m)}{b\beta_H} + \frac{2I_m^*}{(1 + V_m I_H)} + \frac{2(1 + V_m I_H)\alpha_m E_m^*}{b\beta_m I_H^*} + 4I_m^*$$
(3.55)

And

$$B = \begin{cases} \frac{\mu_{H} \left(1 + V_{H} I_{m}\right)}{b\beta_{H}} \left(\frac{S_{H}^{*}}{S_{H}} + \frac{S_{H}}{S_{H}^{*}}\right) + \frac{2I_{m}^{*}}{\left(1 + V_{m} I_{H}\right)} \left(\frac{S_{m}^{*}}{S_{m}}\right) + \frac{\left(1 + V_{m} I_{H}\right)}{b\beta_{m} I_{H}^{*}} \left(\frac{S_{m}^{*}}{S_{m}} + \frac{S_{m}}{S_{m}^{*}}\right) \\ + I_{m}^{*} \left(\frac{S_{H}^{*}}{S_{H}} + \frac{S_{H}}{S_{H}^{*}} \frac{I_{m}}{I_{m}^{*}} \frac{E_{m}^{*}}{E_{m}} + \frac{E_{H}}{E_{H}^{*}} \frac{I_{H}^{*}}{I_{H}} + \frac{E_{m}}{E_{m}^{*}} \frac{I_{m}^{*}}{I_{m}}\right) \end{cases}$$

(3.56)

From (3.55) and (3.56), we observed that A < B then $\dot{L} = \frac{dL}{dt} < 0$ (which mean that L will be negative definite)

$$\dot{L} = \frac{dL}{dt} = 0$$
 if only if $S_{H}^{*} = S_{H}, E_{H}^{*} = E_{H}, I_{H}^{*} = I_{H}, S_{m}^{*} = S_{m}, E_{m}^{*} = E_{m}$ and $I_{m}^{*} = I_{m}$

Thus the largest compact invariant set $\{(S_H^*, E_H^*, I_H^*, S_m^*, E_m^*, I_m^*) \in \Omega : \frac{dL}{dt} = 0\}$ Is just the endemic equilibrium point Ω . Then by Lassalle invariant principle, if A < B, then Ω will be globally asymptotically stable in Ω . This ends the proof.

Numerical Solution and Discussion

We give some numerical simulation from practical point of view, numerical solutions are very important beside analytical study. We study the numerical behavior of the model (2.1) and model (2.3) with initial conditions (2.2). The numerical simulations are conducted using 2014 MATLAB software and the results are given in figures below.

Parameter	Values	Reference
$b_{\scriptscriptstyle H}$	0.000215	Olaniyi and Obabiyi (2013)
b_m	0.07	Olaniyi and Obabiyi (2013)
b	0.5	Derouich et al (2006)
$eta_{\scriptscriptstyle H}$	0.1	Olaniyi and Obabiyi (2013)
eta_m	0.09	Olaniyi and Obabiyi (2013)
V_{H}	0.2	Olaniyi and Obabiyi (2013)
V_m	0.1	Olaniyi and Obabiyi (2013)
$\alpha_{_H}$	0.3	Garba et. al (2008)

α_{m}	0.17	Assumed
μ _H	0.000046	Hiroshi et. al (2016)
μ_m	0.07	Olaniyi and Obabiyi (2013)
γ_{H}	0.09	Hiroshi et al (2016)
${\cal E}_{H}$	0.00001	Assumed
p	0.0003	Assumed
\overline{q}	0.0005	Assumed
S _H	100	Assumed
E _H	20	Assumed
I _H	10	Assumed
R _H	3	Assumed
S _m	1000	Assumed
E _m	30	Assumed
I _m	20	Assumed

Table 1: Model Parameters and Values Used in Simulation

GRAPHS OF THE SIMULATION OF MODEL AT $R_0 < 1$



Fig. 2: The behavior of all the human and mosquito compartments.

The Fig.2 above shows that the infected human increases from 10 to its peak value within the first 10 days it slightly step down within the second 10 days until it gets to a period of 55 days where it became drastically zero throughout the period. All the compartments reduces to zero except R_H which increases with time (that is there is perfect recovering). Biologically it means that zika virus will completely be eradicated from the population as time goes on.



Fig. 3: The behavior of susceptible human and infectious human for different values of b

Fig.3 shows the varying effect of biting rate (b) of mosquitoes on human population, we can observed that an increase in biting rate leads to increase in infectious class while decrease in biting rate leads to decrease in infectious class. Biologically it means that the biting rate of mosquito in our environment has a high level of effect on spread of zika virus on human population.



Fig.4: The behavior of susceptible and infectious human for different values of V_H

Fig.4 shows the varying effects of antibody on human population on human population. We observed that susceptible human (S_H) population drops as a result of infection by infectious mosquito and later stabilized when the human developed antibody (V_H) against the parasite causing zika virus. The magnitude of the infectious human population decreases with increased presence of antibody. Biologically it means that building a strong antibody system reduces the rate of infection on human population.



Fig.5: The behavior of susceptible and infectious mosquito for different values of V_m

Fig.5 similarly shows the effects of antibody on mosquito population. We observed that susceptible mosquito (S_m) population drops as a result of infection by infectious human. The infectious mosquito population decreases with an increase presence of antibody produced by mosquitoes.



Fig.6: The behavior of susceptible and infectious human for different values of μ_m

Fig.6 shows the variation of natural death rate of mosquito. As we reduces the number of death rate of mosquito (that is, using insecticides or any other means), we observed that the level of infectious human reduces.

GRAPH OF THE SIMULATION OF MODEL AT $R_0|_{p=q=0} < 1$



Fig.7: The behavior of all the human and mosquito compartments.

Fig.7 shows that at the initial time there will be little or no infected human but within the first 10 days, the infected human (I_H) increased to its peak level and gradually steps down to zero as time goes on. While R_H shows a perfect recovery. Comparing fig.7 and fig.2 we observed that in fig.2, the zika virus was eradicated from the population within a short period while in fig.7 the zika virus was eradicated in shorter time. This implies that in a population of two transmission the zika virus will take longer time to eradicated than that of one transmission

Summary, Conclusion and Recommendation

Summary

This thesis presents a deterministic model for the transmission dynamics of zika virus with vertical transmission involving seven compartments made up of two different populations (Humans and Mosquitoes). The human population has four compartments which are: susceptible, Exposed, Infected and Recovered compartments. $S_H \rightarrow E_H \rightarrow I_H \rightarrow R_H$. The Mosquito compartments are: Susceptible, Exposed and Infected compartments. $S_m \rightarrow E_m \rightarrow I_m$. We extended the model by taking into account of exposed individuals, nonlinear forces of infection (saturated incidence rates) and vertical transmission. We showed that both human and mosquito develop antibodies through saturated incidence rates. We established a region where the model is epidemiologically feasible and mathematically well-posed (which is the invariant region) and also the positivity of the solution of the model. We showed the existence of disease-free equilibrium (DFE) and endemic equilibrium (EE) points. We also obtained the reproduction number of the model and reproduction number of special case model where we set p=q=0 using the next generation matrix technique. We further developed lyapunov functions to prove the global stability of both disease-free equilibrium (DFE) and endemic equilibrium (EE) when $R_0|_{p=q=0}$ is less than unity and greater than unity respectively.

RESULTS OF SIMULATIONS

The simulation results on the first model show that the varying effects of biting rate b of mosquitoes on human population has a higher effect in zika virus transmission. It can be seen from the graph that the higher the biting rate of mosquito, the higher the zika virus transmission. Also, the more we increase the antibody, the lower the zika virus transmission. Biologically, it means that people need to protect themselves from the bite of mosquitoes so as to reduce the rate of transmission .We also observed that depopulating mosquito leads to decrease in infected population.

The behavior of the simulation under horizontal transmission {that is model (3.9)} for $R_0|_{n=q=0} < 1$

show that at the initial time, there where little or no infected humans but within the first 10 days, the infected humans (I_H) increased to its peak level and gradually steps down to zero as time goes on.

We also finds out that model (3.7) have a higher level of infection because of the recruitment of vertical transmission while model (3.9) has little higher infection. We observed that in Figure 4.1 the zika virus was eradicated from the population within a period of 55 days while that of Figure 4.6 was eradicated within a period of 35 days. This means that the zika virus can easily be controlled in a shorter time in model (3.9) than in model (3.7).

CONCLUSION

- (i) The disease-free equilibrium ε_0 , is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$: The implication of the above result is that the zika virus can be eradicated from the population if there be a bound on the rate of transmission between mosquito and human.
- (ii) The disease-free equilibrium of the model is globally asymptotically stable when $R_0|_{p=q=o} < 1$, with no loss of immunity acquired by the recovered individuals. This shows that the zika-free population is possible.
- (iii)The model has a globally asymptotically stable endemic equilibrium if the threshold parameter, $R_0|_{p=q=0}$, is greater than unity.
- (iv) The numerical simulations were performed to see the effects of the proportions of antibodies

produced by both populations and other key parameters on the spread of the zika virus.

(v) It is also important to note that reducing human-mosquito contact rate plays a big role in inhibiting the prevalence of Zika virus.

RECOMMENDATIONS

- 1. Eating right food and taking plenty of fluid like water can help to boost the level of antibodies in human because humans needs to boost their antibodies production to be able to resist the presence of zika-parasites in the blood stream.
- 2. We can achieve a zika-free state by scaling down mosquito biting rate through the use of insecticides, closing of doors and windows against mosquitoes.
- 3. The government, non-governmental organizations and stakeholders should help in creating awareness because prevention is better than cure.

CONTRIBUTIONS TO KNOWLEDGE

The following are our contributions to knowledge

1. We introduced vertical transmission into the existing model and extending the model from five compartments to seven compartments by adding exposed classes.

2. We have been able to construct linear and nonlinear Lyapunov function to prove the global stability analysis of both DFE and EEP using Zika model.

AREAS OF FURTHER STUDIES

Further research may include investigation of applying stochastic models like Continuous Time Markov Chain as well as Discrete Time Markov Chain Models to verify the effectiveness of the SDE on Zika model. They can also look into Sensitivity Analysis of Zika Virus. In future work also, we may look at the vaccine for Zika Virus and the control measures.

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