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Endemic Equilibrium and Forward Bifurcation in the Mathematical Model for Using Wolbachia to Control Spread of Zika Virus Disease

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Abstract

This paper presents a mathematical model for transmission dynamics of Zika virus disease when Wolbachia is used as bio-control. Zika virus disease is an arboviral disease that spreads through bites of female mosquitoes in the Aedes family especially, Aedes aegypti. Studies have shown that Wolbachia, when present in Aedes aegypt, can prevent mosquitoes from transmitting the Zika virus to humans. A system of nonlinear ordinary differential equations is used to model this bio-control technique, and a bifurcation analysis of the model is conducted. The result shows that the model exhibits forward bifurcation, which confirms the existence of a unique endemic equilibrium in the model when $\mathcal{R}_c > 1$. The existence of forward bifurcation in the model indicates that having $\mathcal{R}_c < 1$ is enough to guarantee the eradication of Zika virus disease using Wolbachia as a bio-control irrespective of initial sizes of infected human and mosquito populations. Plots are provided to show the effect of Wolbachia on the Aedes aegypti population.

Keywords: Zika virus disease, aedes aegypti, Wolbachia, forward bifurcation.
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1. Introduction

Zika virus disease is a flavivirus disease transmitted to humans mainly through bites of infected female aedes agypti mosquitoes [1, 2]. Other ways through which the virus could be transmitted to humans include unprotected sex, unscreened blood transfusion and from mother to her unborn child [3, 4, 5, 6]. Even though Zika virus disease has negligible mortality rate, its 'microcephalic' effect on children born to mothers who are exposed to the virus in their pregnancy cannot be overemphasized [7, 8]. The outbreak of Zika in Brazil produced over 5000 confirmed cases of microcephaly. Apart from microcephaly, the disease has been linked to neurological disorder, Guillain-Barre Syndrome(GBS)[9]. In 2015, the World Health Organization (WHO) declared Zika virus disease, a Public Health Emergency of International Concern because of these health conditions associated with the disease[10].

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Since Zika virus disease has no cure and vaccine, a suggested way to combat the spread of the disease is the use of aedes aegypti mosquitoes infected with Wolbachia as a bio-control agent[11]. Wolbachia is a group of bacteria which naturally inhabits in reproductive tissues of most arthropods. They are transmitted maternally through the cytoplasm of eggs of their host insects[12, 13]. It has been shown experimentally that Wolbachia reduces the ability of host mosquitoes to transmit disease-causing pathogens to humans through bites[14]. This it does by increasing the incubation period(or reducing the incubation rate) of the virus in its host mosquito. This thereby increases the time it takes the mosquito carrying Zika virus to become infectious. Since the adult life of the mosquito is short, most of them carrying the virus die before they complete the incubation period. Therefore, the infected Wolbachia-carrier mosquitoes may not transmit the virus to humans through their bites before they die. Apart from incubation rate reduction, Wolbachia induces cytoplasmic incompatibility(CI), a complex biochemical phenomenon, which helps its host mosquito to invade and eliminate the Wolbachia-free aedes aegypti mosquitoes in the wild [16]. The effect of cytoplasmic incompatibility is non-hatching of eggs when male aedes aegypti with Wolbachia mates with their female counterparts without Wolbachia. The resultant effect of CI is that population of wild aedes aegypti continues to decrease until not many mosquitoes will be available to spread the virus. It should be noted that since aedes aegypti is a natural host to Wolbachia, the mosquitoes have to be manually infected with the bacteria in the laboratory, and then released to mate with the wild ones [15].

In infectious disease modeling, the basic reproductive number, generally denoted as $\mathcal{R}_0$, is one of the most important quantities sought by researchers. It is defined as the average number of people that are infected by a single infected person introduced in a purely susceptible population. The basic reproduction number gives a key measure of a disease's ability to spread in a given population or die out gradually. Specifically, if $\mathcal{R}_0 < 1$, it means that on average, less than one person will be infected by a single infected person introduced in a purely susceptible community. Hence, the infection does not replicate and the disease-free equilibrium is stable. On the other hand, $\mathcal{R}_0 > 1$ indicates that more that one person will be infected on on average. Hence, the infection replicates leading to larger numbers of infections and an unstable disease-free equilibrium. However, in some cases, having $\mathcal{R}_0 < 1$ is not good enough to guarantee eradication of disease in a given population. This happens when backward bifurcation takes place in the model. In backward bifurcation, when $\mathrm{R}_0 < 1$, a small positive unstable endemic equilibrium appears, together with disease-free equilibrium and a larger endemic equilibrium that is locally asymptotically stable. This means that backward bifurcation involves at least two endemic equilibria; a small one that is unstable and a larger one that is stable. This implies that the condition $\mathcal{R}_0 < 1$, is no longer a necessary and sufficient condition for disease eradication in a population. On the other hand, in forward bifurcation, when $\mathcal{R}_0$ crosses the unity from below, a small positive asymptotically stable endemic equilibrium appears and the disease-free equilibrium looses its stability[17]. Hence, the endemic equilibrium only exists when $\mathcal{R}_0 > 1$, and there is no possibility of an endemic equilibrium when $\mathcal{R}_0 < 1$. In this case, bringing the basic reproduction number to below unity is a necessary and sufficient condition for disease eradication in the given population, irrespective of the initial number of infectious populations. The purpose of this paper is to model the use of

Wolbachia in the control of Zika virus disease, and to show that forward bifurcation takes place in the model, which implies the existence of a unique endemic equilibrium which is locally asymptotically stable when $\mathcal{R}_0 > 1$. The remaining part of this paper is organized as follows: in section 2,the proposed model for transmission dynamics and control of Zika virus disease is presented. In section 3, the analysis of the model which include derivation of the control reproduction number, stability analysis of the equilibrium points and bifurcation analysis are shown. Simulation of the model, with discussion of result is presented in section 4. The work is concluded in section 5.

2. THE MATHEMATICAL MODEL

In this section, the system of nonlinear ordinary differential equations which models the dynamics of Zika virus disease in the human and aedes aegypti mosquito populations, including the Wolbachia-infected aedes aegypti used for bio-control is derived. The major assumption in the formulation of this model is that humans may become infected with Zika virus through bites of infectious female Aedes *aegypti* mosquito. Uninfected pregnant female Aedes *aegypti* mosquitoes may acquire Zika virus when they bite infectious human to suck blood. Therefore, the model comprises three populations; human population, population of adult female aedes *aegypti* that are infected with Wolbachia, and population of adult female aedes *aegypti* that are not infected with Wolbachia.

2.1. Zika Dynamics in Human Population

In the human population, we have four(4) compartments; humans that are susceptible to Zika virus disease,$S_h(t)$, Humans that are exposed to Zika virus disease, $E_h(t)$, humans that are infectious with Zika virus disease, $I_h(t)$, and humans that have recovered from Zika virus disease infection, $R_h(t)$. The total population of humans, $T_h(t)$ at any time t is therefore given by $T_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$. We assume that individuals are recruited into the susceptible class in the Zika endemic area at the rate, π_h. The susceptible class of humans may become exposed to Zika virus through bites of infectious Wolbachia-free mosquito bites at the rate b_1, with transmission probability, α_{mh}. The susceptible humans may also become infected with the virus through the bites of infected Wolbachia-carrier mosquitoes at the rate b_2, with a very negligible probability of infection, $\alpha_{mwh} \ll \alpha_{mh}$. At the end of the incubation period, the exposed class becomes infectious at the incubation rate β_h, but recovers at the rate γ. There is a natural death rate σ_h, for all the humans in the population under study, and an additional Zika-induced death rate σ'_h, only for the infectious class of humans.

2.2. Dynamics of Zika virus Disease in the Aedes aegypti Population

In the Wolbachia-free adult female aedes *aegypti* mosquitoes, we have three compartments namely; (i)mosquitoes that are susceptible to Zika virus, $S_m(t)$, (ii) mosquitoes that are exposed Zika virus diseases, $E_m(t)$ and (iii)mosquitoes that are infectious with Zika virus disease, $I_m(t)$. The female Wolbachia-free mosquitoes join the susceptible class through migration at the rate π_m, or through oviposition at the rate μ_m, with κ being the proportion of the eggs that are female. The parameter $q \in (0,1)$ is introduced to represent the effect of cytoplasmic incompatibility. In this regard, we assume that the

proportion q, of the eggs produced by the female Wolbachia-free mosquitoes are viable, while $(1-q)$ are non-viable. This parameter is responsible for the reduction in the population of the wild mosquitoes. Susceptible mosquitoes becomes exposed to Zika virus when they bite humans in the infectious class at the biting rate b_1, with probability of infection α_{hm}. At the expiation of the incubation period, the exposed mosquitoes become infectious at the incubation rate, β_m. The mosquitoes remain infectious throughout their lifetime until they die naturally at the rate, σ_m. Similarly, the female adult Wolbachia-carrier aedes *aegypti* mosquitoes are grouped in the same manner, with the following compartments, the susceptible Wolbachia-carrier class; $S_{mw}(t)$, the exposed Wolbachia-carrier class; $E_{mw}(t)$, and the infectious Wolbachia-carrier class; $I_{mw}(t)$. The dynamics of Zika virus disease in the Wolbachia-carrier mosquito population is similar to that of the Wolbachia-free mosquitoes, except at the infectious stage where the probability of the Wolbachia-carrier mosquitoes to transmit the virus to the susceptible humans is negligible. We also mention that the susceptible Wolbachia-infected mosquitoes are introduced from the laboratory into the Zika-endemic area in the constant rate π_{mw}. Using the above assumptions on the transmission dynamics and control of Zika, we obtain the following system of nonlinear ordinary differential equations which models the dynamics of the disease.

$$
\begin{aligned}
\frac{dS_h(t)}{dt} &= \pi_h - b_1\alpha_{mh}S_h(t)I_m(t) - b_2\alpha_{mwh}I_{mw}(t)S_h(t) - \sigma_h S_h(t),\\
\frac{dE_h(t)}{dt} &= b_1\alpha_{mh}I_m(t)S_h(t) + b_2\alpha_{mwh}I_{mw}(t)S_h(t) - (\beta_h+\sigma_h)E_h(t),\\
\frac{dI_h(t)}{dt} &= \beta_h E_h(t) - (\gamma+\sigma_h+\sigma_h')I_h(t),\\
\frac{dR_h(t)}{dt} &= \gamma I_h(t) - \sigma_h R_h(t),\\
\frac{dS_m(t)}{dt} &= \pi_m + q\kappa\mu_m T_m(t) - b_1\alpha_{hm}S_m(t)I_h - \sigma_m S_m(t),\\
\frac{dE_m(t)}{dt} &= b_1\alpha_{hm}S_m(t)I_h(t) - (\beta_m+\sigma_m)E_m(t),\\
\frac{dI_m(t)}{dt} &= \beta_m E_m(t) - \sigma_m I_m(t),\\
\frac{dS_{mw}(t)}{dt} &= \pi_{mw} + \kappa\mu_{mw}T_{mw}(t) - b_2\alpha_{hmw}S_{mw}(t)I_h(t) - \sigma_m S_{mw}(t),\\
\frac{dE_{mw}(t)}{dt} &= b_2\alpha_{hmw}S_{mw}(t)I_h(t) - (\beta_{mw}+\sigma_m)E_{mw}(t),\\
\frac{dI_{mw}(t)}{dt} &= \beta_{mw}E_{mw}(t) - \sigma_m I_{mw}(t),
\end{aligned}
\tag{2.1}
$$

with the initial solutions given by $X_0 = (S_h^0, E_h^0, {}^0_h, R_h^0, S_m^0, E_m^0, I_m^0, S_{mw}^0, E_{mw}^0, I_{mw}^0)$. Since human and mosqutio populations cannot be negative, we can conclude that the initial solutions are all nonnegative numbers. From (2.1), it can be seen that the total populations of human, Wolbachia-free and Wolbachia-carrier mosquitoes, respectively, satisfy the fol-

lowing system of equations

$$\begin{aligned}\frac{dT_h}{dt} &= \pi_h - \sigma_h T_h - \sigma_h' I_h,\\ \frac{dT_m}{dt} &= \pi_m + (q\kappa\mu_m - \sigma_m)\, T_m,\\ \frac{dT_{mw}}{dt} &= \pi_{mw} + (\kappa\mu_{mw} - \sigma_m)\, T_{mw}.\end{aligned} \tag{2.2}$$

The domain of existence of the solution to this system can be described as $\mathcal{D} = D_1 \cup D_2 \cup D_3$, where $D_1 = \{S_h, E_h, I_h, R_h) \in \mathbb{R}^4_+ | S_h + E_h + I_h + R_h \leqslant T_h\}$,
$D_2 = \{(S_m, E_m, I_m) \in \mathbb{R}^3_+ | S_m + E_m + I_m \leqslant T_m\}$,
and $D_3 = \{(S_{mw}, E_{mw}, I_{mw}) \in \mathbb{R}^3_+ | S_{mw} + E_{mw} + I_{mw} \leqslant T_{mw}\}$.

Theorem 2.1. *The model (2.1) possesses a unique positive solution $\forall t > 0$, which passes through the initial solution $X(0)$, and the set $\mathcal{D}$ is positive invariant.*

Proof: Each right-hand side of the model system has continuous partial derivatives with respect to each state variable. Hence, the system possesses a unique solution $X(t)$ which passes through the initial solution $X(0)$. To show that the solution remains nonnegative, we have, using differential inequality on (2.1), that

$$S_h(t) > S_h^0 e^{-\int^t (b_1\alpha_{mh} I_m(s) + b_2\alpha_{mwh}(s) I_{mw}(s) + \sigma_h) ds} > 0,$$

$E_h(t) > E_h^0 e^{-(\beta_h + \sigma_h)t} > 0$, $I_h(t) > I_h^0 e^{-(\gamma + \sigma_h + \sigma_h')t} > 0$, $R_h(t) > R_h^0 e^{-\sigma_h t} > 0$. Similar methods can be applied to prove that other components of the system have positive solutions in the octant $\mathcal{D}$. To show that $\mathcal{D}$ is positive invariant with respect to the solution of the model system, we have from (2.2), that the total populations satisfy the following inequalities

$$\begin{aligned}0 < \lim_{t\to\infty} \sup T_h(t) &\leqslant \frac{\pi_h}{\sigma_h}\\ 0 < \lim_{t\to\infty} \sup T_m(t) &\leqslant \frac{\pi_m}{\sigma_m - qk\mu_m}\\ 0 < \lim_{t\to\infty} \sup T_{mw}(t) &\leqslant \frac{\pi_{mw}}{\sigma_m - k\mu_{mw}}\end{aligned}$$

The inequalities show the boundedness of total human and mosquito populations. Since no population can be negative, it implies that all solutions enter and remain in the domain $\mathcal{D}$ $\forall t \geqslant 0$. This confirms positive invariance of $\mathcal{D}$.

3. Analysis of the Model

3.1. Disease-free Equilibrium and the Control Reproduction Number

It is easy to show that the steady state of (1) in the absence of Zika virus disease infection is $E_0 = \left(\frac{\pi_h}{\sigma_h}, 0, 0, 0, \frac{\pi_m}{\sigma_m - qk\mu_m}, 0, 0, \frac{\pi_{mw}}{\sigma_M - k\mu_{mw}}, 0, 0\right)$. Also, at disease-free equilibrium, $(T_h^0, T_m^0, T_{mw}^0) = \left(\frac{\pi_h}{\sigma_h}, \frac{\pi_m}{\sigma_m - qk\mu_m}, \frac{\pi_{mw}}{\sigma_m - k\mu_{mw}}\right)$.

The control reproduction number, $\mathcal{R}_c$ of a disease gives on average, the number of people that can be infected by a single index case of the disease when introduced in a disease-free population with a control measure in place[18]. This number gives an indication of effectiveness of a control measure. The control measure is effective if $\mathcal{R}_c < 1$, otherwise it is not effective. The next generation matrix(NGM) approach by Diekmann et al.[19] is applied to determine the reproduction number, which gives $\mathcal{R}_c$ as the spectral radius of a next generation matrix.

Theorem 3.1. *The NGM of the model is of the form*

$$\mathcal{K} = \begin{pmatrix} 0 & \mathcal{K}_{mh} & \mathcal{K}_{mwh} \\ \mathcal{K}_{hm} & 0 & 0 \\ \mathcal{K}_{hmw} & 0 & 0 \end{pmatrix} \tag{3.1}$$

and the control reproduction number is

$$\mathcal{R}_c = \sqrt{\mathcal{K}_{mh}\mathcal{K}_{hm} + \mathcal{K}_{mwh}\mathcal{K}_{hmw}} \tag{3.2}$$

where, $\mathcal{K}_{hm} = \frac{\beta_h b_1 \alpha_{hm} \pi_m}{(\sigma_m - q\kappa\mu_m)(\beta_h + \sigma_h)(\gamma + \sigma_h + \sigma'_h)}$*, is the number of Wolbachia-free mosquitoes that can be infected by one infectious human*
$\mathcal{K}_{hmw} = \frac{\beta_h b_2 \alpha_{hmw} \pi_{mw}}{(\sigma_m - k\mu_{mw})(\beta_h + \sigma_h)(\gamma + \sigma_h + \sigma'_h)}$*, is the number of Wolbachia-carrier mosquitoes that can be infected by one infectious human*
$\mathcal{K}_{mh} = \frac{\beta_m b_1 \alpha_{mh} \pi_h}{\sigma_h \sigma_m (\beta_m + \sigma_m)}$*, is the number of humans that can be infected by one infectious Wolbachia-free mosquitoes*
$\mathcal{K}_{mwh} = \frac{\beta_{mw} b_2 \alpha_{mwh} \pi_h}{\sigma_h \sigma_m (\beta_{mw} + \sigma_m)}$*,is the number of humans that can be infected by one infectious Wolbachia-carrier mosquitoes.*

Proof: The size(3×3) of the next generation matrix (3) corresponds to the 3 populations involved in the dynamics of the disease. Also, the position elements of the matrix is determined by the assumptions that humans can transmit Zika virus to Wolbachia-free and Wolbachia-carrier mosquitoes, and vice versa. The zero entries in the matrix indicate absence of disease transmission. For example, the zeros in the diagonal is an indication of no vertical and horizontal transmission of Zika virus disease in the populations. Moreover, by definition, the NGM is given by $\mathcal{K} = FV^{-1}$, where

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{b_1 \alpha_{mh} \pi_h}{\sigma_h} & 0 & \frac{b_2 \alpha_{mwh} \pi_h}{\sigma_h} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{b_1 \alpha_{hm} \pi_m}{\sigma_m - q\kappa\mu_m} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{b_2 \alpha_{hmw} \pi_{mw}}{\sigma_m - \kappa\mu_{mw}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \beta_h + \sigma_h & 0 & 0 & 0 & 0 & 0 \\ -\beta_h & \gamma + \sigma_h + \sigma'_h & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_m + \sigma_m & 0 & 0 & 0 \\ 0 & 0 & -\beta_M & \sigma_m & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_{mw} + \sigma_m & 0 \\ 0 & 0 & 0 & 0 & -\beta_{mw} & \sigma_m \end{pmatrix}$$

Then, the next generation matrix becomes

$$\mathcal{K} = \begin{pmatrix} 0 & 0 & \frac{\beta_m b_1 \alpha_{mh} \pi_h}{\sigma_h \sigma_m (\beta_m + \sigma_m)} & \frac{b_1 \alpha_{mh} \pi_h}{\sigma_h \sigma_m} & \frac{\beta_{mw} b_2 \alpha_{mwh} \pi_h}{\sigma_h \sigma_m (\beta_{mw} + \sigma_m)} & \frac{b_2 \alpha_{mwh} \pi_h}{\sigma_h \sigma_m} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_h b_1 \alpha_{hm} \pi_m}{\varkappa_1} & \frac{b_1 \alpha_{hm} \pi_m}{\varkappa_2} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_h b_2 \alpha_{hmw} \pi_{mw}}{\varkappa_3} & \frac{b_2 \alpha_{hmw} \pi_{mw}}{\varkappa_4} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{3.3}$$

where

$$\begin{aligned} \varkappa_1 &: = (\sigma_m - q\kappa\mu_m)(\beta_h + \sigma_h)(\gamma + \sigma_h + \sigma_h') \\ \varkappa_2 &: = (\sigma_m - q\kappa\mu_m)(\gamma + \sigma_h + \sigma_h)' \\ \varkappa_3 &: = (\sigma_m - \kappa\mu_m)(\beta_h + \sigma_h)(\gamma + \sigma_h + \sigma_h') \\ \varkappa_4 &: = (\sigma_m - \kappa\mu_m)(\gamma + \sigma_h + \sigma_h'). \end{aligned}$$

We can recover (3.1) from (3.3) by the multiplication of the form, $A\mathcal{K}A^T$, where A is the auxiliary matrix

$$A = \begin{pmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 \end{pmatrix}^T$$

to get

$$\mathcal{K}_s = \begin{pmatrix} 0 & \frac{\beta_m b_1 \alpha_{mh} \pi_h}{\sigma_h \sigma_m (\beta_m + \sigma_m)} & \frac{\beta_{mw} b_2 \alpha_{mwh} \pi_h}{\sigma_h \sigma_m (\beta_{mw} + \sigma_m)} \\ \frac{\beta_h b_1 \alpha_{hm} \pi_m}{\varkappa_1} & 0 & 0 \\ \frac{\beta_h b_2 \alpha_{hmw} \pi_{mw}}{\varkappa_3} & 0 & 0 \end{pmatrix}.$$

Hence, (3.2) follows from the definition, $\mathcal{R}_c := \rho(\mathcal{K}_s)$.□
In the absence of this control, the reproduction number becomes $\mathcal{R}_0 = \sqrt{\mathcal{K}_{mh}\mathcal{K}_{hm}}$, which is the basic reproduction number of the disease. Therefore, for this control method to be considered effective, we must have $\mathcal{R}_c < \mathcal{R}_0$.

3.2. Stability Analysis of the Equilibrium Points

3.2.1. Local Asymptotic Stability of the Disease-Free Equilibrium

The linearization of the model system(2.1) around the equilibrium point, E_0 gives the Jacobian matrix;

$$\mathcal{J}(E_0) = \begin{pmatrix} -\mathcal{J}_{11} & 0 & 0 & 0 & 0 & 0 & -\mathcal{J}_{12} & 0 & 0 & -\mathcal{J}_{13} \\ 0 & -\mathcal{J}_{21} & 0 & 0 & 0 & 0 & \mathcal{J}_{22} & 0 & 0 & \mathcal{J}_{23} \\ 0 & \mathcal{J}_{31} & -\mathcal{J}_{32} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mathcal{J}_{41} & -\mathcal{J}_{42} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mathcal{J}_{51} & 0 & -\mathcal{J}_{52} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mathcal{J}_{61} & 0 & 0 & -\mathcal{J}_{62} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \mathcal{J}_{71} & -\mathcal{J}_{72} & 0 & 0 & 0 \\ 0 & 0 & -\mathcal{J}_{81} & 0 & 0 & 0 & 0 & -\mathcal{J}_{82} & 0 & 0 \\ 0 & 0 & \mathcal{J}_{91} & 0 & 0 & 0 & 0 & 0 & -\mathcal{J}_{92} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mathcal{J}_{101} & -\mathcal{J}_{102} \end{pmatrix} \tag{3.4}$$

where, $\mathcal{J}_{11} = \sigma_h, \mathcal{J}_{12} = \frac{b_1\alpha_{mh}\pi_h}{\sigma_h}$, $\mathcal{J}_{13} = \frac{b_2\alpha_{mwh}\pi_h}{\sigma_h}, \mathcal{J}_{21} = (\beta_h + \sigma_h)$, $\mathcal{J}_{22}\frac{b_1\alpha_{mh}\pi_h}{\sigma_h}$, $\mathcal{J}_{23} = \frac{b_2\alpha_{mwh}\pi_h}{\sigma_h}, \mathcal{J}_{21} = (\beta_h + \sigma_h)$, $\mathcal{J}_{31} = \beta_h$, $\mathcal{J}_{32} = (\gamma + \sigma_h + \sigma_h')$, $\mathcal{J}_{41} = \gamma$, $\mathcal{J}_{42} = \sigma_h$, $\mathcal{J}_{51} = \frac{b_1\alpha_{hm}\pi_m}{\sigma_m - qk\mu_m}$,
$\mathcal{J}_{52} = (\sigma_m - qk\mu_m)$, $\mathcal{J}_{61} = \frac{b_1\alpha_{hm}\pi_m}{\sigma_m - qk\mu_m}, \mathcal{J}_{62} = (\beta_m + \sigma_m)$, $\mathcal{J}_{71} = \beta_m$, $\mathcal{J}_{72} = \sigma_m$,
$\mathcal{J}_{81} = \frac{b_2\alpha_{hmm}\pi_{mw}}{\sigma_m - k\mu_{mw}}, \mathcal{J}_{82} = (\sigma_m - k\mu_{mw})$, $\mathcal{J}_{91} = \frac{b_2\alpha_{hmm}\pi_{mw}}{\sigma_m - k\mu_{mw}}, \mathcal{J}_{92} = (\beta_{mw} + \sigma_m)$,
$\mathcal{J}_{101} = \beta_{mw}$, $\mathcal{J}_{102} = \sigma_m$. The matrix $\mathcal{J}(E_0)$ can be reduced to 6×6 submatrix by observing that
$-\sigma_h, -\sigma_h, -(\sigma_m - qk\mu_m), -(\sigma_m - k\mu_{mw})$ are some of its eigenvalues. Let

$$\mathcal{J}'(E_0) = \begin{pmatrix} -(\beta_h + \sigma_h) & 0 & 0 & \frac{b_1\alpha_{mh}\pi_h}{\sigma_h} & 0 & \frac{b_2\alpha_{mwh}\pi_h}{\sigma_h} \\ \beta_h & -(\gamma + \sigma_h + \sigma_h') & 0 & 0 & 0 & 0 \\ 0 & \frac{b_1\alpha_{hm}\pi_m}{\sigma_m - qk\mu_m} & -(\beta_m + \sigma_m) & 0 & 0 & 0 \\ 0 & 0 & \beta_m & -\sigma_m & 0 & 0 \\ 0 & \frac{b_2\alpha_{hmm}\pi_{mw}}{\sigma_m - k\mu_{mw}} & 0 & 0 & -(\beta_{mw} + \sigma_m) & 0 \\ 0 & 0 & 0 & 0 & \beta_{mw} & -\sigma_m \end{pmatrix}.$$

Note that the matrices F, V and the sub-matrix, $\mathcal{J}'(E_0)$ are such that $F - V = J'(E_0)$. According to Driessche and Watmough[20], the eigenvalues of $F - V$ or equivalently, $J'(E_0)$ are all negative or have negative real parts if $\rho(FV^{-1}) < 1$. This shows that the disease-free equilibrium E_0 is locally asymptotically stable if $\mathcal{R}_c < 1$, and unstable if $\mathcal{R}_c > 1$. This implies that Zika virus disease can be eradicated when $\mathcal{R}_c < 1$, by using this control measure, if the initial size of the compartments are in the basin of attraction of the disease-free equilibrium.

3.2.2. **Existence of Endemic Equilibrium and its Local Stability Analysis**

Let $E_1 = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*, S_{mw}^*, E_{mw}^*, I_{mw}^*)$ where
$S_h^* = \frac{\pi_h}{b_1\alpha_{mh}I_m^* + b_2\alpha_{mwh}I_m^* + \sigma_h}$, $E_h^* = \frac{b_1\alpha_{mh}I_m^* + b_2\alpha_{mwh}I_m^*}{\beta_h + \sigma_h}$, $I_h^* = \frac{\beta_h E_h^*}{\gamma + \sigma_h + \sigma_h'}, R_h^* = \frac{\gamma I_h^*}{\sigma_h}$, $S_m^* = \frac{\pi_m + qk\mu_m T_m^*}{b_1\alpha_{hm}I_h^* + \sigma_m}$,

$E_m^* = \frac{b_1\alpha_{hm}S_m^*I_h^*}{(\beta_m+\sigma_m)}$, $I_m^* = \frac{b_1\beta_m\alpha_{hm}(\pi_m+qk\mu_mT_m^*)I_h^*}{\sigma_m(\beta_m+\sigma_m)(b_1\alpha_{hm}I_h^*+\sigma_m)}$, $S_{mw}^* = \frac{\pi_{mw}+qk\mu_{mw}T_{mw}^*}{b_1\alpha_{hmw}I_h^*+\sigma_m}$,
$E_{mw}^* = \frac{b_2\alpha_{hmw}S_{mw}^*I_h^*}{(\beta_{mw}+\sigma_m)}$, $I_{mw}^* = \frac{b_2\beta_{mw}\alpha_{hmw}(\pi_{mw}+k\mu_{mw}T_{mw}^*)I_h^*}{\sigma_m(\beta_{mw}+\sigma_m)(b_2\alpha_{hmw}I_h^*+\sigma_m)}$, $T_m^* = \frac{\pi_m}{\sigma_m-qk\mu_m}$, $T_{mw}^* = \frac{\pi_{mw}}{\sigma_m-k\mu_{mw}}$,
$T_h^* = \frac{\pi_h-\sigma_h'I_h^*}{\sigma_h}$, be the solution to the following equations

$$\pi_h - b_1\alpha_{mh}S_hI_m - b_2\alpha_{mwh}I_{mw}S_h - \sigma_hS_h = 0, \tag{3.5}$$
$$b_1\alpha_{mh}I_mS_h + b_2\alpha_{mwh}I_{mw}S_h - (\beta_h+\sigma_h)E_h = 0, \tag{3.6}$$
$$\beta_hE_h - (\gamma+\sigma_h+\sigma_h')I_h = 0, \tag{3.7}$$
$$\gamma I_h - \sigma_hR_h = 0, \tag{3.8}$$
$$\pi_m + q\kappa\mu_mT_m - b_1\alpha_{hm}S_m(t)I_h - \sigma_mS_m(t) = 0, \tag{3.9}$$
$$b_1\alpha_{hm}S_m(t)I_h - (\beta_m+\sigma_m)E_m(t) = 0, \tag{3.10}$$
$$\beta_mE_m(t) - \sigma_mI_m(t) = 0, \tag{3.11}$$
$$\pi_{mw} + \kappa\mu_{mw}T_{mw} - b_2\alpha_{hmw}S_{mw}(t)I_h - \sigma_mS_{mw}(t) = 0, \tag{3.12}$$
$$b_2\alpha_{hmw}S_{mw}(t)I_h - (\beta_{mw}+\sigma_m)E_{mw}(t) = 0, \tag{3.13}$$
$$\beta_{mw}E_{mw}(t) - \sigma_mI_{mw}(t) = 0, \tag{3.14}$$

Let I_m^* and I_{mw}^* be written as $I_m^* = \frac{A_1I_h^*}{A_2I_h^*+A_3}$ and $I_{mw}^* = \frac{B_1I_h^*}{B_2I_h^*+B_3}$, respectively,
where, $A_1 = b_1\beta_m\alpha_{hm}(\pi_m+qk\mu_mT_m^*)$, $A_2 = \sigma_m(\beta_m+\sigma_m)b_1\alpha_{hm}$, $A_3 = \sigma_m^2(\beta_m+\sigma_m)$
$B_1 = b_2\beta_{mw}\alpha_{hmw}(\pi_{mw}+k\mu_{mw}T_{mw}^*)$, $B_2 = \sigma_m(\beta_{mw}+\sigma_m)b_2\alpha_{hmw}$, $B_3 = \sigma_m^2(\beta_Mw+\sigma_m)$
Substituting E_h^* into (3.7) and simplifying, gives

$$\frac{\beta_h\pi_h}{\beta_h+\sigma_h}\frac{G_1I_h^{*2}+G_2I_h^*}{G_3I_h^{*2}+G_4I_h^*+G_5} - (\gamma+\sigma_h+\sigma_h')I_h^* = 0, \tag{3.15}$$

where, $G_1 = b_1\alpha_{mh}A_1B_2 + b_2\alpha_{mwh}B_1A_2$, $G_2 = b_1\alpha_{mh}A_1B_3 + b_2\alpha_{mwh}B_1A_3$,
$G_3 = G_1 + \sigma_hA_2B_2$, $G_4 = G_2 + \sigma_h(A_2B_3+A_3B_2)$, $G_5 = \sigma_hA_3B_3$
Rearranging (3.15) gives the cubic equation

$$-C_1I_h^{*3} + C_2I_h^{*2} + C_3I_h^* = 0, \tag{3.16}$$

where, $C_1 = (\beta_h+\sigma_h)(\gamma+\sigma_h+\sigma_h')G_3$, $C_2 = (\beta_h\pi_hG_1 - (\beta_h+\sigma_h)(\gamma+\sigma_h+\sigma_h'))G_4$,
$C_3 = (\beta_h+\sigma_h)(\gamma+\sigma_h+\sigma_h')[\mathcal{R}_c^2-1]$. The trivial solution, $I_h^* = 0$, too (3.16) is the locally asymptotically stable disease-free equilibrium. Since $C_1 > 0$, and using Descartes's rule of signs, Hence, we get the following cases:

i $C_2 < 0$ and $\mathcal{R}_c > 1$, gives a unique endemic equilibrium in the model.
ii $C_2 > 0$ and $\mathcal{R}_c > 1$, gives a unique endemic equilibrium in the model.
iii $C_2 < 0$ and $\mathcal{R}_c < 1$, indicates absence of endemic equilibrium in the model.
iv $C_2 > 0$ and $\mathcal{R}_c < 1$, gives two positive endemic equilibria in the model.

Cases (i) and (ii) show the absence of backward bifurcation in the model since backward bifurcation requires at least two endemic equilibria in order to occur. Case (iii) indicates

that the disease has been completely eradicated in the population. Case (iv) may be an indication of backward bifurcation in the model.

At the endemic equilibrium, E_1, the Jacobian matrix of the right-hand side of (2.1) is

$$\mathcal{J}(E_1) = \begin{pmatrix} -a_1 & 0 & 0 & 0 & 0 & 0 & -a_2 & 0 & 0 & -a_3 \\ c_1 & -c_2 & 0 & 0 & 0 & 0 & c_3 & 0 & 0 & c_4 \\ 0 & d_1 & -d_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & e_1 & -e_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -f_1 & 0 & -f_2 & f_3 & f_4 & 0 & 0 & 0 \\ 0 & 0 & g_1 & 0 & g_2 & -g_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & h_1 & -h_2 & 0 & 0 & 0 \\ 0 & 0 & -j_1 & 0 & 0 & 0 & 0 & -j_2 & j_3 & j_4 \\ 0 & 0 & k_1 & 0 & 0 & 0 & 0 & k_2 & -k_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & m_1 & -m_2 \end{pmatrix} \tag{3.17}$$

where, $a_1 = b_1\alpha_{mh}I^*_m + b_2\alpha_{mwh}I^*_{mw} + \sigma_h$, $a_2 = b_1\alpha_{mh}S^*_h$, $a_3 = b_2\alpha_{mwh}S^*_h$,
$c_1 = b_1\alpha_{mh}I^*_m + b_2\alpha_{mwh}I^*_{mw}$, $c_2 = \beta_h + \sigma_h$, $c_3 = b_1\alpha_{mh}S^*_h$, $c_4 = b_2\alpha_{mwh}S^*_h$,
$d_1 = \beta_h$, $d_2 = \gamma + \sigma_h + \sigma'_h$, $e_1 = \gamma$, $e_2 = \sigma_h$, $f_1 = b_1\alpha_{hm}S^*_m$, $f_2 = b_1\alpha_{hm}I^*_h + \sigma_m - q\kappa\mu_m$,
$f_3 = q\kappa\mu_{mw}$, $f_4 = q\kappa\mu_{mw}$, $g_1 = b_1\alpha_{hm}S^*_m$, $g_2 = b_1\alpha_{hm}I^*_h$, $g_3 = \beta_m + \sigma_m$, $h_1 = \beta_m$, $h_2 = \sigma_m$,
$j_1 = b_2\alpha_{hmw}S^*_{mw}$, $j_2 = b_2\alpha_{hmw}I^*_h + \sigma_m - \kappa\mu_{mw}$, $j_3 = \kappa\mu_{mw}$, $j_4 = \kappa\mu_{mw}$, $k_1 = b_2\alpha_{hmw}S^*_{mw}$,
$k_2 = b_2\alpha_{hmw}I^*_h$, $k_3 = \beta_{mw} + \sigma_m$, $m_1 = \beta_{mw}$, $m_2 = \sigma_m$.

Theorem 3.2 (Gershgorin). *Let $A = a_{ij}$ be a complex $n \times n$ matrix with eigenvalues, λ. For $i \in n$, let $R_i = \sum_{j\neq i} |a_{ij}|$ be the sum of the absolute values of the non-diagonal entries in the i^{th} row. Let $D(a_{ii}, R_i) := |\lambda - a_{ii}| \leqslant R_i$ be a closed disc centered at a_{ii} with radius R_i. Then, the eigenvalues of A belong to the union of the discs, $D(a_{ii}, R_i)$.*

Theorem 3.3. *The endemic equilibrium E_1 is locally asymptotically stable if the Jacobian matrix $J(E_1)$ is strictly diagonally dominant.*

Proof of Theorem 4: Using Gershgorin's theorem, we have that all the eigenvalues, λ of $J'(E_1)$ lie in the closed disc $|\lambda - a_{ii}| \leqslant \sum_{j\neq i} |a_{ij}|$, $i, j = 1, 2, 3, \cdots, 9$. Hence, we have

$$\begin{aligned} -(a_1 + a_2 + a_3) \leqslant \lambda \leqslant a_2 + a_3 - a_1, \\ -(c_1 + c_2 + c_3 + c_4) \leqslant \lambda \leqslant c_1 + c_3 + c_4 - c_2, \\ -(d_1 + d_2) \leqslant \lambda \leqslant d_1 - d_2, \\ -(e_1 + e_2) \leqslant \lambda \leqslant e_1 - e_2, \\ -(f_1 + f_2 + f_3 + f_4) \leqslant \lambda \leqslant f_1 + f_3 + f_4 - f_2, \\ -(g_1 + g_2 + g_3) \leqslant \lambda \leqslant g_1 + g_2 - g_3, \\ -(h_1 + h_2) \leqslant \lambda \leqslant h_1 - h_2 \\ -(j_1 + j_2 + j_3 + j_4) \leqslant \lambda \leqslant j_1 + j_3 + j_4 - j_2, \\ -(k_1 + k_2 + k_3) \leqslant \lambda \leqslant k_1 + k_2 - k_3, \\ -(m_1 + m_2) \leqslant \lambda \leqslant m_1 - m_2. \end{aligned} \tag{3.18}$$

In the above inequalities, each left-hand side is negative, and each right-hand side is negative if $\mathcal{J}'(E_1)$ is strictly diagonally dominant. Therefore, if $\mathcal{J}'(E_1)$ strictly diagonally dominant, Gershgorin's theorem implies that the eigenvalues of $\mathcal{J}'(E_1)$, $\lambda \in [-\Phi_1, -\Phi_2]$, where $-\Phi_1$ and $-\Phi_2$ are respectively, the minimum and maximum of the left-hand and right-hand sides of the inequalities in (3.18). Therefore, the endemic equilibrium E_1 is locally asymptotically stable if the Jacobian matrix, $\mathcal{J}'(E_1)$ is strictly diagonally dominant.

3.3. Existence of Forward Bifurcation in the Model

In subsection 3.2, it is not clear whether the model has a unique endemic equilibrium when $\mathcal{R}_c > 1$. Therefore, bifucation analysis is conducted here to show the existence of one endemic equilibrium and forward bifurcation in the model. This is done by applying the method in Castillo-Chavez and Song[21], which is based on the use of center manifold theory, and requires the computation of left and right eigenvector corresponding to the zero eigenvalue of the Jacobian matrix of the vector field evaluated at the disease-free equilibrium point.

Theorem 3.4. *(Castillo-Chavez and Song (2004)).*
For a given system of ordinary differential equation with a parameter φ:

$$x'(t) = f(x, \varphi), \quad f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}, f \in C^2(\mathbb{R}^n \times \mathbb{R}).$$

Let $J = \frac{\partial f_k(0,0)}{\partial x_j}$ be the Jacobian matrix of $f(x, \varphi)$ evaluated at $(0, 0)$, where $x = 0$ is the equilibrium point of the system. Assume the following hold

1. *zero is a simple eigenvalue of the Jacobian matrix, and all other eigenvalues of J have negative real part.*
2. *J has a nonnegative right eigenvector w and a left eigenvector corresponding to the zero eigenvalue. Let $f_k(x, \varphi)$ denote the kth component of $f(x, \varphi)$ and*

$$\phi_1 = \sum_{k,i,j}^{n} v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j}, \quad \phi_2 = \sum_{k,i}^{n} v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \alpha^*_{mwh}}$$

then

1. *if $\phi_1 > 0, \phi_2 > 0$, then when $\varphi < 0$ with $|\varphi| \ll 1$, $x = 0$ is locally asymptotically stable and $\exists$ a positive equilibrium, and when $0 < |\varphi| \ll 1$, $x = 0$ is unstable, and $\exists$ a negative locally asymptotically stable equilibrium.*
2. *If $\phi_1 < 0$ and $\phi_2 > 0$, when φ changes from negative to positive, $x = 0$ changes from stable to unstable. correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable. particularly, if $\phi_1 < 0$ and $\phi_2 > 0$, then a forward bifurcation occurs at $\varphi = 0$, and if $\phi_1 > 0$ and $\phi_2 > 0$, a backward bifurcation occurs at $\varphi = 0$.*

If we choose α_{mwh} as our bifurcation parameter, then $\mathcal{R}_c = 1$ implies that

$$\alpha_{mwh} = \alpha^*_{mwh} = \frac{1 - \mathcal{K}_{hm}\mathcal{K}_{mh}}{\mathcal{K}'_{mwh}\mathcal{K}_{hmw}}, \text{ where } \mathcal{K}'_{mwh} = \frac{b_2 \beta_{mw} \pi_h}{\sigma_h \sigma_m (\beta_{mw} + \sigma_m)}$$

Let $J(E_0;\alpha^*_{mwh})$ be the Jacobian matrix of $f(x,\alpha^*_{mwh})$ at the disease-free equilibrium E_0. Note that the elements of $J(E_0;\alpha^*_{mwh})$ and $J(E_0)$ are the same, except for J_{13} and J_{23} where α_{mwh} is replaced with α^*_{mwh}. The matrix $J(E_0;\alpha^*_{mwh})$ possesses one zero eigenvalue, while the remaining eigenvalues have negative real part. Therefore, E_0 is non-hyperbolic, and we can apply theorem 3 to analyze the dynamics of the model around the bifurcation parameter value $\alpha_{mwh}=\alpha^*_{mwh}$. The right eigenvector $\tilde{w}=(w_1\ w_2\ w_3\ \cdots w_{10})^T$ and the left eigenvector $\tilde{v}=(v_1\ v_2\ v_3\ \cdots v_{10})^T$ corresponding to the zero eigenvalue satisfy the systems $J(E_0;\alpha^*_{mwh})\tilde{w}=0$ and $\tilde{v}J(E_0;\alpha^*_{mwh})=0$, respectively, where $w_1=-\left(\frac{\beta_m b_1^2\alpha_{hm}\alpha_{mh}S^0_mS^0_h}{\sigma_m(\beta_m+\sigma_m)}+\frac{\beta_{mw}b_2^2\alpha_{hmw}\alpha_{mwh}S^0_{mw}S^0_h}{\sigma_m(\beta_{mw}+\sigma_m)}\right), w_2=\frac{\beta_h}{\gamma+\sigma+\sigma'_h}, w_3=w_3>0, w_4=\frac{\gamma}{\sigma_m}$, $w_5=-\frac{b_1\alpha_{hm}S^0_m}{\sigma_m-qk\mu_m}$, $w_6=\frac{b_1\alpha_{hm}S^0_m}{\beta_m+\sigma_m}$, $w_7=\frac{b_1\beta_m\alpha_{hm}S^0_m}{\sigma_m(\beta_m+\sigma_m)}$, $w_8=-\frac{b_2\alpha_{hmw}S^0_{mw}}{\sigma_m-k\mu_{mw}}$, $w_9=\frac{b_2\alpha_{hmw}S^0_{mw}}{\beta_{mw}+\sigma_m}$,
$w_{10}=\frac{b_2\beta_{mw}\alpha_{hmw}S^0_{mw}}{\sigma(\beta_{mw}+\sigma_m)}$, and $v_3=\left(\frac{\beta_m b_1^2\alpha_{hm}\alpha_{mh}S^0_mS^0_h}{\sigma_m(\beta_m+\sigma_m)(\gamma+\sigma_h+\sigma'_h)}+\frac{\beta_{mw}b_2^2\alpha_{hmw}\alpha_{mwh}S^0_{mw}S^0_h}{\sigma_m(\beta_{mw}+\sigma_m)(\gamma+\sigma_h+\sigma'_h)}\right)$, $v_6=\frac{b_1\beta_m\alpha_{mh}S^0_h}{\sigma_m(\beta_m+\sigma_m)}$, $v_7=\frac{b_1\beta_m\alpha_{mh}S^0_h}{\sigma_m}$, $v_9=\frac{b_2\beta_{mw}\alpha_{mwh}S^0_h}{\sigma_m(\beta_{mw}+\sigma_m)}$, $v_{10}=\frac{b_2\beta_{mw}\alpha_{mwh}S^0_h}{\sigma_m}$. The components v_1,v_4,v_5 and v_8 have zero values since they correspond to the states that are not infected by Zika virus disease [20]. The nonzero second order partial derivatives of $f(x,\alpha^*_{mwh})$ with respect to each relevant variable are
$\frac{\partial^2 f_2(0,0)}{\partial S_h\partial I_m}=b_1\alpha_{mh}$, $\frac{\partial^2 f_2(0,0)}{\partial S_h\partial I_{mw}}=b_2\alpha^*_{mwh}$, $\frac{\partial^2 f_6(0,0)}{\partial I_h\partial S_m}=b_1\alpha_{hm}$, $\frac{\partial^2 f_9(0,0)}{\partial I_h\partial S_{mw}}=b_2\alpha_{hmw}$, $\frac{\partial^2 f_2(0,0)}{\partial S_H\partial\alpha^*_{mwh}}=b_2I_{mw}$,
$\frac{\partial^2 f_2(0,0)}{\partial I_{mw}\partial\alpha^*_{mwh}}=b_2S_h$.
Using the expression for the bifurcation coefficients in theorem 5, we have
$\phi_1=w_1v_2(w_7b_1\alpha_{mh}+w_{10}b_2\alpha^*_{mwh})+v_6w_3w_5b_1\alpha_{hm}+v_9w_3w_8b_2\alpha_{hmw}$ and $\phi_2=v_2w_2b_2(S_h+I_{mw})$.
Since $w_1<0$, $w_5<0$ and $w_8<0$, we see that $\phi_1<0$ and $\phi_2>0$. This shows that forward bifurcation occurs in the model. Hence, the model has a unique endemic equilibrium, E_1 that is locally asymptotically stable for $\mathcal{R}_c>1$.

4. Numerical Experiment and Discussion

Simulation of the model is carried out here to show the impact of introducing Wolbachia-infected mosquitoes on the susceptible wild aedes aegypti mosquitoes. The simulation is carried out in MATLAB© R2010a, where fourth-order Runge-Kutta integration scheme is applied to obtain numerical solution to the non-linear system. The initial values of the state variables and the parameter values used in the simulation are presented in Table 1 and Table 2, respectively. The value of some of the parameters were obtained from the literature, while others are assumed to be within a reasonable and realistic range for the purpose of the simulation. In Table 2, the value of the parameters $b_1, b_2, \beta_{mw}, \alpha_{mw}$ are chosen based on the characteristics of the Wolbachia-infected mosquitoes. When the Wolbachia-infected aedes aegypti have established in the wild, the number of natural/wild aedes aegypti becomes small due to cytoplasmic incompatibility, thereby reducing the biting rate, b_1 of wild mosquitoes, and increasing b_2 of Wolbachia-carrier mosquitoes. The extrinsic incubation period of Zika virus is $\frac{1}{\beta_2}=9$. Based on our assumption that Wolbachia increases the extrinsic incubation period of the virus, we assume that the extrinsic incubation period is doubled in Wolbachia-carrier mosquitoes such that $\frac{1}{\beta_3}=\frac{2}{\beta_2}=18$.

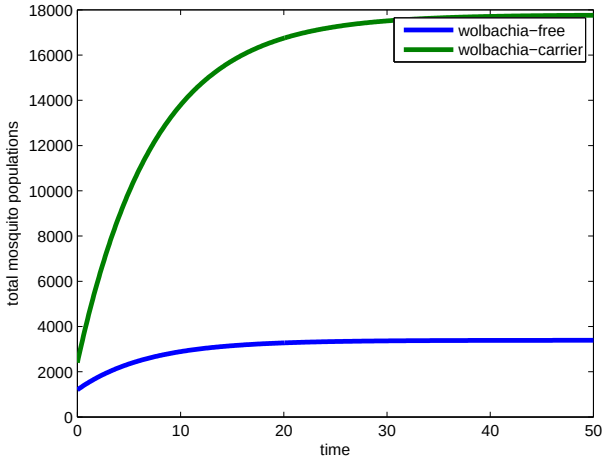
Since the incubation rate of Zika virus in the mosquito is small, we assume that the probability, α_{31} of transmission of Zika virus from Wolbachia-infected mosquito to human is negligibly small. The results of the simulation are given in Figures(1-3). In Figure 1, we see the action of cytoplasmic incompatibility on the population of Wolbachia-free mosquitoes as induced by Wolbachia. In Figure 1(i), the difference between the population of Wolbachia-free and Wolbachia-carrier mosquitoes is clearly seen. This shows that Wolbachia-carrier mosquitoes have suppressed the wild mosquitoes, thereby overtaking its population. Similarly, in Figure 1(ii), the number of susceptible Wolbachia-carrier mosquitoes is higher than the number of susceptible Wolbachia-free mosquitoes. This is also due to cytoplasmic incompatibility. In Figure 2, we have the population of exposed and infectious Wolbachia-carrier and Wolbachia-free mosquitoes. It is noticeable that the populations of exposed and infectious Wolbachia-carrier mosquitoes are higher than those of exposed and infectious Wolbachia-free mosquitoes. However, this does not translate to high exposed and infected humans as seen in Figure 3. This is because even though the infected Wolbachia-carrier mosquitoes have the virus, they have less capability to transmit the virus to humans due to prolonged incubation period of the virus in them. In Figure 3 , we notice initial increase in the populations of exposed and infectious humans in number, which later decrease as time increases. The initial increase in these populations could be attributed to initial period when Wolbachia-infected mosquitoes have not fully established in the wild. During this period, there are more exposed and infectious wild mosquitoes than the Wolbachia-infected mosquitoes, which translates to high number of exposed and infectious humans. However, as time increases when Wolbachia-infected mosquitoes have established fully in the wild, the population of wild mosquitoes begins to reduce, which translates to reduction in the population of exposed and infectious humans.

variables	S_h	E_h	I_h	R_h	S_m	E_m	I_m	S_{mw}	E_{mw}	I_{mw}
initial values	5000	2000	1500	100	1500	300	400	2400	0	0

Table 1: Initial Values

Parameter	*Value*	*Source*	*Parameter*	*Value*	*Source*
π_h	250	assumed	σ'_h	0.00001	assumed
π_m	500	assumed	σ_h	0.0005	assumed
α_{hm}	0.175	[2]	π_{mw}	250	assumed
α_{hmw}	0.0005	assumed	μ_M	0.03	assumed
μ_{mw}	0.03	assumed	σ_M	0.15	[13]
β_h	$\frac{1}{3}$	[22]	κ	0.5	[13]
b_1	0.0075	assumed	γ	0.5	assumed
b_2	0.7	assumed	α_{mwh}	0.001	assumed
β_m	1/9	[23]	α_{mh}	0.0175	assumed
β_{mw}	1/18	assumed	q	0.017	assumed

Table 2: Parameter Values used in this model



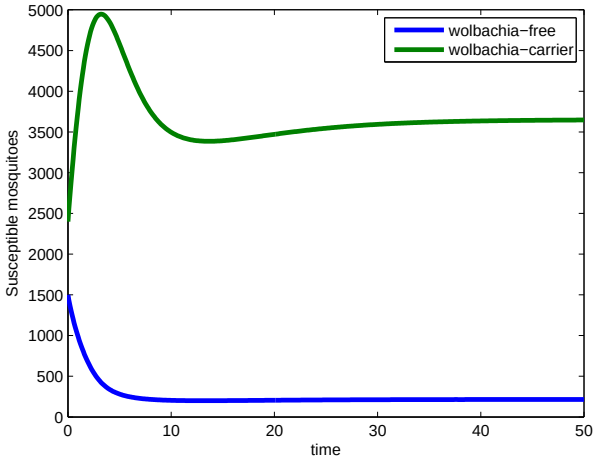
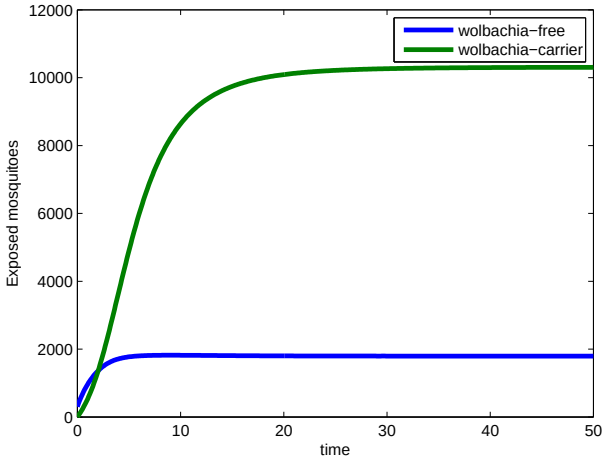


Figure 1: (i)Total mosquitoes population (ii)Susceptible mosquitoes

5. Summary and Conclusion

A mathematical model for the control of Zika virus disease is presented in this paper. The control is based on using aedes aegypti mosquitoes infected with Wolbachia to eliminate natural aedes aegypti in Zika-endemic area. The expression for the control reproduction number is derived, and it is shown that the disease-free equilibrium of the model is locally asymptotically stable if $\mathcal{R}_c < 1$. Bifurcation analysis conducted on the model shows that forward bifurcation takes place in the model, which confirms that the model has only one endemic equilibrium which is locally asymptotically stable when $\mathcal{R}_c > 1$.
In conclusion, the model exhibits forward bifurcation rather than backward bifurcation, which shows that having $\mathcal{R}_c < 1$ is a necessary and sufficient requirement for eradicating Zika virus disease this mosquito control technique is applied, irrespective of the initial sizes of the infected human and mosquito populations. This further confirms the efficacy of using this mosquito control method for eradicating Zika virus disease or any other disease transmitted by aedes aegypti mosquitoes.



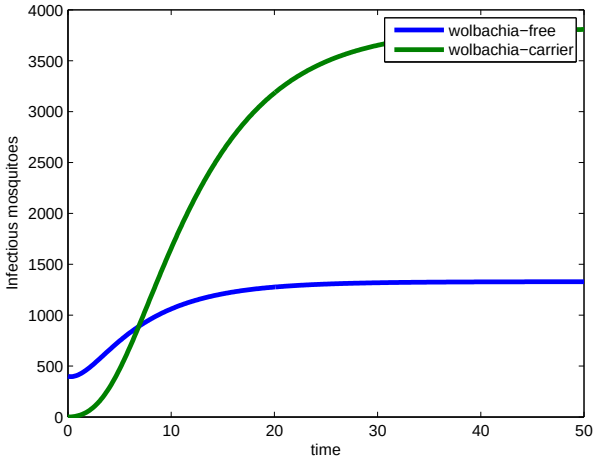
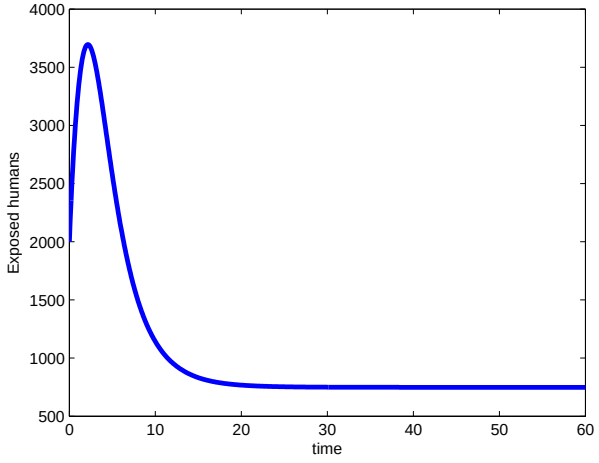


Figure 2: (i) Exposed mosquitoes (ii) infectious mosquitoes



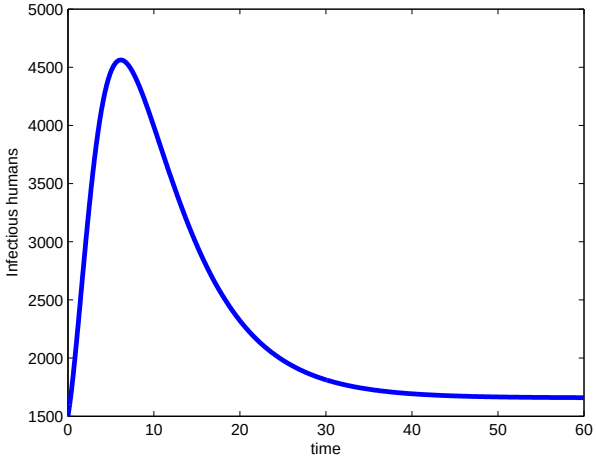


Figure 3: (i) Exposed humans (ii) infectious humans

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