Modeling the transmission dynamics of Zika with sterile insect technique

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Abstract

A deterministic model for the transmission dynamics of Zika is designed and rigorously analysed. A model consisting of mutually exclusive compartments representing the human and mosquito dynamics takes into account both direct (human-human) and indirect modes of transmissions. The basic offspring number of the mosquito population is computed, and condition for existence and stability of equilibria is investigated. Using the centre manifold theory, the model (with and without direct transmission) is shown to exhibit the phenomenon of backward bifurcation (where a locally-asymptotically stable disease free equilibrium co-exists with a locally-asymptotically stable endemic equilibrium) whenever the associated reproduction number is less than unity. The study shows that the models with and without direct transmission exhibit the same qualitative dynamics with respect to the local stability of their associated disease-free equilibrium and backward bifurcation phenomenon. The main cause of the backward bifurcation is identified as Zika induced mortality in humans. Sensitivity (local and global) analysis of the model parameters are conducted to identify crucial parameters that influence the dynamics of the disease. Analysis of the model shows that an increase in the mating rate with sterile mosquito decreases the mosquito population. Numerical simulations, using parameter values relevant to the transmission dynamics of Zika are carried out to support some of the main theoretical findings.

Keywords: Zika virus, Sterile insect technique, Reproduction number, Stability, Equilibrium, Bifurcation.

1 Introduction

Zika is a mosquito borne disease caused by Zika virus (Zv) of genus Flavivirus. The virus was first identified in Uganda in 1947, through a monitoring network of sylvatic yellow fever in rhesus monkeys. Five years later, human infection was identified in Uganda and Tanzania. Since then, Zika outbreaks have been recorded in Africa, Americas, Asia and the Pacific [47]. The disease is primarily transmitted by a bite of an infected mosquito, mainly Aedes aegypti (a mosquito species that transmits yellow fever, dengue, West Nile virus, Chikungunya and Japanese encephalitis virus) [29, 47]. The virus is also transmitted through sexual contact between humans [23, 24, 38, 47], blood transfusion and parental transmission [47]. Recently, incidences of congenital neurological disorder (microcephaly) and auto-immune (Guillain-Barr syndrome) complications have been attributed to growing number of Zika incidences especially in the Americas [41, 47].

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Unfortunately there is no specific treatment for Zika infection available. Fluid replacement therapy is used for individuals with symptoms such as fever, rash or arthralgia [47]. Although there is no effective vaccine for Zika at the moment, a number of candidate vaccines are undergoing various phases of clinical trials. One of the recent is the experimental vaccine known as rZIKV/D4Δ30-713 developed by scientist at the National Institute of Allergy and Infectious Diseases (NIAID), which is being evaluated in a phase 1 clinical trial (initiated in August 2018) [48]. It is acknowledged that mosquito control is one of the most important tools in the control and/or prevention of mosquito borne diseases (such as dengue and Zika). One of the most promising methods to control Zika is the Sterile Insect Technology (SIT), which is non-polluting method of insect control that relies on the release of sterile male mosquitoes. Mating of released sterile males with wild female mosquitoes leads to non-hatching of eggs and this results in the decline of the wild mosquitoes population [3, 19, 32, 42].

Since the introduction of SIT, numerous mathematical models have been developed and used to quantify the impact of SIT on the transmission dynamics of vector borne diseases (VBDs). These models fall into two main categories, namely, the deterministic or process-based models (which represent the dynamics of the disease using differential equations) some of which include [3, 5, 19, 21, 22] (and some of the references therein), and the statistical models (mainly stochastic processes which are typically based on the use of time-series data to describe the correlation or relationship, between VBDs and the vector population). In this study, we extend the model designed in [3] by incorporating additional compartments for infectious mosquitoes (sterilized and non-sterilized) and human population. This allows us to assess the potential impact of sterilization on both mosquito and disease control. Furthermore, the model assumes that disease transmission between humans is possible, the recent findings which confirm the Zika transmission via sexual contact between humans include [23, 24, 37, 38, 47]. The purpose of incorporating sexual transmission is to investigate the potential impact of combined direct (human-to-human) and indirect (human-vector-human) Zika transmission.

The paper is organized as follows. A Zika model, which incorporates the dynamics of mosquitoes (sterilized and non-sterilized) and humans is formulated and analysed in Section 2. Mosquito-only model is analysed in Section 3. Analysis of the model in the absence of direct (human-to-human) transmission is presented in Section 4. The model with direct transmission is analysed in Section 5. Sensitivity analysis and numerical simulations are reported in Sections 6 and 7, respectively.

2 Model formulation

The model assumes a homogeneous mixing of human and vector (mosquito) populations, so that each mosquito bite has equal chance of transmitting the virus to a susceptible human (or acquiring infection from infectious human) in the population. The total human population at time t, denoted by $N_H(t)$ is split into three mutually exclusive compartments of susceptible ($S_H(t)$), infected ($I_H(t)$) and recovered ($R_H(t)$), so that

$$N_H(t) = S_H(t) + I_H(t) + R_H(t).$$

Similarly, the total mosquito population is split into aquatic (immature) and non-aquatic (adult) stages. For mathematical tractability, the aquatic stages (eggs, larvae and pupae) are lumped into one compartment denoted by $A(t)$. The adult mosquito population (non-aquatic stage) at time t is sub-divided into seven mutually exclusive compartments consisting of non-fertilized adult female mosquitoes ($Y(t)$), fertilized non-sterile susceptible females ($F_N(t)$), fertilized sterile susceptible females (those that could lay eggs but do not hatch due to mating with sterile male mosquitoes) ($F_S(t)$), fertilized non-sterile infected females ($F_{NI}(t)$), fertilized sterile infected females ($F_{SI}(t)$), sterile male ($M_S(t)$) and non-sterile male ($M_N(t)$) mosquitoes. Sterile male mosquitoes are injected into the

population at a constant rate. Thus, the total mosquito population at time t is given by

$$N_V(t) = A(t) + Y(t) + F_N(t) + F_S(t) + F_{NI}(t) + F_{SI}(t) + M_N(t) + M_S(t).$$

It is assumed that humans can acquire infection following effective contact with infectious mosquitoes in the F_{NI} or F_{SI} classes at a rate λ_{H1} given by

$$\lambda_{H1} = \beta_{VH}\frac{(F_{NI} + \eta_1 F_{SI})}{N_V}, \tag{1}$$

where $\beta_{VH} = \rho_{VH}\xi_1$ is the effective contact rate between infectious mosquitoes and susceptible humans, it is defined as the product of the transmission probability from an infectious mosquito to susceptible human (ρ_{VH}) and the biting rate of infectious mosquitoes (ξ_1). The modification parameter $0 < \eta_1 < 1$ accounts for the assumed reduction in transmissibility of mosquitoes in F_{SI} class in comparison to those in F_{NI} class. Furthermore, it is assumed that humans can acquire Zika infection from infectious humans (in I_H or R_H class) via sexual contact at a rate λ_{H2} (this is in line with some recent clinical studies which suggest that, high viral load was found in the semen and saliva of recovered patients weeks after recovery, hence, there is high chance of direct vaginal or oral sex transmission by recovered humans [20, 23, 24, 38, 47, 48]). It is worth mentioning that Zika is the first Flavivirus known to be transmitted sexually by infectious humans [24]. Thus

$$\lambda_{H2} = \beta_{HH}\frac{(I_H + \eta_2 R_H)}{N_H},$$

where $\beta_{HH} = \rho_{HH}\xi_2$ is the effective contact rate between infectious and susceptible humans, which is the product of the transmission probability from infectious humans to susceptible humans (ρ_{HH}) and contact rate (usually sexual) between infectious and susceptible humans (ξ_2). The modification parameter $0 < \eta_2 < 1$ accounts for the assumed reduction in transmissibility of recovered humans in comparison to infectious humans, so that the force of infection of humans is given by

$$\lambda_H = \lambda_{H1} + \lambda_{H2} = \beta_{VH}\frac{(F_{NI} + \eta_1 F_{SI})}{N_V} + \beta_{HH}\frac{(I_H + \eta_2 R_H)}{N_H}. \tag{2}$$

Similarly, a susceptible mosquito can acquire Zika infection from an infectious human at a rate λ_V (the force of infection of mosquitoes), given by

$$\lambda_V = \beta_{HV}\frac{I_H}{N_H},$$

where $\beta_{HV} = \rho_{HV}\xi_3$ is the effective contact rate between infectious humans and susceptible mosquitoes; it is defined as the product of the transmission probability from an infectious human to a susceptible mosquito (ρ_{HV}) and the biting rate of susceptible mosquitoes (ξ_3).

2.1 Incidence functions

In this section, the functional form of the incidence functions for the transmission dynamics of Zika will be derived. Using the well known fact that for mosquito borne diseases, the total number of bites made by mosquitoes must be equal to the total number of bites received by humans (see [6, 7, 14, 26, 40] for detailed justification), for the number of bites to be conserved, the following equation must hold

$$\beta_{VH}(N_H, N_V)N_H = \beta_{HV}N_V,$$

hence

$$N_V = \frac{\beta_{VH}(N_H, N_V)}{\beta_{HV}}N_H. \tag{3}$$

Substituting (3) in (1) gives

$$\lambda_{H1} = \frac{\beta_{HV}}{N_H}(F_{NI} + \eta_1 F_{SI}),$$

so that

$$\lambda_H = \lambda_{H1} + \lambda_{H2} = \frac{\beta_{HV}(F_{NI} + \eta_1 F_{SI}) + \beta_{HH}(I_H + \eta_2 R_H)}{N_H}. \tag{4}$$

2.2 Dynamics of human population

The population of susceptible humans is generated by birth or immigration at a constant rate b_H. This population is decreased by acquiring infection after receiving adequate number of bites capable of disease transmission from an infectious mosquito (at the rate λ_{H1}) or via sexual transmission by an infectious human (at the rate λ_{H2}) and by natural death at a rate μ_H. This gives

$$\frac{dS_H}{dt} = b_H - \lambda_H S_H - \mu_H S_H.$$

The population of infectious humans is generated by infection of susceptible humans at the rate λ_H, and decreases due to recovery (at a rate γ_H), natural death (at the rate μ_H) and disease induced death (at a rate δ_H), so that

$$\frac{dI_H}{dt} = \lambda_H S_H - \delta_H I_H - \gamma_H I_H - \mu_H I_H.$$

The population of recovered humans is generated by the recovery of infectious humans (at the rate γ_H) and reduces due to natural death (at the rate μ_H). Thus

$$\frac{dR_H}{dt} = \gamma_H I_H - \mu_H R_H.$$

2.3 Dynamics of mosquito population

The population of mosquitoes in the aquatic stage (eggs, larvae and pupae) is increased through oviposition by reproductive mosquitoes at a rate ϕ_V. This population decreases due to natural death at a rate μ_V (it is assumed that natural death occurs in all mosquito compartments at the rate μ_V), by density dependent death at a rate μ, maturate and move out of aquatic stage at a rate b_V. Thus

$$\frac{dA}{dt} = \phi_V F_{NI} + \phi_V F_N - \mu A^2 - \mu_V A - b_V A.$$

The population of non-sterile male mosquitoes evolves directly from the aquatic stage at a rate $(1 - r)b_V$, and decreases due to natural death. Thus

$$\frac{dM_N}{dt} = (1 - r)b_V A - \mu_V M_N.$$

Sterile male mosquitoes (M_S) are released into the population at a rate $\omega(t)$ at time t. However, due to some environmental and geographical factors that may affect the mixing of sterile and wild mosquitoes, such as location of mosquito breeding site, it is convenient to assume that, only a fraction p of the released mosquitoes will join the wild mosquito population. It is further believed that the sterile mosquitoes are in several ways the same as wild mosquitoes. In particular, they are able to mate with wild female mosquitoes. However, there are some differences, which include a change in mating competitiveness due to irradiation and population distributions (which depend on the released formula that could depends on the breeding site and feeding ground). The differences in mating

competitiveness can be captured by a modification parameter g which represents the mean mating competitiveness of the sterile male mosquitoes [3, 14, 30], so that, if the number of wild mosquitoes equivalent of sterile mosquitoes is given by M_S, then the actual number of released sterile male mosquitoes is $\frac{1}{pg}M_S$. Therefore, the population of sterile male mosquitoes increases at a rate $pg\omega(t)$ at time t (see for instance [3]). This population decreases due to natural death at a rate μ_S, so that

$$\frac{dM_S}{dt} = pg\omega - \mu_S M_S.$$

It is assumed that mating of female mosquitoes with sterile male mosquitoes results to non-hatching of their eggs (that is, they lay infertile eggs). Thus, under the previously stated assumptions and adjustments to M_S, it is convenient to assume that the mosquitoes in the M_S and M_N classes have equal chances of mating. Thus, a female mosquito has probability $\frac{M_S}{M_S+M_N}$ of mating with sterile male mosquito and probability $\frac{M_N}{M_S+M_N}$ of mating with non-sterile male mosquito. Adult female mosquitoes evolve from the aquatic stage at a rate $rb_V A$, they mate with non-sterile male mosquito and progress to F_N compartment at a rate $\frac{\alpha M_N}{M_S+M_N}$, or with a sterile male mosquito and move to F_S compartment at a rate $\frac{\alpha M_S}{M_S+M_N}$ (where α is total mating rate). Note that the total mating rate $\frac{\alpha M_S}{M_S+M_N} + \frac{\alpha M_N}{M_S+M_N} = \alpha$ remain the same. Thus we have

$$\frac{dY}{dt} = rb_V A - \frac{\alpha M_S}{M_N+M_S}Y - \frac{\alpha M_N}{M_N+M_S}Y - \mu_V Y.$$

The population of mosquitoes in the F_N class is generated from compartment Y through mating of female mosquitoes with a non-sterile male mosquitoes (M_N). In order to nourish their eggs before oviposition, they need blood, and hence they will probably bite an infectious human and if acquired infection move to the F_{NI} compartment at the rate λ_V. This population is reduced due to natural death, so that

$$\frac{dF_N}{dt} = \frac{\alpha M_N}{M_N+M_S}Y - \lambda_V F_N - \mu_V F_N.$$

Similarly, the population of mosquitoes in the F_S class is generated through mating of adult female mosquitoes with sterile male mosquitoes. This population is decreased by infections following contact with infectious humans and progress to the F_{SI} compartment at a rate λ_V. This gives

$$\frac{dF_S}{dt} = \frac{\alpha M_S}{M_N+M_S}Y - \lambda_V F_S - \mu_V F_S.$$

The population of mosquitoes in the F_{NI} class is generated by the infection of mosquitoes in F_N class, and are decreased by natural death. Hence

$$\frac{dF_{NI}}{dt} = \lambda_V F_N - \mu_V F_{NI}.$$

Finally, the population of mosquitoes in the F_{SI} class is generated from F_S after biting an infectious human. Thus

$$\frac{dF_{SI}}{dt} = \lambda_V F_{SI} - \mu_V F_{SI}.$$

2.4 Model equations

Since there are only two mating possibilities, either with sterilized or with non-sterilized male mosquitoes, we let $\frac{M_S}{M_N+M_S} = \theta$, so that $\frac{M_N}{M_N+M_S} = 1-\theta$. Thus, the Zika transmission model is given by the following system of non-linear differential equations (a flow diagram of the model is given in Figure 1

and the associated variables and parameters are described in Table 1)

$$\text{Humans}\begin{cases}\dfrac{dS_H}{dt} = b_H - \lambda_H S_H - \mu_H S_H,\\ \dfrac{dI_H}{dt} = \lambda_H S_H - \delta_H I_H - \gamma_H I_H - \mu_H I_H,\\ \dfrac{dR_H}{dt} = \gamma_H I_H - \mu_H R_H,\end{cases}$$

$$\text{Mosquitoes}\begin{cases}\dfrac{dA}{dt} = \phi_V F_{NI} + \phi_V F_N - \mu A^2 - \mu_V A - b_V A,\\ \dfrac{dY}{dt} = r b_V A - \alpha Y - \mu_V Y,\\ \dfrac{dF_N}{dt} = \alpha(1-\theta)Y - \lambda_V F_N - \mu_V F_N,\\ \dfrac{dF_S}{dt} = \alpha\theta Y - \lambda_V F_S - \mu_V F_S,\\ \dfrac{dF_{NI}}{dt} = \lambda_V F_N - \mu_V F_{NI},\\ \dfrac{dF_{SI}}{dt} = \lambda_V F_S - \mu_V F_{SI},\\ \dfrac{dM_N}{dt} = (1-r) b_V A - \mu_V M_N,\\ \dfrac{dM_S}{dt} = pg\omega(t) - \mu_V M_S.\end{cases} \tag{5}$$

Notice that, the last equation of (5) is controlled externally and it is independent of the other compartments. Therefore, given $\omega(t)$ continuous, as a linear equation it has the solution

$$M_S(t) = e^{-\mu_S t}\Big(M_S(0) + \int_0^t e^{\mu_S j} pg\omega(j)dj\Big). \tag{6}$$

It is worth mentioning that model (5) was also considered in a conference proceeding (reference [14] by the same authors). The focus and approach of the two papers are however different. This study gives a thorough rigorous theoretical and constructive analysis of the model, such as the global stability and backward bifurcation property, global sensitivity analysis, we also consider the model with and without human-human Zika transmission. The Zika model (5), to the author's knowledge is the first to incorporate sterile insect technique with both direct and indirect transmission modes. The model extends some Zika transmissions models and sterile insect technique (SIT) models in the literature, such as those in [1, 3, 5, 7, 19, 21, 22, 25, 31], by for instance:

- Incorporating mosquito sterilization in the model for the transmission of Zika, which is not considered in [1, 7, 25, 31].
- Incorporating the aquatic and non-aquatic stages of mosquitoes which allows us to evaluate the effects of the mosquito reproduction and sterilization on disease transmission, which is not considered in [3, 5, 7, 21, 22, 25].
- Allowing for the transmissions of Zika by both infectious and recovered humans, whereas only transmission by infectious human is assumed in [7, 25].

To understand the impact of controlling mosquito population, it is imperative to consider the mosquito-only population in the presence of sterilization. Thus, the following section.

3 Theoretical analysis of the mosquito-only model

Here, we carry out analysis of mosquito population in the absence of interaction with infectious humans, by considering the compartment for the non infectious mosquito population only, given by

$$\begin{aligned}
\frac{dA}{dt} &= \phi_V F_N - \mu A^2 - \mu_V A - b_V A,\\
\frac{dY}{dt} &= r b_V A - \alpha Y - \mu_V Y,\\
\frac{dF_N}{dt} &= \alpha(1-\theta)Y - \mu_V F_N,\\
\frac{dF_S}{dt} &= \alpha\theta Y - \mu_V F_S,\\
\frac{dM_N}{dt} &= (1-r)b_V A - \mu_V M_N.
\end{aligned} \tag{7}$$

3.1 Basic offspring number of the mosquito population

The basic offspring number of the mosquito population is given by

$$N_0 = \frac{\phi_V r b_V \alpha(1-\theta)}{(b_V+\mu_V)(\alpha+\mu_V)\mu_V}. \tag{8}$$

It can be interpreted as follows. A successful oviposition occurs after a female mosquito mates with a non-sterile (wild) male mosquito, which fertilizes and lays eggs. The average duration spent in aquatic stage by mosquito is $\frac{1}{b_V+\mu_V}$ (where b_V is the rate at which mosquitoes transform from aquatic to non-aquatic stage). Let r be the fraction of aquatic mosquitoes that become females, the probability that an egg survives the aquatic stage and becomes an adult female mosquito is

$$\frac{r b_V}{b_V+\mu_V}. \tag{9}$$

Similarly, $\frac{1}{(\alpha+\mu_V)}$ is the average duration spent by a female mosquito in Y compartment. The rate at which a mosquito in compartment Y move to compartment F_N (through mating with male mosquitoes in M_N compartment) is $\alpha(1-\theta)$. Thus, the probability that a female mosquito successfully moves from class Y to class F_N is given by

$$\frac{\alpha(1-\theta)}{(\alpha+\mu_V)}. \tag{10}$$

Furthermore, let the average lifespan of a mosquito in F_N class be $\frac{1}{\mu_V}$, and ϕ_V be its oviposition rate, then the average number of eggs oviposited by each mosquito in F_N compartment during its lifetime is given by

$$\frac{\phi_V}{\mu_V}. \tag{11}$$

The product of the quantities in equations (9), (10) and (11) gives the number of offspring produced by a single female mosquito that mates with a non-sterile male mosquito in its entire lifespan.

Thus, If $N_0 > 1$, then the mosquito population persist, otherwise, if $N_0 \leq 1$ then, the mosquito population goes to extinction and the indirect (human-mosquito-human) transmission can be eliminated.

3.2 Existence and stability of equilibria in mosquito population

Setting the right hand side of the equations of system (7) to zero gives the following equilibria

$$\mathcal{E}_0 = (A^*, Y^*, F_N^*, F_S^*, M_N^*) = \left(A^*, \frac{rb_V A^*}{K_3}, \frac{(1-\theta)\alpha r b_V A^*}{K_3\mu_V}, \frac{\theta\alpha r b_V A^*}{K_3\mu_V}, \frac{(1-r)b_V A^*}{K_3\mu_V}\right), \tag{12}$$

where

$$K_1 = \delta_H + \gamma_H + \mu_H, \quad K_2 = b_V + \mu_V, \text{ and } K_3 = \alpha + \mu_V,$$

and A^* satisfies

$$(A^*)^2 + \frac{K_2}{\mu}\Big[1 - \frac{\phi_V r b_V \alpha(1-\theta)}{K_2 K_3 \mu_V}\Big]A^* = 0,$$

or equivalently

$$A^*\left[A^* + \frac{K_2}{\mu}(1 - N_0)\right] = 0. \tag{13}$$

The roots of A^* are controlled by the magnitude of N_0.

If $N_0 \leq 1$, then, the only biologically meaningful root of equation (13) is $A^* = 0$, which corresponds to the trivial (or mosquito extinction) equilibrium, $\mathcal{E}_1$, given by

$$\mathcal{E}_1 = (A^*, Y^*, F_N^*, F_S^*, M_N^*) = (0, 0, 0, 0, 0). \tag{14}$$

It is worth mentioning that the equilibrium, $\mathcal{E}_1$, is biologically less attractive due to the absence of mosquitoes in the population. However if $N_0 > 1$, then, the system (7), has a non-zero positive equilibrium, $\mathcal{E}_2$, given by

$$\mathcal{E}_2 = \big(A^*, Y^*, F_N^*, F_S^*, M_N^*\big) = \Big(A^*, \frac{b_V r A^*}{K_3}, \frac{b_V r\alpha(1-\theta)A^*}{K_3\mu_V}, \frac{b_V\theta\alpha r A^*}{K_3\mu_V}, \frac{b_V(1-r)A^*}{K_3\mu_V}\Big), \tag{15}$$

where $A^* = \frac{K_2}{\mu}(N_0 - 1) > 0$.

Theorem 1 *For the mosquito-only model* (7)*, the extinction equilibrium, $\mathcal{E}_1$, is globally asymptotically stable (GAS) if $N_0 \leq 1$ and unstable otherwise. In addition, the positive equilibrium, $\mathcal{E}_2$, is locally asymptotically stable if $N_0 > 1$.*

The proof is given in Appendix A ■

The epidemiological implication of Theorem 1 is that the model (7) does not undergo backward bifurcation when $N_0 \leq 1$ (since $\mathcal{E}_1$ is GAS when $N_0 \leq 1$). Thus bringing the value of N_0 to below unity is a sufficient condition for the control of a mosquito population, which could be achieved by increasing the mating rate of sterile mosquitoes (θ).

The full model is now analysed for its dynamical features, by first of all considering the model in the absence of direct (human-human) transmission.

4 Analysis of the model (in the absence of direct transmission)

Here, we analyse the model (5) in the absence of human-human transmission (obtained by setting $\beta_{HH} = 0$), so that, the forces of infections are now given by

$$\lambda_H = \frac{\beta_{HV}(F_{NI} + \eta_1 F_{SI})}{N_H} \text{ and } \lambda_V = \beta_{HV}\frac{I_H}{N_H}. \tag{16}$$

4.1 Disease-free equilibrium (DFE)

The model (5) with (16) has the following disease-free equilibrium

$$\begin{aligned}\mathcal{E}_3 = \Big(S_H^*, I_H^*, R_H^*, A^*, Y^*, F_N^*, F_S^*, F_{NI}^*, F_{SI}^*, M_N^*\Big) = \Big(\frac{b_H}{\mu_H}, 0, 0, \frac{K_2}{\mu}[N_0 - 1], \\ \frac{b_V K_2 r[N_0-1]}{K_3\mu}, \frac{b_V K_2 \alpha r(1-\theta)[N_0-1]}{K_3\mu_V\mu}, \frac{b_V K_2\theta\alpha r[N_0-1]}{K_3\mu_V\mu}, 0, 0, \frac{b_V K_2(1-r)[N_0-1]}{K_3\mu_V\mu}\Big).\end{aligned} \tag{17}$$

Notice that:
If $N_0 \leq 1$, then, the only DFE of the model (5) is the trivial equilibrium (corresponding to human population free of mosquitoes), denoted by $\mathcal{E}_{31}$, given by

$$\mathcal{E}_{31} = (S_H^*, I_H^*, R_H^*, A^*, Y^*, F_N^*, F_S^*, F_{NI}^*, F_{SI}^*, M_N^*) = \left(\frac{b_H}{\mu_H}, 0, 0, 0, 0, 0, 0, 0, 0, 0\right). \tag{18}$$

This coincides with the mosquito extinction equilibrium, $\mathcal{E}_1$, which is shown to be GAS in Theorem 1.

If $N_0 > 1$, then, the system (5), has a non-zero positive disease-free equilibrium, $\mathcal{E}_{32}$ (which corresponds to human population in the presence of mosquitoes), given by

$$\begin{aligned}\mathcal{E}_{32} = \Big(S_H^*, I_H^*, R_H^*, A^*, Y^*, F_N^*, F_S^*, F_{NI}^*, F_{SI}^*, M_N^*\Big) = \Big(\frac{b_H}{\mu_H}, 0, 0, \frac{K_2}{\mu}[N_0 - 1], \\ \frac{b_V K_2 r[N_0-1]}{K_3\mu}, \frac{b_V K_2 \alpha r(1-\theta)[N_0-1]}{K_3\mu_V\mu}, \frac{b_V K_2\theta\alpha r[N_0-1]}{K_3\mu_V\mu}, 0, 0, \frac{b_V K_2(1-r)[N_0-1]}{K_3\mu_V\mu}\Big).\end{aligned} \tag{19}$$

As stated in Section 3.2, the equilibrium $\mathcal{E}_{31}$ is less attractive. Thus, the stability of $\mathcal{E}_{32}$ is now explored.

4.1.1 Local stability of the DFE ($\mathcal{E}_{32}$)

The local stability of the DFE, $\mathcal{E}_{32}$ (for the case when $N_0 > 1$) can be established using the next generation operator method on the system given by model (5). The matrices F (for the new infection terms) and V (of the transition terms) are respectively, given by

$$F = \begin{pmatrix} 0 & 0 & \beta_{HV} & \eta_1\beta_{HV} \\ 0 & 0 & 0 & 0 \\ \beta_{HV}\frac{F_N^*}{N_H^*} & 0 & 0 & 0 \\ \beta_{HV}\frac{F_S^*}{N_H^*} & 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} K_1 & 0 & 0 & 0 \\ -\gamma_H & \mu_H & 0 & 0 \\ 0 & 0 & \mu_V & 0 \\ 0 & 0 & 0 & \mu_V \end{pmatrix}.$$

Following [45], the basic reproduction number of the Zika model (5) about $\mathcal{E}_{32}$, with the forces of infection given by (16) (and $N_0 > 1$) is

$$\mathcal{R}_1 = \rho(FV^{-1}) = \sqrt{\frac{\beta_{HV}^2 b_V \alpha r K_2 (N_0 - 1)\big[\theta\eta_1 + (1-\theta)\big]}{N_H^* K_1 K_3 \mu_V^2 \mu}}. \tag{20}$$

Lemma 1 *The DFE ($\mathcal{E}_{32}$), of the model (5) with (16) (and $N_0 > 1$) is locally-asymptotically stable (LAS) if $\mathcal{R}_1 < 1$, and unstable if $\mathcal{R}_1 > 1$ [45].*

The epidemiological implication of Lemma 2 is that, there will not be a disease outbreak for a small influx of infectious individuals in the community if $\mathcal{R}_1 < 1$, and therefore the disease eventually dies out.

4.2 Interpretation of $\mathcal{R}_1$

In the absence of direct transmission (when $\beta_{HH} = 0$), the threshold quantity ($\mathcal{R}_1$) is defined as the expected number of secondary cases generated by an infected case introduced into a completely susceptible population. It can be interpreted as follows. Susceptible humans can acquire infection following effective contact with infectious mosquitoes (in F_{NI} or F_{SI} classes). The number of human infections generated by mosquitoes in the F_{NI} class (near the DFE) is given by the product of the infection rate of infectious mosquitoes in the F_{NI} class ($\frac{\beta_{HV}}{N_H^*} = \frac{\beta_{HV}\mu_H}{b_H}$), the average duration in the F_{NI} class ($\frac{1}{\mu_V}$), and the probability that a female mosquito survives the fertilized non-sterilized class (F_N) and move to the F_{NI} compartment ($\frac{\alpha(1-\theta)}{\mu_V}$). This gives (noting that $S_H^* = \frac{b_H}{\mu_H}$)

$$\frac{\beta_{HV}\mu_H\alpha(1-\theta)}{b_H\mu_V^2}S_H^* = \frac{\beta_{HV}\alpha(1-\theta)}{\mu_V^2}. \tag{21}$$

Similarly, the number of human infections generated by infectious mosquitoes in the F_{SI} class (near the DFE) is given by the product of the infection rate of mosquitoes in the F_{SI} class ($\frac{\beta_{HV}\eta_1}{N_H^*} = \frac{\beta_{HV}\eta_1\mu_H}{b_H}$), the average duration in the F_{SI} class ($\frac{1}{\mu_V}$), and the probability that a female mosquito survives the fertilized sterilized class (F_S) and move to F_{SI} compartment ($\frac{\alpha\theta}{\mu_V}$), so that

$$\frac{\beta_{HV}\mu_H\alpha\theta\eta_1}{b_H\mu_V^2}S_H^* = \frac{\beta_{HV}\alpha\theta\eta_1}{\mu_V^2}. \tag{22}$$

Therefore, the sum of (21) and (22) gives the average number of new human infections generated by infectious mosquito (sterilized or non-sterilized). This gives

$$\mathcal{R}_{VH} = \frac{\beta_{HV}\alpha(1-\theta)}{\mu_V^2} + \frac{\beta_{HV}\alpha\theta\eta_1}{\mu_V^2} = \frac{\beta_{HV}\alpha\big[\theta\eta_1 + (1-\theta)\big]}{\mu_V^2}. \tag{23}$$

The number of mosquitoes infection generated by infectious human (near the DFE), is given by the product of infection rate of infectious humans ($\frac{\beta_{HV}}{N_H^*} = \frac{\beta_{HV}\mu_H}{b_H}$), and the average duration of humans in the infectious class $\frac{1}{K_1}$, so that (with $Y^* = \frac{A^* b_V r}{K_3}$)

$$\mathcal{R}_{HV} = \frac{\beta_{HV}}{N_H^* K_1}Y^* = \frac{\beta_{HV} b_V \mu_H r}{K_1 K_3 b_H}A^*. \tag{24}$$

The geometric mean of (23) and (24) gives the associated reproduction number (noting that $A^* = \frac{K_2(N_0-1)}{\mu} > 0$)

$$\mathcal{R}_1 = \sqrt{\frac{\beta_{HV}^2 b_V \mu_H \alpha r K_2 (N_0-1)\big[\theta\eta_1 + (1-\theta)\big]}{b_H K_1 K_3 \mu_V^2 \mu}} = \sqrt{\mathcal{R}_{HV}\mathcal{R}_{VH}},$$

where the quantities $\mathcal{R}_{HV}$ and $\mathcal{R}_{VH}$ are the reproduction thresholds associated with Zika transmission from human to mosquitoes and from mosquito to humans, respectively.

4.3 Endemic equilibrium and backward bifurcation

Let,

$$\mathcal{E}_4 = \big(S_H^{**}, I_H^{**}, R_H^{**}, A^{**}, Y^{**}, F_N^{**}, F_S^{**}, F_{NI}^{**}, F_{SI}^{**}, M_N^{**}\big) \tag{25}$$

represents an arbitrary positive endemic equilibrium point of the model (5) in the absence of human-human transmission. Furthermore, let

$$\lambda_H^{**} = \frac{\beta_{HV}F_{NI}^{**} + \beta_{HV}\eta_1 F_{SI}^{**}}{S_H^{**} + I_H^{**} + R_H^{**}} \quad \text{and} \quad \lambda_V^{**} = \beta_{HV}\frac{I_H^{**}}{S_H^{**} + I_H^{**} + R_H^{**}}. \tag{26}$$

be the associated forces of infections at steady-state. Solving the equations of model (5) at steady state gives

$$\begin{aligned}
&S_H^{**} = \frac{b_H}{\lambda_H^{**}+\mu_H}, \quad I_H^{**} = \frac{\lambda_H^{**} b_H}{K_1(\lambda_H^{**}+\mu_H)}, \quad R_H^{**} = \frac{\lambda_H^{**} b_H \gamma_H}{K_1\mu_H(\lambda_H^{**}+\mu_H)},\\
&A^{**} = \frac{K_2}{\mu}\big(N_0-1\big), \quad Y^{**} = \frac{rb_V K_2}{K_3\mu}\big(N_0-1\big), \quad F_N^{**} = \frac{b_V r\alpha(1-\theta)K_2}{K_3\mu(\lambda_V^{**}+\mu_V)}\big(N_0-1\big),\\
&F_S^{**} = \frac{b_V r\alpha\theta K_2}{K_3\mu(\lambda_V^{**}+\mu_V)}\big(N_0-1\big), \quad F_{NI}^{**} = \frac{b_V\lambda_V r\alpha(1-\theta)K_2}{K_3\mu_V\mu(\lambda_V^{**}+\mu_V)}\big(N_0-1\big),\\
&F_{SI}^{**} = \frac{b_V\lambda_V r\alpha\theta K_2}{K_3\mu_V\mu(\lambda_V^{**}+\mu_V)}\big(N_0-1\big), \quad M_N^{**} = \frac{(1-r)b_V K_2}{\mu_V\mu}\big(N_0-1\big).
\end{aligned} \tag{27}$$

Since the endemic equilibrium is dependent on λ_V^{**} and λ_H^{**}, it is imperative to find the possible roots of λ_H^{**}, which can be used to evaluate λ_V^{**}. This can be achieved by substituting $S_H^{**}, I_H^{**}, R_H^{**}, F_{NI}^{**}$ and F_{SI}^{**} from (27) in (26). After some algebraic simplification, it can be shown that, λ_H^{**} satisfies

$$a_0(\lambda_H^{**})^5 + a_1(\lambda_H^{**})^4 + a_2(\lambda_H^{**})^3 + a_3(\lambda_H^{**})^2 + a_4\lambda_H^{**} = 0, \tag{28}$$

where

$$\begin{aligned}
a_0 =&\beta_{HV} b_H^2\mu_H^2 K_1(\mu_H+\gamma_H) + b_H^2 K_1\mu_H\mu_V(\mu_H+\gamma_H)^2,\\
a_1 =&2\beta_{HV} b_H^2\mu_H^3 K_1(\mu_H+\gamma_H) + \beta_{HV} b_H^2\mu_H^3 K_1^2 +\\
&2b_H^2 K_1^2\mu_H^2\mu_V(\mu_H+\gamma_H) + 2b_H^2\mu_H^2 K_1\mu_V(\mu_H+\gamma_H)^2 - \mathcal{R}_1^2 K_1^3 b_H^2\mu_H^2\mu_V,\\
a_2 =&2\beta_{HV} b_H^2 K_1^2\mu_H^4 + \beta_{HV} b_H^2\mu_H^4 K_1(\mu_H+\gamma_H) +\\
&4b_H^2 K_1^2\mu_H^3\mu_V(\mu_H+\gamma_H) + b_H^2 K_1^3\mu_H^3\mu_V + b_H^2\mu_H^3 K_1\mu_V(\mu_H+\gamma_H)^2 - 3\mathcal{R}_1^2 K_1^3 b_H^2\mu_H^3\mu_V,\\
a_3 =&2K_1^3\mu_H^4 b_H^2\mu_V + K_1^2 b_H^2\mu_H^5\beta_{HV} + 2K_1^2 b_H^2\mu_H^4\mu_V(\mu_H+\gamma_H) - 3\mathcal{R}_1^2 K_1^3 b_H^2\mu_H^4\mu_V,\\
a_4 =&b_H^2 K_1^3\mu_H^5\mu_V(1-\mathcal{R}_1^2).
\end{aligned} \tag{29}$$

Clearly, $\lambda_H^{**} = 0$ is a root of (28), which corresponds to the DFE. Notice from (29) that $a_0 > 0$ and $a_4 > 0$ ($a_4 < 0$) whenever $\mathcal{R}_1 < 1$ ($\mathcal{R}_1 > 1$). Further, the signs of the remaining coefficients (a_1, a_2 and a_3) depend on the magnitude of the associated parameters, different possibilities can be obtained by permuting their signs as presented in Table 4.

Theorem 2 *The model* (5) *in the absence of human-human transmission has*
i) Unique endemic equilibrium if $\mathcal{R}_1 > 1$ *as in Cases 2, 4, 8 and 10 in Table 4.*
ii) Two or more endemic equilibrium if $\mathcal{R}_1 < 1$ *as in Cases 3, 5, 7, 9, 11, 13, and 15 in Table 4.*
iii) No endemic equilibrium if $\mathcal{R}_1 < 1$*, as in Case 1 in Table 4.*

Theorem 2 (Case (ii)) indicates the possibility of backward bifurcation (where the locally-asymptotically stable DFE co-exists with a locally-asymptotically stable endemic equilibrium when $\mathcal{R}_1 < 1$) in the model (5) (see, for instance, [26, 27, 28]). Furthermore, this is investigated using the center manifold theory below. We claim the following result.

Theorem 3 *The Zika model* (5) *in the absence of direct transmission undergoes backward bifurcation at* $\mathcal{R}_1 = 1$*, whenever the bifurcation coefficient denoted by* $\tilde{a}$ *given by* (B.3) *in Appendix B is positive.*

The proof is given in Appendix B. ■

The public health implication of backward bifurcation phenomenon of the model (5) is that the classical epidemiological requirement of having the reproduction number ($\mathcal{R}_1$) to be less than unity, while necessary is no longer sufficient for the effective control of the disease. In other words, the backward bifurcation property of the model (5) makes effective Zika control difficult. Further, as a consequence, it is instructive to try to determine the cause of the backward bifurcation phenomenon in the model (5). This is explored below.

4.4 Non-existence of Backward bifurcation

Consider the model (5) with Zika induced death assumed to be negligible (obtained by setting $\delta_H = 0$) so that, K_1 reduces to $\mu_H + \gamma_H$, thus we have the coefficients in (29) reduces to

$$\begin{aligned} a_0 =& \beta_{HV} b_H^2 \mu_H^2 K_1^2 + b_H^2 K_1^3 \mu_H \mu_V, \\ a_1 =& 3\beta_{HV} b_H^2 \mu_H^3 K_1^2 + K_1^3 b_H^2 \mu_H^2 \mu_V (4 - \mathcal{R}_1^2), \quad a_2 = 3\beta_{HV} b_H^2 K_1^2 \mu_H^4 + 3K_1^3 b_H^2 \mu_H^3 \mu_V (2 - \mathcal{R}_1^2) \\ a_3 =& \beta_{HV} K_1^2 b_H^2 \mu_H^5 + K_1^3 b_H^2 \mu_H^4 \mu_V (4 - 3\mathcal{R}_1^2), \quad a_4 = b_H^2 K_1^3 \mu_H^5 \mu_V (1 - \mathcal{R}_1^2). \end{aligned} \tag{30}$$

Clearly $a_0 > 0$, the signs of a_1, a_2, a_3, and a_4 depend on the magnitude of $\mathcal{R}_1$. Noticed that, if $\mathcal{R}_1 \leq 1$, there is no sign change, hence, by Routh-Hurwitz criterion, there is no endemic equilibrium whenever $\mathcal{R}_1 \leq 1$.

Lemma 2 *The Zika model in the absence of direct transmission given by* (5)*, with* $\delta_H = 0$ *has no endemic equilibrium whenever* $\mathcal{R}_1 \leq 1$*.*

The epidemiological implication of Lemma 2 is that the Zika model without direct transmission given by (5), with $\delta_H = 0$ does not undergo backward bifurcation (since the occurrence of backward bifurcation requires the existence of at least two equilibria when $\mathcal{R}_1 < 1$).

Furthermore, it is worth noticing that, substituting $\delta_H = 0$ in the expression for the bifurcation coefficient $\tilde{\mathbf{a}}$, given by equation (B.3) in Appendix B reduces $\tilde{\mathbf{a}}$ reduces to

$$\tilde{\mathbf{a}} = \frac{-2w_2^2 v_2}{N_H^*} \Big[\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}K_1^2}{\mu_H}\big(2 - \mathcal{R}_{HV}\mathcal{R}_{VH}\big) + \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}K_1\beta_{HV}}{\mu_V} \Big] < 0, \tag{31}$$

provided $\mathcal{R}_1 \leq 1$. Thus, it follows from Theorem 4.1 of [8] that, the model (5) does not undergoes backward bifurcation if the disease induced death rate is negligible. This result is similar to that obtained numerically by Chitnis et al [9] in their malaria model.

The Zika model with both direct (human-human) and indirect (human-mosquito-human) transmission is now analysed for its dynamical features. The aim is to find out if incorporating direct transmission will change the dynamics of the disease.

5 Analysis of the model with direct transmission

In this section, we consider the full Zika model in the presence of human-human transmission (i.e with the forces of infection given by (1) and (4)).

5.1 Disease-free equilibrium

The model (5) (with direct transmission) has two disease-free equilibria given by:

$$\mathcal{E}_5 = \big(S_H^*, I_H^*, R_H^*, A^*, Y^*, F_N^*, F_S^*, F_{NI}^*, F_{SI}^*, M_N^*\big) = \left(\frac{b_H}{\mu_H}, 0, 0, 0, 0, 0, 0, 0, 0, 0\right), \tag{32}$$

which occurs when $N_0 \leq 1$, and

$$\begin{aligned} \mathcal{E}_6 =& \Big(S_H^*, I_H^*, R_H^*, A^*, Y^*, F_N^*, F_S^*, F_{NI}^*, F_{SI}^*, M_N^*\Big) = \Big(\frac{b_H}{\mu_H}, 0, 0, \frac{K_2}{\mu}(N_0 - 1), \\ & \frac{rb_V K_2(N_0-1)}{K_3\mu}, \frac{(1-\theta)\alpha r b_V K_2(N_0-1)}{K_3\mu_V\mu}, \frac{\theta\alpha r b_V K_2(N_0-1)}{K_3\mu_V\mu}, 0, 0, \frac{(1-r)b_V K_2(N_0-1)}{K_3\mu_V\mu}\Big), \end{aligned} \tag{33}$$

which is obtained when $N_0 > 1$.

5.2 Local stability of $\mathcal{E}_6$

The local stability of the DFE, $\mathcal{E}_6$, is established using the next generation method on model (5). The F and V matrices about $\mathcal{E}_5$ are respectively given by

$$F = \begin{pmatrix} \beta_{HH} & \eta_2\beta_{HH} & \beta_{HV} & \eta_1\beta_{HV} \\ 0 & 0 & 0 & 0 \\ \beta_{HV}\frac{F_N^*}{N_H^*} & 0 & 0 & 0 \\ \beta_{HV}\frac{F_S^*}{N_H^*} & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} K_1 & 0 & 0 & 0 \\ -\gamma_H & \mu_H & 0 & 0 \\ 0 & 0 & \mu_V & 0 \\ 0 & 0 & 0 & \mu_V \end{pmatrix}.$$

Following [45], the associated reproduction number of the system model (5) (with $N_0 > 1$) denoted by $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \frac{\beta_{HH}\big(\eta_2\gamma_H + \mu_H\big)}{2K_1\mu_H} + \sqrt{\Big[\frac{\beta_{HH}\big(\eta_2\gamma_H + \mu_H\big)}{2K_1\mu_H}\Big]^2 + \frac{\beta_{HV}^2 b_V r\alpha\mu_H K_2(N_0-1)\big[\theta\eta_1 + (1-\theta)\big]}{K_1K_3b_H\mu_V^2\mu}}.$$

$$= \frac{1}{2}\left(\mathcal{R}_{HH} + \sqrt{\mathcal{R}_{HH}^2 + 4\mathcal{R}_{VH}\mathcal{R}_{HV}}\right).$$

where, $\mathcal{R}_{HH}$ is the threshold quantity associated with the direct (human-to-human) transmissions.

Lemma 3 *The disease-free equilibrium ($\mathcal{E}_6$), of model (5) with (1), (4) and $N_0 > 1$ is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$ [45].*

The threshold quantity $\mathcal{R}_{HH}$ can be interpreted as follows. The number of new human-human infections (via sexual transmission), generated by an infectious human (I_H) (near the DFE) is given by the product of the infection rate of infectious human ($\frac{\beta_{HH}S_H^*}{N_H^*}$), and the average duration in the infectious class ($\frac{1}{K_1}$), this gives

$$\frac{\beta_{HH}}{K_1}. \tag{34}$$

Similarly, the number of new human infections generated by humans in the R_H class (near the DFE), is given by the product of the infection rate of infectious human ($\frac{\beta_{HH}\eta_2 S_H^*}{N_H^*}$), the probability that human survives the infectious class I_H and move to recovered class ($\frac{\gamma_H}{K_1}$), and the average duration in the recovered class ($\frac{1}{\mu_H}$), this gives

$$\frac{\beta_{HH}\eta_2\gamma_H}{K_1\mu_H}. \tag{35}$$

Hence, the sum of (34) and (35) gives the threshold quantity associated with the human-human Zika transmissions

$$\mathcal{R}_{HH} = \frac{\beta_{HH}}{K_1} + \frac{\beta_{HH}\eta_2\gamma_H}{K_1\mu_H} = \frac{\beta_{HH}\big(\mu_H + \eta_2\gamma_H\big)}{K_1\mu_H}. \tag{36}$$

Notice that in the absence of direct Zika transmission, $\mathcal{R}_0 = \mathcal{R}_1$. This result is consistent with those obtained in Brauer et al [6] and Chitnis et al [11] for epidemic model of vector borne diseases with both direct and indirect transmissions.

5.3 Endemic equilibrium and backward bifurcation

Let

$$\mathcal{E}_7 = \big(S_H^{***}, I_H^{***}, R_H^{***}, A^{***}, Y^{***}, F_N^{***}, F_S^{***}, F_{NI}^{***}, F_{SI}^{***}, M_N^{***}\big), \tag{37}$$

represents an arbitrary positive endemic equilibrium point (EE) of the model (5). Furthermore, let

$$\lambda_H^{***} = \frac{\beta_{HV}(F_{NI}^{***} + \eta_1 F_{SI}^{***}) + \beta_{HH}(I_H^{***} + \eta_2 R_H^{***})}{S_H^{***} + I_H^{***} + R_H^{***}}, \quad \lambda_V^{***} = \beta_{HV}\frac{I_H^{***}}{S_H^{***} + I_H^{***} + R_H^{***}}$$

be the associated forces of infections at the steady-state. Solving equations of model (5) at the steady-state (with $N_0 > 1$) gives

$$\begin{aligned}
&S_H^{***} = \frac{b_H}{\lambda_H^{***} + \mu_H}, \quad I_H^{***} = \frac{\lambda_H^{***} b_H}{K_1(\lambda_H^{***} + \mu_H)}, \quad R_H^{***} = \frac{\lambda_H^{***} b_H \gamma_H}{K_1 \mu_H (\lambda_H^{***} + \mu_H)},\\
&A^{***} = \frac{K_2}{\mu}\big(N_0 - 1\big), \quad Y^{***} = \frac{r b_V K_2}{K_3 \mu}\big(N_0 - 1\big), \quad F_N^{***} = \frac{b_V r\alpha(1-\theta)K_2}{K_3\mu(\lambda_V^{***} + \mu_V)}\big(N_0 - 1\big),\\
&F_S^{***} = \frac{b_V r\alpha\theta K_2}{K_3\mu(\lambda_V^{***} + \mu_V)}\big(N_0 - 1\big), \quad F_{NI}^{***} = \frac{b_V \lambda_V^{***} r\alpha(1-\theta)K_2}{K_3\mu_V\mu(\lambda_V^{***} + \mu_V)}\big(N_0 - 1\big)\\
&F_{SI}^{***} = \frac{b_V \lambda_V^{***} r\alpha\theta K_2}{K_3\mu_V\mu(\lambda_V^{***} + \mu_V)}\big(N_0 - 1\big), \quad M_N^{***} = \frac{(1-r)b_V K_2}{\mu_V \mu}\big(N_0 - 1\big).
\end{aligned} \tag{38}$$

we claim the following result.

Theorem 4 *The Zika model* (5) *with direct transmission undergoes backward bifurcation at* $\mathcal{R}_0 = 1$*, whenever the bifurcation coefficient denoted by* $\tilde{\boldsymbol{a}}_2$ *given by equation* (39) *is positive.*

Proof. Using similar approach as in the proof of Theorem 3. It can be shown that the associated bifurcation coefficient $\tilde{\mathbf{a}}$, is now given by

$$\begin{aligned}
\tilde{\mathbf{a}}_2 = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) = \frac{-2K_1 w_2^2 v_2}{N_H^*}\Big[\frac{\mathcal{R}_{HH}\big(\gamma_H + \mu_H\big)}{\mu_H} +\\
\frac{2\mathcal{R}_{HV}\mathcal{R}_{VH}\big(\gamma_H + \mu_H\big)}{\mu_H} + \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}\beta_{HV}}{\mu_V} - K_1\Big(\frac{\mathcal{R}_{HH}\mathcal{R}_{VH}\mathcal{R}_{HV}}{\mu_H} + \frac{\mathcal{R}_{HV}^2\mathcal{R}_{VH}^2}{\mu_H}\Big)\Big]
\end{aligned} \tag{39}$$

and

$$\tilde{\mathbf{b}}_2 = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi_V}(0,0) = \frac{2\beta_{HV} v_2 w_2 K_2 b_V (N_0 - 1) r\alpha\Big([1-\theta] + \eta_1\theta\Big)}{N_H^* K_3 \mu_V^3 \mu} > 0. \tag{40}$$

■

Notice that if $\delta_H = 0$, then K_1 reduces to $\gamma_H + \mu_H$ and $\tilde{\mathbf{a}}_2$ reduces to

$$\tilde{\mathbf{a}}_2 = \frac{-2w_2^2 v_2}{N_H^*}\Big[\frac{\mathcal{R}_{HH}K_1^2}{\mu_H}\big(1 - \mathcal{R}_{HV}\mathcal{R}_{VH}\big) + \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}K_1^2}{\mu_H}\big(2 - \mathcal{R}_{HV}\mathcal{R}_{VH}\big) + \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}K_1\beta_{HV}}{\mu_V}\Big] < 0, \tag{41}$$

provided $\mathcal{R}_0 \leq 1$.

Lemma 4 *The Zika model* (5) *does not undergoes backward bifurcation at* $\mathcal{R}_0 = 1$ *if* $\delta_H = 0$.

Thus, as in Section 4, this result completely rules out the existence of backward bifurcation when $\delta_H = 0$.

6 Sensitivity analysis

Sensitivity analysis is a tool used in studying the variation of an output of a model due to change in the input parameters. We perform both local sensitivity analysis (where all other parameters are held at a certain baseline) for the basic reproduction number ($\mathcal{R}_0$), and global sensitivity analysis, where a multidimensional parameter space is studied globally [36].

6.1 Local sensitivity analysis of $\mathcal{R}_0$ with respect to model parameters

The basic reproduction number ($\mathcal{R}_0$) is used to measure the potential impact of a disease. Using elasticity index, we perform local sensitivity analysis of the parameters of $\mathcal{R}_0$. The method is used to measure the percentage change of a parameter say α, with respect to a percentage change of a quantity say $R(\alpha)$. The normalized sensitivity index (elasticity indices) of $R(\alpha)$ with respect to α is [10],

$$\Upsilon_{\alpha}^{R} = \frac{\partial R}{\partial \alpha} \times \frac{\alpha}{R}.$$

Using the parameter values in Table 2, we give the sensitivity index of the parameters for low and high baseline values in Table 3. For both low and high transmission regions, $\mathcal{R}_0$ is most negatively correlated to μ_V, where $\Upsilon_{\mu_V}^{\mathcal{R}_0} = -0.71355$ in low region and $\Upsilon_{\mu_V}^{\mathcal{R}_0} = -0.66732$ in high region, both are followed by b_H and μ (note that $\Upsilon_{b_H}^{\mathcal{R}_0} = \Upsilon_{\mu}^{\mathcal{R}_0}$). Similarly, $\mathcal{R}_0$ is most positively correlated to r then b_V and β_{HH} in both regions. Given that

$$\mathcal{R}_0 = \frac{\mathcal{R}_{HH}}{2} + \sqrt{\left(\frac{\mathcal{R}_{HH}}{2}\right)^2 + \mathcal{R}_{HV}\mathcal{R}_{VH}} \tag{42}$$

where $\mathcal{R}_{HH}$, $\mathcal{R}_{VH}$ and $\mathcal{R}_{HV}$ are as defined in (36), (23) and (24) respectively. The computation of sensitivity index is presented in Appendix C.

6.2 Global sensitivity analysis

Unlike local sensitivity analysis, global sensitivity analysis allows other parameters to vary as the effect of a certain parameter is estimated. Using ranges and baseline values in Table 2 (high baseline), the partial rank correlation coefficient (PRCC) of the model parameters were computed and presented in Figure 8. Total infectious humans ($I_H + R_H$) is taken as the output function. Other parameters considered are defined as $k_2 = b_V + \mu_V$ and $k_3 = \alpha + \mu_V$. Also $k_4 = \alpha\theta$ and $k_8 = \alpha(1-\theta)$ are the rates of fertilization of F_N and F_S compartments, respectively, $k_5 = (1-r)b_v$ and $k_9 = rb_V$ are respectively the rates of maturation to M_N and Y compartments. Input parameters were sampled using Latin Hypercube Sampling (LHS) method (a statistical method for generating a sample of plausible collections of parameter values from a multidimensional distribution), and a total of 1000 simulations were ran. The value of the PRCC in Figure 8 gives the correlation between the parameters and the chosen output ($I_H + R_H$). The parameters with large PRCC values are considered to be the most important (in determining the value/size of the chosen response function). The figures shows that the total infectious humans is most positively correlated to b_H and negatively correlated with ϕ_V, γ_H and k_5 thus they can be targeted in reducing the number of infectious humans.
The Scatter plots of the most sensitive parameters (that is b_H, ϕ_V, γ_H and k_5) are presented in Figure 9. The vertical axis represent the residual of the linear regression between the rank transformed values of the parameters b_H, ϕ_V, γ_H and k_5 and the transformed values of other parameters. The ordinate gives the residual of the linear regression between the rank-transformed values of the output function ($I_H + R_H$) and the transformed values of all other parameters.
The value of the PRCC of the threshold parameters $\mathcal{R}_1$ and $\mathcal{R}_0$ are given in Figures 10 and 11, respectively. In either case, μ_V is the most negatively correlated parameter to the threshold quantities, followed by θ, μ and b_H. Thus, this sensitivity study shows the significance of θ in controlling both $\mathcal{R}_1$ and $\mathcal{R}_0$.

7 Numerical simulations

In this section, the Zika model (5) is simulated using parameter values in Table 2, this is aimed at illustrating some of the established analytical results in the previous sections. Two different set of parameter values are used, the low baseline values give $\mathcal{R}_0 = 0.2461 < 1$, while the high baseline values give $\mathcal{R}_0 = 4.3250 > 1$. Different simulations were obtained using both low and high baseline parameter values for comparison purposes. Initial conditions used through out our simulations are $S_H(0) = 600$, $I_H(0) = 20$, $R_H(0) = 0$, $A(0) = 2400$, $Y(0) = 500$, $F_N(0) = 300$, $F_S(0) = 100$, $F_{NI}(0) = 100$, $F_{SI}(0) = 50$, and $M_N(0) = 150$.
Figures 2 and 3 depict population of infected humans with different initial conditions. Figure 2 shows the convergence of solution profile to the disease-free equilibrium when $\mathcal{R}_0 = 0.2461 < 1$ and Figure 3 shows the convergence of solutions to a non zero equilibrium (endemic equilibrium) when $\mathcal{R}_0 = 4.3250 > 1$. The solution profile of the model (5) showing cumulative number of new Zika cases in humans, with different values of θ (the probability of a female mosquito mating with a sterile male mosquito) is illustrated in Figure 4. The figure shows how increase in the value of θ can drastically reduce the cumulative new human cases. As such, introduction and successful mating of female mosquitoes with sterile male mosquitoes is negatively correlated to new human cases. In Figure 5, the effect of θ on the population of reproductive mosquitoes is shown, as the value of θ increases, total reproductive mosquitoes is reduced.
Figures 6 and 7 give a comparison between solution profile of the model showing total number of adult mosquitoes with varying values of ϕ_V and θ respectively, both have positive effect in reducing the size of adult mosquito population, although θ can be controlled (through increase in the release of sterile male mosquitoes at the right location), ϕ_V is not easily controlled.

Conclusion

In this paper, we design a new deterministic model for the transmission dynamics of Zika in a population consisting of humans and mosquitoes. The model which adopts a standard incidence formulation incorporates the aquatic stage of mosquito development and mosquito sterilization. Some of the key findings of the study are as follows.

1. The mosquito extinction equilibrium, $\mathcal{E}_0$, is shown to be globally-asymptotically stable when the associated threshold quantity (N_0) called the basic offspring number is less than unity.

2. An increase in the mating rate of sterilized mosquitoes, could be sufficient to bring the value of N_0 to value less than unity, there by decreases the mosquito population.

3. The model (with $N_0 > 1$) in the absence of direct transmission undergoes backward bifurcation, where the stable DFE co-exist with a stable endemic equilibrium when the associated reproduction number is less than unity. This study identifies a sufficient condition for the emergence of backward bifurcation in the model, namely disease induced death in humans ($\delta_H = 0$).

4. Similarly, the model with direct transmission also undergoes backward bifurcation at $\mathcal{R}_0 = 1$. The backward bifurcation property can be removed when the Zika-induced mortality in humans is negligible ($\delta_H = 0$). Thus, the major parameter responsible for backward bifurcation in both models (with and without direct transmission) is the disease-induced mortality in humans. This result is similarly shown by Garba et al [26] for dengue model and numerically for a Malaria model by Chitnis et al [9].

5. The DFE of both models (with and without direct transmissions) in the presence of mosquito population (when $N_0 > 1$) are shown to be locally-asymptotically stable when the associated reproduction numbers are less than unity.

6. The two models exhibit the same qualitative dynamics with respect to the local stability of the associated disease-free equilibrium and backward bifurcation phenomenon.

7. Using elasticity index (local sensitivity analysis), it is shown that, the most effective parameter for the control of the basic reproduction number in both areas of high and low transmission is mosquito death rate.

Acknowledgement

The authors acknowledges with thanks the support of South African DST/NRF SARChI chair on Mathematical Models and Methods in Bioengineering and Biosciences (M^3B^2) and DST-NRF Centre of Excellence in Mathematical and Statistical Sciences (CoE-MaSS). The authors are grateful to the anonymous reviewers and handling editor for their constructive comments.

Appendix A: Proof of Theorem 1

Proof. In this appendix, we shall give the proof of the first part of Theorem 1 (GAS of $\mathcal{E}_1$) using similar approach to that in [3]. In particular Theorem 6 of [3], reproduced below for convenience will be used.
Consider $\dot{x} = f(x)$, where $D \subseteq \mathbb{R}^n$ and $f : D \longrightarrow \mathbb{R}^n$ is continuous. Then we have the following result.

Theorem 5 *[3] Let $a, b \in D$ be such that $a < b$, $[a, b] \subseteq D$ and $f(b) \leq 0 \leq f(a)$. Then $\dot{x} = f(x)$ defines a (positive) dynamical system on $[a, b]$. Moreover, if $[a, b]$ contains a unique equilibrium q then q is globally asymptotically stable on $[a, b]$.*

To apply Theorem (5) to system (7), let $p \in \mathbb{R}_+ > \frac{3(\mu_V+b_V)}{\mu}$ and A_p be chosen so large such that

$$\begin{aligned}
A_p &\geq p,\\
F_{N_p} &= \frac{\mu_V + b_V + \mu A_p}{2\phi_V} A_p \geq p,\\
Y_p &= \frac{\mu_V F_{N_p}}{2\alpha(1-\theta)} = \frac{\mu_V(\mu_V + b_V + \mu A_p)}{4\phi_V \alpha(1-\theta)} A_p \geq p,\\
F_{S_p} &= \frac{\theta F_{N_p}}{(1-\theta)} = \frac{\theta(\mu_V + b_V + \mu A_p)}{2\phi_V(1-\theta)} A_p \geq p,\\
M_p &= \frac{2b_v(1-r)A_p}{\mu_V} \geq p.
\end{aligned} \tag{A.1}$$

Further, let $b_p = (A_p, Y_p, F_{N_p}, F_{S_p}, M_p)^T$, and consider the interval $[0, b_p] \in \mathbb{R}^5_+$. Then

$$f(b_p) = \begin{pmatrix} -\frac{(\mu_V+b_V+\mu A_p)}{2} A_p \\ rb_V A_p\left(1 - \frac{\mu_V+b_V+\mu A_p}{4N_0(\mu_V+b_V)}\right) \\ -\mu_V F_p \\ -\frac{\mu_V \phi_V F_{N_p}}{2(1-\theta)} \\ -(1-r)b_v A_p \end{pmatrix} \leq \begin{pmatrix} -\frac{(\mu_V+b_V+\mu A_p)}{2} A_p \\ rb_V A_p\left(1 - \frac{1}{N_0}\right) \\ -\mu_V F_p \\ -\frac{\mu_V \phi_V F_{N_p}}{2(1-\theta)} \\ -(1-r)b_v A_p \end{pmatrix} < 0, \text{ provided } N_0 \leq 1.$$

Therefore in the interval $[a, b] = [0, b_p] \in \mathbb{R}^5_+$, the condition $f(b) \leq 0 \leq f(a) = f(b_p) \leq 0 \leq f(0)$ is

satisfied. However since p is arbitrary, then b_p can be selected larger than any $x \in \mathbb{R}^5_+$. Thus the system defines a positive dynamical system on $\mathbb{R}^5_+$. Moreover if $N_0 \leq 1$, $\mathcal{E}_1$ is unique in $[0, b_p]$ and thus, $\mathcal{E}_1$ is globally asymptotically stable.

For local stability of the non-zero equilibrium $\mathcal{E}_2$, we use the property of eigenvalues of the Jacobian matrix J_1 below.

$$J_1 = \begin{pmatrix} -P_1 & 0 & \phi_V & 0 & 0 \\ rb_V & -K_3 & 0 & 0 & 0 \\ 0 & P_2 & -\mu_V & 0 & 0 \\ 0 & \theta\alpha & 0 & -\mu_V & 0 \\ (1-r)b_V & 0 & 0 & 0 & -\mu_V \end{pmatrix},$$

where $P_1 = 2\mu A^* + \mu_V + b_V = 2K_2(N_0 - 1) + \mu_V + b_V$, and $P_2 = (1-\theta)\alpha$. Notice that the system given by (7) is cooperative on $\mathbb{R}^5_+$, that is, growth in any compartment has positive effect on the growth of other compartments. Equivalently, a system is cooperative if the non-diagonal elements of its Jacobian matrix are non-negative.

Clearly, $-\mu_V$ is an eigenvalue of J_1. The remaining eigenvalues satisfy

$$\lambda^3 + \lambda^2(P_1 + K_3 + \mu_V) + \lambda(K_3\mu_V + P_1K_3 + P_1\mu_V) + P_1K_3\mu_V\big(1 - \frac{b_V\phi_V\alpha r(1-\theta)}{K_3\mu_V(K_2 + 2\mu A^*)}\big) = 0 \quad \text{(A.2)}$$

Applying Routh-Hurwitz criterion and Lienard-Chipart test [33], the roots of a polynomial of degree three are negative if and only if $a_i > 0$ with $i = 0, 1, 2, 3$ and $a_1a_2 - a_3 > 0$.
Its clear from (A.2) that a_0, a_1 and a_2 are positive, while the sign of $a_3 = P_1K_3\mu_V\big(1 - \frac{N_0}{2N_0-1}\big)$ depends on N_0. Also,

$$a_1a_2 - a_3 = (K_3 + P_1 + \mu_V)(K_3\mu_V + P_1K_3 + P_1\mu_V) + P_2b_V\phi_V r - P_1K_3\mu_V > 0.$$

If $N_0 > 1$, then $a_3 > 0$. On the other hand $1 - \frac{N_0}{2N_0-1} < 0$ if and only if $N_0 < 1$, hence, $\mathcal{E}_2$ is locally asymptotically stable. ■

Appendix B: Proof of Theorem 4

Backward bifurcation analysis

Proof. To prove the existence of backward bifurcation for the model given by (5), a method, which is based on the Centre Manifold Theory [8, 45], is used. The following change of variables are made on the model given by (5). Let,

$$\big(S_H, I_H, R_H, A, Y, F_N, F_S, F_{NI}, F_{SI}, M_N\big) = \big(x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9, x_{10}\big),$$

and hence, the total human and mosquito populations are:

$$N_H = x_1 + x_2 + x_3 \quad \text{and} \quad N_V = x_4 + x_5 + x_6 + x_7 + x_8 + x_9 + x_{10}.$$

Using vector notation, we have,

$$X = \big(x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9, x_{10}\big)^T \quad \text{and} \quad \frac{dX}{dt} = \big(f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9, f_{10}\big)^T,$$

and therefore the transformed model (5) is represented by

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = b_H - \big(\frac{\beta_{HV}x_8 + \beta_{HV}\eta_1 x_9 + \beta_{HH}x_2 + \beta_{HH}\eta_2 x_3}{x_1+x_2+x_3}\big)x_1 - \mu_H x_1,\\
\frac{dx_2}{dt} &= f_2 = \big(\frac{\beta_{HV}x_8 + \beta_{HV}\eta_1 x_9 + \beta_{HH}x_2 + \beta_{HH}\eta_2 x_3}{x_1+x_2+x_3}\big)x_1 - x_2K_1,\\
\frac{dx_3}{dt} &= f_3 = \gamma_H x_2 - \mu_H x_3,\\
\frac{dx_4}{dt} &= f_4 = \phi_V x_6 + \phi_V x_8 - \mu x_4^2 - K_2 x_4,\\
\frac{dx_5}{dt} &= f_5 = rb_V x_4 - K_3 x_5,\\
\frac{dx_6}{dt} &= f_6 = \alpha(1-\theta)x_5 - \frac{\beta_{HV}x_2x_6}{x_1+x_2+x_3} - \mu_V x_6,\\
\frac{dx_7}{dt} &= f_7 = \alpha\theta x_5 - \frac{\beta_{HV}x_2x_7}{x_1+x_2+x_3} - \mu_V x_7,\\
\frac{dx_8}{dt} &= f_8 = \frac{\beta_{HV}x_2x_6}{x_1+x_2+x_3} - \mu_V x_8,\\
\frac{dx_9}{dt} &= f_9 = \frac{\beta_{HV}x_2x_7}{x_1+x_2+x_3} - \mu_V x_9,\\
\frac{dx_{10}}{dt} &= f_{10} = (1-r)b_V x_4 - \mu_V x_{10},
\end{aligned} \tag{B.1}$$

so that the forces of infection are given by

$$\lambda_H = \frac{\beta_{HV}x_8 + \beta_{HV}\eta_1 x_9}{x_1+x_2+x_3}x_1 \text{ and } \lambda_V = \frac{\beta_{HV}x_2}{x_1+x_2+x_3}.$$

Let β^*_{HV} be chosen as a bifurcation parameter obtained by solving for $\beta_{HV} = \beta^*_{HV}$, when $\mathcal{R}_0 = 1$, given by

$$\beta^*_{HV} = \sqrt{\frac{K_1 N^*_H}{F^*_N + \eta_1 F^*_S}}. \tag{B.2}$$

The Jacobian of the system (B.1), evaluated at the DFE, $\mathcal{E}_{32}$, is given by

$$J^* = \begin{pmatrix}
-\mu_H & 0 & 0 & 0 & 0 & 0 & 0 & -\beta^*_{HV} & -\beta^*_{HV}\eta_1 & 0\\
0 & -K_1 & 0 & 0 & 0 & 0 & 0 & \beta^*_{HV} & \beta^*_{HV}\eta_1 & 0\\
0 & \gamma_H & -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & -2\mu A^* - K_1 & 0 & \phi_V & 0 & \phi_V & 0 & 0\\
0 & 0 & 0 & rb_V & -K_3 & 0 & 0 & 0 & 0 & 0\\
0 & -\frac{\beta_{HV}F^*_N}{N^*_H} & 0 & 0 & \alpha(1-\theta) & -\mu_V & 0 & 0 & 0 & 0\\
0 & -\frac{\beta_{HV}F^*_S}{N^*_H} & 0 & 0 & \alpha\theta & 0 & -\mu_V & 0 & 0 & 0\\
0 & \frac{\beta_{HV}F^*_N}{N^*_H} & 0 & 0 & 0 & 0 & 0 & -\mu_V & 0 & 0\\
0 & \frac{\beta_{HV}F^*_S}{N^*_H} & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V & 0\\
0 & 0 & 0 & (1-r)b_V & 0 & 0 & 0 & 0 & 0 & -\mu_V
\end{pmatrix},$$

The Jacobian (J^*) of the linearised system has a simple zero eigenvalue (with all other eigenvalues having negative real part). Thus, the centre manifold theory can be used to analyse the dynamics of the system (B.1) around $\beta_{HV} = \beta^*_{HV}$.

For the case were equation (B.2) holds, the matrix J^* has left eigenvectors associated with zero eigenvalue given by $\mathbf{v} = [v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9, v_{10}]^T$

$$v_1 = 0, \quad v_2 = \frac{1}{\mu_H^2 N_H^* \mu_V^2 + \beta_{HV}^2 \mu_H^2 \big(F_N^* + \eta_1 F_S^*\big)}, \quad v_3 = 0, \quad v_4 = 0,$$
$$v_5 = 0, \quad v_6 = 0, \quad v_7 = 0, \quad v_8 = \frac{\beta_{HV} v_2}{\mu_V}, \quad v_9 = \frac{\beta_{HV}\eta_1 v_2}{\mu_V}, \quad v_{10} = 0,$$

and the right eigenvector (of the zero eigenvalue) denoted by $\mathbf{w} = [w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9, w_{10}]^T$ has elements given by:

$$w_1 = -\frac{K_1 w_2}{\mu_H} R_{HV} R_{VH} \quad w_2 = N_H^* \mu_H^2 \mu_V^2, \quad w_3 = \frac{\gamma_H w_2}{\mu_H}, \quad w_4 = 0, \quad w_5 = 0,$$
$$w_6 = -\frac{\beta_{HV} F_N^* w_2}{N_H^* \mu_V}, \quad w_7 = -\frac{\beta_{HV} F_S^* w_2}{N_H^* \mu_V}, \quad w_8 = \frac{\beta_{HV} F_N^* w_2}{N_H^* \mu_V}, \quad w_9 = \frac{\beta_{HV} F_S^* w_2}{N_H^* \mu_V}, \quad w_{10} = 0.$$

It can be shown, by computing the non-zero partial derivatives of the right-hand functions, that the associated backward bifurcation coefficients, $\tilde{a}$ and $\tilde{b}$, are respectively, given by (see Theorem 4.1 in [8])

$$\begin{aligned} \tilde{\mathbf{a}} &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) = \\ &\frac{-2K_1 w_2^2 v_2}{N_H^*}\Big[\mathcal{R}_{HV}\mathcal{R}_{VH}\Big(\frac{2\big(\gamma_H + \mu_H\big)}{\mu_H} + \frac{\beta_{HV}}{\mu_V}\Big) - \frac{K_1 \mathcal{R}_{HV}^2 \mathcal{R}_{VH}^2}{\mu_H}\Big] \end{aligned} \tag{B.3}$$

and

$$\tilde{\mathbf{b}} = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi_V}(0,0) = \frac{\beta_{HV} v_2 w_2 K_2 b_V (N_0 - 1) r\alpha\Big(\big[1-\theta\big] + \eta_1 \theta\Big)}{N_H^* K_3 \mu_V^2 \mu} > 0 \tag{B.4}$$

Since the bifurcation coefficient, b is positive, it follows from Theorem 4.1 in [8] that the Zika model (or its transform equivalent (29)) will undergo backward bifurcation if the bifurcation coefficient, $\tilde{a}$, given by (B.3), is positive. ■

Appendix C: Computing local sensitivity index

The sensitivity index of the parameters are given as follows:

$$
\begin{aligned}
\Upsilon^{\mathcal{R}_0}_{\beta_{HV}} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{\mathcal{R}_0\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{b_H} &= -\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{2\mathcal{R}_0\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{r} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}(2N_0-1)}{2\mathcal{R}_0(N_0-1)\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\mu} &= -\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{2\mathcal{R}_0\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\beta_{HH}} &= \frac{\mathcal{R}_{HH}}{2\mathcal{R}_0}\Big(1+\frac{\mathcal{R}_{HH}}{2\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}\Big),\\
\Upsilon^{\mathcal{R}_0}_{\phi_V} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}N_0}{2\mathcal{R}_0(N_0-1)\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\alpha} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}N_0(K_3-1)^2}{2\mathcal{R}_0K_3^2\alpha(N_0-1)\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\eta_2} &= \frac{\beta_{HH}\eta_2\gamma_H}{2\mathcal{R}_0K_1\mu_H}\Big(1+\frac{\mathcal{R}_{HH}}{2\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}\Big),\\
\Upsilon^{\mathcal{R}_0}_{\eta_1} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}\theta\eta_1}{2\mathcal{R}_0\Big[\theta\eta_1+(1-\theta)\Big]\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\mu_V} &= -\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}\mu_V\Big[K_3+K_2(N_0-1)\Big]}{2K_2K_3\mathcal{R}_0(N_0-1)\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{b_V} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}}{2K_2(N_0-1)\mathcal{R}_0\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}\big(2N_0K_2-2b_V-\mu_V\big),\\
\Upsilon^{\mathcal{R}_0}_{\delta_H} &= -\frac{\mathcal{R}_{HH}\delta_H}{2\mathcal{R}_0K_1}\Big(1+\frac{\mathcal{R}_{HH}}{2\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}\Big)-\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}\delta_H}{2\mathcal{R}_0K_1\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\gamma_H} &= \frac{\beta_{HH}\eta_2\gamma_H}{2\mathcal{R}_0K_1\mu_H}\Big(1+\frac{1}{2\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}\Big)-\frac{\Big(\mathcal{R}_{HH}+2\mathcal{R}_{HV}\mathcal{R}_{VH}\Big)}{4K_1\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}-\frac{\mathcal{R}_{HH}\gamma_H}{2\mathcal{R}_0K_1},\\
\Upsilon^{\mathcal{R}_0}_{\theta} &= \frac{\mathcal{R}_{HV}\mathcal{R}_{VH}\theta\eta_1}{2\mathcal{R}_0[\theta\eta_1+(1-\theta)]\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}-\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}N_0\theta}{2\mathcal{R}_0(N_0-1)(1-\theta)\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}},\\
\Upsilon^{\mathcal{R}_0}_{\mu_H} &= \frac{\beta_{HH}}{2K_1\mathcal{R}_0}-\frac{\mathcal{R}_{HH}(K_1+\mu_H)}{2K_1\mathcal{R}_0}-\frac{(\mathcal{R}_{HH}\mu_H+\mathcal{R}_{HH}K_1-\beta_{HH})}{4\mathcal{R}_0K_1\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}-\frac{\mathcal{R}_{HV}\mathcal{R}_{VH}(K_1-\mu_H)}{2\mathcal{R}_0K_1^2\sqrt{(\frac{\mathcal{R}_{HH}}{2})^2+\mathcal{R}_{HV}\mathcal{R}_{VH}}}.
\end{aligned}
\tag{C.1}
$$

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Table 1: Description of variables and parameters for the model (5).

Variable	**Interpretation**
S_H	Population of susceptible humans
I_H	Population of infected humans
R_H	Population of recovered humans
A	Population of aquatic mosquitoes
M_S	Population of sterile male mosquitoes
M_N	Population of non-sterile male mosquitoes
M	Total male mosquito population
Y	Population of non-fertilized female mosquitoes
F_S	Population of fertilized sterile susceptible female mosquitoes
F_N	Population of fertilized non-sterile susceptible female mosquitoes
F_{SI}	Population of fertilized sterile infected female mosquitoes
F_{NI}	Population of fertilized non-sterile infected female mosquitoes
Parameter	**Interpretation**
b_H	Recruitment rate of humans
γ_H	Recovery rate of humans
μ_H	Natural death rate of humans
δ_H	Disease induced death rate of humans
α	Mating rate of mosquitoes
b_V	Maturation rate of mosquitoes
ϕ_V	Oviposition rate of fertilized female mosquitoes
θ	Mating probability of a sterilized male mosquito
μ_V	Natural death rate of non-sterilized mosquitoes
μ_S	Natural death rate of sterilized male mosquitoes
μ	Density dependent death rate of aquatic mosquitoes
r	Proportion of matured mosquitoes that are female
η_1	Modification parameter for reduced infectiousness of sterilized mosquitoes in comparison to non-sterilized mosquitoes
η_2	Modification parameter for reduction in infectiousness of recovered humans in comparison to infected humans
ρ_{HH}	Transmission probability from infectious to susceptible humans
ρ_{VH}	Transmission probability from infectious mosquitoes to susceptible humans
ρ_{HV}	Transmission probability from infectious humans to susceptible mosquitoes
β_{HH}	Rate of infection from infectious to susceptible humans
β_{VH}	Rate of infection from infectious mosquitoes to susceptible humans
β_{HV}	Rate of infection from infectious humans to susceptible mosquitoes

Table 2: Two sets of parameter values used in numerical simulations, with low baseline values that gives $\mathcal{R}_0 = 0.2461 < 1$, while $\mathcal{R}_0 = 4.3250 > 1$ for the high baseline values

Parameters	Range (day^{-1})	Low baseline	High baseline	References
r	$(0, 1)$	0.5	0.5	[19, 21]
δ_H	0.001	0.001	0.001	[13, 26]
θ	$(0, 1)$	0.2	0.4	assumed
α	$(0, 1)$	0.7	0.7	[19]
μ	0.00001	0.00001	0.00001	[3]
ϕ_V	$100 - 200$	100	120	[12]
b_H	30	30	30	[6]
b_V	$0.05 - 0.1$	0.05	0.08	[17, 18, 19]
η_1	$(0, 1)$	0.5	0.5	assumed
η_2	$(0, 1)$	0.04	0.2	assumed
γ_H	$0.059 - 0.167$	0.14	0.08	[39]
ξ_1	$0.3 - 1$	0.3	0.5	[25, 37, 2]
ξ_2	$0.01 - 0.20$	0.001	0.01	[37]
ξ_3	$0.3 - 1$	0.3	0.5	[37, 2]
μ_V	$0.029 - 0.25$	0.25	0.09	[25, 2]
μ_H	0.00004	0.00004	0.00004	[17, 18, 19]
ρ_{HH}	$0 - 1$	0.02	0.04	[25, 37]
ρ_{VH}	$0.1 - 0.75$	0.2	0.7	[25, 2]
ρ_{HV}	$0.3 - 0.75$	0.3	0.5	[25, 2]

Table 3: Sensitivity index of $\mathcal{R}_0$ with respect to parameters of the model (5) for $\mathcal{R}_0 = 0.2461 < 1$ and $\mathcal{R}_0 = 4.3250 > 1$ using the values of Table 2

Parameter	Low baseline	Sensitivity index	High baseline	Sensitivity index
r	0.5	+0.40271	0.5	+0.41333
δ_H	0.001	−0.00344	0.001	−0.00662
θ	0.2	−0.07344	0.4	−0.18978
γ_H	0.14	−0.19457	0.08	−0.19978
α	0.7	+0.10598	0.7	+0.04709
μ	0.00001	−0.19610	0.00001	−0.20585
ϕ_V	100	+0.20661	120	+0.20749
η_1	0.5	+0.02179	0.5	+0.05146
η_2	0.04	+0.28729	0.2	+0.32992
μ_V	0.2	−0.71355	0.09	−0.66732
μ_H	0.00004	−0.09133	0.00004	−0.12435
b_H	30	−0.19610	30	−0.20585
b_V	0.05	+0.40096	0.08	+0.41274
β_{HV}	0.09	+0.39219	0.25	+0.41169
β_{HH}	0.0001	+0.28934	0.0004	+0.33075

Table 4: Number of possible roots for (28) for $\mathcal{R}_1 < 1$ and $\mathcal{R}_1 > 1$.

Case	a_0	a_1	a_2	a_3	a_4	Value of $\mathcal{R}_1$	Sign change	Real roots
1	+	+	+	+	+	$\mathcal{R}_1 < 1$	0	0
2	+	+	+	+	-	$\mathcal{R}_1 > 1$	1	1
3	+	+	+	-	+	$\mathcal{R}_1 < 1$	2	0, 2
4	+	+	+	-	-	$\mathcal{R}_1 > 1$	1	1
5	+	+	-	+	+	$\mathcal{R}_1 < 1$	2	0, 2
6	+	+	-	+	-	$\mathcal{R}_1 > 1$	3	1, 3
7	+	+	-	-	+	$\mathcal{R}_1 < 1$	2	0, 2
8	+	+	-	-	-	$\mathcal{R}_1 > 1$	1	1
9	+	-	-	-	+	$\mathcal{R}_1 < 1$	2	0, 2
10	+	-	-	-	-	$\mathcal{R}_1 > 1$	1	1
11	+	-	+	-	+	$\mathcal{R}_1 < 1$	4	0, 2, 4
12	+	-	+	-	-	$\mathcal{R}_1 > 1$	3	1, 3
13	+	-	+	+	+	$\mathcal{R}_1 < 1$	2	0, 2
14	+	-	+	+	-	$\mathcal{R}_1 > 1$	3	1, 3
15	+	-	-	+	+	$\mathcal{R}_1 < 1$	2	0, 2
16	+	-	-	+	-	$\mathcal{R}_1 > 1$	3	1, 3

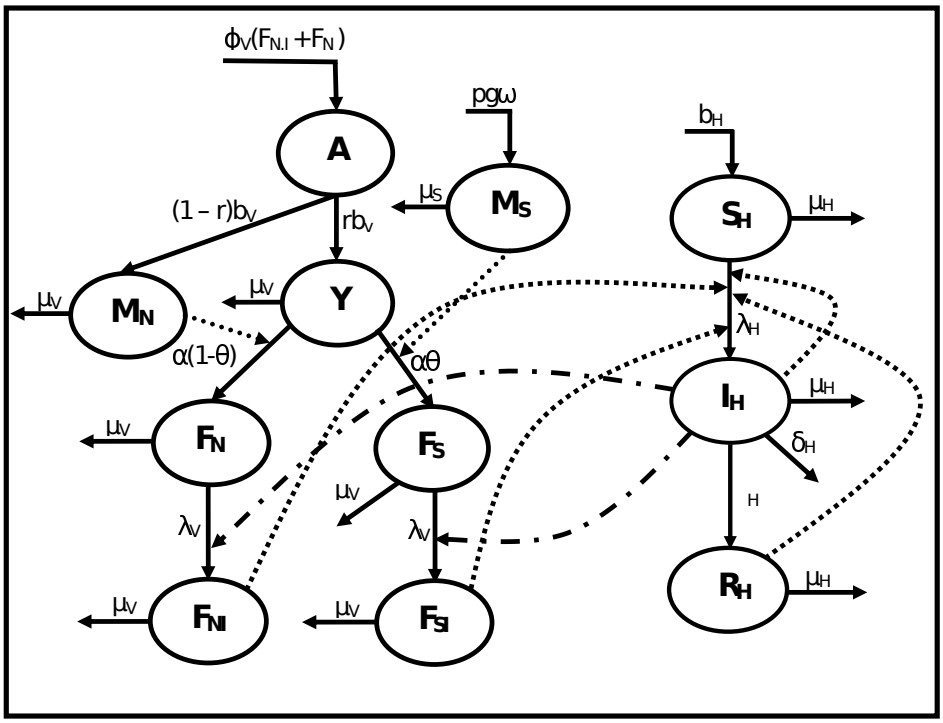

Figure 1: *Schematic diagram of the model* (5).

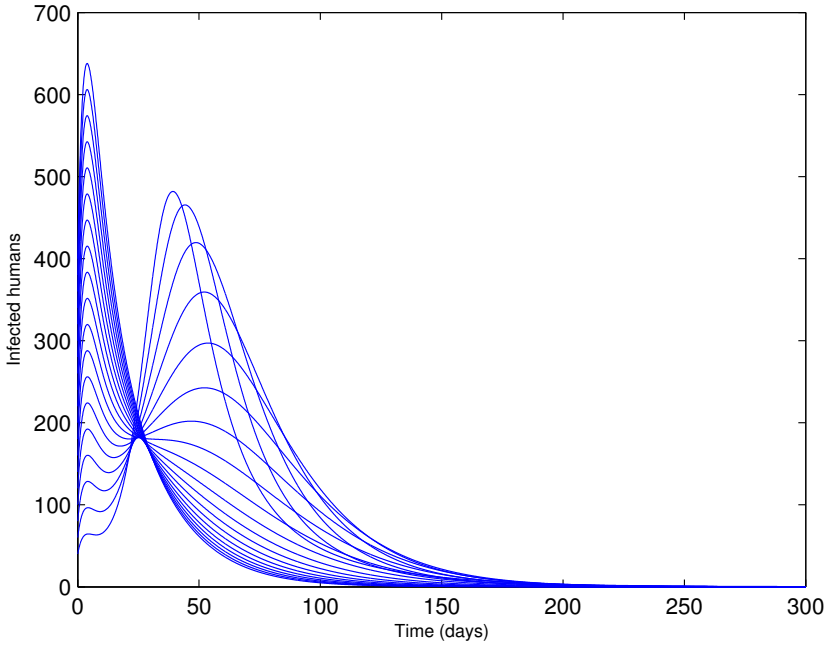

Figure 2: Simulation of the model (5) showing solution profile of infected humans. Parameter values used are as given in Table 2, with different initial conditions so that $\mathcal{R}_0 = 0.2461 < 1$.

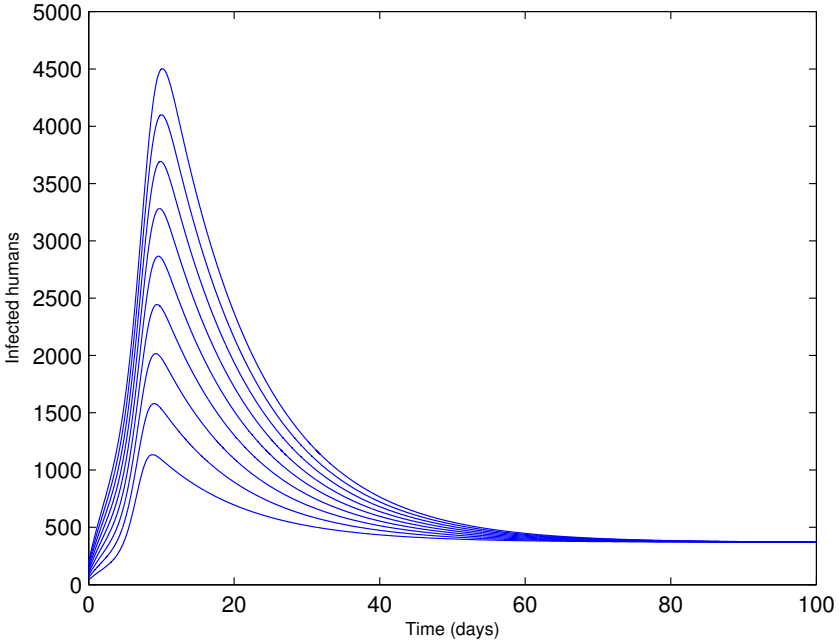

Figure 3: Simulation of the model (5) showing solution profile of infected humans. Parameter values used are as given in Table 2, with different initial conditions so that $\mathcal{R}_0 = 4.3250 > 1$.

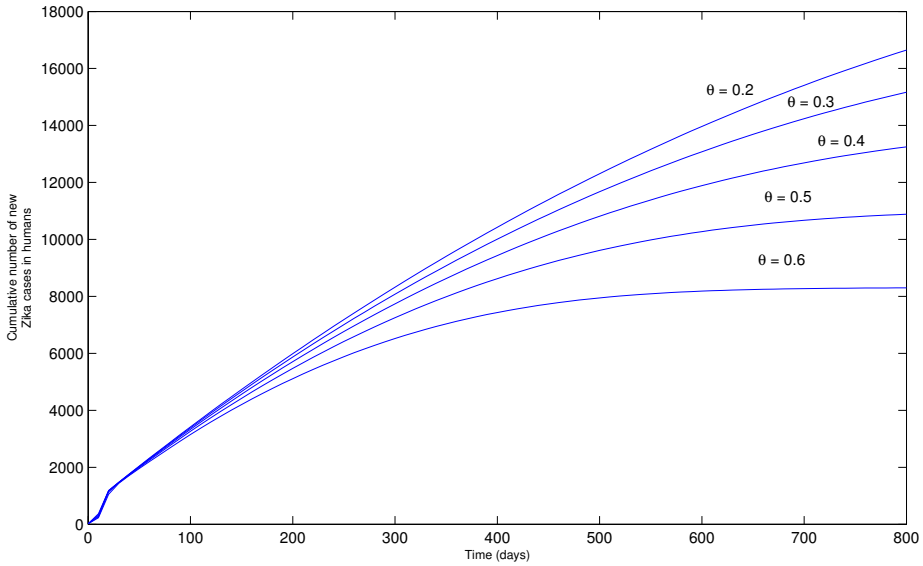

Figure 4: Simulation of the model (5) showing the cumulative number of new cases in human population. Parameter values used are as given in Table 2, with various values of θ (chances of mating with sterilized male mosquitoes).

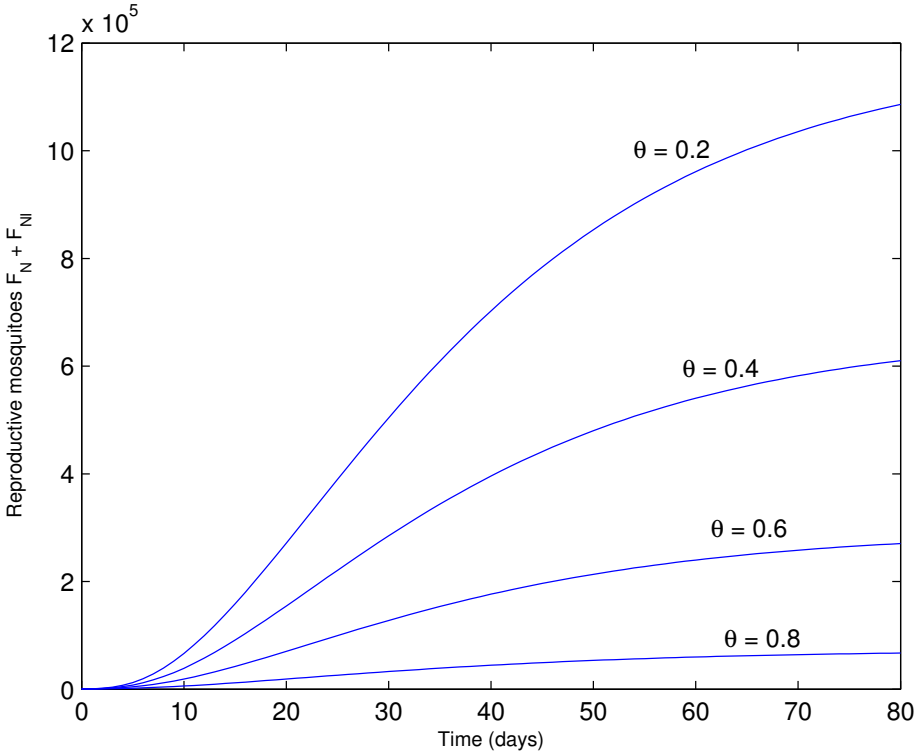

Figure 5: Simulation of the model (5) showing solution profile of reproductive mosquitoes. Parameter values used are as given in Table 2, with $\theta = 0.2$, $\theta = 0.4$, $\theta = 0.6$ and $\theta = 0.8$.

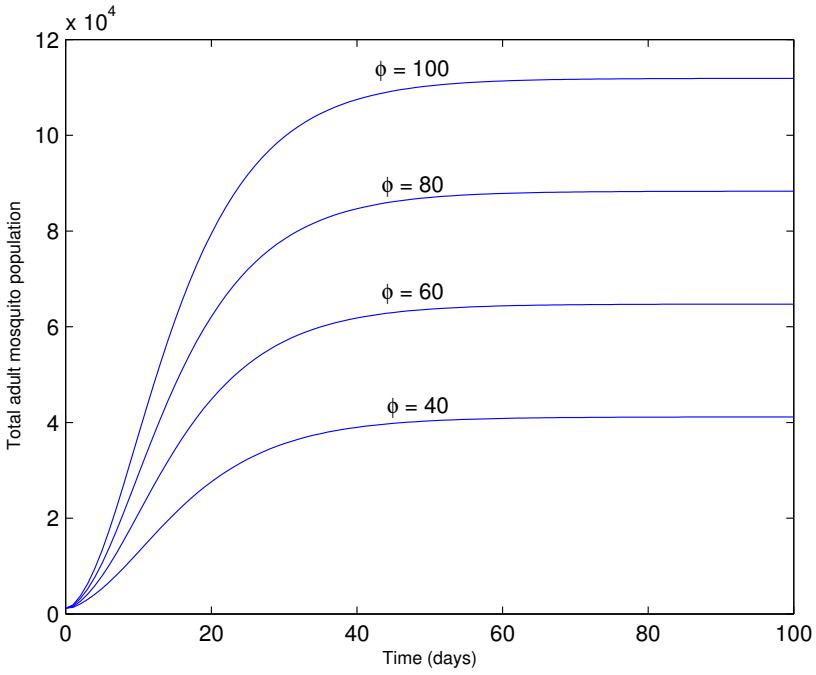

Figure 6: Simulation of the model (5) showing the total number of adult mosquitoes. Parameter values used are as given in Table 2, with $\phi_V = 100$, $\phi_V = 80$, $\phi_V = 60$ and $\phi_V = 40$ which respectively give $N_0 = 19.6491$, $N_0 = 15.7193$, $N_0 = 11.7895$ and $N_0 = 7.8596$.

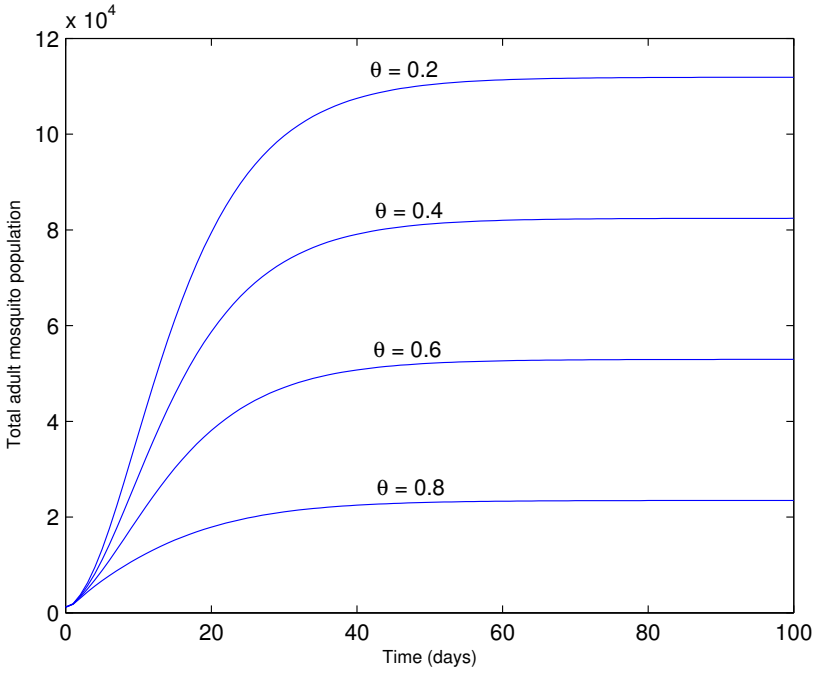

Figure 7: Simulation of the model (5) showing the number of reproductive mosquitoes. Parameter values used are as given in Table 2, with $\theta = 0.2$, $\theta = 0.4$, $\theta = 0.6$ and $\theta = 0.8$ which respectively give $N_0 = 19.6491$, $N_0 = 14.7368$, $N_0 = 9.8246$ and $N_0 = 4.9123$.

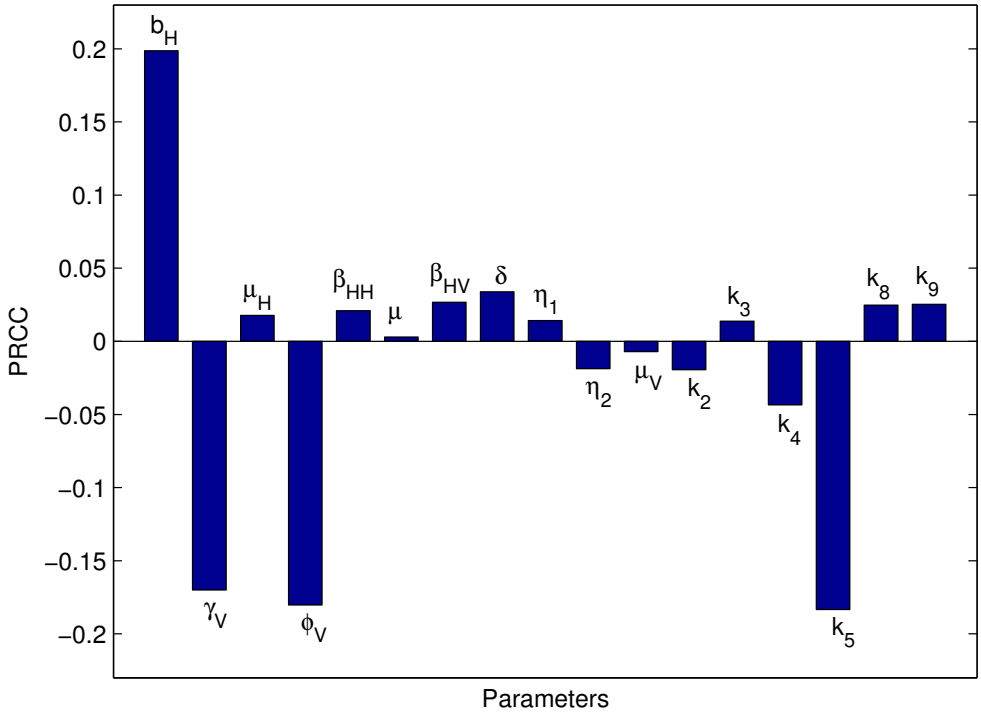

Figure 8: PRCC plots of the various parameters of the model (5), using total infectious humans ($I_H + R_H$) as the output function.

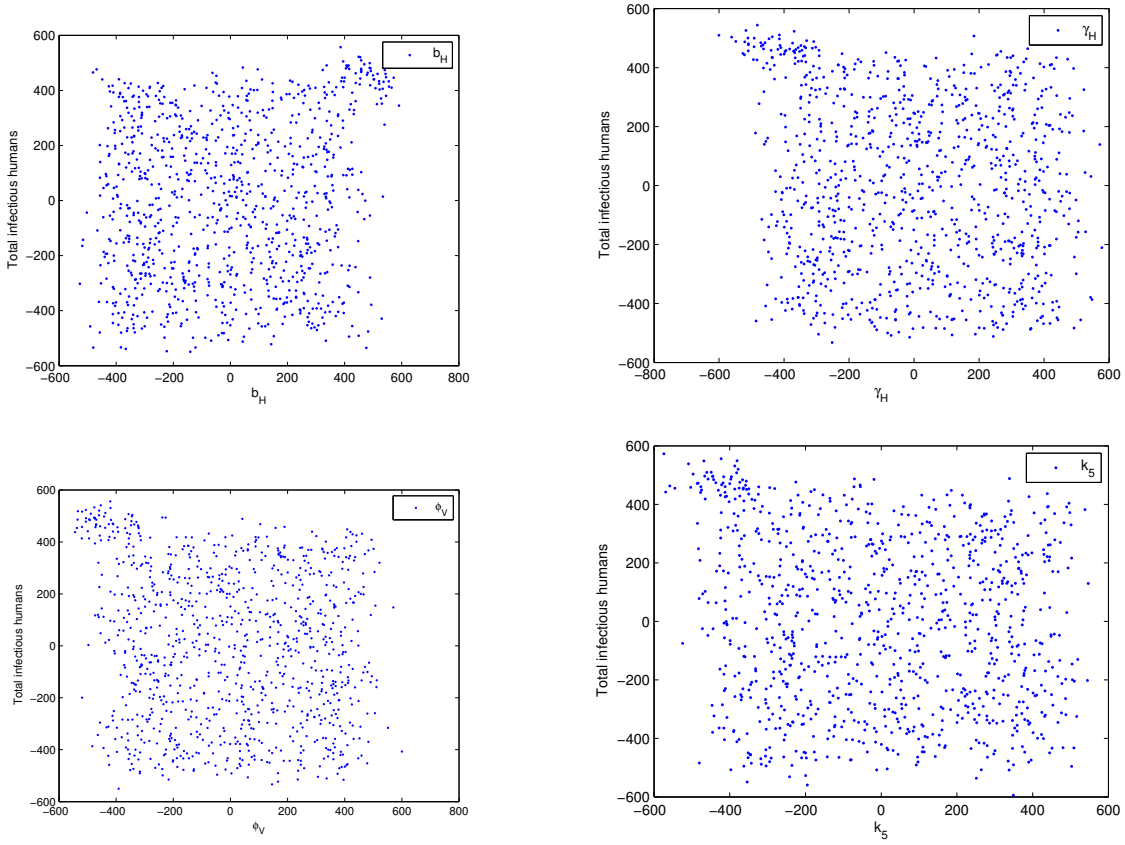

Figure 9: Scatter plots of the most sensitive parameters b_H, γ_H, ϕ_V and k_5.

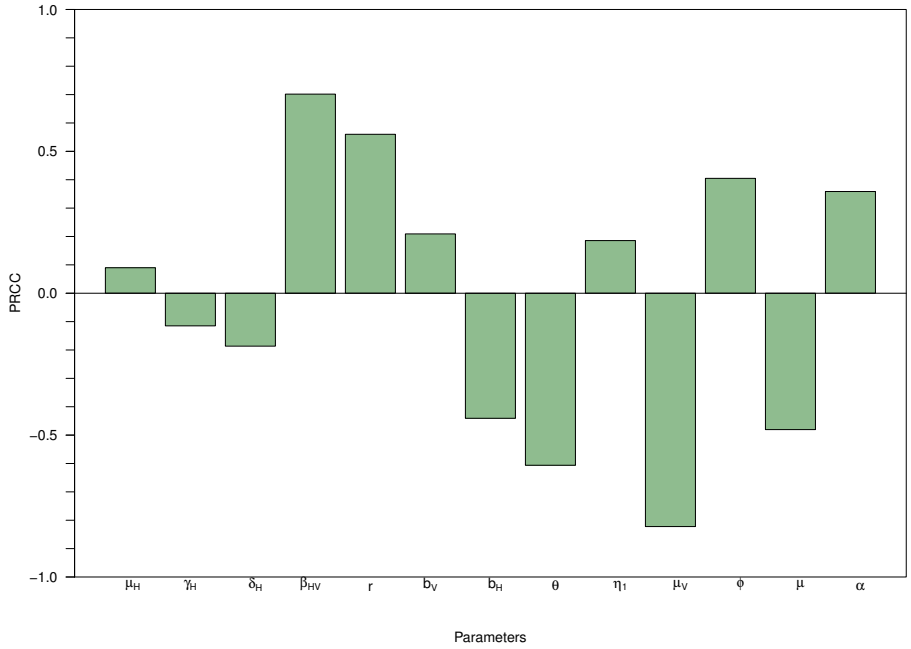

Figure 10: PRCC plots of the various parameters of the Zika model (5), using $\mathcal{R}_1$ as the output function. Parameter ranges used are in Table 2.

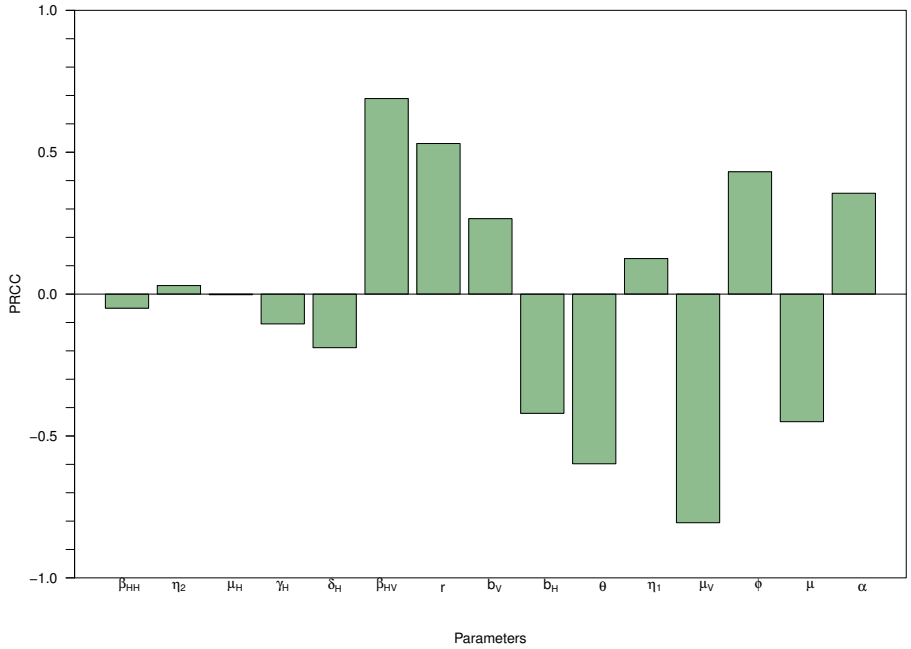

Figure 11: PRCC plots of the various parameters of the Zika model (5), using $\mathcal{R}_0$ as the output function. Parameter ranges used are in Table 2.