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Research article

Dynamics and optimal control of a Zika model with sexual and vertical transmissions

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Abstract: A new transmission model of Zika virus with three transmission routes including human transmission by mosquito bites, sexual transmission between males and females and vertical transmission is established. The basic reproduction number R_0 is derived. When $R_0 < 1$, it is proved that the disease-free equilibrium is globally stable. Furthermore, the optimal control and mitigation methods for transmission of Zika virus are deduced and explored. The MCMC method is used to estimate the parameters and the reasons for the deviation between the actual infection cases and the simulated data are discussed. In addition, different strategies for controlling the spread of Zika virus are simulated and studied. The combination of mosquito control strategies and internal human control strategies is the most effective way in reducing the risk of Zika virus infection.

Keywords: Zika virus; vertical transmission; sexual transmission; the basic reproduction number; optimal control

1. Introduction

The Zika virus is named after it was first discovered in 1947 in rhesus monkeys in Uganda's Zika forest [1]. The Zika virus is widely distributed in the tropics and subtropics. It is mainly transmitted to humans through the bites of infected female aedes mosquitoes [2]. Many people infected with Zika will experience no symptoms or only mild symptoms. Common Zika symptoms are fever, rash, headache, joint pain, red eyes and muscle pain that can last from a few days to a week [3]. The Zika virus was first detected in Uganda and Tanzania in 1952 [4]. Virus activity had been muted and only sporadic cases of Zika virus infection had been reported in equatorial Africa, the Americas, Asia and the Pacific. Since then, there had been a pandemic on the Western Pacific island of Yap in 2007 [5]. In 2013 to 2014, there was a larger outbreak in French Polynesia in the Pacific region, which infected about 32,000 people [6]. Zika spread rapidly in Brazil after the first case in South America was detected there in 2015 [7]. In October of that year, Brazil reported an increase in microcephaly cases. Pregnant women with Zika risk transmitted the virus to their newborns and gave birth to babies with microcephaly or other birth defects [8]. In addition to mosquito-borne transmission, Zika is transmitted sexually, mainly from men to women. Studies have shown that the Zika virus persists in men's semen longer than other body fluids, up to six months [9]. Zika virus can be transmitted to women several months after a man has recovered. Another mode of transmission that has a high impact is vertical transmission, where the virus is passed from an infected pregnant woman to her newborn. This mode of transmission is likely to result in children being born with microcephaly or other severe fetal brain defects. The virus is also spread in laboratories and through blood transfusions [10].

All sorts of sophisticated models have been built to study the spread of Zika. The possible transmission of Zika virus through sexual transmission was first identified by Foy et al. [11]. Gao et al. [12] established a Zika transmission model with mosquito-borne and sexual transmission of the virus, using the Zika epidemic data from Brazil, Colombia and El Salvador for data simulation. It was concluded that sexual transmission was relatively small contributor to Zika virus transmission but that sexual transmission increases the risk of infection and epidemic size and may prolong outbreaks. He et al. [13] simulated Zika virus infections reported in French Polynesia, Colombia, and the State of Bahia in Brazil. By comparing the simulation results, we would be able to better understand the likely evolution of Zika virus, control the spread of the outbreak and prevent potential transmission. Baca-Carrasco et al. [14] proposed three mathematical models including vector transmission, sexual transmission and population migration. The common conclusion was that the level of endemic disease following Zika virus outbreak was very low and sexual transmission contributed to the extent of the outbreak. Agusto et al. [15] established a transmission model of Zika virus in the absence of disease deaths and performed stability analysis. When expanding the model to include mortality due to Zika virus, different model stability analyses were established and numerical simulations were used to assess the importance of sexual transmission to study the dynamics of the disease. Imran et al. [16] established a model considering the vertical transmission of human and mosquito as well as analyzing its dynamic behavior in detail. Denes et al. [17] established a complex transmission model, which distinguished males and females in the mode of sexual transmission. Data simulations used Zika virus data from Costa Rica and Suriname concluded that mosquito birth and death rates were the most important factors in Zika virus transmission, but sexual transmission also had a significant impact on

disease transmission. Ibrahim et al. [18] established a model of Zika virus transmission involving sexually transmitted and asymptomatic carriers. The effects of weather periodicity on mosquito-related parameters were studied. Yuan et al. [19] proposed three modes of transmission of Zika virus, including vector transmission between humans and mosquitoes, transmission by human sexual contact and vertical transmission within mosquitoes. The contribution of each transmission route to the basic reproduction number was analyzed. Numerical simulations confirm that sexual and vertical transmission had different effects on the early and long-term transmission of Zika virus. Busenberg and Cooke [20] earlier discussed the modeling and dynamic analysis of various vertically transmitted diseases. In an $S - I - R$ compartment model, we incorporate vertical transmission into infected category (I_f, I_m) by assuming that a subset of the offspring of infected women $(B_h \frac{\lambda_h I_f}{N_f})$ are infectious at birth, while the remaining newborns $(B_h - B_h \frac{\lambda_h I_f}{N_f})$ enter the susceptible category. Where B_h represents natural birth rate of humans and λ_h represents proportion of offspring with congenital infection of infected females. Li et al. [21] used a similar method to consider vertical transmission. Some literatures have conducted detailed studies on the optimal control strategies for the transmission of Zika virus, studied the impact of different control strategy combinations on the transmission of Zika virus in human populations, and obtained some effective strategies for preventing and controlling the transmission of Zika virus disease [22, 23].

Few previous studies on Zika transmission have dealt with the vertical transmission in humans. However, the people most affected by Zika infection are newborns. It is of great significance to distinguish the sexes and add neonatal infection for analysis. With numerical simulations, it is clear which control measures are in place to rapidly control the spread of Zika disease, and the changes in actual cases in Colombia show that effective control measures can rapidly control the spread of the disease. Motivated by the above discussion, the goal of this paper is to study the joint influence of mosquito-borne transmission, sexual transmission and vertical transmission in human and mosquitoes on the spread of Zika virus. The sections of this paper are as follows. In Section 2, we introduce a Zika transmission model with three transmission modes: mosquito-borne transmission, sexual contact transmission and vertical transmission. In Section 3, we determine the basic reproduction number of the model and study the local asymptotic stability of the disease-free equilibrium as well as the global stability of the disease-free equilibrium in the case of $R_0 < 1$. In Section 4, we derive and explore the expression of the optimal control policy. In Section 5, we provide parameter estimates using actual transmission cases in Colombia, explaining why the actual transmission cases do not fit well with the fitted curve. The number of infected cases is simulated under different control strategies. The paper ends with a discussion in last section.

2. The model formulation

2.1. System description

The total human population $N_h(t)$ consists of female human and male human. Female human consists of three compartments: $S_f(t)$, $I_f(t)$, $R_f(t)$. $S_f(t)$ represents the number of female susceptible

individual, $I_f(t)$ represents the number of female infected individual, $R_f(t)$ represents the number of female recovered individual. Male human consists of four compartments: $S_m(t)$, $I_m(t)$, $I_m^r(t)$, $R_m(t)$. $S_m(t)$ represents the number of male susceptible individuals, $I_m(t)$ represents the number of male infected individuals, $I_m^r(t)$ represents the number of male convalescent individuals who have recovered from the disease but can still transmit it through sexual contact. $R_m(t)$ represents the number of male recovered individual. The total vector population $N_v(t)$ consists of two compartments: $S_v(t)$, $I_v(t)$. $S_v(t)$ represents the number of susceptible mosquitos, $I_v(t)$ represents the number of infected mosquitos. The transmission diagram of the model is shown in Figure 1.

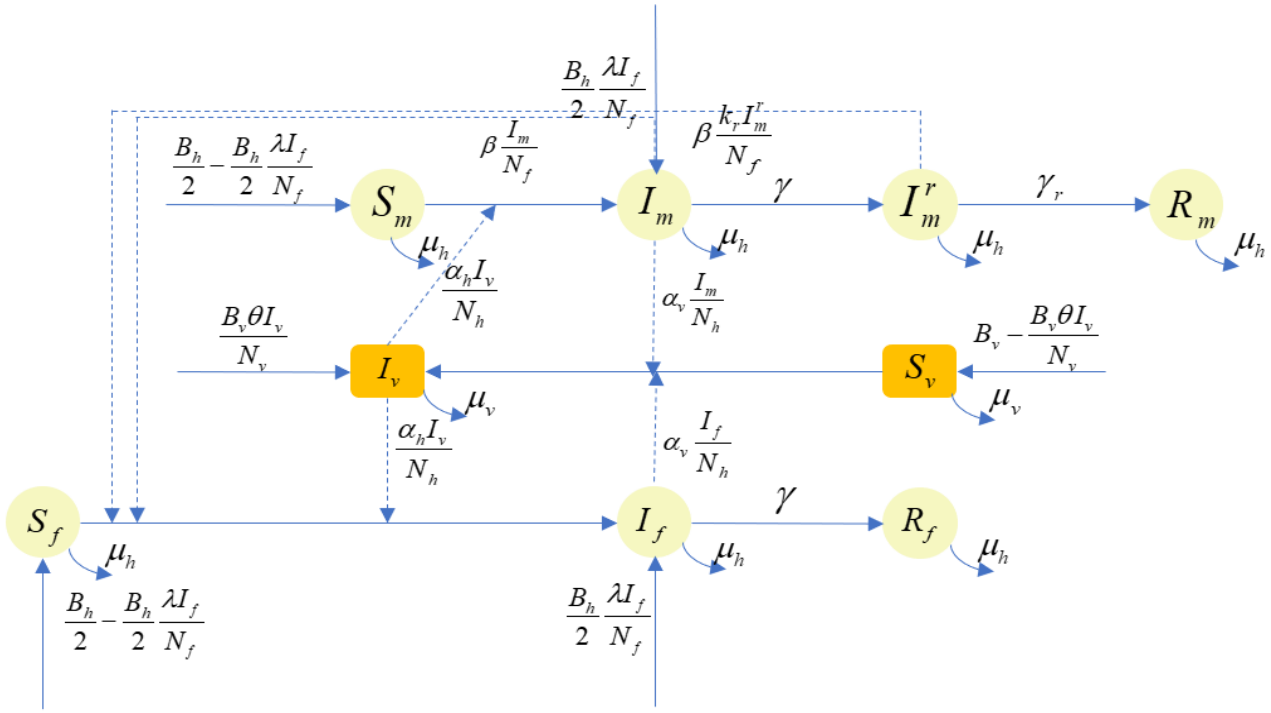

Figure 1. Transfer diagram for the dynamics of Zika. Solid arrows show the progression of infection, and dotted arrows show the direction of human-to-mosquito and human-to-human transmission.

The total human population and the total mosquito population are given by:

$$\begin{aligned} N_f(t) &= S_f(t) + I_f(t) + R_f(t), \\ N_m(t) &= S_m(t) + I_m(t) + I_m^r(t) + R_m(t), \\ N_v(t) &= S_v(t) + I_v(t), \\ N_h(t) &= N_f(t) + N_m(t). \end{aligned}$$

Then the model can be built as follows:

$$
\begin{cases}
\dfrac{\mathrm{d}S_f}{\mathrm{d}t} = \dfrac{B_h}{2} - \dfrac{B_h}{2}Y_1(t) - Y_2(t)S_f - Y_3(t)S_f - \mu_h S_f, \\
\dfrac{\mathrm{d}I_f}{\mathrm{d}t} = \dfrac{B_h}{2}Y_1(t) + Y_2(t)S_f + Y_3(t)S_f - \gamma I_f - \mu_h I_f, \\
\dfrac{\mathrm{d}R_f}{\mathrm{d}t} = \gamma I_f - \mu_h R_f, \\
\dfrac{\mathrm{d}S_m}{\mathrm{d}t} = \dfrac{B_h}{2} - \dfrac{B_h}{2}Y_1(t) - Y_3(t)S_m - \mu_h S_m, \\
\dfrac{\mathrm{d}I_m}{\mathrm{d}t} = \dfrac{B_h}{2}Y_1(t) + Y_3(t)S_m - \gamma I_m - \mu_h I_m, \\
\dfrac{\mathrm{d}I_m^r}{\mathrm{d}t} = \gamma I_m - \gamma_r I_m^r - \mu_h I_m^r, \\
\dfrac{\mathrm{d}R_m}{\mathrm{d}t} = \gamma_r I_m^r - \mu_h R_m, \\
\dfrac{\mathrm{d}S_v}{\mathrm{d}t} = B_v - Y_5(t)S_v - B_v Y_4(t) - \mu_v S_v, \\
\dfrac{\mathrm{d}I_v}{\mathrm{d}t} = Y_5(t)S_v + B_v Y_4(t) - \mu_v I_v.
\end{cases}
\tag{2.1}
$$

We define the functions $Y_1(t), Y_2(t), Y_3(t), Y_4(t), Y_5(t)$ by

$$
\begin{aligned}
Y_1(t) &= \frac{\lambda_h I_f}{N_f}, \\
Y_2(t) &= \beta\frac{I_m + k_r I_m^r}{N_f}, \\
Y_3(t) &= \alpha_h\frac{I_v}{N_h}, \\
Y_4(t) &= \frac{\theta I_v}{N_v}, \\
Y_5(t) &= \alpha_v\frac{I_f + I_m}{N_h}.
\end{aligned}
$$

The model assumes that the population has a constant natural birth rate (B_h) and that the number of males and females is the same, denoting the natural birth rate of males and females by $\frac{B_h}{2}$. When vertical transmission is considered, if a woman is infected with Zika virus during pregnancy, then the individual newborn may also be infected and proportion of congenital infected offspring in infected females is assumed to be λ_h (see, e.g., [21]). $\frac{I_f}{N_f}$ is the proportion of infected women, so $\frac{B_h}{2}\frac{\lambda_h I_f}{N_f}$ represents the number of infected newborns, and $\frac{B_h}{2} - \frac{B_h}{2}\frac{\lambda_h I_f}{N_f}$ indicates susceptible female newborns. The same is true of male representations. Male susceptible individuals (S_m) are infected with Zika virus due to the bite of mosquitos infected with Zika virus, the transmission probability of infected mosquitos biting susceptible human is α_h, thus entering the category of infected individuals (I_m). Male infections decrease due to natural mortality (μ_h) and cure rates (γ) of infected persons. The male enters the convalescent phase (I_m^r), during which the virus will not be present in the male blood, but will be present in the male semen for a long period of time (even up to six months). Sexual contact with others during

this period of time is a risk of transmitting the virus to others. Due to metabolism, the virus will be metabolized out of the body and eventually transformed into male recovered individual (R_m) at the recovery rate of γ_r. Female susceptible individuals (S_f) enter the infected category (I_f) by acquiring the virus through the bite of infected mosquitos with transmission probability (α_h) and through sexual contact with transmission probability (β). Female infections decrease due to natural mortality (μ_h) and cure rates (γ) of infected persons. Recovered women were transferred to the recovered category (R_f).

Similarly, susceptible mosquitoes (S_v) become infected by biting infected human with transmission probability (α_v). The proportion of the offspring of infected mosquitoes (I_v) will be infected and assume that the proportion of infected female offspring is θ. Consider the expression of vertical propagation similar to that of humans. It is assumed that the mosquito will not recover after infection. The parameters description of the Zika model are summarized in Table 1.

Table 1. The parameters description of the Zika model.

Parameter	Description(Units)
B_h	Natural birth rate of humans ($days^{-1}$)
μ_h	Natural death rate of humans ($days^{-1}$)
β	Sexual transmission infection rate from infected humans to susceptible humans ($days^{-1}$)
α_h	Baseline value of transmission rate from mosquitoes to humans($days^{-1}$)
α_v	Baseline value of transmission rate from humans to mosquitoes ($days^{-1}$)
k_r	Relative human-to-human transmissibility of convalescent to symptomatic humans (none)
γ	Human recovery rate ($days^{-1}$)
γ_r	Recovery rate of convalescent humans ($days^{-1}$)
λ_h	Proportion of offspring with congenital infection of infected females (none)
B_v	Baseline value of mosquito birth rate ($days^{-1}$)
$\frac{1}{\mu_v}$	Mosquito lifespan(days)
θ	Proportion of offspring congenital infection of infected female mosquitoes (none)

By reference [24], Theorems 1.1.8 and 1.1.9 of Wiggins [25] for each nonnegative initial condition there is a unique, non-negative solution.

Lemma 1. *Let the initial value $W(0) \geq 0$, where $W(t) = (S_f, I_f, R_f, S_m, I_m, I_m^r, R_m, S_v, I_v)$. Then the solutions $W(t)$ of model (2.1) are non-negative for all time $t \geq 0$. Furthermore*

$$\limsup_{t\to\infty} N_h(t) = \frac{B_h}{\mu_h}, \quad \limsup_{t\to\infty} N_v(t) = \frac{B_v}{\mu_v}.$$

Proof. Let $t_1 = sup\{t > 0 : W(t) > 0\}$, by the first equation of model (2.1) that

$$\frac{\mathrm{d}S_f}{\mathrm{d}t} = \frac{B_h}{2} - \frac{B_h}{2}Y_1(t) - Y_2(t)S_f - Y_3(t)S_f - \mu_h S_f,$$

we get

$$S_f(t_1) = S_f(0)e^{-(Y_2(u)+Y_3(u)+\mu_h)t_1} + e^{-(Y_2(u)+Y_3(u)+\mu_h)t_1}e^{\int_0^{t_f}-(Y_2(u)+Y_3(u))\mathrm{d}u+\mu_h t_1}$$
$$\int_0^{t_f}(\frac{B_h}{2} - \frac{B_h}{2}Y_1(u))\mathrm{d}u > 0.$$

It can similarly be shown that $W(t) > 0$ for all $t > 0$. It can be represented as $0 < S_f \leq N_h(t), 0 < I_f \leq N_h(t), 0 < R_f \leq N_h(t), 0 < S_m \leq N_h(t), 0 < I_m \leq N_h(t), 0 < I_m^r \leq N_h(t), 0 < R_m \leq N_h(t), 0 < S_v \leq N_v(t), 0 < I_v \leq N_v(t)$.

Supplementary model (2.1) are given as

$$\frac{\mathrm{d}N_h}{\mathrm{d}t} = B_h - \mu_h N_h.$$

$$\frac{\mathrm{d}N_v}{\mathrm{d}t} = B_v - \mu_v N_v.$$

Hence,

$$\frac{B_h}{\mu_h} = \liminf_{t\to\infty} N_h \leq \limsup_{t\to\infty} N_h = \frac{B_h}{\mu_h},$$

$$\frac{B_v}{\mu_v} = \liminf_{t\to\infty} N_v \leq \limsup_{t\to\infty} N_v = \frac{B_v}{\mu_v}.$$

as required.

Model (2.1) is analyzed in a biologically meaningful feasible domain. Consider the feasible region

$$W_h = \left\{ S_f, I_f, R_f, S_m, I_m, I_m^r, R_m. : N_h \leq \frac{B_h}{\mu_h} \right\},$$

$$W_v = \left\{ S_v, I_v : N_v \leq \frac{B_v}{\mu_v} \right\}.$$

Lemma 2. *The region $W = W_h \times W_v \subset \mathbb{R}_+^7 \times \mathbb{R}_+^2$ is positively invariant and attracts all positive orbits in W.*

Proof. Following steps to establish positive invariance of W. The rate of change in the population is obtained by supplementary model (2.1) to give

$$\frac{\mathrm{d}N_h}{\mathrm{d}t} = B_h - \mu_h N_h,$$

$$\frac{\mathrm{d}N_v}{\mathrm{d}t} = B_v - \mu_v N_v.$$

Therefore, it can be obtained

$$N_h = N_h(0)e^{-\mu_h t} + \frac{B_h}{\mu_h}(1 - e^{-\mu_h t}),$$

$$N_v = N_v(0)e^{-\mu_v t} + \frac{B_v}{\mu_v}(1 - e^{-\mu_v t}).$$

Particularly, $N_h \leq \frac{B_h}{\mu_h}$, if $N_h(0) \leq \frac{B_h}{\mu_h}$. Similarly, $N_v \leq \frac{B_v}{\mu_v}$, if $N_v(0) \leq \frac{B_v}{\mu_v}$. Thus, the set W is a positive invariant.

In order to prove that the set W is attractive, note that $\frac{\mathrm{d}N_h}{\mathrm{d}t} < 0$ and $\frac{\mathrm{d}N_v}{\mathrm{d}t} < 0$ if $N_h(0) > \frac{B_h}{\mu_h}$ and $N_v(0) > \frac{B_v}{\mu_v}$ respectively. Thus, either the solution enters W in finite time, or $N_h(t)$ and $N_v(t)$ approach $\frac{B_h}{\mu_h}$ and $\frac{B_v}{\mu_v}$ respectively, and therefore the variables of the infection class I_f, I_m, I_m^r and I_v tend to 0. Thus, the set W is attractive and all solutions in $\mathbb{R}_+^9$ eventually enter W.

3. Analysis of the model

3.1. Local stability of disease-free equilibrium

Obviously, the system (2.1) always has a disease-free equilibrium which is given by:

$$\begin{aligned}\varepsilon_0 &= (S_f^*, I_f^*, R_f^*, S_m^*, I_m^*, I_m^{r*}, R_m^*, S_v^*, I_v^*)\\ &= (\frac{B_h}{2\mu_h}, 0, 0, \frac{B_h}{2\mu_h}, 0, 0, 0, \frac{B_v}{\mu_v}, 0).\end{aligned}$$

Using the next-generation matrix method [24] to prove local stability of disease free equilibrium, we construct the transmission vector $\mathcal{F}$ representing the new infections flowing only into the infected compartments given by

$$\mathcal{F} = \left(\frac{B_h}{2}\frac{\lambda_h I_f}{N_f} + \beta\frac{I_m + k_r I_m^r}{N_f}S_f + \frac{\alpha_h I_v}{N_h}S_f, \frac{B_h}{2}\frac{\lambda_h I_f}{N_f} + \frac{\alpha_h I_v}{N_h}S_m, 0, \frac{B_v\theta I_v}{N_v} + \alpha_v\frac{I_f + I_m}{N_h}S_v\right)^T,$$

the transition vector $\mathcal{V}$ represent the outflow from the infectious compartments in system (2.1) is given by

$$\mathcal{V} = \left(\gamma I_f + \mu_h I_f, \gamma I_m + \mu_h I_m, \gamma_r I_m^r + \mu_h I_m^r - \gamma I_m, \mu_v I_v\right)^T$$

Substitute the value of the disease-free equilibrium $N_h = \frac{B_h}{\mu_h}$ and $N_f = N_m = \frac{B_h}{2\mu_h}$,we compute the Jacobian F from $\mathcal{F}$ given by

$$F = \begin{pmatrix} \lambda_h\mu_h & \beta & \beta k_r & \frac{\alpha_h}{2} \\ \lambda_h\mu_h & 0 & 0 & \frac{\alpha_h}{2} \\ 0 & 0 & 0 & 0 \\ \frac{\alpha_v\mu_h B_v}{B_h\mu_v} & \frac{\alpha_v\mu_h B_v}{B_h\mu_v} & 0 & \theta\mu_v \end{pmatrix},$$

and the Jacobian V from $\mathcal{V}$ given by

$$V = \begin{pmatrix} \gamma + \mu_h & 0 & 0 & 0 \\ 0 & \gamma + \mu_h & 0 & 0 \\ 0 & -\gamma & \gamma_r + \mu_h & 0 \\ 0 & 0 & 0 & \mu_v \end{pmatrix}.$$

Thus, the characteristic polynomial available from of the generation matrix FV^{-1} is

$$\lambda^4 - \frac{k_1\theta + \mu_h\lambda_h}{k_1}\lambda^3 - \frac{\mu_h(B_h\beta k_r\mu_v^2\theta\lambda_h + B_v k_2\alpha_h\alpha_v)}{k_1 B_h\mu_v^2 k_2}\lambda^2 + \frac{1}{2}\frac{\mu_h\beta\gamma_r k_r(2B_h\mu_v^2\theta\lambda_h - B_v\alpha_h\alpha_v)}{k_1 B_h\mu_v^2 k_2}\lambda = 0,$$

where $k_1 = \gamma + \mu_h, k_2 = \gamma_r + \mu_h$. We can solve for four eigenroots, one of which is 0, two complex roots, one real root, and find the absolute value of the largest root.

Hence, the basic reproduction number of model (2.1) is

$$R_0 = \rho(FV^{-1}) = \frac{\frac{1}{6}\frac{1}{k_1 B_h \mu_v k_2}\sqrt[3]{\psi_1 + 6\sqrt{-\frac{1}{B_h k_2}3\mu_h\psi_2}} + \frac{2}{3}\psi_3}{\sqrt[3]{k_1\mu_v(\psi_1 + 6\sqrt{-\frac{1}{B_h k_2}3\mu_h\psi_2}k_1)B_h^2k_2^2} + \frac{1}{3}\frac{k_1\theta+\mu_h\lambda_h}{k_1}}$$

where

$$\begin{aligned}
\psi_1 =& -72B_h\beta k_1^2k_r\mu_h\mu_v^3\theta\gamma_r\lambda_h + 36B_h\beta k_1k_r\mu_h^2\mu_v^3\gamma_r\lambda_h^2 + 8B_hk_1^3k_2\mu_v^3\theta^3 - 12B_hk_1k_2\mu_h\mu_v^3\theta^2\lambda_h \\
&- 12B_hk_1k_2\mu_h^2\mu_v^3\theta\lambda_h^2 + 8B_hk_2\mu_h^3\mu_v^3\lambda_h^3 + 54B_v\beta k_1^2k_r\mu_h\mu_v\alpha_h\alpha_v\gamma_r + 36B_vk_1^2k_2\mu_h\mu_v\theta\alpha_h\alpha_v \\
&+ 36B_vk_1k_2\mu_h^2\mu_v\alpha_h\alpha_v\lambda_h, \\
\psi_2 =& 16B_h^3\beta^3k_1k_r^3\mu_h^2\mu_v^6\gamma_r^3\lambda_h^3 - 32B_h^3\beta_2k_1^2k_2k_r^2\mu_h\mu_v^6\theta_2\gamma_r^2\lambda_h^2 + 32B_h^2\beta^2k_1k_2k_r^2\mu_h^2\mu_v^6\theta\gamma_r^2\lambda_h^3 \\
&+ 4B_h^3\beta^2k_2k_r^2\mu_h^3\mu_v^6\gamma_r^2\lambda_h^4 + 16B_h^3\beta k_1^3k_2^2k_r\mu_v^6\theta^4\gamma_r\lambda_h - 32B_h^3\beta k_1^2k_2^2k_r\mu_h\mu_v^6\theta_3\gamma_r\lambda_h^3 \\
&+ 8B_h^3\beta k_1k_2^2k_r\mu_h^2\mu_v^6\theta^2\gamma_r\lambda_h^3 + 8B_h^3\beta k_2^2k_r\mu_h^3\mu_v^6\theta\gamma_r\lambda_h^4 + 4B_h^3k_1^2k_2^3\mu_h\mu_v^6\theta^4\lambda_h^2 \\
&- 8B_h^3k_1k_2^3\mu_h^2\mu_v^6\theta_3\lambda_h^3 + 4B_h^3k_2^2\mu_h^3\mu_v^6\theta^2\lambda_h^4 + 72B_h^2B_v\beta^2k_1^2k_2k_r^2\mu_h\mu_v^4\theta\alpha_h\alpha_h\alpha_v^6\gamma_r^2\lambda_h \\
&+ 12B_h^2B_v\beta^2k_1k_2k_r^2\mu_h^2\mu_v^4\alpha_h\alpha_v\gamma_r^2\lambda_h^2 - 8B_h^2B_v\beta k_1^3k_2^2k_r\mu_v^4\theta^3\alpha_h\alpha_v\gamma_r \\
&+ 92B_h^2B_v\beta k_1^2k_2^2k_r\mu_h\mu_v^4\theta^2\alpha_h\alpha_v\gamma_r\lambda_h + 4B_h^2B_v\beta k_1k_2^2k_r\mu_h^2\mu_v^4\theta\alpha_h\alpha_v\gamma_r\lambda_h^2 \\
&- 8B_h^2B_vk_1^2k_2^3\mu_h\mu_v^4\theta^3\alpha_h\alpha_v\lambda_h + 32B_h^2B_vk_1k_2^3\mu_h^2\mu_v^4\theta^2\alpha_h\alpha_v\lambda_h^2 - 8B_h^2B_vk_2^3\mu_h^3\mu_v^4\theta\alpha_h\alpha_v\lambda_h^3 \\
&- 27B_hB_v^2\beta^2k_1^2k_2k_r^2\mu_h\mu_v^2\alpha_h^2\alpha_v^2\gamma_r^2 - 36B_hB_v^2\beta k_1^2k_2^2k_r\mu_h\mu_v^2\theta\alpha_h^2\alpha_v^2\gamma_r \\
&+ 12B_hB_v^2\beta k_1k_2^2k_r\mu_h^2\mu_v^2\gamma_r\lambda_h + 4B_hB_v^2k_1^2k_2^3\mu_h\mu_v^2\theta^2\alpha_h^2\alpha_v^2 - 40B_hB_v^2k_1k_2^3\mu_h^2\mu_v^2\theta\alpha_h^2\alpha_v^2\lambda_h \\
&+ 4B_hB_v^2k_2^3\mu_h^3\mu_v^2\alpha_h^2\alpha_v^2\lambda_h^2 + 16B_v^3k_1k_2^3\mu_h^2\alpha_h^3\alpha_v^3, \\
\psi_3 =& 2B_h\beta k_1k_r\mu_h\mu_v^2\gamma_r\lambda_h + B_hk_1^2k_2\mu_v^2\theta^2 + B_hk_1k_2\mu_h\mu_v^2\theta\lambda_h + B_hk_2\mu_h^2mu_v^2\lambda_h^2 + 3B_vk_1k_2\mu_h\alpha_h\alpha_v.
\end{aligned}$$

Using Theorem 2 of [24], we obtain the local stability of disease-free equilibrium.

Theorem 3.1.1. *The disease-free equilibrium ε_0 is locally asymptotically stable for $R_0 < 1$ and is unstable for $R_0 > 1$.*

3.2. Global stability of disease-free equilibrium

Using the method which is applied in [26–29], we obtain global stability of disease-free equilibrium. In order to make the population size of Zika extinction independent of the initial value, we establish the global stability of the disease-free equilibrium point, considering a feasible region

$$W_1 = \{X \in W : S_f \le S_f^*, S_m \le S_m^*, S_v \le S_v^*\},$$

where $X = S_f, I_f, R_f, S_m, I_m, I_m^r, R_m, S_v, I_v$

Lemma 3. *The W_1 is positively invariant for model (2.1).*

Proof. For the the first equation of the model (2.1), where $S_f^* = \frac{B_h}{2\mu_h}$

$$\begin{aligned}\frac{\mathrm{d}S_f}{\mathrm{d}t} &= \frac{B_h}{2} - \frac{B_h}{2}Y_1(t) - Y_2(t)S_f - Y_3(t)S_f - \mu_h S_f \\ &\leq \frac{B_h}{2} - \mu_h S_f \\ &\leq \mu_h[\frac{B_h}{2\mu_h} - S_f] \\ &= \mu_h\left(S_f^* - S_f\right).\end{aligned} \tag{3.1}$$

Further,

$$S_f(t) \leq S_f^* - \left(S_f^* - S_f(0)\right)e^{-\mu_h t}.$$

Hence, if $S_f(0) \leq S_f^*$ for $t \geq 0$, then $S_f(t) \leq S_f^*$ for $t \geq 0$. Similarly, it can be obtained if $S_m(0) \leq S_m^*$ for $t \geq 0$, then $S_m(t) \leq S_m^*$ for $t \geq 0$ and if $S_v(0) \leq S_v^*$ for $t \geq 0$, then $S_v(t) \leq S_v^*$ for $t \geq 0$. Therefore, in general the field W_1 is a positive invariant set and attracts all solutions of model (2.1) in $\mathbb{R}_+^9$.

Remark 3.2.1. *The W_1 is a special field that needs to be satisfied when $R_0 < 1$, which represents the basin of attraction.*

Theorem 3.2.1. *The disease-free equilibrium ε_0 is globally asymptotically stable for $R_0 < 1$.*

Proof. To prove the global stability of the disease-free equilibrium, let $X = \left(S_f, R_f, S_m, R_m, S_v\right)$ and $Z = \left(I_f, I_m, I_m^r, I_v\right)$. Therefore, the grouping system can be expressed as

$$\begin{aligned}\frac{\mathrm{d}X}{\mathrm{d}t} &= F(X, 0), \\ \frac{\mathrm{d}Z}{\mathrm{d}t} &= G(X, Z).\end{aligned} \tag{3.2}$$

where, $F(X, 0)$ is the right side of $\dot{S_f}, \dot{R_f}, \dot{S_m}, \dot{R_m}, \dot{S_v}$ when $I_f = I_m = I_m^r = I_v = 0$ and $G(X, Z)$ is the right side of $\dot{I_f}, \dot{I_m}, \dot{I_m^r}, \dot{I_v}$.

Next, we consider simplifying the system $\frac{\mathrm{d}X}{\mathrm{d}t} = F(X, 0)$:

$$\begin{aligned}\frac{\mathrm{d}S_f}{\mathrm{d}t} &= \frac{B_h}{2} - \mu_h S_f, \\ \frac{\mathrm{d}R_f}{\mathrm{d}t} &= -\mu_h R_f, \\ \frac{\mathrm{d}S_m}{\mathrm{d}t} &= \frac{B_h}{2} - \mu_h S_m,. \\ \frac{\mathrm{d}R_m}{\mathrm{d}t} &= -\mu_h R_m, \\ \frac{\mathrm{d}S_v}{\mathrm{d}t} &= B_v - \mu_v S_v.\end{aligned} \tag{3.3}$$

It is easy to get an equilibrium for system (3.3),

$$X^* = \left(S_f^*, R_f^*, S_m^*, R_m^*, S_v^*\right) = \left(\frac{B_h}{2\mu_h}, 0, \frac{B_h}{2\mu_h}, 0, \frac{B_v}{\mu_v}\right).$$

Showing that X^* is a globally stable equilibrium in W_1. For this, by the first and second equations of (3.3) we get

$$\begin{aligned} S_f(t) &= \frac{B_h}{2\mu_h} + (S_f(0) - \frac{B_h}{2\mu_h})e^{-\mu_h t}, \\ R_f(t) &= R_f(0)e^{-\mu_h t}. \end{aligned} \tag{3.4}$$

Taking the limits of $S_f(t)$ and $R_f(t)$ at $t \to \infty$, we have

$$\lim_{t\to\infty} S_f(t) = \frac{B_h}{2\mu_h} \quad \text{and} \quad \lim_{t\to\infty} R_f(t) = 0.$$

Similarly, it can be shown that $\lim_{t\to\infty} S_m(t) = \frac{B_h}{2\mu_h}$, $\lim_{t\to\infty} R_m(t) = 0$, and $\lim_{t\to\infty} S_v(t) = \frac{B_v}{\mu_v}$. These asymptotic dynamics are independent of initial in W. Therefore, the convergence of the solution of (3.3) is global in W_1. Further, according to [28] we require $G(X, Z)$ to satisfy the two stated conditions:

$$\begin{aligned} &(i). \quad G(X, 0) = 0, \\ &(ii). \quad G(X, Z) = D_z G(X^*, 0) - \hat{G}(X, Z), \quad \hat{G}(X, Z) \geq 0, \end{aligned}$$

where $(X^*, 0) = \left(\frac{B_h}{2\mu_h}, 0, 0, \frac{B_h}{2\mu_h}, 0, 0, 0, \frac{B_v}{\mu_v}, 0\right)$ and $D_z G(X^*, 0)$ is the Jacobian of the $G(X, Z)$ at $(X^*, 0)$, which is an M-matrix(the off-diagonal elements are nonnegative).
Thus,

$$D_z G(X^*, 0) = \begin{pmatrix} -\gamma - \mu_h + \frac{B_h\lambda_h}{2N_f^*} & \frac{\beta S_f^*}{N_f^*} & \frac{\beta k_r S_f^*}{N_f^*} & \frac{\alpha_h}{N_h^*} S_f^* \\ \frac{B_h\lambda_h}{2N_f^*} & -\gamma - \mu_h & 0 & \frac{\alpha_h}{N_h^*} S_m^* \\ 0 & \gamma & -\gamma_r - \mu_h & 0 \\ \frac{\alpha_v S_v^*}{N_h^*} & \frac{\alpha_v S_v^*}{N_h^*} & 0 & \frac{B_v\theta}{N_v^*} - \mu_v \end{pmatrix},$$

and

$$\hat{G}(X, Z) = \begin{pmatrix} \xi_1 I_f + \xi_2 I_m + \xi_3 I_m^r + \xi_4 I_v \\ \xi_1 I_f + \xi_5 I_v \\ 0 \\ \xi_6 I_v + \xi_7 (I_f + I_m) \end{pmatrix},$$

where, $\xi_1 = \frac{B_h\lambda_h}{2N_f^*}(1 - \frac{N_f^*}{N_f})$, $\xi_2 = \frac{\beta S_f^*}{N_f^*}\left(1 - \frac{S_f N_f^*}{N_f S_f^*}\right)$, $\xi_3 = \frac{\beta k_r S_f^*}{N_f^*}\left(1 - \frac{S_f N_f^*}{N_f S_f^*}\right)$, $\xi_4 = \frac{\alpha_h S_f^*}{N_h^*}\left(1 - \frac{S_f N_h^*}{N_h S_f^*}\right)$, $\xi_5 = \frac{\alpha_h S_m^*}{N_h^*}\left(1 - \frac{S_m N_h^*}{N_h S_m^*}\right)$, $\xi_6 = \frac{B_v\theta}{N_v^*}(1 - \frac{N_v^*}{N_v})$. $\xi_7 = \frac{\alpha_v S_v^*}{N_h^*}\left(1 - \frac{S_v N_h^*}{N_h S_v^*}\right)$.

Further, $S_f^* = \frac{B_h}{2\mu_h}, S_m^* = \frac{B_h}{2\mu_h}, S_v^* = \frac{B_v}{\mu_v}, N_h^* = \frac{B_h}{\mu_h}$. We have in that $S_f \leq S_f^*, S_m \leq S_m^*, S_v \leq S_v^*$. Hence, if the human population is in a state of equilibrium, we can get $(1 - \frac{N_f^*}{N_f}) > 0, \left(1 - \frac{S_f N_f^*}{N_f S_f^*}\right) > 0, \left(1 - \frac{S_f N_h^*}{N_h S_f^*}\right) > 0, \left(1 - \frac{S_m N_h^*}{N_h S_m^*}\right) > 0, (1 - \frac{N_v^*}{N_v}) > 0$ and $\left(1 - \frac{S_v N_h^*}{N_h S_v^*}\right) > 0$. Therefore, $\hat{G} \geq 0$. Then, according to the theorem in [28], the global stability of the disease-free equilibrium can be obtained. This would indicate that Zika virus will die out over time and remain stable globally.

4. The optimal control problems

4.1. Existence of optimal control

We add four time-dependent control variables to the model corresponding to four mitigation strategies. In order to derive the necessary conditions for the existence of optimal control, we use Pontryagin's maximum principle [30]. Abimbade et al. [31] and Olaniyi et al. [32] refers to the derivation of the optimal control problem.

In model (4.1), four control strategies $u_1(t), u_2(t), u_3(t), u_4(t)$ are added to extend model (2.1) to obtain model(4.1). $u_1(t)$ denotes the use of mosquito nets and other methods to reduce human exposure to mosquitoes; $u_2(t)$ means to improve the media, internet and other publicity efforts to enhance human awareness and reduce the probability of sexual transmission; $u_3(t)$ advocates delaying pregnancy and reducing the number of babies born with abnormalities. $u_4(t)$ means the use of insecticides and other methods to reduce mosquito populations. Here, we assume that the control set is

$$U = \{(u_1, u_2, u_3, u_4 | u_i(t) \in L^\infty[0, t_f], 0 \le u_i(t) \le c_i, 0 \le c_i \le 1, i = 1, ..., 4\}.$$

The optimal control model is given as

$$\begin{aligned}
\frac{\mathrm{d}S_f}{\mathrm{d}t} &= \frac{B_h}{2} - (1-u_3(t))\frac{B_h}{2}Y_1(t) - (1-u_2(t))Y_2(t)S_f - (1-u_1(t))Y_3(t)S_f - \mu_h S_f,\\
\frac{\mathrm{d}I_f}{\mathrm{d}t} &= (1-u_3(t))\frac{B_h}{2}Y_1(t) + (1-u_2(t))Y_2(t)S_f + (1-u_1(t))Y_3(t)S_f - \gamma I_f - \mu_h I_f,\\
\frac{\mathrm{d}R_f}{\mathrm{d}t} &= \gamma I_f - \mu_h R_f,\\
\frac{\mathrm{d}S_m}{\mathrm{d}t} &= \frac{B_h}{2} - (1-u_3(t))\frac{B_h}{2}Y_1(t) - (1-u_1(t))Y_3(t)S_m - \mu_h S_m,\\
\frac{\mathrm{d}I_m}{\mathrm{d}t} &= (1-u_3(t))\frac{B_h}{2}Y_1(t) + (1-u_1(t))Y_3(t)S_m - \gamma I_m - \mu_h I_m,\\
\frac{\mathrm{d}I_m^r}{\mathrm{d}t} &= \gamma I_m - \gamma_r I_m^r - \mu_h I_m^r,\\
\frac{\mathrm{d}R_m}{\mathrm{d}t} &= \gamma_r I_m^r - \mu_h R_m,\\
\frac{\mathrm{d}S_v}{\mathrm{d}t} &= B_v - B_v Y_4(t) - (1-u_1(t))Y_5(t)S_v - u_4(t)S_v - \mu_v S_v,\\
\frac{\mathrm{d}I_v}{\mathrm{d}t} &= B_v Y_4(t) + (1-u_1(t))Y_5(t)S_v - u_4(t)I_v - \mu_v I_v.
\end{aligned} \tag{4.1}$$

Due to non-negative initial conditions and bounded Lebesgue measurable control, this system has non-negative bounded solutions [33]. We consider an optimal control problem to minimize the objective functional

$$J = \int_0^{t_f} \left[A_1 I_f + A_2 I_m + A_3 I_m^r + A_4 I_v + \frac{1}{2}(\eta_1 u_1^2 + \eta_2 u_2^2 + \eta_3 u_3^2 + \eta_4 u_4^2) \right] \mathrm{d}t. \tag{4.2}$$

In Eq (4.2), A_1, A_2, A_3 and A_4 represent the weights of human infected and mosquito infected, respectively. The weights η_1, η_2, η_3 and η_4 are measures of the costs associated with the control variables u_1, u_2, u_3 and u_4, respectively.

Theorem 4.1.1. *We consider the objective functional J given by Eq (4.2) with $(u_1, u_2, u_3, u_4) \in U$ subject to the control model (4.1) with initial conditions. There exists $u^*(t) = \{u_1^*, u_2^*, u_3^*, u_4^*\} \in U$ such that*

$$J(u_1^*, u_2^*, u_3^*, u_4^*) = min\{J(u_1, u_2, u_3, u_4)|(u_1, u_2, u_2, u_4) \in U\}.$$

Proof. We can use the result of [33] to prove the existence of an optimal control problem.

By its definition, we know that the control set U is closed and convex, and the integrand is also convex on U. Obviously these state and control variables are non-negative. The control system is bounded, which means the compactness of optimal control. Furthermore, there exists a constant $\zeta > 1$ and positive values z_1, and z_2, such that

$$J(u_1, u_2, u_3, u_4) \geq z_1(|u_1|^2 + |u_2|^2 + |u_3|^2 + |u_4|^2)^{\frac{\zeta}{2}} - z_2,$$

which completes the existence of the optimal control. A method to prove the existence of optimal control is proposed in [34].

4.2. Characterization of optimal controls

To find an optimal solution we consider the Lagrangian function of the optimal control problem. The Lagrangian function is

$$L = A_1 I_f + A_2 I_m + A_3 I_m^r + A_4 I_v + \frac{1}{2}(\eta_1 u_1^2 + \eta_2 u_2^2 + \eta_3 u_3^2 + \eta_4 u_4^2).$$

For model (4.1), we derive the necessary conditions for optimal control according to the Pontryagin maximum principle. The corresponding Hamiltonian function is

$$
\begin{aligned}
&H(S_f, I_f, R_f, S_m, I_m, I_m^r, R_m, S_v, I_v, u_1, u_2, u_3, u_4, \lambda_i)\\
&=L(I_f, I_m, I_m^r, I_v) + \lambda_1\frac{\mathrm{d}S_f}{\mathrm{d}t} + \lambda_2\frac{\mathrm{d}I_f}{\mathrm{d}t} + \lambda_3\frac{\mathrm{d}R_f}{\mathrm{d}t} + \lambda_4\frac{\mathrm{d}S_m}{\mathrm{d}t} + \lambda_5\frac{\mathrm{d}I_m}{\mathrm{d}t}\\
&\quad + \lambda_6\frac{\mathrm{d}I_m^r}{\mathrm{d}t} + \lambda_7\frac{\mathrm{d}R_m}{\mathrm{d}t} + \lambda_8\frac{\mathrm{d}S_v}{\mathrm{d}t} + \lambda_9\frac{\mathrm{d}I_v}{\mathrm{d}t}\\
&=A_1I_f + A_2I_m + A_3I_m^r + A_4I_v + \frac{1}{2}(\eta_1u_1^2 + \eta_2u_2^2 + \eta_3u_3^2 + \eta_4u_4^2)\\
&\quad + \lambda_1\left[\frac{B_h}{2} - (1-u_3(t))\frac{B_h}{2}Y_1(t) - (1-u_2(t))Y_2(t)S_f - (1-u_1(t))Y_3(t)S_f - \mu_hS_f\right]\\
&\quad + \lambda_2\left[(1-u_3(t))\frac{B_h}{2}Y_1(t) + (1-u_2(t))Y_2(t)S_f + (1-u_1(t))Y_3(t)S_f - \gamma I_f - \mu_hI_f\right]\\
&\quad + \lambda_3\left[\gamma I_f - \mu_hR_f\right]\\
&\quad + \lambda_4\left[\frac{B_h}{2} - (1-u_3(t))\frac{B_h}{2}Y_1(t) - (1-u_1(t))Y_3(t)S_m - \mu_hS_m\right]\\
&\quad + \lambda_5\left[(1-u_3(t))\frac{B_h}{2}Y_1(t) + (1-u_1(t))Y_3(t)S_m - \gamma I_m - \mu_hI_m\right]\\
&\quad + \lambda_6\left[\gamma I_m - \gamma_rI_m^r - \mu_hI_m^r\right]\\
&\quad + \lambda_7\left[\gamma_rI_m^r - \mu_hR_m\right]\\
&\quad + \lambda_8\left[B_v - B_vY_4(t) - (1-u_1(t))Y_5(t)S_v - u_4(t)S_v - \mu_vS_v\right]\\
&\quad + \lambda_9\left[B_vY_4(t) + (1-u_1(t))Y_5(t)S_v - u_4(t)I_v - \mu_vI_v\right],
\end{aligned}
$$

where $\lambda_i, i = 1, ..., 9$ are adjoint variables.

Theorem 4.2.1. *Given an optimal control $(u_1^*, u_2^*, u_3^*, u_4^*)$, and let $S_f, I_f, R_f, S_m, I_m, I_m^r, R_m, S_v$ and I_v be the state solutions for model (4.1). Thus, there exist adjoint variables $\lambda_i, i = 1, ..., 9$ satisfying*

$$
\begin{aligned}
\frac{d\lambda_1}{dt} =&(\lambda_1-\lambda_2)\left[(1-u_2(t))Y_2+(1-u_1(t))Y_3\right]+\lambda_1\mu_h\Big],\\
\frac{d\lambda_2}{dt} =&-A_1+(\lambda_1-\lambda_2)(1-u_3(t))\frac{B_h\lambda_h}{2N_f}+(\lambda_2-\lambda_3)\gamma+\lambda_2\mu_h+(\lambda_4-\lambda_5)(1-u_3(t))\frac{B_h\lambda_h}{2N_f}\\
&+(\lambda_8-\lambda_9)(1-u_1(t))\frac{\alpha_v}{N_h}S_v,\\
\frac{d\lambda_3}{dt} =&\lambda_3\mu_h,\\
\frac{d\lambda_4}{dt} =&(\lambda_4-\lambda_5)(1-u_1(t))Y_3+\lambda_4\mu_h,\\
\frac{d\lambda_5}{dt} =&-A_2+(\lambda_1-\lambda_2)(1-u_2(t))\frac{\beta}{N_f}S_f+(\lambda_5-\lambda_6)(\gamma)+\lambda_5\mu_h+(\lambda_8-\lambda_9)(1-u_1(t))\frac{\alpha_v}{N_h}S_v,\\
\frac{d\lambda_6}{dt} =&-A_3+(\lambda_1-\lambda_2)(1-u_2(t))\frac{\beta k_r}{N_f}S_f+(\lambda_6-\lambda_7)\gamma_r+\lambda_6\mu_h,\\
\frac{d\lambda_7}{dt} =&\lambda_7\mu_h,\\
\frac{d\lambda_8}{dt} =&(\lambda_8-\lambda_9)(1-u_1(t))Y_5+\lambda_8(\mu_v+u_4(t)),\\
\frac{d\lambda_9}{dt} =&-A_4+(\lambda_1-\lambda_2)(1-u_1(t))\frac{\alpha_h}{N_h}S_f+(\lambda_4-\lambda_5)(1-u_1(t))\frac{\alpha_h}{N_h}S_m+(\lambda_8-\lambda_9)\frac{B_v\theta}{N_v}\\
&+\lambda_9(\mu_v+u_4(t)).
\end{aligned}
$$

The boundary conditions are

$$\lambda_i(t_f)=0, i=1,...,9.$$

Furthermore, the optimal controls u_1^*, u_2^*, u_3^* and u_4^* are represented by

$$
\begin{aligned}
u_1^*(t) &= min\{max\{u_1^c,0\},1\},\\
u_2^*(t) &= min\{max\{u_2^c,0\},1\},\\
u_3^*(t) &= min\{max\{u_3^c,0\},1\},\\
u_4^*(t) &= min\{max\{u_4^c,0\},1\},
\end{aligned}
\tag{4.3}
$$

where

$$
\begin{aligned}
u_1^c &= \frac{(\lambda_2-\lambda_1)Y_3S_f+(\lambda_5-\lambda_4)Y_3S_m+(\lambda_9-\lambda_8)Y_5S_v}{\eta_1},\\
u_2^c &= \frac{(\lambda_2-\lambda 1)Y_2S_f}{\eta_2},\\
u_3^c &= \frac{(\lambda_2-\lambda_1)\frac{B_h}{2}Y_1+(\lambda_5-\lambda_4)\frac{B_h}{2}Y_1}{\eta_3},\\
u_4^c &= \frac{\lambda_8S_v+\lambda_9I_v}{\eta_4}.
\end{aligned}
$$

Hence,

$$u_i^* = \begin{cases} 0, & if \quad u_i^c \leq 0, \\ u_i^c, & if \quad 0 < u_i^c < 1, \\ 1, & if \quad u_i^c \geq 1, \end{cases}$$

where $i = 1, ...4$.

Proof. The result of the adjoint system can be obtained from Pontryagin's principle

$$\frac{\mathrm{d}\lambda_i}{\mathrm{d}t} = -\frac{\partial H}{\partial x},$$

where $x = S_f, I_f, R_f, S_m, I_m, I_m^r, R_m, S_v$ and I_v. The boundary conditions are $\lambda_i^*(t_f) = 0, i = 1, ..., 9$.

To derive the characterization of the optimal control given by Eq (4.3), we solve the equations on the interior of the control set,

$$\frac{\partial H}{\partial u_1} = 0, \frac{\partial H}{\partial u_2} = 0, \frac{\partial H}{\partial u_3} = 0, \frac{\partial H}{\partial u_4} = 0.$$

Plugging the bounds for the controls, we obtain the desired characterization.

5. A case study for colombia

5.1. Numerical results

In this section, we apply our model to study Zika virus transmission cases in Colombia during 2015 to 2017. Based on the actual data of Zika virus transmission, numerical experiments are carried out using matlab and the parameters of the model is estimated using the Markov Chain Monte Carlo (MCMC) procedure. According to [35] data, we know that the average number of births per day in Colombia is about 1826.81 and the mortality rate is 0.0000368. It has been reported that it will take three to seven days for infected individuals with Zika to recover. Therefore, the recovery rate is set to 1/4. We know from [36, 37] that γ_r is 0.06. We choose a set of values of parameters in Table 2.

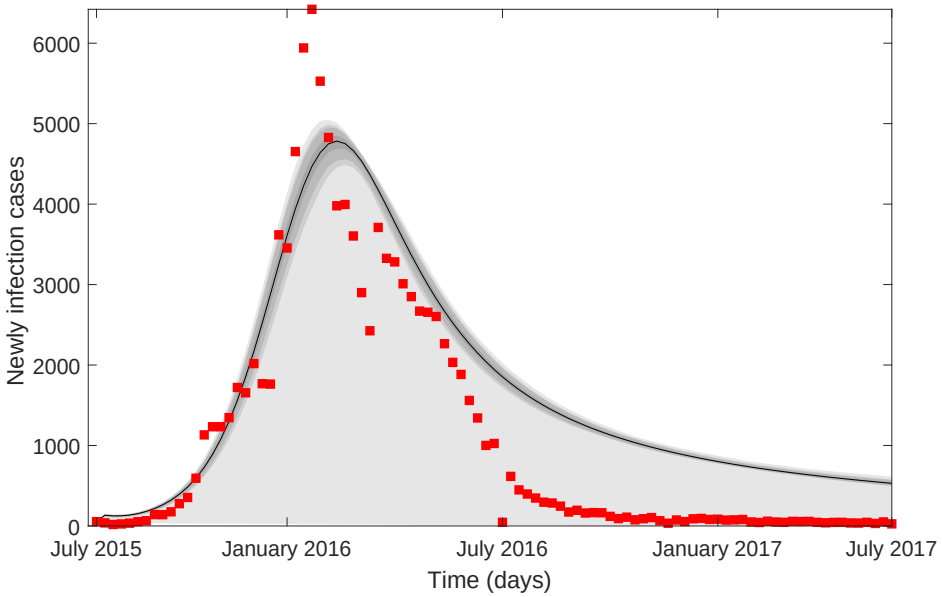

Figure 2. It represents the fitting results of Colombia's cases from 2015 to 2017 (no control measures), where the red dots represent the actual number of infections, the black line represents the fitted cases, and the gray areas from brightest to darkest represent the 50, 90, 95, and 99% posterior limits of the system.

According to Table 2, we know that transmission rate from infected humans to susceptible humans (β) is 0.046586, baseline value of transmission rate from mosquitoes to humans (α_h) is 0.63722 and baseline value of transmission rate from humans to mosquitoes (α_v) is 0.6893. About vertical transmission, proportion of offspring congenital infection of infected female mosquitoes (θ) is 0.0021348 and proportion of offspring with congenital infection of infected females (λ_h) is 0.0050859. The probability is relatively small, but it has caused serious consequences, not only the financial impact on society but also the impact on life and health. Therefore, we must pay attention to it.

Clearly, fitting results Figure 2, the data simulation does not match the actual data very well. According to [7], we found that in June 2016, the World Health Organization developed a plan and implemented measures to address the threat posed by Zika virus, including in Colombia, which also implemented measures to reduce the probability of transmission through sexual contact and human mosquito contact. This is the reason for the rapid decline in the actual number of cases from June 2016. Therefore, we're going to do a piecewise simulation(see Figure 3). Before June 2016, transmission rate from infected humans to susceptible humans (β) is 0.046586, baseline value of transmission rate from mosquitoes to humans (α_h) is 0.63722 and baseline value of transmission rate from humans to mosquitoes (α_v) is 0.6893. After June 2016, transmission rate from infected humans to susceptible humans (β) is 0.045953, baseline value of transmission rate from mosquitoes to humans (α_h) is 0.19573 and baseline value of transmission rate from humans to mosquitoes (α_v) is 0.30891. The values of other parameters are shown in Table 2. In conclusion, control measures can quickly bring the disease under control. In the next section, we will discuss the impact of several control measures on the transmission of Zika virus.

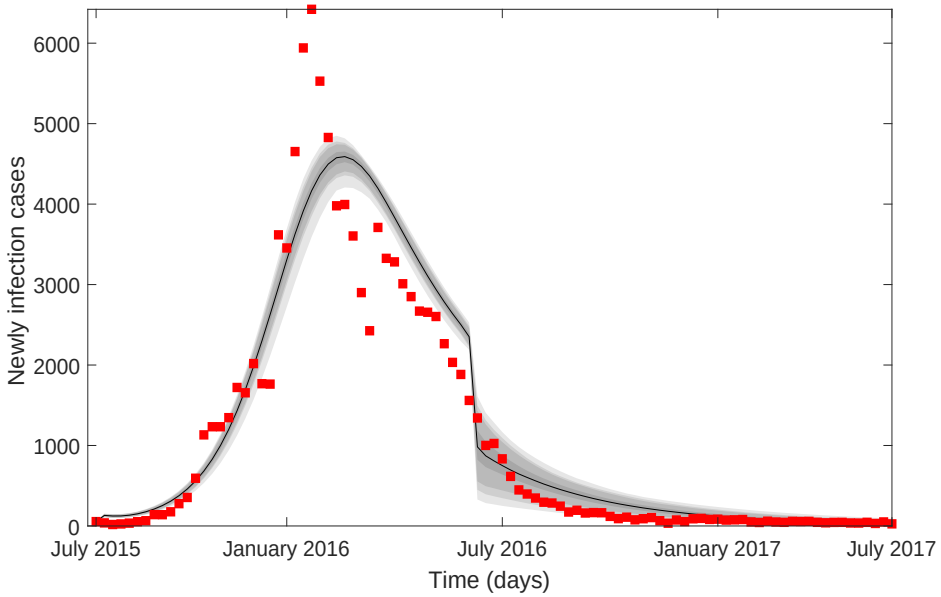

Figure 3. It represents the fitting results of Colombia's cases from 2015 to 2017 (add control measures), where the red dots represent the actual number of infections, the black line represents the fitted cases, and the gray areas from brightest to darkest represent the 50, 90, 95, and 99% posterior limits of the system.

Table 2. The parameters description of the Zika model.

Parameter	Range	Value	Source
B_h	-	1826.81	[35]
μ_h	-	0.0000368	[35]
β	0.01-0.1	0.046586	MCMC
α_h	0.03-0.75	0.63722	MCMC
α_v	0.09-0.75	0.6893	MCMC
k_r	0.2-0.8	0.4249	MCMC
γ	1/3-1/7	1/4	Assumed
γ_r	0.01-0.07	0.06	[36, 37]
λ_h	0.001-0.3	0.0054773	MCMC
B_v	200-5000	4506	MCMC
$\frac{1}{\mu_v}$	4-35	8	Assumed
θ	0-0.004	0.0021348	MCMC

5.2. Analysis of control measures

In this section, we examine the impact of several control measures on the prevalence of the disease and the change in the number of infected people under optimal control. We take the initial value

$S_f = 30000, I_f = 200, R_f = 500, S_m = 30000, I_m = 150, I_m^r = 100, R_m = 400, S_v = 2000, I_v = 100$. We choose the parameter values as follows, $B_h = 1826.81, B_v = 4506, \mu_h = 3.65 * 10^{-5}, \mu_v = \frac{1}{8}, \beta = 0.046586, \alpha_h = 0.63722, \alpha_v = 0.6893, k_r = 0.4249, \gamma = \frac{1}{4}, \gamma_r = 0.06, \lambda_h = 0.0054773, \theta = 0.0021348$. In order to compare the effectiveness of control measures, we conduct simulations as follows.

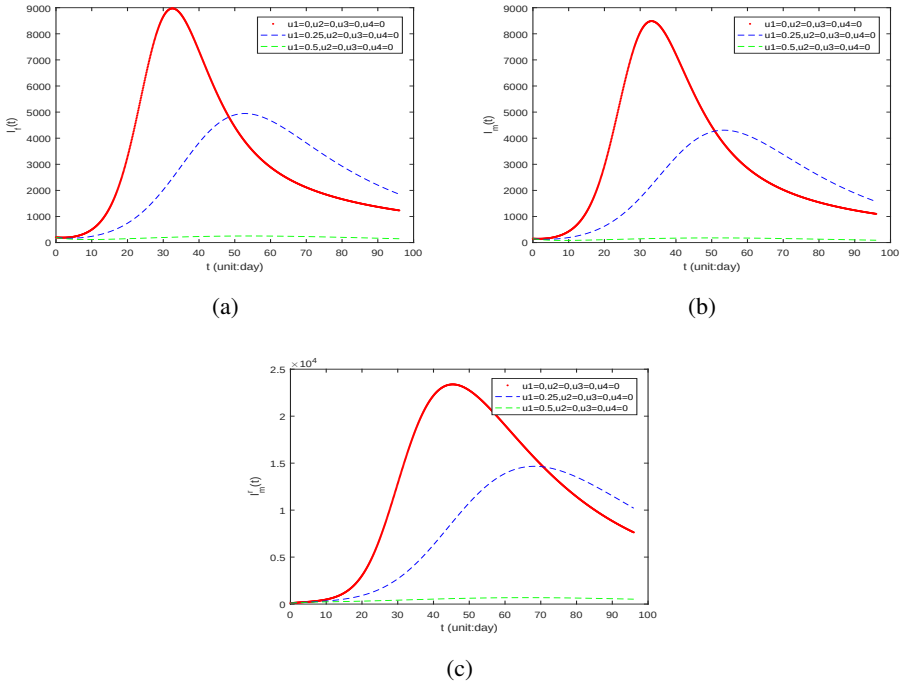

Figure 4. Effects of reducing human exposure to mosquitoes with none($u_1 = 0$), mild ($u_1 = 0.25$) and moderate ($u_1 = 0.5$) on the time varying plots of the number of I_f, I_m, I_m^r.

To study the impact of different control measures on Zika virus outbreak, we will simulate cases over time in female susceptible individual, male susceptible individual and male convalescent individuals under different control measures. Figure 4 shows the change of the number of infected persons (I_f, I_m, I_m^r) over time when only the control measure u_1 is added and other control measures are zero. As can be seen from the figure, when no control measures are taken, the peak of infected people will be brought to a high degree and then the number will decrease. Reduction of human mosquito exposure rate ($u_1 = 0.25$) will greatly reduce the peak of infection with slight control, but its impact on Zika virus will be reduced in later stages. Under moderate control of u_1 the disease will not break out or become extinct and will remain at a very low incidence. Figure 5 shows the change of the number of infected persons (I_f, I_m, I_m^r) over time when control measure u_4 is added and other control measures are zero. As can be seen from the figure, only mild implementation of control measures ($u_4 = 0.2$) is required to prevent the outbreak of disease and gradually eliminate the disease over time. Figure 6 shows the change in the number of infected persons (I_f, I_m, I_m^r) when only u_2 is added and other control measures are zero. As can be seen from the figure, when u_2 control measures are moderate, the peak value of infected females (I_f) is slightly reduced and the effect on infected females (I_m) and convalescent men (I_m^r) is not significant. Comparing Figures 4–6, it is clear that controlling mosquito transmission to

humans is more effective than controlling sexual transmission.

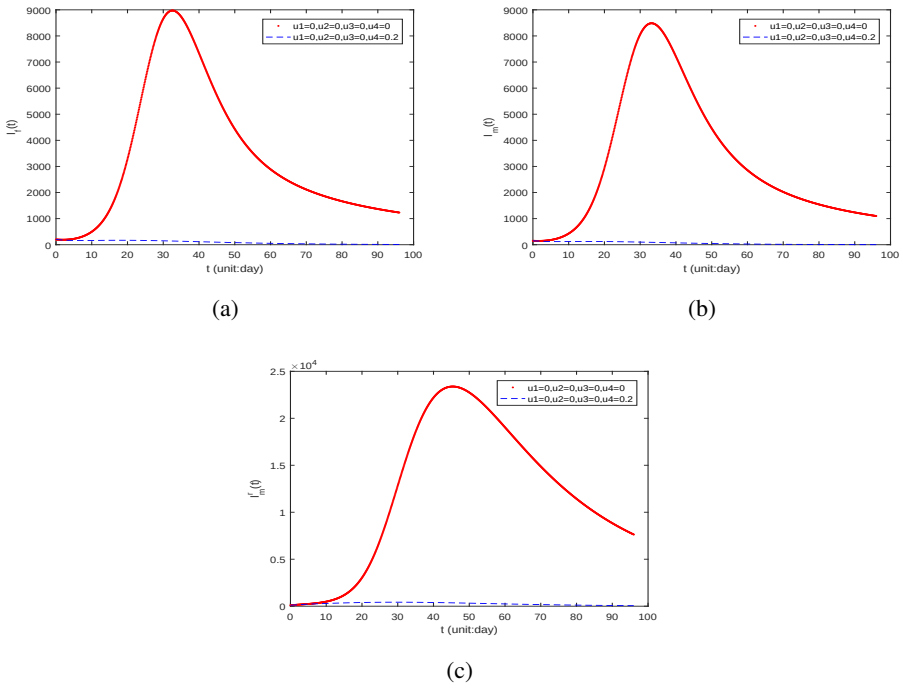

Figure 5. Effects of reducing mosquito populations with mild ($u_4 = 0.2$) on the time varying plots of the number of I_f, I_m, I_m^r.

Figure 7 shows the change in the number of infected persons (I_f, I_m, I_m^r) when only control measure $u3$ is added and other control measures are zero. As can be seen from the figure, due to the large number of infected people in the outbreak of the disease, only implementing the control strategy of delaying pregnancy ($u_3 = 0.5$) has little impact on the number of infected people. Figure 8 shows the change in the number of infected persons (I_f, I_m, I_m^r) over time when all four control measures were implemented. As can be seen from the figure, only minor control measures are needed to quickly control the outbreak and wipe out the disease in a short time. Figure 9 shows the change of the number of infected persons (I_f, I_m, I_m^r) over time under the optimal control measures. We know that under optimal control, the disease rarely breaks out and is most effectively controlled.

Reducing the mosquito population is the most effective way to control the spread of the disease by comparing several control measures. In conclusion, rapid control of a Zika outbreak requires a combination of mosquito-borne control strategies and internal human control strategies. The strategy of delaying pregnancy may be effective because of the greater adverse effects of Zika virus infection in newborns and the possibility that delaying pregnancy may reduce the number of infections in newborns.

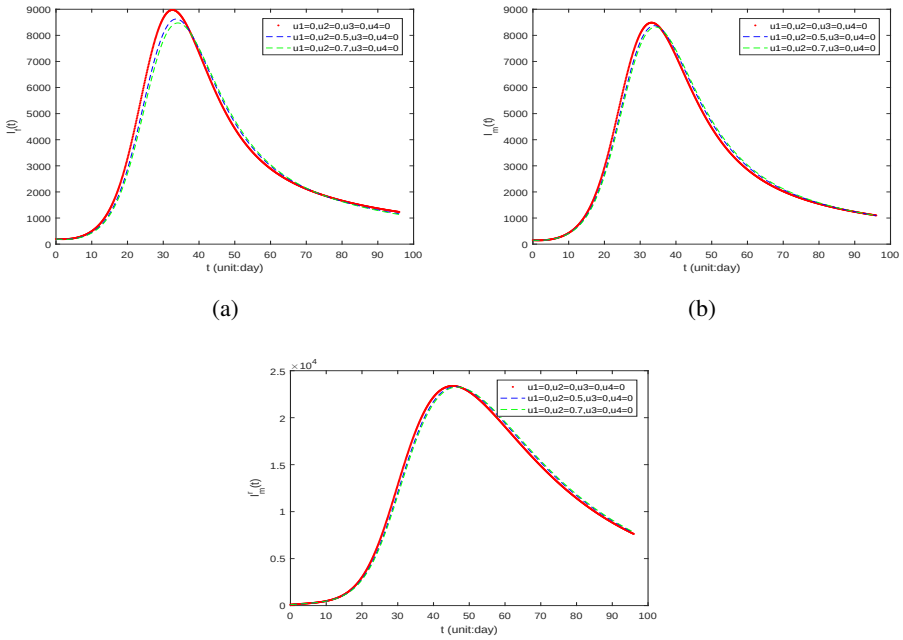

Figure 6. Effects of reducing the probability of sexual transmission with moderate ($u_2 = 0.5$) and strict ($u_2 = 0.7$) on the time varying plots of the number of I_f, I_m, I_m^r.

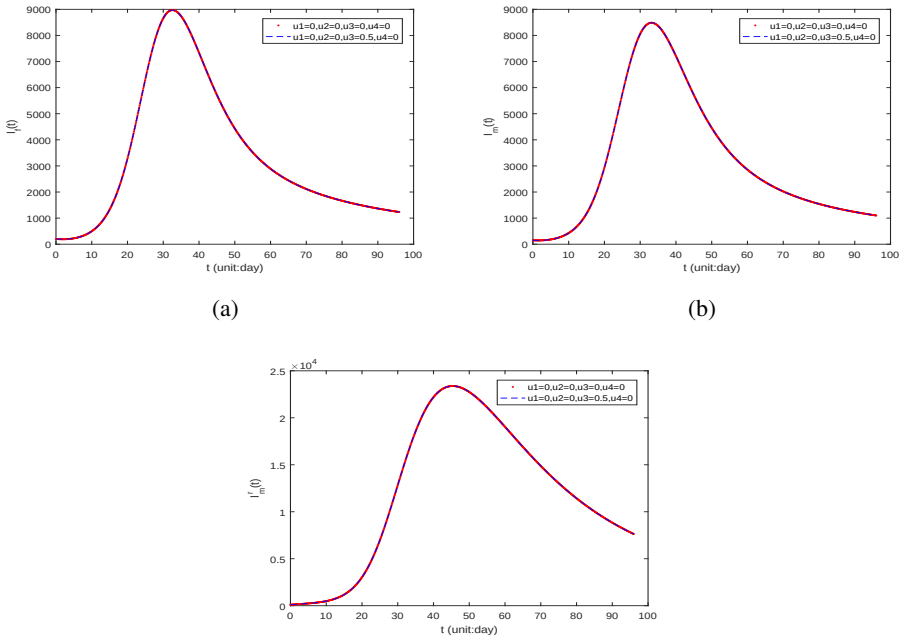

Figure 7. Effects of delaying pregnancy on the time varying plots of the number of I_f, I_m, I_m^r.

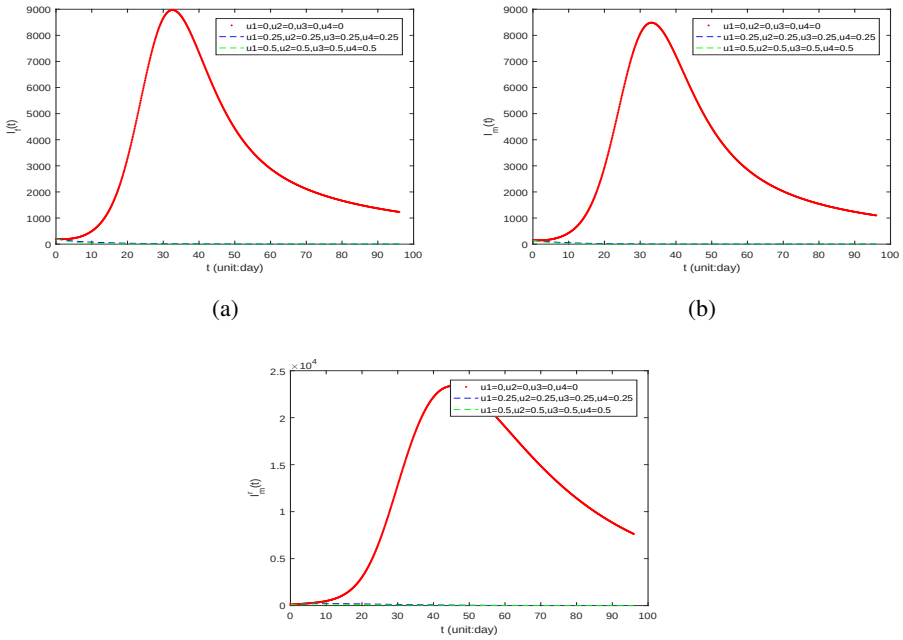

Figure 8. Effects of reducing the probability of sexual transmission, delayed pregnancy and reduce mosquito populations with none ($u_1 = 0, u_2 = 0, u_3 = 0, u_4 = 0$), mild ($u_1 = 0.25, u_2 = 0.25, u_3 = 0.25, u_4 = 0.25$) and moderate ($u_1 = 0.5, u_2 = 0.5, u_3 = 0.5, u_4 = 0.5$) on the time varying plots of the number of I_f, I_m, I_m^r.

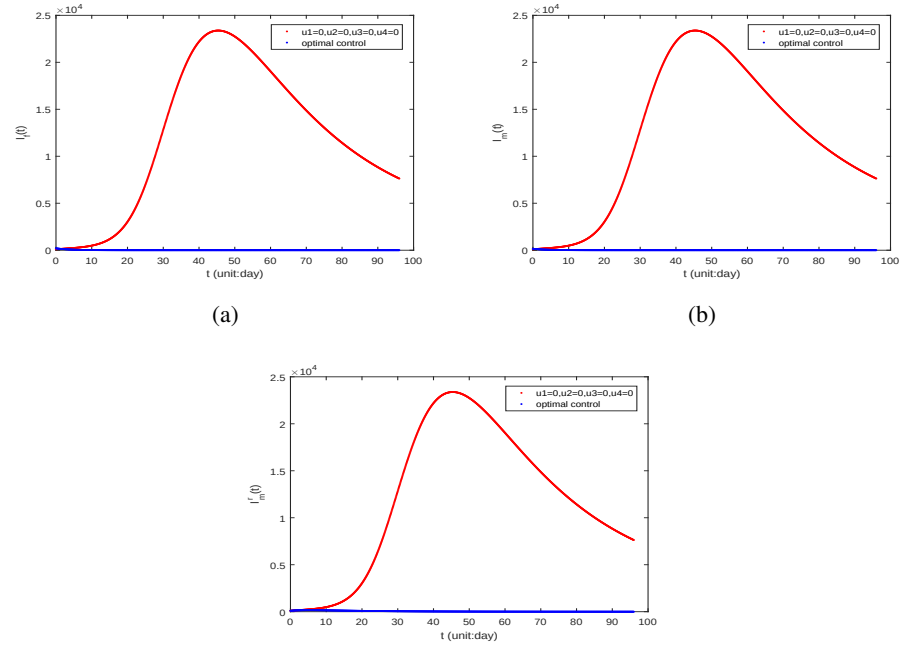

Figure 9. Time varying plots of the number of I_f, I_m, I_m^r under optimal control.

6. Discussion and conclusions

We develop a Zika virus transmission model with mosquito-borne transmission, sexual transmission and vertical transmission. Because sexual transmission of Zika is mainly male to female, we have differentiated the genders to more accurately represent the method of sexual transmission. Vertical transmission distinguishes between vertical transmission by mosquitoes and vertical transmission by humans. The basic reproduction number is obtained by the next generation matrix method. The global stability of the disease-free equilibrium is derived. The existence and mathematical expression of optimal control are obtained by using Pontryagin's maximum principle.

Based on the data of Zika virus in Colombia from 2015 to 2017, the unknown parameters of the model are estimated by MCMC algorithm. The reason why the fitted curve was not consistent with the actual cases was that the corresponding control measures adopted in Columbia in June 2016 led to a rapid decline in the number of infections after June 2016. A comparison of the four control strategies revealed that a combination of mosquito vector control and internal human control is necessary to bring the disease under control quickly. So in order to quickly control the spread of the Zika virus, we need to not only control the mosquito population and avoid contact with mosquitoes, but also increase awareness of disease transmission in humans and reduce sexual transmission. The Zika virus can cause severe brain defects in newborns and control strategies to delay pregnancy may reduce the number of infections in newborns. However, the change in the number of neonatal infected persons is not clearly indicated in this paper, which will likely be related to follow-up work in this respect.

Acknowledgments

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Conflict of interest

The authors declare that they have no conflict of interest.

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