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Dynamics of Zika Virus Model with Nonlinear Incidence and Optimal Control Strategies

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Abstract: We formulate and analyze Zika virus transmission model with three nonlinear forces of infection from infected mosquito, asymptomatic and symptomatic humans. The sensitivity indexes of the associated parameters of the model with respect to the basic reproduction number are calculated to identify intervention strategies for prevention and control of Zika virus. Multiple time-dependent optimal controls are considered. The analysis based on the use of optimal control theory made popular by Pontryagin's maximum principle is carried out, and the resulting optimality system is quantitatively simulated to investigate the impact of the controls on the dynamics of Zika virus. In addition, the effects of non-linearity of the forces of infection and other key parameters on the disease transmission are illustrated.

Keywords: Zika virus, Basic reproduction number, Non-autonomous model, Optimal control

1 Introduction

In recent times, Zika virus (ZIKV) disease has been found to be an additional source of concerns to the public health. The disease can be transmitted to humans through sexual intercourse, and primarily through an intermediate vector – infected female *Aedes* mosquito. According to the World Health Organization (WHO), 69 countries or territories of the world have reported mosquito-borne ZIKV transmission while 13 countries or territories have reported human-to-human transmission of ZIKV [34]. More often than not, infections with Zika are asymptomatic because only 20% of infected humans develop symptoms such as mild fever, skin rashes, conjuctivitis, muscle and joint pain [9].

The emergence of Zika virus disease in humans occurred in 1952 in Uganda and the United Republic of Tanzania. Since then, there have been several outbreaks of ZIKV in Africa, Americas, Asia and the Pacific, with the first large outbreak reported from the Island of Yap (Federated States of Micronesia) in 2007 [35]. It has been projected that Brazil among the Americas will have the largest total number of infections by more than three fold, due to a combination of its size and suitability for transmission [29]. Complications like microcephaly and Guillain-Barré syndrome have been attributed to the effects of ZIKV infections while links to other neurological disorders are also being investigated ([5], [7], [22], [35]).

Mathematical epidemiological models have been developed to broaden the understanding of the transmission dynamics of diseases. More importantly, the models play great roles in influencing the decision-making processes regarding intervention strategies for preventing and controlling the emergence and reemergence of the disease. A number of mathematical studies on ZIKV transmission dynamics have been carried out lately. Kucharski *et al* [15] used a compartmental mathematical model to examine the 2013–14 outbreak on the six major archipelagos of French Polynesia. Gao *et al* [11] studied a model to investigate the impact of mosquito-borne and sexual transmission on the spread and control of ZIKV. In [14], the stability analysis of infectious state of ZIKV in many types of population was presented with a view to taking necessary precautions against upcoming epidemic.

Agusto *et al* [1] analyzed a deterministic model of ZIKV by incorporating human vertical transmission of the virus, the birth of babies with microcephaly and asymptomatically infected individuals. Padmanabhan *et al* [30] considered ZIKV model that incorporates both sexual and vector transmission modes with constant

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preventive parameters. Readers may also see ([13], [16], [20], [26]) among others for some mathematical perspectives on ZIKV transmission dynamics. In another perspective, the time-dependent optimal control functions have been incorporated into a few ZIKV models with a view to exploring prevention and control measures for the disease spread ([3], [8], [19], [32]).

In this paper, a mathematical model is designed to analyse the ZIKV transmission dynamics with nonlinear forces of infection induced by infected mosquito, asymptomatic and symptomatic infectious humans. The optimal control analysis is performed on the non-autonomous version of the formulated model. The effects of the nonlinearity of the incidence terms and the five control variables, including human-mosquito contact prevention, human-human sexual contact prevention, routine check for asymptomatic individuals, treatment and mosquito reduction strategies, in preventing and controlling the spread process of the ZIKV disease are investigated.

The rest of the paper is arranged as follows. Section 2 presents the formulation of the autonomous model with its basic qualitative properties. In Section 3, the sensitivity indexes of the parameters of the model with respect to the basic reproduction number are investigated. In Section 4, optimal control analysis of the non-autonomous model based on Pontryagin's maximum principle is carried out. Section 5 provides the discussion of the quantitative results obtained from simulations. In Section 6, the paper is wrapped up with concluding remarks.

2 Model formulation

The human population comprises four compartments, namely susceptible $S_H(t)$ (number of humans who are liable to be infected with ZIKV at time t), asymptomatic infected $A_H(t)$ (number of humans infected with ZIKV without showing symptoms of the disease but are capable of infecting both humans and mosquitoes at time t), symptomatic infected $I_H(t)$ (number of infected humans with ZIKV symptoms and are capable of transmitting the disease to both humans and mosquitoes at time t) and recovered $R_H(t)$ (those recovered from the ZIKV infection spontaneously or therapeutically). Then, the total human population at time t, denoted by $N_H(t)$ is given by $N_H(t) = S_H(t) + A_H(t) + I_H(t) + R_H(t)$.

The total vector (mosquito) population at time t, denoted by $N_V(t)$, is sub-classified into susceptible $S_V(t)$ (number of mosquitoes not yet infected with ZIKV but are capable of being infected by both asymptomatic and symptomatic infectious humans at time t) and infected $I_V(t)$ (number of infectious mosquitoes that are capable of transmitting ZIKV to the susceptible humans at time t), so that $N_V(t) = S_V(t) + I_V(t)$.

Let the recruitment terms for human and mosquito populations (both assumed susceptible) be represented, respectively by Λ_H and Λ_V. Since Zika virus is a vector-borne and sexually transmitted disease, then the susceptible human population can be infected through the bite of the infectious female *Aedes* mosquito (at a rate $\beta_1 b$), and through sexual intercourse with both symptomatic (at a rate β_2) and asymptomatic infected individuals (at a rate β_3). Where β_1, β_2 and β_3 are the disease transmission probabilities caused by I_V, I_H and A_H respectively, and b is the mosquito biting rate. It is apt to assume that $\beta_2 \leq \beta_3$ since infectious humans who show the symptoms of ZIKV disease are less likely to engage in sexual intercourse than the asymptomatic individuals. Further, the infection of the susceptible human population generates either the population of asymptomatic or symptomatic infectious humans. If it is assumed that $0 < \theta < 1$ is the fraction of the susceptible human population who becomes asymptomatic due to infection, then the remaining fraction $(1-\theta)$ is symptomatic due to infection. The population of the recovered human R_H is generated by the spontaneous recovery of both asymptomatic and symptomatic infectious individuals at rates γ_H and σ_H respectively. The natural death rate of human is represented by μ_H.

On the other hand, the population of the susceptible mosquito is reduced by infection due to effective contact with symptomatic (at a rate $\beta_2 b$) and asymptomatic (at a rate $\beta_3 b$) humans. As a result of the infection of the susceptible mosquito, the population of the infectious mosquito is produced. Let μ_V be the natural death rate for both susceptible and infectious mosquito populations.

Based on the foregoing assumptions, a system of ordinary differential equations describing the ZIKV transmission is given by

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \left(\tfrac{\beta_1 b I_V}{1+\alpha_1 I_V} + \tfrac{\beta_2 I_H}{1+\alpha_2 I_H} + \tfrac{\beta_3 A_H}{1+\alpha_3 A_H}\right) S_H \\
&\quad -\mu_H S_H \\
\frac{dA_H}{dt} &= \theta\left(\tfrac{\beta_1 b I_V}{1+\alpha_1 I_V} + \tfrac{\beta_2 I_H}{1+\alpha_2 I_H} + \tfrac{\beta_3 A_H}{1+\alpha_3 A_H}\right) S_H \\
&\quad -(\gamma_H + \mu_H) A_H \\
\frac{dI_H}{dt} &= (1-\theta)\left(\tfrac{\beta_1 b I_V}{1+\alpha_1 I_V} + \tfrac{\beta_2 I_H}{1+\alpha_2 I_H} + \tfrac{\beta_3 A_H}{1+\alpha_3 A_H}\right) S_H \\
&\quad -(\sigma_H + \mu_H) I_H \\
\frac{dR_H}{dt} &= \gamma_H A_H + \sigma_H I_H - \mu_H R_H \\
\frac{dS_V}{dt} &= \Lambda_V - \left(\tfrac{\beta_2 b I_H}{1+\alpha_2 I_H} + \tfrac{\beta_3 b A_H}{1+\alpha_3 A_H}\right) S_V - \mu_V S_V \\
\frac{dI_V}{dt} &= \left(\tfrac{\beta_2 b I_H}{1+\alpha_2 I_H} + \tfrac{\beta_3 b A_H}{1+\alpha_3 A_H}\right) S_V - \mu_V I_V,
\end{aligned} \tag{1}$$

where the nonlinear forces of infection induced by infectious mosquito, symptomatic and asymptomatic infectious humans are given, respectively, by $\frac{I_V}{1+\alpha_1 I_V}$, $\frac{I_H}{1+\alpha_2 I_H}$ and $\frac{A_H}{1+\alpha_3 A_H}$ which are of saturated form introduced by Capasso and Serio [6]. Noting that α_1, α_2

and α_3 are positive saturation constants that determine the level at which the force of infection saturates. This nonlinear force of infection of saturated form which can also be referred to as Holling-type II (see, e.g., [18], [31]) has been used in vector-borne related disease (malaria) models ([24], [27]). The choice of this form is informed by the fact that the number of effective contacts (sexual or vector transmission) between susceptible and infectious individuals may saturate at high infective levels due to crowding of infectious individuals or due to the precautionary strategies put in place by the susceptible individuals.

2.1 Basic properties

Since model (1) monitors human and vector populations, all its associated parameters are nonnegative. It thus remains to show that the state variables of the model (1) are nonnegative.

Theorem 1. *The solutions $S_H(t)$, $A_H(t)$, $I_H(t)$, $R_H(t)$, $S_V(t)$, $I_V(t)$, of the ZIKV model (1) with nonnegative initial data $S_H(0)$, $A_H(0)$, $I_H(0)$, $R_H(0)$, $S_V(0)$, $I_V(0)$, remain nonnegative for all time $t > 0$.*

Proof. The first equation of model (1) can be written as $\frac{dS_H}{dt} + \left(\frac{\beta_1 bI_V}{1+\alpha_1 I_V} + \frac{\beta_2 I_H}{1+\alpha_2 I_H} + \frac{\beta_3 A_H}{1+\alpha_3 A_H} + \mu_H\right) S_H \geq 0$, so that

$$\frac{d}{dt}\left[S_H(t)\exp\left(\int_0^t \frac{\beta_1 bI_V(\zeta)}{1+\alpha_1 I_V(\zeta)} + \frac{\beta_2 I_H(\zeta)}{1+\alpha_2 I_H(\zeta)} + \frac{\beta_3 A_H(\zeta)}{1+\alpha_3 A_H(\zeta)} d\zeta + \mu_H t\right)\right] \geq 0. \tag{2}$$

Integrating (2) gives

$$S_H(t) \geq S_H(0)\exp\left[-\left(\int_0^t \frac{\beta_1 bI_V(\zeta)}{1+\alpha_1 I_V(\zeta)} + \frac{\beta_2 I_H(\zeta)}{1+\alpha_2 I_H(\zeta)} + \frac{\beta_3 A_H(\zeta)}{1+\alpha_3 A_H(\zeta)} d\zeta + \mu_H t\right)\right] > 0. \tag{3}$$

In a similar manner, it can be shown that other state variables $A_H(t)$, $I_H(t)$, $R_H(t)$, $S_V(t)$ and $I_V(t)$ are nonnegative for all $t > 0$. □

Next, consider the biologically feasible region defined by $\mathfrak{D} = \mathfrak{D}_H \times \mathfrak{D}_V \subset \mathbb{R}_+^4 \times \mathbb{R}_+^2$, where

$$\mathfrak{D}_H = \left\{(S_H, A_H, I_H, R_H) \in \mathbb{R}_+^4 : N_H \leq \frac{\Lambda_H}{\mu_H}\right\}$$

and

$$\mathfrak{D}_V = \left\{(S_V, I_V,) \in \mathbb{R}_+^2 : N_V \leq \frac{\Lambda_V}{\mu_V}\right\}.$$

It can be shown that $\mathfrak{D}$ is a positive invariant region.

Theorem 2. *The region $\mathfrak{D}$ is positively invariant with respect to the model (1).*

Proof. The rate of change of the total human population is given by $\frac{dN_H}{dt} = \Lambda_H - \mu_H N_H$ which on solving yields $N_H(t) = N_H(0)e^{-\mu_H t} + \frac{\Lambda_H}{\mu_H}(1 - e^{-\mu_H t})$. A similar approach for the total mosquito population gives $N_V(t) = N_V(0)e^{-\mu_V t} + \frac{\Lambda_V}{\mu_V}(1 - e^{-\mu_V t})$. It follows that $N_H(t) \to \frac{\Lambda_H}{\mu_H}$ and $N_V(t) \to \frac{\Lambda_V}{\mu_V}$ as $t \to \infty$. In particular, $N_H(t) \leq \frac{\Lambda_H}{\mu_H}$ if $N_H(0) \leq \frac{\Lambda_H}{\mu_H}$ and $N_V(t) \leq \frac{\Lambda_V}{\mu_V}$ if $N_V(0) \leq \frac{\Lambda_V}{\mu_V}$. Hence, $\mathfrak{D}$ is positively invariant. □

Therefore, it is sufficient to study the dynamics of model (1) in region $\mathfrak{D}$ where the model can be considered as being epidemiologically and mathematically well-posed [12].

3 Basic reproduction number and sensitivity analysis

An important epidemiological threshold which measures the spread potential of an infectious disease in a given population is a basic reproduction number, usually denoted by $\mathscr{R}_0$. For ZIKV model (1), $\mathscr{R}_0$ can be defined as the average number of secondary infections, in a completely susceptible human or mosquito population, caused by a typical infectious (mosquito or symptomatic or asymptomatic) individual over its period of infectiousness. Using the technique and notation in [33], the matrix F of the new infection terms and matrix V of the remaining transition terms are given, respectively, by

$$F = \begin{pmatrix} \theta\beta_3\frac{\Lambda_H}{\mu_H} & \theta\beta_2\frac{\Lambda_H}{\mu_H} & \theta\beta_1\frac{\Lambda_V}{\mu_V} \\ (1-\theta)\beta_3\frac{\Lambda_H}{\mu_H} & (1-\theta)\beta_2\frac{\Lambda_H}{\mu_H} & (1-\theta)\beta_1\frac{\Lambda_V}{\mu_V} \\ \beta_3\frac{\Lambda_V}{\mu_V} & \beta_2\frac{\Lambda_V}{\mu_V} & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \gamma_H + \mu_H & 0 & 0 \\ -\alpha & \sigma_H + \mu_H & 0 \\ 0 & 0 & \mu_V \end{pmatrix}.$$

Consequently, the basic reproduction number of the ZIKV model (1) is given by

$$\mathscr{R}_0 = \rho(FV^{-1}) = \frac{1}{2}\left[\frac{\Lambda_H \mathscr{R}_*}{\mu_H} + \sqrt{\mathscr{R}_*\left(\frac{\Lambda_H^2 \mathscr{R}_*}{\mu_H^2} + \frac{4\beta_1 b^2 \Lambda_V^2}{\mu_V^3}\right)}\right], \tag{4}$$

where

$$\mathscr{R}_* = \frac{(1-\theta)\beta_2}{(\sigma_H + \mu_H)} + \frac{\theta\beta_3}{(\gamma_H + \mu_H)}.$$

It is necessary at this stage to carry out sensitivity analysis of the model (1) with a view to determining the relative change in the basic reproduction number $\mathscr{R}_0$ to the relative change in its associated parameters. In what follows, the normalized forward sensitivity index of $\mathscr{R}_0$ that depends differentiably on any of its parameter p is defined as

$$\Upsilon_p^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial p} \times \frac{p}{\mathscr{R}_0}. \tag{5}$$

Based on the values of the parameters in Table 1, the sensitivity indexes presented in Table 2 with respect to all the parameters in (4) are calculated using (5). The parameters with positive sensitivity index indicate that an increase (or decrease) in the value of each of the parameters leads to a corresponding increase (or decrease) in the basic reproduction number. Conversely, the parameters with negative index means that an increase (or decrease) in the value of the parameters causes a decrease (or increase) in the basic reproduction number of the ZIKV model (1).

Table 2: Sensitivity indexes of $\mathscr{R}_0$ with respect to the model parameters

Parameter	Sensitivity index
Λ_H	0.03972
Λ_V	0.96028
β_1	0.48014
β_2	0.03999
β_3	0.47987
b	0.96028
θ	0.31991
μ_H	−0.03984
μ_V	−1.44042
γ_H	−0.47976
σ_H	−0.03998

For example, increasing the natural death rate of mosquito, μ_V, by 10% decreases the basic reproduction number, $\mathscr{R}_0$, by 14%. Hence, sensitivity analysis, when carried out on a disease model, helps in focusing on appropriate intervention strategies for preventing and controlling the spread of the disease [25].

Table 1: Description of variables and parameters

Variable	Description			
$S_H(t)$	Population of susceptible humans			
$A_H(t)$	Asymptomatic infectious humans population			
$I_H(t)$	Symptomatic infectious humans population			
$R_H(t)$	Population of recovered humans			
$S_V(t)$	Population of susceptible mosquitoes			
$I_V(t)$	Population of infectious mosquitoes			
$N_H(t)$	Total human population			
$N_V(t)$	Total mosquito population			
Parameter	Description	Range	Baseline value	Source
Λ_H	Human recruitment term		0.000011	[19]
Λ_V	Mosquito recruitment term	0.05-0.5	0.13	[27]
β_1	Transmission probability per contact by infectious mosquito	0-0.75	0.4	[11], [19], [30]
β_2	Transmission probability per contact by symptomatic infectious human	0-0.5	0.05	[19]
β_3	Transmission probability per contact by asymptomatic infectious human	0-1	0.15	Assumed
b	Mosquito biting rate		0.5	[1], [11]
θ	Fraction of susceptible human that becomes asymptomatic due to infection	0-1	0.8	Assumed
μ_H	Human natural death rate	$\frac{1}{80\times365} - \frac{1}{45\times365}$	0.000046	[3], [21]
μ_V	Mosquito natural death rate	0.05-0.5	0.066	[4], [24]
γ_H	Recovery rate for asymptomatic human	0.05-0.33	0.2	Assumed
σ_H	Recovery rate for symptomatic human	0.0667-0.33	0.2	[11]

4 Analysis of optimal control problem

Arising from the results of the sensitivity analysis, time-dependent optimal control measures are incorporated into the model (1) in this section. And optimal control theory based on the Pontryagin's maximum principle [28] is employed to obtain the necessary conditions for the optimal strategies aimed at preventing and controlling the disease spread. Thus, the following five optimal control variables are considered:

(i). The control $u_1(t)$ which represents a measure for preventing mosquito-borne ZIKV transmission through the use of insecticide-treated bed nets and skin repellent lotions.
(ii). The control $u_2(t)$ which represents a measure for preventing human-to-human sexual ZIKV transmission through the use of condoms.
(iii). The control $u_3(t)$ represents a surveillance measure or routine check for identifying and treating asymptomatic ZIKV infected individuals.
(iv). The control $u_4(t)$ represents a treatment measure for symptomatic individuals.
(v). The control $u_5(t)$ represents a mosquito-reduction strategy targeted at the mosquito breeding sites and indoors through the use of residual spraying.

Based on the use of the aforementioned five control variables, the autonomous model (1) becomes ZIKV model (6) governed by a non-autonomous system of ordinary differential equations of the form:

$$\begin{cases} \frac{dS_H}{dt} = \Lambda_H - \left(\frac{(1-u_1(t))\beta_1 bI_V}{1+\alpha_1 I_V} + \frac{(1-u_2(t))\beta_2 I_H}{1+\alpha_2 I_H}\right) S_H \\ \quad + \left(\frac{(1-u_2(t))\beta_3 A_H}{1+\alpha_3 A_H}\right) S_H - \mu_H S_H \\ \frac{dA_H}{dt} = \theta\left(\frac{(1-u_1(t))\beta_1 bI_V}{1+\alpha_1 I_V} + \frac{(1-u_2(t))\beta_2 I_H}{1+\alpha_2 I_H} + \frac{(1-u_2(t))\beta_3 A_H}{1+\alpha_3 A_H}\right) S_H \\ \quad -(\gamma_H + k_1 u_3(t) + \mu_H) A_H \\ \frac{dI_H}{dt} = (1-\theta)\left(\frac{(1-u_1(t))\beta_1 bI_V}{1+\alpha_1 I_V} + \frac{(1-u_2(t))\beta_2 I_H}{1+\alpha_2 I_H}\right) S_H \\ \quad +(1-\theta)\left(\frac{(1-u_2(t))\beta_3 A_H}{1+\alpha_3 A_H}\right) S_H - \sigma_H I_H \\ \quad +(k_2 u_4(t) + \mu_H) I_H \\ \frac{dR_H}{dt} = (\gamma_H + k_1 u_3(t)) A_H + (\sigma_H + k_2 u_4(t)) I_H - \mu_H R_H \\ \frac{dS_V}{dt} = (1-u_5(t))\Lambda_V - \left(\frac{(1-u_1(t))\beta_2 bI_H}{1+\alpha_2 I_H} + \frac{(1-u_1(t))\beta_3 bA_H}{1+\alpha_3 A_H}\right) S_V \\ \quad -(\mu_V + c_0 u_5(t)) S_V \\ \frac{dI_V}{dt} = \left(\frac{(1-u_1(t))\beta_2 bI_H}{1+\alpha_2 I_H} + \frac{(1-u_1(t))\beta_3 bA_H}{1+\alpha_3 A_H}\right) S_V \\ \quad -(\mu_V + c_0 u_5(t)) I_V, \end{cases} \tag{6}$$

where k_1, k_2 and c_0 are positive rate constants. The goal of the optimal control strategies is to minimize the number of infectious humans (asymptomatic and symptomatic) and the vector population while keeping the costs of applying the controls; $u_1(t)$, $u_2(t)$, $u_3(t)$, $u_4(t)$ and $u_5(t)$, as low as possible. To do this, the use is made of the objective functional J given by

$$J = \int_0^{t_f} \left(B_1 A_H + B_2 I_H + B_3 (S_V + I_V) + \frac{1}{2}\left(c_1 u_1^2 + c_2 u_2^2 + c_3 u_3^2 + c_4 u_4^2 + c_5 u_5^2\right)\right) dt, \tag{7}$$

where B_1, B_2, B_3, c_1, c_2, c_3, c_4 and c_5 are positive weight constants. The term $c_1 u_1^2$ represents the cost associated with mosquito-human contact protection efforts where $c_2 u_2^2$ represents the cost associated with human-human sexual contact protection efforts. Further, the terms $c_3 u_3^2$, $c_4 u_4^2$ and $c_5 u_5^2$ represent the cost associated with routine check efforts for diagnosing asymptomatic individuals, treatment efforts for symptomatic patients and mosquito-reduction efforts respectively. The costs of controls have been chosen to be quadratic in accordance with what is in other literature on epidemic models ([2], [21], [23], [36]).

Of interest is to seek an optimal control quintuple u_1^*, u_2^*, u_3^*, u_4^* and u_5^* such that

$$J(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*) = \min\{J(u_1, u_2, u_3, u_4, u_5) : u_1, u_2, u_3, u_4, u_5 \in \mathscr{U}\}, \tag{8}$$

where the control set is defined by
$\mathscr{U} = \{u_i : 0 \le u_i(t) \le 1, \text{Lebesgue measurable}, \in [0, t_f]\}$ for $i = 1, ..., 5$.

4.1 Existence of an optimal control

To determine the existence of an optimal control to the non-autonomous model governed by (6), a result from Fleming and Rischel [10] is employed, where the following properties must be satisfied:

P1. The control set is convex and closed.
P2. The right hand side of the state system is bounded by a linear function in the state and control variables.
P3. The integrand of the objective functional is convex with respect to the control, and
P4. There exist constants $b_1, b_2 > 0$ and $b_3 > 1$ such that the objective functional is bounded below by $b_1\left(|u_i|^2\right)^{\frac{b_3}{2}} - b_2$.

One sees that P1 is satisfied by the definition of the control set $u_1, u_2, u_3, u_4, u_5 \in \mathscr{U}$. Considering Theorem 2, the state variables are *priori* bounded so that the right hand side of the non-autonomous system given by (6) satisfies P2. Furthermore, the integrand in the objective functional J obtained by (7) is convex on $\mathscr{U}$ satisfying P3. Finally, P4 is satisfied since the state variables are bounded, then some constants $b_1, b_2 > 0$ and $b_3 > 1$ can be obtained satisfying the bound,

$\mathscr{L} \ge b_1\left(|u_1|^2 + |u_2|^2 + |u_3|^2 + |u_4|^2 + |u_5|^2\right)^{\frac{b_3}{2}} - b_2$, where $\mathscr{L}$ is the Lagrangian referred to as the integrand of the objective functional given by (7).

Arising from the above analysis, the following existence result is claimed.

Lemma 1. *If the objective functional J given by (7) is defined on a set of bounded control $\mathscr{U}$ and is subject to the non-autonomous system (6) with initial conditions at $t=0$, then $\exists$ an optimal control $u^*=(u_1^*,u_2^*,u_3^*,u_4^*,u_5^*)$ such that $J(u^*)=\min\{J(u_i):u_i\in\mathscr{U}\}$, for $i=1,2,...,5$.*

The Pontryagin's maximum principle [28] converts system (6), with (7) and (8) into a problem of minimizing pointwise a Hamiltonian $\mathscr{H}$, with respect to u_1, u_2, u_3, u_4 and u_5 of the form given by

$$\begin{aligned}\mathscr{H} &= B_1A_H+B_2I_H+B_3(S_V+I_V)\\ &+\tfrac{1}{2}\left(c_1u_1^2+c_2u_2^2+c_3u_3^2+c_4u_4^2++c_5u_5^2\right)\\ &+\lambda_1\left[\Lambda_H-\left(\tfrac{(1-u_1)\beta_1bI_V}{1+\alpha_1I_V}+\tfrac{(1-u_2)\beta_2I_H}{1+\alpha_2I_H}\right.\right.\\ &\left.\left.+\tfrac{(1-u_2)\beta_3A_H}{1+\alpha_3A_H}\right)S_H-\mu_HS_H\right]\\ &+\lambda_2\left[\theta\left(\tfrac{(1-u_1)\beta_1bI_V}{1+\alpha_1I_V}+\tfrac{(1-u_2)\beta_2I_H}{1+\alpha_2I_H}\right.\right.\\ &\left.\left.+\tfrac{(1-u_2)\beta_3A_H}{1+\alpha_3A_H}\right)S_H-(\gamma_H+k_1u_3+\mu_H)A_H\right]\\ &+\lambda_3\left[(1-\theta)\left(\tfrac{(1-u_1)\beta_1bI_V}{1+\alpha_1I_V}+\tfrac{(1-u_2)\beta_2I_H}{1+\alpha_2I_H}\right.\right.\\ &\left.\left.+\tfrac{(1-u_2)\beta_3A_H}{1+\alpha_3A_H}\right)S_H-(\sigma_H+k_2u_4+\mu_H)I_H\right]\\ &+\lambda_4[(\gamma_H+k_1u_3)A_H+(\sigma_H+k_2u_4)I_H\\ &-\mu_HR_H]\\ &+\lambda_5\left[(1-u_5)\Lambda_V-\left(\tfrac{(1-u_1)\beta_2bI_H}{1+\alpha_2I_H}\right.\right.\\ &\left.\left.+\tfrac{(1-u_1)\beta_3bA_H}{1+\alpha_3A_H}\right)S_V-(\mu_V+c_0u_5)S_V\right]\\ &+\lambda_6\left[\left(\tfrac{(1-u_1)\beta_2bI_H}{1+\alpha_2I_H}+\tfrac{(1-u_1)\beta_3bA_H}{1+\alpha_3A_H}\right)S_V\right.\\ &\left.-(\mu_V+c_0u_5)I_V\right],\end{aligned}\tag{9}$$

where λ_i, $i=1,2,...,6$, are the adjoint variables. The next result presents the adjoint system and characterization of the optimal control.

Theorem 3. *Given an optimal control quintuple $(u_1^*,u_2^*,u_3^*,u_4^*,,u_5^*)$ that minimizes J over $\mathscr{U}$ and solutions of the associated non-autonomous system (6), there exist adjoint variables λ_i, $i=1,2,...,6$, satisfying*

$$\begin{cases}\frac{d\lambda_1}{dt}=(\lambda_1-\theta\lambda_2-(1-\theta)\lambda_3)\left[\left(\frac{(1-u_1)\beta_1bI_V}{1+\alpha_1I_V}+\frac{(1-u_2)\beta_2I_H}{1+\alpha_2I_H}\right.\right.\\ \qquad\left.\left.+\frac{(1-u_2)\beta_3A_H}{1+\alpha_3A_H}\right)+\mu_H\lambda_1\right]\\ \frac{d\lambda_2}{dt}=(\lambda_1-\theta\lambda_2-(1-\theta)\lambda_3)\frac{(1-u_2)\beta_3S_H}{(1+\alpha_3A_H)^2}\\ \qquad+(\lambda_5-\lambda_6)\frac{(1-u_1)\beta_3bS_V}{(1+\alpha_3A_H)^2}+(\lambda_2-\lambda_4)(\gamma_H+k_1u_3)\\ \qquad+\mu_H\lambda_2-B_1\\ \frac{d\lambda_3}{dt}=(\lambda_1-\theta\lambda_2-(1-\theta)\lambda_3)\frac{(1-u_2)\beta_2S_H}{(1+\alpha_2I_H)^2}\\ \qquad+(\lambda_5-\lambda_6)\frac{(1-u_1)\beta_2bS_V}{(1+\alpha_2I_H)^2}+(\lambda_3-\lambda_4)(\sigma_H+k_2u_4)\\ \qquad+\mu_H\lambda_3-B_2\\ \frac{d\lambda_4}{dt}=\mu_H\lambda_4\\ \frac{d\lambda_5}{dt}=(\lambda_5-\lambda_6)(1-u_1)b\left(\frac{\beta_2I_H}{1+\alpha_2I_H}+\frac{\beta_3A_H}{1+\alpha_3A_H}\right)\\ \qquad+(\mu_V+c_0u_5)\lambda_5-B_3\\ \frac{d\lambda_6}{dt}=(\lambda_1-\theta\lambda_2-(1-\theta)\lambda_3)\frac{(1-u_1)\beta_1bS_H}{(1+\alpha_1I_V)^2}\\ \qquad+(\mu_V+c_0u_5)\lambda_6-B_3\end{cases}\tag{10}$$

with transversality conditions

$$\lambda_i(t_f)=0,\ i=1,2,...,6.\tag{11}$$

Then, the optimal controls $(u_1^*,u_2^*,u_3^*,u_4^*,u_5^*)$ are given by

$$\begin{cases}u_1^*=\min\left\{\max\left\{0,\frac{1}{c_1}\left[((1-\theta)\lambda_3+\theta\lambda_2-\lambda_1)\frac{\beta_1bI_VS_H}{1+\alpha_1I_V}\right.\right.\right.\\ \qquad\left.\left.\left.+(\lambda_6-\lambda_5)\left(\frac{\beta_2bI_HS_V}{1+\alpha_2I_H}+\frac{\beta_3bA_HS_V}{1+\alpha_3A_H}\right)\right]\right\},1\right\},\\ u_2^*=\min\left\{\max\left\{0,\frac{1}{c_2}\left[(\theta\lambda_2-\lambda_1)\frac{\beta_2I_HS_H}{1+\alpha_2I_H}\right.\right.\right.\\ \qquad\left.\left.\left.+((1-\theta)\lambda_3-\lambda_1)\frac{\beta_3A_HS_H}{1+\alpha_3A_H}\right]\right\},1\right\},\\ u_3^*=\min\left\{\max\left\{0,\frac{(\lambda_2-\lambda_4)k_1A_H}{c_3}\right\},1\right\},\\ u_4^*=\min\left\{\max\left\{0,\frac{(\lambda_3-\lambda_4)k_2I_H}{c_4}\right\},1\right\},\\ u_5^*=\min\left\{\max\left\{0,\frac{(\Lambda_V+c_0S_V)\lambda_5+c_0I_V\lambda_6}{c_5}\right\},1\right\}.\end{cases}\tag{12}$$

Proof. To do this, the use is made of the Hamiltonian $\mathscr{H}$ given by (9) following Pontryagin's maximum principle [28]. The differential equations (10) governing the adjoint variables λ_i, $i=1,...,6$, are obtained by taking the partial derivatives of $\mathscr{H}$ with respect to the corresponding state

variables, so that

$$\frac{d\lambda_1}{dt} = -\frac{\partial \mathscr{H}}{\partial S_H}, \quad \frac{d\lambda_2}{dt} = -\frac{\partial \mathscr{H}}{\partial A_H},$$

$$\frac{d\lambda_3}{dt} = -\frac{\partial \mathscr{H}}{\partial I_H}, \quad \frac{d\lambda_4}{dt} = -\frac{\partial \mathscr{H}}{\partial R_H},$$

$$\frac{d\lambda_5}{dt} = -\frac{\partial \mathscr{H}}{\partial S_V}, \quad \frac{d\lambda_6}{dt} = -\frac{\partial \mathscr{H}}{\partial I_V},$$

with terminal conditions (11). Further, the characterization of the optimal control given by (12) is derived by solving the partial differential equations; $\frac{\partial \mathscr{H}}{\partial u_1} = 0$, $\frac{\partial \mathscr{H}}{\partial u_2} = 0$, $\frac{\partial \mathscr{H}}{\partial u_3} = 0$, $\frac{\partial \mathscr{H}}{\partial u_4} = 0$ and $\frac{\partial \mathscr{H}}{\partial u_5} = 0$ for u_1^*, u_2^*, u_3^*, u_4^* and u_5^* respectively. This completes the proof. □

5 Simulations and discussion of results

The optimality system (state equations (6) coupled with adjoint equations (10)) is solved using an iterative method of order four Runge-Kutta scheme. The state equations are solved forward in time with initial guess for the controls over the simulated time. Owing to the transversality conditions (11), the adjoint equations are solved backward in time using the current iteration solutions of the state equations. Then the controls are updated by using a convex combination of the previous controls and the value from the characterization (12). This process continues until the difference between the values of unknowns at the previous iteration and that of the present iteration is negligibly small [17].

Using the parameter values provided in Table 1 such that $\mathscr{R}_0 = 2.03374 > 1$ with initial conditions $S_H(0) = 500$, $A_H(0) = 30$, $I_H(0) = 20$, $R_H(0) = 10$, $S_V(0) = 1000$ and $I_V(0) = 50$. The values of the weight constants in the objective functional (7) are chosen as $B_1 = 20$, $B_2 = 500$, $B_3 = 30$, $c_1 = 10$, $c_2 = 10$, $c_3 = 20$, $c_4 = 15$ and $c_5 = 20$. The effects of the optimal control strategies on the dynamical spread of ZIKV represented by model (6) are illustrated by considering the following few scenarios combined with the mosquito-reduction strategy:

Scenario 1: Prevention (u_1) and mosquito reduction measure (u_5)

The combined effects of the use of controls u_1 and u_5, for minimizing the objective functional (7), on the dynamical spread of ZIKV are shown in Figures 1(a)–1(d). It is observed that the sizes of asymptomatic humans decrease in the presence of controls u_1 and u_2 when compared with the case without controls. As expected, the magnitude of symptomatic infectious humans also reduces with control. Similarly, the population sizes of both susceptible and infectious mosquitoes decrease with control when compared with the case without control.

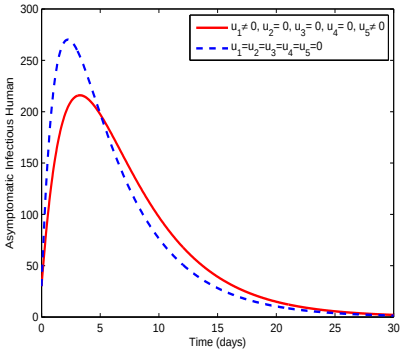

Fig. 1(a): Simulations of model (6) showing effects of controls u_1 and u_5 on asymptomatic human population.

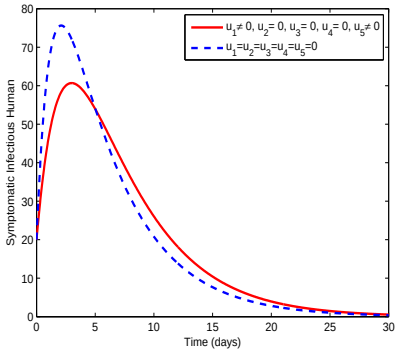

Fig. 1(b): Simulations of model (6) showing effects of controls u_1 and u_5 on symptomatic human population.

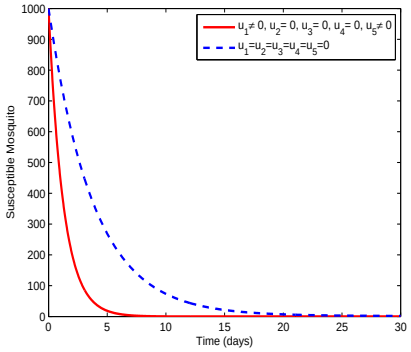

Fig. 1(c): Simulations of model (6) showing effects of controls u_1 and u_5 on susceptible mosquito population.

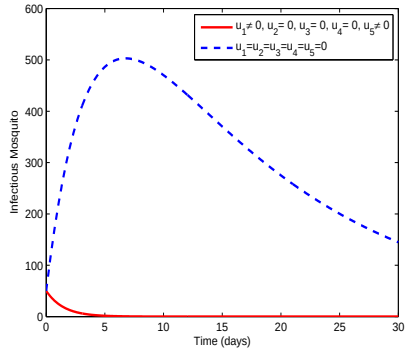

Fig. 1(d): Simulations of model (6) showing effects of controls u_1 and u_5 on infectious mosquito population.

Scenario 2: Prevention (u_1), routine check (u_3) and mosquito-reduction measure (u_5)

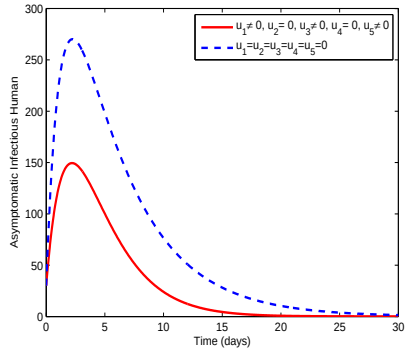

Fig. 2(a): Simulations of model (6) showing effects of controls u_1, u_3 and u_5 on asymptomatic human population.

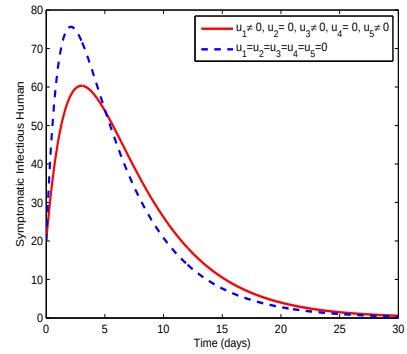

Fig. 2(b): Simulations of model (6) showing effects of controls u_1, u_3 and u_5 on symptomatic human population.

Figures 2(a)–2(d) illustrate how the combination of the controls u_1, u_3 and u_5 minimizes the objective functional (7). In this scenario with the inclusion of u_3, the number of asymptomatic infectious humans in Figure 2(a) decreases more rapidly with control, especially when compared with Figure 1(a), than the case without control. Moreover, the numbers of symptomatic humans, susceptible and infectious mosquitoes decline in the presence of control when compared with the case without control.

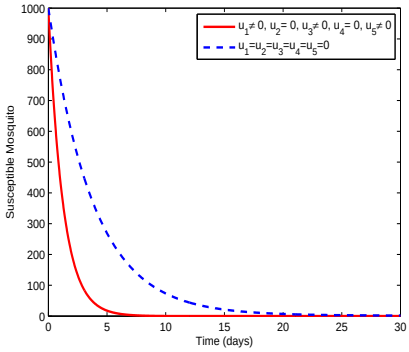

Fig. 2(c): Simulations of model (6) showing effects of controls u_1, u_3 and u_5 on susceptible mosquito population.

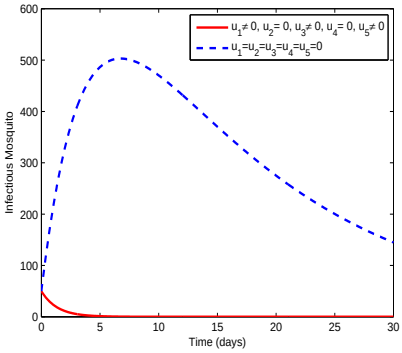

Fig. 2(d): Simulations of model (6) showing effects of controls u_1, u_3 and u_5 on infectious mosquito population.

Scenario 3: Prevention (u_1), routine check (u_3), treatment (u_4) and mosquito-reduction measure (u_5)

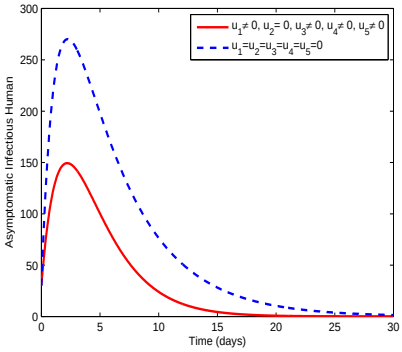

Fig. 3(a): Simulations of model (6) showing effects of controls u_1, u_3, u_4 and u_5 on asymptomatic human population.

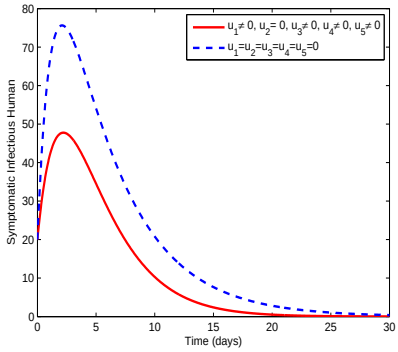

Fig. 3(b): Simulations of model (6) showing effects of controls u_1, u_3, u_4 and u_5 on symptomatic human population.

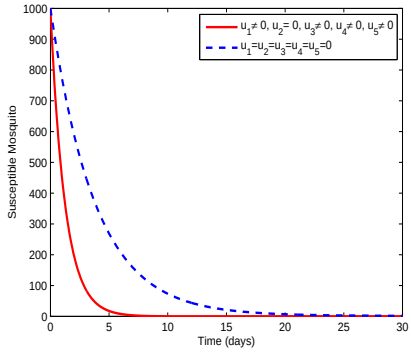

Fig. 3(c): Simulations of model (6) showing effects of controls u_1, u_3, u_4 and u_5 on susceptible mosquito population.

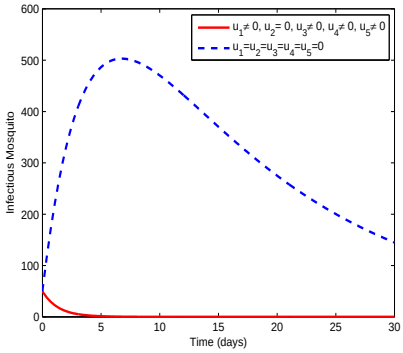

Fig. 3(d): Simulations of model (6) showing effects of control u_1, u_3, u_4 and u_5 on infectious mosquito population.

Due to the inclusion of control u_4 in scenario 3, the number of symptomatic infectious humans in Figure 3(b) diminish more rapidly with control, especially when compared with Figure 2(b), than the case without control. Whereas, the effects observed on asymptomatic humans, susceptible and infectious mosquitoes in Figures 3 are the same when compared with the scenario in Figures 2 accordingly.

Scenario 4: Prevention (u_2) and mosquito reduction measure (u_5)

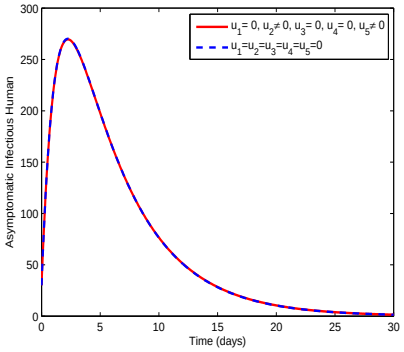

Fig. 4(a): Simulations of model (6) showing effects of controls u_2 and u_5 on asymptomatic human population.

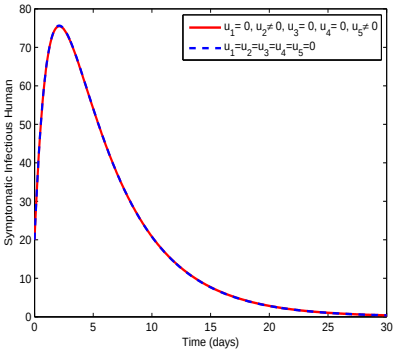

Fig. 4(b): Simulations of model (6) showing effects of controls u_2 and u_5 on symptomatic human population.

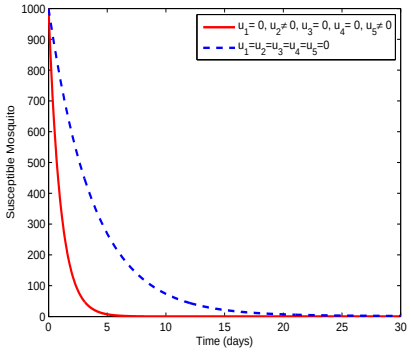

Fig. 4(c): Simulations of model (6) showing effects of controls u_2 and u_5 on susceptible mosquito population.

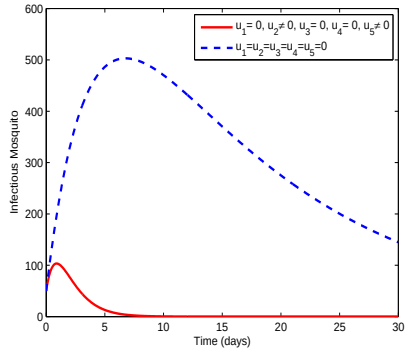

Fig. 4(d): Simulations of model (6) showing effects of controls u_2 and u_5 on infectious mosquito population.

There is no significant difference in the numbers of asymptomatic and symptomatic humans between the case with control and the case without control in Figures 4(a) and 4(b). This shows that the use of control u_2 is less important in curtailing ZIKV when compared with the use of control u_1 as shown in Figures 1.

Scenario 5: Prevention (u_2), routine check (u_3), treatment (u_4) and mosquito-reduction measure (u_5)

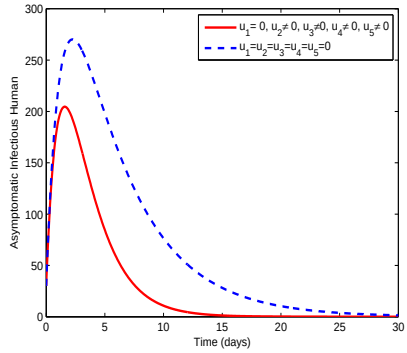

Fig. 5(a): Simulations of model (6) showing effects of controls u_2, u_3, u_4 and u_5 on asymptomatic human population.

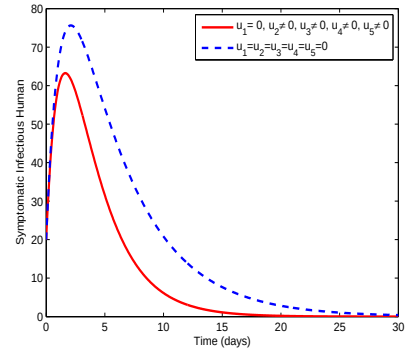

Fig. 5(b): Simulations of model (6) showing effects of controls u_2, u_3, u_4 and u_5 on symptomatic human population.

With the optimal control strategies used in Figures 5(a)–5(d), the reduction in the numbers of asymptomatic and symptomatic infectious individuals as well as the reduction in the number of infectious mosquito are less when compared with the optimal control strategies in Figure 3. This again confirms that combination of intervention strategies without the use of control u_1 may not yield the desired results in curtailing ZIKV spread.

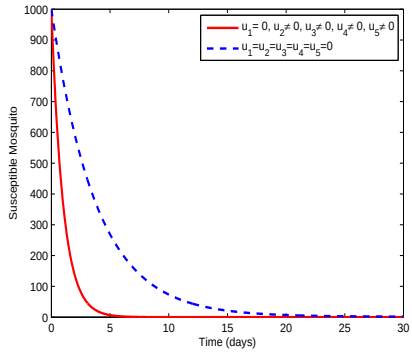

Fig. 5(c): Simulations of model (6) showing effects of controls u_2, u_3, u_4 and u_5 on susceptible mosquito population.

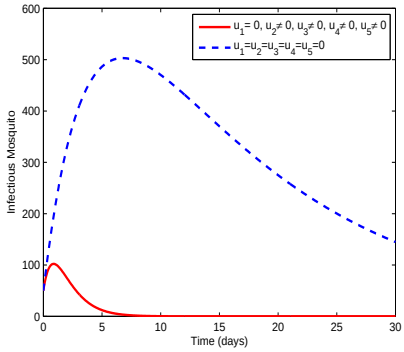

Fig. 5(d): Simulations of model (6) showing effects of controls u_2, u_3, u_4 and u_5 on infectious mosquito population.

Scenario 6: Prevention (u_1 and u_2), routine check (u_3), treatment (u_4) and mosquito reduction measure (u_5)

The effects of using all the optimal control strategies in optimizing the objective functional (7) are illustrated in Figures 6(a)–6(d). It is shown that the sizes of both asymptomatic and symptomatic infectious humans reduce when control is applied. Figure 6(c) shows that the total size of the mosquito population, N_V, decreases more rapidly with control than the case without control. Control profiles in Figure 6(d) reveal that the optimal control u_1 is at the upper bound for about 12 days before dropping to the lower bound while the control u_2 is initially at the lower bound till around 9 days plus few hours before it is sustained at the upper bound until 28th day and then

drops back to the lower bound. Further, the control u_3 is at the upper bound for t = 19 days before dropping to the lower bound. The control u_4 is at the upper bound till about 25 days before reducing to the lower bound. The last control u_5 is at the upper bound for 10 days before it is sustained around 0.6 and gradually reduces to the lower bound at final time.

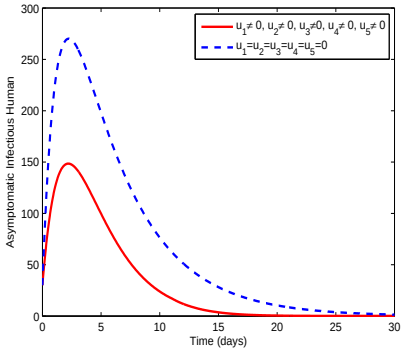

Fig. 6(a): Simulations showing the combined effects of all controls on asymptomatic human population.

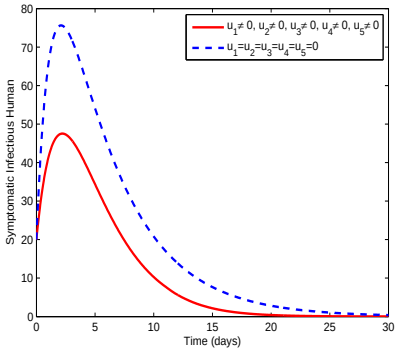

Fig. 6(b): Simulations showing the combined effects of all controls on symptomatic human population.

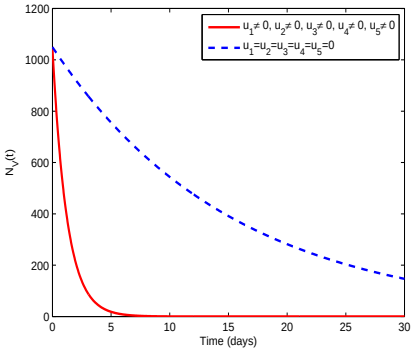

Fig. 6(c): Simulations showing the combined effects of all controls on the total mosquito population size.

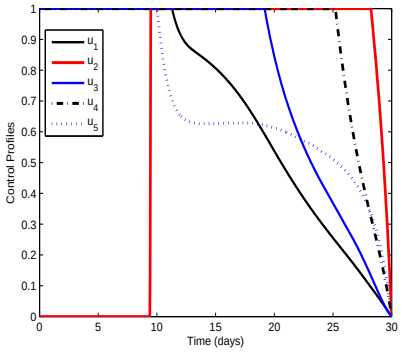

Fig. 6(d): Simulations of model (6) showing the profile of all the control functions.

Figures 7(a)–7(c) illustrate the influence of the saturation constants α_1, α_2 and α_3 of the nonlinear incidence rates on the behaviour of asymptomatic and symptomatic humans as well as the infectious mosquito population. It is shown that as the saturation constants increase, the sizes of the infectious humans and mosquito populations decrease accordingly.

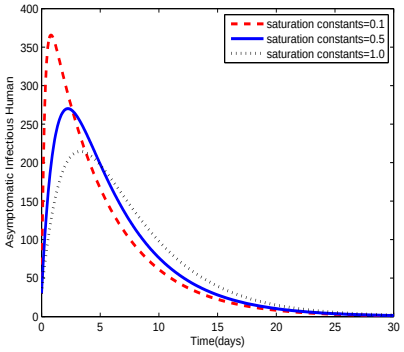

Fig. 7(a): Simulations of system (1) showing the effect of the saturation constants on the behaviour of asymptomatic human population.

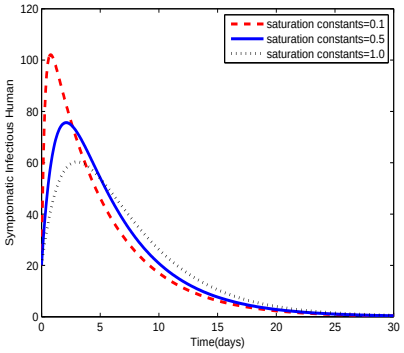

Fig. 7(b): Simulations of system (1) showing the effect of the saturation constants on the behaviour of symptomatic human population.

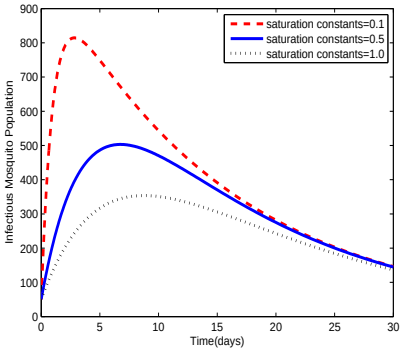

Fig. 7(c): Simulations of system (1) showing the effect of the saturation constants on the behaviour of infectious mosquito population.

Further investigation to justify the need for preventive and control measures is carried out in Figures 8(a)–8(d). It is observed in Figure 8(a) that the magnitude of the symptomatic infectious human population increases as the value of β_3 increases from $\beta_3 = 0.02(\mathscr{R}_0 = 0.8897)$ via $\beta_3 = 0.15(\mathscr{R}_0 = 2.0337)$ to $\beta_3 = 0.25(\mathscr{R}_0 = 2.6128)$.

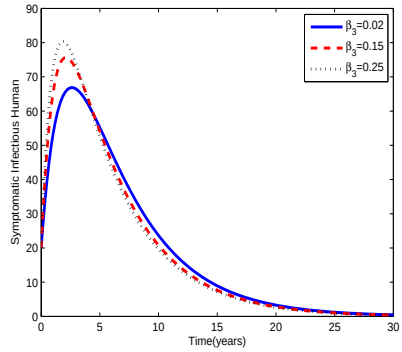

Fig. 8(a): Simulations investigating the influence of β_3 on the magnitude of the symptomatic infectious human population.

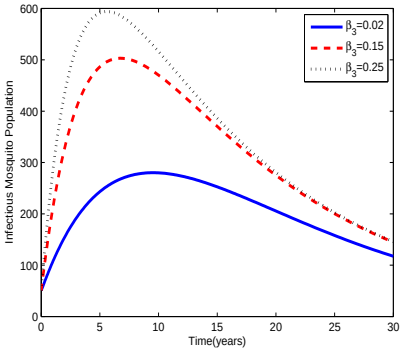

Fig. 8(b): Simulations investigating the influence of β_3 on the magnitude of the infectious mosquito population.

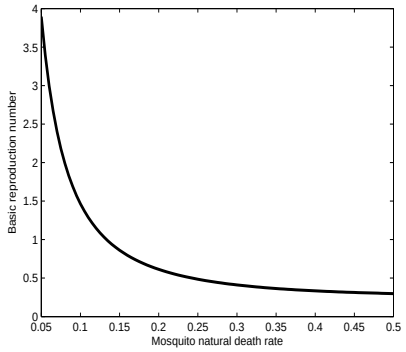

Fig. 8(c): Simulations investigating the influence of μ_V on the basic reproduction number of the ZIKV model

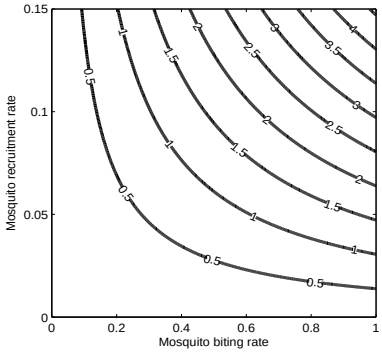

Fig. 8(d): 2D-contour plot of $\mathscr{R}_0$ as a function of Λ_V and b.

Similar feature is observed in Figure 8(b) on the magnitude of the infectious mosquito population. In addition, Figure 8(c) shows that $\frac{\partial \mathscr{R}_0}{\partial \mu_V} < 0$, which implies that intensifying mosquito-reduction effort will go a long way in decreasing the ZIKV spread in the population. In another perspective, a 2D-contour plot of the basic reproduction number $\mathscr{R}_0$ as a function of mosquito recruitment rate Λ_V and mosquito biting rate b is shown in Figure 8(d). It can be seen that effort that forbids the presence of mosquito in the population has the capacity to bring the value of $\mathscr{R}_0$ below unity leading to the reduction of ZIKV burden in the population. However, any attitude that encourages proliferation of mosquito population would bring the basic reproduction number to a value greater than unity as depicted in Figure 8(d).

6 Conclusion

A mathematical model, representing the ZIKV transmission dynamics with nonlinear forces of infection induced by infected mosquito, asymptomatic and symptomatic infectious humans, has been developed and analyzed. With sensitivity analysis carried out on the model parameters, five intervention strategies signaling a possible reduction of the basic reproduction number of the disease are identified. Conditions for the optimal control of these strategies, which include

human-mosquito contact prevention, human-human sexual contact protection, routine check for asymptomatic individuals, treatment and mosquito reduction strategies, are derived and analyzed based on the use of optimal control theory. Therefore, the results of the analysis aided by simulations show that the combination of the five intervention strategies could help in preventing and controlling the transmission of the Zika virus disease. Moreover, the sizes of the infected mosquito, asymptomatic and symptomatic infectious humans in the population could be inhibited by increase in the saturation constants of the nonlinear forces of infection.

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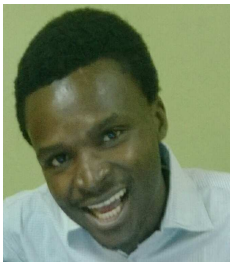
References

[1] F. B. Agusto, S. Bewick, W. F. Fagan, Mathematical Model of Zika Virus with Vertical Transmission, Infectious Disease Modelling, Vol. 2, No. 2, pp. 244–267 (2017).
[2] E. A. Bakare and C. R. Nwozo, Effect of Control Strategies on the Optimal Control Analysis of a Human-Mosquito Model for Malaria Under the Influence of Infective Immigrants, International Journal of Mathematics and Computation, Vol. 26, No. 1, pp. 51–73 (2015).
[3] E. Bonyah and K. O. Okosun, Mathematical Modeling of Zika Virus, Asian Pacific Journal of Tropical Disease, Vol. 6, No. 9, pp. 673–679 (2016).
[4] K. Blayneh, Y. Cao and H. Kwon, Optimal Control of Vector-Borne Diseases: Treatment and Prevention, Discrete and Continuous Dynamical Sys. Series B, Vol. 11, No. 3, pp. 587–611 (2009).
[5] V. M. Cao-Lormeau, A. Blake, S. Mons, *et al.*, Guillain-Barré Syndrome Outbreak Associated with Zika Virus Infection in French Polynesia: A Case-Control Study, Lancet, Vol. 387, No. 10027, pp. 1531–1539 (2016).
[6] V. Capasso and G. Serio, A Generalization of the Kermack-McKendrick Deterministic Epidemic Model, Mathematical Biosciences, Vol. 42, pp. 43–61 (1978).
[7] S. Cauchemez, M. Besnard, P. Bompard, *et al.*, Association Between Zika Virus and Microcephaly in French Polynesia, 2013-15: A Retrospective Study, Lancet, Vol. 387, pp. 2125–2132 (2016).
[8] C. Ding, N. Tao and Y. Zhu, A Mathematical Model of Zika Virus and its Optimal Control, Proceedings of the 35th Chinese Control Conference, pp. 2642–2645 (2016).
[9] M. R. Duffy, T. H. Chen, W. T. Hancock, *et al.*, Zika Virus Outbreak on Yap Island, Federated States of Micronesia, New England Journal of Medicine, Vol. 360, No. 24, pp. 2536–2543 (2009).
[10] W. H. Fleming and R. W. Richel, Derteministic and Stochastic Optimal Control. Springer: New York, 1975.
[11] D. Gao, Y. Lou, D. He, *et al.*, Prevention and Control of Zika as a Mosquito-Borne and Sexually Transmitted Disease: A Mathematical Modeling Analysis, Scientific Reports, Vol. 6, No. 28070, (2016).
[12] H. W. Hethcote, The Mathematics of Infectious Diseases, SIAM Review Vol. 42, No. 4, pp. 599–653 (2000).
[13] R. Isea and K. E. Lonngren, A Preliminary Mathematical Model for the Dynamic Transmission of Dengue, Chikungunya and Zika, American Journal of Modern Physics and Application, Vol. 3, No. 2, pp. 11–15 (2016).
[14] M. Khalid and F. S. Khan, Stability Analysis of Deterministic Mathematical Model for Zika Virus, British Journal of Mathematics & Computer Science, Vol. 19, No. 4, pp. 1–10 (2016).
[15] A. J. Kucharski, S. Funk, R. M. Eggo, *et al.*, Transmission Dynamics of Zika Virus in Island Populations: A Modelling Analysis of the 2013-14 French Polynesia Outbreak, PLOS Neglected Tropical Diseases, Vol. 10, No. 5, (2016).
[16] J. Lamwong and P. Pongsumpun, The Zika Virus Transmission Model, International Journal of Bioscience, Biochemistry and Bioinformatics, Vol. 7, No. 2, pp. 66–73 (2017).
[17] S. Lenhart and J. T. Workman, Optimal Control Applied to Biological Models. Chapman & Hall: London, 2007.
[18] S. M. Moghadas and A. B. Gumel, Global Stability of a Two-Stage Epidemic Model with Generalized Non-linear Incidence, Mathematics and Computers in Simulation, Vol. 60, pp. 107–118 (2002).
[19] A. A. Momoh and A. Fügenschuh, Optimal Control of Intervention Strategies and Cost Effectiveness Analysis for a Zika Virus Model, Operations Research for Health Care, Vol. 18, pp. 99–111 (2018).
[20] S. C. Mpeshe, N. Nyerere and S. Sanga, Modeling Approach to Investigate the Dynamics of Zika Virus Fever: A Neglected Disease in Africa, Int. J. Adv. Appl. Math. and Mech., Vol. 4, No. 3, pp. 14–21 (2017).
[21] G. G. Mwanga, H. Haario and V. Capasso, Optimal Control Problems of Epidemic Systems with Parameter Uncertainties: Application to a Malaria Two-Age-Classes Transmission Model with Asymptomatic Carriers, Mathematical Biosciences Vol. 261, pp. 1–12 (2015).
[22] E. Oehler, L. Watrin, P. Larre, *et al.*, Zika Virus Infection Complicated by Guillain-Barré Syndrome–Case Report, French Polynesia, December 2013, Euro Surveillance, Vol. 19, No. 9 (2014).
[23] D. Okuonghae and V. U. Aihie, Optimal Control Measures for Tuberculosis Mathematical Models Including Immigration and Isolation of Infective, Journal of Biological Systems, Vol. 18, No. 1, pp. 17–54 (2010).
[24] S. Olaniyi and O. S. Obabiyi, Mathematical Model for Malaria Transmission Dynamics in Human and Mosquito Populations with Nonlinear Forces of Infection, Int. J. Pure Appl. Math., Vol. 88, No. 1, pp. 125–156 (2013).
[25] S. Olaniyi and O. S. Obabiyi, Qualitative Analysis of Malaria Dynamics with Nonlinear Incidence Function, Applied Mathematical Sciences, Vol. 8, No. 78, pp. 3889–3904 (2014).
[26] T. O. Oluyo and M. O. Adeyemi, Mathematical Analysis of Zika Epidemic model, IOSR Journal of Mathematics, Vol. 12, No. 6, pp. 21–33 (2016).

[27] M. Ozair, A. A. Lashari, I. H. Jung and K. O. Okosun, Stability Analysis and Optimal Control of a Vector-Borne Disease with Nonlinear Incidence, Discrete Dynamics in Nature and Society, Article ID 595487, pp. 1–21 (2012).
[28] L. S. Pontryagin, V. G. Boltyanskii, R. V. Gamkrelidze, E. F. Mishchenko, The Mathematical Theory of Optimal Processes. Wiley: New York, 1962.
[29] T. A. Perkins, A. S. Siraj, C. W. Ruktanonchai, M. U. G. Kraemer and A. J. Tatem, Model-Based Projections of Zika Virus Infections in Childbearing Women in the Americas, Nature Microbiology, Vol. 1, No. 16126, pp. 1–8 (2016).
[30] P. Padmanabhan, P. Seshaiyer and C. Castillo-Chavez, Mathematical Modeling, Analysis and Simulation of the Spread of Zika with Influence of Sexual Transmission and Preventive Measeures, Letters in Biomathematics, Vol. 4, No. 1, pp. 148–166 (2017).
[31] G. P. Sahu and J. Dhar, Analysis of an SVEIS Epidemic Model with Partial Temporary Immunity and Saturation Incidence Rate, Applied Mathematical Modelling, Vol. 36, pp. 908–923 (2012).
[32] N. H. Shah, Z. A. Patel and B. M. Yeolekar, Preventions and Controls on Congenital Transmissions of Zika: Mathematical Analysis, Applied Mathematics, Vol. 8, pp. 500–519 (2017).
[33] P. van den Driessche and J. Watmough, Reproduction Numbers and sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission, Mathematical Biosciences, Vol. 180, pp. 29–48 (2002).
[34] WHO - World Health Organization, WHO's response to Zika virus and its associated complications, WHO Press: Geneva, 2016.
[35] WHO - World Health Organization, Zika Virus Fact Sheet, Online, Retrieved in May 2017.
[36] Y. Yang, S. Tang, X. Ren, H. Zhao and C. Guo, Global Stability and Optimal Control for a Tuberculosis Model with Vaccination and Treatment, Discrete and Continuous Dynamical Systems Series, Vol. 21, No. 3, pp. 1009–1022 (2016).



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