Journal of Applied Analysis and Computation
Volume 17, Number 1, February 2027, 349–373
Website:http://www.jaac-online.com
DOI:10.11948/20260018

GLOBAL DYNAMICS OF A TIME-DELAYED ZIKA VIRUS MODEL WITH INCUBATION PERIOD

Yuxi Li[1] and Fuxiang Li[1,†]

Abstract Zika virus (ZIKV), primarily transmitted by *Aedes* mosquitoes, can cause severe neurological complications and has triggered multiple outbreaks worldwide in recent years. In this paper, we develop a time-delayed transmission model incorporating the human incubation period and an exposed host compartment. The basic reproduction number R_0 is derived, and the local asymptotic stability of both the disease-free and endemic equilibria is established. By constructing suitable Lyapunov functionals, the global asymptotic stability of both equilibria is further demonstrated. Numerical simulations further illustrate the theoretical results, showing that the disease dies out when $R_0 < 1$ and persists when $R_0 > 1$. Furthermore, an optimal control framework involving five intervention measures is formulated and analyzed. Numerical results show that the proposed control strategies effectively reduce disease transmission, with vaccination and treatment exhibiting the greatest impact among the considered interventions. In addition, sensitivity analysis reveals the relative influence of model parameters on R_0. Overall, this model enhances our understanding of ZIKV transmission dynamics and provides theoretical support for the design of effective control strategies.

Keywords Zika virus, time delay, global stability, Lyapunov functional, optimal control.

MSC(2010) 34K20, 37N25, 92D30.

1. Introduction

Zika virus (ZIKV), a mosquito-borne flavivirus, has emerged as a significant global health threat since its discovery in Uganda's Zika Forest in 1947. Transmission primarily occurs through the bite of infected *Aedes aegypti* and *Aedes albopictus* mosquitoes, which are also vectors for dengue and chikungunya viruses [4]. Since it was first reported, only a small number of cases were documented in Africa, until a large outbreak occurred on Yap Island in Oceania in 2007 [7, 15]. In 2013, French Polynesia experienced the largest recorded outbreak of Zika fever [27]. By 2016, ZIKV had already spread across South America, Asia and Oceania [1, 3]. In recent years, studies have increasingly found that ZIKV can cause more serious clinical sequelae, including severe neurological complications such as meningitis, meningoencephalitis, and Guillain-Barré syndrome [20]. The absence of licensed vaccines or anfiviral therapies underscores the urgency of effective intervention and control strategies to mitigate future epidemics.

Mathematical modeling has been instrumental in elucidating mosquito-borne disease transmission mechanisms [10, 13, 16, 26, 30, 31]. Zhang and Sandamali [33] proposed a ZIKV transmission model using a Poisson point process for sexual contacts, proved the global stability of the disease-free equilibrium, and demonstrated that the sexual route induces backward and Hopf

[†]The corresponding author.
[1]School of Science, China University of Geosciences (Beijing), 100083 Beijing, China
Email: hlgpn51567@gmail.com(Y. Li), fuxiang_li@cugb.edu.cn(F. Li)

bifurcations. Wang et al. [29] formulated a ZIKV model to study the effects of mosquito-to-human and sexual transmission on disease dynamics and solved the optimal control problem of the Zika model. Recently, Li and Zhao [17] built a reaction–diffusion model of ZIKV with seasonality and spatial heterogeneity, and obtained the global dynamics of the model. For more information on ZIKV transmission, we refer readers to [11, 21, 28] and the references therein.

A crucial aspect of accurately modeling the spread of ZIKV is accounting for the host incubation period—the time delay between exposure to the virus and their progression to the infectious stage [19]. Clinical studies indicate that the human incubation period of ZIKV typically ranges from 3 to 14 days following the bite of an infected mosquito [22]. Some frameworks adopt an SIR structure for the human population coupled with an SI structure for the mosquito population. Denu and Son [2] developed a five-compartment ZIKV transmission model and demonstrated optimal strategies, including human-vector contact prevention and human treatment. Motivated by the work in [2], our model incorporates a time-delay for the host incubation period and an exposed host compartment, providing a more realistic description of disease progression and transmission dynamics.

The remainder of this paper is organized as follows. In Section 2, we establish a six-dimensional dynamical system with time delay caused by the incubation period. In Section 3, we derive the basic reproduction number and the disease-free equilibrium, and prove the existence of the endemic equilibrium. In Section 4, we prove the local asymptotic stability of both the disease-free equilibrium and endemic equilibrium, and further establish their global asymptotic stability by constructing suitable Lyapunov functionals. In Section 5, we introduce five control functions representing different intervention measures, formulate the optimal control problem, and derive the corresponding optimality conditions. In Section 6, we perform numerical simulations to evaluate the effectiveness of the control strategies and perform a sensitivity analysis of R_0 with respect to the model parameters.

2. The model

We divide the human and mosquito populations into six compartments: susceptible (S_h), exposed (E_h), infectious (I_h), and recovered (R_h) humans, as well as susceptible (S_v) and infectious (I_v) mosquitoes. The total human population is denoted by $H(t)$ at time t, that is, $H(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$. Let δ_1 denote the human recruitment rate. Susceptible humans acquire ZIKV infection through bites from infected mosquitoes, with the corresponding incidence given by $\frac{abS_hI_v}{H}$, or through sexual transmission, with incidence $\frac{\beta S_hI_h}{H}$. The human incubation period is assumed to be τ. The proportion of newly infected humans entering the exposed compartment is denoted by θ, $0 < \theta < 1$. All human compartments are subject to a natural death rate η, and infected humans recover at rate γ. In this study, *Aedes aegypti* is considered as the vector species. Let δ_2 denote the mosquito recruitment rate. Susceptible mosquitoes become infected by humans infected with ZIKV, with the incidence term given by $\frac{acpI_hS_v}{H}$. Mosquitoes die naturally at a rate ν. The transmission dynamics of ZIKV are governed by the following six-dimensional nonlinear delay differential equations:

$$\frac{\mathrm{d}S_h(t)}{\mathrm{d}t} = \delta_1 - \left(\frac{abS_h(t)I_v(t)}{H(t)} + \frac{\beta S_h(t)I_h(t)}{H(t)}\right) - \eta S_h(t),$$

$$\frac{\mathrm{d}E_h(t)}{\mathrm{d}t} = \theta\frac{abS_h(t)I_v(t) + \beta S_h(t)I_h(t)}{H(t)} - \theta e^{-\eta\tau}\frac{abS_h(t-\tau)I_v(t-\tau) + \beta S_h(t-\tau)I_h(t-\tau)}{H(t-\tau)}$$

$$
\begin{aligned}
&\quad - \eta E_h(t),\\
\frac{\mathrm{d}I_h(t)}{\mathrm{d}t} &= \theta e^{-\eta\tau}\frac{abS_h(t-\tau)I_v(t-\tau)+\beta S_h(t-\tau)I_h(t-\tau)}{H(t-\tau)} - (\eta+\gamma)I_h(t),\\
\frac{\mathrm{d}R_h(t)}{\mathrm{d}t} &= (1-\theta)\frac{abS_h(t)I_v(t)+\beta S_h(t)I_h(t)}{H(t)} + \gamma I_h(t) - \eta R_h(t),\\
\frac{\mathrm{d}S_v(t)}{\mathrm{d}t} &= \delta_2 - \frac{acpI_h(t)S_v(t)}{H(t)} - \nu S_v(t),\\
\frac{\mathrm{d}I_v(t)}{\mathrm{d}t} &= \frac{acpI_h(t)S_v(t)}{H(t)} - \nu I_v(t).
\end{aligned}
\tag{2.1}
$$

Table 1. Parameter descriptions of system (2.1).

Parameter	**Description (unit)**
a	Mosquito biting rate (day^{-1})
b	Probability of transmission from an infectious mosquito to a susceptible human per bite (dimensionless)
c	Probability of transmission from an infectious human to a susceptible mosquito per bite (dimensionless)
β	Human-to-human transmission rate due to sexual transmission (day^{-1})
θ	Proportion of newly infected humans entering the exposed class (dimensionless)
η	Human natural death rate (day^{-1})
γ	Human recovery rate (day^{-1})
p	Relative human-to-mosquito transmission probability of an infected human to a susceptible mosquito per bite (dimensionless)
ν	Mosquito mortality rate (day^{-1})
δ_1	Human recruitment rate (individuals/day)
δ_2	Mosquito recruitment rate (mosquitoes/day)
τ	Human incubation delay (day)

Table 1 illustrates the parameters of system (2.1) and presents their definitions. All parameters in system (2.1) are positive, with $\tau \geq 0$. To ensure the continuity of solutions of system (2.1), we impose the following compatibility condition:

$$E_h(0) = \int_{-\tau}^{0} \theta\frac{abS_h(s)I_v(s)+\beta S_h(s)I_h(s)}{H(s)}e^{\eta s}ds.$$

By the uniqueness of solutions, $E_h(t)$ can be expressed as follows:

$$E_h(t) = \int_{t-\tau}^{t} \theta\frac{abS_h(s)I_v(s)+\beta S_h(s)I_h(s)}{H(s)}e^{-\eta(t-s)}ds. \tag{2.2}$$

The phase space of system (2.1) is

$$C_+ = \{\phi \in C = C([-\tau,0],\mathbb{R}^6_+) : \phi_1(\Theta)+\phi_2(\Theta)+\phi_3(\Theta)+\phi_4(\Theta) > 0, \forall \Theta \in [-\tau,0],$$

$$\phi_2(0) = \int_{-\tau}^{0} \theta \frac{ab\phi_1(s)\phi_6(s) + \beta\phi_1(s)\phi_3(s)}{\phi_1(s) + \phi_2(s) + \phi_3(s) + \phi_4(s)} e^{\eta s} ds\},$$

where C denotes the Banach space of continuous functions from $[-\tau, 0]$ to $\mathbb{R}^6_+$, equipped with the supremum norm.

3. Basic results

Lemma 3.1. *The solution $y(t) = (S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), I_v(t))^T$ of system (2.1) with the initial condition $\phi \in C_+$ is existent, unique, non-negative, and ultimately bounded on $[0, +\infty)$.*

Proof. For any $\phi \in C_+$, we define

$$f(\phi) = (f_1(\phi), f_2(\phi), f_3(\phi), f_4(\phi), f_5(\phi), f_6(\phi))^T$$
$$= \begin{pmatrix} \delta_1 - \left(\dfrac{ab\phi_1(0)\phi_6(0)}{H(0)} + \dfrac{\beta\phi_1(0)\phi_3(0)}{H(0)} \right) - \eta\phi_1(0) \\ \theta \dfrac{ab\phi_1(0)\phi_6(0) + \beta\phi_1(0)\phi_3(0)}{H(0)} - \theta e^{-\eta\tau} \dfrac{ab\phi_1(-\tau)\phi_6(-\tau) + \beta\phi_1(-\tau)\phi_3(-\tau)}{H(-\tau)} - \eta\phi_2(0) \\ \theta e^{-\eta\tau} \dfrac{ab\phi_1(-\tau)\phi_6(-\tau) + \beta\phi_1(-\tau)\phi_3(-\tau)}{H(-\tau)} - (\eta + \gamma)\phi_3(0) \\ (1-\theta) \dfrac{ab\phi_1(0)\phi_6(0) + \beta\phi_1(0)\phi_3(0)}{H(0)} + \gamma\phi_3(0) - \eta\phi_4(0) \\ \delta_2 - \dfrac{acp\phi_3(0)\phi_5(0)}{H(0)} - \nu\phi_5(0) \\ \dfrac{acp\phi_3(0)\phi_5(0)}{H(0)} - \nu\phi_6(0) \end{pmatrix}.$$

Note that $f(\phi)$ is continuous and Lipschitz in ϕ on any compact subset of C_+. By the existence and uniqueness theorem for solutions of delay differential equations (see, e.g., [12, 14]), the solution $(S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), I_v(t))^T$ of system (2.1), corresponding to any initial function $\phi \in C_+$, is uniquely defined on its maximal interval $[0, T_\phi)$ of existence.

Furthermore, from equation (2.2), we also observe that there exists $t_0 \geq 0$ such that $E_h(t) \geq 0$, $\forall t \in [0, t_0]$, whenever $S_h(t) \geq 0$, $I_h(t) \geq 0$, $I_v(t) \geq 0$ and $t \in [0, t_0] \subseteq [0, T_\phi)$. If $\phi_i(0) = 0$ for some $i \in \{1, 3, 4, 5, 6\}$, then $f_i(\phi) \geq 0$. By [25, Theorem 5.2.1] and its proof, it immediately follows that $S_h(t)$, $I_h(t)$, $R_h(t)$, $S_v(t)$, and $I_v(t)$ remains nonnegative for all $t \in [0, T_\phi)$. Together with the integral form (2.2), it yields $E_h(t) \geq 0$, $\forall t \in [0, T_\phi)$. Thus the solution $y(t) = (S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), I_v(t))^T$ of system (2.1) with $\phi \in C_+$ is non-negative for all $t \in [0, T_\phi)$.

By summing the equations for human compartments of system (2.1), we obtain

$$\begin{aligned} \frac{\mathrm{d}H(t)}{\mathrm{d}t} =& \frac{\mathrm{d}(S_h(t) + E_h(t) + I_h(t) + R_h(t))}{\mathrm{d}t} \\ =& \delta_1 - \eta(S_h(t) + E_h(t) + I_h(t) + R_h(t)) \\ =& \delta_1 - \eta H(t). \end{aligned}$$

For the vector compartments, summing their equations gives

$$\frac{\mathrm{d}(S_v(t) + I_v(t))}{\mathrm{d}t} = \delta_2 - \nu(S_v(t) + I_v(t)).$$

It is easily seen that the solution of system (2.1) is bounded for all $t \in [0, T_\phi)$. By [12], it yields $T_\phi = +\infty$. Thus, it has

$$\begin{aligned} &\lim_{t\to+\infty} H(t) = \frac{\delta_1}{\eta} := H^*, \\ &\lim_{t\to+\infty} (S_v(t) + I_v(t)) = \frac{\delta_2}{\nu}. \end{aligned} \tag{3.1}$$

Therefore, it evidently follows that the solution exists uniquely, and remains non-negative and bounded for any initial condition $\phi \in C_+$ on $[0, +\infty)$. In fact, it is easy to obtain that C_+ is a positive invariant set for system (2.1). □

Note that the total human population $H(t)$ asymptotically approaches a constant H^*. Therefore, system (2.1) admits the following limiting system:

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} =\delta_1 - \left(\dfrac{abS_h(t)I_v(t)}{H^*} + \dfrac{\beta S_h(t)I_h(t)}{H^*}\right) - \eta S_h(t), \\ \dfrac{\mathrm{d}E_h(t)}{\mathrm{d}t} =\theta\dfrac{abS_h(t)I_v(t) + \beta S_h(t)I_h(t)}{H^*} - \theta e^{-\eta\tau}\dfrac{abS_h(t-\tau)I_v(t-\tau) + \beta S_h(t-\tau)I_h(t-\tau)}{H^*} \\ \qquad\qquad - \eta E_h(t), \\ \dfrac{\mathrm{d}I_h(t)}{\mathrm{d}t} =\theta e^{-\eta\tau}\dfrac{abS_h(t-\tau)I_v(t-\tau) + \beta S_h(t-\tau)I_h(t-\tau)}{H^*} - (\eta+\gamma)I_h(t), \\ \dfrac{\mathrm{d}R_h(t)}{\mathrm{d}t} =(1-\theta)\dfrac{abS_h(t)I_v(t) + \beta S_h(t)I_h(t)}{H^*} + \gamma I_h(t) - \eta R_h(t), \\ \dfrac{\mathrm{d}S_v(t)}{\mathrm{d}t} =\delta_2 - \dfrac{acpI_h(t)S_v(t)}{H^*} - \nu S_v(t), \\ \dfrac{\mathrm{d}I_v(t)}{\mathrm{d}t} =\dfrac{acpI_h(t)S_v(t)}{H^*} - \nu I_v(t). \end{cases} \tag{3.2}$$

In order to more easily establish the global stability of system (2.1), we first study the limiting system (3.2). Since the equations for $S_h(t)$, $I_h(t)$, $S_v(t)$, and $I_v(t)$ are independent of $E_h(t)$ and $R_h(t)$ in system (3.2), we only need to consider the following subsystem:

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} = \delta_1 - \left(\dfrac{abS_h(t)I_v(t)}{H^*} + \dfrac{\beta S_h(t)I_h(t)}{H^*}\right) - \eta S_h(t), \\ \dfrac{\mathrm{d}I_h(t)}{\mathrm{d}t} = \theta e^{-\eta\tau}\dfrac{abS_h(t-\tau)I_v(t-\tau) + \beta S_h(t-\tau)I_h(t-\tau)}{H^*} - (\eta+\gamma)I_h(t), \\ \dfrac{\mathrm{d}S_v(t)}{\mathrm{d}t} = \delta_2 - \dfrac{acpI_h(t)S_v(t)}{H^*} - \nu S_v(t), \\ \dfrac{\mathrm{d}I_v(t)}{\mathrm{d}t} = \dfrac{acpI_h(t)S_v(t)}{H^*} - \nu I_v(t). \end{cases} \tag{3.3}$$

The initial function of system (3.3) is defined by

$$\phi = (\phi_1, \phi_2, \phi_3, \phi_4)^T \in C([-\tau, 0], \mathbb{R}^4_+) := W_+.$$

As a fundamental epidemiological parameter, the basic reproduction number R_0 quantifies the expected number of secondary infections generated by infected individuals and vectors introduced into a fully susceptible population. To determine R_0, we employ the next-generation

matrix method [5, 6] by constructing the new infection matrix F and transition matrix V, and then compute the spectral radius of matrix FV^{-1}. Define

$$F = \begin{pmatrix} \theta e^{-\eta\tau}\beta & \theta e^{-\eta\tau}ab \\ \dfrac{acp\eta\delta_2}{\delta_1\nu} & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \eta+\gamma & 0 \\ 0 & \nu \end{pmatrix}.$$

Then the next-generation matrix is

$$FV^{-1} = \begin{pmatrix} \dfrac{\theta e^{-\eta\tau}\beta}{\eta+\gamma} & \dfrac{\theta e^{-\eta\tau}ab}{\nu} \\ \dfrac{acp\eta\delta_2}{\delta_1\nu(\eta+\gamma)} & 0 \end{pmatrix}.$$

The spectral radius of matrix FV^{-1}, denoted as R_0, is computed as follows:

$$R_0 = \frac{1}{2}\left[\frac{\theta e^{-\eta\tau}\beta}{\eta+\gamma} + \sqrt{\left(\frac{\theta e^{-\eta\tau}\beta}{\eta+\gamma}\right)^2 + \frac{4\theta e^{-\eta\tau}a^2bcp\delta_2\eta}{\delta_1\nu^2(\eta+\gamma)}}\right].$$

The disease-free equilibrium of system (3.3) is given by

$$E_0 = (S_h^0, 0, S_v^0, 0),$$

where $S_h^0 = \frac{\delta_1}{\eta}$, $S_v^0 = \frac{\delta_2}{\nu}$.

Lemma 3.2. *System (3.3) admits a unique endemic equilibrium E^* if and only if $R_0 > 1$.*

Proof. From system (3.3), we can derive the following equations for the equilibrium equations:

$$\begin{cases} \delta_1 - \dfrac{abS_h^*I_v^* + \beta S_h^*I_h^*}{H^*} - \eta S_h^* = 0, \\ \theta e^{-\eta\tau}\dfrac{abS_h^*I_v^* + \beta S_h^*I_h^*}{H^*} - (\eta+\gamma)I_h^* = 0, \\ \delta_2 - \dfrac{acp}{H^*}I_h^*S_v^* - \nu S_v^* = 0, \\ \dfrac{acp}{H^*}I_h^*S_v^* - \nu I_v^* = 0. \end{cases} \tag{3.4}$$

After refining and organizing, we obtain

$$S_v^* = \frac{\delta_2 H^*}{acpI_h^* + \nu H^*}, \quad I_v^* = \frac{acp\delta_2 I_h^*}{\nu(acpI_h^* + \nu H^*)},$$
$$S_h^* = \frac{\delta_1\nu H^*(acpI_h^* + \nu H^*)}{a^2bcp\delta_2 I_h^* + \nu(\beta I_h^* + \eta H^*)(acpI_h^* + \nu H^*)},$$

and

$$a_1 I_h^{*2} + a_2 I_h^* + a_3 = 0, \tag{3.5}$$

where

$$
\begin{aligned}
a_1 &= (\eta+\gamma)\nu\beta acp > 0,\\
a_2 &= -\delta_1\theta e^{-\eta\tau}\nu\beta acp + (\eta+\gamma)(a^2bcp\delta_2 + \nu\eta H^* acp + \nu^2\beta H^*)\\
&= -H^*\eta\theta e^{-\eta\tau}\nu\beta acp + (\eta+\gamma)(a^2bcp\delta_2 + \nu\eta H^* acp + \nu^2\beta H^*)\\
&= -H^*\eta\nu acp(R_0-1)\xi + acp\eta\theta e^{-\eta\tau}\frac{a^2bcp\delta_2}{\nu} + (\eta+\gamma)(a^2bcp\delta_2 + \nu^2\beta H^*),\\
a_3 &= -\nu^2 H^*\delta_1\left(\theta e^{-\eta\tau}(\frac{a^2bcp\delta_2}{\nu^2 H^*} + \beta) - (\eta+\gamma)\right)\\
&= -\nu^2 H^*\delta_1(R_0-1)\xi,\\
\xi &= \left(\frac{1}{2}\sqrt{(\theta e^{-\eta\tau}\beta)^2 + \frac{4\theta e^{-\eta\tau}a^2bcp\delta_2(\eta+\gamma)}{H^*\nu^2}} + (\eta+\gamma) - \frac{1}{2}\theta e^{-\eta\tau}\beta\right) > 0.
\end{aligned}
$$

Clearly, when $R_0 > 1$, we have $a_3 < 0$. Since $a_1 > 0$, it follows from Vieta's formula that the two roots of equation (3.5) have opposite signs. Thus, equation (3.5) has a unique positive root, denoted I_h^*. On the other hand, when $R_0 \le 1$, we have $a_2 > 0$, $a_3 \ge 0$, and hence, equation (3.5) has no positive roots. From the above analysis, it is known that system (3.3) has a unique endemic equilibrium $E^* = (S_h^*, I_h^*, S_v^*, I_v^*)$ if and only if $R_0 > 1$. □

4. Stability analysis of equilibria

4.1. Local stability

Theorem 4.1. *For any $\tau \ge 0$, the disease-free equilibrium E_0 of system (3.3) is locally asymptotically stable when $R_0 < 1$ and is unstable when $R_0 > 1$.*

Proof. The characteristic matrix associated with the linearized system at the disease-free equilibrium E_0 is given by:

$$
J(E_0) = \begin{pmatrix}
\lambda+\eta & \beta & 0 & ab\\
0 & \lambda+\eta+\gamma-\theta e^{-\eta\tau}\beta e^{-\lambda\tau} & 0 & -\theta e^{-\eta\tau}abe^{-\lambda\tau}\\
0 & \dfrac{acp\delta_2}{H^*\nu} & \lambda+\nu & 0\\
0 & -\dfrac{acp\delta_2}{H^*\nu} & 0 & \lambda+\nu
\end{pmatrix}.
$$

Then the characteristic equation of the linearized system is

$$
G(\lambda) = (\lambda+\eta)(\lambda+\nu)\left((\lambda+\eta+\gamma-\theta e^{-\eta\tau}\beta e^{-\lambda\tau})(\lambda+\nu) - \theta e^{-\eta\tau}e^{-\lambda\tau}\frac{a^2bcp\delta_2}{H^*\nu}\right) = 0. \quad (4.1)
$$

Clearly, equation (4.1) admits two negative real roots, $-\eta$ and $-\nu$. The remaining characteristic roots are determined by the following equation:

$$
(\lambda+\eta+\gamma)(\lambda+\nu) = \theta e^{-\eta\tau}\beta e^{-\lambda\tau}(\lambda+\nu) + \theta e^{-\eta\tau}e^{-\lambda\tau}\frac{a^2bcp\delta_2}{H^*\nu}. \quad (4.2)
$$

Assume, by contradiction, that all roots of equation (4.2) have a nonnegative real part. Rearranging equation (4.2), we obtain

$$\lambda+\eta+\gamma=\theta e^{-\eta\tau}\beta e^{-\lambda\tau}+\theta e^{-\eta\tau}e^{-\lambda\tau}\frac{a^2bcp\delta_2}{H^*\nu(\lambda+\nu)}.$$

On one hand, by the properties of complex modulus, it yields

$$|\lambda+\eta+\gamma|\geq\eta+\gamma.$$

On the other hand, by the triangle inequality and the fact that $\mathrm{Re}(\lambda)\geq 0$, where $\mathrm{Re}(\lambda)$ denotes the real part of λ, we have

$$\begin{aligned}&\left|\theta e^{-\eta\tau}\beta e^{-\lambda\tau}+\theta e^{-\eta\tau}e^{-\lambda\tau}\frac{a^2bcp\delta_2}{H^*\nu(\lambda+\nu)}\right|\\ \leq&\,\theta e^{-\eta\tau}\beta+\theta e^{-\eta\tau}\frac{a^2bcp\delta_2}{H^*\nu^2}\\ =&\,(R_0-1)\xi+\eta+\gamma,\end{aligned}$$

where

$$\xi=\left(\frac{1}{2}\sqrt{(\theta e^{-\eta\tau}\beta)^2+\frac{4\theta e^{-\eta\tau}a^2bcp\delta_2(\eta+\gamma)}{H^*\nu^2}}+(\eta+\gamma)-\frac{1}{2}\theta e^{-\eta\tau}\beta\right)>0.$$

Since $R_0<1$, we obtain

$$(R_0-1)\xi+\eta+\gamma<\eta+\gamma,$$

which contradicts the previous inequality. Thus, all characteristic roots have negative real parts, which means that E_0 is locally asymptotically stable when $R_0<1$.

When $R_0>1$ and $\tau\geq 0$, we can obtain

$$G(0)=\eta\nu^2(1-R_0)\xi<0,\ \lim_{\lambda\to+\infty}G(\lambda)=+\infty.$$

By the continuity of $G(\lambda)$, equation (4.1) has a positive real root. Hence the disease-free equilibrium E_0 is unstable. □

Theorem 4.2. *For any $\tau\geq 0$, the endemic equilibrium E^* of system (3.3) is locally asymptotically stable when $R_0>1$.*

Proof. Let

$$A=\frac{abI_v^*+\beta I_h^*}{H^*},B=\frac{\beta S_h^*}{H^*},C=\theta e^{-\eta\tau},D=\frac{abS_h^*}{H^*},E=\frac{acpS_v^*}{H^*},F=\frac{acpI_h^*}{H^*}.$$

The characteristic matrix of the linearized system at the endemic equilibrium E^* is given by:

$$J(E^*)=\begin{pmatrix}\lambda+\eta+A & B & 0 & D\\ -CAe^{-\lambda\tau} & \lambda+\eta+\gamma-CBe^{-\lambda\tau} & 0 & -CDe^{-\lambda\tau}\\ 0 & E & \lambda+\nu+F & 0\\ 0 & -E & -F & \lambda+\nu\end{pmatrix}.$$

Thus, the characteristic equation of system (3.3) at E^* is given by

$$\begin{aligned}&(\lambda+\nu)\big[(\lambda+\eta+A)(\lambda+\nu+F)(\lambda+\eta+\gamma)-(\lambda+\eta)(\lambda+\nu+F)CBe^{-\lambda\tau}\\&-(\lambda+\eta)CDEe^{-\lambda\tau}\big]=0.\end{aligned}\tag{4.3}$$

The above equation has a negative real root $-\nu$, and the remaining roots satisfy

$$\begin{aligned}&(\lambda+\eta+A)(\lambda+\nu+F)(\lambda+\eta+\gamma)\\=&(\lambda+\eta)\left[(\lambda+\nu+F)\frac{\theta e^{-\eta\tau}\beta S_h^*}{H^*}+\frac{\theta e^{-\eta\tau}a^2bcpS_h^*S_v^*}{H^{*2}}\right]e^{-\lambda\tau}.\end{aligned}$$

That is,

$$\frac{\lambda+\eta+A}{\lambda+\eta}=\left(\frac{\theta e^{-\eta\tau}\beta S_h^*}{(\lambda+\eta+\gamma)H^*}+\frac{\theta e^{-\eta\tau}a^2bcpS_h^*S_v^*}{(\lambda+\nu+F)(\lambda+\eta+\gamma)H^{*2}}\right)e^{-\lambda\tau}.\tag{4.4}$$

Assume, by contradiction, that all roots of equation (4.4) have a nonnegative real part. Taking the modulus of both sides of equation (4.4), we have

$$\left|\frac{\lambda+\eta+A}{\lambda+\eta}\right|>1.$$

On the other hand, substituting the results obtained in equation (3.4) into equation (4.4), we have

$$\begin{aligned}&\left|\left(\frac{\theta e^{-\eta\tau}\beta S_h^*}{(\lambda+\eta+\gamma)H^*}+\frac{\theta e^{-\eta\tau}a^2bcpS_h^*S_v^*}{(\lambda+\nu+F)(\lambda+\eta+\gamma)H^{*2}}\right)e^{-\lambda\tau}\right|\\\le&\frac{\theta e^{-\eta\tau}\beta S_h^*}{(\eta+\gamma)H^*}+\frac{\theta e^{-\eta\tau}a^2bcpS_h^*S_v^*}{\nu(\eta+\gamma)H^{*2}}\\=&\frac{\beta\nu(acpI_h^*+\nu H^*)}{a^2bcp\delta_2+\beta\nu(acpI_h^*+\nu H^*)}+\frac{a^2bcp\delta_2}{a^2bcp\delta_2+\beta\nu(acpI_h^*+\nu H^*)}\\=&1.\end{aligned}$$

Clearly, this is a contradiction. Thus, all characteristic roots of equation (4.3) have negative real parts, which implies that the endemic equilibrium E^* is locally asymptotically stable when $R_0>1$. □

4.2. Global stability

Next we investigate the global asymptotic stability of E_0 and E^* in terms of R_0 by formulating suitable Lyapunov functionals. Adopting an argument similar to that in Lemma 3.1, it follows that, for any initial condition $\phi\in W_+$, system (3.3) admits a unique non-negative and ultimately bounded solution on $[0,+\infty)$. Set $F(u)=u-1-\ln u$, $u\in(0,+\infty)$. Then $F(u)\ge 0$ for all $u\in(0,+\infty)$ and $\min_{0<u<+\infty}F(u)=F(1)=0$. The following theorem characterizes the global stability of the disease-free equilibrium E_0 of system (3.3).

Theorem 4.3. *For any $\tau\ge 0$, the disease-free equilibrium E_0 of system (3.3) is globally attractive when $R_0\le 1$ and globally asymptotically stable when $R_0<1$ in $\mathcal{L}_1=\{\phi\in W_+:\phi_1(0)>0,\phi_3(0)>0\}$.*

Proof. For any $\phi \in \mathcal{L}_1$, let $z(t) = (S_h(t), I_h(t), S_v(t), I_v(t))^T$ be the solution of system (3.3). In fact, we can show that $S_h(t) > 0$ and $S_v(t) > 0$ for all $t \geq 0$, and hence, $\mathcal{L}_1$ is a positive invariant set for system (3.3). We define the following Lyapunov functional V_1 on $\mathcal{L}_1$:

$$V_1(\phi) = \tilde{V}_1(\phi(0)) + \frac{\theta e^{-\eta\tau} ab}{H^*} \int_{-\tau}^{0} \phi_1(s)\phi_4(s)ds + \frac{\theta e^{-\eta\tau}\beta}{H^*} \int_{-\tau}^{0} \phi_1(s)\phi_2(s)ds,$$

where

$$\tilde{V}_1(\phi(0)) = \theta e^{-\eta\tau} S_h^0 F\left(\frac{\phi_1(0)}{S_h^0}\right) + \phi_2(0) + \frac{\theta e^{-\eta\tau} ab}{\nu} S_v^0 F\left(\frac{\phi_3(0)}{S_v^0}\right) + \frac{\theta e^{-\eta\tau} ab}{\nu}\phi_4(0).$$

It is easy to verify that V_1 is positive definite with respect to E_0. It is also easy to know that V_1 is continuous on $\mathcal{L}_1$ and satisfies condition (ii) of [23, Lemma 3.1] on $\partial\mathcal{L}_1 = \overline{\mathcal{L}_1}\backslash\mathcal{L}_1$. For all $t \geq 0$, calculating the derivation of V_1 along the solution $z(t)$ of system (3.3), it yields that

$$\begin{aligned}
\dot{V}_1(z_t) =& \theta e^{-\eta\tau}\left(1 - \frac{S_h^0}{S_h(t)}\right)\left(\eta S_h^0 - \frac{abS_h(t)I_v(t) + \beta S_h(t)I_h(t)}{H^*} - \eta S_h(t)\right) \\
&+ \frac{\theta e^{-\eta\tau}}{H^*}\left(abS_h(t-\tau)I_v(t-\tau) + \beta S_h(t-\tau)I_h(t-\tau)\right) - (\eta+\gamma)I_h(t) \\
&+ \frac{\theta e^{-\eta\tau} ab}{\nu}\left(1 - \frac{S_v^0}{S_v(t)}\right)\left(\nu S_v^0 - \frac{acpI_h(t)S_v(t)}{H^*} - \nu S_v(t)\right) \\
&+ \frac{\theta e^{-\eta\tau} ab}{\nu}\left(\frac{acpI_h(t)S_v(t)}{H^*} - \nu I_v(t)\right) + \frac{\theta e^{-\eta\tau}}{H^*}\big(abS_h(t)I_v(t) \\
&+ \beta S_h(t)I_h(t) - abS_h(t-\tau)I_h(t-\tau) - \beta S_h(t-\tau)I_v(t-\tau)\big) \\
=& -\theta e^{-\eta\tau}\eta\frac{(S_h(t) - S_h^0)^2}{S_h(t)} - \theta e^{-\eta\tau} ab\frac{(S_v(t) - S_v^0)^2}{S_v(t)} \\
&+ \left(\theta e^{-\eta\tau}(\beta + \frac{a^2bcp\delta_2}{\nu^2 H^*}) - (\eta+\gamma)\right) I_h(t) \\
=& -\theta e^{-\eta\tau}\eta\frac{(S_h(t) - S_h^0)^2}{S_h(t)} - \theta e^{-\eta\tau} ab\frac{(S_v(t) - S_v^0)^2}{S_v(t)} + (R_0 - 1)\Big(\eta + \gamma \\
&- \frac{1}{2}\theta e^{-\eta\tau}\beta + \frac{1}{2}\sqrt{(\theta e^{-\eta\tau}\beta)^2 + \frac{4\theta e^{-\eta\tau} a^2 bcp\delta_2(\eta+\gamma)}{H^*\nu^2}}\Big) I_h(t).
\end{aligned}$$

Hence, if $R_0 \leq 1$, we have $\dot{V}_1 \leq 0$ for all $t \geq 0$. From the stability theorem in [12, 14], E_0 is stable for $\tau \geq 0$ if $R_0 \leq 1$. Note that $\dot{V}_1 = 0$ if and only if $S_h(t) = S_h^0$ and $S_v(t) = S_v^0$. Let M be the largest invariant set in the set $\Omega_1 = \{\phi \in \overline{\mathcal{L}_1} : V_1 < +\infty \text{ and } \dot{V}_1 = 0\}$. Then $M \subseteq \Omega_1 \subseteq \{\phi \in \overline{\mathcal{L}_1} : \phi_1(0) = S_h^0, \phi_3(0) = S_v^0\}$. By the invariance of M and system (3.3), we can obtain $M = \{E_0\}$. It then follows from [23, Lemma 3.1] (LaSalle's invariance principle) that the disease-free equilibrium E_0 is globally attractive when $R_0 \leq 1$. When $R_0 < 1$, the local asymptotic stability of E_0 has been established in Theorem 4.1, and hence, E_0 is globally asymptotically stable when $R_0 < 1$. □

Subsequently, the following theorem establishes the global stability of the endemic equilibrium E^* of system (3.3).

Theorem 4.4. *For any $\tau \geq 0$, the endemic equilibrium E^* of system (3.3) is globally asymptotically stable when $R_0 > 1$ in $\mathcal{L}_2 = \{\phi \in W_+ : \phi_i(0) > 0,\ i = 1, 2, 3, 4\}$.*

Proof. For any $\phi \in \mathcal{L}_2$, let $z(t) = (S_h(t), I_h(t), S_v(t), I_v(t))^T$ be the solution of system (3.3). In fact, we can show that $S_h(t) > 0$, $I_h(t) > 0$, $S_v(t) > 0$ and $I_v(t) > 0$ for all $t \geq 0$, and hence, $\mathcal{L}_2$ is a positive invariant set for system (3.3). Define the following Lyapunov functional V_2 on $\mathcal{L}_2$:

$$V_2(\phi) = \tilde{V}_2(\phi(0)) + \frac{\theta e^{-\eta\tau} ab}{H^*} S_h^* I_v^* \int_{-\tau}^{0} F\left(\frac{\phi_1(s)\phi_4(s)}{S_h^* I_v^*}\right) ds + \frac{\theta e^{-\eta\tau}\beta}{H^*} S_h^* I_h^* \int_{-\tau}^{0} F\left(\frac{\phi_1(s)\phi_2(s)}{S_h^* I_h^*}\right) ds,$$

where

$$\tilde{V}_2(\phi(0)) = \theta e^{-\eta\tau} F\left(\frac{\phi_1(0)}{S_h^*}\right) + F\left(\frac{\phi_2(0)}{I_h^*}\right) + \frac{e^{-\eta\tau} b S_h^* I_v^*}{c p I_h^* S_v^*} F\left(\frac{\phi_3(0)}{S_v^*}\right) + \frac{e^{-\eta\tau} b S_h^* I_v^*}{c p I_h^* S_v^*} F\left(\frac{\phi_4(0)}{I_v^*}\right).$$

Obviously, V_2 is positive definite with respect to E^* and continuous on $\mathcal{L}_2$, and satisfies condition (ii) of [23, Lemma 3.1] on $\partial\mathcal{L}_2 = \overline{\mathcal{L}_2}\backslash\mathcal{L}_2$. For all $t \geq 0$, calculating the derivation of V_2 along the solution $z(t)$ of system (3.3), it follows that

$$\begin{aligned}
\dot{V}_2(z_t) &= \theta e^{-\eta\tau}\left(1 - \frac{S_h^*}{S_h(t)}\right)\dot{S}_h(t) + \left(1 - \frac{I_h^*}{I_h(t)}\right)\dot{I}_h(t) \\
&\quad + \frac{\theta e^{-\eta\tau} b S_h^* I_v^*}{c p I_h^* S_v^*}\left(1 - \frac{S_v^*}{S_v(t)}\right)\dot{S}_v(t) + \frac{\theta e^{-\eta\tau} b S_h^* I_v^*}{c p I_h^* S_v^*}\left(1 - \frac{I_v^*}{I_v(t)}\right)\dot{I}_v(t) \\
&\quad + \frac{\theta e^{-\eta\tau} ab}{H^*} S_h(t) I_v(t) - \frac{\theta e^{-\eta\tau} ab}{H^*} S_h(t-\tau) I_v(t-\tau) + \frac{\theta e^{-\eta\tau}\beta}{H^*} S_h(t) I_h(t) \\
&\quad - \frac{\theta e^{-\eta\tau}\beta}{H^*} S_h(t-\tau) I_h(t-\tau) + \frac{\theta e^{-\eta\tau} ab}{H^*} S_h^* I_v^* \ln\frac{S_h(t-\tau) I_v(t-\tau)}{S_h(t) I_v(t)} \\
&\quad + \frac{\theta e^{-\eta\tau}\beta}{H^*} S_h^* I_h^* \ln\frac{S_h(t-\tau) I_h(t-\tau)}{S_h(t) I_h(t)} \\
&= -\theta e^{-\eta\tau}\eta\frac{(S_h^* - S_h(t))^2}{S_h(t)} - \theta e^{-\eta\tau}\nu\frac{b S_h^* I_v^*}{c p I_h^* S_v^*}\frac{(S_v^* - S_v(t))^2}{S_v(t)} \\
&\quad + \frac{\theta e^{-\eta\tau} ab}{H^*} S_h^* I_v^*\left(4 - \frac{S_h^*}{S_h(t)} - \frac{S_v^*}{S_v(t)} - \frac{I_h(t) S_v(t) I_v^*}{I_h^* S_v^* I_v(t)} - \frac{S_h(t-\tau) I_v(t-\tau) I_h^*}{S_h^* I_v^* I_h(t)}\right. \\
&\quad \left. + \ln\frac{S_h(t-\tau) I_v(t-\tau)}{S_h(t) I_v(t)}\right) + \frac{\theta e^{-\eta\tau}\beta}{H^*} S_h^* I_h^*\left(2 - \frac{S_h^*}{S_h(t)} - \frac{S_h(t-\tau) I_h(t-\tau)}{S_h^* I_h(t)}\right. \\
&\quad \left. + \ln\frac{S_h(t-\tau) I_h(t-\tau)}{S_h(t) I_h(t)}\right).
\end{aligned}$$

Since

$$\begin{aligned}
\ln\frac{S_h(t-\tau) I_v(t-\tau)}{S_h(t) I_v(t)} &= \ln\frac{S_h^*}{S_h(t)} + \ln\frac{S_v^*}{S_v(t)} + \ln\frac{I_h(t) S_v(t) I_v^*}{I_h^* S_v^* I_v(t)} + \ln\frac{S_h(t-\tau) I_v(t-\tau) I_h^*}{S_h^* I_v^* I_h(t)}, \\
\ln\frac{S_h(t-\tau) I_h(t-\tau)}{S_h(t) I_h(t)} &= \ln\frac{S_h^*}{S_h(t)} + \ln\frac{S_h(t-\tau) I_h(t-\tau)}{S_h^* I_h(t)},
\end{aligned}$$

we obtain

$$\begin{aligned}\dot{V}_2 =& -\theta e^{-\eta\tau}\eta\frac{(S_h^*-S_h(t))^2}{S_h(t)}-\theta e^{-\eta\tau}\nu\frac{bS_h^*I_v^*}{cpI_h^*S_v^*}\frac{(S_v^*-S_v(t))^2}{S_v(t)}\\&-\frac{\theta e^{-\eta\tau}ab}{H^*}S_h^*I_v^*\bigg(F(\frac{S_h^*}{S_h(t)})+F(\frac{S_v^*}{S_v(t)})+F(\frac{I_h(t)S_v(t)I_v^*}{I_h^*S_v^*I_v(t)})\\&+F(\frac{S_h(t-\tau)I_v(t-\tau)I_h^*}{S_h^*I_v^*I_h(t)})\bigg)-\frac{\theta e^{-\eta\tau}\beta}{H^*}S_h^*I_h^*\bigg(F(\frac{S_h^*}{S_h(t)})\\&+F(\frac{S_h(t-\tau)I_h(t-\tau)}{S_h^*I_h(t)})\bigg)\\\leq&0.\end{aligned}$$

Thus, E^* is stable for $\tau\geq 0$ by [12, 14]. Note that $\dot{V}_2=0$ if and only if $S_h(t)=S_h^*$, $S_v(t)=S_v^*$, $I_h(t)=I_h^*$, $I_v(t)=I_v^*$. Let M be the largest invariant set in the set $\Omega_2=\{\phi\in\overline{\mathcal{L}_2}:V_2<+\infty$ and $\dot{V}_2=0\}$. Clearly, $M=\{E^*\}$. It then follows from [23, Lemma 3.1] (LaSalle's invariance principle) that the endemic equilibrium E^* is globally attractive. Since E^* is locally asymptotically stable by Theorem 4.2, it follows that E^* is globally asymptotically stable when $R_0>1$. □

Note that system (2.1) has a unique positive equilibrium $\tilde{E}^*=(S_h^*,E_h^*,I_h^*,R_h^*,S_v^*,I_v^*)$ if $R_0>1$, where $E_h^*=\frac{\theta(1-e^{-\eta\tau})}{\eta}\frac{abS_h^*I_v^*+\beta S_h^*I_h^*}{H^*}$ and $R_h^*=\frac{(1-\theta)}{\eta}\frac{abS_h^*I_v^*+\beta S_h^*I_h^*}{H^*}+\frac{\gamma}{\eta}I_h^*$. By the theory of asymptotically semiflows [34, Section 3.2] and the chain transitive sets [34, Theorems 1.2.1 and 1.3.1 and Lemma 1.2.2], we can lift the global stability for system (3.3) to system (2.1) (also see, e.g., [17, Theorem 2.1]).

Theorem 4.5. *For any $\tau\geq 0$, the following statements are true.*

(i) The disease-free equilibrium $(S_h^0,0,0,0,S_v^0,0)$ of system (2.1) is globally attractive when $R_0\leq 1$ and globally asymptotically stable when $R_0<1$ in $L_1=\{\phi\in C_+:\phi_1(0)>0,\phi_5(0)>0\}$.

(ii) The endemic equilibrium $\tilde{E}^*$ of system (2.1) is globally asymptotically stable when $R_0>1$ in $L_2=\{\phi\in C_+:\phi_i(0)>0,\ i=1,2,3,4,5,6\}$.

5. Optimal control strategy

In this section, according to Pontryagin's Minimum Principle [9] for systems with delays, we formulate an optimal control framework that incorporates the following five key intervention strategies:

u_1 denotes personal protection measures that reduce human-vector contact (e.g., bed nets or repellents);

u_2 denotes the effort to detect infected individuals and reduce their transmission potential through isolation (e.g., diagnostic screening);

u_3 denotes vaccination campaigns to reduce the number of susceptible individuals (e.g., targeted immunization programs);

u_4 denotes treatment for infected individuals (e.g., supportive therapy or hospitalization);

u_5 denotes the effort of vector population control (e.g., insecticides, biological agents, or environmental management).

Hence, based on system (2.1), we introduce the following corresponding optimal control system:

$$\begin{cases} \dfrac{\mathrm{d}S_h(t)}{\mathrm{d}t} = \delta_1 - \left(\dfrac{(1-u_1(t))abS_h(t)I_v(t)}{H(t)} + \dfrac{(1-u_2(t))\beta S_h(t)I_h(t)}{H(t)}\right) \\ \qquad\qquad - (\eta + u_3(t))S_h(t), \\ \dfrac{\mathrm{d}E_h(t)}{\mathrm{d}t} = \theta\dfrac{(1-u_1(t))abS_h(t)I_v(t) + (1-u_2(t))\beta S_h(t)I_h(t)}{H(t)} \\ \qquad\qquad - \theta e^{-\eta\tau}\dfrac{(1-u_1(t))abS_h(t-\tau)I_v(t-\tau) + (1-u_2(t))\beta S_h(t-\tau)I_h(t-\tau)}{H(t-\tau)} \\ \qquad\qquad - \eta E_h(t), \\ \dfrac{\mathrm{d}I_h(t)}{\mathrm{d}t} = \theta e^{-\eta\tau}\dfrac{(1-u_1(t))abS_h(t-\tau)I_v(t-\tau) + (1-u_2(t))\beta S_h(t-\tau)I_h(t-\tau)}{H(t-\tau)} \\ \qquad\qquad - (\eta + \gamma + u_4(t))I_h(t), \\ \dfrac{\mathrm{d}R_h(t)}{\mathrm{d}t} = (1-\theta)\dfrac{(1-u_1(t))abS_h(t)I_v(t) + (1-u_2(t))\beta S_h(t)I_h(t)}{H(t)} \\ \qquad\qquad + (\gamma + u_4(t))I_h(t) - \eta R_h(t) + u_3(t)S_h(t), \\ \dfrac{\mathrm{d}S_v(t)}{\mathrm{d}t} = \delta_2 - (1-u_1(t))\dfrac{acpI_h(t)S_v(t)}{H(t)} - (\nu + u_5(t))S_v(t), \\ \dfrac{\mathrm{d}I_v(t)}{\mathrm{d}t} = (1-u_1(t))\dfrac{acpI_h(t)S_v(t)}{H(t)} - (\nu + u_5(t))I_v(t). \end{cases} \tag{5.1}$$

Our purpose is to minimize the objective function, which is given by

$$\begin{aligned} J(u_1,u_2,u_3,u_4,u_5) = \int_0^{t_f} \Big[& k_1S_h(t) + k_2I_h(t) + k_3S_v(t) + k_4I_v(t) \\ & + \frac{1}{2}\left(A_1u_1^2(t) + A_2u_2^2(t) + A_3u_3^2(t) + A_4u_4^2(t) + A_5u_5^2(t)\right)\Big]\mathrm{d}t, \end{aligned}$$

where t_f is the fixed final time and k_i $(i = 1,2,3,4)$ are the weight constants of the susceptible humans, infected humans, susceptible mosquitoes, and infected mosquitoes, respectively. Meanwhile, the constants A_i $(i = 1,2,3,4,5)$ represent the positive weighting factors of $u_i(t)$, respectively. The quadratic cost terms $\frac{A_i}{2}u_i^2(t)$ quantify the implementation costs of the control strategies. Thus, J is formulated to quantify the susceptible and infected populations of humans and mosquitoes during the epidemic, together with the implementation costs of the corresponding control measures. Let the control space U be

$$\begin{aligned} U = \{ & u_i(t) : \ u_i(t) \text{ is Lebesgue measurable}, \quad 0 \le u_i(t) \le u_{i\max} \le 1, \\ & i = 1,2,3,4,5, \quad t \in [0,t_f]\}, \end{aligned}$$

where u_{imax} represents the maximum achievable value of the control $u_i(t)$. Our aim is to find the optimal controls $(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*)$ that satisfy

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

To derive the optimal solution, the corresponding Hamiltonian for the control problem is given by

$$\begin{aligned}K(y,y_\tau,u,\lambda,t)=&\,k_1S_h(t)+k_2I_h(t)+k_3S_v(t)+k_4I_v(t)+\frac{1}{2}\sum_{i=1}^{5}A_iu_i^2(t)\\&+\lambda_1(t)\frac{\mathrm{d}S_h(t)}{\mathrm{d}t}+\lambda_2(t)\frac{\mathrm{d}E_h(t)}{\mathrm{d}t}+\lambda_3(t)\frac{\mathrm{d}I_h(t)}{\mathrm{d}t}+\lambda_4(t)\frac{\mathrm{d}R_h(t)}{\mathrm{d}t}\\&+\lambda_5(t)\frac{\mathrm{d}S_v(t)}{\mathrm{d}t}+\lambda_6(t)\frac{\mathrm{d}I_v(t)}{\mathrm{d}t},\end{aligned}$$

where $\lambda_i(t)$ $(i=1,2,\cdots,6)$ is the adjoint variables, $\lambda(t)=(\lambda_1(t),\lambda_2(t),\lambda_3(t),\lambda_4(t),\lambda_5(t),\lambda_6(t))$, $u(t)=(u_1(t),\ u_2(t),u_3(t),u_4(t),u_5(t))$, $y(t)=(S_h(t),E_h(t),I_h(t),R_h(t),S_v(t),I_v(t))^T$, $y_\tau(t)=(S_{h\tau}(t),E_{h\tau}(t),\ I_{h\tau}(t),R_{h\tau}(t),\ S_{v\tau}(t),I_{v\tau}(t))^T$ with $y_\tau(t)=y(t-\tau)$. By applying Pontryagin's Minimum Principle for systems with delays [9], we obtain the following result.

Theorem 5.1. *Suppose that $(u_1^*(t),u_2^*(t),u_3^*(t),u_4^*(t),u_5^*(t))$ is an optimal control and that $(S_h^*(t),E_h^*(t),I_h^*(t),R_h^*(t),\ S_v^*(t),I_v^*(t))$ is the corresponding state solution of system (5.1). Then there exist adjoint variables $\lambda_i(t)$ $(i=1,2,\cdots,6)$ that satisfy the following equations:*

$$\begin{aligned}\frac{\mathrm{d}\lambda_1(t)}{\mathrm{d}t}=&-k_1+\frac{((1-u_1(t))abI_v^*(t)+(1-u_2(t))\beta I_h^*(t))\,(E_h^*(t)+I_h^*(t)+R_h^*(t))}{H^*(t)^2}\\&\times(\lambda_1(t)-\lambda_2(t)\theta-\lambda_4(t)+\lambda_4(t)\theta)+\lambda_1(t)(\eta+u_3(t))-\lambda_4(t)u_3(t)\\&+\frac{((1-u_1(t+\tau))abI_v^*(t)+(1-u_2(t+\tau))\beta I_h^*(t))\,(E_h^*(t)+I_h^*(t)+R_h^*(t))}{H^*(t)^2}\\&\times\chi_{[0,t_f-\tau]}(\lambda_2(t+\tau)-\lambda_3(t+\tau))\theta e^{-\eta\tau},\\\frac{\mathrm{d}\lambda_2(t)}{\mathrm{d}t}=&\,\lambda_2(t)\eta,\\\frac{\mathrm{d}\lambda_3(t)}{\mathrm{d}t}=&-k_2+\frac{(1-u_2(t))\beta S_h^*(t)(S_h^*(t)+E_h^*(t)+R_h^*(t))}{H^*(t)^2}(\lambda_1(t)-\lambda_2(t)\theta-\lambda_4(t)\\&+\lambda_4(t)\theta)+\frac{(1-u_2(t+\tau))\beta S_h^*(t)(S_h^*(t)+E_h^*(t)+R_h^*(t))}{H^*(t)^2}\chi_{[0,t_f-\tau]}(\lambda_2(t+\tau)\\&-\lambda_3(t+\tau))\theta e^{-\eta\tau}+\frac{(1-u_1(t))acpS_v^*(t)(S_h^*(t)+E_h^*(t)+R_h^*(t))}{H^*(t)^2}(\lambda_5(t)\\&-\lambda_6(t))+\lambda_3(t)(\eta+\gamma+u_4(t)),\\\frac{\mathrm{d}\lambda_4(t)}{\mathrm{d}t}=&\,\lambda_4(t)\eta,\\\frac{\mathrm{d}\lambda_5(t)}{\mathrm{d}t}=&-k_3+(\lambda_5(t)-\lambda_6(t))\frac{(1-u_1(t))acpI_h^*(t)}{H^*(t)}+\lambda_5(t)(\nu+u_5(t)),\\\frac{\mathrm{d}\lambda_6(t)}{\mathrm{d}t}=&-k_4+(\lambda_1(t)-\lambda_2(t)\theta-\lambda_4(t)(1-\theta))\frac{(1-u_1(t))abS_h^*(t)}{H^*(t)}\\&+\chi_{[0,t_f-\tau]}(\lambda_2(t+\tau)-\lambda_3(t+\tau))\theta e^{-\eta\tau}\frac{(1-u_1(t+\tau))abS_h^*(t)}{H^*(t)}\\&+\lambda_6(t)(\nu+u_5(t)),\end{aligned}$$

with transversality conditions $\lambda_i(t_f)=0$, $i=1,2,\cdots,6$, where the characteristic function is

defined as

$$\chi_{[0,t_f-\tau]}(t)=\begin{cases}1, & \text{if } t\in[0,t_f-\tau],\\ 0, & \text{otherwise.}\end{cases}$$

In addition, the corresponding optimal controls are given as follows:

$$\begin{aligned}
u_1^*(t)&=\min\left\{\max\left\{\frac{\Omega(t)}{A_1},0\right\},u_{1\max}\right\},\\
u_2^*(t)&=\min\left\{\max\left\{\frac{\Delta(t)}{A_2},0\right\},u_{2\max}\right\},\\
u_3^*(t)&=\min\left\{\max\left\{\frac{(\lambda_1(t)-\lambda_4(t))S_h^*(t)}{A_3},0\right\},u_{3\max}\right\},\\
u_4^*(t)&=\min\left\{\max\left\{\frac{(\lambda_3(t)-\lambda_4(t))I_h^*(t)}{A_4},0\right\},u_{4\max}\right\},\\
u_5^*(t)&=\min\left\{\max\left\{\frac{\lambda_5(t)S_v^*(t)+\lambda_6(t)I_v^*(t)}{A_5},0\right\},u_{5\max}\right\},
\end{aligned}$$

where

$$\begin{aligned}
\Omega(t)&=(\theta\lambda_2(t)-\lambda_1(t)-(\theta-1)\lambda_4(t))\frac{abS_h^*(t)I_v^*(t)}{H^*(t)}+(\lambda_3(t)\\
&\quad-\lambda_2(t))\theta e^{-\eta\tau}\frac{abS_h^*(t-\tau)I_v^*(t-\tau)}{H^*(t-\tau)}+(\lambda_6(t)-\lambda_5(t))\frac{acpI_h^*(t)S_v^*(t)}{H^*(t)},\\
\Delta(t)&=(\theta\lambda_2(t)-\lambda_1(t)-(\theta-1)\lambda_4(t))\frac{\beta S_h^*(t)I_h^*(t)}{H^*(t)}\\
&\quad+(\lambda_3(t)-\lambda_2(t))\theta e^{-\eta\tau}\frac{\beta S_h^*(t-\tau)I_h^*(t-\tau)}{H^*(t-\tau)}.
\end{aligned}$$

Proof. Applying Pontryagin's Minimum Principle formulated in [9], there exist adjoint variables $\lambda_1(t)$, $\lambda_2(t)$, $\lambda_3(t)$, $\lambda_4(t)$, $\lambda_5(t)$, $\lambda_6(t)$ that satisfy the following equations:

$$\begin{cases}
\dfrac{\mathrm{d}\lambda_1(t)}{\mathrm{d}t}=-\dfrac{\partial K}{\partial S_h}-\chi_{[0,t_f-\tau]}(t)\dfrac{\partial K}{\partial S_{h\tau}}(t+\tau), & \lambda_1(t_f)=0,\\
\dfrac{\mathrm{d}\lambda_2(t)}{\mathrm{d}t}=-\dfrac{\partial K}{\partial E_h}, & \lambda_2(t_f)=0,\\
\dfrac{\mathrm{d}\lambda_3(t)}{\mathrm{d}t}=-\dfrac{\partial K}{\partial I_h}-\chi_{[0,t_f-\tau]}(t)\dfrac{\partial K}{\partial I_{h\tau}}(t+\tau), & \lambda_3(t_f)=0,\\
\dfrac{\mathrm{d}\lambda_4(t)}{\mathrm{d}t}=-\dfrac{\partial K}{\partial R_h}, & \lambda_4(t_f)=0,\\
\dfrac{\mathrm{d}\lambda_5(t)}{\mathrm{d}t}=-\dfrac{\partial K}{\partial S_v}, & \lambda_5(t_f)=0,\\
\dfrac{\mathrm{d}\lambda_6(t)}{\mathrm{d}t}=-\dfrac{\partial K}{\partial I_v}-\chi_{[0,t_f-\tau]}(t)\dfrac{\partial K}{\partial I_{v\tau}}(t+\tau), & \lambda_6(t_f)=0.
\end{cases}$$

Based on the constraints imposed on the control set, we derive

$$\begin{cases} \dfrac{\partial K}{\partial u_1} = A_1 u_1^*(t) - \Omega(t) = 0 & \text{at } u_1 = {u_1}^*, \\ \dfrac{\partial K}{\partial u_2} = A_2 u_2^*(t) - \Delta(t) = 0 & \text{at } u_2 = {u_2}^*, \\ \dfrac{\partial K}{\partial u_3} = A_3 u_3^*(t) - (\lambda_1(t) - \lambda_4(t)) S_h^*(t) = 0 & \text{at } u_3 = {u_3}^*, \\ \dfrac{\partial K}{\partial u_4} = A_4 u_4^*(t) - (\lambda_3(t) - \lambda_4(t)) I_h^*(t) = 0 & \text{at } u_4 = {u_4}^*, \\ \dfrac{\partial K}{\partial u_5} = A_5 u_5^*(t) - \lambda_5(t) S_v^*(t) - \lambda_6(t) I_v^*(t) = 0 & \text{at } u_5 = {u_5}^*. \end{cases}$$

Hence, we have

$$u_1^*(t) = \begin{cases} 0, & \dfrac{\Omega(t)}{A_1} < 0, \\ \dfrac{\Omega(t)}{A_1}, & 0 \le \dfrac{\Omega(t)}{A_1} \le u_{1\max}, \\ u_{1\max}, & \dfrac{\Omega(t)}{A_1} > u_{1\max}, \end{cases}$$

$$u_2^*(t) = \begin{cases} 0, & \dfrac{\Delta(t)}{A_2} < 0, \\ \dfrac{\Delta(t)}{A_2}, & 0 \le \dfrac{\Delta(t)}{A_2} \le u_{2\max}, \\ u_{2\max}, & \dfrac{\Delta(t)}{A_2} > u_{2\max}, \end{cases}$$

$$u_3^*(t) = \begin{cases} 0, & \dfrac{(\lambda_1(t) - \lambda_4(t)) S_h^*(t)}{A_3} < 0, \\ \dfrac{(\lambda_1(t) - \lambda_4(t)) S_h^*(t)}{A_3}, & 0 \le \dfrac{(\lambda_1(t) - \lambda_4(t)) S_h^*(t)}{A_3} \le u_{3\max}, \\ u_{3\max}, & \dfrac{(\lambda_1(t) - \lambda_4(t)) S_h^*(t)}{A_3} > u_{3\max}, \end{cases}$$

$$u_4^*(t) = \begin{cases} 0, & \dfrac{(\lambda_3(t) - \lambda_4(t)) I_h^*(t)}{A_4} < 0, \\ \dfrac{(\lambda_3(t) - \lambda_4(t)) I_h^*(t)}{A_4}, & 0 \le \dfrac{(\lambda_3(t) - \lambda_4(t)) I_h^*(t)}{A_4} \le u_{4\max}, \\ u_{4\max}, & \dfrac{(\lambda_3(t) - \lambda_4(t)) I_h^*(t)}{A_4} > u_{4\max}, \end{cases}$$

$$u_5^*(t) = \begin{cases} 0, & \dfrac{\lambda_5(t) S_v^*(t) + \lambda_6(t) I_v^*(t)}{A_5} < 0, \\ \dfrac{\lambda_5(t) S_v^*(t) + \lambda_6(t) I_v^*(t)}{A_5}, & 0 \le \dfrac{\lambda_5(t) S_v^*(t) + \lambda_6(t) I_v^*(t)}{A_5} \le u_{5\max}, \\ u_{5\max}, & \dfrac{\lambda_5(t) S_v^*(t) + \lambda_6(t) I_v^*(t)}{A_5} > u_{5\max}. \end{cases}$$

□

6. Numerical simulations

In this section, we present numerical simulations to illustrate the transmission dynamics of ZIKV and support our theoretical findings. In addition, we investigate optimal control strategies and perform sensitivity analysis of the basic reproduction number R_0.

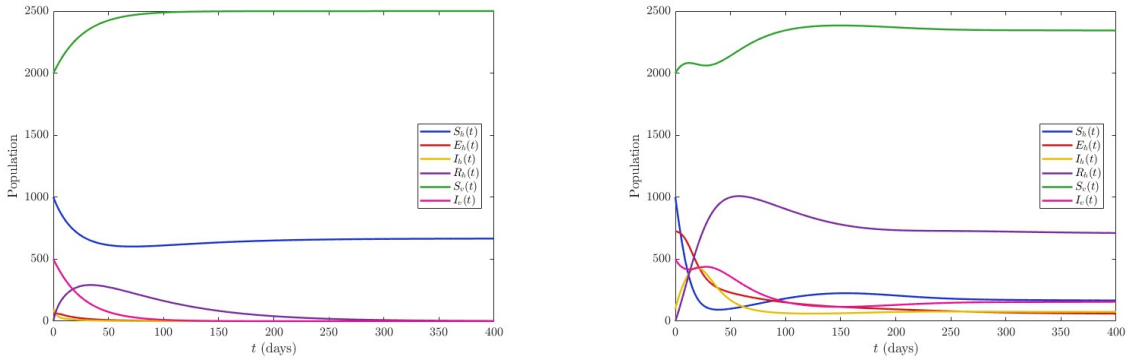
6.1. Long-term behavior

We perform numerical simulations using MATLAB to investigate the transmission dynamics of ZIKV. Using the parameter values listed in the first column of Table 2, we calculate $R_0 = 0.247 < 1$. With initial conditions $[S_h(\theta), I_h(\theta), R_h(\theta), S_v(\theta), I_v(\theta)] = [1000, 100, 0, 2000, 500]$ for all $\theta \in [-\tau, 0]$ and the initial value $E_h(0)$ calculated to be 63.5 from equation (2.2), the system dynamics are simulated over 400 days. The temporal variations in compartmental populations are shown in Figure 1(a). The results demonstrate that all epidemic compartments (E_h, I_h and I_v) asymptotically approach zero, while the susceptible human and vector populations converge, respectively, to the corresponding components of the disease-free equilibrium E_0.

Table 2. Model parameter values.

Parameters	Value 1	Value 2	Range	Unit	Reference
a	0.4	0.6	0.3-1	day^{-1}	[8]
b	0.1	0.3	0.1-0.75	dimensionless	[8]
c	0.4	0.3	0.3-0.75	dimensionless	[8]
β	0.015	0.3	0.001-0.4	day^{-1}	[8]
θ	0.35	0.7	0.1-0.8	dimensionless	[8]
η	0.015	0.01	0.0073-0.018	day^{-1}	[2]
γ	0.3	0.06	0.01-0.33	day^{-1}	[2]
p	0.1	0.2	0-0.5	dimensionless	[8]
ν	0.04	0.04	0.04-0.09	day^{-1}	[24]
δ_1	10	10	>1	individuals/day	[2]
δ_2	100	100	>1	mosquitoes/day	[2]
τ	10	10	3-14	day	[18]

We employ the parameter values given in the second column of Table 2 and then calculate $R_0 = 3.704 > 1$. With initial conditions $[S_h(\theta), I_h(\theta), R_h(\theta), S_v(\theta), I_v(\theta)] = [1000, 100, 0, 2000, 500]$ for all $\theta \in [-\tau, 0]$ and $E_h(0) = 726.7$ calculated from equation (2.2), the system dynamics are simulated over 400 days. The temporal variations in compartmental populations are shown in Figure 1(b). All human and vector populations converge to the endemic equilibrium. The infected compartments remain positive at the endemic equilibrium, indicating that ZIKV persists in the population. These numerical results validate the analysis of the endemic equilibrium, confirming the system's behavior when $R_0 > 1$. Keeping all other parameters fixed, τ is set to 4, 8 and 12. The trajectories of the I_h and I_v are shown in Figure 2. As τ increases, both the transient peak and the equilibrium level of I_h decrease. Likewise, increasing τ reduces the magnitude of I_v over time and leads to a lower endemic level, despite a slightly slower approach to equilibrium.



(a) The solution curves when $R_0 = 0.247 < 1$

(b) The solution curves when $R_0 = 3.704 > 1$

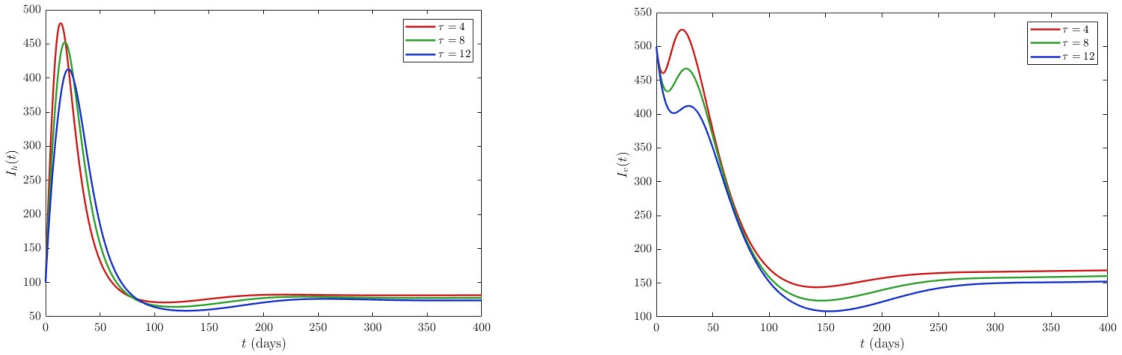
Figure 1. The solution curves of system (2.1).

6.2. Optimal control

We investigate the disease transmission dynamics under the proposed control measures to evaluate the effectiveness of the optimal control strategy. We set $k_1 = 1, k_2 = 20, k_3 = 1, k_4 = 1; A_1 = 1, A_2 = 1, A_3 = 2, A_4 = 3, A_5 = 1;\ u_{1\max} = 0.6, u_{2\max} = 0.6, u_{3\max} = 0.8, u_{4\max} = 0.3, u_{5\max} = 0.8$. Using the parameter values in the second column of Table 2, we perform numerical simulations and present the dynamics of the main compartments in Figure 3. Compared with the uncontrolled case, the susceptible human population S_h is maintained at a lower level under optimal control, while the optimal control strategy rapidly drives both the infected human population I_h and the infected mosquito population I_v to near zero. Meanwhile, the recovered human population R_h remains at a higher level under optimal control. These results indicate that the proposed control strategy can effectively suppress disease transmission by reducing infection levels and increasing the recovered population.

Figure 4(a) shows the numerical simulation results for individual control measures. Among these, treatment (u_4) appears to be the most effective, producing a rapid decline in infections with no apparent infection peak, and closely approximating the outcome of the overall optimal control strategy. Vaccination (u_3) appears to be the second most effective measure, also accelerating the decline of infected populations, although a small peak still occurs in the early stages of the outbreak. The other three control measures, when implemented individually, also exhibit certain inhibitory effects on disease transmission. However, none of these single interventions achieves the same level of suppression as the combined optimal control strategy.

In practice, implementing all control measures simultaneously may not always be feasible due to limited resources, financial constraints, or insufficient healthcare capacity. To further evaluate the effectiveness of different intervention schemes, we consider several combinations of control measures by allowing only selected controls to be optimized while setting the remaining controls to zero. Figure 4(b) illustrates the resulting dynamics of the infected human population under different control combinations. In regions where vaccination and treatment resources are limited, access to pharmaceutical interventions may be restricted. Under such circumstances, non-pharmaceutical measures, including reducing human–mosquito contact through mosquito nets and repellents (u_1), isolating infectious individuals (u_2), and controlling mosquito populations with insecticides (u_5), can still effectively reduce disease transmission. Although these strategies are less effective than vaccination or treatment, they can substantially slow the initial



(a) I_h with different time delays

(b) I_h with different time delays

Figure 2. Dynamics of I_h and I_v with different time delays.

growth of the epidemic and lower the infection peak, thereby providing a practical alternative when medical resources are constrained.

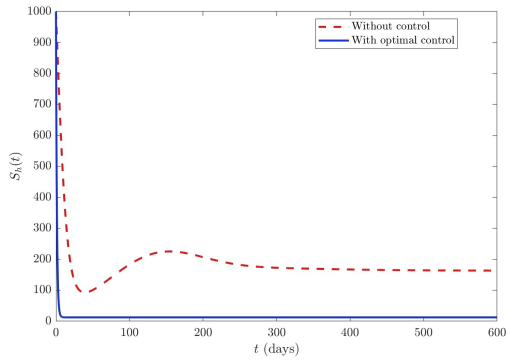
6.3. Sensitivity analysis

A global sensitivity analysis of the basic reproduction number R_0 is performed using Latin hypercube sampling (LHS) and partial rank correlation coefficients (PRCCs). Using the biologically plausible parameter ranges provided in Table 2, model parameters are sampled by LHS, and the corresponding values of R_0 are calculated. PRCCs are calculated to quantify the strength and direction of the monotonic association between each parameter and R_0. The resulting sensitivity ranking is shown in Figure 5(a). The vertical axis represents the PRCC values, where positive (negative) values indicate that a parameter is positively (negatively) correlated with R_0.

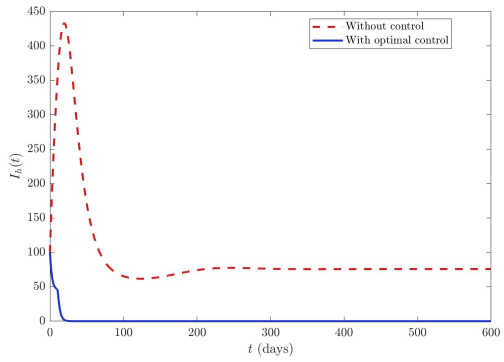
The sensitivity analysis reveals that R_0 is most positively sensitive to the proportion of newly infected individuals entering the exposed class (θ) and the mosquito recruitment rate (δ_2), indicating that increasing the proportion of newly infected individuals entering the exposed class and increasing the vector recruitment rate can substantially enhance disease transmission. The human-to-mosquito transmission probability (p) and the biting rate (a) also exert positive effects on R_0, reflecting the noticeable impact of cross-species transmission and contact frequency on disease transmission dynamics, whereas the human incubation period (τ), mosquito mortality rate (ν), human recruitment rate (δ_1), and especially the human recovery rate (γ) are negatively correlated with R_0.

These results provide important insights for disease control. In particular, interventions aimed at reducing the incidence of infection and strengthening treatment measures may have a significant positive impact on controlling the spread of ZIKV. Measures that limit vector proliferation and human–mosquito contact can substantially reduce transmission, while improvements in treatment and case management can further decrease the disease burden. Together, these interventions contribute to reducing R_0 and maintaining it below the epidemic threshold.

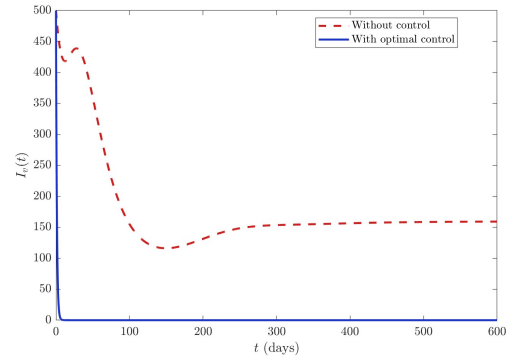
To further investigate the combined influence of the two most influential parameters, θ and γ, we conduct a joint sensitivity analysis of R_0. As shown in Figure 5(b), R_0 increases with increasing θ and decreasing γ, respectively. The largest values of R_0 occur when the proportion of newly infected individuals entering the exposed class is high and the recovery rate is low, whereas the opposite combination of parameter values leads to the smallest values of R_0. To



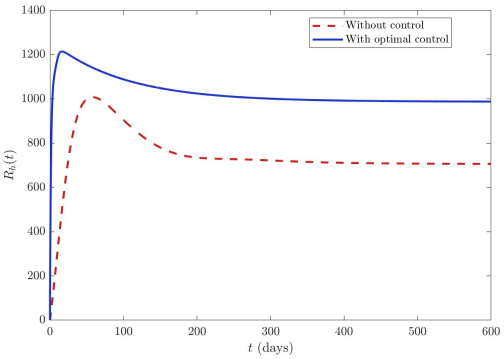
(a) S_h with and without optimal control



(b) I_h with and without optimal control



(c) I_v with and without optimal control



(d) R_h with and without optimal control

Figure 3. Dynamics of main compartments with and without optimal control.

quantify and compare their relative contributions, we further derive the following analytical expression. For any parameter ς related to R_0, the elasticity of R_0 is given as follows:

$$\varepsilon_{\varsigma}^{R_0} = \frac{\partial R_0}{\partial \varsigma} \cdot \frac{\varsigma}{R_0}. \tag{6.1}$$

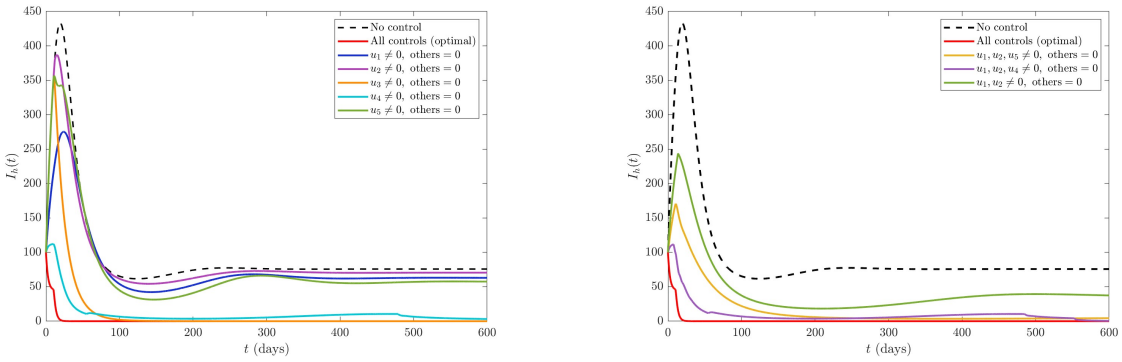
According to the explicit expression of R_0 and equation (6.1), we have

$$\varepsilon_{\theta}^{R_0} = \frac{1}{2}\left[\frac{e^{-\eta\tau}\beta}{\eta+\gamma} + \frac{\theta\left(\frac{e^{-\eta\tau}\beta}{\eta+\gamma}\right)^2 + \frac{2e^{-\eta\tau}a^2bcp\delta_2\eta}{\delta_1\nu^2(\eta+\gamma)}}{\sqrt{\left(\frac{\theta e^{-\eta\tau}\beta}{\eta+\gamma}\right)^2 + \frac{4\theta e^{-\eta\tau}a^2bcp\delta_2\eta}{\delta_1\nu^2(\eta+\gamma)}}}\right] \cdot \frac{\theta}{R_0},$$

$$\varepsilon_{\gamma}^{R_0} = \frac{1}{2}\left[-\frac{\theta e^{-\eta\tau}\beta}{(\eta+\gamma)^2} - \frac{\frac{(\theta e^{-\eta\tau}\beta)^2}{(\eta+\gamma)^3} + \frac{2\theta e^{-\eta\tau}a^2bcp\delta_2\eta}{\delta_1\nu^2(\eta+\gamma)^2}}{\sqrt{\left(\frac{\theta e^{-\eta\tau}\beta}{\eta+\gamma}\right)^2 + \frac{4\theta e^{-\eta\tau}a^2bcp\delta_2\eta}{\delta_1\nu^2(\eta+\gamma)}}}\right] \cdot \frac{\gamma}{R_0}.$$

It can be easily observed that

$$|\varepsilon_{\theta}^{R_0}| = \frac{\eta+\gamma}{\gamma}|\varepsilon_{\gamma}^{R_0}| > |\varepsilon_{\gamma}^{R_0}|.$$



(a) Comparison of individual control measures

(b) Comparison of combined control measures

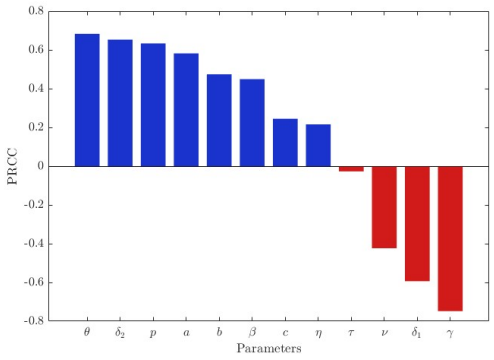
Figure 4. Dynamics of infected human population with non-global optimal control.

The above equation implies that R_0 is more sensitive to θ than to γ. This finding highlights the importance of surveillance and early intervention to reduce transmission before individuals progress to the infectious stage. Moreover, Figure 5(c) illustrates that R_0 decreases as τ increases, suggesting that a longer human incubation period slows the spread of the disease. Thus, these results imply that control efforts should focus on reducing the proportion of newly infected individuals entering the exposed class, enhancing recovery through effective treatment strategies, and limiting transmission opportunities.

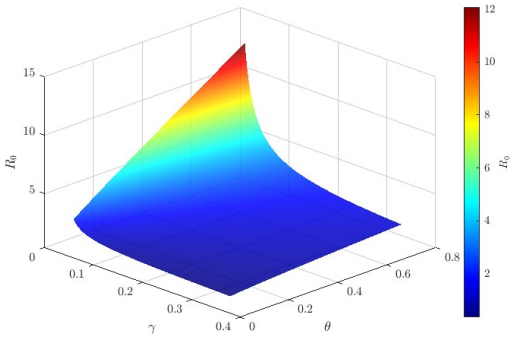
7. Conclusions

In this paper, we proposed a ZIKV transmission model with time delay and investigated its dynamical properties. Our theoretical results demonstrated that the long-term dynamics of the ZIKV transmission are characterized by the threshold value R_0. By constructing appropriate Lyapunov functionals and applying the LaSalle invariance principle, we established the global stability properties of the equilibria. Specifically, when $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, indicating that the ZIKV transmission will eventually die out. Conversely, when $R_0 > 1$, the endemic equilibrium becomes globally asymptotically stable, implying the persistent presence of the disease. Furthermore, by using Pontryagin's Minimum Principle for systems with delays, we have studied the optimal control problem and evaluated the effects of intervention measures, providing insights into effective approaches for mitigating ZIKV transmission.

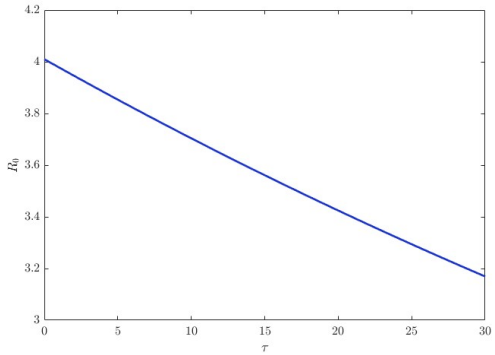
The numerical simulations further supported the theoretical results, illustrating the behavior of the system under different parameter configurations. This not only validated the theoretical analysis but also offered a clearer understanding of the ZIKV transmission dynamics. Further, we constructed a control model incorporating five control measures: reduction of human–vector contact, early detection and isolation of infected individuals, vaccination, treatment, and vector population control. We derived the adjoint equations for the adjoint variables λ and the characterization equations for the optimal controls u, thereby establishing the optimal control system. Numerical simulations demonstrated that the proposed control strategies effectively reduce the number of infected individuals and suppress disease transmission. Under the selected parameter settings, treatment exhibits the greatest impact on epidemic mitigation, followed by vaccination, while the remaining measures also contribute positively to disease control. These



(a) PRCC values of R_0



(b) R_0 vs θ and γ



(c) R_0 vs τ

Figure 5. Sensitivity analysis of R_0 with respect to various parameters.

results highlight the effectiveness and practicality of the proposed optimal control strategies in mitigating ZIKV outbreaks.

A sensitivity analysis based on PRCCs was conducted to identify the key parameters affecting R_0. The analysis quantified the sensitivity of R_0 to model parameters and time delay. Overall, the proposed model not only enhances the understanding of ZIKV transmission dynamics but also provides a theoretical basis for evaluating and optimizing public health intervention strategies. These findings emphasize the importance of mathematical modeling in understanding ZIKV transmission dynamics and guiding the development of effective prevention and intervention strategies.

Finally, we note some limitations of the current model and possible directions for future research. The global stability of the original system (2.1) without resorting to the limit system (3.3) remains to be established. Moreover, real-world populations often exhibit complex network structures, such as scale-free or small-world networks, which can qualitatively alter disease transmission dynamics [32]. Extending the model to incorporate such network structures would be a valuable direction for future work.

Acknowledgments

We are grateful to the referees for their valuable comments and suggestions which led to an improvement of our original manuscript.

References

[1] R. Buathong, L. Hermann, B. Thaisomboonsuk, W. Rutvisuttinunt, C. Klungthong, P. Chinnawirotpisan, W. Manasatienkij, A. Nisalak, S. Fernandez, I. K. Yoon, P. Akrasewi and T. Plipat, *Detection of Zika virus infection in Thailand, 2012–2014*, Am. J. Trop. Med. Hyg., 2015, 93, 380–383.

[2] D. Denu and H. Son, *Analysis and optimal control of a deterministic Zika virus model*, J. Nonlinear Sci. Appl., 2022, 15, 88.

[3] D. Diallo, A. A. Sall, C. T. Diagne, O. Faye, O. Faye, Y. Ba, K. A. Hanley, M. Buenemann, S. C. Weaver and M. Diallo, *Zika virus emergence in mosquitoes in Southeastern Senegal, 2011*, PLoS ONE, 2014, 9, e109442.

[4] G. W. A. Dick, S. F. Kitchen and A. J. Haddow, *Zika virus (I). Isolations and serological specificity*, Trans. R. Soc. Trop. Med. Hyg., 1952, 46, 509–520.

[5] P. van den Driessche, *Reproduction numbers of infectious disease models*, Infect. Dis. Model., 2017, 2, 288–303.

[6] P. van den Driessche and J. Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci., 2002, 180, 29–48.

[7] M. R. Duffy, T.-H. Chen, W. T. Hancock, et al., *Zika virus outbreak on Yap Island, Federated States of Micronesia*, N. Engl. J. Med., 2009, 360, 2536–2543.

[8] 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*, Sci. Rep., 2016, 6, 28070.

[9] L. Göllmann, D. Kern and H. Maurer, *Optimal control problems with delays in state and control variables subject to mixed control-state constraints*, Optim. Control Appl. Methods, 2009, 30, 341–365.

[10] S. Guo, M. He and F. Li, *Threshold dynamics of a time-delayed dengue virus infection model incorporating vaccination failure and exposed mosquitoes*, Appl. Math. Lett., 2025, 161, 109366.

[11] J. Gutkes, N. P. Krabbe, K. Ausderau and E. L. Mohr, *Macaque models of prenatal and postnatal Zika virus exposure and developmental outcomes*, J. Pediatric Infect. Dis. Soc., 2025, 14, piaf024.

[12] J. Hale and S. M. V. Lunel, *Introduction to Functional Differential Equations*, Springer, New York, 1993.

[13] M. Jamal, S. Batool, I. Ahmed, et al., *Mathematical modeling of Zika virus with vertical transmission in the presence of Wolbachia-infected mosquitoes*, J. Appl. Math. Comput., 2025, 71, 605–625.

[14] Y. Kuang, *Delay Differential Equations with Applications in Population Dynamics*, Academic Press, Boston, MA, 1993.

[15] R. S. Lanciotti, O. L. Kosoy, J. J. Laven, J. O. Velez, A. J. Lambert, A. J. Johnson, S. M. Stanfield and M. R. Duffy, *Genetic and serologic properties of Zika virus associated with an epidemic, Yap State, Micronesia, 2007*, Emerg. Infect. Dis., 2008, 14, 1232–1239.

[16] J. P. Romero-Leiton, E. K. E. Laisón, R. Alfaro, E. J. Parmley, J. Arino, K. R. Acharya and B. Nasri, *Exploring Zika's dynamics: A scoping review journey from epidemic to equations through mathematical modelling*, Infect. Dis. Model., 2025, 10, 536–558.

[17] F. Li and X.-Q. Zhao, *Global dynamics of a reaction–diffusion model of Zika virus transmission with seasonality*, Bull. Math. Biol., 2021, 83, 43.

[18] E. R. Krow-Lucal, B. J. Biggerstaff and J. E. Staples, *Estimated incubation period for Zika virus disease*, Emerg. Infect. Dis., 2017, 23, 841–845.

[19] F. N. MacNamara, *Zika virus: A report on three cases of human infection during an epidemic of jaundice in Nigeria*, Trans. R. Soc. Trop. Med. Hyg., 1954, 48, 139–145.

[20] A. R. Plourde and E. M. Bloch, *A literature review of Zika virus*, Emerg. Infect. Dis., 2016, 22, 1185–1192.

[21] R. Rakkiyappan, V. Latha, R. Preethi and F. A. Rihan, *A fractional-order model for Zika virus infection with multiple delays*, Complexity, 2019, 2019, 4178073.

[22] R. F. Relich and M. Loeffelholz, *Zika virus*, Clin. Lab. Med., 2017, 37, 253–267.

[23] Y. Saito, T. Hara and W. Ma, *Necessary and sufficient conditions for permanence and global stability of a Lotka–Volterra system with two delays*, J. Math. Anal. Appl., 1999, 236, 534–556.

[24] D. P. Shutt, C. A. Manore, S. Pankavich, A. T. Porter and S. Y. Del Valle, *Estimating the reproductive number, total outbreak size, and reporting rates for Zika epidemics in South and Central America*, Epidemics, 2017, 21, 63–79.

[25] H. L. Smith, *Monotone Dynamical Systems: An Introduction to the Theory of Competitive and Cooperative Systems*, American Mathematical Society, Providence, 1995.

[26] H. Song, S. Jin, G. J. Yang, N. W. H. Lim, J. T. Lim and B. L. Dickens, *Modelling the impact and cost-effectiveness of upcoming Zika virus vaccines on congenital Zika syndrome*, Vaccine, 2025, 48, 126728.

[27] J. Tognarelli, S. Ulloa, E. Villagra, et al., *A report on the outbreak of Zika virus on Easter Island, South Pacific, 2014*, Arch. Virol., 2016, 161, 665–668.

[28] J. Tran and W. A. Woldegerima, *Singular perturbation analysis of a two-time scale model of vector-borne disease: Zika virus model as a case study*, Chaos Solitons Fractals, 2025, 194, 116209.

[29] X. Wang, M. Shen, Y. Xiao and L. Rong, *Optimal control and cost-effectiveness analysis of a Zika virus infection model with comprehensive interventions*, Appl. Math. Comput., 2019, 359, 165–185.

[30] Y. Wang, Q. Yan and L. Liu, *Multi-scale modeling and analysis of vaccine prevention and control strategies for Chikungunya disease*, Adv. Contin. Discret. Models, 2026, 2026, 4.

[31] Z. Wang, S. Cai, G. Chen, et al., *Dynamics of dengue fever transmission model with multi-stage and fluctuations*, Mathematics, 2024, 12, 2491.

[32] Y. Wu, Z. Zhang, L. Song and C. Xia, *Global stability analysis of two strains epidemic model with imperfect vaccination and immunity waning in a complex network*, Chaos Solitons Fractals, 2024, 179, 114414.

[33] W. Zhang and C. T. Sandamali, *Modeling and analysis of low-level transmission ZIKV dynamics via a Poisson point process on sexual transmission route*, J. Appl. Anal. Comput., 2025, 13, 1044–1069.

[34] X.-Q. Zhao, *Dynamical Systems in Population Biology, Second Edition*, Springer-Verlag, New York, 2017.

Received January 2026; Accepted July 2026; Available online August 2026.