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Research article

A modified optimal control for the mathematical model of dengue virus with vaccination

Puntipa Pongsumpun[1], Jiraporn Lamwong[2], I-Ming Tang[3] and Puntani Pongsumpun[1,*]

[1] Department of Mathematics, School of Science, King Mongkut's Institute of Technology Ladkrabang, Bangkok 10520, Thailand
[2] Department of Applied Basic Subjects, Thatphanom College, Nakhon Phanom University, Nakhon Phanom 48000, Thailand
[3] Department of Physics, Faculty of Science, Mahidol University, Bangkok 10400, Thailand

* **Correspondence:** Email: puntani.po@kmitl.ac.th; Tel: +6623298000.

Abstract: The dengue viruses (of which there are four strains) are the causes of three illnesses of increasing severity; dengue fever (DF), dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). Recently, dengue fever has reached epidemic proportion in several countries. Strategies or preventative methods have to be developed to combat these epidemics. This can be done by development of vaccines or by preventing the transmission of the virus. The latter approach could involve the use of mosquito nets or insecticide spraying. To determine which strategy would work, we test the strategy using mathematical modeling to simulate the effects of the strategy on the dynamics of the transmission. We have chosen the Susceptible-Exposed-Infected-Recovered (SEIR) model and the Susceptible-Exposed-Infected (SEI) model to describe the human and mosquito populations, repectively. We use the Pontryagin's maximum principle to find the optimal control conditions. A sensitivity analysis revealed that the transmission rate (γ_h, γ_v), the birth rate of human population (μ_h), the constant recruitment rate of the vector population (A) and the total human population (N_h) are the most influential factors affecting the disease transmission. Numerical simulations show that the optimal controlled infective responses, when implemented, cause the convergence to zero to be faster than that in uncontrolled cases.

Keywords: dengue; mathematical model; optimal control; simulations; Lyapunov function; stabilities; sensitivity
Mathematics Subject Classification: 00A71

1. Introduction

Dengue is a vector-borne infectious disease that has rapidly spread to all regions of the world, mainly in tropical and sub-tropical countries. In the recent decades, the global incidence of dengue outbreak has grown dramatically around the world, and some places, dengue is now a greater threat to people than coronavirus disease 2019 (COVID-19). Dengue is a challenging disease in developing countries due to a shortage of medical personnel and many other economic constraints [1–3]. The number of dengue cases reported to the World Health Organization (WHO) has increased more than ten times over the last two decades, from 0.5 million cases in 2000, to 5.2 million cases in 2019. Between the years 2000 and 2015, children had the highest mortality rate [4]. In 2019, WHO listed dengue as one of the top ten major threats to global health [5]. In Thailand, dengue is currently listed as a "neglected tropical disease". The number of new dengue cases of infection has gradually increased [6]. Thailand is ranked among the thirty countries with the highest number of dengue fever outbreak in the world. It was found that among the population with dengue fever outbreaks around the world, more than three-thirds of the world's infected population is in Southeast Asia. In 2023, Thailand has had a serious out-break of dengue fever because of the rainy season and El Niño phenomenon, which is a major threat to public health [1]. Dengue has been becoming a major public health problem in Thailand.

Dengue is caused by one of four known strains of dengue virus, i.e., DENV-1, DENV-2, DENV-3 and DENV-4. Dengue virus can be transmitted by the bites of infected female *Aedes* mosquitoes. The main vector is the mosquito *Aedes aegypti*, while the other *Aedes* species such as *Aedes albopictus*, *Aedes polynesiensis* and *Aedes scutellaris* have a limited capacity to serve as dengue vectors [7,8]. WHO has classified dengue disease into dengue fever (DF), dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) [6]. The three stages of dengue disease symptoms are different. The symptoms of dengue fever are those of a mild cold. Dengue hemorrhagic fever (DHF) can cause serious blood discharge from the blood vessel and when the blood pressure drops rapidly (shock), it becomes dengue shock syndrome (DSS) [9,10]. In most cases, people are rarely found to have DSS and DHF, with the latter leading to death [11]. Currently, there are no effective antiviral drugs and vaccines for treating and controlling dengue [6]. For this reason, we focus on the use of mathematical modeling to describe the dynamics of the transmission of DF in Thailand and use it to qualitatively analyze the epidemic in the country.

Several mathematical modeling studies have been done to develop and identify the transmission dynamics of dengue in the human population [8,10,12–21]. The SEIR model is known as the basic mathematical model for disease dynamics. At any given time, a population of size N is categorized into sub-populations, e.g., those who are susceptible to the infection (S), those who are exposed to the virus (E), those who are infectious and can transmit the virus to others (I) and those who have recovered (R). The purpose of this study is to construct a mathematical model that describes the transmission dynamics in Thailand and use the model to control the spread of the disease in Thailand. Creating an optimal control model for the spread of the disease and finding an optimal control strategy are now sought to help reduce the number of infected populations, control the spread of the disease and reduce the cost of management related to disease control. In conclusion, the use of the control strategy for disease prevention is a guideline to help prevent and control the spread of the disease.

Much research has already been done. For example, in 2006, Nishiura [12] summarized the recent researches about mathematical and statistical approaches on dengue epidemiology regardless of the mathematical background. They also stated that the operational researchers have very little to work on

when compared to those in mathematical modeling, due to the impossibility of alternating the health treatment in real life. In 2010, Supriatna et al. [10] studied mathematical models on dengue transmission with four strains, which are denoted by dengue I-IV. They formulated mathematical models of the critical vaccination level for indirect transmission of the disease. The mathematical model was created to consider the transmission of dengue fever by taking into account the iceberg phenomenon, dividing the illnesses into asymptomatic, mild and severe infections. In 2014, Isea and Puerta [13] constructed a dengue model of the transmission dynamics in each dengue strain with a SEIR-SEI model. The paper indicates that the model analyzing each of the four strains is very similar to that of the two-strain model, thus it can be said that analytical studies of four dengue strains of the epidemiology are not necessary.

In 2015, Phaijoo and Gurung [14] presented a mathematical model of dengue fever with the transmission dynamics of dengue disease and absence of awareness in host population. The effect of awareness parameters were included in the transmission dynamics of this dengue modeling. In 2017, Pongsumpun [15] studied the transmission of dengue disease by formulating a mathematical model. The transmission of dengue disease between human and vector population was considered in the dengue model. In 2019, Pongsumpun et al. [16] developed a control mechanism in the model of the dengue disease by including the effects of vertical transmission in their numerical analyses of the optimal control in the model. Sungchasit and Pongsumpun [17] investigated the development of dengue disease infection from dengue fever (DF) to dengue hemorrhagic fever (DHF) by using the SEIR model for humans and SEI model for mosquitoes. In 2021, Khan and Fatmawati [18] displayed a mathematical model of dengue fever with hospitalization to explain the dynamics of the infection. The results showed that the protection from mosquitoes and using insecticide spray can significantly lessen the dengue infection and may reduce the spread of infectious disease in the community. In 2022, Affandi et al. [19] proposed mathematical models with optimal control that predicted a decrease in the spread of dengue disease when the controls were used. Schaum et al. [20] developed a mathematical model for the epidemic dengue that is based on a Markov–like continuous-time process by using a simple six state automaton to represent the mixed human-mosquito population. Li and Gao have done several studies [21–25] using mathematical models and optimal control for epidemic control and online game addiction modeling. Li and Gao [21] studied transmission of the COVID-19 epidemic by formulating mathematical models and optimal control. They found that vaccination, isolation and nucleic acid testing are three control measures of the optimal control measure to control the spread of mutated COVID-19 (Delta strain) with imperfect vaccination. Gao and Li [22] examined mathematical model of novel coronavirus pneumonia (COVID-19) to investigate the effect of the Chinese government's epidemic prevention in China. They concluded that intervention by the Chinese government was effective in preventing the spread of COVID-19. Gao and Li [23] used mathematical models and optimal control theory to apply a game addiction model considering family education. Their research has found that both avoiding video game addiction and controlling the epidemic of adolescent game addiction can be controlled with adequate financial resources and increased family education. In 2023, Gao and Li [24] constructed the new fractional model for online game addiction and simulated online gaming addiction in China. They generated partial differential equations involving Caputo derivatives of fractional variable order for controlling the spread of online games. Gao and Li [25] used an 11-dimensional mathematical model for studying the co-transmission of Omicron and Delta strains of COVID-19. Vaccination with the third dose of a vaccine, isolation and national nucleic acid testing are control measures that can reduce the number of infected people in the optimal control. The optimal control of the spread of the Omicron variant of COVID-19 virus when vaccination against this virus was performed was recently done by Lamwong, Pongsumpun, Tang and

Wongvanich, [53]. In that study, it was found that optimal control could be achieved where the most effective control strategy is controlling the rate of vaccinated people and immunity achieved from vaccination to reduce the spread of COVID-19. The spread can be minimized by planning and control strategy. Therefore, mathematical modeling has proven to be a powerful tool for simulating the results, making predictions and gaining insights into the behavior of biological systems [24].

The data for the dengue disease in Thailand from 2013 to 2022 [26] are shown in Figure 1. It shows that the dengue cases are high in the rainy season (May to October) because Aedes mosquitoes breed a lot in the rainy season. But in big cities such as Bangkok, this disease may be found throughout the year.

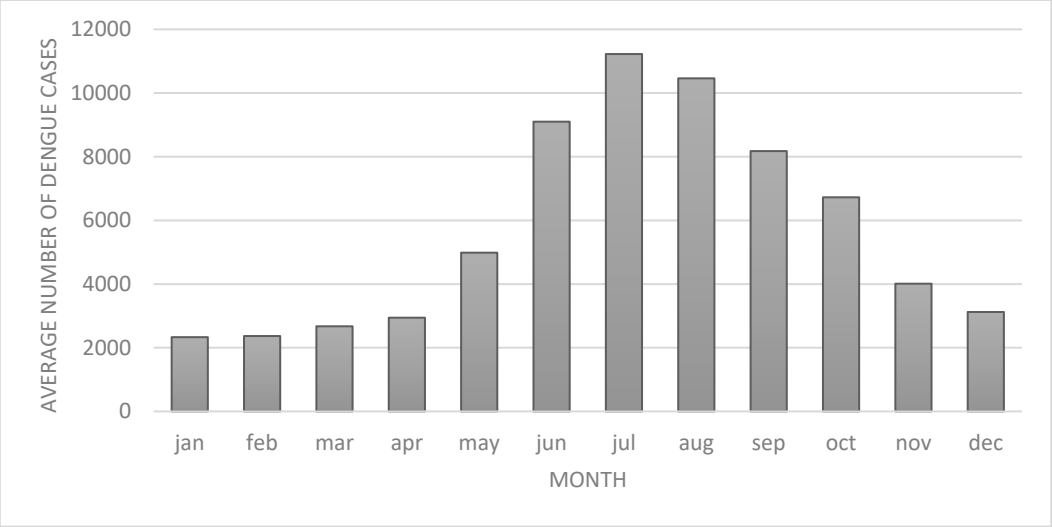


Figure 1. Number of dengue cases in Thailand from 2013 to 2022 [26].

In recent years, several vaccines have been developed for the treatment of dengue fever. Sanofi Pasteur's Dengvaxia® (CYD-TDV) is the only licensed dengue vaccine approved by the United States (US) Food and Drug Administration (FDA). Data from tested cases indicate that the CYD-TDV vaccine can give a high level of protection (82%) against dengue infection for 9–16 year-old children [27,28]. But this vaccine has disadvantages because it depends on the serostatus of the recipient [29]. There are other dengue vaccine candidates in ongoing clinical testing with promising results, including TDV (TAK-003) and TV003/TV005. To develop a dengue vaccine, it should provide protection against all four dengue serotypes, which can protect from risks of antibody-dependent enhancement [30,31]. Dengue vaccine acceptance has obviously changed during coronavirus (COVID-19) outbreak. For this reason, it may have an impact on the use of dengue fever vaccines. Mathematical modelling is a method to test whether a given intervention strategy will help in alleviating the course of an epidemic. There may be many strategies that could accomplish this. The aims of these strategies could be saving of money, the better allotment of resources or the combination of both. The optimal control approach method is one way to determine which set of strategies is best to control the dengue fever epidemic when vaccination against the fever is taking place.

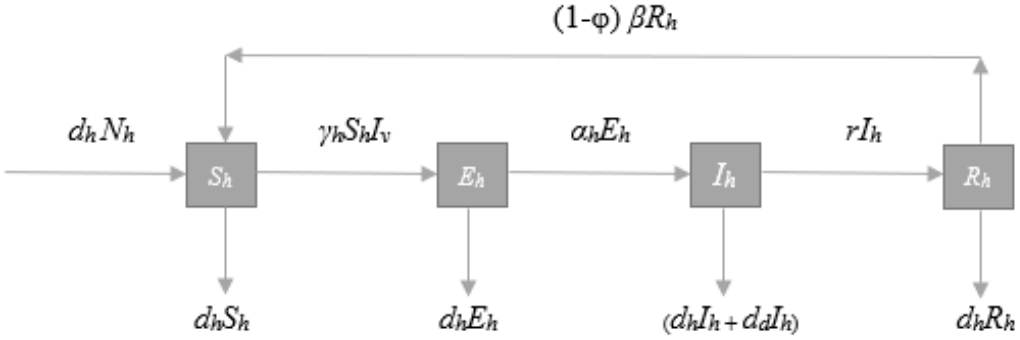
In this study, the dynamical model of dengue disease is constructed by separating the populations into human and mosquito populations. The human population is modeled using the susceptible-exposed-infectious-recovered (SEIR) framework, whereas the mosquito's population is modeled through the susceptible-exposed-infectious (SEI) framework. Dynamical analyses of the presented

model are given, including local and global stability analyses. An optimal controller was designed based on the Pontryagin maximum principle to minimize the number of infected humans. The strategy used in designing the Hamiltonian objective function in this study has been modified from the typical approaches found in the literature. More emphasis has been placed on prevention strategies, such as using mosquito nets, window screens and insecticide spraying, rather than treatment strategies, such as taking medication and vaccination [16,43,44]. The former strategies are more commonly used in the rural areas of Thailand, where it is more economical to seek the prevention strategies in lieu of the medical treatment strategies.

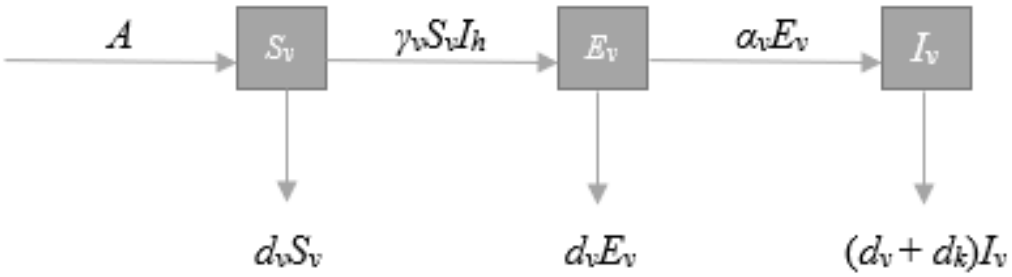
2. Materials and methods

2.1. Mathematical model

We separate the human into susceptible, exposed, infectious and recovered populations (SEIR). Mosquitoes are separated into susceptible, exposed and infectious populations (SEI). The relationship between human and mosquitoes are shown as in Figure 2. The variables and parameters for human and mosquito populations are defined in Table 1 and Table 2.



(a) Human populations



(b) Mosquito populations

Figure 2. Diagram showing the relationship between human and mosquitoes.

We can write the dynamical equations as follows:

$$\frac{dS_h}{dt} = d_h N_h - \gamma_h S_h I_v + (1-\varphi)\beta R_h - d_h S_h, \tag{1}$$

$$\frac{dE_h}{dt} = \gamma_h S_h I_v - (\alpha_h + d_h) E_h, \tag{2}$$

$$\frac{dI_h}{dt} = \alpha_h E_h - (r + d_h + d_d) I_h, \tag{3}$$

$$\frac{dR_h}{dt} = rI_h - (1-\varphi)\beta R_h - d_h R_h, \tag{4}$$

and

$$N_h = S_h + E_h + I_h + R_h. \tag{5}$$

Table 1. Definition of variables and parameters for human population.

Variables and parameters	Description
μ_h	The birth and natural death rate of human population,
N_h	The total human population,
γ_h	The transmission rate of dengue virus from human to vector population,
d_d	The disease death rate of human population,
α_h	The incubation rate of dengue virus in humans,
r	The recovery rate of dengue virus,
β	The rate at which the recovery human changed to be susceptible human,
ϕ	The vaccine efficiency,
S_h	The susceptible human population,
E_h	The exposed human population,
I_h	The infectious human population,
R_h	The recovered human population.

For the mosquito population, we have

$$\frac{dS_v}{dt} = A - \gamma_v S_v I_h - d_v S_v, \tag{6}$$

$$\frac{dE_v}{dt} = \gamma_v S_v I_h - (\alpha_v + d_v) E_v, \tag{7}$$

$$\frac{dI_v}{dt} = \alpha_v E_v - (d_v + d_k) I_v, \tag{8}$$

and

$$N_v = S_v + E_v + I_v. \tag{9}$$

Table 2. Definition of variables and parameters for mosquito population.

Variables and parameters	Description
N_v	The total vector population,
A	The constant recruitment rate of vector population,
γ_v	The transmission rate of dengue disease from vector to human population,
d_v	The natural death rate of vector population,
d_k	The disease death rate of vector population,
α_v	The incubation rate of dengue virus in vector population,
S_v	The susceptible human population,
E_v	The exposed human population,
I_v	The infectious human population.

3. Analysis of the model

3.1. The equilibrium points of the model

Definition 1. The equilibrium point $X^* \in R^n$ is defined as $\frac{dX}{dt} = f(t,x)$. And $f(t,x) = 0$ for all *t*.

We assume that the dynamics of a system is described by a differential equation. An essential step in the analysis of a differential equation is the determination of equilibrium points and the study of their stability. Equilibrium points of the model are determined by setting the right-hand side of Eqs (1)–(4) and (6)–(8) to zero. Then the disease-free equilibrium point is defined by

$$B_0^* = \left(\text{ɴʜ} , 0,0,0,\frac{A}{d_v}, 0,0\right). \tag{10}$$

The endemic equilibrium point is defined by $B_1^* = \left(S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*\right)$, where

$$S_h^* = \frac{((\alpha_h+d_h)(\alpha_v+d_v)(d_k+d_v)(d_d+d_h+r)H}{\alpha_h\gamma_v T}, \tag{11}$$

$$E_h^* = \frac{P(d_d+d_h+r)}{\alpha_h T}, \tag{12}$$

$$I_h^* = \frac{P}{T}, \tag{13}$$

$$R_v^* = \frac{rP}{(\beta+d_h+\beta\varphi)T}, \tag{14}$$

$$S_v^* = \frac{U}{\alpha_v\gamma_h H}, \tag{15}$$

$$E_v^* = \frac{P}{\alpha_V(\alpha_v+d_v)\gamma_h H}, \tag{16}$$

$$I_v^* = \frac{P}{(\alpha_v+d_v)(d_k+d_v)\gamma_h H}, \tag{17}$$

and

$$H = \left((\alpha_h + d_h)(d_d + d_h)d_v + \alpha_h d_h\gamma_v N_h\right)(\beta + d_h + \beta\varphi) + d_h d_v(\alpha_h + \beta + d_h - \beta\varphi)\mathrm{r}, \tag{18}$$

$$P = d_h(\beta(1-\varphi) + d_h)(d_h d_v(\alpha_v + d_v)(d_k + d_v)(d_d+d_h+\mathrm{r})+\alpha_h(d_v^2(d_k+d_v)(d_d+d_h+\mathrm{r}) +\alpha_v(A\gamma_h\gamma_v N_h+d_v(d_k+d_v)(d_d+d_h+\mathrm{r})))), \tag{19}$$

$$T = \gamma_v(d_h(d_h(\alpha_v + d_v)(d_k + d_v) + A\alpha_v\gamma_h)((\beta(1-\varphi) + d_h)(d_d+d_h+\mathrm{r}) + \alpha_h (d_h(d_h(d_k + d_v) + A\gamma_h)(d_d + d_h + r)+\beta(1-\varphi)((d_d + d_h)(d_h(d_k + d_v) + A\gamma_h) +d_h(d_k + d_k)r)))), \tag{20}$$

$$U = d_h(d_h(\alpha_v + d_v)(d_k + d_v) + A\alpha_v\gamma_h)((\beta(1-\varphi) + d_h)(d_d+d_h+\mathrm{r}) + \alpha_h(\alpha_v(d_d + d_h) (d_h(d_k+d_k)+A\gamma_h)(\beta(1-\varphi) + d_h)+ (\alpha_v d_h(A\gamma_h+(d_k + d_v)(\beta(1-\varphi) + d_h))\mathrm{r}+d_h d_v (d_k + d_v)(\beta(1-\varphi) + d_h)(d_d+d_h+\mathrm{r})). \tag{21}$$

3.2. The basic reproduction number

The basic reproductive number (R_0) is the key measure to estimate the ability of the disease to spread. It is determined as the average number of secondary transmissions from one infected case. If R_0 is more than 1, the disease epidemic is growing. The values of R_0 have important implications for controlling disease. It indicates the level of mitigation efforts needed to bring an epidemic under control. Mitigation measures include rapid case identification, quarantine measures and physical distancing to prevent secondary transmissions. It brings down the effective transmission coefficient. The basic reproductive number was evaluated by using the next-generation method [20,37,38,40,44]. The states E_h, I_h, E_v and I_v are

$$\begin{vmatrix} \boldsymbol{Gains\ to\ E_h} & \boldsymbol{\gamma_h S_h I_v} \\ \boldsymbol{Gains\ to\ I_h} & \boldsymbol{0} \\ \boldsymbol{Gains\ to\ E_v} & \boldsymbol{\gamma_v S_v I_h} \\ \boldsymbol{Gains\ to\ I_v} & \boldsymbol{0} \end{vmatrix}, \quad \begin{vmatrix} \boldsymbol{Losses\ from\ E_h} & \boldsymbol{(\alpha_h + d_h)E_h} \\ \boldsymbol{Losses\ from\ I_h} & \boldsymbol{-\alpha_h E_h + (r + d_h + d_d)I_h,} \\ \boldsymbol{Losses\ from\ E_v} & \boldsymbol{(\alpha_v + d_v)E_v} \\ \boldsymbol{Losses\ from\ I_v} & \boldsymbol{-\alpha_v E_v + (d_v + d_k)I_v} \end{vmatrix}.$$

Where $\boldsymbol{F}$ is the Jacobian matrix of the gains matrix and $\boldsymbol{V}$ is the Jacobian matrix of the losses matrix. Thus we have

$$\boldsymbol{F} = \begin{bmatrix} \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\gamma_h S_h} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{\gamma_v S_v} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} \end{bmatrix}, V = \begin{bmatrix} \boldsymbol{\alpha_h + d_h} & \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{-\alpha_h} & \boldsymbol{r + d_h + d_d} & \boldsymbol{0} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{\alpha_v + d_v} & \boldsymbol{0} \\ \boldsymbol{0} & \boldsymbol{0} & \boldsymbol{-\alpha_v} & \boldsymbol{d_v + d_k} \end{bmatrix}.$$

Disease-free equilibrium point:

$$B_0^* = (S^*_h, E^*_h, I^*_h, R^*_h, S^*_v, E^*_v, I^*_v) = \left(\mathcal{N}_h, 0,0,0, \frac{A}{d_v}, 0,0 \right)$$

Since $R = FV^{-1}$, $R_0$is the eigenvalue of the matrix R considered from the most positive eigenvalue. So the formula shall be:

$$R_0 = \sqrt{\frac{A\gamma_h\gamma_v N_h \alpha_h \alpha_v}{d_v(d_k+d_v)(d_d+d_h+r)(d_h+\alpha_h)(d_v+\alpha_v)}}. \tag{22}$$

3.3. Global stability analysis

The global stability analysis of two equilibrium points of Eqs (1)–(7) is established as follows:

Theorem 1. The disease free equilibrium point B_0^* of Eqs (1)–(4) and (6)–(8) is global symptomatically stable in θ if $R_0 < 1$.

We let $$\gamma_h = \frac{D_V}{N_h} \quad \text{and} \quad \gamma_V = \frac{d_h d_V}{A}. \tag{23}$$

Proof. We consider the Lyapunov function defined by

$$L = (S_h - S_h^* \ln S_h) + E_h + I_h + R_h + (S_v - S_v^* \ln S_v) + E_v + I_v$$

$$\frac{d}{dt}L = (d_h N_h - \gamma_h S_h I_v + (1 - \varphi)\beta R_h - d_h S_h)\left(1 - \frac{S_h^*}{S_h}\right) + (\gamma_h S_h I_v - (\alpha_h + d_h)E_h) + (\alpha_h E_h$$

$$(r + d_h + d_d)I_h) + \left(rI_h - \left((1 - \varphi)\beta + d_h\right)R_h\right) + (A - \gamma_v S_v I_h - d_v S_v)\left(1 - \frac{S_v^*}{S_v}\right)$$
$$+(\gamma_v S_v I_h - (\alpha_v + d_v)E_v) + (\alpha_V E_V - (d_v + d_k)I_v)$$

$$= -(-d_h N_h - A + d_h(S_h + E_h + I_h + R_h) + d_v(S_v + E_v + I_v) + d_d I_h + d_k I_v) + \frac{S_h^*}{S_h}(-d_h N_h$$
$$-(1 - \varphi)\beta R_h + d_h S_h + \gamma_h S_h I_v) + \frac{S_v^*}{S_v}(-A + \gamma_v I_h S_v + d_v S_v)$$

$$= -(d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)$$
$$\beta R_h \frac{S_h^*}{S_h} + \gamma_h I_v S_h^* + A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + \gamma_v I_h S_v^*$$

We substitute Eq (23), and we have

$$\frac{d}{dt}L = -(d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)\beta R_h$$
$$\frac{S_h^*}{S_h} + \frac{dv}{N_h} I_V S_h^* + A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + \frac{d_h d_V}{A} I_h S_v^*. \quad (24a)$$

At the disease free equilibrium point $S_h^* = N_h$, $S_v^* = \frac{A}{d_v}$, after we substitute them into Eq (24a) then

$$\frac{d}{dt}L = -(d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)$$
$$\beta R_h \frac{(N_h)}{S_h} + \frac{dv}{N_h} I_V (N_h) + A\left(2 - \frac{S_V^*}{S_V} - \frac{S_V}{S_V^*}\right) + \frac{d_h d_V}{A} I_h \left(\frac{A}{d_V}\right)$$

$$= -(d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)$$
$$\beta R_h \frac{(N_h)}{S_h} + d_V I_v + A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + d_h I_h$$

$$= -(d_h(E_h + R_h) + d_v E_v + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right)N - (1 - \varphi)\beta R_h \frac{(N_h)}{S_h}$$
$$+ A\left(2 - \frac{S_V^*}{S_V} - \frac{S_V}{S_V^*}\right)$$

$$= -(d_h(E_h + R_h) + d_v E_v + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)\beta R_h \frac{(N_h)}{S_h}$$
$$+ A\left(2 - \frac{S_V^*}{S_V} - \frac{S_V}{S_V^*}\right)$$

$$= -(d_h(E_h + R_h) + d_vE_v + d_dI_h + d_kI_v) - d_hN_h\left(\frac{(-S_h + S_h^*)^2}{S_hS_h^*}\right) - (1 - \varphi)\beta R_h\frac{(N_h)}{S_h}$$
$$-A\left(\frac{(-S_v + S_v^*)^2}{S_vS_v^*}\right)$$

$$\frac{d}{dt}L = -\left[(d_h(E_h + R_h) + d_vE_v + d_dI_h + d_kI_v + d_hN_h\left(\frac{(-S_h+S_h^*)^2}{S_hS_h^*}\right) + (1 - \varphi)\beta R_h\frac{Nh}{S_h} + A\left(\frac{(-S_v+S_v^*)^2}{S_vS_v^*}\right)\right] \leq 0. \quad (24b)$$

From LaSalle's invariant principal [36,37] and knowing that $\frac{d}{dt}L$ is not positive, we can say that the disease-free equilibrium point B_0^* is global asymptotically stable in θ if $R_0 < 1$.

Theorem 2. The endemic equilibrium point B_1^* of Eqs (1)–(4) and (6)–(8) is global asymptotically stable in θ if $R_0 > 1$.

We let $\gamma_h = \frac{d_v\alpha_h\gamma_vT}{((\alpha_h+d_h)(\alpha_v+d_v)(d_k+d_v)(d_d+d_h+r)H}$ and $\gamma_v = \frac{d_h\alpha_v\gamma_hH}{U}$. (25)

Proof. The Lyapunov function is defined by

$$M = (S_h - S_h^* ln S_h) + E_h + I_h + R_h + (S_v - S_v^* ln S_v) + E_v + I_v,$$

$$\frac{d}{dt}M = (d_hN_h - \gamma_hS_hI_v + (1 - \varphi)\beta R_h - d_hS_h)\left(1 - \frac{S_h^*}{S_h}\right) + (\gamma_hS_hI_v - (\alpha_h + d_h)E_h) + (\alpha_hE_h$$
$$-(r + d_h + d_d)I_h) + (rI_h - ((1 - \varphi)\beta + d_h)R_h) + (A - \gamma_vS_vI_h - d_vS_v)\left(1 - \frac{S_v^*}{S_v}\right)$$
$$+(\gamma_vS_vI_h - (\alpha_v + d_v)E_v) + (\alpha_v E_v - (d_v + d_k)I_v)$$
$$= -(-d_hN_h - A + d_h(S_h + E_h + I_h + R_h) + d_v(S_v + E_v + I_v) + d_dI_h + d_kI_v) +$$
$$\frac{S_h^*}{S_h}(-d_hN_h - (1 - \varphi)\beta R_h + d_hS_h + \gamma_hS_hI_v) + \frac{S_v^*}{S_v}(-A + \gamma_vI_hS_v + d_vS_v)$$

$$= -(d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_dI_h + d_kI_v) + d_hN_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) -$$
$$(1 - \varphi)\beta R_h\frac{S_h^*}{S_h} + \gamma_hI_vS_h^* + A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + \gamma_vI_hS_v^*.$$

We substitute Eq (25) then

$$\frac{d}{dt}M = -(d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_dI_h + d_kI_v) + d_hN_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right)$$
$$-(1 - \varphi)\beta R_h\frac{S_h^*}{S_h} + \frac{d_v\alpha_h\gamma_vT}{((\alpha_h + d_h)(\alpha_v + d_v)(d_k + d_v)(d_d + d_h + r)H}I_vS_h^*$$
$$+A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + \frac{d_h\alpha_v\gamma_hH}{U}I_hS_v^*$$

We substitute the endemic equilibrium point $S_h^* = \frac{((\alpha_h+d_h)(\alpha_v+d_v)(d_k+d_v)(d_d+d_h+r)H}{\alpha_h\gamma_v T}$ and $S_v^* = \frac{U}{\alpha_v\gamma_h H}$ into Eq (26) and we get

$$\frac{d}{dt}M = -(\ d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) -$$

$$(1 - \varphi)\beta R_h \frac{S_h^*}{S_h} + \frac{d_v\alpha_h\gamma_v T}{((\alpha_h+d_h)(\alpha_v+d_v)(d_k+d_v)(d_d+d_h+r)H} I_v \frac{((\alpha_h+d_h)(\alpha_v+d_v)(d_k+d_v)(d_d+d_h+r)H}{\alpha_h\gamma_v T}$$

$$+A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + \frac{d_h\alpha_v\gamma_h H}{U} I_h \frac{U}{\alpha_v\gamma_h H}$$

$$= -(\ d_h(E_h + I_h + R_h) + d_v(E_v + I_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)$$

$$\beta R_h \frac{S_h^*}{S_h} + d_v I_v + A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right) + d_h I_h$$

$$= -(\ d_h(E_h + R_h) + d_v(E_v) + d_d I_h + d_k I_v) + d_h N_h\left(2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}\right) - (1 - \varphi)\beta R_h \frac{S_h^*}{S_h} +$$

$$A\left(2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}\right)$$

$$= -(\ d_h(E_h + R_h) + d_v(E_v) + d_d I_h + d_k I_v) - d_h N_h\left(\frac{(-S_h + S_h^*)^2}{S_h S_h^*}\right) - (1 - \varphi)\beta R_h \frac{S_h^*}{S_h} -$$

$$A\left(\frac{(-S_v + S_v^*)^2}{S_v S_v^*}\right)$$

$$\frac{d}{dt}M = -\ (\ d_h(E_h + R_h) + d_v(E_v) + d_d I_h + d_k I_v + d_h N_h\left(\frac{(-S_h + S_h^*)^2}{S_h S_h^*}\right) + (1 - \varphi)\beta R_h \frac{S_h^*}{S_h}$$

$$+\ A\left(\frac{(-S_v + S_v^*)^2}{S_v S_v^*}\right))$$

$$\leq 0. \tag{27}$$

We will see that $\frac{d}{dt}M$ is nonpositive if $R_0 > 1$, implying that the endemic equilibrium B_1^* is globally asymptotically stable in θ.

3.4. Numerical analysis

In this section, we simulate the numerical results for the Eqs (1)–(4) and (6)–(8). The values of each parameter are given in Table 3. Note that the basic reproduction number for the disease free state is determined to be 0.61, while the basic reproduction number for the endemic state is 610.69.

We see that the trajectories of all the system states converge to the disease-free state (1000, 0, 0, 0, 120, 0, 0) when $R_0 < 1$ and that these trajectories converge to the endemic point (0.01, 399, 200, 9400, 8, 5240, 3790) when $R_0 > 1$ as shown in Figures 3 and 4. When we vary the transmission rate of dengue virus from human to vector population and the different vaccine efficiency, we will see that the time of the epidemic peak and the length of outburst are different, as shown in Figures 5, 6 and 7.

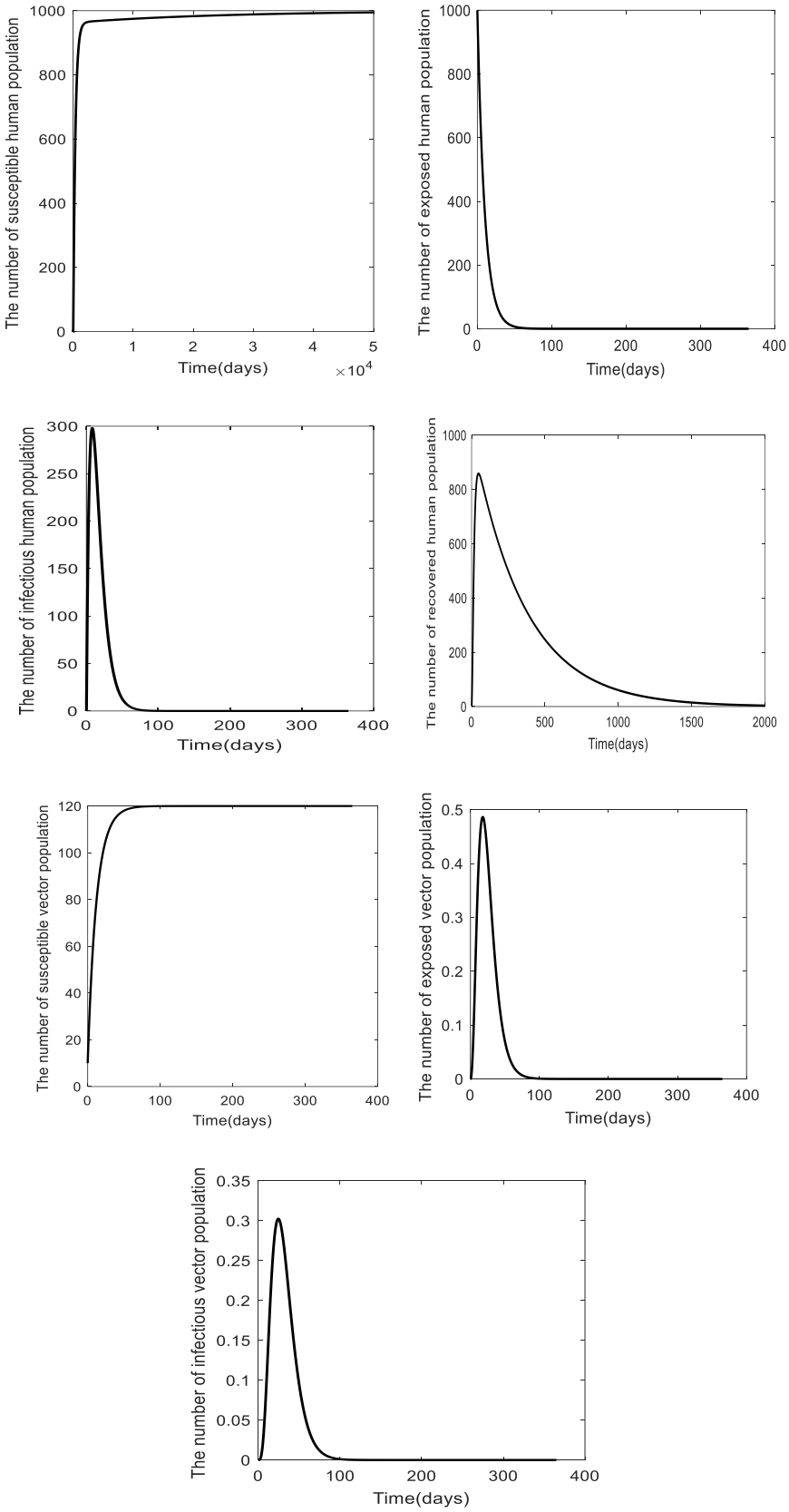


Figure 3. The time series solutions for the susceptible, exposed, infectious and recovered human populations and susceptible, exposed and infectious mosquito populations for the disease free state.

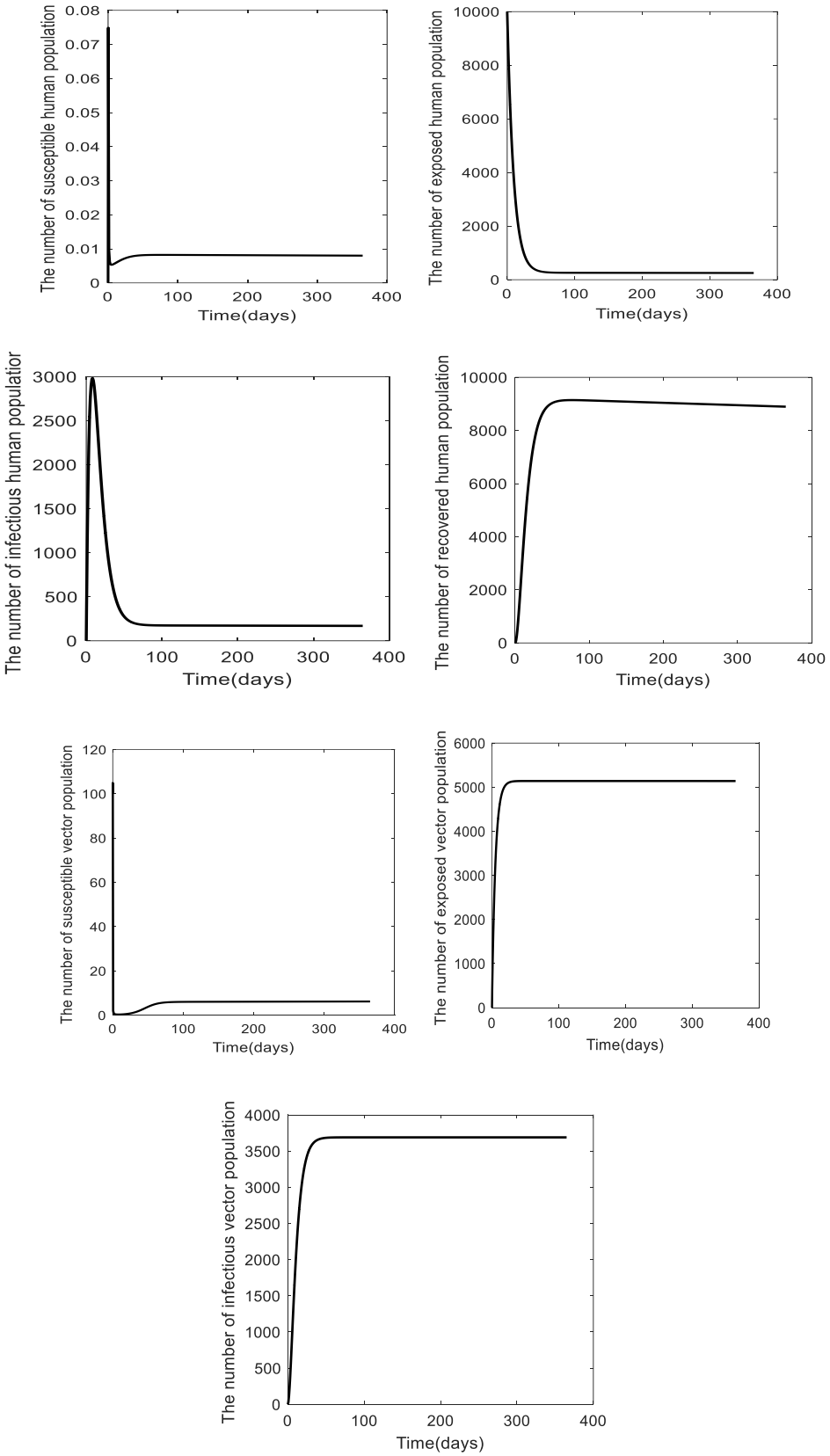


Figure 4. The time series solutions for the susceptible, exposed, infectious and recovered human populations and susceptible, exposed and infectious mosquito populations for the disease endemic state.

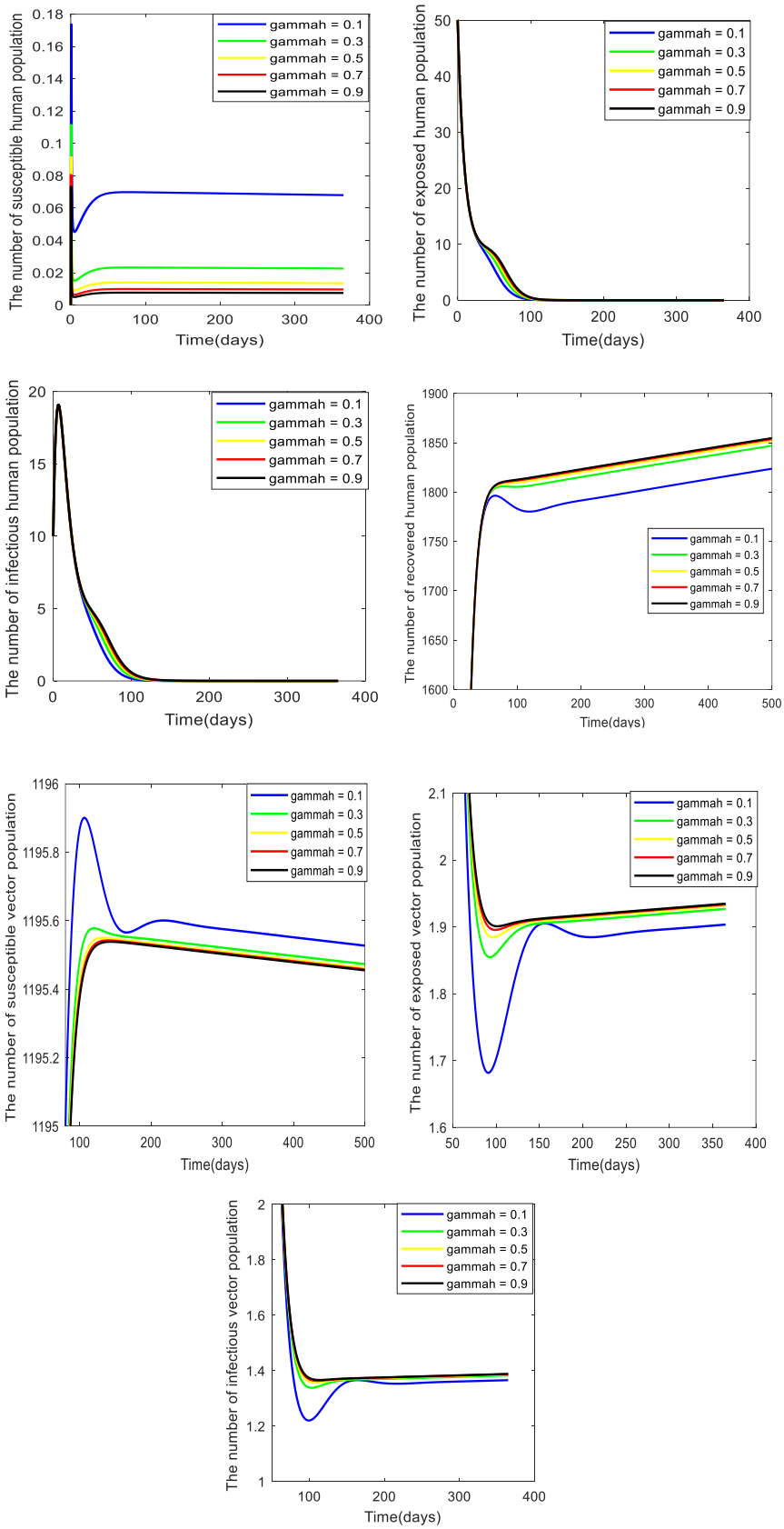


Figure 5. The time series solutions for the susceptible, exposed, infectious and recovered human populations and susceptible, exposed and infectious mosquito populations for the different transmission rates of dengue virus from human to vector population.

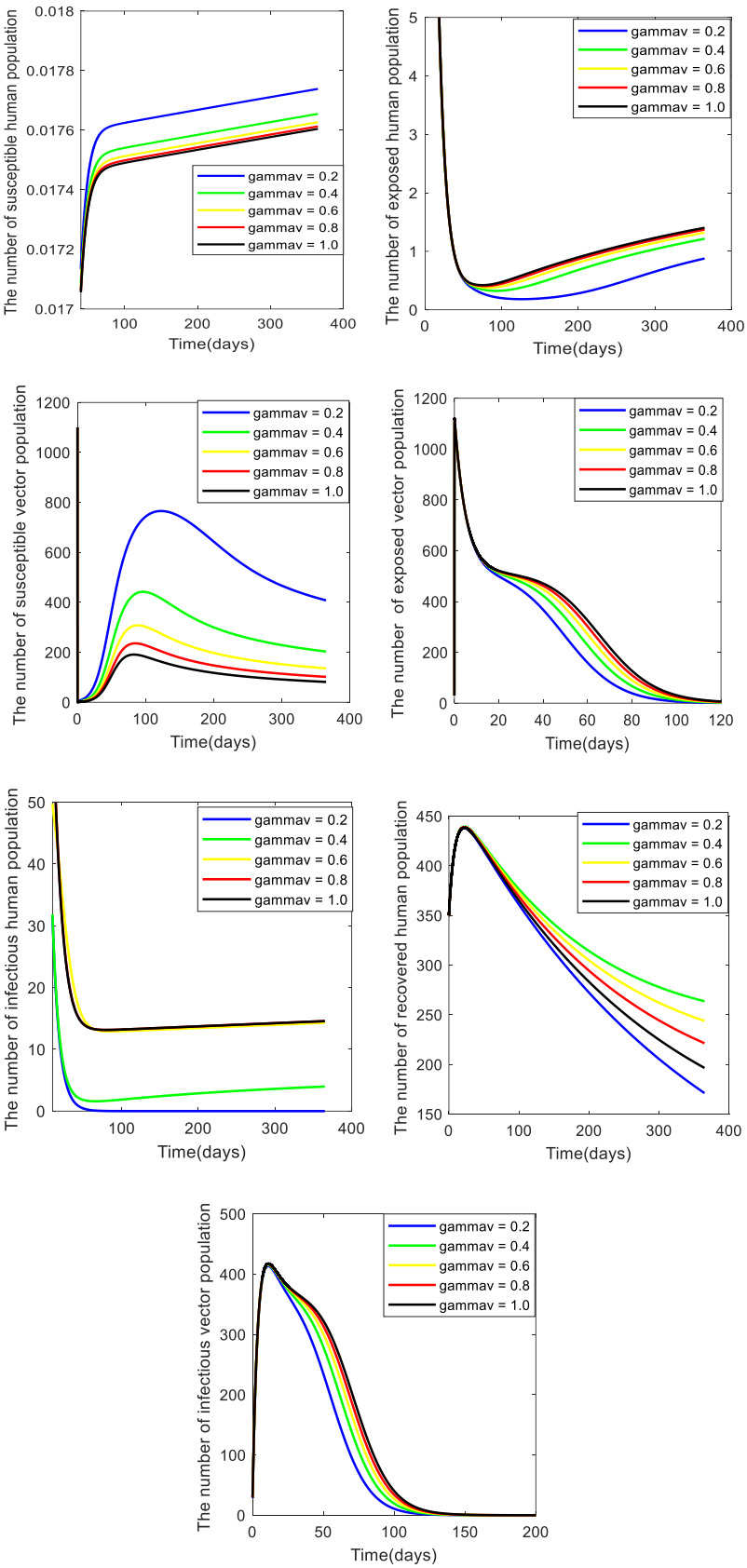


Figure 6. The time series solutions for the susceptible, exposed, infectious and recovered human populations and susceptible, exposed and infectious mosquito populations for the different transmission rates of dengue virus from vector to human population.

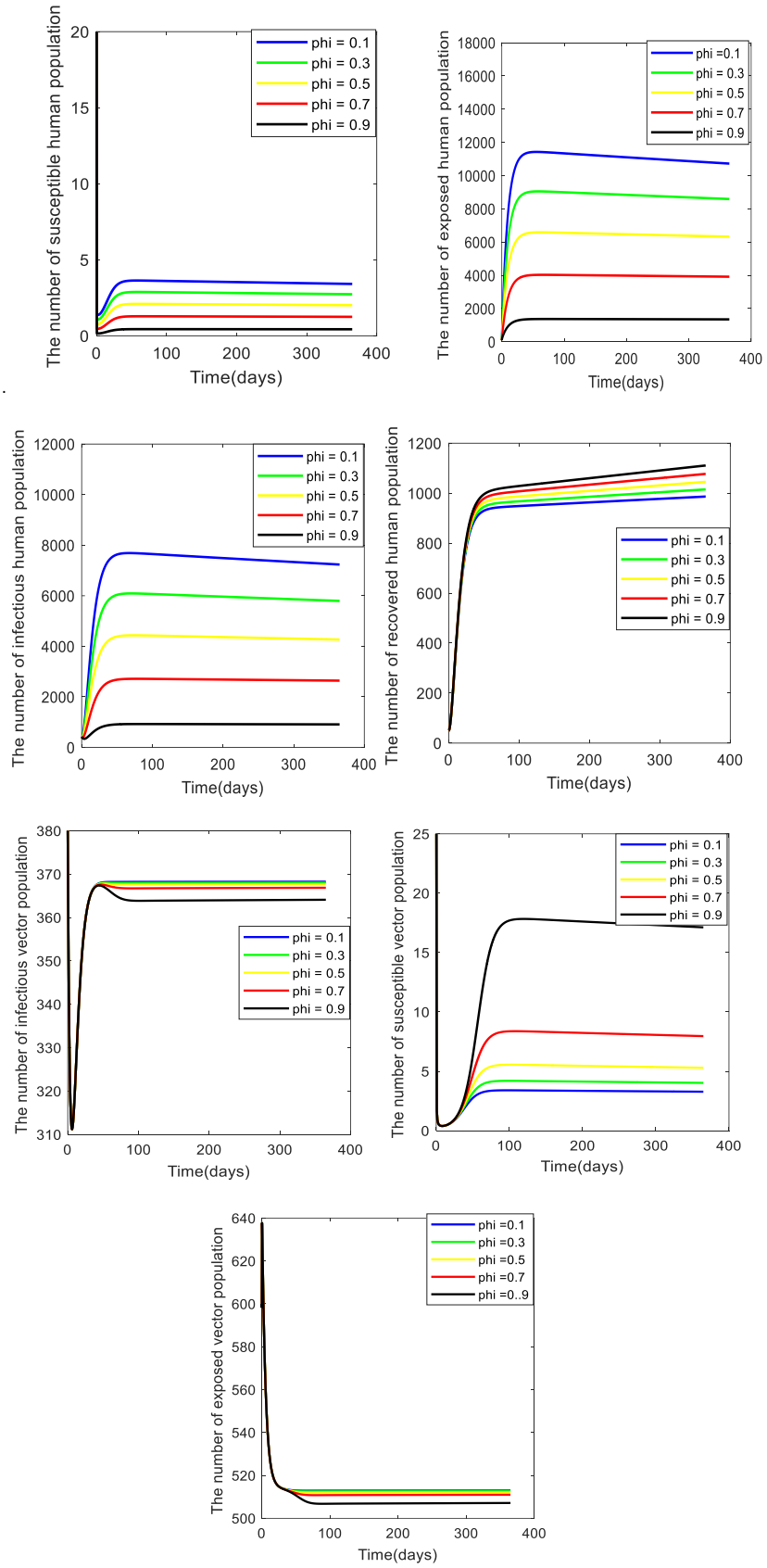


Figure 7. The time series solutions for the susceptible, exposed, infectious and recovered human populations and susceptible, exposed and infectious mosquito populations for the different vaccine efficiency.

Table 3. The value of each parameter used in the numerical simulations.

Parameters	Disease-free	Endemic state	References
N_h	1,000	10,000	assumed
γ_h	0.000025	0.025	assumed
β	1/(180)	1/(180)	[38–43]
α_h	1/10	1/10	[38–43]
r	1/7	1/7	[38–43]
μ_h	1/(70*365)	1/(70*365)	[38–43]
φ	0.5	0.5	assumed
d_h	1/(70*365)	1/(70*365)	[38–43]
d_d	1/(6*30)	1/(6*30)	[38–43]
A	10	100	assumed
γ_v	0.000005	0.05	assumed
d_v	1/12	1/12	[38–43]
d_k	1/14	1/14	assumed
α_v	1/9	1/9	[38–43]
R_0	0.61	610.69	-

3.5. Sensitivity analysis of parameters

In this section, the results of the sensitivity analysis of the basic reproductive number (R_0) is presented. This will help us to know which parameters will have the most significant impact on the numerical simulation results of the model. The sensitivity analysis allows us to determine the importance of each parameter to the spread of dengue fever and this helps public health authorities to focus on appropriate intervention strategies to prevent and control the spread of the disease, which can be calculated from the following formula [46]:

$$\Upsilon_{\rho}^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0},$$

where ρ is the epidemic parameter. For each parameter, we can determine the normalized forward sensitivity index of R_0, which is derived as follows:

$$\Upsilon_{N_h}^{R_0} = \Upsilon_{\gamma_h}^{R_0} = \Upsilon_{A}^{R_0} = \Upsilon_{\gamma_v}^{R_0} = \frac{1}{2},$$

$$\Upsilon_{\alpha_h}^{R_0} = \frac{d_h}{2(d_h+\alpha_h)},$$

$$\Upsilon_{\alpha_v}^{R_0} = \frac{d_v}{2(d_v+\alpha_v)},$$

$$\Upsilon_{d_h}^{R_0} = -\frac{d_h(d_d+2d_h+r+\alpha_h)}{2(d_d+d_h+r)(d_h+\alpha_h)},$$

$$\Upsilon_{r}^{R_0} = -\frac{r}{2(d_d+d_h+r)},$$

$$\Upsilon_{d_k}^{R_0} = -\frac{d_k}{2(d_k+d_v)},$$

$$\Upsilon_{d_d}^{R_0} = -\frac{d_d}{2(d_d+d_h+r)},$$

$$\Upsilon_{d_v}^{R_0} = -\frac{d_k(2d_v+\alpha_v)+d_v(3d_v+2\alpha_v)}{2(d_k+d_v)(d_v+\alpha_v)}.$$

The data in Table 3 show the parameters used for numerical simulations. The results of calculating the sensitivity of the basic reproductive number of each parameter are shown in Table 4.

Table 4. Sensitivity values of the basic reproduction numbers.

Parameter	Sensitivity index
$\boldsymbol{\gamma_h}$	0.5
$\boldsymbol{\alpha_h}$	0.000196
$\boldsymbol{A}$	0.5
$\boldsymbol{\gamma_v}$	0.5
$\boldsymbol{N_h}$	0.5
$\boldsymbol{\alpha_v}$	0.214286
$\boldsymbol{d_h}$	−0.000327
$\boldsymbol{r}$	−0.481157
$\boldsymbol{d_k}$	−0.230769
$\boldsymbol{d_d}$	−0.018712
$\boldsymbol{d_v}$	−0.983516

From Table 4, it can be seen that 6 of the 11 parameters have positive values: $\gamma_h, \alpha_h, A, \gamma_v, N_h$ and α_v. The negative parameters are $\alpha_v, d_h, r, d_k, d_d$ and d_v. From the model, it was suggested that dengue control was most likely to be achieved by decreasing the values of γ_h, A, γ_v and N_h.

4. Optimal control problem

So far, we have determined the dynamics of the transmission of dengue fever described by our dynamical model using a particular set of parameters. To reduce the transmission of dengue virus, we used the control functions such as u_1 and u_2, which can lead the values of the parameters to change. We define u_1 to represent the prevention from the dengue infection by using clothes, mosquito nets and window screens. The control function u_2 represents insecticide spraying.

$$\frac{dS_h}{dt} = d_h N_h - \big(1-u_1(t)\big) r_h S_h I_v + (1-\varphi)\beta R_h - d_h S_h, \tag{28}$$

$$\frac{dE_h}{dt} = \big(1-u_1(t)\big)\gamma_h S_h I_v - (\alpha_h + d_h) E_h, \tag{29}$$

$$\frac{dI_h}{dt} = \alpha_h E_h - (r + d_h + d_d) I_h, \tag{30}$$

$$\frac{dR_h}{dt} = r I_h - (1-\varphi)\beta R_h - d_h R_h, \tag{31}$$

$$\frac{dS_v}{dt} = A - \big(1 - u_1(t)\big)\gamma_v S_v I_h - d_v S_v - u_2(t) S_v, \tag{32}$$

$$\frac{dE_v}{dt} = \big(1 - u_1(t)\big)\gamma_v S_v I_h - (\alpha_v + d_v) E_v - u_2(t) E_v, \tag{33}$$

$$\frac{dI_v}{dt} = \alpha_v E_v - (d_v + d_k) I_v - u_2(t) I_v. \tag{34}$$

We used the optimal control problem by using Pontryagin's maximum principle [45–52] to reduce the number of infectious populations. We determined the form of the following objective function:

$$J(u_1(t), u_2(t)) = min \int_0^T \Big(A_1 I_h + A_2 I_v + \frac{1}{2} A_3 u_1^2(t) + \frac{1}{2} A_4 u_2^2(t) \Big). \tag{35}$$

The objective function defined in Eq (35) depends on the number I_h and I_v. We note that A_1, A_2, A_3 and A_4 are the weight constants. We solve the optimal control problem by Lagrangian and Hamiltonian approaches, then we get:

$$L(I_h, I_v, u_1(t), u_2(t)) = A_1 I_h + A_2 I_v + \frac{1}{2} A_3 u_1^2(t) + \frac{1}{2} A_4 u_2^2(t). \tag{36}$$

Theorem 3. For problem-solving guidelines with S_h, E_h, I_h, R_h, S_v, E_v and I_v and a suitable control $u^* = (u_1^*(t), u_2^*(t))$ for the initial problems (28)–(34) that minimize $J(u_1(t), u_2(t))$, there exists an adjoint variable $\lambda_i; i = 1,2,3,4,5,6,7$ under the control equations:

$$\frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial \psi}. \tag{37}$$

We define $\psi = (S_h, E_h, I_h, R_h, S_v, E_v, I_v)$ with the transversality condition given as $\lambda_i(t)$=0; $i = 1,2,3,4,5,6,7$ and

$$u^*{}_1 = \begin{cases} 0 & if \ \frac{(\lambda_2 - \lambda_1) r_h S_h I_v + (\lambda_6 - \lambda_5)\gamma_v S_v I_h}{A_3} \leq 0 \\ \frac{(\lambda_2 - \lambda_1) r_h S_h I_v + (\lambda_6 - \lambda_5)\gamma_v S_v I_h}{A_3} & if \ \frac{(\lambda_2 - \lambda_1) r_h S_h I_v + (\lambda_6 - \lambda_5)\gamma_v S_v I_h}{A_3} < u_1^{max} \\ u_1^{max} & if \ \frac{(\lambda_2 - \lambda_1) r_h S_h I_v + (\lambda_6 - \lambda_5)\gamma_v S_v I_h}{A_3} \geq u_1^{max} \end{cases} \tag{38}$$

$$u^*{}_2 = \begin{cases} 0 & if \ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} \leq 0 \\ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} & if \ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} < u_2^{max} \\ u_2^{max} & if \ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} \geq u_2^{max} \end{cases} \tag{39}$$

Proof. We define the Hamiltonian function as follows:

$$H = L(I_h, I_v, u_1, u_2) + \lambda_1 \frac{dS_h}{dt} + \lambda_2 \frac{dE_h}{dt} + \lambda_3 \frac{dI_h}{dt} + \lambda_4 \frac{dR_h}{dt} + \lambda_5 \frac{dS_v}{dt} + \lambda_6 \frac{dE_v}{dt} + \lambda_7 \frac{dI_v}{dt}$$

where

$$L(I_h, I_v, u_1(t), u_2(t)) = A_1 I_h + A_2 I_v + \frac{1}{2} A_3 u_1^2(t) + \frac{1}{2} A_4 u_2^2(t)$$

is the Lagrangian of the control problem. We have

$$H = A_1 I_h + A_2 I_v + \frac{1}{2}A_3 u_1^2 + \frac{1}{2}A_4 u_2^2$$
$$+\lambda_1\left[d_h N_h - \left(1-u_1(t)\right)r_h S_h I_v + (1-\varphi)\beta R_h - d_h S_h\right]$$
$$+\lambda_2\left[\left(1-u_1(t)\right)\gamma_h S_h I_v - (\alpha_h + d_h)E_h\right]$$
$$+\lambda_3[\alpha_h E_h - (r + d_h + d_d)I_h]$$
$$+\lambda_4[rI_h - (1-\varphi)\beta R_h - d_h R_h]$$
$$+\lambda_5\left[A - \left(1-u_1(t)\right)\gamma_v S_v I_h - d_v S_v - u_2(t)S_v\right]$$
$$+\lambda_6\left[\left(1-u_1(t)\right)\gamma_v S_v I_h - (\alpha_v + d_v)E_v - u_2(t)E_v\right]$$
$$+\lambda_7[\alpha_v E_v - (d_v + d_k)I_v - u_2(t)I_v]. \quad (40)$$

The adjoint functions can be defined as

$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial S_h} = \lambda_1(t)\left(\left(1-u^*{}_1(t)\right)r_h I_v + d_h\right) - \lambda_2(t)\left(\left(1-u_1{}^*(t)\right)\gamma_h I_v\right),$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial E_h} = \lambda_2(t)(\alpha_h + d_h) - \lambda_3(t)\alpha_h,$$

$$\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial I_h} = \lambda_3(t)(r + d_h + d_d) - \lambda_4(t)r + (\lambda_5(t) - \lambda_6(t))(1-u^*{}_1(t))\gamma_v S_v - A_1,$$

$$\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial R_h} = -\lambda_1(t)(1-\varphi)\beta + \lambda_4(t)\left((1-\varphi)\beta + d_h\right),$$

$$\frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial S_v} = \lambda_5(t)\left(\left(1-u^*{}_1(t)\right)\gamma_v I_h + d_v + u^*{}_2(t)\right) - \lambda_6(t)\left(1-u^*{}_1(t)\right)\gamma_v I_h,$$

$$\frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial E_v} = \lambda_6(t)(d_v + \alpha_v + u^*{}_2(t)) - \lambda_7(t)\alpha_v \ ,$$

$$\frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial I_v} = \left(\lambda_1(t) - \lambda_2(t)\right)\left(\left(1-u^*{}_1(t)\right)r_h S_h\right) + \lambda_7(t)\left(d_v + d_k + u^*{}_2(t)\right) - A_2.$$

The suitable controls $u^*{}_1(t)$ and $u^*{}_2(t)$ depends on $\frac{\partial H}{\partial u_j} = 0$ for all $j = 1,2$ at $u_j = u^*{}_j$.

Therefore,

$\frac{\partial H}{\partial u_1} = A_3 u_1 - (\lambda_2 - \lambda_1)r_h S_h I_v - (\lambda_6 - \lambda_5)\gamma_v S_v I_h$ then $u^*{}_1 = \frac{(\lambda_2-\lambda_1)r_h S_h I_v + (\lambda_6-\lambda_5)\gamma_v S_v I_h}{A_3}$,

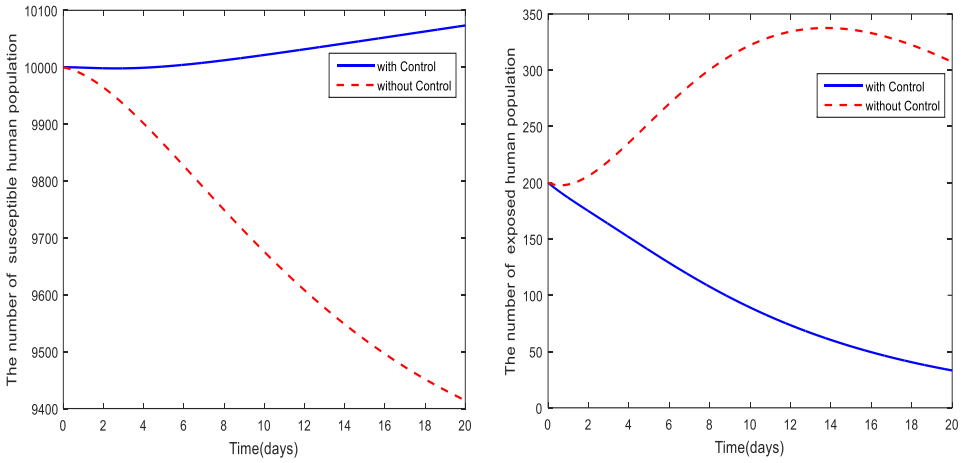
$\frac{\partial H}{\partial u_2} = A_4 u_2 - \lambda_5 S_v - \lambda_6 E_v - \lambda_7 I_v$ then $u^*{}_2 = \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4}$.

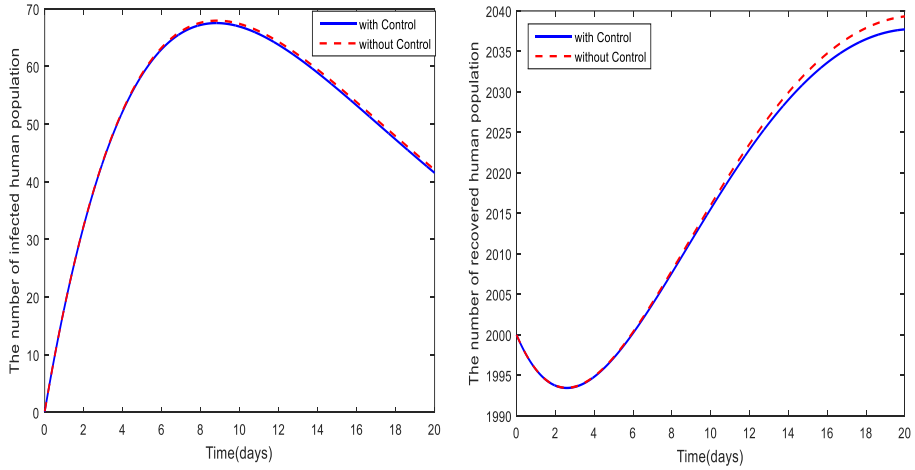
The optimal control function can be defined as follows:

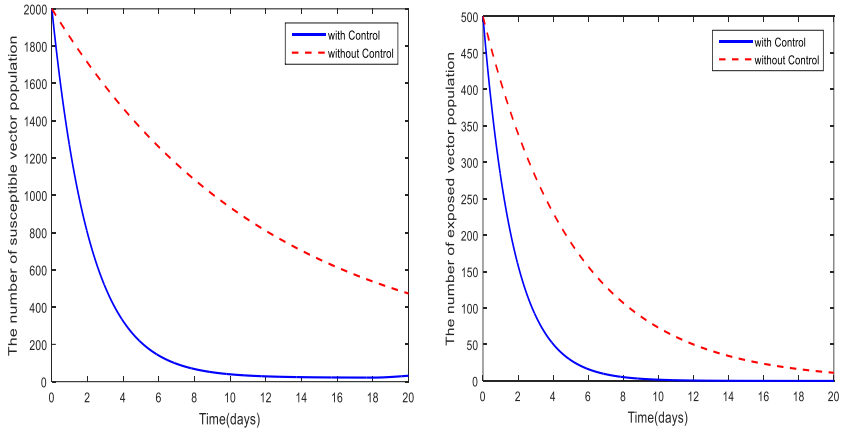
$$u^*_1 = \begin{cases} 0 & if \quad \dfrac{(\lambda_2-\lambda_1)r_h S_h I_v + (\lambda_6-\lambda_5)\gamma_v S_v I_h}{A_3} \leq 0 \\ \dfrac{(\lambda_2-\lambda_1)r_h S_h I_v + (\lambda_6-\lambda_5)\gamma_v S_v I_h}{A_3} & if \quad \dfrac{(\lambda_2-\lambda_1)r_h S_h I_v + (\lambda_6-\lambda_5)\gamma_v S_v I_h}{A_3} < u_1^{max} \\ u_1^{max} & if \quad \dfrac{(\lambda_2-\lambda_1)r_h S_h I_v + (\lambda_6-\lambda_5)\gamma_v S_v I_h}{A_3} \geq u_1^{max} \end{cases}$$

$$u^*_2 = \begin{cases} 0 & if \ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} \leq 0 \\ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} & if \ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} < u_2^{max} \\ u_2^{max} & if \ \frac{\lambda_5 S_v + \lambda_6 E_v + \lambda_7 I_v}{A_4} \geq u_2^{max} \end{cases}$$

We show the numerical analysis of the optimal control in Figures 8 and 9. The numerical solutions were given by Runge–Kutta forward–backward sweep method [46]. Time was supposed as 20 days. The control weighted values were $A_1 = 100$, $A_2 = 200$, $A_3 = 300$ and $A_4 = 400$. We can see that the cases with control converge to an equilibrium point faster than the cases without the optimal control.

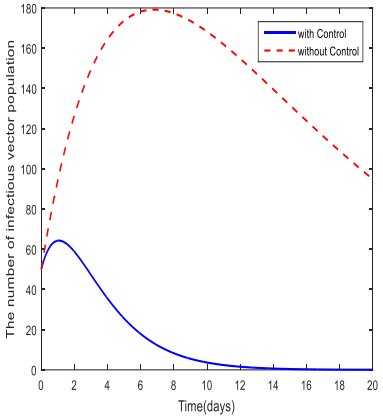




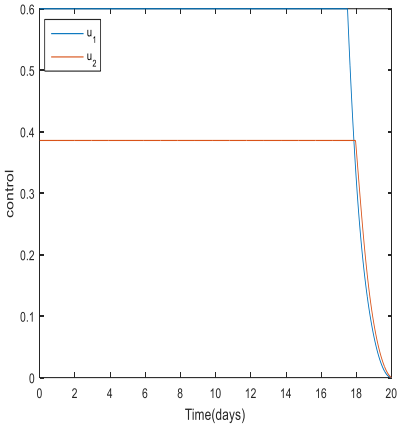


(e)

(f)

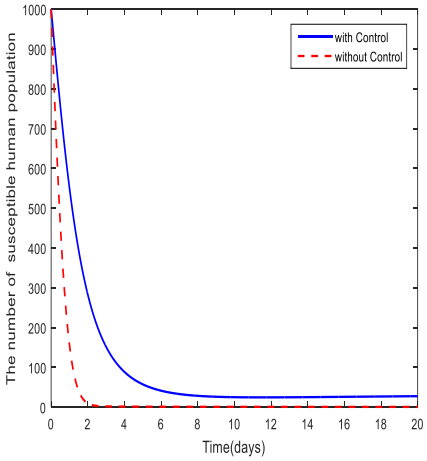


(g)

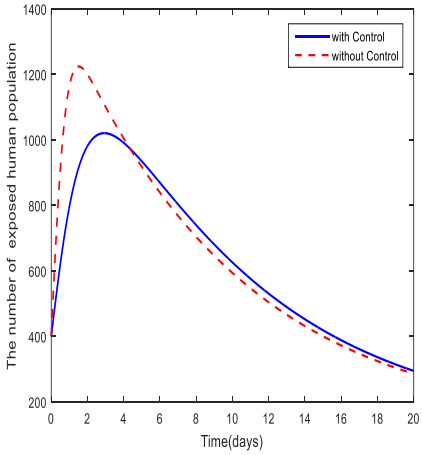


(h)

Figure 8. The comparison between cases with control and without control. (a) The time series solutions for the susceptible human, (b) the time series solutions for the exposed human, (c) the time series solutions for the infectious human, (d) the time series solutions for the recovered human populations, (e) the time series solutions for the susceptible mosquito populations, (f) the time series solutions for the exposed mosquito populations, (g) the time series solutions for the infectious mosquito populations for the disease free state and (h) the control effects $u_1(t)$ and $u_2(t)$.



(a)



(b)

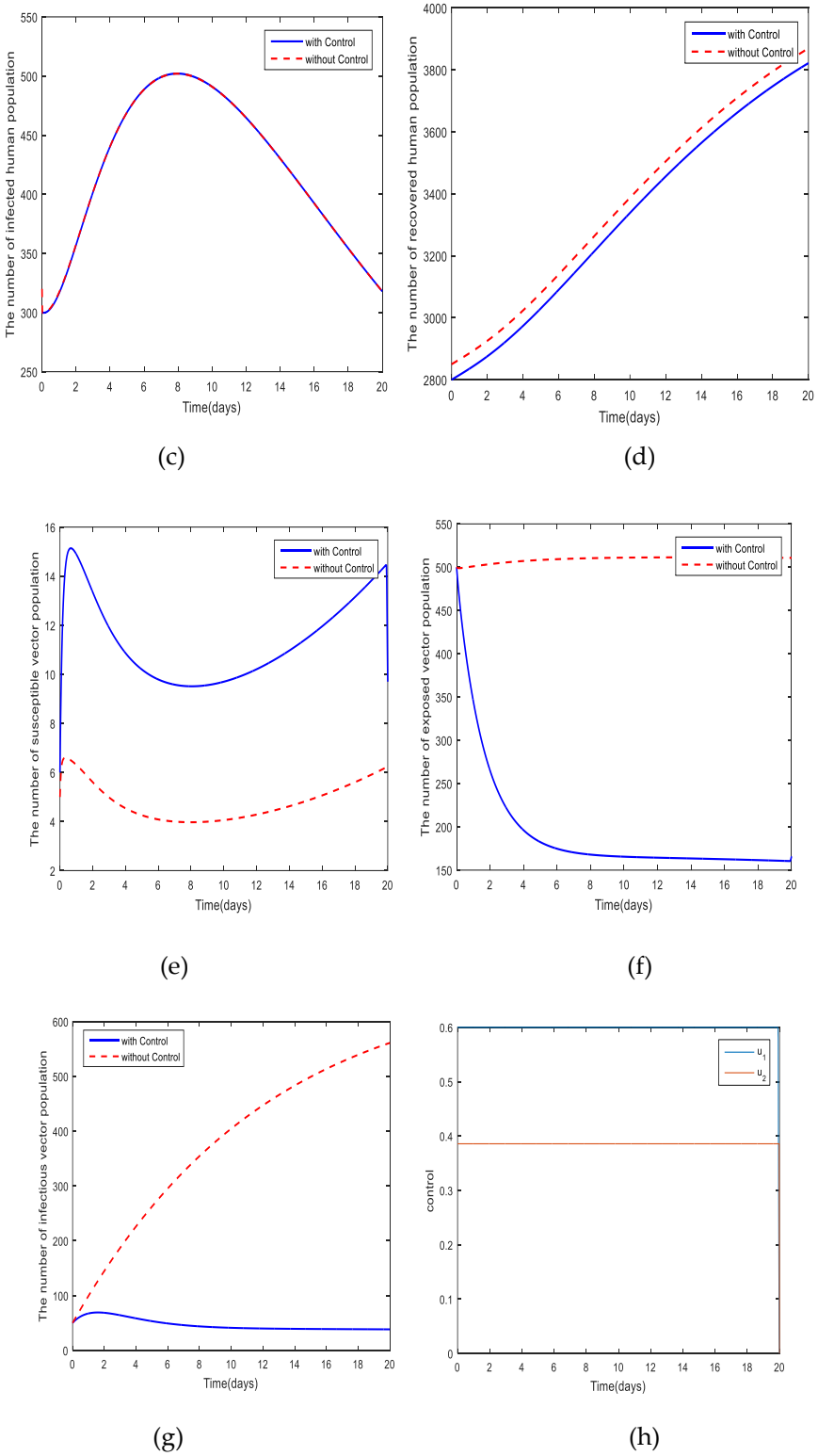


Figure 9. The comparison between cases with control and without control. (a) The time series solutions for the susceptible human, (b) the time series solutions for the exposed human, (c) the time series solutions for the infectious human, (d) the time series solutions for the recovered human populations, (e) the time series solutions for the susceptible mosquito populations, (f) the time series solutions for the exposed mosquito populations, (g) the time series solutions for the infectious mosquito populations for the endemic state and (h) the control effects $u_1(t)$ and $u_2(t)$.

5. Discussion and conclusions

The optimal control of the dengue virus when vaccination against this virus was carriered out was determined. The disease dynamics of the human population was modeled using the SEIR model, while the mosquito population was modeled with a SEI model. The models were then analyzed by using standard dynamical modeling. Disease free and endemic equilibrium points were found. The stability of equilibrium points was determined by the Lyapunov function. The basic reproduction number was found. If $R_0 < 1$, the disease-free state is stable. If $R_0 > 1$, the endemic state is stable. A sensitivity analysis of the basic reproduction number was also performed. It is found that for the developed models, the dengue control is likely to be achieved with the decrement of $\boldsymbol{\gamma_h}$ (the transmission rate of dengue virus from human to vector population), $\mu_{\boldsymbol{h}}$ (the birth and natural death rate of human population), $\boldsymbol{A}$ (the constant recruitment rate of vector population), $\boldsymbol{\gamma_v}$ (the transmission rate of dengue disease from vector to human population) and $\boldsymbol{N_h}$ (the total human population). Based on these findings, the optimal control problem was then formulated. The strategies for the control of the dengue infection centred on the prevention of dengue virus through the use of mosquito nets and window screens, as well as insecticides sprayings, rather than treatment measures such as medication and vaccination. Such control measures are more appropriate for the rural areas of Thailand, where it is more economical to use these strategies. Simulation results in Figures 8 and 9 show that the controlled response trajectories converge to the equilibrium point faster than the ones without the control. Hence the developed mathematical model, along with the designed control strategies could yield an important guideline for the Thai Ministry of Public Health, as they attempt to contain the outbreaks that happen in the rural areas, such as in the northeastern part of the country. Further research could consider the multiple patch counterparts of the developed model, to enable ways of constraining the dengue outbreaks optimally where there are movements from a higher populated rural area to a lower populated one.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflicts of interest

The authors declare that no conflict of interest exist in the publication of this paper.

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