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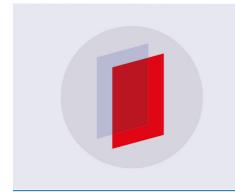
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### Optimal control for the transmission dynamics of malaria disease model

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Abstract. In this work a modified SEIR-SVEVIV model that described the dynamics transmission of malaria disease was proposed and analyzed. The standard method is used to analyze the behaviors of the proposed model. The results shown that there were two equilibrium points; disease free and endemic equilibrium point. The qualitative results are depended on a basic reproductive number  $(R_0)$ . We obtained the basic reproductive number by using the next generation method technique and finding the spectral radius. Routh-Hurwitz criteria is used for determining the stabilities of the model. If R<sub>0</sub><1, then the diseases free equilibrium point is local asymptotically stable: that is the disease will died out, but if  $R_0 > 1$ , then the endemic equilibrium is local asymptotically stable. After that the SEIR-SVEVIV model is modified from the first model by adding the optimal control functions that includes two times - dependent control functions with one minimizing the contract between the susceptible human and the infected vector and the other, minimizing the population of the infected human. The result from the numerical solutions of the models are shown and compared for supporting the analytic results.

#### 1. Introduction

Malaria is caused by parasites that are transmitted to people through the bites of infected female mosquitoes. P. falciparum is the most deadly malaria parasite and the most prevalent in Africa, where malaria cases and deaths are heavily concentrated. The first symptoms of malaria – fever, headache, chills and vomiting – usually appear between 10 and 15 days after the mosquito bite. Without prompt treatment, P. falciparum malaria can progress to severe illness and death. WHO recommends a multipronged strategy to prevent, control and eliminate malaria. Key interventions include: the use of insecticide-treated mosquito nets and indoor residual spraying, diagnostic testing, and treatment of confirmed cases with effective anti-malarial medicines. In recent years, these measures have dramatically lowered the malaria burden in many settings. Malaria transmission continues in many countries around the world however, and causes hundreds of thousands of deaths each year (WHO) [1]. Malaria situation in Thailand week 1-43 on 1 Sep 2017, from 1 Jan 2017 to 1 Sep 2017. No. of Thai malaria cases is about 7,902 and No. of foreigner malaria cases is 2,976.By total that decrease 35.31% from the year 2016 [2]. Mathematical models have become an important tool for understanding the spread and control of disease because of this disease is caused by virus, therefore no drug can cure this disease specificially [3-7]. This paper is organized respectively as follows. In section 2, we present an SEIR-SVEVIV model for malaria. The standard method is used to analyze the behaviors of the proposed model. The analysis of optimization problem is presented in section 3. In section4, we give a

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numerical appropriate method and the simulation corresponding results. Finally, the conclusions are summarized in section 5.

#### 2. Material and method

#### 2.1 Material

In this paper, we study the transmission of malaria diseases through mathematical modell. By using the standard method to analyze the behaviours of the model which was adopted from [8]. The population consist of two groups: human population N and population vector  $N_y$ . Human population be divided into four disease-state compartments: susceptible individuals (S), people who can catch the disease; exposed individuals (E), people whose body is a host for the infectious agent but are not yet able to transmit the disease; infectious individuals (I), people who have the disease and can transmit the disease; recovered individuals (R), people who have recovered from the disease. Population vector or mosquitoes N<sub>y</sub> are divided into three groups of mosquitoes: mosquitoes population  $S_{\nu}$ , the exposed mosquitoes population  $E_{\nu}$  and the infected mosquitoes population  $I_{\nu}$ . In this study, we assumed that there are numbers of people in the populations that have already infected by the virus while others have not. It is also assumed that the transmission of the virus continues in the population but number of mosquitoes as the vector is constant. People and mosquitoes are categorized in one group at a time. Then we obtained the transmission model as shown by a system of ordinary differential equations as follows.

Human Population;

$$\frac{dS}{dt} = A - \left(\frac{b\beta SI_{\upsilon}}{1 + \upsilon I_{\upsilon}}\right) - \mu S + \varphi R \tag{1}$$

$$\frac{dE}{dt} = \left(\frac{b\beta SI_{\nu}}{1 + \nu I_{\nu}}\right) - (\mu + \alpha)E \tag{2}$$

$$\frac{dI}{dt} = \alpha E - (\mu + \gamma + \delta)I \tag{3}$$

$$\frac{dR}{dt} = \gamma vI - (\mu + \varphi)R \tag{4}$$

Vector Population;

$$\frac{dS_{_{\boldsymbol{v}}}}{dt} = B - (\frac{b\beta_{_{\boldsymbol{v}}}S_{_{\boldsymbol{v}}}I}{1+\upsilon_{_{\boldsymbol{v}}}I}) + \mu_{_{\boldsymbol{v}}}S_{_{\boldsymbol{v}}} \tag{5}$$

$$\frac{dE_{v}}{dt} = \left(\frac{b\beta_{v}S_{v}I}{1 + \upsilon_{v}I}\right) - (\mu_{v} + \alpha_{v})E_{v} \tag{6}$$

$$\frac{dI_{v}}{dt} = \alpha_{v}E_{v} - (\mu_{v} + \delta_{v})I_{v}$$
 (7)

Where; 
$$S + E + I + R = N = \frac{A}{\mu}$$
 and  $S_v + E_v + I_v = N_v = \frac{B}{\mu_v}$ ,

S(t) is the susceptible human population at time t

E(t) is the exposed human population at time t

I(t) is the infected human population at time t

R(t) is the recovered human population at time t

N is the total number of human population,

S<sub>o</sub>(t) is the susceptible mosquitoes population at time t

 $E_{\nu}(t)$  is the exposed mosquitoes population at time t

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 $I_{v}(t)$  is the infected mosquitoes population at time t

N<sub>v</sub> is the total number of mosquitoes population,

b is average bite of mosquitoes that potentially infected

A is the constant recruitment rate of the human,

B is the constant recruitment rate of the mosquito,

 $\mu$  is the death rate of human population,

 $\mu_{\rm p}$  is the death rate of mosquitoes population,

 $\beta$  is the probability that a bite by an infectious mosquito results in transmission of disease to human

 $\beta_{\nu}$  Probability that a bite results in transmission of parasite to a susceptible mosquito

 $\alpha$  is the rate of progression of humans from the exposed state to the infectious state

 $\alpha_{_{V}}$  is the rate of progression of mosquitoes from the exposed state to the infectious state

 $\gamma$  is the recovery rate for humans from the infectious state to the recovered state

 $\delta$  is the disease-induced death rate of human

 $\delta_{v}$  is the disease-induced death rate of mosquito

 $\varphi$  is the rate of loss of immunity in humans

υ is an antibody produced by human in response to the incidence of infection caused by mosquito

 $v_{\rm v}$  is an antibody produced by mosquito in response to the incidence of infection caused by human

u is the contract rate between the infectious mosquito and susceptible human

v is the rate at which infectious human are treated at each time period

#### 2.1.1 Basic properties of the model

Positivity and Boundedness of the Solution

**Theorem 1.** If the initial data  $S \ge 0$ ,  $E \ge 0$ ,  $I \ge 0$ ,  $R \ge 0$  and  $S_v \ge 0$ ,  $E_v \ge 0$ ,  $I_v \ge 0$  then the solution  $(S, E, I, R, S_v, E_v, I_v)$  of the malaria control model (1) are non-negative for all t > 0. Therefore,

$$\lim_{t \to \inf} \sup N(t) \leq \frac{A}{\mu}, \lim_{t \to \infty} \sup N_{v}(t) \leq \frac{B}{\mu_{v}}, \text{ Where } S + E + I + R = N = \frac{A}{\mu}, S_{v} + E_{v} + I_{v} = N_{v} = \frac{B}{\mu_{v}}$$

The Invariant Region

**Theorem 2.** The region  $\Omega = \Omega_h \cup \Omega_v \subset R^4 \times R^3$  is positively invariant for the model with non-negative initial condition in  $R^7$ , where

$$\Omega_h = \{ (S, E, I, R) \in R^4 \mid S + E + I + R \le \frac{A}{\mu} \}, \Omega_v = \{ (S_v, E_v, I_v) \in R^3 \mid S_v + E_v + I_v \le \frac{B}{\mu_v} \}.$$

The equilibrium points for  $(S^*,E^*,I^*,R^*,S^*_{\nu},E^*_{\nu},I^*_{\nu})$  are found by setting the right hand side of each equations (1-7) equal to zero. We obtained two equilibrium points as follows;

$$S = \frac{A + \phi R}{(\mu + \frac{b\beta I_{v}}{1 + \nu I})}, E = \frac{(\frac{b\beta SI_{v}}{1 + \nu I_{v}})}{(\mu + \alpha)}, I = \frac{\alpha E}{(\mu + \gamma + \delta)}, R = \frac{\gamma I}{\mu + \phi},$$

$$S_{v} = \frac{B}{\frac{b\beta_{v}I}{1+\nu_{v}I} + \mu_{v}}, E_{v} = \frac{\frac{b\beta_{v}S_{v}I}{1+\nu_{v}I}}{\alpha_{v} + \mu_{v}}, I_{v} = \frac{(\alpha_{v}E_{v})}{\mu_{v} + \delta_{v}}$$

#### 2.1.1.1 Disease Free Equilibrium Point $(E_0)$ :

In the absence of the disease in the community, there are I=0 and  $I_{\nu}=0$ , we obtained  $E_0(S,E,I,R,S_{\nu},E_{\nu},I_{\nu})$  where

$$S = \frac{A}{\mu}, E = 0, I = 0, R = 0, S_v = \frac{B}{\mu_v}, E_v = 0, I_v = 0$$

#### 2.1.1.2 Endemic Equilibrium Point (E<sub>1</sub>):

In case the disease is presented in the community, I>0 and  $I_{\upsilon}>0$ , we obtained,  $E_{\iota}(S^{*},E^{*},I^{*},R^{*},S^{*}_{\upsilon},E^{*}_{\upsilon},I^{*}_{\upsilon})$  where;

$$S^{*} = \frac{A + \phi R^{*}}{(\mu + \frac{b\beta I_{v}^{*}}{1 + vI_{v}^{*}})}, E^{*} = \frac{(\frac{b\beta S^{*}I_{v}^{*}}{1 + vI_{v}^{*}})}{(\mu + \alpha)}, I^{*} = \frac{\alpha E^{*}}{(\mu + \gamma + \delta)}, R^{*} = \frac{\gamma I^{*}}{\mu + \phi}, S_{v}^{*} = \frac{B}{\frac{b\beta_{v}I^{*}}{1 + v_{v}I^{*}} + \mu_{v}}, E_{v}^{*} = \frac{\frac{b\beta_{v}S_{v}^{*}I^{*}}{1 + v_{v}I^{*}}}{\alpha_{v} + \mu_{v}}, I_{v}^{*} = \frac{(\alpha_{v}E_{v}^{*})}{\mu_{v} + \delta_{v}}$$

#### 2.1.2 Basic Reproductive Number (R<sub>0</sub>)

We obtained a basic reproductive number by using the next generation method [9]. By rewriting the equations (1)-(7) in matrix form;

$$\frac{dX}{dt} = F(X) - V(X) \tag{16}$$

Where F(X) is the non-negative matrix of new infection terms and V(X) is the non-singular matrix of remaining transfer terms.

And setting;

$$F = \left[\frac{\partial F_{i}(E_{0})}{\partial X_{i}}\right], V = \left[\frac{\partial V_{i}(E_{0})}{\partial X_{i}}\right]$$
(17)

for all i, j = 1, 2, 3, 4, 5, 6, 7 be the Jacobean matrix of F(X) and V(X) at  $E_0$ . The basic reproductive number  $(R_0)$  is the number of secondary case generate by a primary infectious case (Van den Driessche and Watmough, 2002). It can be evaluated through the formula;

$$\rho(FV^{-1}) \tag{18}$$

Where  $FV^{-1}$  is called the next generation matrix and  $\rho(FV^{-1})$  is the spectral radius (largest eigenvalues) of  $FV^{-1}$ . Then we get the reproduction number  $R_0$  where,

$$R_0 = \frac{(b^2 \beta \alpha \alpha_v AB)}{\mu(\mu + \alpha)(\gamma + \delta + \mu)(\delta_v + \mu_v)(\alpha_v + \mu_v)}$$
(19)

Finally, Routh-Hurwitz criteria is used for determining the stabilities of the model. If  $R_0 < 1$ , then the disease free equilibrium point is local asymptotically stable: that is the disease will died out, but if  $R_0 > 1$ , then the endemic equilibrium is local asymptotically stable. In this paper, we use optimal control this method as part of control measures for malaria disease. From the system of equations (1-

7), we include two controls u and v that represent, respectively, the effort rate that reduces the contract between the infectious vector and the susceptible individuals and the rate at with infectious human are treated at each time period. The mathematical system with controls is given by the nonlinear differential equations subject to non-negative initial conditions as the following;

$$\frac{dS}{dt} = A - (\frac{ub\beta SI_{v}}{1 + \nu I_{v}}) - \mu S + \varphi R \tag{1}$$

$$\frac{dE}{dt} = \left(\frac{ub\beta SI_{v}}{1 + vI_{v}}\right) - (\mu + \alpha)E \tag{2}$$

$$\frac{dI}{dt} = \alpha E - (\mu + \gamma + \delta + v)I \tag{3}$$

$$\frac{dR}{dt} = \gamma v I - (\mu + \varphi) R \tag{4}$$

$$\frac{dS_{v}}{dt} = B - (\frac{b\beta_{v}S_{v}I}{1 + v_{v}I}) + \mu_{v}S_{v}$$
 (5)

$$\frac{dE_{v}}{dt} = \left(\frac{b\beta_{v}S_{v}I}{1 + v_{v}I}\right) - (\mu_{v} + \alpha_{v})E_{v}$$
 (6)

$$\frac{dI_{v}}{dt} = \alpha_{v} E_{v} - (\mu_{v} + \delta_{v}) I_{v}$$
 (7)

Where, S(0) > 0,  $E(0) \ge 0$ ,  $I(0) \ge 0$ ,  $R(0) \ge 0$ ,  $S_v(0) > 0$ ,  $E_v(0) \ge 0$  and  $I_v(0) \ge 0$ 

#### 2.2 method

2.2.1 Optimal control for the transmission dynamics of malaria disease model

In this section we use the optimal control theory to analyze the behavior of the system of equations (20). The objective one is to minimize the contract between susceptible human and the infected vector and the other is to minimize the population of the infected human. Mathematically, for a fixed terminal time  $t_f$ , the problem is to minimize the objective functional;

$$J(u,v) = \int_{0}^{t_{f}} \left[ S(t) + I(t) + \frac{B_{1}}{2}u^{2}(t) + \frac{B_{2}}{2}v^{2}(t) \right] dt$$
 (21)

The parameter  $B_1 \ge 0$  and  $B_2 \ge 0$  denote weights that balance the size of the terms for a fixed terminal time  $t_f$ . Hence we are interested in finding an optimal control pair  $u^*$  and  $v^*$ , such that:

$$J(u^*, v^*) = \min \{J(u, v): (u, v) \varepsilon U\}$$
(22)

Where,  $U = \{(u, v) \mid 0 < u(t) < 1, 0 < v(t) < 1, t \in [0, t_f], \text{ wand } v \text{ are Lebesgue measurable}\}$ 

Next, applying the Pontryagin's Maximum Principle (Kirschner et al.,1997),we derive necessary conditions for our optimal control and corresponding state variables, including the two control functions. Therefore we have seven corresponding adjoint variables where  $\lambda_1$  corresponds to S,  $\lambda_2$  corresponds to S,  $\lambda_3$  corresponds to S,  $\lambda_4$  corresponds to S, and S, S, are corresponds to S, and S, and S, and S, are corresponds to S, and S, are corresponds to S,

The Hamiltonian equation is formed by allowing each of the adjoint variables correspond to each of the state variables accordingly and combining the result with the objective functional as below:

$$H = S(t) + I(t) + \frac{B_1}{2}u^2(t) + \frac{B_2}{2}v^2(t) + \sum_{i=1}^{7} \lambda_i f_i$$
 (23)

Where  $f_i$  is the right hand side of the differential equation of the  $i^{th}$  state variables.

The adjoint equations are formed by taking the derivative of the Hamiltonian with respect to each of the state variables as follow; By applying the Pontryagin's maximum principle [10] and the existence result of optimal control from [11] we obtain the following theorem:

**Theorem** 3 There exists an optimal control  $(u^*, v^*) \in U$ , and corresponding solution  $S^*, E^*, I^*, R^*, S^*_v, E^*_v$  and  $I^*_v$  that minimize J(u, v) over U. And there exists adjoint functions  $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$  and  $\lambda_7$  verifying;

$$\lambda_1' = -1 + \lambda_1 \left(\mu + \frac{ub\beta I_v}{1 + vI_v}\right) - \lambda_2 \left(\frac{ub\beta I_v}{1 + vI_v}\right), \lambda_2' = \lambda_2 \left(\mu + \alpha\right) - \lambda_3 \alpha,$$

$$\begin{split} \lambda_{3}' &= \lambda_{3} \left( \mu + \gamma + \delta + v \right) - \lambda_{4} \gamma v + \lambda_{5} \left( \frac{(1 + v_{v}I)(b\beta_{v}S_{v}) + b\beta_{v}S_{v}Iv_{v}}{(1 + v_{v}I)^{2}} \right) \\ &- \lambda_{6} \left( \frac{(1 + v_{v}I)(b\beta_{v}S_{v}) - b\beta_{v}S_{v}Iv_{v}}{(1 + v_{v}I)^{2}} \right) + \lambda_{7} (\mu_{v} + \delta_{v}) - 1 \end{split}$$

$$\lambda_4' = -\lambda_1 \varphi + \lambda_4 (\mu + \varphi) , \lambda_5' = \lambda_5 (\frac{b\beta_{\nu}I}{1 + \nu I} + \mu_{\nu}) - \lambda_6 (\frac{b\beta_{\nu}I}{1 + \nu I}),$$

$$\lambda_{6}' = \lambda_{6}(\alpha_{v} + \mu_{v}) - \lambda_{7}\alpha_{v}, \ \lambda_{7}' = (\lambda_{1} - \lambda_{2})(\frac{(1 + vI_{v})(ub\beta S) - (ub\beta SI_{v}v)}{(1 + vI_{v})^{2}}) + \lambda_{7}(\mu_{v} + \delta_{v})$$

With the transversality conditions;  $\lambda_1(t_f) = \lambda_2(t_f) = \lambda_3(t_f) = \lambda_4(t_f) = \lambda_5(t_f) = \lambda_5(t_f) = \lambda_7(t_f) = 0$ , and the optimize control  $(u^*, v^*)$  is given by

$$u^* = \min \left\{ 1, \max \left\{ 0, \frac{((\lambda_1 - \lambda_2)(b\beta I_v)S^*)}{B_1(1 + vI_v)} \right\} \right\}$$
$$v^* = \min \left\{ 1, \max \left\{ 0, \frac{(\lambda_3 - \lambda_4)(I^* - \gamma)}{B_2} \right\} \right\}.$$

#### Proof.

The existence of optimal control can be proved by using the results from [3]. The adjoint equations and transversality conditions can be obtained by using Pontryagin's Maximum Principle such that,

$$\begin{split} \lambda_1' &= -1 + \lambda_1 (\mu + \frac{ub\beta I_v}{1 + vI_v}) - \lambda_2 (\frac{ub\beta I_v}{1 + vI_v}) \ , \ \lambda_2' = -\frac{\partial H}{\partial E} = \lambda_2 \left(\mu + \alpha\right) - \lambda_3 \alpha \ , \\ \lambda_3' &= \lambda_3 \left(\mu + \gamma + \delta + v\right) - \lambda_4 \gamma v + \lambda_5 (\frac{(1 + v_v I)(b\beta_v S_v) + b\beta_v S_v I v_v}{(1 + v_v I)^2}) - \lambda_6 (\frac{(1 + v_v I)(b\beta_v S_v) - b\beta_v S_v I v_v}{(1 + v_v I)^2}) \\ &+ \lambda_7 (\mu_v + \delta_v) - 1 \\ \lambda_4' &= -\frac{\partial H}{\partial R} - \lambda_1 \varphi + \lambda_4 (\mu + \varphi) \ , \ \lambda_5' = -\frac{\partial H}{\partial E_v} = \lambda_5 (\frac{b\beta_v I}{1 + v_v I} + \mu_v) - \lambda_6 (\frac{b\beta_v I}{1 + v_v I}) \ , \\ \lambda_6' &= -\frac{\partial H}{\partial I_v} = \lambda_6 (\alpha_v + \mu_v) - \lambda_7 \alpha_v \ , \ \lambda_7' = (\lambda_1 - \lambda_2) (\frac{(1 + vI_v)(ub\beta S) - (ub\beta SI_v v)}{(1 + vI_v)^2}) + \lambda_7 (\mu_v + \delta_v) \ , \end{split}$$

The optimal control pair  $(u^*, v^*)$  are obtained by finding the derivative of the Hamiltonian equation with respect to the control variables, equating to zero, and solving equation. Then we get;

$$\frac{\partial H}{\partial u} = B_1 u(t) - \frac{\lambda_1 b \beta S I_v}{1 + v I_v} + \frac{\lambda_2 b \beta S I_v}{1 + v I_v} = 0.$$

Then the optimal value for 
$$u$$
 is;  $u^* = \frac{(\lambda_1 - \lambda_2)(b\beta I_v^*S^*)}{B_1(1 + vI_v^*)}$ . And  $\frac{\partial H}{\partial v} = B_2 v(t) - \lambda_3 I + \lambda_4 \gamma I = 0$ ,

Hence the optimal value for v is;  $v^* = \frac{(\lambda_3 - \lambda_4)(I^*)(1 - \gamma)}{R}$ 

By the bounds in U of the control, the optimal control pair  $(u^*, v^*)$  is given by

$$u^* = \min\left\{1, \max\left\{0, \frac{(\lambda_1 - \lambda_2)(b\beta I_v^* S^*)}{B_1(1 + v I_v^*)}\right\}\right\}, v^* = \min\left\{1, \max\left\{0, \frac{(\lambda_3 - \lambda_4)(I^*)(1 - \gamma)}{B_2}\right\}\right\}.$$
(24)

For supporting analytic results we need to resolve the optimal control model numerically.

#### 3. Results

In this section we present the numerical simulations obtained by solving numerically from the following optimality system;

$$\frac{dS}{dt} = A - \left(\frac{u^*b\beta SI_v}{1 + \nu I_v}\right) - \mu S + \omega R \qquad (1), \qquad \frac{dS_v}{dt} = B - \left(\frac{b\beta_v S_v I}{1 + \nu_v I}\right) + \mu_v S_v \qquad (5)$$

$$\frac{dE}{dt} = \left(\frac{u^*b\beta SI_v}{1 + \nu I_v}\right) - (\mu + \alpha)E \qquad (2), \qquad \frac{dE_v}{dt} = \left(\frac{b\beta_v S_v I}{1 + \nu_v I}\right) - (\mu_v + \alpha_v)E_v \qquad (6)$$

$$\frac{dI}{dt} = \alpha E - (\mu + \gamma + \delta + v^*)I \qquad (3), \qquad \frac{dI_v}{dt} = \alpha_v E_v - (\mu_v + \delta_v)I_v \qquad (7)$$

$$\frac{dR}{dt} = \gamma v^* I - (\mu + \varphi) R \tag{4},$$

$$\begin{split} \lambda_{1}' &= -\frac{\partial H}{\partial S} - 1 + \lambda_{1} (\mu + \frac{ub\beta I_{v}}{1 + v I_{v}}) - \lambda_{2} (\frac{ub\beta I_{v}}{1 + v I_{v}}) \;\;,\; \lambda_{2}' = -\frac{\partial H}{\partial E} = \lambda_{2} \left(\mu + \mu\right) - \lambda_{3} \alpha \;\;,\\ \lambda_{3}' &= \lambda_{3} \left(\mu + \gamma + \delta + v\right) - \lambda_{4} \gamma v + \lambda_{5} (\frac{(1 + v_{v}I)(b\beta_{v}S_{v}) + b\beta_{v}S_{v}Iv_{v}}{(1 + v_{v}I)^{2}}) \\ - \lambda_{6} (\frac{(1 + v_{v}I)(b\beta_{v}S_{v}) - b\beta_{v}S_{v}Iv_{v}}{(1 + v_{v}I)^{2}}) + \lambda_{7} (\mu_{v} + \delta_{v}) - 1,\\ \lambda_{4}' &= -\frac{\partial H}{\partial R} - \lambda_{1} \varphi + \lambda_{4} (\mu + \varphi) \;\;, \lambda_{5}' = -\frac{\partial H}{\partial E_{v}} = \lambda_{5} (\frac{b\beta_{v}I}{1 + v_{v}I} + \mu_{v}) - \lambda_{6} (\frac{b\beta_{v}I}{1 + v_{v}I}),\\ \lambda_{6}' &= -\frac{\partial H}{\partial I_{v}} = \lambda_{6} (\alpha_{v} + \mu_{v}) - \lambda_{7} \alpha_{v} \;\;, \lambda_{7}' = (\lambda_{1} - \lambda_{2}) (\frac{(1 + vI_{v})(u^{*}b\beta S) - (u^{*}b\beta SI_{v}v)}{(1 + vI_{v})^{2}}) + \lambda_{7} (\mu_{v} + \delta_{v}) \;,\\ \text{With} \;\; S(0) &= S_{0}, E(0) = E_{0}, I(0) = I_{0}, R(0) = R_{0}, S_{v}(0) = S_{v_{0}}, E_{v}(0) = E_{v_{0}} and I_{v}(0) = I_{v_{0}} \;\; \text{and} \\ \lambda_{i} \left(t_{f}\right) &= 0, (i = 1, 2, 3, 4, 5, 6, 7) \end{split}$$

Since ,there were initial condition for the state variables and terminal conditions for the adjoint variabless and the optimality system is two-point boundary value problem, with separated boundary conditions at t=0 and  $t_f$ . Then we use the semi-implicit finite difference method to solve the optimality system (20). We partition the interval  $\begin{bmatrix} t_0, t_f \end{bmatrix}$  at the point  $t_i = t_0 + ih(i = 0,1,2,...,n)$ , where h is the time step such that  $t_n = t_f$ . And we define the state and adjoint variable;  $S(t), E(t), I(t), R(t), S_v(t), E_v(t), I_v(t), \lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7$  and the control u and v in terms of nodal points  $S_i, E_i, I_i, R_i, S_{v_i}, E_{v_i}, I_{v_i}, \lambda_1^i, \lambda_2^i, \lambda_3^i, \lambda_4^i, \lambda_5^i, \lambda_6^i, \lambda_7^i, u^i$  and  $v^i$ . After that we use the combination of forward and backward difference approximation. The simulations at endemic state were carried out using the following values taken from table 1, with initial condition;  $S(0)=200, E(0)=40, I(0)=20, R(0)=0, S_v(0)=2000, E_v(0)=40$  and  $I_v(0)=60$ . and results show below

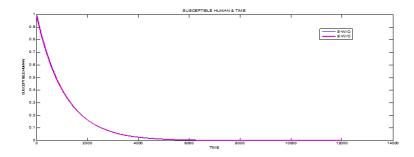
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**Table 1.** Parameters values used in numerical simulation at endemic state.

Parameters	Description	Value
μ	Death rate of human populations	0.0000548 day <sup>-1</sup>
A	Recruitment rate of susceptible human	0.000215
$\mu_{\rm v}$	Death rate of vector populations	0.0667 day <sup>-1</sup>
В	Constant inflow of infective rate	0.07
b	The average bite of mosquitoes that potentially infected	0.02
φ	the rate of loss of immunity in humans	0.09
δ	The disease-induced death rate of human	0.001
$\delta_{ m v}$	Proportional rates of mosquitoes exposed to the virus infection	0.01
γ	the duration of infection in the body	0.05
β	Probability that a bite by an infectious mosquito results in transmission of disease to human	0.1
$\beta_{\rm v}$	Probability that a bite results in transmission of parasite to a susceptible mosquito	0.09
N	Number of human populations	10000
N <sub>v</sub>	Number of mosquitoes populations	20000
α	The rate of progression of humans from the exposed state to the infectious state	0.0588
$\alpha_{_{\scriptscriptstyle V}}$	The rate of progression of mosquitoes from the exposed state to the infectious state	0.0556
u	The effort rate that reduces the contract between the infectious vector and the susceptible individuals	0 < u < 1
V	The rate at with infectious human are treated at each time period.	0 < v < 1

3.1 Numerical results for a modified SEIR-SVEVIV model for each state variable with and without control u and v.



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#### Figure 1 Represent time series of susceptible individuals (S) with and without controls.

It's show the number of susceptible individuals (S) with controls decreased faster than the number of susceptible individuals (S) without controls.

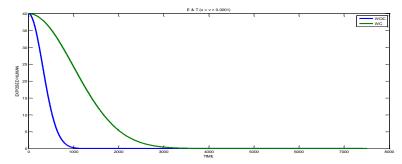


Figure 2 Represent time series of exposed individuals (E) with and without controls.

It's show the number of exposed individuals (S) with controls are increased faster than the number of exposed individuals (S) without controls.

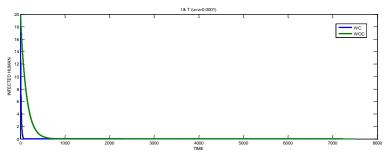


Fig. 3 Represent time series of infectious individuals (I) with and without controls.

It's show that the number of infected individuals (I) with controls are decreased rapidly.

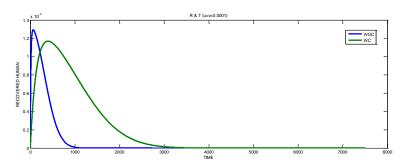


Fig. 4 Represent time series of recovered human (R) with and without controls.

It's show the number of recovered human (R) with controls are increased more faster than the number of recovered human (R) without controls.

#### 4. Conclusions

In this paper, a modified SEIR-SVEVIV model for transmission dynamics of malaria disease was proposed and analyzed in order to better understand the transmission and spread of the malaria disease and tried to find an effective strategy for its prevention. The qualitative results are depended on a basic reproductive number  $(R_0)$ . We obtained the basic reproductive number by using the next generation method technique and finding the spectral radius. Routh-Hurwitz criteria is used for determining the stabilities of the model. If  $R_0 < 1$ , then the diseases free equilibrium point is local asymptotically stable:

that is the disease will died out, but if  $R_0 > 1$ , then the endemic equilibrium is local asymptotically stable. To reduce the contract between the susceptible human and the infected vector and the other, minimizing the population of the infected human. The optimal control theory has been applied. By using the Pontryagin's maximum principle, the explicit expression of the optimal controls was obtained. Simulation results indicate that the numbers of infectious individuals (I) are decreased after control, but the number of recovered human (E) increased after control.

#### References

- [1] WHO. Fact sheets: Dengue and dengue haemorrhagicfever.http://www.who.int/mediacentre/factsheets/fs117/en/ (2014).
- [2] <a href="http://www.thaivbd.org/n/histories/view/2662">http://www.thaivbd.org/n/histories/view/2662</a>
- [3] Derouich, M. and Boutayeb, A., Dengue Fever: Mathematical modelling and computer simulation. *Applied Mathematics and Computation*. **177**(2), 528–544 (2006).
- [4] Derouich, M. A., Boutayeb and Twizell, E. H., A Model of Dengue Fever. *Brunel Universty*, *England*. p. 1–10 (2003).
- [5] Pongsumpun, P., Transmission model for dengue disease with and without the effect of extrinsic incubation period. *KMITL Sci. Tech.* J. Thailand **6**, 74–82 (2006).
- [6] Pongsumpun, P. and K. Patanarapelert., Infection risk to travelers going to dengue fever endemic regions. *Mahidol University*, Thailand **35**, 155–159 (2004).
- [7] Pongsumpun, P. and Tang, I., Transmission of dengue hemorrhagic fever in age structured population. *Mathematical and Computer Modelling* **37**, 949–961 (2003).
- [8] Ang, K. C. and Li, Z., Modeling the spread of dengue in Singapore. In Conference Proceedings for The International Congress on Modeling and Simulation 1999, Hamilton, New Zealand, December 1999, Vol. 2, p. 555–560 (2002).
- [9] Van den Driessche, P and Watmough,J (2002). Reproductive numbers and sub-threshold endemic equilibria for compartment models of disease transmission.Math. Biosci.;180;29-48
- [10] L. S. Pontryagin, V. G. Boltyanskii, R. V.Gamkrelidze, E. F. Mishchenko, The Mathematical Theory of Optimal Processes, Wiley, New York, 1962.
- [11] H.Raj joshi, S. Lenhart, M Y Li, L. Wang, Optimal control methods applied to disease models-Contemporary Mathematics, Volume 410, 2006

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