Dengue Fever with Two Strains in Thailand

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***Abstract*—In this paper, a mathematical model of dengue fever with two strains is developed. Analysis of the model reveals the existence of four equilibrium points, which belong to the region of biological interest. One of the equilibrium points corresponds to the disease–free state, the other three equilibria correspond to the two states where just one strain is present, and the state where both strain coexist, respectively. The model has a local asymptotically stable, disease-free equilibrium (DFE) wherever the maximum of the associated reproduction numbers of the two strains (denoted by R_0^s) is less than unity. The proposed model is used to forecast transmission dengue in Thailand.**

***Index Terms*—Dengue, strains, reproduction number, mathematical model, Thailand.**

I. Introduction

Dengue is one of the several emerging tropical diseases which progressively spread geographically to virtually all tropical countries. It is transmitted by the bite of an *Aedes* mosquito infected with any one of the four dengue viruses. Theses infected mosquitoes pass the disease to susceptible humans. It is known individuals who recover from one serotype become permanently immune to it, but may become partially-immune or temporarily-immune to the other serotypes [1]-[3]. Epidemiological evidence suggests that an important risk factor for dengue is the presence of preexisting antibodies at subneutralizing level [4]. This led to the formulation of secondary infection or immune enhancement hypothesis.

In Thailand, dengue epidemics have occurred every year in the last 40 years [1]. Thus, the model of dengue with two strains is formulated for predicting the transmission dynamics of dengue in Thailand.

II. Model Description

The model is formulated based on the following assumptions.

1) The total human population at time t, denoted by $N_H(t)$, is divided into twelve sub-populations, so that

$$N_H(t) = S_H(t) + E_{H1}(t) + E_{H2}(t) + I_{H1}(t) + I_{H2}(t) + R_{H1}(t) + R_{H2}(t) + E_{H12}(t) + E_{H21}(t) + I_{H12}(t) + I_{H21}(t) + R_{H22}(t). \quad (1)$$

2) Once a mosquito is infected it never recovers and it cannot be reinfection with a difference serotype of virus. Secondary infection, therefore, may take place only in human population [1]. Thus, the total mosquito population at time t, denoted by $N_v(t)$, is split into five sub-populations, so that

$$N_V(t) = S_V(t) + E_{V1}(t) + E_{V2}(t) + I_{V1}(t) + I_{V2}(t). \quad (2)$$

3) The recruitment rate of human and vector populations are denoted by Π_H and Π_V, respectively.

4) Flow from the susceptible to the infected class of both populations (human and mosquito), for each strain, depend on the biting rate of the mosquitoes, the transmission probabilities, as well as the number of infectives and susceptibles of each population [5], [6]. It assumed that the transmission probability from an infected human to a susceptible mosquito must equal the transmission probability from an infected mosquito to a susceptible human $(\rho_{VH} = \rho_{HV})$. Then, the rate of infection with strain 1 or strain 2 per susceptible human for primary infection are given by

$$\beta_1 = \frac{C_{HV}}{N_H}\left(\eta_{V1}E_{V1} + I_{V1}\right), \quad (3)$$

$$\beta_2 = \frac{C_{HV}}{N_H}\left(\eta_{V2}E_{V2} + I_{V2}\right), \quad (3)$$

where, $C_{HV} = \rho_{HV}b$, b and ρ_{HV} denote the average number of bites per mosquito per unit time and the transmission probability from an infected human to a susceptible mosquito, respectively. The parameter $\eta_{vi} \in (0, 1)$, $i = 1, 2$, account for reduction in transmissibility of exposed mosquitoes relative to infectious mosquitoes.

Similarly, primary infection of mosquito, infection rate per susceptible mosquito with strain 1 or strain 2 are given by

$$\beta_{v1} = \frac{C_{HV}}{N_H}\left(\eta_{H1}\left([ES]+[ER]\right)+[IS]+[IR]\right), \quad (5)$$

$$\beta_{v2} = \frac{C_{HV}}{N_H}\left(\eta_{H2}\left([SE]+[RE]\right)+[SI]+[RI]\right). \quad (4)$$

where, $\eta_{Hi} \in (0, 1)$, $i = 1, 2$, accounts for the reduction in transmissibility with strain i of exposed human relative to infectious human.

Manuscript received November 9, 2013; revised January 15, 2014. This work was supported by the Center of Excellence in Mathematics, the Commission on Higher Education, Thailand, and the Higher Education Research Promotion and National Research University Project of Thailand, Office of the Higher Education Commission.

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5) Immunity to reinfection with a previously experienced serotype hold lifelong. The antibody dependent enhancement (ADE) which is a negative immune reaction occurs after the temporal cross immunity period. Then, secondary infection with strain i are produced at a rate $\lambda_i\beta_i$, $i=1, 2$, where λ_i is enhancement multiple for strain i. If $0 \le \lambda_j < 1$, primary infection confer partial or total immunity to strain j for $j=1, 2$. If $\lambda_j > 0$, primary infection increase susceptibility to strain j due to immune enhancement. If $\lambda_j = 1$, primary infections do not alter the susceptibility to secondary infections [7], [8].
6) All subpopulations of human and mosquito die at the same rate μ_H and μ_V, respectively.
7) The parameter σ_i, $i=1, 2$ denotes the transfer from exposed human to infectious human. The parameter σ_{Vi}, $i=1, 2$ denotes the transfer rate from exposed vector to infectious mosquito.
8) The per capita mortality rate of dengue virus with strain i for infectious human and infectious mosquito are δ_i, $i=1, 2$ and δ_{Vi}, $i=1, 2$, respectively.
9) The recovery rate of infectious human with strain i is γ_i, $i=1, 2$.

With above assumptions, the model of dengue with two strains described by the following system of nonlinear differential equations

$$\frac{dS_H}{dt} = \Pi_H - (\beta_1 + \beta_2) S_H - \mu_H S_H, \tag{7}$$

$$\frac{dE_{H1}}{dt} = \beta_1 S_H - (\sigma_1 + \mu_H) E_{H1}, \tag{5}$$

$$\frac{dE_{H2}}{dt} = \beta_2 S_H - (\sigma_2 + \mu_H) E_{H2}, \tag{6}$$

$$\frac{dI_{H1}}{dt} = \sigma_1 E_{H1} - (\gamma_1 + \delta_1 + \mu_H) I_{H1}, \tag{10}$$

$$\frac{dI_{H2}}{dt} = \sigma_2 E_{H2} - (\gamma_2 + \delta_2 + \mu_H) I_{H2}, \tag{11}$$

$$\frac{dR_{H1}}{dt} = \gamma_1 I_{H1} - (\lambda_2\beta_2 + \mu_H) R_{H1}, \tag{7}$$

$$\frac{dR_{H2}}{dt} = \gamma_2 I_{H2} - (\lambda_1\beta_1 + \mu_H) R_{H2}, \tag{8}$$

$$\frac{dE_{H12}}{dt} = \lambda_2\beta_2 R_{H2} - (\sigma_2 + \mu_H) E_{H12}, \tag{9}$$

$$\frac{dE_{H21}}{dt} = \lambda_1\beta_1 R_{H1} - (\sigma_1 + \mu_H) E_{H21}, \tag{15}$$

$$\frac{dI_{H12}}{dt} = \sigma_2 E_{H12} - (\gamma_2 + \delta_2 + \mu_H) I_{H12}, \tag{16}$$

$$\frac{dI_{H21}}{dt} = \sigma_1 E_{H21} - (\gamma_1 + \delta_1 + \mu_H) I_{H21}, \tag{10}$$

$$\frac{dR_{H22}}{dt} = \gamma_1 I_{H21} + \gamma_2 I_{H12} - \mu_H R_{H22}, \tag{11}$$

$$\frac{dS_v}{dt} = \Pi_v - (\beta_{v1} + \beta_{v2}) S_v - \mu_v S_v, \tag{12}$$

$$\frac{dE_{v1}}{dt} = \beta_{v1} S_v - (\sigma_{v1} + \mu_v) E_{v1}, \tag{13}$$

$$\frac{dE_{v2}}{dt} = \beta_{v2} S_v - (\sigma_{v2} + \mu_v) E_{v2}, \tag{14}$$

$$\frac{dI_{v1}}{dt} = \sigma_{v1} E_{v1} - (\delta_{v1} + \mu_v) I_{v1}, \tag{22}$$

$$\frac{dI_{v2}}{dt} = \sigma_{v2} E_{v2} - (\delta_{v2} + \mu_v) I_{v2}. \tag{15}$$

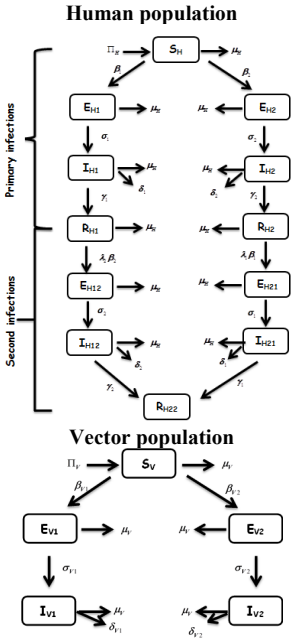


Fig. 1. Schematic diagram of the system (7)-(23).

TABLE I: DESCRIPTION OF VARIABLE OF THE SYSTEM (7)-(23)

Variables	*Description*
S_H	Humans susceptible to both strains
E_{H1}	Humans exposed with strain 1 in primary infection
E_{H2}	Humans exposed with strain 2 in primary infection
I_{H1}	Humans infected with strain 1 in primary infection
I_{H2}	Humans infected with strain 2 in primary infection
R_{H1}	Humans recovered with strain 1 in primary infection
R_{H2}	Humans recovered with strain 2 in primary infection
E_{H12}	Humans exposed to strain 2 in secondary infection
E_{H21}	Humans exposed to strain 1 in secondary infection
I_{H12}	Humans infected to strain 2 in secondary infection
I_{H21}	Humans infected to strain 1 in secondary infection
R_{H22}	Humans recovered with both strain
S_V	Mosquitoes susceptible to both strains
E_{V1}	Mosquitoes exposed with strain 1
E_{V2}	Mosquitoes exposed with strain 2
I_{V1}	Mosquitoes infected with strain 1
I_{V2}	Mosquitoes infected with strain 2

Complete interaction and a schematic flow diagram of human and mosquito populations are depicted in Fig. 1, and the associated variables and parameters are described in Tables I, respectively.

III. Analysis of the Model

A. Basic Properties of the Model

From of biological considerations, the system (7)-(23) is studied in the region of biological interest

$$T = T_1 \cup T_2 \subset \mathbb{R}_+^{12} \times \mathbb{R}_+^5,$$

where

$$T_1 = \Big\{ \big(S_H, E_{H1}, E_{H2}, I_{H1}, I_{H2}, R_{H1}, R_{H2}, E_{H12}, E_{H21}, I_{H12}, I_{H21}, R_{H22}\big) \in \mathbb{R}_+^{12} : N_H \le \frac{\Pi_H}{\mu_H} \Big\}$$

and $T_2 = \left\{ \big(S_V, E_{V1}, E_{V2}, I_{V1}, I_{V2}\big) \in \mathbb{R}_+^5 : N_V \le \frac{\Pi_V}{\mu_V} \right\}.$

It can be shown that the closed set T is positively invariant and attracting for the system(7)-(23), see more detail in [9]. Hence, it is sufficient to study the dynamics of the system (7)-(23) in T.

B. Disease Free Equilibrium and the Basic Reproduction Number

In the absence of infection (that is, all infected components are zero), the system has a disease-free equilibrium, DFE, obtained by setting the right-hand sides of (7)-(23) to zero, is given by

$$\begin{aligned} E_0^* &= \big(S_H^*, E_{H1}^*, E_{H2}^*, I_{H1}^*, I_{H2}^*, R_{H1}^*, R_{H2}^*, E_{H12}^*, E_{H21}^*, \\ &\quad I_{H12}^*, I_{H21}^*, R_{H22}^*, S_V^*, E_{V1}^*, E_{V2}^*, I_{V1}^*, I_{V2}^*\big) \\ &= \left(\frac{\Pi_H}{\mu_H}, 0,0,0,0,0,0,0,0,0,0,0, \frac{\Pi_v}{\mu_v}, 0,0,0,0 \right). \end{aligned} \tag{16}$$

The local stability of E_0^* can be established using the next generation operator method [10], we have

$$R_0^s = \max\{R_1, R_2\}, \tag{17}$$

where, R_1 and R_2 are the associated reproduction numbers for strain 1 and strain 2, respectively, given by

$$R_i = \sqrt{\frac{C_{HV}^2 \Pi_V \mu_H (\eta_{Hi} B_i + \sigma_i)(\eta_{Vi} D_i + \sigma_{Vi})}{\Pi_H \mu_V A_i B_i C_i D_i}}, \tag{18}$$

For $i = 1, 2,$ where, $A_1 = \sigma_1 + \mu_H$, $A_2 = \sigma_2 + \mu_H$, $B_1 = \gamma_1 + \delta_1 + \mu_H$, $B_2 = \gamma_2 + \delta_2 + \mu_H$, $C_1 = \sigma_{V1} + \mu_V$, $C_2 = \sigma_{V2} + \mu_V$, $D_1 = \delta_{V1} + \mu_V$ and $D_2 = \delta_{V1} + \mu_V$.

The following Theorem, using theorem 2 in [10], is established.

Theorem 1. The DFE, E_0^* of the system (7)-(23) is locally asymptotically stable (LAS) if $R_0^s < 1$, and unstable if $R_0^s > 1$.

The threshold quantity R_0^s given in (17) is called basic reproduction number of the model. It represents the average number of secondary cases of strain *i,* produced by a single infective of strain *i,* in completely susceptible population [9].

The results of Theorem 1, indicates that the disease will be eradicated from the community if $R_0^s < 1$, that is both R_1 and R_2 are less than unity. Therefore, in the event of an endemic is to determine the condition that can make $R_0^s < 1$, which is of great public health interest.

C. The Existence of Endemic Equilibrium

The equilibria of the system (7)-(23) are difficult to be expressed in closed form, an approach in [11] for investigating the existence of endemic equilibrium of the system is as follow.

Let,

$$\begin{aligned} E^* &= \big(S_H^*, E_{H1}^*, E_{H2}^*, I_{H1}^*, I_{H2}^*, R_{H1}^*, R_{H2}^*, E_{H12}^*, \\ &\quad E_{H21}^*, I_{H12}^*, I_{H21}^*, R_{H22}^*, S_V^*, E_{V1}^*, E_{V2}^*, I_{V1}^*, I_{V2}^*\big) \end{aligned} \tag{19}$$

be any positive equilibria where at the last one of the infected variable of the system (7)-(23) is non-zero. Further, the forces of infection with strain 1 and strain 2 at steady state are given by, respectively,

$$\beta_1^* = \frac{C_{HV}}{N_H^*}\big(\eta_{v1} E_{V1}^* + I_{V1}^*\big), \tag{20}$$

$$\beta_2^* = \frac{C_{HV}}{N_H^*}\big(\eta_{v2} E_{V2}^* + I_{V2}^*\big). \tag{21}$$

Setting all derivative in the system (7)-(23) equal to zero and solving the obtain results give the positive equilibrium in terms of β_1^* and β_2^* as follow,

$$S_H^* = \frac{\Pi_H}{\beta_1^* + \beta_2^* + \mu_H}, \; E_{H1}^* = \frac{\Pi_H \beta_1^*}{A_1\big(\beta_1^* + \beta_2^* + \mu_H\big)},$$

$$E_{H2}^* = \frac{\Pi_H \beta_2^*}{A_2\big(\beta_1^* + \beta_2^* + \mu_H\big)}, \; I_{H1}^* = \frac{\Pi_H \sigma_1 \beta_1^*}{A_1 B_1\big(\beta_1^* + \beta_2^* + \mu_H\big)},$$

$$I_{H1}^* = \frac{\Pi_H \sigma_1 \beta_1^*}{A_1 B_1\big(\beta_1^* + \beta_2^* + \mu_H\big)}, \; I_{H1}^* = \frac{\Pi_H \sigma_1 \beta_1^*}{A_1 B_1\big(\beta_1^* + \beta_2^* + \mu_H\big)},$$

$$R_{H2}^* = \frac{\Pi_H \sigma_2 \gamma_2 \beta_2^*}{A_2 B_2 \mu_H\big(\lambda_1 \beta_1^* + \mu_H\big)\big(\beta_1^* + \beta_2^* + \mu_H\big)},$$

$$R_{H2}^* = \frac{\Pi_H \sigma_2 \gamma_2 \beta_2^*}{A_2 B_2 \mu_H\big(\lambda_1 \beta_1^* + \mu_H\big)\big(\beta_1^* + \beta_2^* + \mu_H\big)},$$

$$E^*_{H21}=\frac{\Pi_H\sigma_2\gamma_2\lambda_1\beta_1^*\beta_2^*}{A_1A_2B_2\left(\lambda_1\beta_1^*+\mu_H\right)\left(\beta_1^*+\beta_2^*+\mu_H\right)},$$

$$I^*_{H12}=\frac{\Pi_H\sigma_1\sigma_2\gamma_1\lambda_2\beta_1^*\beta_2^*}{A_1A_2B_1B_2\left(\lambda_2\beta_2^*+\mu_H\right)\left(\beta_1^*+\beta_2^*+\mu_H\right)}, \tag{22}$$

$$I^*_{H21}=\frac{\Pi_H\sigma_1\sigma_2\gamma_2\lambda_1\beta_1^*\beta_2^*}{A_1A_2B_1B_2\left(\lambda_1\beta_1^*+\mu_H\right)\left(\beta_1^*+\beta_2^*+\mu_H\right)},$$

$$R^*_{H22}=\frac{\Pi_H\sigma_1\sigma_2\beta_1^*\beta_2^*\left\{\gamma_1\lambda_2\left(\lambda_1\beta_1^*+\mu_H\right)+\gamma_2\lambda_1\left(\lambda_2\beta_2^*+\mu_H\right)\right\}}{A_1A_2B_1B_2\left(\lambda_1\beta_1^*+\mu_H\right)\left(\lambda_2\beta_2^*+\mu_H\right)\left(\beta_1^*+\beta_2^*+\mu_H\right)}$$

$$S^*_V=\frac{\Pi_V}{\beta^*_{V1}+\beta^*_{V2}+\mu_V},\ E^*_{V1}=\frac{\beta^*_{V1}\Pi_V}{C_1\left(\beta^*_{V1}+\beta^*_{V2}+\mu_V\right)},$$

$$E^*_{V2}=\frac{\Pi_v\beta^*_{V2}}{C_2\left(\beta^*_{V1}+\beta^*_{V2}+\mu_V\right)},\ I^*_{V1}=\frac{\Pi_V\sigma_{V1}\beta^*_{V1}}{C_1D_1\left(\beta^*_{V1}+\beta^*_{V2}+\mu_V\right)},$$

$$I^*_{V2}=\frac{\Pi_V\sigma_{V2}\beta^*_{V2}}{C_2D_2\left(\beta^*_{V1}+\beta^*_{V2}+\mu_V\right)}.$$

where β^*_{V1} and β^*_{V2} are determined by (5) and (6) at steady state.

Substituting the expressions (30) into the force of infections β_1^* and β_2^* given in (28) and (29), respectively, yields the fixed points problem

$$x=\phi(x)=\begin{bmatrix}\phi_1\left(\beta_1^*,\beta_2^*\right)\\ \phi_2\left(\beta_1^*,\beta_2^*\right)\end{bmatrix} \tag{23}$$

where,

$$\beta_1^*=\phi_1\left(\beta_1^*,\beta_2^*\right)=\frac{C_{HV}\left(\eta_{V1}D_1+\sigma_{V1}\right)\Pi_V\beta^*_{V1}}{N_H^*C_1D_1\left(\beta^*_{V1}+\beta^*_{V2}+\mu_V\right)}, \tag{24}$$

$$\beta_2^*=\phi_2\left(\beta_1^*,\beta_2^*\right)=\frac{C_{HV}\left(\eta_{V2}D_2+\sigma_{V2}\right)\Pi_v\beta^*_{V2}}{N_H^*C_2D_2\left(\beta^*_{V1}+\beta^*_{V2}+\mu_V\right)}. \tag{33}$$

and $x=\begin{bmatrix}\beta_1^*\\ \beta_2^*\end{bmatrix}$.

Thus, solving (31) for β_1^* and β_2^* substituting obtained results into expression (30), yield three endemic equilibria of the model as in the following.

1) Competitive exclusion

Strain 1-only boundary equilibrium, denoted by E_1^*, occurs whenever $R_2<1<R_1$ so that $R_0^s>1$. It is given by is given by

$$E_1^*=\left(S_H^*,E^*_{H1},0,I^*_{H1},0,R^*_{H1},0,0,0,0,0,0,S_V^*,E^*_{V1},0,I^*_{V1},0\right) \tag{25}$$

where, $S_H^*=\frac{\Pi_H}{\beta_1^*+\mu_H}$, $E^*_{H1}=\frac{\Pi_H\beta_1^*}{A_1\left(\beta_1^*+\mu_H\right)}$,

$$I^*_{H1}=\frac{\Pi_H\sigma_1\beta_1^*}{A_1B_1\left(\beta_1^*+\mu_H\right)},\ R^*_{H1}=\frac{\Pi_H\sigma_1\gamma_1\beta_1^*}{A_1B_1\mu_H\left(\beta_1^*+\mu_H\right)},$$

$$S^*_V=\frac{\Pi_V}{\beta^*_{V1}+\mu_V},\ E^*_{V1}=\frac{\beta^*_{V1}\Pi_V}{C_1\left(\beta^*_{V1}+\mu_V\right)},\ I^*_{V1}=\frac{\Pi_V\sigma_{V1}\beta^*_{V1}}{C_1D_1\left(\beta^*_{V1}+\mu_V\right)}.$$

Strain 2-only boundary equilibrium, denoted by E_2^*, occurs whenever $R_1<1<R_2$ so that $R_0^s>1$. It is given by

$$E_2^*=\left(S_H^*,0,E^*_{H2},0,I^*_{H2},0,R^*_{H2},0,0,0,0,0,S_V^*,0,E^*_{V2},0,I^*_{V2}\right) \tag{26}$$

where $S_H^*=\frac{\Pi_H}{\beta_2^*+\mu_H}$, $E^*_{H2}=\frac{\Pi_H\beta_2^*}{A_2\left(\beta_2^*+\mu_H\right)}$,

$$I^*_{H2}=\frac{\Pi_H\sigma_2\beta_2^*}{A_2B_2\left(\beta_2^*+\mu_H\right)},\ R^*_{H2}=\frac{\Pi_H\sigma_2\gamma_2\beta_2^*}{A_2B_2\mu_H\mu_H\left(\beta_2^*+\mu_H\right)},$$

$$S^*_V=\frac{\Pi_V}{\beta^*_{V2}+\mu_V},\ S^*_V=\frac{\Pi_V}{\beta^*_{V2}+\mu_V},\ I^*_{V2}=\frac{\Pi_V\sigma_{V2}\beta^*_{V2}}{C_2D_2\left(\beta^*_{V2}+\mu_V\right)}.$$

2) Co-existence equilibrium

Co-existence equilibrium, denoted by E_{12}^*, occurs whenever $R_1>1$ and $R_2>1$ so that $R_0^s>1$. it is given by E_{12}^* as given in (27)

D. Stability Analysis

The equilibrium in (34) and (35) are a second type of equilibrium which is competitive exclusion. These equilibriums occur when one strain in the populations is stronger than the other strain, causing the weaker strain to die out. Only one strain will be present in the long term of the system (7)-(23). Further, a third type of equilibrium is co-existence. This equilibrium occurs when two strains are present in populations.

To analyze the stability of all endemic equiibria, we find that large explicit solution for all endemic equilibrium points given in (27), (34) and (35), respectively, make proving stability complicate. Thus, stability of endemic equilibria are verified by using numerical simulations of the system (7)-(23) are as discuss in next section. The simulation is carried out into four experiments.

IV. Numerical Simulations

To illustrate the dynamics of the model are simulated with a set of parameter values in Table II and the parameters C_{HV}, η_{V1}, η_{V2}, η_{H1} and η_{H2} are vary. The initial values for experiment 1, 2 and 3 are $S_H(0)=5000$, $E_{H1}(0)=10$,

$E_{H2}(0)=50, I_{H1}(0)=20,\quad I_{H2}(0)=60,\quad R_{H1}(0)=70000,$
$R_{H2}(0)=80000,\quad E_{H12}(0)=800,\quad E_{H21}(0)=200,$
$I_{H12}(0)=300,$
$I_{H21}(0)=100,\quad R_{H22}(0)=600000,\quad S_V(0)=60000,$
$E_{V1}(0)=100,\quad E_{V2}(0)=2000,\quad I_{V1}(0)=100, I_{V2}(0)=300.$

The simulations are carried out into four experiments.

Experiment 1: Infection dies out both strain i and stain j

The system (7)-(23) is first simulated using the parameter values in Table II with $C_{HV}=0.8$, $\eta_{v1}=0.1$, $\eta_{v2}=0.3$ and $\eta_{H1}=\eta_{H2}=0.66$. It follows that reproduction numbers $R_1=0.6031$, $R_2=0.8430$ so that $R_0^s<1$. The time series simulations of the total number of infected with strain 1 and strain 2 are depicted in Fig. 2. These results show that the persistence of strain 1 and strain 2 are dies out.

Experiment 2: Infection with strain *i* and die out strain *j*

The time series simulations of the infected human population with strain 1 and strain 2 are depicted in Fig. 3 and 4, respectively. These result depict the infected human population with strain 1 establishes itself at steady state (see Fig. 3 a)), while the infected human population with strain 2 declines to zero (see Fig. 3 b)). This is verified the persistence of dengue with strain 1 while strain 2 dies out when $R_0^s>1$ (that is $R_1>1$ and $R_2<1$). Similarly, the persistence of strain 2 while strain 1 dies out when $R_1<1$ and $R_2>1$ (that is $R_0^s>1$), see Fig. 4**.**

In summary, the model undergoes competitive exclusion, with strain *i* driving out strain *j* if with strain *i* driving out strain *j* when $R_i>1$ and $R_j<1$.

Experiment 3: Infection with both strains

When the reproduction number of each strain exceeds unity, co-existence equilibrium exists as predicted in Fig. 5. These results show that the infected human population with strain, which has the higher reproduction number, will dominate, but not drive out the infection human population with other strain.

Experiment 4: Comparison of simulation results and epidemiological data

The simulation results of the model are compared to reported values the dengue cases of some province in Thailand such as Chachoengsao, Roiet, Zone 13 (Nakonnayok, Prachinburi, Chachoengsao, Sakaeo, Samutprakan) and Zone 18 (Kamphaengphet, Nakornsawan, Phichit, Utaitani). The parameter used in simulation is given in Table II, except the parameter $C_{HV}, \eta_{Hi}, \eta_{Vi}, \sigma_i, \sigma_{Vi}$, for $i=1,2$ and initial conditions are varied in each considered area as follows.

For Chachoengsao: $C_{HV}=1.8, \eta_{H1}=\eta_{V1}=0.385,$ $\eta_{H2}=\eta_{V2}=0.49, \sigma_1=\sigma_{V1}=0.5,$ and $\sigma_{V2}=0.01.$

So that $R_o^S>1$ (that is $R_2=4.0322>R_1=3.4247>1$) and initial values are
$S_H(0)=679370, E_{H1}(0)=50,\ E_{H2}(0)=50,$
$I_{H1}(0)=150,\quad R_{H1}(0)=2000,\quad R_{H2}(0)=2000,$
$E_{H12}(0)=100,$
$E_{H21}(0)=100,\ I_{H12}(0)=150,\ I_{H21}(0)=150,\ R_{H22}(0)=0,$
$S_v(0)=5000,\quad E_{v1}(0)=100, E_{v2}(0)=100,\quad I_{v1}(0)=150,$
$I_{v2}(0)=150.$

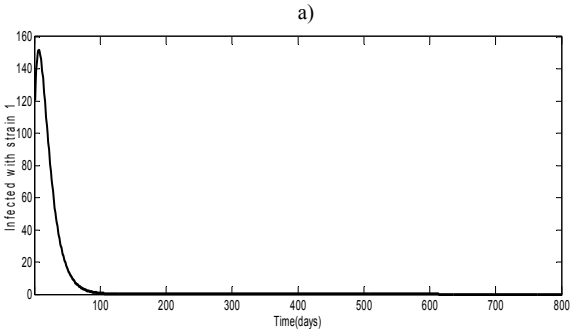
For Roiet: $C_{HV}=1.8, \eta_{H1}=\eta_{V1}=0.385,\ \eta_{H2}=0.49,$ $\eta_{V2}=0.49,\ \sigma_1=\sigma_2=0.2,$ and $\sigma_{V1}=\sigma_{V2}=0.1$ so that $R_o^S>1$ (that is $R_1=2.3188>R_2=2.1216>1$) and initial values are
$S_{H1}(0)=140000, E_{H1}(0)=3, E_{H2}(0)=3,\ I_{H1}(0)=30,$
$R_{H1}(0)=5000,\ R_{H2}(0)=3000,\ E_{H12}(0)=0, E_{H21}(0)=0,$
$I_{H12}(0)=30,\ I_{H21}(0)=50,\ I_{H21}(0)=50,\ S_V(0)=60000,$
$E_{V1}(0)=30, E_{V2}(0)=10,\ I_{V1}(0)=120,\ I_{V2}(0)=90.$

For Zone 3: $C_{HV}=1.8, \eta_{H1}=\eta_{V1}=0.385,\ \eta_{H2}=0.49,$ $\eta_{V2}=0.49,\ \sigma_1=\sigma_2=0.1428,$ and $\sigma_{V1}=\sigma_{V2}=0.1$ so that $\sigma_{V1}=\sigma_{V2}=0.1$ (that is $R_1=4.2600>R_2=2.3872>1$) and initial values are
$S_H(0)=60000, E_{H1}(0)=0, E_{H2}(0)=0,\quad I_{H1}(0)=50,$
$I_{H2}(0)=50,\quad R_{H1}(0)=400000,\quad R_{H2}(0)=20000,$
$E_{H12}(0)=0,\quad E_{H21}(0)=0,\quad I_{H12}(0)=50, I_{H21}(0)=50,$
$R_{H22}(0)=300000,\quad S_V(0)=70000,$
$E_{V1}(0)=10, E_{V2}(0)=10, I_{V1}(0)=100,\ I_{V2}(0)=100.$

For Zone 18: $C_{HV}=1.8, \eta_{H1}=\eta_{V1}=0.385,\ \eta_{H2}=0.49,$ $\eta_{V2}=0.49,\ \sigma_1=\sigma_{V1}=0.5, \sigma_2=0.05, \sigma_{V2}=0.01$ So that $R_o^S>1$ (that is $R_2=4.0322>R_1=3.4247>1$) and initial values are $S_H(0)=60000,\quad E_{H1}(0)=0,\ E_{H2}(0)=1,$
$I_{H1}(0)=50,$
$I_{H2}(0)=1,\ R_{H1}(0)=600000,\ R_{H2}(0)=70000,\ E_{H12}(0)=0,$
$E_{H21}(0)=1,\ I_{H12}(0)=0, I_{H21}(0)=1,\ R_{H22}(0)=70000,$
$S_V(0)=60000,\ E_{V1}(0)=1,\ E_{V2}(0)=1, I_{V1}(0)=1, I_{V2}(0)=1.$

All simulation results are displayed in Fig. 6. It is seen that the trend of the number of infected humans is similar but not fit well to real dengue data that is reported from the bureau of epidemiology, Thailand. Whereas the model can predict the duration of outbreak.

a)



b)

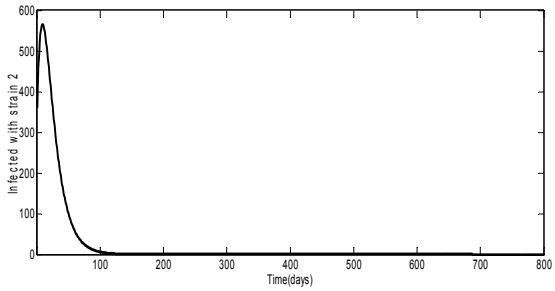
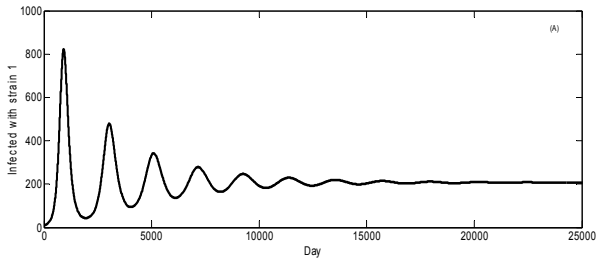


Fig. 2. Time series plots for the system (7)-(23) for parameter $C_{HV}=0.8,\eta_{v1}=0.1,\eta_{v2}=0.3,\ \eta_{H1}=\eta_{H2}=0.66$ with the other parameter values used in Table II, so that $R_0^s<1\ \left(R_1=0.6031<R_2=0.8430<1\right)$ a) Total number of individuals infected with strain 1. b) Total number of individuals infected with strain 2.

a)



b)

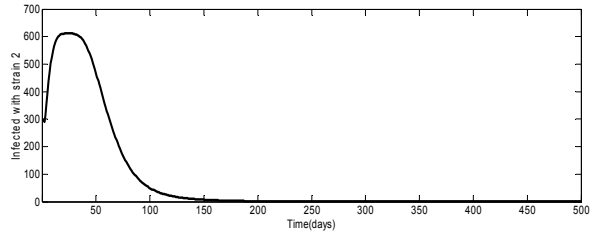


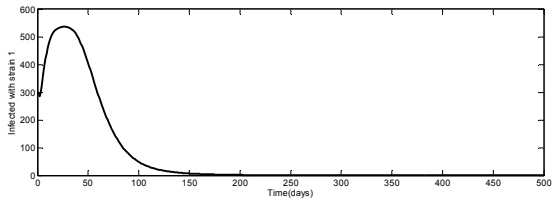
Fig. 3. Time series plots for the system (7)-(23) for parameter $C_{HV}=1.8,\eta_{v1}=0.8,\ \eta_{v2}=0.02,\eta_{H1}=0.8,\eta_{H2}=0.05$ with the other parameter values used in Table II, so that $R_0^s<1\ \left(R_2=0.9148<1<R_1=2.6723\right)$ a) Total number of individuals infected with strain 1. b) Total number of individuals infected with strain 2.

V. Conclusion

The model of dengue with two strains is formulated to gain insight into their dynamical features. The study shows the following

1) The associated reproduction number of the two strains denoted by R_0^S, is derived and given in (25).
2) The disease-free equilibrium of the model is locally asymptotically stable whenever $R_0^S<1$ (that is the associated reproductive number value for each strain is less than one). This indicates that the number of infected population will be brought to zero if public health measures that make the threshold R_0^S to a value less than unity are carried out.
3) The model has three equilibrium types: disease-free, competitive exclusion, and co-existence.
4) The model undergoes competitive exclusion in the sense that the persistence of strain *i*, while strain *j* dies out whenever $R_i>1>R_j,\ \left(i,j=1,2,i\neq j\right)$.
5) The complexity of model makes finding an explicit point for co-existence equilibrium impractical. However, the numerical simulation is evidence that co-existence equilibrium is stable whenever $R_i>R_j>1,$ $\left(i,j=1,2,i\neq j\right)$. the results also state that the strain with the higher reproduction number will dominate, but not drive out the other strain.
6) When the model is use to can predict the transmission of dengue in some areas of Thailand by comparing with the real dengue data. The results show that the model can predict the duration of outbreak but not fit well to the number of infected population. This may be the model not including the seasonal variation.

a)



b)

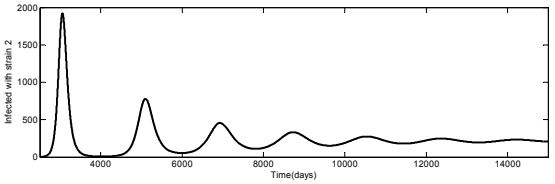
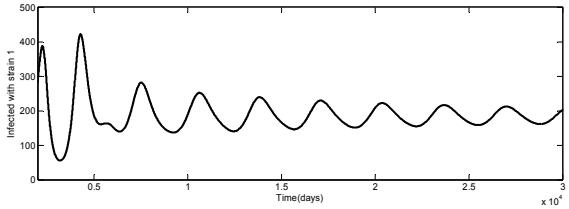


Fig. 4. Time series plots for the Model (7)-(23) for parameter $C_{HV}=1.8,\eta_{v1}=0.02,\quad \eta_{v2}=0.9,\eta_{H1}=0.03,\eta_{H2}=0.8$ with the other parameter values used in Table II, so that $R_0^s<1\ \left(R_1=0.9118<1<R_2=3.0981\right)$ a) Total number of individuals infected with strain 1. b) Total number of individuals infected with strain 2.

a)



b)

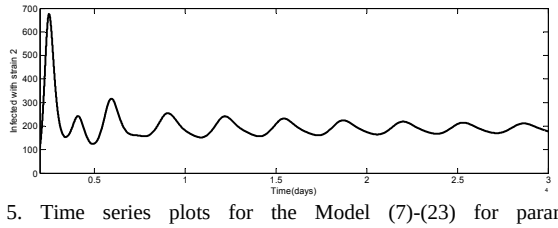
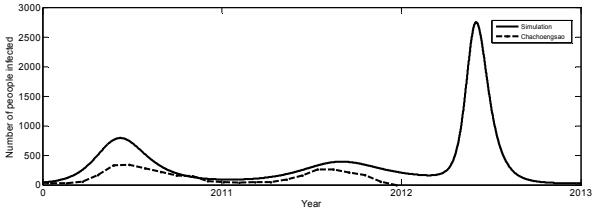


Fig. 5. Time series plots for the Model (7)-(23) for parameter $C_{HV}=1.8,\eta_{v1}=0.8,\ \eta_{v2}=0.5,\eta_{H1}=0.8,\eta_{H2}=0.5$ with the other parameter values used in Table II, so that $R_0^s<1\ \left(R_2=4.4929>R_1=4.1336>1\right)$ a) Total number of individuals infected with strain 1. b) Total number of individuals infected with strain 2.

a)



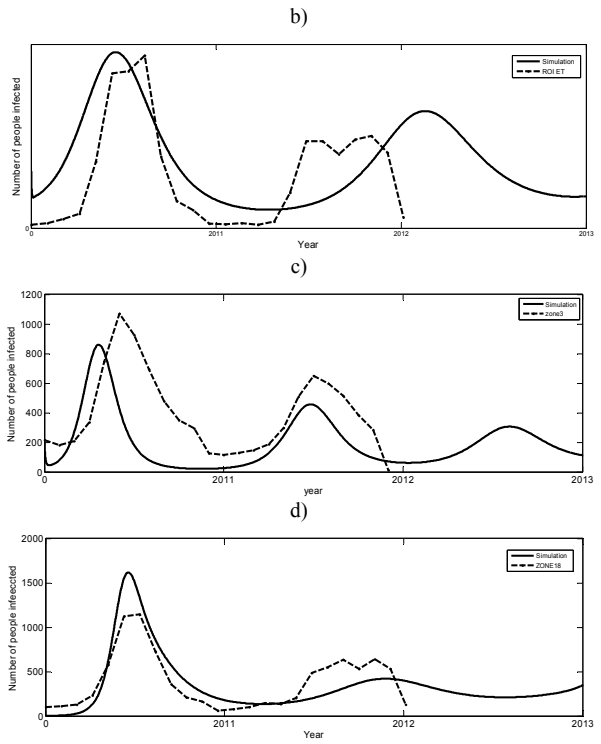


Fig. 6. Comparison of simulation results (solid lines) and reported valued from the bureau of epidemiology, Thailand (dotted lines) for infection case. a) Chachoengsao, b) Roi et, c) Zone 13 and d) Zone 18.

TABLE II: PARAMETER OF THE SYSTEM (7)-(23)

Parameters	*Description*	*Values*
Π_H	Recruitment rate of human	30 day^{-1} [11]
Π_v	Recruitment rate of mosquitoes	4000 day^{-1} [11]
μ_H	Natural death rate of humans	1/70 years^{-1}[11]
μ_v	Natural death rate of mosquitoes	1/14 days^{-1} [1]
b	Biting rate	0.33-1days^{-1} [1]
ρ_{HV}	Transmission probability from infected humans to susceptible mosquitoes	0.75 days^{-1} [12]
ρ_{VH}	Transmission probability from infected mosquitoes to susceptible humans	0.75 days^{-1} [12]
$\delta_i\,(i=1,\,2)$	Disease-induced death rate for humans infected with strain i	0.134 day^{-1} [12]
$\delta_{vi}\,(i=1,\,2)$	Disease-induced death rate for mosquitoes infected with strain i	0.077 day^{-1} [12]
$\gamma_i\,(i=1,\,2)$	Infectious period in humans with strain i	0.1428 day^{-1} [3]
$\sigma_i\,(i=1,\,2)$	Incubation period in human with strain i	0.5 day^{-1} [3]
$\sigma_{vi}\,(i=1,\,2)$	Incubation period in mosquito with strains i	0.1 day^{-1} [3]
$\lambda_i\,(i=1,\,2)$	Enhancement multiple for the strain i	0.66
$\eta_{Hi}\,(i=1,\,2)$	Modification parameter in human	(0, 1) [12]
$\eta_{vi}\ \ (i=1,\,2)$	Modification parameter in mosquito	(0, 1) [12]
C_{HV}	Infection rate	Assume

ACKNOWLEDGMENT

This research is (partially) supported by the Center of Excellence in Mathematics, the Commission on Higher Education, Thailand, and the Higher Education Research Promotion and National Research University Project of Thailand, Office of the Higher Education Commission (Under NRU-CSEC Project).

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