

TRANSMISSION MODEL OF CHIKUNGUNYA FEVER IN THE PRESENCE OF TWO SPECIES OF *Aedes* MOSQUITOES

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ABSTRACT

In 2008 there was a large outbreak of Chikungunya fever in the south of Thailand. Chikungunya fever is a febrile disease transmitted to humans by the bite of infected *Aedes* mosquitoes. The symptoms of this disease are a sudden onset of a fever, chills, headache, nausea, vomiting, joint pain with or without swelling, low back pain and rash. In this study we study the effects of there being two species of *Aedes* mosquito (*Aedes aegypti* and *Aedes albopictus*) present. In this study, we assume that both the human and mosquito populations are constant. A dynamical model of Chikungunya fever is proposed and analyzed. The Routh-Hurwitz criteria are used to determine the stability of the model. The conditions which would lead to either the disease free equilibrium state or the disease endemic equilibrium state to exist is determined. The numerical simulations are done in order to illustrate the behaviors of transmission of disease for different values of parameters. It is shown that the destruction of breeding sites could be an effective method to control this disease.

Keywords: Mathematical Model, Chikungunya Fever, Basic Reproductive Number, Equilibrium Point, *Aedes* Mosquitoes

1. INTRODUCTION

Chikungunya fever is an emerging, mosquito-borne disease caused by an *alphavirus* of *Togaviridae* family, which first was isolated in 1953 in Tanzania (PAHO, 2011). The symptoms of this disease are a high fever, extreme joints pains and an irritating skin rash (Tilston *et al.*, 2009). This disease is self-limiting and some patients suffered from along-lasting arthritis akin (Kucharz and Cebula-Byrska, 2012). In the major outbreak of this disease in 2005 on the island of Réunion, 244,000 out of a population of 775,000 inhabitants reported that they had experienced these symptoms (Moulay *et al.*, 2011). Chikungunya Virus (CHIKV) is transmitted to humans by the bite of an infected *Aedes* mosquito, widespread in some tropical

regions. The *Ae. albopictus* mosquito is a highly competent vector for this virus (Poletti *et al.*, 2011). A genetic change at position 226 in the gene for the glycoprotein E1/E2 created a mutated Chikungunya virus strain which had an increased capability for replication in the *Ae. albopictus* mosquito (Tsetsarkin, 2009).

Mathematical modeling is a useful tool for understanding and describing the transmission of this disease. Pongsumpun (2010) has studied the effects of there being a seasonal variation in the number of mosquitoes on the transmission of this disease. The results showed that length of the outbreak would be shorter if the basic reproduction number would be higher. Poletti *et al.* (2011) developed a model describing the temporal dynamics of the vector depending on climate factors, coupled to an epidemic transmission model describing the

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spread of the epidemic in both humans and mosquitoes. The cumulative number of notified cases predicted by the model was 185 on average, in good agreement with observed data. They found that the basic reproduction number was in the range of 1.8-6. Yakob and Clements (2013) proposed a simple model of the virus transmission between humans and mosquitoes. This model is fitted with data from Reunion epidemic, with basic reproductive number is 4.1. In a latter study, Moulay and Pigne (2013) proposed a metapopulation model to represent both a high-resolution humans and mosquitoes. Numerical results showed the impact of the geographical environment and populations' mobility on the spread of the disease.

In Asia and the Indian Ocean region the main CHIKV vectors are the *Ae. aegypti* and *Ae. albopictus* mosquitoes. *Ae. albopictus* is particularly resilient and it can survive in both rural and urban area. It has shown a remarkable capacity to adapt to human being and to urbanization, allowing the *Ae. albopictus* mosquito to supersede *Ae. aegypti* mosquito. *Ae. albopictus* has a relatively longer life expectancy about 4 to 8 weeks and a flight radius of 400-600 m. It is aggressive biter and is diurnal. Preechaporn *et al.* (2007) investigated the seasonal prevalence of *Ae. aegypti* and *Ae. albopictus* in three topographical areas (mangrove, rice paddy and mountainous areas) in both wet and dry seasons in Nakhon Si Thammarat province, Thailand. *Ae. aegypti* larval indices were higher than the *Ae. Albopictus* larval indices in all three topographical areas during both seasons. In this study, we have formulated and analyze the Chikungunya fever model in which both species of the *aedes* mosquitoes are present. In most countries, only one species are present. In Thailand however both species are present.

2. MATERIALS AND METHODS

2.1. Model Formulation

In our model, we assume that human population and mosquito population are constant denoted by N_h and N_m respectively. The dynamics of the disease is depicted in the compartment diagram, **Fig. 1**.

The human population is divided into the susceptible human (\bar{S}_h), the infected human (\bar{I}_h) and the recover human population (\bar{R}_h) compartment. The mosquito population is divided into four compartments, the susceptible *Ae. aegypti* mosquito (\bar{S}_{m1}), the infected *Ae. aegypti* mosquito (\bar{I}_{m1}), the susceptible *Ae. albopictus* mosquito (\bar{S}_{m2}) and the infected *Ae. albopictus* mosquito (\bar{I}_{m2}) compartment, the recovered

compartments for the mosquito does not exist, since the mosquitoes do not recover after they are infected for over all their life.

The transmission dynamics of the Chikungunya fever are described by the following ordinary differential equations:

$$\frac{d\bar{S}_h}{dt} = \lambda_h N_h - (\gamma_{m1} \bar{I}_{m1} + \gamma_{m2} \bar{I}_{m2}) \bar{S}_h - \mu_h \bar{S}_h \quad (1a)$$

$$\frac{d\bar{I}_h}{dt} = (\gamma_{m1} \bar{I}_{m1} + \gamma_{m2} \bar{I}_{m2}) \bar{S}_h - (r_h + \mu_h) \bar{I}_h \quad (1b)$$

$$\frac{d\bar{R}_h}{dt} = r_h \bar{I}_h - \mu_h \bar{R}_h \quad (1c)$$

$$\frac{d\bar{S}_{m1}}{dt} = A_1 - \gamma_h \bar{I}_h \bar{S}_{m1} - \mu_{m1} \bar{S}_{m1} \quad (1d)$$

$$\frac{d\bar{I}_{m1}}{dt} = \gamma_h \bar{I}_h \bar{S}_{m1} - \mu_{m1} \bar{I}_{m1} \quad (1e)$$

$$\frac{d\bar{S}_{m2}}{dt} = A_2 - \gamma_h \bar{I}_h \bar{S}_{m2} - \mu_{m2} \bar{S}_{m2} \quad (1f)$$

$$\frac{d\bar{I}_{m2}}{dt} = \gamma_h \bar{I}_h \bar{S}_{m2} - \mu_{m2} \bar{I}_{m2} \quad (1g)$$

The Equation (1c, 1d and 1f) are dropped since it will be assumed that the human and mosquito population are constant. i.e., $\bar{R}_h = N_h - \bar{S}_h - \bar{I}_h$, $\bar{S}_{m1} = N_{m1} - \bar{I}_{m1}$ and $\bar{S}_{m2} = N_{m2} - \bar{I}_{m2}$. The number of dependent variables reduced to four, which we pick to be S_h, I_h, I_{m1}, I_{m2} .

To analyze the model by normalizing the Equation (1a-1g) and defining new variables Equation 2a-2d:

$$\begin{aligned} S_h &= \frac{\bar{S}_h}{N_h}, I_h = \frac{\bar{I}_h}{N_h}, R_h = \frac{\bar{R}_h}{N_h} \\ S_{m1} &= \frac{\bar{S}_{m1}}{N_{m1}} = \frac{\bar{S}_{m1}}{A_1 / (\mu_{m1})}, I_{m1} = \frac{\bar{I}_{m1}}{N_{m1}} = \frac{\bar{I}_{m1}}{A_1 / (\mu_{m1})} \\ S_{m2} &= \frac{\bar{S}_{m2}}{N_{m2}} = \frac{\bar{S}_{m2}}{A_2 / (\mu_{m2})}, I_{m2} = \frac{\bar{I}_{m2}}{N_{m2}} = \frac{\bar{I}_{m2}}{A_2 / (\mu_{m2})} \end{aligned} \quad (2a)$$

Since the total human and mosquito populations are constant, thus the time rate of change of human population equal to zero, i.e., $\frac{d\bar{S}_h}{dt} + \frac{d\bar{I}_h}{dt} + \frac{d\bar{R}_h}{dt} = 0$.

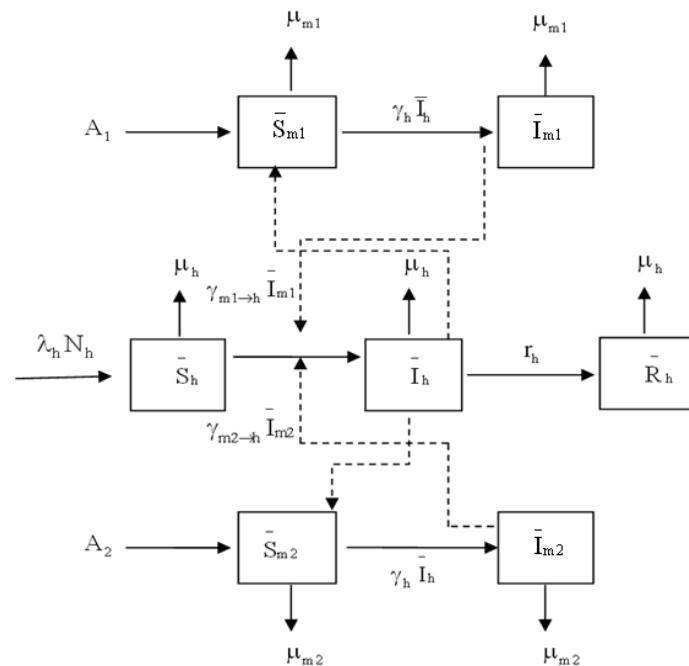


Fig. 1. Diagram for the dynamical transmission of Chikungunya fever

It means that the birth rate and the death rate of human population are equal, that is $\lambda_h = \mu_h$. The total number of *Ae. aegypti* mosquito and *Ae. albopictus* mosquito at equilibrium equal to $\frac{A_1}{\mu_{m1}}$ and $\frac{A_2}{\mu_{m2}}$, respectively.

where, N_h is the total human population:

- N_{m1} (N_{m2}) is the total *Ae. aegypti* (*Ae. albopictus*) mosquito population
- S_h , I_h , R_h is the number of susceptible, infected, recovered human population, respectively
- S_{m1} , (I_{m1}) is the number of susceptible (infected) *Ae. aegypti* mosquito population
- S_{m2} (I_{m2}) is the number of susceptible (infected) *Ae. albopictus* mosquito population
- λ_h (μ_h) is the birth (death) rate of human population
- A_1 (A_2) is the recruit rate of *Ae. aegypti* (*Ae. albopictus*) mosquito population
- $\gamma_{m1 \rightarrow h}$ ($\gamma_{m2 \rightarrow h}$) is the transmission rate of CHIKV from infected *Ae. aegypti* (*Ae. albopictus*) mosquito to human population
- r_h is the recovery rate of human population
- b is the biting rate of mosquito population
- $\beta_{h \rightarrow m1}$ ($\beta_{h \rightarrow m2}$) is the probability that CHIV will be transmitted from infected human population to *Ae. aegypti* (*Ae. albopictus*) mosquito population

- $\gamma_{h \rightarrow m1}$ is the transmission rate of CHIKV from infected human population to susceptible *Ae. aegypti* mosquito population, i.e.:

$$\gamma_{h \rightarrow m1} = \frac{b\beta_{h \rightarrow m1}}{N_m} \quad (2b)$$

$\gamma_{h \rightarrow m2}$ is the transmission rate of CHIKV from infected human population to susceptible *Ae. albopictus* mosquito population, i.e.:

$$\gamma_{h \rightarrow m2} = \frac{b\beta_{h \rightarrow m2}}{N_m} \quad (2c)$$

We assume that the transmission rate of CHIV from infected human population to both mosquitoes are equal, $\gamma_{h \rightarrow m1} = \gamma_{h \rightarrow m2} = \gamma_h$ and:

$$N_m = N_{m1} + N_{m2} \quad (2d)$$

μ_{m1} (μ_{m2}) is the death rate of *Ae. aegypti* (*Ae. albopictus*) mosquito population.

The reduced model is depicted as following:

$$\begin{aligned} \frac{dS_h}{dt} = & \mu_h(1 - S_h) - \gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})I_{m1}S_h \\ & - \gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})I_{m2}S_h \end{aligned} \quad (3)$$

$$\frac{dI_h}{dt} = \gamma_{m1 \rightarrow h} (A_1 / \mu_{m1}) I_{m1} S_h + \gamma_{m2 \rightarrow h} (A_2 / \mu_{m2}) I_{m2} S_h - (r_h + \mu_h) I_h \quad (4)$$

$$\frac{dI_{m1}}{dt} = \gamma_h N_h I_h (1 - I_{m1}) - \mu_{m1} I_{m1} \quad (5)$$

$$\frac{dI_{m2}}{dt} = \gamma_h N_h I_h (1 - I_{m2}) - \mu_{m2} I_{m2} \quad (6)$$

2.2. Analysis of the Model

2.2.1. Equilibrium Points

We first determine the equilibrium points and investigate their stability. It is found that the system has two possible equilibrium points: the disease free equilibrium point and an endemic equilibrium point. Two equilibrium points are found by setting the RHS of Equation 3-6 to zero. Doing this, we obtained.

2.3. Disease Free Equilibrium Point (E_0)

In the absence of disease, that is $I_h = 0$, $I_{m1} = 0$, $I_{m2} = 0$. Equation 3 reduces to:

$$\frac{dS_h}{dt} = \mu_h (1 - S_h)$$

The solution of this equation is $S_h = 1$. The disease free state becomes $E_0 = (1, 0, 0, 0)$.

2.4. Endemic Equilibrium Point (E_1)

In the case where the disease is presented, we will have $I_h \neq 0$, $I_{m1} \neq 0$, $I_{m2} \neq 0$. This gives:

$$I_h^* = \frac{M_2 P_1 + M_1 P_2}{P_1 + P_2} \quad (7)$$

$$I_{m1}^* = \frac{M_1 P_2 + M_2 P_1}{M_1 P_2 + M_2 P_1 + M_1 (P_1 + P_2)} \quad (8)$$

$$I_{m2}^* = \frac{M_1 P_2 + M_2 P_1}{M_1 P_2 + M_2 P_1 + M_2 (P_1 + P_2)} \quad (9)$$

$$S_h^* = \frac{M_{11} M_{22}}{N_{11} M_{22} + N_{22} M_{11} + M_{11} M_{22}} \quad (10)$$

With:

$$P_1 = \frac{\gamma_{m1 \rightarrow h} A_1}{\mu_h \mu_{m1}}, P_2 = \frac{\gamma_{m2 \rightarrow h} A_2}{\mu_h \mu_{m2}}$$

$$M_1 = \frac{\mu_{m1 \rightarrow h}}{\gamma_h N_h}, M_2 = \frac{\mu_{m2 \rightarrow h}}{\gamma_h N_h}, M_{11} = M_1 P_2 + M_2 P_1 + M_1 (P_1 + P_2)$$

$$M_{22} = M_1 P_2 + M_2 P_1 + M_2 (P_1 + P_2), N_{11} = P_1 (M_1 P_2 + M_2 P_1)$$

And:

$$N_{22} = P_2 (M_1 P_2 + M_2 P_1)$$

The endemic equilibrium point becomes $E_1(S_h^*, I_h^*, I_{m1}^*, I_{m2}^*)$.

2.5. Basic Reproductive Number

The basic reproductive number is obtained by the next generation method. In the notations of Driessche and Watmough (2002), we start with:

$$\frac{dX}{dt} = \Pi_i - \Omega_i$$

where, Π_i is the matrix of new infectious and Ω_i is the matrix for the transfers between the compartments in the infective equations. Specifically, we have:

$$X = \begin{bmatrix} S_h \\ I_h \\ I_{m1} \\ I_{m2} \end{bmatrix}, \Pi_i = \begin{bmatrix} 0 & \gamma_{m1 \rightarrow h} (A_1 / \mu_{m1}) S_h I_{m1} + \gamma_{m2 \rightarrow h} (A_2 / \mu_{m2}) S_h I_{m2} \\ 0 & 0 \\ 0 & 0 \end{bmatrix}$$

And:

$$V_i = \begin{bmatrix} -\mu_h + \gamma_{m1 \rightarrow h} (A_1 / \mu_{m1}) S_h I_{m1} + \gamma_{m2 \rightarrow h} (A_2 / \mu_{m2}) S_h I_{m2} + \mu_h S_h \\ (r_h + \mu_h) I_h \\ -\gamma_h N_h I_h + \gamma_h N_h I_h I_{m1} + \mu_{m1} I_{m1} \\ -\gamma_h N_h I_h + \gamma_h N_h I_h I_{m2} + \mu_{m2} I_{m2} \end{bmatrix}$$

where, $F = \left[\frac{\partial \Pi_i(E_0)}{\partial X_j} \right]$ and $V = \left[\frac{\partial \Omega_i(E_0)}{\partial X_j} \right]$ for all $i, j = 1, 2$,

3, 4 are the Jacobian matrix of Π and Ω at E_0 . The basic reproductive number, R_0 , is the threshold for the stability of the disease free equilibrium E_0 . It can be calculated by noting that:

$$R_0 = \rho(FV^{-1})$$

where, FV^{-1} is called the next generation matrix and $\rho(FV^{-1})$ is the spectral radius (dominant eigenvalue) of the matrix FV^{-1} . For our model, the Jacobian matrices are:

$$F(E_0) = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_{m1 \rightarrow h} (A_1 / \mu_{m1}) & \gamma_{m2 \rightarrow h} (A_2 / \mu_{m2}) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

And:

$$V(E_0) = \begin{bmatrix} \mu_h & 0 & \gamma_{m1 \rightarrow h}(A_1 / \mu_{m1}) & \gamma_{m2 \rightarrow h}(A_2 / \mu_{m2}) \\ 0 & r_h + \mu_h & 0 & 0 \\ 0 & -\gamma_h N_h & \mu_{m1} & 0 \\ 0 & -\gamma_h N_h & 0 & \mu_{m2} \end{bmatrix}$$

The inverse of V is:

$$V^{-1}(E_0) = \begin{bmatrix} \frac{1}{\mu_h} & -\frac{\gamma_h N_h \gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})}{\mu_h(r_h + \mu_h)\mu_{m1}} & -\frac{\gamma_h N_h \gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})}{\mu_h(r_h + \mu_h)\mu_{m2}} & \frac{\gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})}{\mu_h \mu_{m1}} & -\frac{\gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})}{\mu_h \mu_{m2}} \\ 0 & \frac{1}{r_h + \mu_h} & 0 & 0 & 0 \\ 0 & \frac{\gamma_h N_h}{\mu_{m1}(r_h + \mu_h)} & \frac{1}{\mu_{m1}} & 0 & 0 \\ 0 & -\frac{\gamma_h N_h}{\mu_{m2}(r_h + \mu_h)} & 0 & \frac{1}{\mu_{m2}} & 0 \end{bmatrix}$$

This leads to:

$$FV^{-1}(E_0) = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & -\frac{\gamma_h N_h \gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})}{(r_h + \mu_h)\mu_{m1}} & -\frac{\gamma_h N_h \gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})}{(r_h + \mu_h)\mu_{m2}} & \frac{\gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})}{\mu_{m1}} & \frac{\gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})}{\mu_{m2}} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

Thus:

$$\rho(FV^{-1}(E_0)) = \frac{\gamma_h N_h \gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})}{(r_h + \mu_h)\mu_{m1}} + \frac{\gamma_h N_h \gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})}{(r_h + \mu_h)\mu_{m2}}$$

And so:

$$R_0 = \frac{\gamma_h N_h \gamma_{m1 \rightarrow h}(A_1 / \mu_{m1})}{(r_h + \mu_h)\mu_{m1}} + \frac{\gamma_h N_h \gamma_{m2 \rightarrow h}(A_2 / \mu_{m2})}{(r_h + \mu_h)\mu_{m2}}$$

2.6. Local Asymptotical Stability

The local stability of an equilibrium point is determined from the Jacobian matrix of the system of ordinary differential Equation 3-6 evaluated at each equilibrium point. The Jacobian matrix at the disease free state E_0 is Equation 11:

$$J_0 = \begin{bmatrix} -\mu_h & 0 & -\frac{\gamma_{m1 \rightarrow h} A_1}{\mu_{m1}} & -\frac{\gamma_{m2 \rightarrow h} A_2}{\mu_{m2}} \\ 0 & -(r_h + \mu_h) & \frac{\gamma_{m1 \rightarrow h} A_1}{\mu_{m1}} & \frac{\gamma_{m2 \rightarrow h} A_2}{\mu_{m2}} \\ 0 & \gamma_h N_h & -\mu_{m1} & 0 \\ 0 & \gamma_h N_h & 0 & -\mu_{m2} \end{bmatrix} \quad (11)$$

The eigenvalues of the J_0 are obtained by solving $\det(J_0 - \lambda I) = 0$. We obtained the characteristic Equation 12:

$$(\lambda + \mu_h)(\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3) = 0 \quad (12)$$

Where:

$$\begin{aligned} a_1 &= r_h + \mu_h + \mu_{m1} + \mu_{m2}, \\ a_2 &= (r_h + \mu_h)(\mu_{m1} + \mu_{m2}) + \mu_{m1} \mu_{m2} - \frac{\gamma_h N_h \gamma_{m1 \rightarrow h} A_1}{\mu_{m1}} \\ &\quad - \frac{\gamma_h N_h \gamma_{m2 \rightarrow h} A_2}{\mu_{m2}}, \\ a_3 &= (r_h + \mu_h)(\mu_{m1} \mu_{m2}) - \frac{\gamma_h N_h \gamma_{m1 \rightarrow h} A_1 \mu_{m2}}{\mu_{m1}} \\ &\quad - \frac{\gamma_h N_h \gamma_{m2 \rightarrow h} A_2 \mu_{m1}}{\mu_{m2}} \end{aligned} \quad (13)$$

One of eigenvalues is $\lambda_1 = -\mu_h < 0$. The other three eigenvalues are the solutions of the cubic equation $\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0$. The roots of this equation are negative if the coefficients satisfied the three conditions of Routh-Hurwitz criteria (Allen, 2006). E_0 will be locally asymptotically stable when the coefficients satisfy the conditions $a_1 > 0$ Equation 14:

$$\begin{aligned} a_3 &> 0 \\ a_1 a_2 &> a_3 \end{aligned} \quad (14)$$

Looking at the coefficients, Equation 13, we see that a_1 is always positive and a_3 will be positive when Equation 15:

$$\frac{\gamma_h N_h \gamma_{m1 \rightarrow h} A_1 \mu_{m2}}{\mu_{m1}} + \frac{\gamma_h N_h \gamma_{m2 \rightarrow h} A_2 \mu_{m1}}{\mu_{m2}} < (r_h + \mu_h)(\mu_{m1} \mu_{m2}) \quad (15)$$

Thus, the disease free equilibrium point will be locally asymptotically stable when $R_0 < 1$.

Where Equation 16:

$$\begin{aligned} R_0 &= R_{01} + R_{02} \\ R_{01} &= \frac{\gamma_h N_h \gamma_{m1 \rightarrow h} A_1}{(r_h + \mu_h)(\mu_{m1} \mu_{m1})}, R_{02} = \frac{\gamma_h N_h \gamma_{m2 \rightarrow h} A_2}{(r_h + \mu_h)(\mu_{m2} \mu_{m2})} \end{aligned} \quad (16)$$

2.6. Disease Endemic Equilibrium Point

To determine the stability of the endemic equilibrium point, E_1 , we first evaluate the Jacobian matrix at E_1 to get Equation 17:

$$J_1 = \begin{bmatrix} -\mu_h - \frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} - \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}} & 0 & \frac{\gamma_{m1 \rightarrow h} A_1 S_h^*}{\mu_{m1}} & \frac{\gamma_{m2 \rightarrow h} A_2 S_h^*}{\mu_{m2}} \\ \frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}} & -(r + \mu_h) & \frac{\gamma_{m1 \rightarrow h} A_1 S_h^*}{\mu_{m1}} & \frac{\gamma_{m2 \rightarrow h} A_2 S_h^*}{\mu_{m2}} \\ 0 & \gamma_h N_h - \gamma_h N_h I_{m1}^* & -\gamma_h N_h I_{m1}^* - \mu_{m1} & 0 \\ 0 & \gamma_h N_h - \gamma_h N_h I_{m2}^* & 0 & -\gamma_h N_h I_{m2}^* - \mu_{m2} \end{bmatrix} \quad (17)$$

where, $S_h^*, I_h^*, I_{m1}^*, I_{m2}^*$ are given by Equation 7-10. The characteristic equation of Jacobian matrix at E_1 , becomes Equation 18:

$$\lambda^4 + b_1 \lambda^3 + b_2 \lambda^2 + b_3 \lambda + b_4 = 0 \quad (18)$$

Where Equation 19:

$$\begin{aligned} b_1 &= \mu_{m1} + \mu_{m2} + r_h + 2\mu_h + 2\gamma_h N_h I_h^* + \frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}}, \\ b_2 &= (\mu_{m2} + \gamma_h N_h I_h^*)(\mu_{m1} + r_h + \mu_h + \gamma_h N_h I_h^*) + (r_h + \mu_h)(\mu_{m1} + \gamma_h N_h I_h^*) - (\gamma_h N_h)(1 - I_{m1}^*) \left(\frac{\gamma_{m1 \rightarrow h} A_1}{\mu_{m1}} S_h^* \right) \\ &\quad - (\gamma_h N_h)(1 - I_{m2}^*) \left(\frac{\gamma_{m2 \rightarrow h} A_2}{\mu_{m2}} S_h^* \right) + (\mu_h + \frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}}) [\mu_{m1} + \mu_{m2} + r_h + \mu_h + 2\gamma_h N_h I_h^*] \\ b_3 &= (r_h + \mu_h)(\mu_{m1} + \gamma_h N_h I_h^*)(\mu_{m2} + r_h N_h I_h^*) - (\mu_{m1} + \gamma_h N_h I_h^*)(\gamma_h N_h)(1 - I_{m2}^*) \left(\frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}} \right) \\ &\quad - (\mu_{m2} + \gamma_h N_h I_h^*)(\gamma_h N_h)(1 - I_{m1}^*) \left(\frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} \right) - (\mu_h + \frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}}) \times \\ &\quad \{ -(\mu_{m2} + \gamma_h N_h I_h^*)(\mu_{m1} + r_h + \mu_h + \gamma_h N_h I_h^*) - (r_h + \mu_h)(\mu_{m1} + \gamma_h N_h I_h^*) + (\gamma_h N_h)[(1 - I_{m2}^*) \left(\frac{\gamma_{m2 \rightarrow h} A_2 S_h^*}{\mu_{m2}} \right) + (1 - I_{m1}^*) \left(\frac{\gamma_{m1 \rightarrow h} A_1 S_h^*}{\mu_{m1}} \right)] \} \\ &\quad - (\frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}})(\gamma_h N_h)[(1 - I_{m2}^*) \left(\frac{\gamma_{m2 \rightarrow h} A_2 S_h^*}{\mu_{m2}} \right) + (1 - I_{m1}^*) \left(\frac{\gamma_{m1 \rightarrow h} A_1 S_h^*}{\mu_{m1}} \right)] \\ b_4 &= -(\mu_h + \frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}}) \\ &\quad \{ -(r_h + \mu_h)(\mu_{m1} + \gamma_h N_h I_h^*)(\mu_{m2} + \gamma_h N_h I_h^*) + \gamma_h N_h S_h^*[(1 - I_{m1}^*)(\mu_{m2} + \gamma_h N_h I_h^*) \left(\frac{\gamma_{m1 \rightarrow h} A_1}{\mu_{m1}} \right) + (1 - I_{m2}^*)(\mu_{m1} + \gamma_h N_h I_h^*) \left(\frac{\gamma_{m2 \rightarrow h} A_2}{\mu_{m2}} \right)] \} \\ &\quad - (\frac{\gamma_{m1 \rightarrow h} A_1 I_{m1}^*}{\mu_{m1}} + \frac{\gamma_{m2 \rightarrow h} A_2 I_{m2}^*}{\mu_{m2}})(\gamma_h N_h S_h^*)[(1 - I_{m1}^*)(\mu_{m2} + \gamma_h N_h I_h^*) \left(\frac{\gamma_{m1 \rightarrow h} A_1}{\mu_{m1}} \right) + (1 - I_{m2}^*)(\mu_{m1} + \gamma_h N_h I_h^*) \left(\frac{\gamma_{m2 \rightarrow h} A_2}{\mu_{m2}} \right)] \end{aligned} \quad (19)$$

The four eigenvalues of $\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4 = 0$ will have negative real part if they satisfy the Routh-Hurwitz criteria. Thus, E_1 will be locally asymptotically stable for $R_0 > 1$ when:

$$\begin{aligned} b_1 &> 0 \\ b_3 &> 0 \\ b_4 &> 0 \\ b_1b_2b_3 &> b_3^2 + b_1^2b_4 \end{aligned} \quad (20)$$

$$\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4 = 0$$

Satisfies the following conditions Equation 20:

3. RESULTS

The parameters used in the numerical simulation are given in **Table 1**.

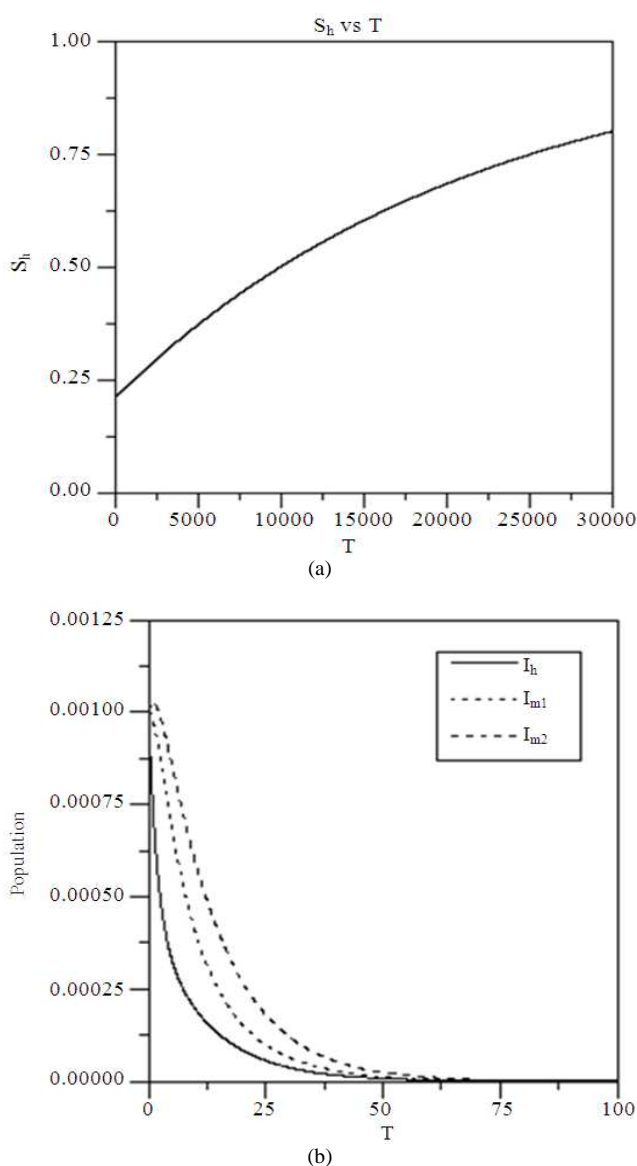


Fig. 2. Time series of (a) susceptible human (S_h); (b) infected human (I_h), infected *Ae. aegypti* mosquito (I_{m1}), infected *Ae. albopictus* mosquito (I_{m2}) proportions. The values of parameters are in the text and $\lambda_1 = -0.628528$, $\lambda_2 = -0.186016$, $\lambda_3 = -0.0283587$, $\lambda_4 = -0.00457$, $R_0 = 0.000548521 < 1$. We see that the solutions converge to the disease free equilibrium state as shown

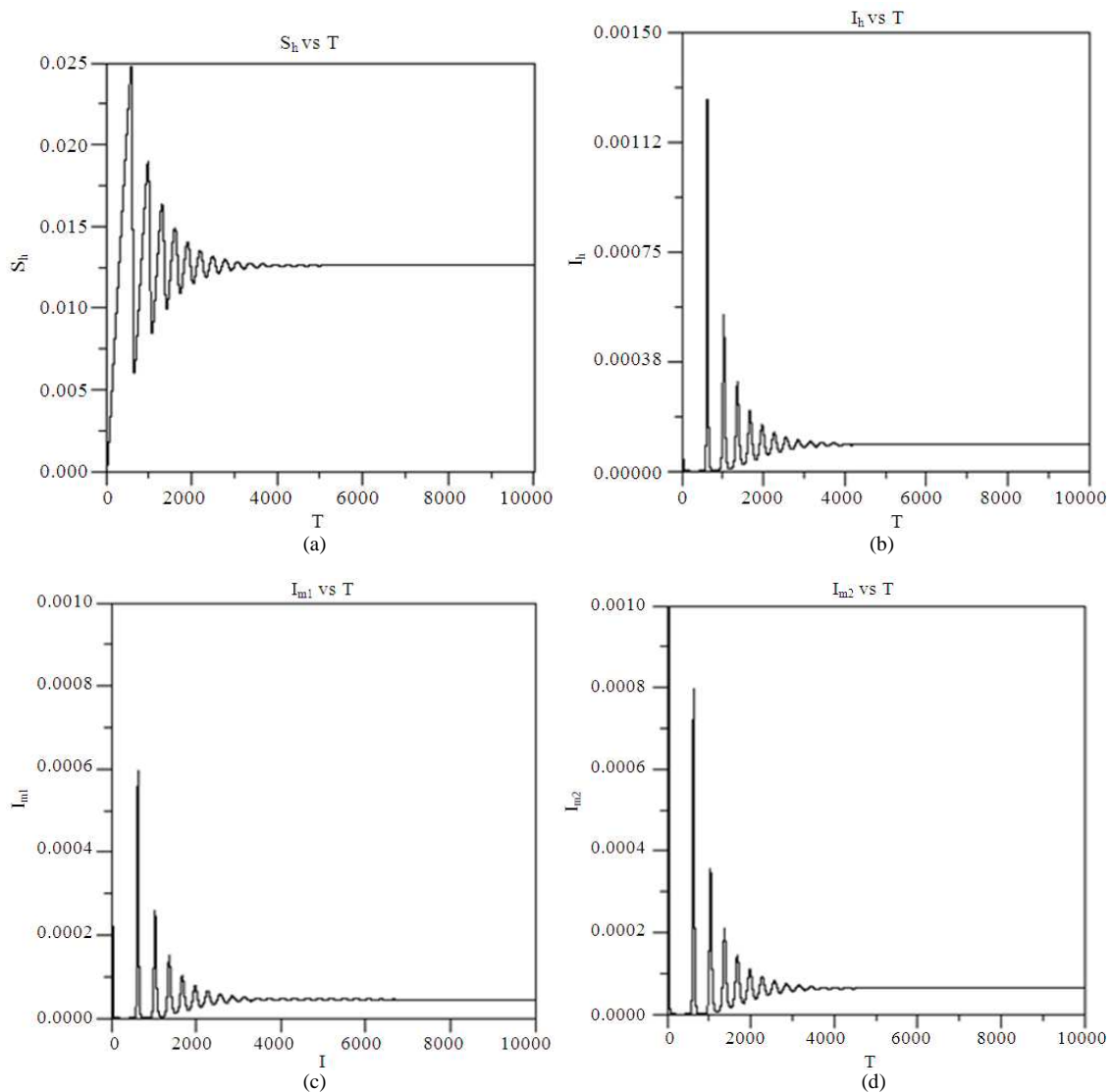


Fig. 3. Time series of (a) susceptible human, (b) infected human, (c) infected *Ae. aegypti* mosquito and (d) infected *Ae. albopictus* mosquito. The values of parameters are in the text, the solutions converge to the endemic equilibrium state E_1 as shown

Table 1. Parameter values used in numerical simulations at disease free state

Parameters	Descriptions	Values
μ_h	Birth (death) rate of human	$1/(65 \times 365) \text{ day}^{-1}$
N_h	Total number of human population	20000
A_1	Recruitment rate of <i>Ae. aegypti</i> mosquito	90 day^{-1}
A_2	Recruitment rate of <i>Ae. albopictus</i> mosquito	100 day^{-1}
$\gamma_{m1 \rightarrow h}$	Transmission rate of CHIKV from <i>Ae. aegypti</i> to human	0.0002 day^{-1}
$\gamma_{m2 \rightarrow h}$	Transmission rate of CHIKV from <i>Ae. albopictus</i> to human	0.003 day^{-1}
γ_h	Transmission rate of CHIKV from human to mosquito	0.00001 day^{-1}
r_h	Recovery rate of human	$1/2 \text{ day}^{-1}$
μ_{m1}	Death rate of <i>Ae. aegypti</i> mosquito	$1/5 \text{ day}^{-1}$
μ_{m2}	Death rate of <i>Ae. albopictus</i> mosquito	$1/7 \text{ day}^{-1}$
b	Biting rate of mosquito	1 day^{-1}

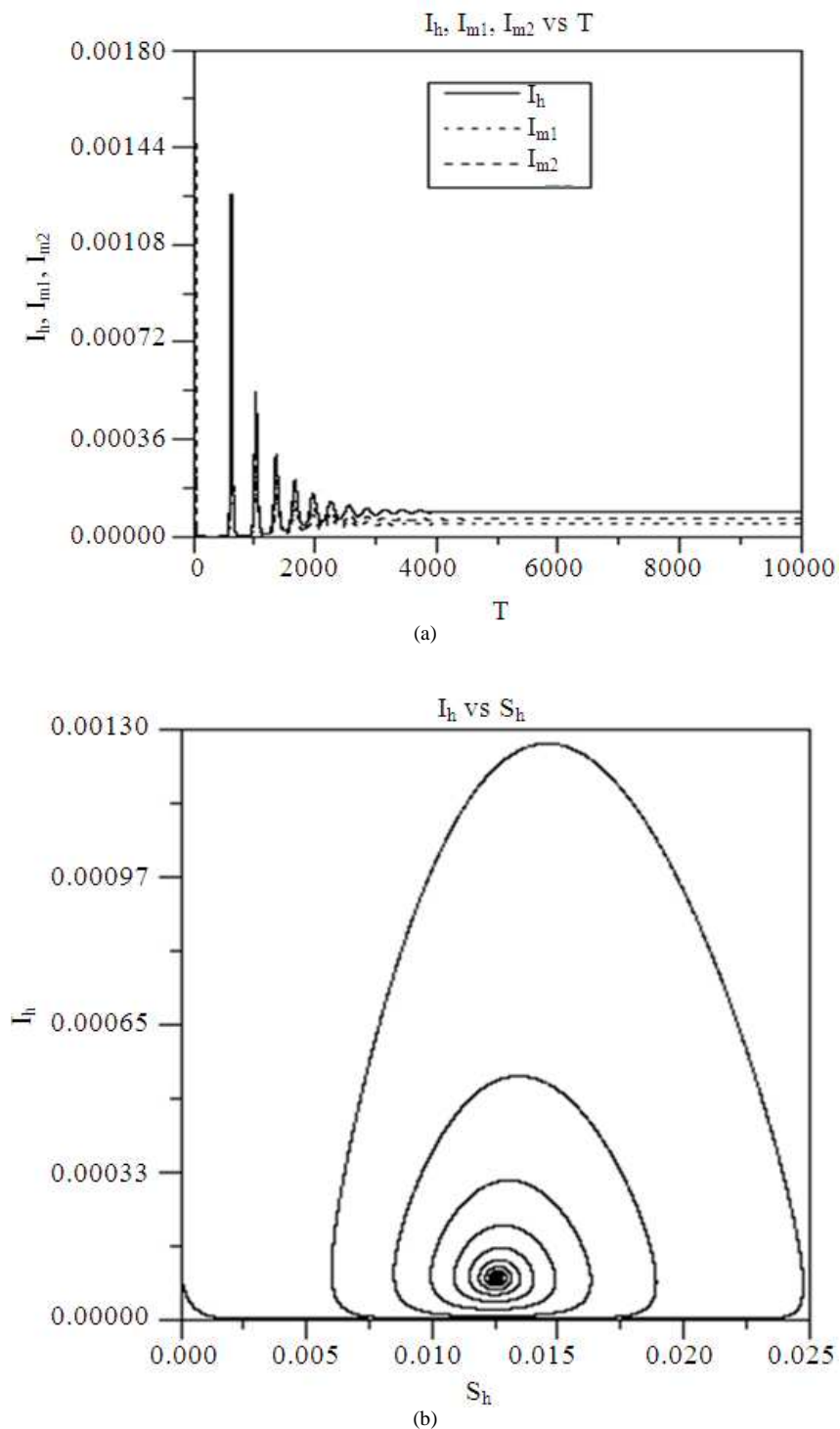


Fig. 4. (a) Time series of infected human, infected *Ae. aegypti* mosquito and infected *Ae. albopictus* mosquito, proportions. (b) Phase portrait of (S_h-I_h) plan with parameters as given in text, the trajectory approach to the endemic state E_1 as shown

3.1. Stability of Disease Free State

Using the values of parameters listed in **Table 1**, we find the eigenvalues and basic reproductive number to be:

$$\lambda_1 = -0.628528, \lambda_2 = -0.186016, \lambda_3 = -0.0283587, \\ \lambda_4 = -0.00457, R_0 = 0.000548521 < 1$$

Since all of these eigenvalues are to be negative and the basic reproductive number is less than one, the equilibrium state will be the disease free state, E_0 as is shown in **Fig. 2**.

3.2. Stability of Endemic State

We change the value of the recruitment rate of mosquito to $A_1 = 5,000$, $A_2 = 10,000$ and keep the other values of parameters to be those given in **Table 1**, we find the eigenvalues to be $\lambda_1 = -0.6285525$, $\lambda_2 = -0.186016$, $\lambda_3 = -0.0283579$, $\lambda_4 = -0.004557$ and the basic reproductive number to be $R_0 = 4571.01 > 1$. Since all of these eigenvalues are to be negative and the basic reproductive number to be greater than one, the equilibrium endemic state will be locally asymptotically stable E_1 as seen in **Fig. 3**.

4. DISSCUSSION

We formulated the transmission model of Chikungunya fever by taking into account the presence of two species of *Aedes* mosquitoes. We found two equilibrium points: Disease free state and endemic disease state. In disease free state, it will be local stability when $R_0 < 1$ and the endemic disease state will be local stability when $R_0 > 1$. The basic reproductive number is:

$$R_o = R_{o1} + R_{o2} = \frac{\gamma_h N_h \gamma_{m1 \rightarrow h} A_1}{(r_h + \mu_h)(\mu_{m1})} + \frac{\gamma_h N_h \gamma_{m2 \rightarrow h} A_2}{(r_h + \mu_h)(\mu_{m2})}$$

It represents the number of secondary case that one case can produce.

5. CONCLUSION

In this study, we have analyzed a transmission model for Chikungunya fever in which two species of mosquitoes are present. This is important to the modeling of this disease in Thailand since both the *Ae. aegypti* and *Ae. albopictus* mosquitoes are present. In most other countries, only one specie of the *Aedes*

mosquito is present. Again in Thailand, it was found that both species can inhabit the same breeding sites. By reducing the number of the breeding sites, the recruitment rates of both *Aedes* mosquitoes would be reduced. From **Fig. 2**, changing the recruitment rate A_1 and A_2 to 5,000 and 10,000, respectively, we find that the time behaviors of the infected human, the *Ae. aegypti* and the *Ae. albopictus* mosquitoes decrease sinsodially to the endemic state (**Fig. 4a**). **Figure 4b** shows the trajectory of the human population in the S_h - I_h phase space. The changes seen in the time behaviors shown in **Fig. 3** and **4** show that the destruction of the breeding cause equilibrium state to change from being an endemic equilibrium state to a disease free equilibrium state.

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