Dynamical Transmission Model of Chikungunya in Thailand

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Abstract**—**One of the important tropical diseases is Chikunkunya. This disease is transmitted between the human by the insect-borne virus, of the genus *Alphavirus.* It occurs in Africa, Asia and the Indian subcontinent. In Thailand, the incidences due to this disease are increasing every year. In this study, the transmission of this disease is studied through dynamical model analysis.

Keywords**—**Chikunkunya, dynamical model, Endemic region, Routh-Hurwitz criteria.

I. Introduction

MATHEMATICS have become a major tool for studying the evolution of vector-borne diseases [1-3]. For instance, several temporal deterministic models have been proposed for dengue [4-6], but, to our knowledge, only very few for Chikungunya [7].

Chikungunya is caused by the chikungunya virus, which is classified in the family Togaviridae, genus *Alphavirus*. It is spread by the biting of the infected Aedes mosquito. Most commonly, the mosquitoes involved are *Aedes aegypti* and *Aedes albopictus*. Humans are thought to be the major source, or reservoir, of chikungunya virus for mosquitoes. Therefore, the mosquito usually transmits the disease by biting an infected person and then biting someone else. An infected person cannot spread the infection directly to other persons.

Chikungunya is characterized by an abrupt onset of fever frequently accompanied by joint pain. Other common signs and symptoms include muscle pain, headache, nausea, fatigue and rash. The joint pain is often very debilitating, but usually ends within a few days or weeks. Most patients recover fully, but in some cases joint pain may persist for several months, or even years.

There is no vaccine or specific antiviral treatment currently available for chikungunya fever. Treatment is applied to the symptomatic patients and can include rest, fluids, and medicines to relieve symptoms of fever and aching such as ibuprofen, naproxen, acetaminophen, or paracetamol.

Chikungunya virus was first isolated in Tanzania during a 1952 to 1953 epidemic [8]. The first appearance of the virus in Southeast Asia by isolation during an intense epidemic of dengue fever and dengue hemorrhagic fever in Bangkok, Thailand, in 1958 [9]. Clinically, the disease can resemble classical dengue fever and in dengue endemic countries, this can give rise to confusion and misdiagnosis. The virus continued to be active in Thailand until the 1970s, after which it almost disappeared.

In 1988 evidence of chikungunya transmission in Thailand re-emerged, but the pattern was one of occasional outbreaks rather than severe epidemic disease. In 2008, a total of 2,233 cases of chikungunya fever were reported from 4 provinces of southern Thailand, Narathiwat, Pattani, Yala and Songkhla. The reported cases of chikungunya fever increased gradually week by week until the end of 2008 with the highest incidence of about 400 cases per week [10].

Studying the mathematical model for Chikunkunya began in 1970 [11]. After that, there is no study about the Chikunkunya model. Because there is the re-emergence of this disease in 2007, the study of the model for this disease is started again. N.Bacaer [12] studied the basic reproductive number of this disease by considering the mosquito population. In 2008, Y. Dumont and F. Chiroleu [13] formulated the mathematical model for human and mosquito population and find the stability condition for the disease free and endemic state.

The abundance of the mosquitoes in Thailand depends on the season and the temperature of the environment. In this paper, we formulate and analyze the Chikunkunya model by incorporating the effect of the season which effect to the number of the mosquitoes in Thailand.

II. Mathematical Model

Our model classifies the human into three epidemiological states:

S_h is the number of susceptible human,

I_h is the number of infectious human,

R_h is the number of recovered human.

We assume that the total human population (N_T) is constant, and $N_T = S_h + I_h + R_h$.

The mosquitoes are classified in three classes:

S_{vi} is the number of susceptible vector in i season,

E_{vi} is the number of exposed vector in i season,

I_{vi} is the number of infectious vector in i season,

when $i = 1, 2, 3$ if

$i = 1$ means winter season ,

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$i = 2$ means rainy season ,
$i = 3$ means summer season.
We obtain the following system equations

$$\frac{dS_h}{dt} = \lambda N_T - (\mu_h + \gamma_h I_v)S_h,$$
$$\frac{dI_h}{dt} = \gamma_h S_h I_v - (\mu_h + r)I_h,$$
$$\frac{dR_h}{dt} = rI_h - \mu_h R_h, \quad (1)$$
$$\frac{dS_{vi}}{dt} = A_i - (\gamma_v I_h + \mu_v)S_{vi},$$
$$\frac{dE_{vi}}{dt} = \gamma_v S_{vi} I_h - (\mu_v + q)E_{vi},$$
$$\frac{dI_{vi}}{dt} = qE_{vi} - \mu_v I_{vi}$$

where $I_v = I_{v1} + I_{v2} + I_{v3}$

with

λ is the birth rate of the human population,
μ_h is the death rate of the human population,
γ_h is the transmission rate from vector to human population,
r is the recoverey rate of the human population,
A_i is the constant recruitment rate of the vector population at the i^{th} season,
μ_v is the death rate of the vector population,
γ_v is the transmission rate from human to vector population,
q is the rate at which the exposed vector change to be the infectious vector.

Using the fact $N_T = S_h + I_h + R_h$, $N_{v1} = S_{v1} + E_{v1} + I_{v1}$, $N_{v2} = S_{v2} + E_{v2} + I_{v2}$ and $N_{v3} = S_{v3} + E_{v3} + I_{v3}$ are constants. We let $\overline{S_h} = \frac{S_h}{N_T}$, $\overline{I_h} = \frac{I_h}{N_T}$, $\overline{R_h} = \frac{R_h}{N_T}$,

$$\overline{S_{v1}} = \frac{S_{v1}}{N_{v1}}, \ \overline{E_{v1}} = \frac{E_{v1}}{N_{v1}}, \ \overline{R_{v1}} = \frac{R_{v1}}{N_{v1}}, \ \overline{S_{v2}} = \frac{S_{v2}}{N_{v2}}$$
$$\overline{E_{v2}} = \frac{E_{v2}}{N_{v2}}, \overline{R_{v2}} = \frac{R_{v2}}{N_{v2}}, \ \overline{S_{v3}} = \frac{S_{v3}}{N_{v3}}, \ \overline{E_{v3}} = \frac{E_{v3}}{N_{v3}} \text{ and}$$
$$\overline{R_{v3}} = \frac{R_{v3}}{N_{v3}}. \quad (2)$$

The total human population and the numbers of vector in i^{th} season are assumed to be constant, then the rates of change for all population equal to zero. From setting the rate of change for each population equals to zero, we obtained

$\lambda = \mu_h$, $N_{v1} = A_1/\mu_v$, $N_{v2} = A_2/\mu_v$ and $N_{v3} = A_3/\mu_v$.

Then the normalized equations become:

$$\frac{d\overline{S_h}}{dt} = \mu_h - (\mu_h + \gamma_h(\overline{I_{v1}}N_{v1} + \overline{I_{v2}}N_{v2} + \overline{I_{v3}}N_{v3}))\overline{S_h},$$
$$\frac{d\overline{I_h}}{dt} = \gamma_h \overline{S_h}(\overline{I_{v1}}N_{v1} + \overline{I_{v2}}N_{v2} + \overline{I_{v3}}N_{v3}) - (\mu_h + r)\overline{I_h},$$
$$\frac{d\overline{E_{v1}}}{dt} = \gamma_v(1 - \overline{E_{v1}} - \overline{I_{v1}})\overline{I_h}N_T - (\mu_v + q)\overline{E_{v1}},$$
$$\frac{d\overline{I_{v1}}}{dt} = q\overline{E_{v1}} - \mu_v \overline{I_{v1}}, \quad (3)$$
$$\frac{d\overline{E_{v2}}}{dt} = \gamma_v(1 - \overline{E_{v2}} - \overline{I_{v2}})\overline{I_h}N_T - (\mu_v + q)\overline{E_{v2}},$$
$$\frac{d\overline{I_{v2}}}{dt} = q\overline{E_{v2}} - \mu_v \overline{I_{v2}},$$
$$\frac{d\overline{E_{v3}}}{dt} = \gamma_v(1 - \overline{E_{v3}} - \overline{I_{v3}})\overline{I_h}N_T - (\mu_v + q)\overline{E_{v3}},$$
$$\frac{d\overline{I_{v3}}}{dt} = q\overline{E_{v3}} - \mu_v \overline{I_{v3}}$$

where

$\overline{R_h} = 1 - \overline{S}h - \overline{I}h$, $\overline{S_{v1}} = 1 - \overline{E_{v1}} - \overline{I_{v1}}$, $\overline{S_{v2}} = 1 - \overline{E_{v2}} - \overline{I_{v2}}$ and $\overline{S_{v3}} = 1 - \overline{E_{v3}} - \overline{I_{v3}}$. (4)

III. ANALYSIS OF THE MODEL

A. Analytical Solutions

The equilibrium states $(S_h^*, I_h^*, E_{v1}^*, I_{v1}^*, E_{v2}^*, I_{v2}^*, E_{v3}^*, I_{v3}^*)$ are found by setting the right hand side of (3) to zero, then we obtain: $E_1 = (1,0,0,0,0,0,0,0)$ as the disease free equilibrium state and $E_2 = (S_h^*, I_h^*, E_{v1}^*, I_{v1}^*, E_{v2}^*, I_{v2}^*, E_{v3}^*, I_{v3}^*)$ as the endemic equilibrium state:
where

$$S_h^* = \frac{1}{1 + \beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*},$$
$$I_h^* = \frac{(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)}{\sigma_1(1 + \beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)},$$
$$E_{v1}^* = \frac{(1 - I_{v1}^*)(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)}{(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)(1 + \beta_4\sigma_1) + \beta_4\sigma_1},$$
$$I_{v1}^* = \frac{q(1 - I_{v1}^*)(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)}{\mu_v((\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)(1 + \beta_4\sigma_1) + \beta_4\sigma_1)},$$
$$E_{v2}^* = \frac{(1 - I_{v2}^*)(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)}{(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)(1 + \beta_4\sigma_1) + \beta_4\sigma_1},$$
$$I_{v2}^* = \frac{q(1 - I_{v2}^*)(\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)}{\mu_v((\beta_1 I_{v1}^* + \beta_2 I_{v2}^* + \beta_3 I_{v3}^*)(1 + \beta_4\sigma_1) + \beta_4\sigma_1)},$$

$$E^*_{v3} = \frac{(1-I^*_{v3})(\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3})}{(\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3})(1+\beta_4\sigma_1) + \beta_4\sigma_1},$$

$$I^*_{v3} = \frac{q(1-I^*_{v3})(\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3})}{\mu_v((\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3})(1+\beta_4\sigma_1) + \beta_4\sigma_1)},$$

where

$$\beta_1 = \frac{\gamma_h N_{v1}}{\mu_h},\ \beta_2 = \frac{\gamma_h N_{v2}}{\mu_h},\ \beta_3 = \frac{\gamma_h N_{v3}}{\mu_h}, \beta_4 = \frac{\mu_v + q}{\gamma_v N_T}$$

and $\sigma_1 = \frac{\mu_h + r}{\mu_h}$.

The characteristic equation of (3) for the disease free equilibrium state is

$$(\mu_h+\lambda)(\mu_v+\lambda)^2(\beta_4\mu_v N_T+\lambda)^2(\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0) = 0$$

where

$$a_2 = \sigma_1\mu_h + (1+N_T\beta_4)\mu_v,$$

$$a_1 = \mu_v((1+N_T\beta_4)\sigma_1\mu_h + N_T\beta_4\mu_v),$$

$$a_0 = N_T\mu_h(-q(\beta_1+\beta_2+\beta_3)\gamma_v + \beta_4\sigma_1\mu_v^2).$$

The eigenvalues are

$$\lambda_1 = -\mu_h,\ \lambda_2 = \lambda_3 = -\mu_v,\ \lambda_4 = \lambda_5 = -\beta_4\mu_v N_T.$$

The remaining three eigenvalues can be found from equation

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0.$$

From the Routh-Hurwitz stability condition, the eigenvalues λ_6, λ_7 and λ_8 are negative when

1) $a_2 > 0$,

2) $a_0 > 0$ and

3) $a_2 a_1 > a_0$.

The first condition $a_2 = \sigma_1\mu_h + (1+N_T\beta_4)\mu_v$ is always positive. The second condition, $a_0 = N_T\mu_h(-q(\beta_1+\beta_2+\beta_3)\gamma_v + \beta_4\sigma_1\mu_v^2)$ is positive when $\beta_4\sigma_1\mu_v^2 > q(\beta_1+\beta_2+\beta_3)\gamma_v$ or

$$R_0 = \frac{q(N_{v1}+N_{v2}+N_{v3})\gamma_h\gamma_v^2 N_T}{(\mu_h+r)(\mu_v+q)\mu_v^2} < 1.$$

The last condition, $a_2a_1 - a_0 = qN_T(\beta_1+\beta_2+\beta_3)\gamma_v\mu_h + (1+N_T\beta_4)\mu_v(\sigma_1\mu_h+\mu_v)(\sigma_1\mu_h+N_T\beta_4\mu_v)$ is always positive.

From our evaluations, we found that all eigenvalues have negative real parts for $R_0 < 1$. By the local stability theorem [5], the disease free state is local asymptotically stable for $R_0 < 1$.

The characteristic equation of (3) for the endemic disease state is

$$(\lambda^2 + c_1\lambda + c_2)^2(\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4) = 0$$

where

$$c_1 = (1+N_T\beta_4)\mu_v + N_T\gamma_v I^*_h,$$

$$c_0 = N_T(\beta_4\mu_v^2 + \gamma_v(q+\mu_v)I^*_h),$$

$$b_1 = (1+N_T\beta_4)\mu_v + N_T\gamma_v I^*_h + \mu_h(1+\sigma_1+\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3}),$$

$$b_2 = N_T(\beta_4\mu_v^2 + \gamma_v(q+\mu_v)I^*_h) + \mu_h(\mu_v + N_T\beta_4\mu_v + N_T\gamma_v I^*_h)$$
$$(1+\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3}) + \sigma_1\mu_h(\mu_v + N_T\beta_4\mu_v +$$
$$N_T\gamma_v I^*_h + \mu_h(1+\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3})),$$

$$b_3 = \mu_h\mu_v((1+N_T\beta_4)\sigma_1\mu_h + N_T\beta_4\mu_v) + N_T\mu_h(-q(\beta_1 +$$
$$\beta_2+\beta_3)\gamma_v + \beta_4\sigma_1\mu_v^2\mu_h(\sigma_1\mu_h\mu_v(\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3}) +$$
$$N_T(\beta_4\mu_v(\sigma_1\beta_4 + \mu_v)(\beta_1 I^*_{v1} + \beta_2 I^*_{v2} + \beta_3 I^*_{v3}) +$$
$$\gamma_v(I^*_h(q + q\beta_3 + q\sigma_1 + \sigma_1\mu_h + \mu_v + \sigma_1\mu_v + \beta_1(q + (q+\sigma_1\mu_h+\mu_v)I^*_{v1}) +$$
$$\beta_2(q + (q+\sigma_1\mu_h+\mu_v)I^*_{v2}) + \beta_3(q + (q+\sigma_1\mu_h+\mu_v)I^*_{v3}) + q(\beta_1+\beta_2+\beta_3)R^*_h +$$

$$b_4 = N_T\mu_h^2(-q(\beta_1+\beta_2+\beta_3)\gamma_v + \beta_4\sigma_1\mu_v^2) + N_T\mu_h^2(q\beta_3\gamma_v I^*_h + q\gamma_v\sigma_1 I^*_h +$$
$$\sigma_1\gamma_v\mu_v I^*_h + \beta_3\beta_4\sigma_1\mu_v^2 I^*_{v3} + q\beta_3\gamma_v\sigma_1 I^*_h I^*_{v3} + \beta_3\gamma_v\mu_v\sigma_1 I^*_h I^*_{v3} +$$
$$q\beta_3\gamma_v R^*_h + q\beta_3\gamma_v(I^*_{v3} + E^*_{v3})S^*_h + \beta_1(\beta_4\sigma_1\mu_v^2 I^*_{v1} + \gamma_v(I^*_h(q +$$
$$\sigma_1(q+\mu_v)I^*_{v1}) + q(R^*_h + (I^*_{v1} + E^*_{v1})S^*_h)) + \beta_2(\beta_4\sigma_1\mu_v^2 I^*_{v2}$$
$$+\gamma_v(I^*_h(q + \sigma_1(q+\mu_v)I^*_{v2}) + q(R^*_h + (I^*_{v2} + E^*_{v2})S^*_h))).$$

The eigenvalues are given by

$$\lambda_1 = \lambda_2 = \frac{1}{2}(-\mu_v - N_T\beta_4\mu_v - N_T\gamma_v I^*_h$$
$$-\sqrt{(\mu_v + N_T\beta_4\mu_v + N_T\gamma_v I^*_h)^2 - 4(N_T\beta_4\mu_v^2 + qN_T\gamma_v I^*_h + N_T\gamma_v\mu_v I^*_h)}$$

$$\lambda_3 = \lambda_4 = \frac{1}{2}(-\mu_v - N_T\beta_4\mu_v - N_T\gamma_v I^*_h$$
$$+\sqrt{(\mu_v + N_T\beta_4\mu_v + N_T\gamma_v I^*_h)^2 - 4(N_T\beta_4\mu_v^2 + qN_T\gamma_v I^*_h + N_T\gamma_v\mu_v I^*_h)}$$

Since

$$\mu_v + N_T\beta_4\mu_v + N_T\gamma_v I^*_h >$$
$$\sqrt{(\mu_v + N_T\beta_4\mu_v + N_T\gamma_v I^*_h)^2 - 4(N_T\beta_4\mu_v^2 + qN_T\gamma_v I^*_h + N_T\gamma_v\mu_v I^*_h)}$$

So, $\lambda_1, \lambda_2, \lambda_3, \lambda_4$ are always negative.

The remaining eigenvalues are calculated from

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

From the Routh-Hurwitz criteria, the eigenvalues $\lambda_5, \lambda_6, \lambda_7$ and λ_8 are negative when

1) $b_1 > 0$,

2) $b_3 > 0$,

3) $b_4 \geq 0$ and

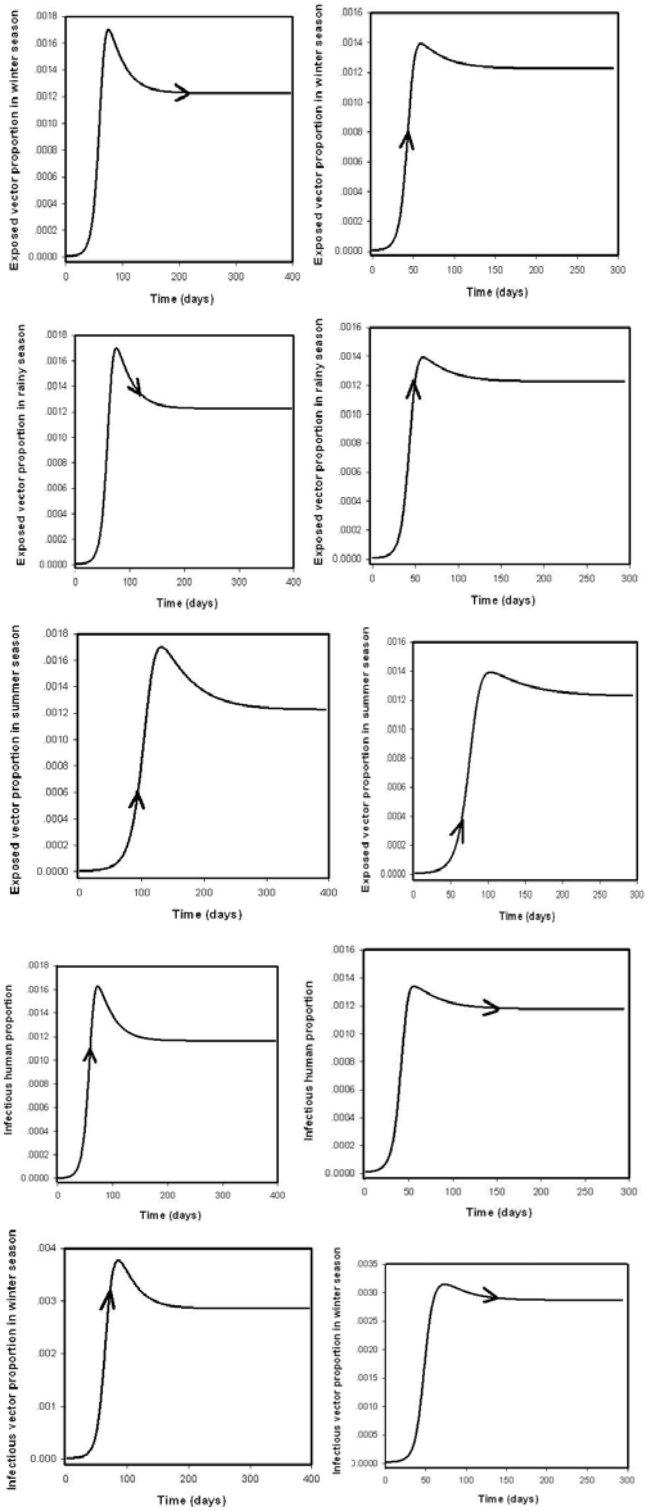
4) $b_1 b_2 b_3 > b_3^2 + b_1^2 b_4$.

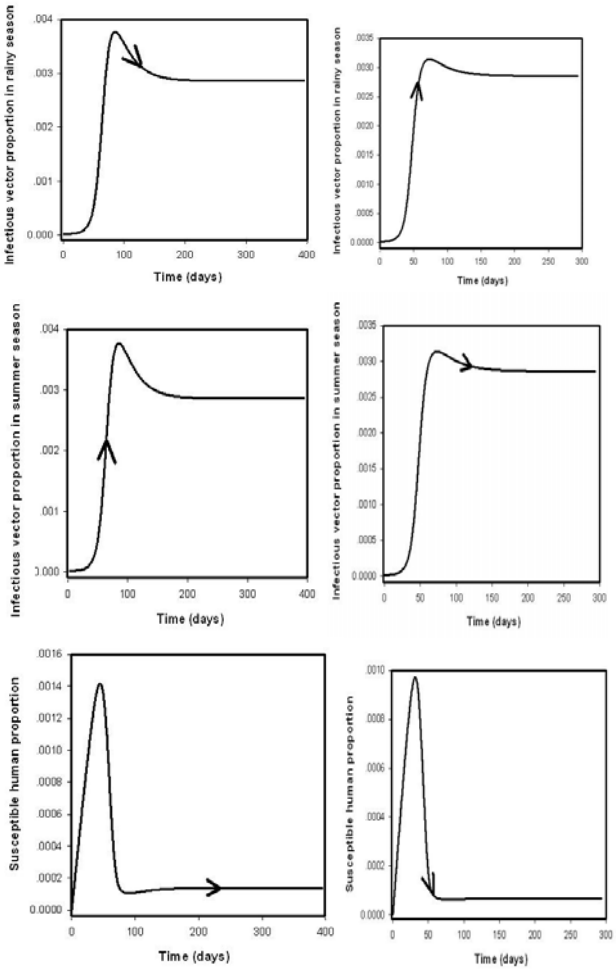
From the calculations, we found that the above conditions satisfied when $R_0 > 1$. So the endemic disease state is local asymptotically stability for $R_0 > 1$.

B. Numerical Solutions

In this section, we use the numerical methods to simulate the results of system (3). The comparison for the different threshold conditions (R_0) of the endemic equilibrium state is presented. The parameters in this simulation are: $\mu_h = 1/(365\times 70)$ corresponds to the life expectancy of 70 years for human, $q = 1/3$ correspond to the 3 days which the exposed vector change to be the infectious vector,

$r = 1/30$ correspond to the recovery of 30 days, $\mu_v = 1/7$ correspond to the life expectancy of 7 days for vector [11].





1a) 1b)

Fig. 1. Numerical solutions of the system (3), demonstrate the time series , $\overline{S_h}, \overline{I_h}, \overline{E_{v1}}, \overline{I_{v1}}, \overline{E_{v2}}, \overline{I_{v2}}, \overline{E_{v3}}, \overline{I_{v3}}$ respectively for $N_T = 25,000$, $N_{v1} = 5,000$, $N_{v3} = 10,000$, $\mu_h = 1/(365 \times 70)$ per day, $\mu_v = 1/7$ per day, $r = 1/30$ per day, $q = 1/3$ per day, $\gamma_h = 0.00001$, $\gamma_v = 0.00002$.

a) $R_0 = 1.00878$. The fractions of populations spiral to the endemic state (0.000139935, 0.00117262, 0.00122622, 0.0028612, 0.00122622, 0.0028612, 0.00122622, 0.0028612).

b) $R_0 = 2.05713$. The fractions of populations spiral to the endemic state (0.000068335, 0.00117271, 0.00122631, 0.00286139, 0.00122631, 0.00286139, 0.00122631, 0.00286139).

IV. DISCUSSION AND CONCLUSION

The transmission of Chikunkunya in human and mosquitoes is studied through the method of dynamical modeling. Routh-Hurwitz stability condition is used for determining the behavior of the population. The threshold condition (R_0) is found to classify the stability property of each equilibrium state. We compare the dynamical behavior of each individual population in fig.1. We can see that the shorter days of outbreak will occur when the threshold number is higher.

Furthermore, the individual vector proportions are shown as varying of the transmission rate from human to vector in fig.2

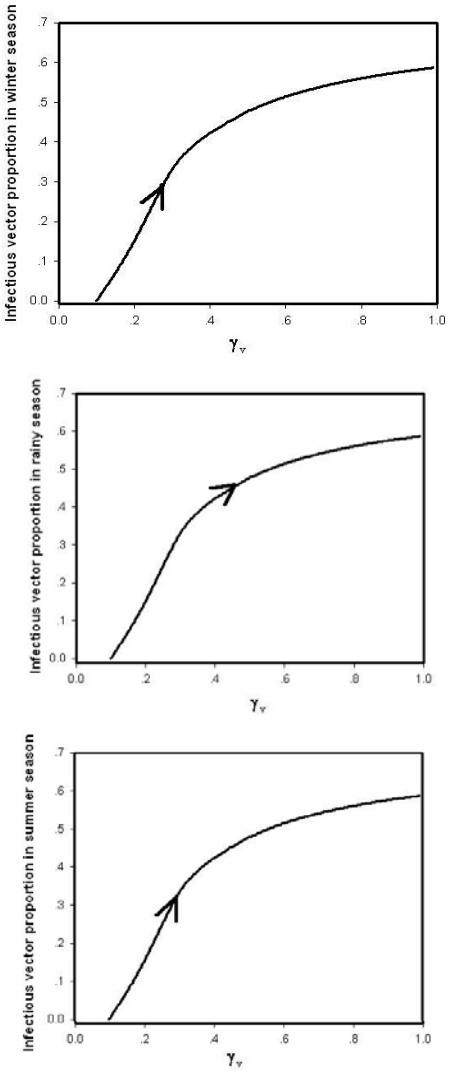


Fig 2. The individual vector proportion as varying of the transmission rate of the disease from human to vector. The parameters are similarly as fig.1.

We can see that when the transmission rate of the disease from human to vector is higher, the individual vector proportions are higher. This corresponds to the fact that the transmission rate of the disease should be reduced to decrease the fraction of the infectious vector. Consequently, the outbreak of this disease will be eliminated.

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