Mathematical Modeling for Dengue Transmission with the Effect of Season

R. Kongnuy., and P. Pongsumpun

Abstract—Mathematical models can be used to describe the transmission of disease. Dengue disease is the most significant mosquito-borne viral disease of human. It now a leading cause of childhood deaths and hospitalizations in many countries. Variations in environmental conditions, especially seasonal climatic parameters, effect to the transmission of dengue viruses the dengue viruses and their principal mosquito vector, *Aedes aegypti*. A transmission model for dengue disease is discussed in this paper. We assume that the human and vector populations are constant. We showed that the local stability is completely determined by the threshold parameter, B_0 . If B_0 is less than one, the disease free equilibrium state is stable. If B_0 is more than one, a unique endemic equilibrium state exists and is stable. The numerical results are shown for the different values of the transmission probability from vector to human populations.

Keywords—Dengue disease, mathematical model, season, threshold parameters.

I. INTRODUCTION

DENGUE d Fever (DF), Dengue Haemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS) are increasingly important public health problems in the tropical and subtropical areas. Dengue has been recognized in over 100 countries and 2.5 billion people live in areas where dengue is endemic [1]. Dengue disease caused by four distinct serotypes virus known as DEN-1, DEN-2, DEN-3 and DEN-4. It is transmitted to the human by biting of the infected female *Aedes* mosquitoes as the primary mosquito vector. Infection by any single type apparently produces permanent immunity to it, but only temporary cross immunity to the others. The mosquitoes never recover from the infection since their infective period ends with their death [2].

Infection with one of these viruses characteristically results in fever, headache and rash. The clinical spectrum can vary, however, from asymptomatic to more severe infections with bleeding and shock. The manifestations of DHF include hemorrhage and shock, which is the result of a sudden loss of intravascular volume consequent to vascular leakage. As no vaccine presently exists, the only method of controlling or

preventing dengue and DHF is to combat the mosquito vectors [1].

Environments, climatic variables such as temperature, humidity, season and precipitation significantly influence mosquito development. Temperature affects to the development of the mosquitoes, as well as dengue viral development. Dengue infection is endemic in Thailand and many other tropical countries. Fig. 1 shows the incidence rate of dengue disease per 1,000,000 reported to the Division of Epidemiology, Ministry of Public Health classified by month, Thailand during the period 1999-2008. As we see, dengue cases generally peak in June, July and August.

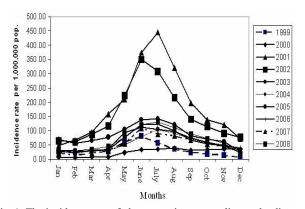


Fig. 1 The incidence rate of dengue patients according to the disease severity month-by-month between 1999 and 2008 [3].

Mathematical modeling of infectious disease has a long history. The starting point is generally taken to be a paper by Daniel Bernoulli [4] on the prevention of smallpox by inoculation. To control the dengue effectively, we should understand the dynamics of the disease transmission and take into account all of the relevant details, such as the dynamics of the Esteva and Vargas [5] developed a model for the dengue disease transmission and included the dynamics of the Aedes aegypti mosquitoes into a standard SIR (susceptible-infective-recover) epidemic model of a single population. Their model shows that there is a threshold number which is a function of Aedes equilibrium population size and of the Aedes recruitment rate, above which the disease will be endemic and below which the disease will vanish.

In this paper, we develop a mathematical model as an interesting tool for the understanding of the dengue transmission for the difference season. Our interest here is to derive and analyze the model taking into account the

P. Pongsumpun is with the Department of Mathematics, Faculty of Science, King Mongkut's Institute of Technology Ladkrabang, Chalongkrung road, Ladkrabang, Bangkok 10520, Thailand (corresponding author to provide phone: 662-737-3000 ext. 6196; fax: 662-326-4344 ext. 284; e-mail: kppuntan@ kmitl.ac.th)*.

R. Kongnuy is with the Department of Mathematics, Faculty of Science, King Mongkut's Institute of Technology Ladkrabang, Chalongkrung road, Ladkrabang, Bangkok 10520, Thailand, (e-mail: s9062852@kmitl. ac.th).

seasonality dengue compartment in the transmission model.

In the next section, we formulate the model. In section 3, we analyze the model. The equilibrium states of this system and their stability are obtained. Finally, we give the numerical results and conclusion.

II. THE MATHEMATICAL MODEL

In our model, we assume that the human and vector population have constant size. Let N_T and N_v be the human and vector population sizes. The mathematical model for this transmission is based on the transmission diagram in Fig. 2.

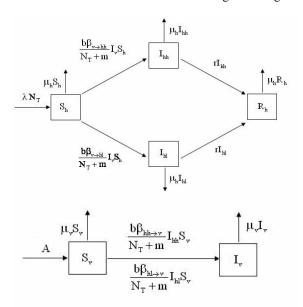


Fig. 2 Transmission diagram of dengue disease with season.

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The human population is subdivided into the susceptible S_h , the infectious human in high epidemic season I_{hh} (in June, July and August), the infectious human in low epidemic season I_{hh} (every months except June, July and August),and the recovered R_h . The vector population is subdivided into the susceptible S_v and the infectious I_v . The dynamical equations for human population are

$$\frac{dS_{_h}}{dt} = \lambda N_{_T} - (\mu_{_h} + \frac{b\beta_{_{v \to hh}}}{N_{_T} + m} I_{_v} + \frac{b\beta_{_{v \to hl}}}{N_{_T} + m} I_{_v}) S_{_h} \tag{1}$$

$$\frac{dI_{hh}}{dt} = \frac{b\beta_{\nu \to hh}}{N_{\scriptscriptstyle T} + m} I_{\scriptscriptstyle \nu} S_h - (\mu_h + r) I_{hh} \eqno(2)$$

$$\frac{dI_{hl}}{dt} = \frac{b\beta_{v \to hl}}{N_{\scriptscriptstyle T} + m} I_{\scriptscriptstyle V} S_h - (\mu_h + r) I_{hl} \tag{3} \label{eq:dIhl}$$

$$\frac{dR_{h}}{dt} = r(I_{hh} + I_{hl}) - \mu_{h}R_{h} \tag{4}$$

and the dynamical equations for vector population are as follows

$$\frac{dS_{v}}{dt} = A - (\mu_{v} + \frac{b\beta_{hh \to v}}{N_{x} + m}I_{hh} + \frac{b\beta_{hl \to v}}{N_{x} + m}I_{hl})S_{v}$$
 (5)

$$\frac{dI_{\rm v}}{dt} = (\frac{b\beta_{\rm hh\to v}}{N_{_{\rm T}}+m}I_{\rm hh} + \frac{b\beta_{\rm hl\to v}}{N_{_{\rm T}}+m}I_{\rm hl})S_{\rm v} - \mu_{\rm v}I_{\rm v} \tag{6}$$

The equation for $R_{_h}$ and $S_{_v}$ in (4)-(5) can be eliminated since at time t, we have $S_{_h}+I_{_{hh}}+I_{_{hl}}+R_{_h}=N_{_T}$ and $S_{_v}+I_{_v}=N_{_v}$. To simplify the mathematical analysis of this study, we normalize the equations (1)-(6) by defining new variables

$$\begin{split} S_h &= \overline{S_h} \big/ N_{_T} \;, \quad I_{_{hh}} &= \overline{I_{_{hh}}} \big/ N_{_T} \;, \quad I_{_{hl}} &= \overline{I_{_{hl}}} \big/ N_{_T} \;, \quad R_{_{h}} &= \overline{R_{_{h}}} \big/ N_{_T} \;, \\ S_{_{v}} &= \overline{S_{_{v}}} \big/ N_{_{v}} &= \overline{S_{_{v}}} \big/ (A \big/ \mu_{_{v}}) \; \text{and} \; I_{_{v}} &= \overline{I_{_{v}}} \big/ N_{_{v}} &= \overline{I_{_{v}}} \big/ (A \big/ \mu_{_{v}}) \;. \end{split}$$

The total human and vector populations are constant, thus rates of change for total human and vector populations equal to zero. This gives birth and death rates are equal for the human population, the total vector population equals to A/μ_{ν} . We obtain the equations as follows:

$$\frac{d\overline{S_h}}{dt} = \mu_h - (\mu_h + \frac{b\beta_{v \to hh}}{N_{_T} + m} \overline{I_v}(A/\mu_v) + \frac{b\beta_{v \to hl}}{N_{_T} + m} \overline{I_v}(A/\mu_v)) \overline{S_h} \qquad (7)$$

$$\frac{d\overline{I_{hh}}}{dt} = \frac{b\beta_{v \to hh}}{N_T + m} \overline{I_v} (A/\mu_v) \overline{S_h} - (\mu_h + r) \overline{I_{hh}}$$
 (8)

$$\frac{d\overline{I_{hl}}}{dt} = \frac{b\beta_{v \to hl}}{N_T + m} \overline{I_v} (A/\mu_v) \overline{S_h} - (\mu_h + r) \overline{I_{hl}}$$
 (9)

$$\frac{d\overline{I_{v}}}{dt} = \left(\frac{b\beta_{hh\to v}}{N_{T} + m}\overline{I_{hh}}N_{T} + \frac{b\beta_{hl\to v}}{N_{T} + m}\overline{I_{hl}}N_{T}\right)(1 - \overline{I_{v}}) - \mu_{v}\overline{I_{v}}$$
(10)

where

 N_{T} is the total human population,

N_v is the total vector population,

S_b is the number of susceptible human population,

 I_{hh} is the number of infectious human in high epidemic season.

 I_{hl} is the number of infectious human in low epidemic season,

R_h is the number of recover human population,

S_v is the number of susceptible vector population,

I_v is the number of infectious vector population,

 μ_h is the death rate in the human population,

 λ is the birth rate in the human population,

 μ_{v} is the death rate in the vector population,

 $\beta_{v \to hh}$ is the transmission probability from vector to human

(in high epidemic season), $\beta_{v \to hl} \quad \text{is the transmission probability from vector to human} \\ \quad \text{(in low epidemic season),}$

 $\beta_{hh\to v}$ is the transmission probability from human (in high epidemic season) to vector,

 $\beta_{hl \to v}$ is the transmission probability from human (in low epidemic season) to vector,

r is the recover rate in the human population

b is the biting rate of vector.

The our model, we define the transmission probability from vector to human (in high epidemic season) and the transmission probability from human (in high epidemic season) to vector are more than 0.5 but not more than 1 $0.5 < \beta_{v \to hh}$, $\beta_{hh \to v} \le 1)$ and the transmission probability from vector to human (in low epidemic season) and the transmission probability from human (in low epidemic season) to vector are more than or equal zero but not more than 0.5 $(0 \le \beta_{v \to hl}, \beta_{hl \to v} \le 0.5)$ with the new two conditions $\overline{S_h} + \overline{I_{hh}} + \overline{I_{hl}} + \overline{R_h} = 1$ and $\overline{S_v} + \overline{I_v} = 1$.

III. ANALYSIS OF THE MODEL

A. Equilibrium Point

In this section we will find the equilibrium points of equations (7)-(10) in the region of Ω , with

$$\Omega = \left\{ (\overline{S_h}, \overline{I_{hh}}, \overline{I_{hl}}, \overline{I_{\nu}}) \middle| 0 \leq \overline{S_h}, \overline{I_{hh}}, \overline{I_{hl}}, \overline{I_{\nu}} \leq 1 \right\}.$$

Direct calculation shows that equations (7)-(10) has two possible equilibrium points: the disease free equilibrium point and a unique endemic equilibrium point. Two equilibrium points are found by setting the right hand side of (7)-(10) equal to zero. This gives

- 1) the disease free equilibrium point $E_0 = (1,0,0,0)$ and
- 2) the endemic disease equilibrium point $E_1 = (S_h^*, I_{hh}^*, I_{hl}^*, I_{h}^*)$

where

$$S_{h}^{*} = \frac{\tau_{1}\tau_{2}}{\tau_{1}\tau_{2} + M_{2}I_{v}^{*}}$$
 (11)

$$I_{hh}^{*} = \frac{\lambda_{1}\tau_{2}I_{v}^{*}}{M_{1}(\tau_{1}\tau_{2} + M_{2}I_{v}^{*})}$$
 (12)

$$I_{hl}^* = \frac{\lambda_2 \tau_1 I_v^*}{M_1(\tau_1 \tau_2 + M_2 I_v^*)}$$
 (13)

$$I_{v}^{*} = \frac{M_{3} - M_{1}\tau_{1}\tau_{2}}{M_{2} + M_{1}M_{2}}$$
 (14)

with

$$\begin{split} &\tau_{1} = \frac{b\beta_{v\to hh}N_{T}}{\mu_{v}\left(N_{T}+m\right)} \quad , \; \tau_{2} = \frac{b\beta_{v\to hl}N_{T}}{\mu_{v}\left(N_{T}+m\right)} \, , \; \tau_{3} = \frac{b\beta_{hh\to v}N_{T}}{\mu_{v}\left(N_{T}+m\right)} \, , \\ &\tau_{4} = \frac{b\beta_{hl\to v}N_{T}}{\mu_{v}\left(N_{T}+m\right)} \, , \; \lambda_{1} = \frac{b^{2}\beta_{v\to hh}^{2}N_{T}(A/\mu_{v})}{\mu_{v}\mu_{h}\left(N_{T}+m\right)^{2}} \quad , \\ &\lambda_{2} = \frac{b^{2}\beta_{v\to hl}^{2}N_{T}(A/\mu_{v})}{\mu_{v}\mu_{h}\left(N_{T}+m\right)^{2}} \quad , M_{1} = \frac{\mu_{h}+r}{\mu_{h}} \, , \; M_{2} = \lambda_{1}\tau_{2} + \lambda_{2}\tau_{1} \, , \\ &M_{3} = \lambda_{1}\tau_{2}\tau_{3} + \lambda_{2}\tau_{1}\tau_{4} \, . \end{split}$$

B. Local Asymptotical Stability

The local stability of an equilibrium point is determined from the Jacobian matrix of the right hand side of the above set of differential equations evaluated at the equilibrium point.

C. Local Asymptotical Stability

For the equations defined by (7)-(10), the Jacobian matrix evaluated at E_0 is the 4×4 matrix given by

$$\begin{bmatrix} -\mu_{h} & 0 & 0 & -\frac{\mu_{h}\lambda_{1}}{\tau_{1}} - \frac{\mu_{h}\lambda_{2}}{\tau_{2}} \\ 0 & -\mu_{h}M_{1} & 0 & \frac{\mu_{h}\lambda_{1}}{\tau_{1}} \\ 0 & 0 & -\mu_{h}M_{1} & \frac{\mu_{h}\lambda_{2}}{\tau_{2}} \\ 0 & \mu_{u}\tau_{2} & \mu_{u}\tau_{4} & -\mu_{u} \end{bmatrix}$$
(15)

The eigenvalues are obtained by solving the matrix equation, $\det(J - \lambda I_4) = 0$. To evaluate the determinant, we obtained the following characteristic equation

$$(\lambda + \mu_h)(\lambda + \mu_h M_1)(\lambda^2 + a_1 \lambda + a_2) = 0$$
 (16) where

 $a_1 = \mu_h M_1 + \mu_v \,, \tag{17}$

$$a_{1} = \mu_{h} M_{1} + \mu_{v}, \qquad (17)$$

$$a_{2} = \mu_{h} \mu_{v} \left(\frac{M_{1} \tau_{1} \tau_{2} - \tau_{2} \tau_{3} \lambda_{1} - \tau_{1} \tau_{4} \lambda_{2}}{\tau_{1} \tau_{2}} \right) \qquad (18)$$

Looking at the characteristic equation, (16), we see that two of the eigenvalues are $-\mu_h$ and $-\mu_h M_1$.

Both of them are negative. Next, we will check the sign of other eigenvalues. From $\lambda^2 + a_1\lambda + a_2 = 0$, the two conditions of Routh-Hurwitz criteria [6] for local asymptotical stability in second order characteristic polynomial equation are

- i) $a_1 > 0$,
- ii) $a_2 > 0$.

After we check the stability of the equilibrium point, we can see a_1 is always positive and a_2 is positive when $\frac{\tau_2\tau_3\lambda_1+\tau_1\tau_4\lambda_2}{M_1\tau_1\tau_2}<1\,.$ Moreover, we found that the disease free

equilibrium point is locally stable for $B_0 = \frac{\tau_2\tau_3\lambda_1 + \tau_1\tau_4\lambda_2}{M_1\tau_1\tau_2} < 1 \, .$

D. Disease Endemic Equilibrium point

The stability of the endemic disease equilibrium point, E_1 , like that of E_0 , is determined by looking at the eigenvalues of the Jacobian evaluated at E_0 . The Jacobian for this equilibrium point is

$$\begin{bmatrix} -\mu_{h} - \frac{\mu_{h}\lambda_{1}}{\tau_{1}} \Gamma_{v}^{*} - \frac{\mu_{h}\lambda_{2}}{\tau_{2}} \Gamma_{v}^{*} & 0 & 0 & -\frac{\mu_{h}\lambda_{1}}{\tau_{1}} S_{h}^{*} - \frac{\mu_{h}\lambda_{2}}{\tau_{2}} S_{h}^{*} \\ \frac{\mu_{h}\lambda_{1}}{\tau_{1}} \Gamma_{v}^{*} & -\mu_{h}M_{1} & 0 & \frac{\mu_{h}\lambda_{1}}{\tau_{1}} S_{h}^{*} \\ \frac{\mu_{h}\lambda_{2}}{\tau_{2}} \Gamma_{v}^{*} & 0 & -\mu_{h}M_{1} & \frac{\mu_{h}\lambda_{2}}{\tau_{2}} S_{h}^{*} \\ 0 & \mu_{v}\tau_{v}(1-1_{v}^{*}) & \mu_{v}\tau_{d}(1-1_{v}^{*}) -\mu_{v}\tau_{v}1_{h}^{*} - \mu_{v}\tau_{d}1_{h}^{*} - \mu_{v} \end{bmatrix}$$

$$(19)$$

where $S_h^*, I_{hh}^*, I_{hl}^*$ and I_v^* are given by equation (11)-(14). The characteristic equation for the Jacobian matrix evaluated at the second equilibrium state, given by (7)-(10), is

$$(\lambda + \mu_h M_1)(\lambda^3 + b_2 \lambda^2 + b_1 \lambda + b_0) = 0$$
 (20)

It can be seen that one eigenvalue is $-\mu_h M_1$. Next we found the other eigenvalues by solving the equation

$$\lambda^{3} + b_{2}\lambda^{2} + b_{1}\lambda + b_{0} = 0 \tag{21}$$

with

$$b_2 = \tau_2(\tau_1\tau_2((1+M_1)\mu_h + \mu_v I_{h3}^*) + \mu_h M_2 I_v^*)/\tau_1, \qquad (22)$$

 $b_{_{1}}=\tau_{_{2}}(\mu_{_{h}}(M_{_{1}}(\tau_{_{1}}\tau_{_{2}}(\mu_{_{h}}+\mu_{_{v}}I_{_{h3}}^{^{*}})+\mu_{_{h}}M_{_{2}}I_{_{v}}^{^{*}}+$

$$\mu_{v}(I_{h3}^{*}(\tau_{1}\tau_{2}+M_{2}I_{v}^{*})+M_{3}(-1+I_{v}^{*})S_{h}^{*})))/\tau_{1}, \qquad (23)$$

$$b_0 = \tau_2(\mu_h^2 \mu_v(M_1 I_{h3}^*(\tau_1 \tau_2 + M_2 I_v^*) + M_3(-1 + I_v^*) S_h^*) / \tau_1 \qquad (24)$$

By using Routh-Hurwitz criteria [6], each equilibrium point is locally stable if the following conditions are satisfied,

- i) $b_2 > 0$
- ii) $b_0 > 0$
- iii) $b_{1}b_{1} > b_{0}$

We can see b_2 is always positive. For the second and the third conditions we shown these conditions by using the following figures

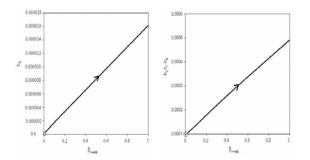


Fig. 3 The parameter space for the endemic equilibrium point which satisfies the Routh-Hurwitz criteria with the value of parameters: respectively, for with $\mu_h=1/(365\times65)~\text{day}^\text{-1},~\mu_v=(1/14)~\text{day}^\text{-1},~\Lambda=10,000$, $N_T=100,000$, b=1/3, r=1/3, $m=0,\beta_{v\to hl}=0.4$, $\beta_{hh\to v}=0.7$, $\beta_{hl\to v}=0.4$ and $0\le\beta_{v\to hh}\le1$.

We found that E_0 is locally stable for $B_0 < 1$ and E_1 is locally stable for $B_0 > 1$.

E. Numerical Simulation

In this paper, we are interested in the transmission of dengue disease with the effect of season. The values of the parameter used in this study are as follows: $\mu_h=1/(365\times65)$ per day corresponds to a life expectancy of 65 years in human. The mean life of mosquito is 14 days; $\mu_v=(1/14)$ per day. The recovery rate equals to 1/3 per day. We assume that no alternative host. The conditions of parameters are $\beta_{v\to hh}>\beta_{v\to hl},~\beta_{hh\to v}>\beta_{hl\to v}$, $0.5<\beta_{v\to hh},\beta_{hh\to v}\leq 1$ and $0\leq\beta_{v\to hl},\beta_{hl\to v}\leq 0.5$.

The other parameters are arbitrarily chosen. We presented the numerical solutions of (7)-(10) for the endemic equilibrium state on the following figures.

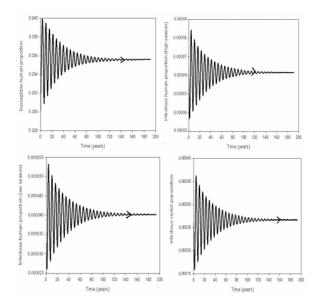


Fig. 4 Numerical solutions of (7)-(10), demonstrate the times series of $\overline{S_h}$, $\overline{I_{hh}}$ $\overline{I_{hl}}$ and $\overline{I_v}$ respectively, for $B_0=4.24613$, $B_0^{'}=2.06061$ with $\mu_h=1/(365\times65)$ day-1, $\mu_v=(1/14)$ day-1, A=10,000, $N_T=100,000$, b=1/3, r=1/3, m=0, $\beta_{v\to hh}=0.7$, $\beta_{v\to hl}=0.4$, $\beta_{hh\to v}=0.7$, $\beta_{hl\to v}=0.4$. The fractions of populations oscillate to the endemic state (0.235571, 0.0000615, 0.0000351,0.0002664).

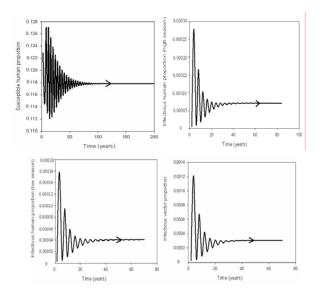


Fig. 5 Numerical solutions of (7)-(10), demonstrate the times series of \overline{S}_h , \overline{I}_{hh} \overline{I}_{hl} and \overline{I}_v respectively, for $B_0=8.49226$, $B_0^\prime=2.91415 \text{ with } \mu_h=1/(365\times65) \text{ day}^{\text{-}1}, \ \mu_v=(1/14) \text{ day}^{\text{-}1}, \\ A=20,000$, $N_T=100,000, b=1/3, r=1/3, m=0, \beta_{v\to hh}=0.7, \\ \beta_{v\to hl}=0.4, \beta_{hh\to v}=0.7, \beta_{hl\to v}=0.4$. The fractions of populations

oscillate to the endemic state (0.117791,0.00007098, 0.00004056,0.000307486).

IV. DISCUSSION AND CONCLUSION

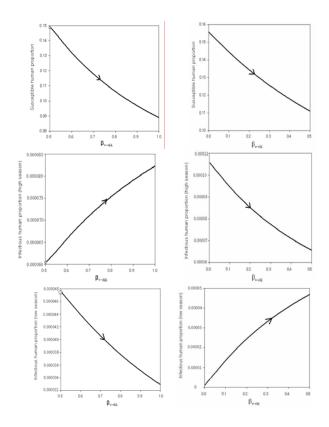
We obtain the threshold parameter for the model as

$$\begin{split} B_{0} &= \frac{\tau_{2}\tau_{3}\lambda_{1} + \tau_{1}\tau_{4}\lambda_{2}}{M_{1}\tau_{1}\tau_{2}} \\ &= \frac{b^{2}\beta_{v\to hh}\beta_{hh\to v}N_{T}A}{\mu_{v}^{2}(N_{T}+m)^{2}(\mu_{h}+r)} + \frac{b^{2}\beta_{v\to hl}\beta_{hl\to v}N_{T}A}{\mu_{v}^{2}(N_{T}+m)^{2}(\mu_{h}+r)} \end{split} \tag{25}$$

Analysis of this model reveals the existence of two equilibrium points. One is the disease free equilibrium and it is locally asymptotically stable if and only if $B_0 < 1$. The another one equilibrium point is the endemic equilibrium point. This equilibrium point will be a locally asymptotically stable endemic point if and only if $B_0 > 1$.

$$Let~~B_0=\frac{\tau_2\tau_3\lambda_1+\tau_1\tau_4\lambda_2}{M_1\tau_1\tau_2}~~is~~the~~threshold~~parameter.~~The$$

quantity $\widetilde{B_0} = \sqrt{B_0}$ is called the basic reproductive number of the disease, since it represents the average number of secondary cases that one case can produce if introduced into a susceptible population. We consider the time series of human and vector populations when the transmission probability from vector to human (in high epidemic season) and the transmission probability from human to vector (in low epidemic season) are difference. We show in Fig. 6.



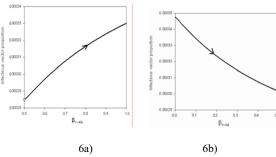


Fig. 6 Numerical solutions of (7)-(10) demonstrate the equilibrium solution of $\overline{S_h}$, $\overline{I_{hh}}$ $\overline{I_{hl}}$ and $\overline{I_v}$ respectively, for $B_0 > 1$

$$\begin{split} &(6a) \ with \ \mu_h = 1/(365 \times 65) \ day^{\text{-1}}, \ \mu_v = (1/14) \ day^{\text{-1}}, \\ &A = 10,000 \ , N_T = 100,000, b = 1/3, r = 1/3, m = 0, \ 0.5 < \beta_{v \to hh} \le 1, \\ &\beta_{v \to hl} = 0.4, \beta_{hh \to v} = 0.7, \beta_{hl \to v} = 0.4 \ . \\ &(6b) \ with \ \mu_h = 1/(365 \times 65) \ day^{\text{-1}}, \ \mu_v = (1/14) \ day^{\text{-1}}, \\ &A = 10,000 \ , N_T = 100,000, b = 1/3, r = 1/3, m = 0, \ \beta_{v \to hh} = 0.7, \\ &0 \le \beta_{v \to hl} \le 0.5, \beta_{hh \to v} = 0.7, \beta_{hl \to v} = 0.4 \ . \end{split}$$

The basic reproductive number of the disease for Fig. 4 and Fig. 5 equal to 2.06061 and 2.91415, respectively. Periods of the oscillations as the simulations approach the endemic equilibrium point are estimated by means of the solutions of the linearized system, obtain 6.07 years for Fig. 4 and 3.99 years for Fig. 5.

Moreover, we compare the transmission of dengue disease for the different transmission probability from vector to human (in high epidemic season)and the transmission probability from vector to human (in low epidemic season).

We can see from the value of B_0 , if the transmission of dengue disease for the different transmission probability from vector to human (in high epidemic season) is higher, this means that the infectious human proportion in high epidemic and infectious vector proportion are high. For the transmission probability from vector to human (in low epidemic season) is high, the infectious human proportion in low endemic interval is high too.

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