Dengue Transmission Model between Infant and Pregnant Woman with Antibody

R. Kongnuy, and P. Pongsumpun

Abstract—Dengue, a disease found in most tropical and subtropical areas of the world. It has become the most common arboviral disease of humans. This disease is caused by any of four serotypes of dengue virus (DEN1-DEN4). In many endemic countries, the average age of getting dengue infection is shifting upwards, dengue in pregnancy and infancy are likely to be encountered more frequently. The dynamics of the disease is studied by a compartmental model involving ordinary differential equations for the pregnant, infant human and the vector populations. The stability of each equilibrium point is given. The epidemic dynamic is discussed. Moreover, the numerical results are shown for difference values of dengue antibody.

Keywords—Dengue antibody, infant, pregnant human, mathematical model.

I. INTRODUCTION

MATHEMATICAL model can provide an alternative quality assessment of dynamic of the infectious disease.

The results of the interested model can help us to gain insight into the factor controlling the persistence and stability of transmitted viral infections within large human communities and lead to a better understanding the viral and host interaction proteomics. In 1999 and 2003, Esteva and Vargas [1]-[2] propose two mathematical models for the transmission of dengue fever. They established the global stability of the endemic equilibrium state. They discussed the vector population in term of the threshold condition, which governs the existence and stability of the endemic equilibrium state.

Dengue fever is caused by four closely related but distinct serotypes known as DEN-1, DEN-2, DEN-3, and DEN-4, in the genus *Flavivirus* of family *Flaviviridae* (single-strand, nonsegmented RNA virus), which can be distinguished by serological methods [3]. Infection with one serotype confers immunity to the infected serotype for a long period, but not to other serotypes. Humans may therefore be infected with the dengue virus up to four times. The most important vector is the *Aedes aegypti* mosquitoes, a day-biting time mosquitoes

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R. Kongnuy is with the Department of Mathematics and Computer Science, Faculty of Science, King Mongkut's Institute of Technology Ladkrabang, Chalongkrung road, Ladkrabang, Bangkok 10520, Thailand (e-mail: s9062852@kmitl.ac.th). which is usually found in dark place inside human housing. The incubation period of dengue disease is normally 3-8 days. The virus is detected in human subjects 6-18 hours before the onset of symptoms and viremia ends as the fever abates [4]. Infection with dengue virus may be asymptomatic or may cause undifferentiated febrile illness, dengue fever (DF) . DF is the most common in older children and adults and occasionally with unusual hemorrhagic, while dengue hemorrhagic fever (DHF) is the most common in school age children having secondary response. DHF is characterized by the onset of an acute fever and associated non-specific constitutional signs and symptoms. In addition, the potentially fatal complication of dengue called Dengue hemorrhagic fever (DHF) can occur.

The global epidemiology and the dynamics of transmission of dengue virus have changed dramatically in South-east Asia since World War II [5]. The disease is now highly endemic in more than 100 tropical countries, and the number of cases has increased dramatically during the past three decades[5]-[7]. Worldwide, it is estimated that up to 100 million cases of DF and 250,000 cases of DHF occur annually [8]. Dengue infection is endemic in Thailand and many other tropical countries. Fig. 1 shows the annual number of dengue cases reported to the Division of Epidemiology, Ministry of Public Health, Thailand during the period 1997-2007. As we see, there were many severe cases with the peak increased in 2001.

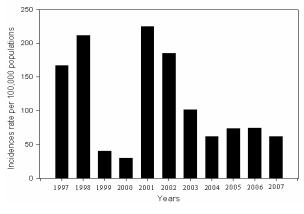


Fig. 1 The distribution of DHF patients according to the disease severity between 1997 and 2007 [9]

In Thailand, DHF is common in children (less than 15 years) and cause a significant number of deaths From 1997 to 2007, 9,412 infants were diagnosed with dengue infection,

this was approximated 1.3 % of all reported cases of dengue infection [9]. DHF is less common during infancy but when it does occur, the mortality is higher than in older children [10]. In Bangkok, Thailand, infants less than 12 months of age are infected with dengue virus were at high risk for DHF if maternal antibodies to dengue virus were present at subneutralizing levels. This led to the theory that DHF is caused by antibody enhancement of viral infection [11]-[13]. As it is mentioned above, infant is an interesting group for this study.

For maternal antibody to dengue virus in infants are disappeared in 3% by two months of age, in 19% by four months of age, in 72% by six months of age, in 92% by nine months of age, and in 100% by 12 months of age [14]. So that, the maternal antibodies to dengue virus disappeared in the first year of life. In our model, infant is defined as the baby who age not more than 12 months. Fig. 2 shows the number of infant cases in Thailand.

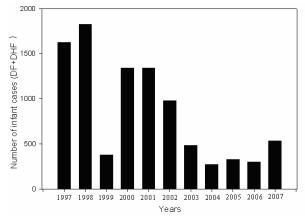


Fig. 2 The number of infant patients according to the disease severity between 1997 and 2007 [9]

In this paper, we are interested in the transmission model between pregnant woman and infant, maternal antibodies to dengue is incorporated into the model. The aims of this paper are to construct and analysis the transmission of dengue disease between pregnant woman and infant with dengue antibody.

II. MATHEMATICAL MODEL

To study the transmission of dengue virus infection, we divide the human population into two classes, pregnant woman and infant classes. Each class is divided into three subclasses, susceptible, infected and recovered human. The vector population is separated into two classes, susceptible and infected vector populations because it never recovers from infection. We assume the susceptible pregnant woman is never infected with dengue virus and infant is defined as the baby who age not more than 12 months.

In our model, the dynamics of each component of human and vector are given by

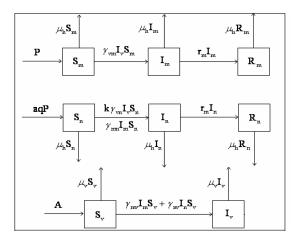


Fig. 3 The diagram of mathematical model for the transmission of dengue disease in pregnant woman, infant human and vector populations

where the parameters in the above diagram are defined as

- $\boldsymbol{S}_{\boldsymbol{m}}$ is the number of susceptible pregnant woman,
- $I_{\rm m}$ is the number of infected pregnant woman,
- \mathbf{R}_{m} is the number of recovered pregnant woman,
- \mathbf{S}_{n} is the number of susceptible infant,
- I_n is the number of infected infant,
- \mathbf{R}_{n} is the number of recovered infant,
- \mathbf{S}_{v} is the number of susceptible vector,
- I_{v} is the number of infected vector,
- a is the percentage of infant who be not die while pregnant,
- q is the average number of infant which one woman can have in each time of pregnancy,
- P is the constant recruitment rate of pregnant woman,
- N_{v} is the total adult mosquitoes,
- μ_h is the average constant death rate of pregnant woman,
- μ_v is the average constant death rate of vector,
- γ_{vm} is the transmission rate of dengue virus from vector to mother and the mother is infected,
- γ_{vn} is the transmission rate of dengue virus from vector to infant and infant is infected,
- γ_{mv} is the transmission rate of dengue virus from mother to vector and vector is infected,
- γ_{nv} is the transmission rate of dengue virus from infant to vector and vector is infected,
- γ_{mn} is the transmission rate of dengue virus from mother to infant and infant is infected,
- $\mathbf{r}_{\rm m}~$ is the constant rate at which human populations

recovers,

 N_m is the total number of pregnant woman,

 N_n is the total number of infant,

- A is the adult mosquito recruitment rate,
- D_{nm} is the percentage of dengue antibody which infant received from mother in the beginning,
- k is the probability of dengue virus which infant received from the biting of infected vector in the beginning,
- b is the average rate of biting per mosquito per day,
- h is the number of alternative hosts available as blood sources,
- β_{vm} is the transmission probability from vector to pregnant woman,
- β_{vn} is the transmission probability from vector to infant,
- β_{mv} is the transmission probability from pregnant woman to vector,
- β_{nv} is the transmission probability from infant to vector,
- β_{mn} is the transmission probability from pregnant woman to infant,

where
$$k = \frac{100 - D_{nm}}{100}$$
, $S_m + I_m + R_m = N_m$,

 $\mathbf{S}_{n}+\mathbf{I}_{n}+\mathbf{R}_{n}=\mathbf{N}_{n}$, and $\,\mathbf{S}_{v}+\mathbf{I}_{v}=\mathbf{N}_{v}$.

The model for dengue disease in pregnant woman and infant human are governed by the following equations:

$$\begin{aligned} \frac{dS_{m}}{dt} = P - (\mu_{h} + \gamma_{vm}I_{v})S_{m}, \\ \frac{dI_{m}}{dt} = \gamma_{vm}I_{v}S_{m} - (\mu_{h} + r_{m})I_{m}, \\ \frac{dR_{m}}{dt} = r_{m}I_{m} - \mu_{h}R_{m}, \\ \frac{dS_{n}}{dt} = aqP - (\mu_{h} + k\gamma_{vn}I_{v} + \gamma_{mn}I_{m})S_{n} , \qquad (1) \\ \frac{dI_{n}}{dt} = (k\gamma_{vn}I_{v} + \gamma_{mn}I_{m})S_{n} - (\mu_{h} + r_{m})I_{n}, \\ \frac{dR_{n}}{dt} = r_{m}I_{n} - \mu_{h}R_{n}, \\ \frac{dS_{v}}{dt} = A - (\mu_{v} + \gamma_{mv}I_{m} + \gamma_{nv}I_{n})S_{v}, \\ \frac{dI_{v}}{dt} = (\gamma_{mv}I_{m} + \gamma_{nv}I_{m})S_{v} - \mu_{v}I_{v} \\ \text{with three conditions } S_{m} + I_{m} + R_{m} = N_{m}, \\ S_{n} + I_{n} + R_{n} = N_{n} \text{ and } S_{v} + I_{v} = N_{v}. \end{aligned}$$

We assumed the total populations remains constant and each group of population also remain constant, we obtain

$$\begin{split} \mathbf{N}_{\mathrm{T}} &= \frac{(\mathbf{P} + \mathbf{a}\mathbf{q}\mathbf{P})}{\mu_{\mathrm{h}}}, \ \mathbf{N}_{\mathrm{m}} = \frac{\mathbf{P}}{\mu_{\mathrm{h}}}, \ \mathbf{N}_{\mathrm{n}} = \frac{\mathbf{a}\mathbf{q}\mathbf{P}}{\mu_{\mathrm{h}}} \ \text{and} \ \mathbf{N}_{\mathrm{v}} = \frac{\mathbf{A}}{\mu_{\mathrm{v}}} \,. \end{split}$$
We normalize (1) by letting $\overline{\mathbf{S}_{\mathrm{m}}} = \frac{\mathbf{S}_{\mathrm{m}}}{\mathbf{N}_{\mathrm{m}}}, \ \overline{\mathbf{I}_{\mathrm{m}}} = \frac{\mathbf{I}_{\mathrm{m}}}{\mathbf{N}_{\mathrm{m}}}, \\ \overline{\mathbf{R}_{\mathrm{m}}} = \frac{\mathbf{R}_{\mathrm{m}}}{\mathbf{N}_{\mathrm{m}}}, \ \overline{\mathbf{S}_{\mathrm{n}}} = \frac{\mathbf{S}_{\mathrm{n}}}{\mathbf{N}_{\mathrm{n}}}, \ \overline{\mathbf{I}_{\mathrm{n}}} = \frac{\mathbf{I}_{\mathrm{n}}}{\mathbf{N}_{\mathrm{n}}}, \ \overline{\mathbf{R}_{\mathrm{n}}} = \frac{\mathbf{R}_{\mathrm{n}}}{\mathbf{N}_{\mathrm{n}}}, \ \overline{\mathbf{S}_{\mathrm{v}}} = \frac{\mathbf{S}_{\mathrm{v}}}{\mathbf{N}_{\mathrm{v}}}, \\ \text{and} \ \overline{\mathbf{I}_{\mathrm{v}}} = \frac{\mathbf{I}_{\mathrm{v}}}{\mathbf{N}_{\mathrm{v}}}, \ \text{then our equations become} \\ \frac{d\overline{\mathbf{S}_{\mathrm{m}}}}{dt} = \mu_{\mathrm{h}} - (\mu_{\mathrm{h}} + \gamma_{\mathrm{vm}}\overline{\mathbf{I}_{\mathrm{v}}}(\mathbf{A}/\mu_{\mathrm{v}}))\overline{\mathbf{S}_{\mathrm{m}}}, \\ \frac{d\overline{\mathbf{I}_{\mathrm{m}}}}{dt} = \gamma_{\mathrm{vm}}\overline{\mathbf{I}_{\mathrm{v}}}(\mathbf{A}/\mu_{\mathrm{v}}))\overline{\mathbf{S}_{\mathrm{m}}} - (\mu_{\mathrm{h}} + \mathbf{r}_{\mathrm{m}})\overline{\mathbf{I}_{\mathrm{m}}}, \\ \frac{d\overline{\mathbf{S}_{\mathrm{n}}}}{dt} = \mu_{\mathrm{h}} - (\mu_{\mathrm{h}} + \mathbf{k}\gamma_{\mathrm{vn}}\overline{\mathbf{I}_{\mathrm{v}}}(\mathbf{A}/\mu_{\mathrm{v}}) + \gamma_{\mathrm{mn}}\overline{\mathbf{I}_{\mathrm{m}}}\mathbf{N}_{\mathrm{m}})\overline{\mathbf{S}_{\mathrm{n}}}, \quad (3) \\ \frac{d\overline{\mathbf{I}_{\mathrm{n}}}}{dt} = (\mathbf{k}\gamma_{\mathrm{vn}}\overline{\mathbf{I}_{\mathrm{v}}}(\mathbf{A}/\mu_{\mathrm{v}}) + \gamma_{\mathrm{nn}}\overline{\mathbf{I}_{\mathrm{m}}}\mathbf{N}_{\mathrm{m}})\overline{\mathbf{S}_{\mathrm{n}}} - (\mu_{\mathrm{h}} + \mathbf{r}_{\mathrm{m}})\overline{\mathbf{I}_{\mathrm{n}}}, \\ \frac{d\overline{\mathbf{I}_{\mathrm{v}}}}{dt} = \gamma_{\mathrm{mv}}\overline{\mathbf{I}_{\mathrm{m}}}\mathbf{N}_{\mathrm{m}}(1 - \overline{\mathbf{I}_{\mathrm{v}}}) + \gamma_{\mathrm{nv}}\overline{\mathbf{I}_{\mathrm{m}}}\mathbf{N}_{\mathrm{m}}(1 - \overline{\mathbf{I}_{\mathrm{v}}}) - \mu_{\mathrm{v}}\overline{\mathbf{I}_{\mathrm{v}}}, \\ \text{with the new three conditions } \overline{\mathbf{S}_{\mathrm{m}}} + \overline{\mathbf{I}_{\mathrm{m}}} = 1, \\ \overline{\mathbf{S}_{\mathrm{n}}} + \overline{\mathbf{I}_{\mathrm{n}}} = 1 \ \text{and} \ \overline{\mathbf{S}_{\mathrm{v}}} + \overline{\mathbf{I}_{\mathrm{v}}} = 1. \quad (4) \end{array}$

III. ANALYSIS OF THE MATHEMATICAL MODEL

A. Equilibrium States

The equilibrium points are obtained by setting the right hand side of all equations in (3) equal to zero. Doing this, we get two equilibrium points are

i) disease free equilibrium point:

$$\mathbf{E}_1 = (1, 0, 1, 0, 0) \tag{5}$$

ii) endemic equilibrium point

$$\mathbf{E}_{2} = (\mathbf{S}_{m}^{*}, \mathbf{I}_{m}^{*}, \mathbf{S}_{n}^{*}, \mathbf{I}_{n}^{*}, \mathbf{I}_{v}^{*})$$
(6)

where

$$\mathbf{S}_{m}^{*} = \frac{\beta_{1}}{\mathbf{I}_{v1}^{*}},$$
(7)

$$I_{m}^{*} = \frac{R_{1}I_{v}^{*}}{M_{1}I_{v1}^{*}},$$
(8)

$$\mathbf{S}_{n}^{*} = \frac{\beta_{2} \mathbf{M}_{1} \mathbf{I}_{v1}^{*}}{\beta_{2} \mathbf{M}_{1} \mathbf{I}_{v1}^{*} + \beta_{1} \mathbf{M}_{1} \mathbf{k} \mathbf{R}_{2} \mathbf{I}_{v}^{*} (\mathbf{I}_{v}^{*} + 1) + \beta_{2} \theta_{1} \mathbf{R}_{1} \mathbf{I}_{v}^{*}}, \qquad (9)$$

$$\mathbf{I}_{n}^{*} = \frac{\beta_{2} \Gamma_{v}^{*} (k \mu_{v} \beta_{2} \beta_{1} M_{1} (1 + \Gamma_{v}) + \mu_{h} \theta_{1} R_{1}))}{\mu_{h} M_{1} (\beta_{2} M_{1} I_{v1}^{*} + k M_{1} R_{2} I_{v}^{*} I_{v1}^{*} + \beta_{2} \theta_{1} R_{1} I_{v}^{*})}, \quad (10)$$

where I_v^* is the positive solution of

$$b_3 I_v^{*^3} + b_2 I_v^{*^2} + b_1 I_v^* + b_0 = 0.$$
 (11)

This corresponds to Descartes' Rule of Signs, there exists one positive solution exactly,

with
$$\beta_1 = \frac{b\beta_{vm}N_m}{\mu_v(N_T + h)}, \beta_2 = \frac{b\beta_{vn}N_n}{\mu_v(N_T + h)},$$

 $R_1 = \frac{b^2\beta_{vm}^2N_m(A/\mu_v)}{\mu_v\mu_h(N_T + h)^2}, R_2 = \frac{b^2\beta_{vn}^2N_n(A/\mu_v)}{\mu_v\mu_h(N_T + h)^2},$
 $M_1 = \frac{\mu_h + r_m}{\mu_h}, \ \theta_1 = \frac{\gamma_{mn}N_m}{\mu_h}, \ \theta_2 = \gamma_{mv}N_m,$
 $\theta_3 = \gamma_{nv}N_n, \ I_{v1}^* = \beta_1 + R_1I_v^*,$
such that

$$\begin{split} b_{3} &= kM_{1}R_{1}^{2}(\beta_{2}^{2}\theta_{3}\mu_{v} + R_{2}\mu_{h}(\theta_{2} + M_{1}\mu_{v})) \,, \\ b_{2} &= R_{1}(-(M_{1}(kR_{2}(R_{1} - \beta_{1}) - R_{1}\beta_{2})\theta_{2} - \\ R_{1}\beta_{2}\theta_{1}(\theta_{2} + \theta_{3}))\mu_{h} + M_{1}(2k\beta_{1}(\beta_{2}^{2}\theta_{3} + M_{1}R_{2}\mu_{h}) \\ &+ R_{1}\beta_{2}(-k\beta_{2}\theta_{3} + (M_{1} + \theta_{1})\mu_{h}))\mu_{v}) \,, \\ b_{1} &= -R_{1}(M_{1}(kR_{2}\beta_{1} + (R_{1} - \beta_{1})\beta_{2})\theta_{2} + \beta_{2}\theta_{1}(-\beta_{1}\theta_{3} + \\ R_{1}(\theta_{2} + \theta_{3}))\mu_{h} + M_{1}\beta_{1}(k\beta_{1}(\beta_{2}^{2}\theta_{3} + M_{1}R_{2}\mu_{h}) + \\ R_{1}\beta_{2}(-2k\beta_{2}\theta_{3} + (2M_{1} + \theta_{1})\mu_{h}))\mu_{v}, \\ b_{0} &= \beta_{1}\beta_{2}(-R_{1}(M_{1}\theta_{2} + \theta_{1}\theta_{3})\mu_{h} + \\ M_{1}\beta_{1}(-k\beta_{2}\theta_{3} + M_{1}\mu_{h})\mu_{v}) \,. \end{split}$$

B. Disease Free Equilibrium Point

For the system defined by (3), the Jacobian matrix evaluated at E_1 is the 5x5 matrix given by

$$\mathbf{J}_{E_{1}} = \begin{bmatrix} -\mu_{h} & 0 & 0 & 0 & -\frac{\mu_{h}\mathbf{R}_{1}}{\beta_{1}} \\ 0 & -\mu_{h}\mathbf{M}_{1} & 0 & 0 & \frac{\mu_{h}\mathbf{R}_{1}}{\beta_{1}} \\ 0 & -\mu_{h}\theta_{1} & -\mu_{h} & 0 & -\frac{\mu_{h}\mathbf{k}\mathbf{R}_{2}}{\beta_{2}} \\ 0 & \mu_{h}\theta_{1} & 0 & -\mu_{h}\mathbf{M}_{1} & \frac{\mu_{h}\mathbf{k}\mathbf{R}_{2}}{\beta_{2}} \\ 0 & \theta_{2} & 0 & \theta_{3} & -\mu_{v} \end{bmatrix}$$

The eigenvalues can be found by solving the following characteristic equation $det(J_{E_1} - \lambda I_5) = 0$, which is $(\mu_h + \lambda)^2 (\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0) = 0$, (13)

$$\mathbf{a}_2 = 2\mathbf{M}_1\mathbf{\mu}_{\mathbf{h}} + \mathbf{\mu}_{\mathbf{v}},$$

with

$$a_{1} = \mu_{h} (M_{1}^{2} \mu_{h} + 2M_{1} \mu_{v} - \frac{R_{1} \theta_{2}}{\beta_{1}} - \frac{kR_{2} \theta_{3}}{\beta_{2}}),$$

$$a_{0} = \mu_{h}^{2} (M_{1}^{2} \mu_{v} - \frac{R_{1} M_{1} \theta_{2}}{\beta_{1}} - \frac{kR_{2} M_{1} \theta_{3}}{\beta_{2}} - \frac{R_{1} \theta_{1} \theta_{3}}{\beta_{1}}).$$

The eigenvalues are $\lambda_1 = \lambda_2 = -\mu_h$, λ_3, λ_4 and λ_5 are found by solving the equation $\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$.

It can be seen that the coefficients a_2, a_1 and a_0 satisfy the Routh-Hurwitz criteria for local stability [15] i) $a_2 > 0$,

ii)
$$a_0 > 0$$
, (14)

iii)
$$a_2 a_1 > a_0$$

when $R_0 < 1$. This means that all eigenvalues will be negative, leading to the first equilibrium point being locally stable where $R_0 = \frac{R_1 M_1 \theta_2 \beta_2 + k R_2 \beta_1 M_1 \theta_3 + R_1 \beta_2 \theta_1 \theta_3}{\beta_1 \beta_2 M_1^2 \mu_v} < 1$.

C. The Endemic Disease Equilibrium Point

The local stability of the endemic disease equilibrium point E_2 is governed by the matrix

$$\mathbf{J}_{E_2} = \begin{bmatrix} -\mu_h - \frac{\mu_h R_1 I_v^*}{\beta_1} & 0 & 0 & 0 & -\frac{\mu_h R_1 S_m^*}{\beta_1} \\ \frac{\mu_h R_1 I_v^*}{\beta_1} & -\mu_h M_1 & 0 & 0 & \frac{\mu_h R_1 S_m^*}{\beta_1} \\ 0 & -\mu_h \theta_l S_n^* & -\mu_h - \frac{\mu_h k R_2 I_v^*}{\beta_2} - \mu_h \theta_l I_m^* & 0 & -\frac{\mu_h k R_2 S_m^*}{\beta_2} \\ 0 & \mu_h \theta_l S_n^* & \frac{\mu_h k R_2 I_v^*}{\beta_2} + \mu_h \theta_l I_m^* & -\mu_h M_1 & \frac{\mu_h k R_2 S_m^*}{\beta_2} \\ 0 & \theta_2 (1 - I_v^*) & 0 & \theta_3 (1 - I_v^*) & -\theta_2 I_m^* - \theta_3 I_n^* - \mu_v \end{bmatrix}$$

where $\mathbf{S}_{m}^{*}, \mathbf{I}_{m}^{*}, \mathbf{S}_{n}^{*}, \mathbf{I}_{n}^{*}$ and \mathbf{I}_{v}^{*} are given by (7)-(11). The characteristic equation for the Jacobian matrix is $(\lambda + \mathbf{M}_{1}\mu_{h})(\lambda^{4} + \mathbf{d}_{3}\lambda^{3} + \mathbf{d}_{2}\lambda^{2} + \mathbf{d}_{1}\lambda + \mathbf{d}_{0}) = 0$. (15) It can be seen that $\lambda_{1} = -\mathbf{M}_{1}\mu_{h}, \lambda_{2}, \lambda_{3}, \lambda_{4}$ and λ_{5} are

found by solving the equation $\lambda^4 + d_3 \lambda^3 + d_2 \lambda^2 + d_1 \lambda + d_0 = 0.$

The stability of the endemic equilibrium point can be determined by using Routh-Hurwitz criteria as follows:

i)
$$d_3 > 0$$
,
ii) $d_1 > 0$, (16)
iii) $d_0 \ge 0$,
iv) $d_3 d_2 d_1 \ge d_1^2 + d_3^2 d_0$.

The endemic equilibrium point is locally stability when

$$R_{0} = \frac{R_{1}M_{1}\theta_{2}\beta_{2} + kR_{2}\beta_{1}M_{1}\theta_{3} + R_{1}\beta_{2}\theta_{1}\theta_{3}}{\beta_{1}\beta_{2}M_{1}^{2}\mu_{v}} > 1.$$
(17)

We present the above four conditions by using the following figures.

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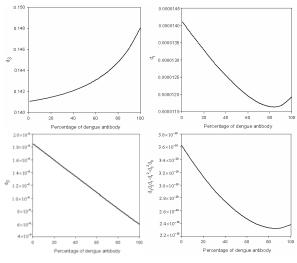


Fig. 4 The parameter space for the endemic equilibrium point which satisfies the Routh-Hurwitz criteria with the value of parameters are A = 500,000, h = 0, k = 0.5, N_T = 10,000, N_m = 5,000, N_n = 5,000, r_m = 1/3 day⁻¹ $\beta_{vm} = \beta_{vn} = \beta_{mv} = \beta_{nv} = 0.1$, $\beta_{mn} = 0.001$, $b = 1/3 day^{-1} \mu_h = 1/(365 \times 70) day^{-1}$, $\mu_v = 1/14 day^{-1}$

The quantity

$$\mathbf{R}_{0}^{'} = \sqrt{\mathbf{R}_{0}} = \sqrt{\frac{\mathbf{R}_{1}\mathbf{M}_{1}\mathbf{\theta}_{2}\beta_{2} + \mathbf{k}\mathbf{R}_{2}\beta_{1}\mathbf{M}_{1}\mathbf{\theta}_{3} + \mathbf{R}_{1}\beta_{2}\mathbf{\theta}_{1}\mathbf{\theta}_{3}}{\beta_{1}\beta_{2}\mathbf{M}_{1}^{2}\mu_{v}}}$$
(18)

is the basic reproduction number, defined as the average number of new infections generated by a single infectious individual in fully susceptible population [16]-[17]. The estimation of \mathbf{R}_0 can determine if dengue sustain its chain of transmission. \mathbf{R}_0 is also referred to as the threshold parameter [18]. It is the most common measure for quantifying the strength of epidemics.

D. Numerical Results

Numerical solutions are shown for comparing the transmission of dengue disease on the free and endemic regions. The values of the parameters used in this study are $\mu_h = 1/(365 \times 70)$ per day, corresponding to a life expectancy of 70 years; $\mu_v = 1/14$ per day, corresponding to a mosquito mean life of 14 days; b = 1/3, one bite providing enough blood meal for three days and there is no alternative host (h = 0). The other parameters are arbitrarily chosen.

The numerical solutions of (3) are shown in following figures.

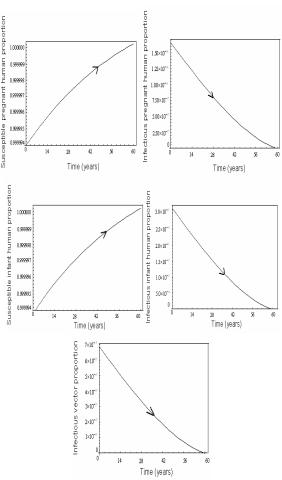
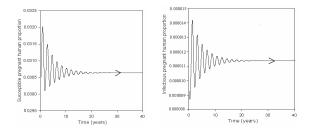


Fig. 5 Numerical solutions of system (3) demonstrate solution of $\overline{S_m}$, $\overline{I_m}$, $\overline{S_n}$, $\overline{I_n}$, $\overline{I_v}$, respectively, for $R_0 < 1$ with A = 140, h = 0, k = 0.5, $N_T = 10,000$, $N_m = 5,000$, $N_n = 5,000$, $\beta_{vm} = \beta_{vn} = \beta_{mv} = \beta_{nv} = 0.1$, $\beta_{mn} = 0.001$, $\mu_h = 1/(365 \times 70) \text{ day}^{-1}$, $b = 1/3 \text{ day}^{-1}$, $r_m = 1/3 \text{ day}^{-1}$, $R_0 = 0.274669$, $\mu_v = 1/14 \text{ day}^{-1}$. The fractions of populations approach to the disease free state



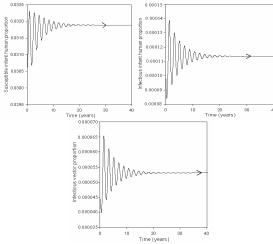


Fig. 6 Numerical solutions of system (3) demonstrate solution of $\overline{S_m}$, $\overline{I_m}$, $\overline{S_n}$, $\overline{I_n}$, $\overline{I_v}$, respectively, for $R_0 > 1$ with $N_n = 5,000$, A = 500,000, h = 0, k = 0.5, $N_T = 10,000$, $N_m = 5,000$, $\beta_{mn} = 0.001$, $b = 1/3 \text{ day}^{-1}$, $\beta_{vm} = \beta_{vn} = \beta_{mv} = \beta_{nv} = 0.1$, $\mu_h = 1/(365 \times 70) \text{ day}^{-1}$, $r_m = 1/3 \text{ day}^{-1}$, $\mu_v = 1/14 \text{ day}^{-1}$, $R_0 = 16.4146$. The fractions of populations converge to the endemic disease state.

Next section, we will compare the numerical solution behaviors when maternal antibodies are difference.

IV. DISCUSSION AND CONCLUSION

The mathematical model which we analyze in our study, the pregnant, infant human and the vector population are assumed to have constant sizes. The number of secondary infections, which can result from one primary infection, is defined from the square root of \mathbf{R}_0 . The quantity $\mathbf{R}'_0 = \sqrt{\mathbf{R}_0}$ is the basic reproductive number of the disease where

$$R_{0} = \frac{R_{1}M_{1}\theta_{2}\beta_{2} + kR_{2}\beta_{1}M_{1}\theta_{3} + R_{1}\beta_{2}\theta_{1}\theta_{3}}{\beta_{1}\beta_{2}M_{1}^{2}\mu_{v}},$$

$$= \frac{R_{1}\theta_{2}}{\beta_{1}M_{1}\mu_{v}} + \frac{kR_{2}\theta_{3}}{\beta_{2}M_{1}\mu_{v}} + \frac{R_{1}\theta_{1}\theta_{3}}{\beta_{1}M_{1}^{2}\mu_{v}}$$

$$= \frac{b\gamma_{mv}\beta_{vm}N_{m}(A/\mu_{v})}{\mu_{v}(N_{T}+h)(\mu_{h}+r_{m})} + \frac{b\beta_{vm}(A/\mu_{v})\gamma_{mn}\gamma_{nv}N_{n}N_{m}}{\mu_{v}(N_{T}+h)(\mu_{h}+r_{m})^{2}\mu_{v}}$$

$$+ \frac{k\gamma_{nv}b\beta_{vn}N_{n}(A/\mu_{v})}{\mu_{v}(N_{T}+h)(\mu_{h}+r_{m})}.$$
(19)
We a consider the third term

We consider the third term

$$\frac{k\gamma_{nv}b\beta_{vn}N_{n}(A/\mu_{v})}{\mu_{v}(N_{T}+h)(\mu_{h}+r_{m})} = \frac{kb\beta_{nv}}{(N_{T}+h)} \cdot \frac{b\beta_{vn}N_{n}(A/\mu_{v})}{\mu_{v}(N_{T}+h)(\mu_{h}+r_{m})} .$$
(20)

It indicates the number of secondary infant cases with maternal antibodies (k). The infective infancy introduced into the susceptible infancy is bitten by $\frac{b(A/\mu_v)}{(N_T + h)} \cdot \frac{1}{(\mu_h + r_m)}$

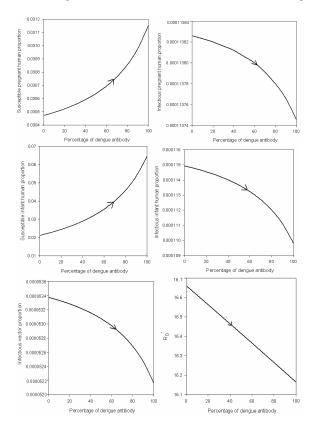
mosquitoes,
$$\beta_{vn} \cdot \frac{b(A/\mu_v)}{(N_T + h)} \cdot \frac{1}{(\mu_h + r_m)}$$
 of these mosquitoes

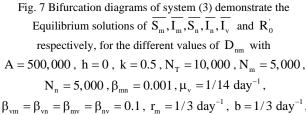
becomes infectious. One of these infectious mosquitoes; b $\begin{pmatrix} N_n \end{pmatrix}$ will in turn hits. Multiplying this number by

 $\frac{b}{\mu_v} \cdot \left(\frac{N_n}{N_T + h} \right)$ will in turn bite. Multiplying this number by

 $k\beta_{nv}$, we get the third term, this number present the infected infancy.

Moreover, we compare the transmission of this disease in human, vector population and the basic reproductive number when the dengue antibodies are difference. We show in Fig. 7.





$$= \beta_{vn} = \beta_{mv} = \beta_{nv} = 0.1, r_m = 1/3 \text{ day}^{-1}, b = 1/3 \text{ day}^{-1}$$
$$\mu_h = 1/(365 \times 70) \text{ day}^{-1}$$

From Fig. 5, all proportions of populations approach to the disease free state (1,0,1,0,0). Fig. 6, the fractions of populations spiral to the endemic disease states (0.0306372, 0.000113806, 0.031889, 0.000113659, 0.0000530724). The imaginary part of complex root of the eigenvalue is approximately 0.0666672. The period of oscillation is 95 days.

The bifurcation diagrams demonstrate the equilibrium solutions of all populations and R_0 for the different values of D_{nm} . If the percentage of dengue antibody which infant received from mother in the beginning is increase, the normalized susceptible pregnant and infant human proportions are increases. But the normalized infectious pregnant, infectious infant, infectious vector population and the basic reproductive number are decreases.

In conclusion, vertical transmission of dengue virus may lead to a full-blown illness in the infant similar to that seen in children and adult cases. Hence, early diagnosis and management of this potentially lethal condition is necessary to reduce perinatal morbidity and mortality, especially in communities where dengue is endemic.

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