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Mathematical Model for Dengue Disease with Maternal Antibodies

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***Abstract*—**Mathematical models can be used to describe the dynamics of the spread of infectious disease between susceptibles and infectious populations. Dengue fever is a re-emerging disease in the tropical and subtropical regions of the world. Its incidence has increased fourfold since 1970 and outbreaks are now reported quite frequently from many parts of the world. In dengue endemic regions, more cases of dengue infection in pregnancy and infancy are being found due to the increasing incidence. It has been reported that dengue infection was vertically transmitted to the infants. Primary dengue infection is associated with mild to high fever, headache, muscle pain and skin rash. Immune response includes IgM antibodies produced by the 5th day of symptoms and persist for 30-60 days. IgG antibodies appear on the 14th day and persist for life. Secondary infections often result in high fever and in many cases with hemorrhagic events and circulatory failure. In the present paper, a mathematical model is proposed to simulate the succession of dengue disease transmission in pregnancy and infancy. Stability analysis of the equilibrium points is carried out and a simulation is given for the different sets of parameter. Moreover, the bifurcation diagrams of our model are discussed. The controlling of this disease in infant cases is introduced in the term of the threshold condition.

***Keywords*—**Dengue infection, equilibrium states, maternal antibodies, pregnancy and infancy.

I. Introduction

AMONG emerging disease, dengue is one of the most important. It occurs in epidemics in Southeast Asia and Western Pacific Regions and comprises a major public health problem. Dengue infection is classified into three categories: Dengue fever (DF), Dengue hemorrhagic fever (DHF) and Dengue shock syndrome (DSS). Dengue fever (DF) is a benign, acute febrile syndrome and it is generally confined to tropical areas and characterized by myalgia or arthralgia, exanthema, leucopenia and lymphadenopathy. Dengue hemorrhagic fever (DHF) is a severe febrile disease of children and adolescents characterized by sudden onset of fever, nausea, vomiting, hepatomegaly, petechial hemorrhagic, epitaxis, melaena and a tendency to delvelop dengue shock sysdrome (DSS) on the fifth to seventh day of illness with significant mortality.

DF, DHF and DSS are caused by dengue virus of the genus Flavivirus, family Flaviviridae. It has four serotypes including dengue virus type 1, 2, 3 and 4 [1]. Infection in humans by one serotype produces life-long immunity against reinfection by the same serotype, but only temporary and partial protection against the other serotypes [2].

The female Aedes aegypti is the major vector for dengue virus transmission. It has been recently shown that infected mosquito requires longer time to acquire blood meal and that may contribute to the efficient transmission of the disease [3]. Longer feeding periods are more likely to be interrupted by the host. It will increase the chance of the infected mosquito who feed on additional hosts.

A primary infection elicits a classic primary-type immunologic response characterized by the initially appearance of dengue antibodies of the immunoglobulin M (IgM) class [4]. Antibody of this immunoglobulin class neutralizes dengue virus and inhibits hemagglutination, but it does not fix complement [5]. Infection with a second member of the genus Flavivirus elicits a secondary-type antibody response. These antibodies fix complement and are predominantly of the immunoglobulin G (IgG) class and raised to antigenic determinants shared by the sequential infecting pairs, and so react broadly with many members of the family [4].

The clinical syndrome was first described in 1779 as “joint fever” by David Bylon in Java [6]. In 1780, Benjamin Rush described an epidemic in Philadelphia under the name “breakbone fever”. Since the 18th century, dengue disease are recurred as epidemic worldwide [7]. However, the hemorrhagic form of this disease was first recognized as a new disease in the Phillipines in 1953 and subsequently became endemic and epidemic in many areas of tropical Asia [8].

Dengue hemorrhagic fever is now an increasing public health problem in most of the countries of tropical areas of the Southeast Asia and Pacific Regions. This disease is the leading cause of hospitalization and death in children in at least eight tropical Asia countries [2]. In Thailand, DHF was first recognized as an epidemic disease of children in Bangkok metropolitan in 1958. There were 2,148 cases with 240 deaths reported. The incidence increased to 5,947 cases in 1962 when the disease started to spread to other big cities, where the communications with Bangkok were accessible. In 1984-1985, the incidences number has reached its highest peak ever

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for over 28 years. There were 69,101 cases with 496 deaths in 1984 and 80,811 cases with 505 deaths in 1985 [8]. The total reported cases were expressed in the form of the averaging of five years (except for the period of 2006-2007) which show overall trend. The table I shows a steadily rise over the whole period between 1961 and 2007. The rate of cases is increasing. The higher rates of increase of 2.6 and 2.7 folds for the early sixties and seventies represented the spread of DHF.

TABLE I
DENGUE HEMORRHAGIC FEVER, THAILAND 1961-2007. FIVE YEAR AVERAGES TOTAL OF REPORTED CASES

Year	Five-year total	Average cases/yr	Ratio increase
1961-1965	20,480	4,096	2.60
1966-1970	25,743	5,148	1.26
1971-1975	69,530	13,906	2.70
1976-1980	115,792	23,158	1.66
1981-1985	224,857	44,971	1.94
1986-1990	395,444	79,089	1.76
1991-1995	263,671	52,734	0.67
1996-2000	313,015	62,603	1.19
2001-2005	402,840	80,568	1.29
2006-2007*	102,410	51,205	0.64

*Two-year period

While DHF/DSS, a serious clinical condition occurs mostly in children between the age of 2 and 9 living in Asia or Asian Pacific region. However, in Southeast Asia where is a hyperendemic area, children below 1 year of age can also develop DHF/DSS [9]. The following figure shows the number of infant cases according to dengue disease between 1997 and 2007 [10].

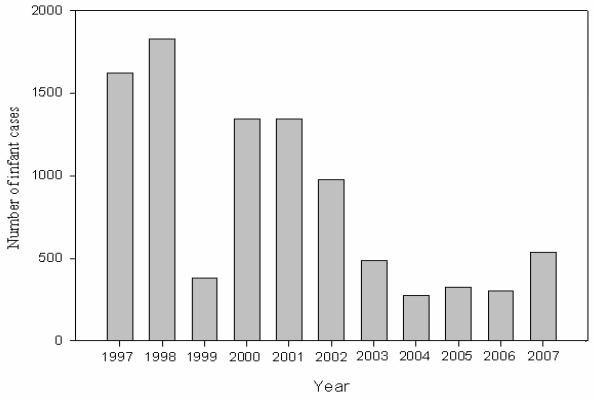


Fig. 1 The number of infant cases due to dengue disease between 1997 and 2007 .

Maternal antibodies have also been shown to be a risk factor for DHF in infants. Because preexisting antibodies have long been held as the causative agent for this enhancement in secondary infections, this is commonly called antibody-dependent enhancement (ADE) [11]-[13]. Infants less than 12 months of age in Bangkok, Thailand infected with dengue viruses 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 [14]-[16]. The severe manifestations occur in infants, they might have acquired antibodies to two dengue virus serotypes by passive transfer of maternal antibodies and sequential exposure to primary infections at early age [17].

Mathematical models have become important tools in analyzing the spread and control of infectious diseases. The process of model formulation clarifies assumptions, variables and parameters. Epidemical modeling can contribute the design and analysis of epidemiological surveys, suggest crucial data that should be collected, identify trends, make general forecasts and estimate the uncertainty in forecasts [18]-[19].

In 1998, Esteva and Vargas [20] proposed the mathematical model for the transmission of dengue fever. They established the global stability of the endemic equilibrium. They discussed the vector population in term of the threshold condition which governs the existence and stability of the endemic equilibrium.

In our studied [21], we formulated mathematical model when the population is separated into pregnant, non-pregnant human and vector classes. The purpose of this study is to study the transmission of dengue disease in a population containing the pregnant, non-pregnant, infant and incorporate effects of the maternal antibody into mathematical model.

Since maternal antibodies of 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 [22]. So that, the maternal dengue antibody disappears in infants by the age of 12 months. The most appropriated age for vaccination with a live-attenuated dengue vaccine in an endemic area is one year of age. The purpose of this paper is to use the mathematical models in understanding and controlling dengue disease in infants, which includes maternal antibody to dengue virus in infant population. In section 2, we propose a mathematical model for describing the transmission of dengue disease in pregnancy and infancy classes. Next section, the analytical result of the model is shown. Finally, section 4 consists of our discussion, conclusion and the numerical solutions of the model.

II. MATHEMATICAL MODEL

Our model is based on susceptible-infected-recovered or SIR model. Maternal antibody to dengue virus is incorporated into our model. The transmission dynamic is described as follows. We divide the human population into two categories, pregnant woman and infant categories. Pregnant woman category is divided into three subcategories, susceptible, infected and recovered classes. Infant population is separated into three subcategories, susceptible, infected and recovered classes. Infected infant is classified into two subgroups, first group is the infectious infant who age not more than 6 months and the second group is the infectious infant who age more than 6 months but not more than 12 months. The vector population is divided into two categories, susceptible and infected mosquitoes, since the mosquitoes never recover from

infection. We assume each category has constant size, susceptible pregnant woman is never infected with dengue virus and infant is defined as the baby who age not more than 12 months.

The dynamic of human population can be described by the following equations

$$\frac{dS_m}{dt} = P - \mu_H S_m - \gamma_{vm} I_v S_m \quad (1.1)$$

$$\frac{dI_m}{dt} = \gamma_{vm} I_v S_m - (\mu_H + r_m) I_m \quad (1.2)$$

$$\frac{dR_m}{dt} = r_m I_m - \mu_H R_m \quad (1.3)$$

$$\frac{dS_n}{dt} = aqP - (\mu_H + k_1\gamma_{vn} I_v + \gamma_{mn} I_m + k_2\gamma_{vn} I_v) S_n \quad (1.4)$$

$$\frac{dI_{n1}}{dt} = (k_1\gamma_{vn} I_v + \gamma_{mn} I_m) S_n - (\mu_H + r_m) I_{n1} \quad (1.5)$$

$$\frac{dI_{n2}}{dt} = k_2\gamma_{vn} I_v S_n - (\mu_H + r_m) I_{n2} \quad (1.6)$$

and $$\frac{dR_n}{dt} = r_m (I_{n1} + I_{n2}) - \mu_H R_n . \quad (1.7)$$

For the mosquito populations, the dynamic of mosquito population can be described as the following equations

$$\frac{dS_v}{dt} = A - (\mu_v + \gamma_{mv} I_m + \gamma_{nv} I_{n1} + \gamma_{nv} I_{n2}) S_v \quad (1.8)$$

$$\frac{dI_v}{dt} = (\gamma_{mv} I_m + \gamma_{nv} I_{n1} + \gamma_{nv} I_{n2}) S_v - \mu_v I_v \quad (1.9)$$

where

S_m represented the number of susceptible pregnant human population,

I_m represented the number of infectious pregnant human population,

R_m represented the number of recovered pregnant human population,

S_n represented the number of susceptible infant population,

I_{n1} represented the number of infectious infant population who age not more than 6 months,

I_{n2} represented the number of infectious infant population who age more than 6 months but not more than 12 months,

R_n represented the number of recovered infant population,

S_v represented the number of susceptible vector population,

I_v represented the number of infectious vector population,

a represented the percentage of infant who be not die while pregnant,

q represented the average number of infant which each woman can have in each time of pregnancy,

P represented the constant recruitment rate of pregnant woman,

N_v represented the total adult mosquitoes,

μ_{H} represented the average constant death rate of pregnant woman,

μ_{v} represented the average constant death rate of vector population,

γ_{vm} represented the transmission rate of dengue virus from vector to mother and the mother is infected,

γ_{vn} represented the transmission rate of dengue virus from vector to infant and infant is infected,

γ_{mv} represented the transmission rate of dengue virus from mother to vector and the vector is infected,

γ_{nv} represented the transmission rate of dengue virus from infant to vector and vector is infected,

γ_{mn} represented the transmission rate of dengue virus from mother to infant and infant is infected,

β_{vm} represented the transmission probability from vector to mother,

β_{vn} represented the transmission probability from vector to infant,

β_{mv} represented the transmission probability from mother to vector,

β_{nv} represented the transmission probability from infant to vector,

β_{mn} represented the transmission probability from mother to infant,

r_m represented the constant rate at which human populations recovers,

N_T represented the total number of human population,

N_m represented the total number of pregnant woman,

N_n represented the total number of infant,

N_v represented the total number of vector population,

A represented the adult mosquito recruitment rate,

D_{nm1} represented the percentage of dengue antibody which infant who age not more than 6 months received from mother in the beginning,

D_{nm2} represented the percentage of dengue antibody which infant who age more than 6 months received from mother in the beginning,

k_1 represented the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby not more than 6 months,

k_2 represented the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby more than 6 months.

We assume $k_1 = \frac{100 - D_{nm1}}{100}, k_1 = \frac{100 - D_{nm2}}{100}$ and $k_2 > k_1$. Therefore, our model consists of equations (1.1)-(1.9) with three conditions $S_m + I_m + R_m = N_m$, $S_n + I_{n1} + I_{n2} + R_n = N_n$ and $S_v + I_v = N_v$.

Introducing the normalized parameters $\overline{S_m} = \frac{S_m}{N_m}$, $\overline{I_m} = \frac{I_m}{N_m}, \overline{R_m} = \frac{R_m}{N_m}, \overline{S_n} = \frac{S_n}{N_n}, \overline{I_{n1}} = \frac{I_{n1}}{N_n}, \overline{I_{n2}} = \frac{I_{n2}}{N_n}$, $\overline{R_n} = \frac{R_n}{N_n}, \overline{S_v} = \frac{S_v}{N_v}$, and $\overline{I_v} = \frac{I_v}{N_v}$, equations (1.1) to (1.9) reduce to

$$\frac{d\overline{S_m}}{dt} = \mu_H - (\mu_H + \gamma_{vm}(A/\mu_v)\overline{I_v})\overline{S_m} \quad (2.1)$$

$$\frac{d\overline{I_m}}{dt} = \gamma_{vm}(A/\mu_v)\overline{I_v}\,\overline{S_m} - (\mu_H + r_m)\overline{I_m} \quad (2.2)$$

$$\frac{d\overline{S_n}}{dt} = \mu_H - (\mu_h + k_1\gamma_{vn}(A/\mu_v)\overline{I_v} + \gamma_{mn}\overline{I_m}N_m + k_2\gamma_{vn}(A/\mu_v)\overline{I_v})\overline{S_n} \quad (2.3)$$

$$\frac{d\overline{I_{n1}}}{dt} = (k_1\gamma_{vn}(A/\mu_v)\overline{I_v} + \gamma_{mn}\overline{I_m}N_m)\overline{S_n} - (\mu_H + r_m)\overline{I_{n1}} \quad (2.4)$$

$$\frac{d\overline{I_{n2}}}{dt} = k_2\gamma_{vn}(A/\mu_v)\overline{I_v}\,\overline{S_n} - (\mu_H + r_m)\overline{I_{n2}} \quad (2.5)$$

and $$\frac{d\overline{I_v}}{dt} = (\gamma_{mv}\overline{I_m}N_m + \gamma_{nv}\overline{I_{n1}}N_n + \gamma_{nv}\overline{I_{n2}}N_n)(1-\overline{I_v}) - \mu_v\overline{I_v} \quad (2.6)$$

where $\gamma_{vm} = \frac{b\beta_{vm}}{N_T + h}$, $\gamma_{vn} = \frac{b\beta_{vn}}{N_T + h}$, $\gamma_{mv} = \frac{b\beta_{mv}}{N_T + h}$, $\gamma_{nv} = \frac{b\beta_{nv}}{N_T + h}$ and $\gamma_{mn} = \beta_{mn}$.

The dynamic equations for R_m, R_n and S_v are not needed since $\overline{S_m} + \overline{I_m} + \overline{R_m} = 1, \overline{S_n} + \overline{I_{n1}} + \overline{I_{n2}} + \overline{R_n} = 1$ and $\overline{S_v} + \overline{I_v} = 1$. The requirements that N_T, N_m, N_n and N_v are constant lead to the conditions that $(P + aqP) = \mu_H N_T$, $P = \mu_H N_m$, $aqP = \mu_H N_n$ and $A = \mu_v N_v$.

III. Analysis of the Mathematical Model

A. Equilibrium Points

The equilibrium points are obtained by setting the right hand side of equations (2.1)-(2.6) equal to zero. Doing this, we get two equilibrium points

i) the disease free state

$$E_1 = (1,0,1,0,0,0) \quad (3.1)$$

ii) the endemic disease state

$$E_2 = (S_m^*, I_m^*, S_n^*, I_{n1}^*, I_{n2}^*, I_v^*) \quad (3.2)$$

where

$$S_m^* = \frac{\beta_1}{\beta_1 + R_1 I_v^*}, \quad (3.3)$$

$$I_m^* = \frac{R_1\beta_1}{M_1(\beta_1 + R_1 I_v^*)}, \quad (3.4)$$

$$S_n^* = \frac{\beta_2 M_1(\beta_1 + R_1 I_v^*)}{(\beta_1 + R_1 I_v^*)(\beta_2 M_1 + k_1 R_2 M_1 I_v^* + k_2 R_2 M_1 I_v^*) + \beta_2\theta_1 R_1 I_v^*}, \quad (3.5)$$

$$I_{n1}^* = \frac{k_1 R_2 M_1 I_v^*(\beta_1 + R_1 I_v^*) + \beta_2\theta_1 R_1 I_v^*}{M_1((\beta_1 + R_1 I_v^*)(\beta_2 M_1 + k_1 R_2 M_1 I_v^* + k_2 R_2 M_1 I_v^*) + \beta_2\theta_1 R_1 I_v^*)} \quad (3.6)$$

$$I_{n2}^* = \frac{k_2 R_2 I_v^*(\beta_1 + R_1 I_v^*)}{(\beta_1 + R_1 I_v^*)(\beta_2 M_1 + k_1 R_2 M_1 I_v^* + k_2 R_2 M_1 I_v^*) + \beta_2\theta_1 R_1 I_v^*} \quad (3.7)$$

with I_v^* are solutions of

$$b_3(I_v^*)^3 + b_2(I_v^*)^2 + b_1(I_v^*) + b_0 = 0 \quad (3.8)$$

where

$$b_3 = (k_1 + k_2)M_1 R_1^2 R_2(\theta_2 + \theta_3 + M_1\mu_v), \quad (4.1)$$

$$b_2 = R_1((k_1 + k_2)M_1 R_2 T_1 + R_1\beta_2(\theta_1(\theta_2 + \theta_3) + M_1^2\mu_v + M_1(\theta_2 + \theta_1\mu_v))) \quad (4.2)$$

$$b_1 = (k_1 + k_2)M_1 R_2\beta_1 T_1 + R_1\beta_2(-\theta_1(-\beta_1\theta_3 + R_1(\theta_2 + \theta_3)) + 2M_1^2\beta_1\mu_v + M_1(-R_1\theta_2 + \beta_1(\theta_2 + \theta_1\mu_v))) \quad (4.3)$$

$$b_0 = \beta_1(-R_1\beta_2\theta_1\theta_3 - M_1(R_1\beta_2\theta_2 + (k_1 + k_2)R_2\beta_1\theta_3) + M_1^2\beta_1\beta_2\mu_v) \quad (4.4)$$

where

$T_1 = (-R_1(\theta_2 + \theta_3) + \beta_1(\theta_2 + 2\theta_3 + M_1\mu_v))$,

$T_2 = (-R_1(\theta_2 + 2\theta_3) + \beta_1(\theta_3 + M_1\mu_v))$

with

$\beta_1 = \frac{b\beta_{vm}N_m}{\mu_v(N_T + h)}$, $\beta_2 = \frac{b\beta_{vn}N_n}{\mu_v(N_T + h)}$, $R_1 = \frac{b^2\beta_{vm}^2 N_m(A/\mu_v)}{\mu_v\mu_H(N_T + h)^2}$,

$R_2 = \frac{b^2\beta_{vn}^2 N_n(A/\mu_v)}{\mu_v\mu_H(N_T + h)^2}$, $M_1 = \frac{\mu_H + r_m}{\mu_H}$, $\theta_1 = \frac{\gamma_{mn}N_m}{\mu_H}$,

$\theta_2 = \gamma_{mv}N_m$ and $\theta_3 = \gamma_{nv}N_n$.

After we check the sign of b_3, b_2 and b_1 are positive. b_0 is negative when $\frac{R_1\beta_2\theta_1\theta_3 + M_1R_1\beta_2\theta_2 + (k_1 + k_2)M_1R_2\beta_1\theta_3}{M_1^2\beta_1\beta_2\mu_v} > 1$. So the solutions of (3.8) exist one positive solution that correspondence with (3.8) following Descartes' Rule of Signs.

B. Local Asymptotical Stability

The local stability for each equilibrium point is determined from the signs of eigenvalues of the Jacobian matrix of the right hand side of the above set of differential equations. If all eigenvalues have negative real part, then that equilibrium point is local stability [22].

C. Disease Free State

For the system defined by (2.1) to (2.6), the Jacobian matrix evaluated at E_1 is the 6x6 matrix given by

$$J_{E_1}=\begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & -\frac{\mu_H R_1}{\beta_1} \\ 0 & -\mu_H M_1 & 0 & 0 & 0 & \frac{\mu_H R_1}{\beta_1} \\ 0 & -\mu_H\theta_1 & -\mu_H & 0 & 0 & -\frac{k_1\mu_H R_2}{\beta_2}-\frac{k_2\mu_H R_2}{\beta_2} \\ 0 & \mu_H\theta_1 & 0 & -\mu_H M_1 & 0 & \frac{k_1\mu_H R_2}{\beta_2} \\ 0 & 0 & 0 & 0 & -\mu_H M_1 & \frac{k_2\mu_H R_2}{\beta_2} \\ 0 & \theta_2 & 0 & \theta_3 & \theta_3 & -\mu_v \end{bmatrix}$$

The eigenvalues are obtained by solving the characteristic equation; $\det(J_{E_1}-\lambda I_6)=0$ where I_6 is the identity matrix size 6x6. The characteristic equation for the disease free state is given by

$$(\lambda+\mu_H)^2(\lambda+M_1\mu_H)(\lambda^3+a_2\lambda^2+a_1\lambda+a_0)=0 \qquad (6.1)$$

where

$$a_2=2M_1\mu_H+\mu_v, \qquad (6.2)$$

$$a_1=\frac{\mu_H(-R_1\beta_2\theta_2+\beta_1(-(k_1+k_2)R_2\theta_3+M_1\beta_2(M_1\mu_H+2\mu_v)))}{\beta_1\beta_2} \qquad (6.3)$$

$$a_0=\frac{\mu_H^2(-R_1\beta_2\theta_1\theta_3-M_1(R_1\beta_2\theta_2+(k_1+k_2)R_2\beta_1\theta_3)+M_1^2\beta_1\beta_2\mu_v)}{\beta_1\beta_2} \qquad (6.4)$$

From the characteristic equation (6.1), the first three eigenvalues are $\lambda_1=\lambda_2=-\mu_H$ and $\lambda_3=-M_1\mu_H$. The remaining three eigenvalues are found by solving $\lambda^3+a_2\lambda^2+a_1\lambda+a_0=0$.

These eigenvalues are negative when the coefficients a_0, a_1 and a_2 satisfy the Routh-Hurwitz criteria [23]

i) $a_2>0$,

ii) $a_0>0$,

iii) $a_2a_1>a_0$,

We can see that a_2 is always positive. Next, we consider the second and third conditions. We found that $a_0>0$ and $a_2a_1-a_0>0$ when

$$M_1^2\beta_1\beta_2\mu_v>R_1\beta_2\theta_1\theta_3+M_1R_1\beta_2\theta_2+(k_1+k_2)M_1R_2\beta_1\theta_3$$

or $$\frac{R_1\beta_2\theta_1\theta_3+M_1R_1\beta_2\theta_2+(k_1+k_2)M_1R_2\beta_1\theta_3}{M_1^2\beta_1\beta_2\mu_v}<1.$$

All three conditions of Routh-Hurwitz criteria are satisfied for $R_0<1$, where

$$R_0=\frac{R_1\beta_2\theta_1\theta_3+M_1R_1\beta_2\theta_2+(k_1+k_2)M_1R_2\beta_1\theta_3}{M_1^2\beta_1\beta_2\mu_v}.$$

This means that all eigenvalues will be negative, leading to the disease free state being locally stable.

D. Endemic Disease State

The local stability of the endemic state, E_2, is governed by the matrix

$$J_{E_2}-\lambda I_6=\begin{bmatrix} a_{11} & 0 & 0 & 0 & 0 & a_{16} \\ a_{21} & a_{22} & 0 & 0 & 0 & a_{26} \\ 0 & a_{32} & a_{33} & 0 & 0 & a_{36} \\ 0 & a_{42} & a_{43} & a_{44} & 0 & a_{46} \\ 0 & 0 & a_{53} & 0 & a_{55} & a_{56} \\ 0 & a_{62} & 0 & a_{64} & a_{65} & a_{66} \end{bmatrix}$$

where

$$a_{11}=-\mu_H-\frac{\mu_H R_1 I_v^*}{\beta_1}-\lambda,\ a_{16}=-\frac{\mu_H R_1 S_m^*}{\beta_1},\ a_{21}=\frac{\mu_H R_1 I_v^*}{\beta_1}$$

$$a_{22}=a_{44}=a_{55}=-\mu_H M_1-\lambda,\ a_{26}=\frac{\mu_H R_1 S_m^*}{\beta_1},\ a_{32}=-\mu_H\theta_1 S_n^*,$$

$$a_{33}=-[\mu_H+\frac{k_1\mu_H R_2 I_v^*}{\beta_2}+\frac{k_2\mu_H R_2 I_v^*}{\beta_2}+\mu_H\theta_1 I_m^*]-\lambda,$$

$$a_{36}=-\frac{k_1\mu_H R_2 S_n^*}{\beta_2}-\frac{k_2\mu_H R_2 S_n^*}{\beta_2},\ a_{42}=\mu_H\theta_1 S_n^*$$

$$a_{43}=\frac{k_1\mu_H R_2 I_v^*}{\beta_2}+\mu_H\theta_1 I_m^*, a_{46}=\frac{k_1\mu_H R_2 S_n^*}{\beta_2}, a_{53}=\frac{k_2\mu_H R_2 I_v^*}{\beta_2},$$

$$a_{56}=\frac{k_2\mu_H R_2 S_n^*}{\beta_2},\ a_{62}=\theta_2(1-I_v^*),\ a_{64}=a_{65}=\theta_3(1-I_v^*),$$

$$a_{66}=-\theta_2 I_m^*-\theta_3 I_{n1}^*-\theta_3 I_{n2}^*-\mu_v-\lambda.$$

The characteristic equation for the endemic state is given by

$$(\lambda+M_1\mu_H)(\lambda^5+a_4\lambda^4+a_3\lambda^3+a_2\lambda^2+a_1\lambda+a_0)=0 \qquad (7.1)$$

where

$$a_4=\frac{\beta_1\beta_2(\mu_v+\theta_2 I_m^*+\mu_H I_{n1}^*+\theta_3 I_{N1}^*)+(k\beta_1+R_1\beta_2)\mu_H I_v^*}{\beta_1\beta_2} \qquad (7.2)$$

$$a_3=\frac{\mu_H(-R_1\beta_2\theta_2+\beta_1(-k\theta_3+\beta_2((1+M_1(4+M_1))\mu_H+2Q_1\mu_v)))}{\beta_1\beta_2}$$

$$+\frac{1}{\beta_1\beta_2}(\mu_H(R_1(k\mu_H I_v^{*2}+\beta_2((\mu_H(I_{m1}^*-1)+I_{N2}^*)I_v^*+\theta_2 I_{m2}^*))$$

$$+\beta_1(\beta_2(\theta_2 I_m^* I_{m1}^*+2Q_1\theta_3 I_{N1}^*+\theta_1 I_m^*(\mu_1+\theta_3 I_{N1}^*))+k((\mu_1+\theta_2 I_m^*)I_v^*$$

$$+\theta_3 I_{v4}^*)))) \qquad (7.3)$$

$$a_2=((\mu_H^2(kQ_3+\beta_2(R_1(-(Q_2+\theta_1)\theta_2-\theta_1\theta_3+\beta_1(\mu_v+M_1(2Q_1\mu_v$$

$$+(4+M_1)\mu_v)))))/\beta_1\beta_2)+\frac{1}{\beta_1\beta_2}(\mu_H^2(\beta_1(\beta_2(\theta_2 I_{mv}^*(1+4M_1$$

$$+M_1^2+Q_4\theta_1 I_m^*)+\theta_3 I_{n1}^*+\theta_1 I_m^* I_{N1}^*+\theta_3 I_{m4}^* I_{n2}^*+M_1^2(\theta_1\mu_H I_m^*+\theta_3 I_{N1}^*)$$

$$+2M_1(2\theta_3 I_{N1}^*+\theta_1 I_m^*(\mu_H+I_{N2}^*)))+k((I_{m3}^*+M_1(Q_2\mu_H+2I_{m3}^*))I_v^*$$

$$+\theta_3(I_{N1}^* I_{v1}^*+Q_2 R_{n1}^*)))+R_1(\beta_2((\mu_v+M_1(Q_2\mu_H+2\mu_v))I_v^*+Q_4\theta_3 I_{N1}^* I_v^*$$

$$+\theta_2(\theta_1 I_m^{*2} I_{v7}^*+(Q_2+\theta_1)R_m^*+(\theta_1+Q_2 I_v^*)S_m^*+I_m^*(2+M_1 I_{v6}^*+\theta_1 R_m^*$$

$$+I_v^*(1+\theta_3 S_m^*)))+\theta_1(\mu_2 I_m^* I_v^*+\theta_3(I_m^*(1+I_{N1}^* I_v^*)+R_m^*+S_m^* I_{v3}^*)))$$

$$+k(I_v^*(\mu_v I_v^*+\theta_2 I_{m2}^*+\theta_3 I_{v4}^*)+S_v^*)))) \qquad (7.4)$$

$$a_1 = ((\mu_H^3(-k_1R_2(Q_4\beta_1\theta_3 + Q_1R_1(\theta_2+\theta_3)) - k_2R_2(Q_4\beta_1\theta_3 + R_1(Q_2\theta_2 + Q_1\theta_3)) - \beta_2(R_1((1+\theta_1+M_1(2+\theta_1))\theta_2 + 2\theta_1\theta_3) - M_1\beta_1(M_1\mu_H + 2Q_1\mu_v))))/\beta_1\beta_2) + \frac{1}{\beta_1\beta_2}(\mu_H^3(\beta_2(M_1\beta_1(\theta_2 I_m^*(2Q_1 + Q_2\theta_1 I_m^*) + 2\theta_3 I_{N1}^* + \theta_1 I_m^* I_{N2}^*) + M_1(2\theta_3 I_{N1}^* + \theta_1 I_m^*(\mu_H + I_{N2}^*))) + R_1(M_1(2\mu_v + M_1(\mu_H+\mu_v))I_v^* + M_1Q_2\theta_3 I_{N1}^* I_v^* + \theta_2(\theta_1 I_m^{*2}(I_{m1}^* - 1) + (1+\theta_1+M_1(2+\theta_1))R_m^* + (Q_1\theta_1 + Q_4 I_v^*)S_m^* + I_m^*(1+\theta_1 R_m^* + M_1(2+Q_2 I_v^* + \theta_1 R_m^*) + Q_1\theta_1 I_v^* S_m^*)) + \theta_1(M_1\mu_3 I_m^* I_v^* + 2\theta_3(I_m^*(1 + M_1 I_{N1}^* I_v^*) + R_m^* + S_m^*(I_{N1}^* + R_n^* + I_v^* + S_n^*))))) + k_2R_2(\beta_1(M_1(\mu_3 + M_1\mu_v + Q_2\theta_2 I_m^*)I_v^* + \theta_3(I_{n1}^* I_v^*) + I_{n2}^* I_{v2}^*) + Q_4 R_{n1}^*)) + R_1(I_v^*(M_1\mu_3 I_v^* + \theta_3(I_{n2}^* I_{v5}^* + I_{n1}^*(1+M_1 I_{v6}^*) + Q_1 + \theta_3(Q_1 I_n^* + R_n^* + M_1(2I_{N1}^* I_v^* + R_n^*) + Q_1 I_v^* S_n^*)) + Q_1 R_{n1}^*)) + Q_1\theta_3 S_v^* + \theta_2(R_m^* + I_v^*(1 + M_1 R_m^* + Q_1 I_v^* S_m^*) + (M_1 + R_m^* + 2S_m^*))S_v^* + I_m^*(1+M_1 I_v^*)I_{v6}^* + S_v^*)))) + k_1R_2(M_1^2 I_v^*(\beta_1(\mu_h + I_{m3}^* + \theta_3 I_{N1}^*) + R_1\mu_H I_v^*) + \beta_1\theta_3 I_{v3}^* + R_1(I_v^*(\theta_2 I_{m1}^* + \theta_3 I_{v3}^*) + S_{v1}^*) + M_1(2\beta_1(I_{m3}^* I_v^* + \theta_3 I_{v4}^*) + R_1(2\mu_v I_v^{*2} + \theta_3(I_v^*)(I_{n2}^* + I_{n1}^*(I_{n1}^* I_{v6}^* + R_n^* + I_v^*(2I_{n2}^* + S_n^*)) + S_v^*) + \theta_2(I_m^*(1 + 2I_v^{*2}) + R_m^* + S_m^*(I_v^{*2} + S_v^*)))))) \quad (7.5)$$

$$a_0 = ((-\mu_H^4(k_2M_1R_2(R_1\theta_2 + (R_1+\beta_1)\theta_3) + k_1M_1R_2(R_1\theta_3 + R_1(\theta_2+\theta_3)) + \beta_2(R_1(M_1(1+\theta_1)\theta_2 + \theta_1\theta_3) - M_1^2\beta_1\mu_v)))/\beta_1\beta_2) + \frac{1}{\beta_1\beta_2}(\mu_H^4(M_1^2(R_1 I_{m3}^* + \theta_3 I_{N1}^*)I_v^*(\beta_2 I_{m4}^* + kI_v^*) + \beta_1(\beta_2(\theta_2 I_m^*)I_{m4}^* + \theta_3 I_{N1}^* + \theta_1 I_m^* I_{N2}^*) + k(\theta_1 I_m^* + I_{N2}^*)I_v^*)) + R_1\beta_2\theta_1\theta_3(I_m^* + R_m^* + S_m^* I_{v3}^* + M_1(k\beta_1\theta_3 I_{v3}^* + R_1(\beta_2\theta_2(\theta_1 I_m^{*2} + (1+\theta_1)R_m^* + (\theta_1 + I_v^*)S_m^* + I_m^*(1 + \theta_1(R_m^* + I_v^* S_m^*))) + k(I_v^*(\theta_2 R_{m1}^* + \theta_3 I_{v3}^*) + S_{v1}^*))))). \quad (7.6)$$

where

$(I_{n1}^* + I_{n2}^*) = I_{N1}^*$, $(R_n^* + I_v^* S_n^*) = R_{n1}^*$, $I_m^* + R_m^* + I_v^* S_m^* = R_{m1}^*$,
$(2 + M_1 + (1+2M_1)I_v^*) = I_{v1}^*$, $(1 + M_1(2 + (2+M_1)I_v^*) = I_{v2}^*$,
$(I_{N1}^* + R_n^* + I_v^* S_n^*) = I_{v3}^*$, $(1 + M_1 + 2M_1 I_v^*) = I_{v5}^*$, $1 + 2I_v^* = I_{v6}^*$,
$(I_{n2}^* + I_{n1}^*(1+I_v^*) + R_n^* + I_v^*(I_{n2}^* + S_n^*)) = I_{v4}^*$, $1 + I_v^* = I_{v7}^*$,
$1 + \theta_1 I_m^* = I_{m4}^*$, $(2 + 2M_1 + \theta_1 I_m^*) = I_{m1}^*$, $\mu_v + \theta_2 I_m^* = I_{m3}^*$,
$(I_m^*(1+I_v^*) + R_m^* + I_v^* S_m^*) = I_{m2}^*$, $(\theta_2 + \theta_3)S_v^* = S_{v1}^*$, $(k_1 + k_2)R_2 = k$,
$(1+2M_1)\mu_H + \mu_v = \mu_1$, $(2M_1\mu_H + \mu_v) = \mu_2$, $(M_1\mu_H + 2\mu_v) = \mu_3$.

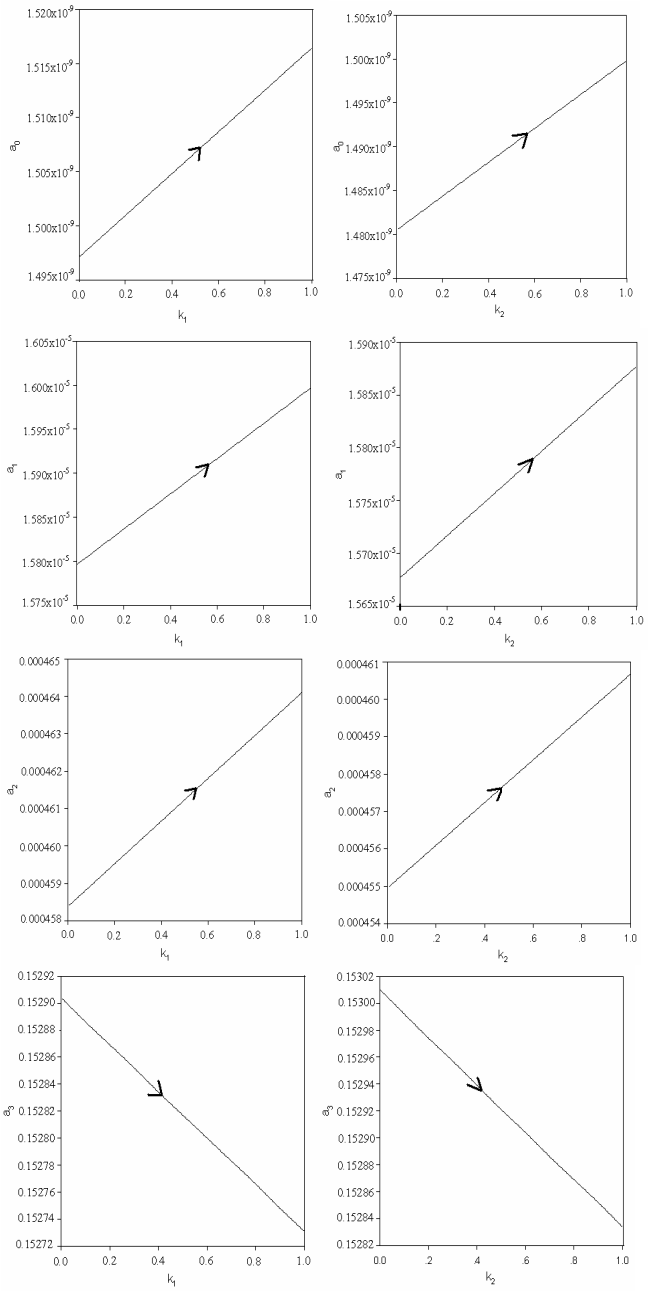
The first eigenvalue is $\lambda_1 = -M_1\mu_H$. It is always negative, the other eigenvalues $\lambda_2, \lambda_3, \lambda_4, \lambda_5$ and λ_6 are found by solving $\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + \lambda = 0$. To determine the local stability of the endemic equilibrium state, we need to check the signs of all eigenvalues for the endemic equilibrium state. The stability of the endemic equilibrium state can be determined by using Routh-Hurwitz criteria as follows:

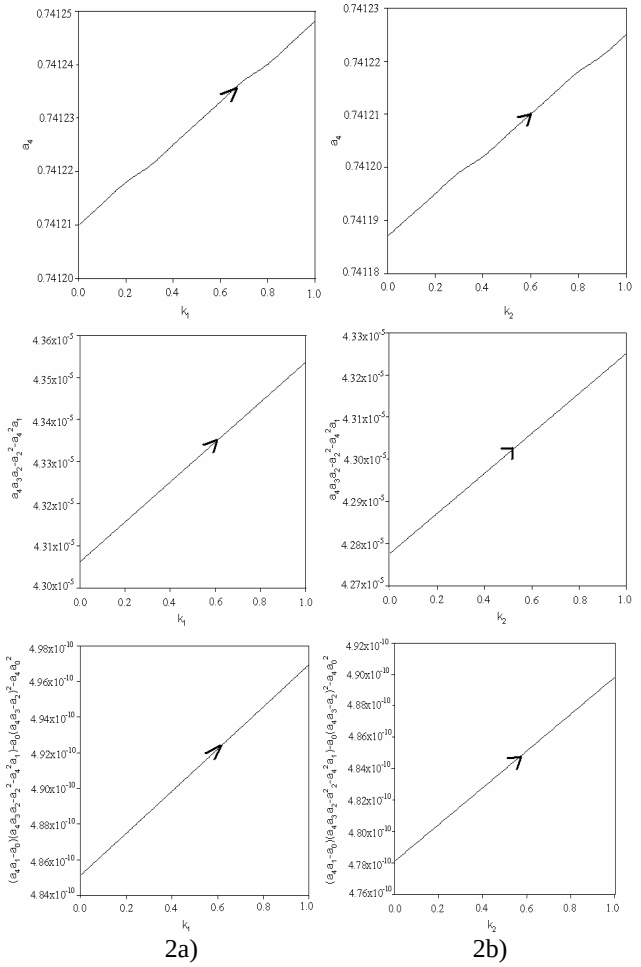
i) $a_i > 0, \forall i, i = 0,1,2,3,4$,

ii) $a_4a_3a_2 > a_2^2 + a_4^2a_1$,

iii) $(a_4a_1 - a_0)(a_4a_3a_2 - a_2^2 - a_4^2a_1) > a_0(a_4a_3 - a_2)^2 + a_4a_0^2$.

We present the above three conditions by using the following figures, by mapping out the regions in $a_i - k_j$ phase space, $(a_4a_3a_2 - a_2^2 - a_4^2a_1) - k_j$ phase space and $((a_4a_1 - a_0)(a_4a_3a_2 - a_2^2 - a_4^2a_1) - a_0(a_4a_3 - a_2)^2 - a_4a_0^2) - k_j$ phase space in which the three above conditions are found when $i = 0,1,2,3,4$, $j = 1,2$.





2a) 2b)

Fig. 2. The parameter space for the endemic equilibrium point which satisfies the Routh-Hurwitz criteria with the value of parameters: $\mu_H = 0.000039139\ day^{-1}$, $N_T = 10,000$, $\mu_v = 0.071428571\ day^{-1}$, $b = 0.33333\ day^{-1}$, $h = 0$, $r_m = 0.33333\ day^{-1}$, $N_m = 5,000$, $N_n = 5,000$, $A = 20,000$, $\beta_{\text{vm}} = 0.1$, $\beta_{\text{vn}} = 0.1$, $\beta_{\text{mv}} = 0.1$, $\beta_{\text{nv}} = 0.1$, $\beta_{\text{mn}} = 0.01$, 2a) $k_2 = 0.9,\ 0 \le k_1 \le 1$, 2b) $k_1 = 0.3,\ 0 \le k_2 \le 1$.

Thus, the endemic equilibrium state is locally stable when $R_0 > 1$.

E. Numerical Results

In this section, the numerical solutions are shown for the disease free and endemic regions. Parameters are used in this study correspond to the real life observations. $\mu_H = 0.000039139$ per day corresponds to life expectancy of 70 years for human population. Since maternal antibodies level of dengue virus in infants are disappeared in 72% by six months of age. So that, we assume $k_2 > k_1$. Total human population (N_T) is assumed to be equal to 10,000. $\beta_{vm}, \beta_{vn}, \beta_{mv}, \beta_{nv}$ and β_{mn} are arbitrary chosen; $\beta_{vm} = \beta_{vn} = \beta_{mv} = \beta_{nv} = 0.1$ and $\beta_{mn} = 0.01$. The mean life of mosquito is 14 days that is $\mu_v = 0.071428571$ per day. The biting rate of the vector population is 1/3 per day; We assume each category has constant size, susceptible pregnant woman is never infected with dengue virus and infant is defined as the baby who age not more than 12 months.

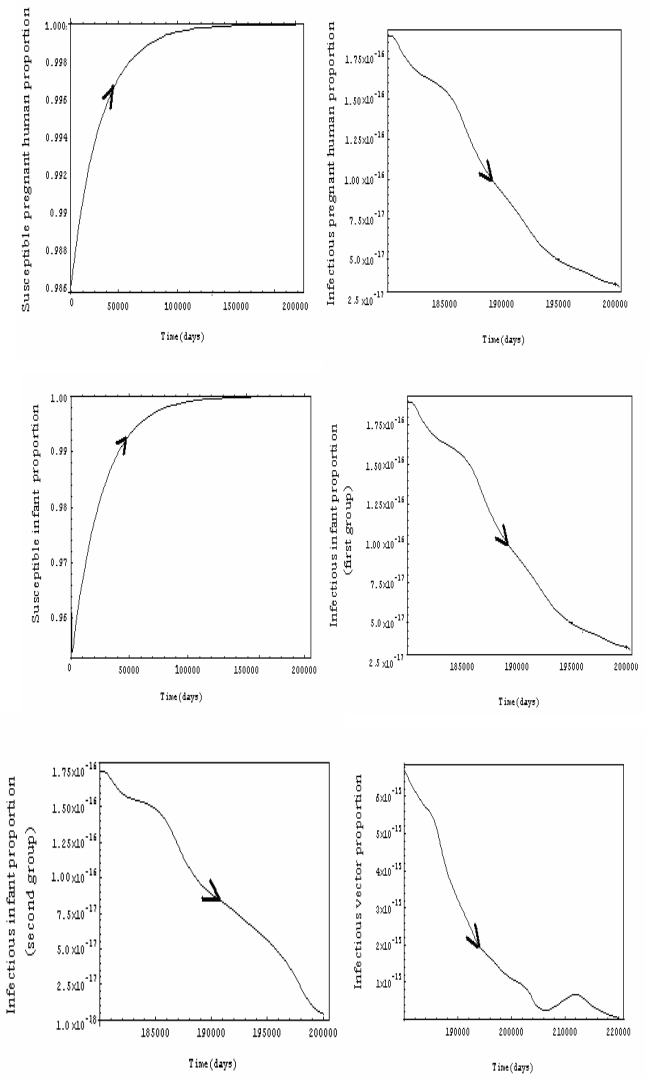


Fig. 3. Time series of susceptible pregnant human, infectious pregnant human, susceptible infant human, first infectious group of infant , second infectious group of infant and infectious vector proportions, respectively. The values of parameters are $\mu_H = 0.000039139\ day^{-1}$, $N_T = 10,000$, $\mu_v = 0.071428571\ day^{-1}$, $b = 0.33333\ day^{-1}$, $h = 0$, $r_m = 0.33333\ day^{-1}$, $N_m = 5,000$, $N_n = 5,000$, $A = 200$, $\beta_{\text{vm}} = 0.1$, $\beta_{\text{vn}} = 0.1$, $\beta_{\text{mv}} = 0.1$, $\beta_{\text{nv}} = 0.1$, $\beta_{\text{mn}} = 0.01$ $k_1 = 0.3, k_2 = 0.9, R_0 = 0.994142, R_0^{'} = 0.997066$. The fractions of populations oscillate to the disease free equilibrium state $(1,0,1,0,0,0)$.

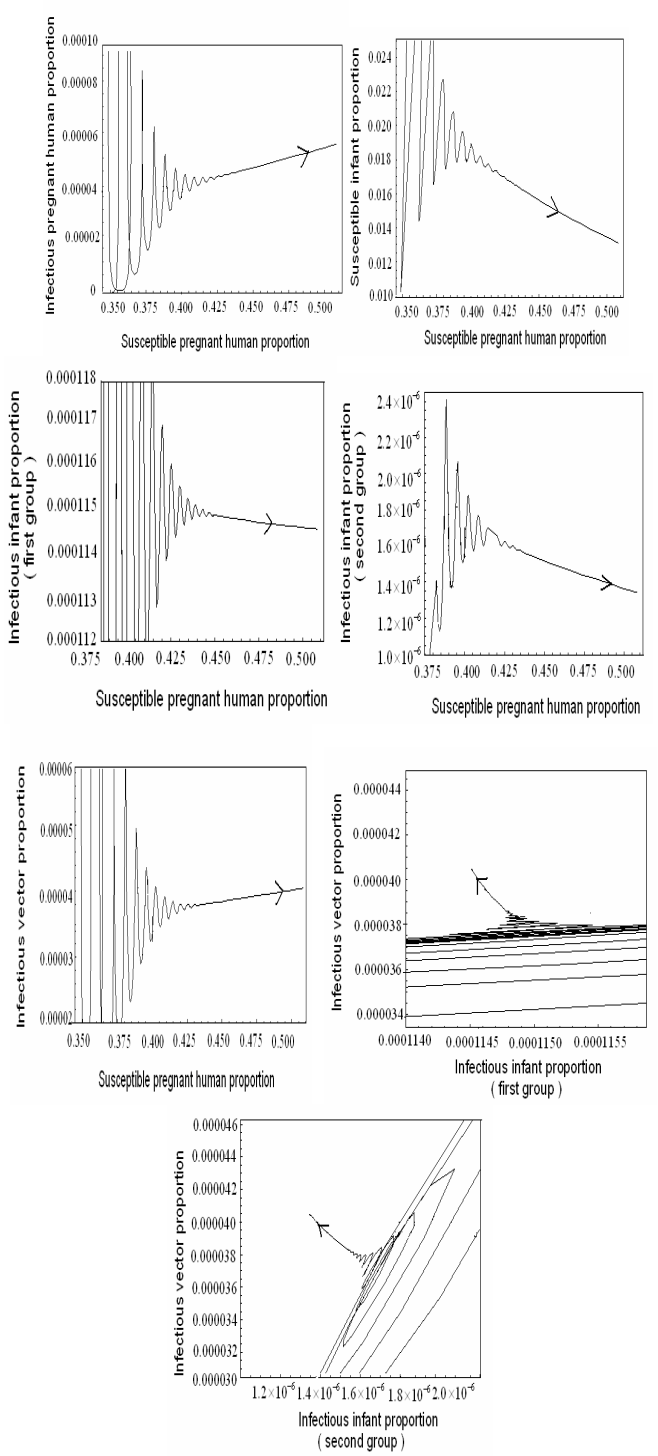


Fig. 4. Numerical solutions demonstrate the solution trajectories, projected onto $(\overline{S_m}, \overline{I_m}), (\overline{S_m}, \overline{S_n}), (\overline{S_m}, \overline{I_{n1}}),$ $(\overline{S_m}, \overline{I_{n2}}), (\overline{S_m}, \overline{I_v}), (\overline{I_{n1}}, \overline{I_v}), (\overline{I_{n2}}, \overline{I_v})$ for $R_0 > 1$ respectively, with the value of parameters are

$\mu_H = 0.000039139\ day^{-1}$, $N_T = 10,000$,

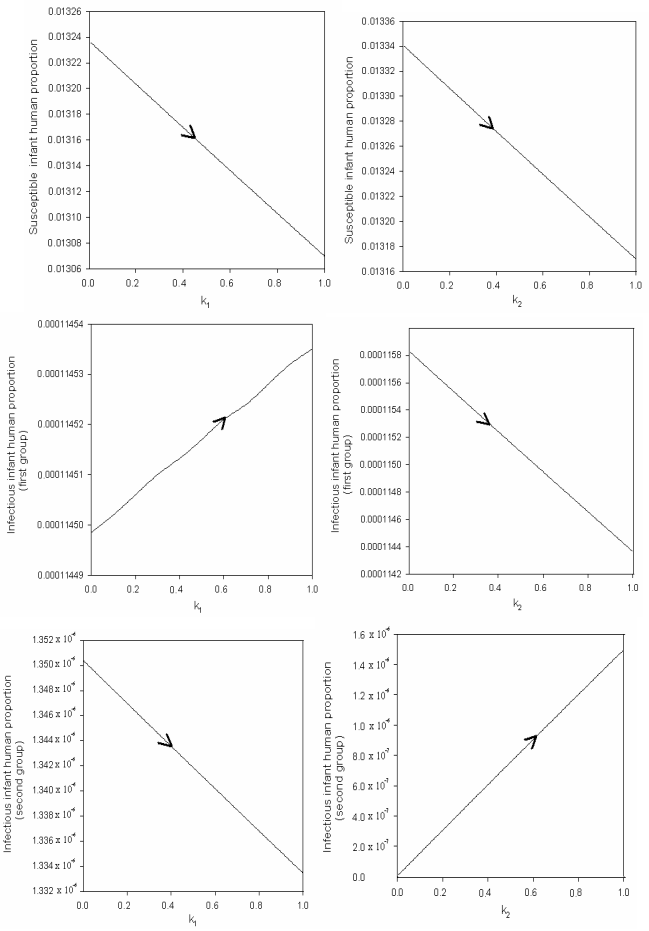
$\mu_v = 0.071428571\ day^{-1}$, $b = 0.33333\ day^{-1}$, $h = 0$,

$r_m = 0.33333\ day^{-1}$, $N_m = 5,000$, $N_n = 5,000$, $A = 20,000$,

$\beta_{vm} = 0.1$, $\beta_{vn} = 0.1$, $\beta_{mv} = 0.1$, $\beta_{nv} = 0.1$, $\beta_{mn} = 0.01$

$k_1 = 0.3, k_2 = 0.9, R_0 = 99.4142, R_0' = 9.97066$. The fractions of populations oscillate to the endemic disease equilibrium state.

Furthermore, we consider the numerical solutions of infant populations when the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby not more than 6 months (k_1) and the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby more than 6 months (k_2) are difference. We show these trajectories in Fig. 5.



5a) 5b)

Fig. 5. Numerical solutions demonstrate the solution trajectories, projected onto $(\overline{S_n}, k_1), (\overline{S_n}, k_2), (\overline{I_{n1}}, k_1),$ $(\overline{I_{n1}}, k_2)(\overline{I_{n2}}, k_1), (\overline{I_{n2}}, k_2)$ with the values of parameters are same as Fig. 4, except the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby not more than 6 months (k_1) and the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby more than 6 months (k_2).

5a) $k_2 = 0.9, 0 \le k_1 \le 1$, 5b) $k_2 = 0.3, 0 \le k_2 \le 1$.

IV. Discussion and Conclusion

In this study, pregnancy, infancy with maternal dengue antibody and the vector populations are assumed to have constant size. The threshold number is defined by R_0 where

$$R_0 = \frac{R_1\beta_2\theta_1\theta_3 + M_1R_1\beta_2\theta_2 + (k_1+k_2)M_1R_2\beta_1\theta_3}{M_1^2\beta_1\beta_2\mu_v}$$

or

$$R_0 = \frac{b\beta_{vm}(A/\mu_v)\gamma_{mn}N_m\gamma_{nv}N_n}{\mu_v(N_T+h)(\mu_H+r_m)^2} + \frac{b\beta_{vm}(A/\mu_v)\gamma_{mv}N_m}{\mu_v(N_T+h)(\mu_H+r_m)} + \frac{(k_1+k_2)b\beta_{vn}(A/\mu_v)\gamma_{nv}N_n}{\mu_v(N_T+h)(\mu_H+r_m)} \quad . \tag{8}$$

The square root of this number represents the average number of secondary cases that one case can produce if introduced into susceptible population. This model, we are interested in dengue virus transmission between pregnant woman and infant with maternal dengue antibody. We consider the third term, it represented the number of secondary infant case in first and second groups (who age not more than 6 months and more than 6 months), respectively with the percentage of dengue antibody which infant who age not more than 6 months received from mother in the beginning, and the percentage of dengue antibody which infant who age more than 6 months received from mother in the beginning. If these values are higher, then the probability of dengue virus which infant received from the biting of infected vector are decreasing. For a disease to be capable of invading and establishing itself in a host population, this threshold number must be greater than one, then every successive generation will diminish inside until its number approach zero.

We can see from fig. 3, the susceptible pregnant human, infectious pregnant human, susceptible infant human, infectious infant human (first group), infectious infant human (second group), infectious vector proportions approach to the disease free equilibrium state (1,0,1,0,0,0) respectively for $R_0 < 1$. The imaginary part of the complex root of eigenvalue is approximately 0.148614. From fig. 4, the fraction of populations spiral to the endemic disease state $(0.508777, 0.0000576711, 0.0131868, 0.00011451, 0.00000134528, 0.0000404878)$ when $R_0 > 1$.

Fig. 5. shows $(\overline{S_n}, k_1), (\overline{S_n}, k_2), (\overline{I_{n1}}, k_1), (\overline{I_{n1}}, k_2)(\overline{I_{n2}}, k_1), (\overline{I_{n2}}, k_2)$ moving towards their equilibrium state when the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby not more than 6 months and the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby more than 6 months are difference. We can see the trajectories spiraling towards the different endemic disease state (fig. 5a). Susceptible infant human, infectious infant (second group) human populations decrease and the proportion of infectious infant (first group) human population increase when the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby not more than 6 months is higher. When the probability of dengue virus which infant received from the biting of infected vector in the beginning for baby more than 6 months (k_2) is higher, susceptible infant human and infectious infant (first group) human populations decrease but the proportion of infectious infant (second group) human population increases.

The bifurcation diagrams of equations (2.1)-(2.6) are shown in the following figures.

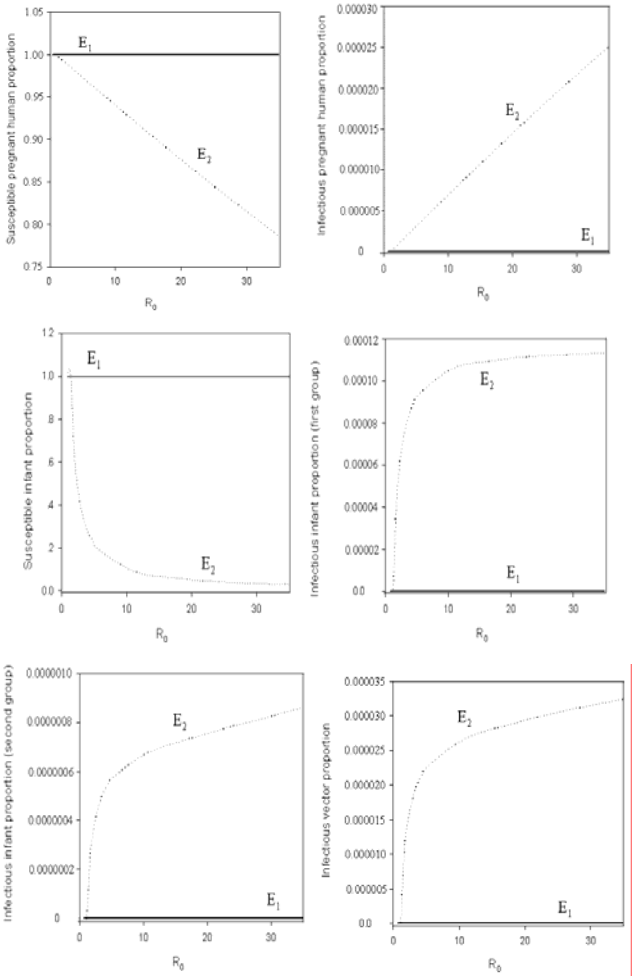


Fig. 6. Bifurcation diagrams of equations (2.1)-(2.6) demonstrate the equilibrium solutions of $\overline{S_m}, \overline{I_m}, \overline{S_n}, \overline{I_{n1}}, \overline{I_{n2}}, \overline{I_v}$, respectively, for the different values of R_0 with

$\mu_H = 0.000039139\ day^{-1}$, $N_T = 10,000$,
$\mu_v = 0.071428571\ day^{-1}$, $b = 0.33333\ day^{-1}$, $h = 0$,
$r_m = 0.33333\ day^{-1}$, $N_m = 5,000$, $N_n = 5,000$,
$\beta_{vm} = 0.1$, $\beta_{vn} = 0.1$, $\beta_{mv} = 0.1$, $\beta_{nv} = 0.1$, $\beta_{mn} = 0.01$
$k_1 = 0.3, k_2 = 0.9$. ——— represents the unstable solutions and ..... represents the stable solutions.

The bifurcation diagrams demonstrate the equilibrium solutions of all populations for the different values of R_0, they represented the stable and unstable solutions. We can see that, for $R_0 < 1, E_1$ will be stable and for $R_0 > 1, E_2$ will be stable.

If the threshold number is greater than one, the normalized susceptible pregnant and susceptible infant proportions are decreases. The normalized infectious pregnant human, infectious infant human (first group), infectious infant human (second group) and infectious vectors increase. If this reproductive number (R_0) is less than unity (one), then the proportions of infectious pregnant human, infectious infant human (first group), infectious infant human (second group) and infectious vector converge to the disease free state. The ultimate goal of any control effort is to reduce (8) below one [24, 25, 26, 27, 28, 29], then the infection will eventually die out and not persist in that community. There may be some secondary cases, but these will decrease with time. If we can reduce the biting rate of the vector, then the threshold number as defined in (8) will be smaller. This will reduce the outbreaks of dengue disease in infants

Management of dengue hemorrhagic fever in pregnancy should be conservative, symptomatic and carry on through the shock stage. The critical period usually passes within 24 to 48 hours. When a pregnant or parturient woman develops signs consistent with dengue disease, the diagnosis in her offspring should be considered even if the infant appears well in the first several days of life. Symptomatic and supportive treatments under close observation are the mainstay of treatment. Other infections, bacterial or viral, can cause clinical features and hematologic changes similar to those of dengue virus infection. The occurrence of subclinical infections may lend further confusion to the situation.

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