Model for the transmission of dengue disease in pregnant and non-pregnant patients

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Abstract**—** Recently, there has been a notable increase in dengue fever and dengue hemorrhagic fever cases in both the very young and in aged adults. Dengue pregnant women had been increasingly reported. Many infants have severe and may suffer from complications and even death because of difficulties in early diagnosis and improper management. In this study, we present the mathematical model for describing the transmission of dengue disease in pregnant and non-pregnant humans. The different transmission probabilities of dengue disease to pregnancy and non-pregnancy are considered. We analyze our model by dynamical analysis method. The numerical simulations are shown to confirm our results. The basic reproductive rate of the disease is discussed.

Keywords**—** basic reproductive rate, dengue disease, locally stable, pregnant human.

I. INTRODUCTION

MATHEMATICAL modeling of disease transmission has a long history. In 1911, an epidemiology model for malaria transmission was developed by Ross [1]. Mac Donald [2] later added a layer of biological realism to the model by providing careful interpretation and estimation of the parameter, which should go into the model. Mc Kenzie [3] has pointed out that the utility of a model depends not as much on how well a mathematical job has been accomplished but on how well a particular question has been translated. If one is interested in disease transmission, it is imperative that the model describes as closely as possible the characteristics of the disease being transmitted. Dengue disease is a mosquito-borne disease caused by dengue virus. Four serotypes of dengue virus exist, namely DEN1, DEN2, DEN3 and DEN4. Infection by one type of the virus confirms permanent immunity to further infections by the infecting strain and temporary immunity to the others. The disease is usually found in tropical region of the world. This disease can be transmitted to human by biting of infected Aedes Aegypti mosquitoes [4]. DF is characterized by the rapid development of the illness that may last from five to seven days with headache, joint and muscle pain and a rash [5]. The illness is characterized by a sudden onset of fever, intense headache, joint and muscle pain, loss of appetite, vomiting and diarrhea, and rash. Dengue hemorrhagic fever(DHF) is the severe form of dengue fever. It is usually the result of a second infection in a person having pre-existing antibodies to a different stain. DHF is associated with loss of appetite, vomiting, high fever, headache and abdominal pain. Shock and circulatory failure may occur. DF may occur in people of all ages who are exposed to infected mosquitoes. DHF is one of the emerging viral diseases spreading throughout the tropical regions of the world. From its first appearance in the Philippines in 1953, it has been estimated that there are between 50 and 100 million cases per year, with approximately 10,000 infant deaths due to this disease [6]. The most dengue infections occur during childhood but some adults may remain susceptible to infection. About 30 percent of dengue infection is reported in patients more than 15 years old [7]. Some pregnant women may also be susceptible to dengue and if they experience dengue infection, they can transmit the dengue viruses to their babies. In 1989, the first 5 reported neonates of vertical dengue infection were born in Tahiti [8]. Since then, there have been 12 additional cases reported from Thailand, Malaysia and France [8]-[15]. Esteva and Vargas [16]-[18] introduced a mathematical model to provide a qualitative assessment for the problem. They used the Susceptible-Infected-Recovered (SIR) model for describing the transmission of dengue disease. For better understanding of the dengue transmission in pregnant and non-pregnant humans, the mathematical model is presented in this study.

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II. MATHEMATICAL MODEL

We proposed a new model to study the transmission of dengue virus infection by introducing pregnant and non-pregnant classes into the SIR model [16]. The model is based on the following assumptions. The total human populations have constant sizes which are classified into two groups, pregnancy and non-pregnancy. Each group has constant size and it is divided into three classes, susceptible, infectious and recovered human populations. The vector population is divided into two groups, susceptible and infectious mosquitoes, with the mosquitoes never recover from the infection.

The model considers the rate of change for eight variables:

$\overline{S_{H_P}}$ is the number of susceptibles pregnancy,

$\overline{I_{H_P}}$ is the number of infectives pregnancy,

$\overline{R_{H_P}}$ is the number of immunes pregnancy,

$\overline{S_{H_n}}$ is the number of susceptibles non-pregnancy,

$\overline{I_{H_n}}$ is the number of infectives non-pregnancy,

$\overline{R_{H_n}}$ is the number of immunes non-pregnancy,

$\overline{S_V}$ is the number of susceptibles vector,

$\overline{I_V}$ is the number of infectives vector .

The rates of change for each class of pregnancy, non-pregnancy and vector populations are given by;

$$\begin{aligned}
&\frac{d\overline{S_{H_P}}}{dt} = \mu_H(N_{H_P} - \overline{S_{H_P}}) - \alpha\beta_{H_n}\overline{S_{H_P}}\,\overline{I_V}\frac{b}{N_H+m},\\
&\frac{d\overline{I_{H_P}}}{dt} = \alpha\beta_{H_n}\overline{S_{H_P}}\,\overline{I_V}\frac{b}{N_H+m} - (\mu_H+\gamma_H)\overline{I_{H_P}},\\
&\frac{d\overline{R_{H_P}}}{dt} = \gamma_H\overline{I_{H_P}} - \mu_H\overline{R_{H_P}},\\
&\frac{d\overline{S_{H_n}}}{dt} = \mu_H(N_{H_n} - \overline{S_{H_n}}) - \beta_{H_n}\overline{S_{H_n}}\,\overline{I_V}\frac{b}{N_H+m},\\
&\frac{d\overline{I_{H_n}}}{dt} = \beta_{H_n}\overline{S_{H_n}}\,\overline{I_V}\frac{b}{N_H+m} - (\mu_H+\gamma_H)\overline{I_{H_n}},\\
&\frac{d\overline{R_{H_n}}}{dt} = \gamma_H\overline{I_{H_n}} - \mu_H\overline{R_{H_n}},\\
&\frac{d\overline{S_V}}{dt} = A - \mu_V\overline{S_V} - \beta_{V_n}\overline{S_V}\frac{b}{N_H+m}(\ell\overline{I_{H_P}} + \overline{I_{H_n}}),\\
&\frac{d\overline{I_V}}{dt} = \beta_{V_n}\overline{S_V}\frac{b}{N_H+m}(\ell\overline{I_{H_P}} + \overline{I_{H_n}}) - \mu_V\overline{I_V},
\end{aligned} \tag{1}$$

where N_H is the number of the human population,

N_{H_P} is the number of the pregnancy,

N_{H_n} is the number of the non-pregnancy,

N_V is the number of the vector,

A is the constant recruitment rate of mosquitoes,

μ_H is the average constant death rate of the human population,

μ_V is the average constant death rate of vector,

m is the number of alternative hosts available as blood sources,

β_{H_P} is the transmission probability from vector to pregnancy,

β_{H_n} is the transmission probability from vector to non-pregnancy,

α is the ratio between transmission probability from vector to pregnancy and transmission probability from vector to non-pregnancy,

γ_H is the constant rate at which an infected human recovers,

b is the average number of biting per mosquito per day,

p is the percentage of the human to be pregnant,

β_{V_P} is the transmission probability from pregnancy to vector,

β_{V_n} is the transmission probability from non-pregnancy to vector,

ℓ is the ratio between transmission probability from pregnancy to vector and the transmission probability from non-pregnancy to vector,

with the three conditions $N_{H_P} = \overline{S_{H_P}} + \overline{I_{H_P}} + \overline{R_{H_P}}$, $N_{H_n} = \overline{S_{H_n}} + \overline{I_{H_n}} + \overline{R_{H_n}}$ and $N_V = \overline{S_V} + \overline{I_V}$. (2)

The total population remains constant. Thus, there is no change of rate for each population. These indicate that $\frac{dN_H}{dt} = 0$, $\frac{dN_{H_P}}{dt} = 0$, $\frac{dN_{H_n}}{dt} = 0$ and $\frac{dN_V}{dt} = 0$ an $\beta_{H_P} = \alpha\beta_{H_n}$, $\frac{pN_H}{100} = N_{H_P}$, $\frac{(100-p)}{100}N_H = N_{H_n}$, $\beta_{V_P} = \ell\beta_{V_n}$ and $N_{H_P} + N_{H_n} = N_H$.

We now normalize (1) by letting

$$S_P = \frac{\overline{S_{H_P}}}{N_{H_P}},\ I_P = \frac{\overline{I_{H_P}}}{N_{H_P}},\ R_P = \frac{\overline{R_{H_P}}}{N_{H_P}},\ S_n = \frac{\overline{S_{H_n}}}{N_{H_n}},\ I_n = \frac{\overline{I_{H_n}}}{N_{H_n}},$$

$$R_n = \frac{\overline{R_{H_n}}}{N_{H_n}},\ S_V = \frac{\overline{S_V}}{N_V} = \frac{\overline{S_V}}{(A/\mu_V)} \text{ and } I_V = \frac{\overline{I_V}}{N_V} = \frac{\overline{I_V}}{(A/\mu_V)}.$$

These give

$$\begin{aligned}
&\frac{dS_P}{dt} = \mu_H(1-S_P) - \alpha\beta_{H_n}S_PI_V(A/\mu_V)\frac{b}{N_H+m},\\
&\frac{dI_P}{dt} = \alpha\beta_{H_n}S_PI_V(A/\mu_V)\frac{b}{N_H+m} - (\mu_H+\gamma_H)I_P,\\
&\frac{dS_n}{dt} = \mu_H(1-S_n) - \beta_{H_n}S_nI_V(A/\mu_V)\frac{b}{N_H+m},\\
&\frac{dI_n}{dt} = \beta_{H_n}S_nI_V(A/\mu_V)\frac{b}{N_H+m} - (\mu_H+\gamma_H)I_n,\\
&\frac{dI_V}{dt} = \beta_{V_n}(1-I_V)\frac{b}{N_H+m}(\ell I_PN_{H_P} + I_nN_{H_n}) - \mu_vI_V
\end{aligned} \tag{3}$$

with the three conditions

$$S_P + I_P + R_P = 1,\ S_n + I_n + R_n = 1 \text{ and } S_V + I_V = 1. \tag{4}$$

III. ANALYSIS OF THE MATHEMATICAL MODEL

A. *Analytical Results*

The equilibrium points are found by setting the right side of (3) equal to zero. This gives

1) The disease free equilibrium point $E_1 = (1,0,1,0,0)$ and
2) The endemic disease equilibrium point $E_2 = (S_P^*, I_P^*, S_n^*, I_n^*, I_V^*)$ where

$$S_P^* = \frac{\beta_1}{\beta_1 + \beta_2 I_V^*}, \tag{5}$$

$$I_P^* = \frac{\beta_1\beta_2 I_V^*}{(\beta_1 + \beta_2 I_V^*)(\beta_1 + \beta_3)}, \tag{6}$$

$$S_n^* = \frac{\alpha\beta_1}{\alpha\beta_1 + \beta_2 I_V^*}, \quad (7)$$

$$I_n^* = \frac{\beta_1\beta_2 I_V^*}{(\alpha\beta_1 + \beta_2 I_V^*)(\beta_1 + \beta_3)}, \quad (8)$$

$$I_V^* = \frac{-a_1 + \sqrt{a_1^2 - 4a_2a_0}}{2a_2} \quad (9)$$

where

$$a_0 = \left(\frac{\beta_1}{\beta_2}\right)^2 \alpha M_3 - \alpha\left(\frac{\beta_1}{\beta_2}\right)M_1 - \left(\frac{\beta_1}{\beta_2}\right)M_2, \quad (10)$$

$$a_1 = \frac{\alpha\beta_1}{\beta_2}M_1 + \frac{\beta_1}{\beta_2}M_2 + \frac{\beta_1}{\beta_2}M_3 + \frac{\alpha\beta_1}{\beta_2}M_3 - M_1 - M_2, \quad (11)$$

$$a_2 = M_1 + M_2 + M_3, \quad (12)$$

such that $\beta_1 = \mu_H(N_H + m)$, $\beta_2 = \alpha\beta_{H_n}(A/\mu_V)b$,

$$\beta_3 = \gamma_H(N_H + m), \quad (13)$$

and $M_1 = \beta_{V_n} b\ell\beta_1\beta_2^2 N_{H_p}$, $M_2 = \beta_{V_n} b\beta_1\beta_2^2 N_{H_n}$

$$M_3 = \mu_V(N_H + m)(\beta_1 + \beta_3)\beta_2^2. \quad (14)$$

-Disease Free State

The stability of each equilibrium point is determined from linearizing equations in (3) about the equilibrium point examining the eigenvalues of the resulting Jacobian matrix. We now consider the eigenvalues of the Jacobian matrix at each equilibrium point. If all eigenvalues for each equilibrium state have negative real parts then that equilibrium state is locally stable. The local stability of the disease free equilibrium E_1 is governed by the matrix

$$\begin{bmatrix} -\mu_H & 0 & 0 & 0 & -\alpha\beta_{H_n}\frac{b}{N_H+m}(A/\mu_V) \\ 0 & -\mu_H-\gamma_H & 0 & 0 & \alpha\beta_{H_n}\frac{b}{N_H+m}(A/\mu_V) \\ 0 & 0 & -\mu_H & 0 & -\beta_{H_n}\frac{b}{N_H+m}(A/\mu_V) \\ 0 & 0 & 0 & -\mu_H-\gamma_H & \beta_{H_n}\frac{b}{N_H+m}(A/\mu_V) \\ 0 & \beta_{V_n}\frac{b}{N_H+m}\ell N_{H_p} & 0 & \beta_{V_n}\frac{b}{N_H+m}N_{H_n} & -\mu_V \end{bmatrix} \quad (15)$$

The eigenvalues are

$\lambda_{1,3} = -\mu_H$, $\lambda_2 = -\mu_H - \gamma_H$,

$$\lambda_4 = \frac{-c_1 - \sqrt{c_1^2 - 4c_0}}{2}, \lambda_5 = \frac{-c_1 + \sqrt{c_1^2 - 4c_0}}{2}$$

where

$$c_0 = \mu_H\mu_V + \gamma_H\mu_V - \beta_{V_n}N_{H_n}[\frac{b}{N_H+m}]^2\beta_{H_n}(A/\mu_V)$$

$$-\alpha\ell\beta_{V_n}N_{H_p}[\frac{b}{N_H+m}]^2\beta_{H_n}(A/\mu_V),$$

$c_1 = \mu_H + \mu_V + \gamma_H$.

It can be seen that λ_1, λ_2, λ_3 and λ_4 have negative real parts.

Next, we will check the sign of eigenvalues λ_5. λ_5 has negative real part when

$\sqrt{c_1^2 - 4c_0} < c_1$ or $c_1^2 - 4c_0 < c_1^2$.

So that

$$\mu_H\mu_V + \gamma_H\mu_V - \beta_{V_n}N_{H_n}[\frac{b}{N_H+m}]^2\beta_{H_n}(A/\mu_V)$$

$$-\alpha\ell\beta_{V_n}N_{H_p}[\frac{b}{N_H+m}]^2\beta_{H_n}(A/\mu_V) > 0$$

or $B_A = \frac{\beta_2(M_2 + \alpha M_1)}{\beta_1\alpha M_3} < 1$.

Therefore the disease free equilibrium point is locally stable for $B_A < 1$.

-EndemicDisease State

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

$$\begin{bmatrix} -\mu_H-\rho & I_V^* & 0 & 0 & -\rho S_P^* \\ \rho & -\mu_H-\gamma_H & 0 & 0 & \rho S_P^* \\ 0 & 0 & -\mu_H-\frac{\rho}{\alpha}I_V^* & 0 & -\frac{\rho}{\alpha}S_n^* \\ 0 & 0 & \frac{\rho}{\alpha}I_V^* & -\mu_H-\gamma_H & \frac{\rho}{\alpha}S_n^* \\ 0 & k-kI_V^* & 0 & l-lI_V^* & -kI_P^*-lI_n^*-\mu_V \end{bmatrix} \quad (16)$$

where $\rho = \alpha\beta_{H_n}\frac{b}{N_H+m}(A/\mu_V)$,

$k = \beta_{V_n}\frac{b}{N_H+m}\ell N_{H_p}$ and

$l = \beta_{V_n}\frac{b}{N_H+m}N_{H_n}$.

For the endemic equilibrium point, $E_2 = (S_P^*, I_P^*, S_n^*, I_n^*, I_V^*)$, the eigenvalues are found by solving the characteristic equation

$$\lambda^5 + d_4\lambda^4 + d_3\lambda^3 + d_2\lambda^2 + d_1\lambda + d_0 = 0. \quad (17)$$

To determine the local stability of the endemic equilibrium point, we need to check the signs of all eigenvalues for the endemic equilibrium point. The stability of the endemic equilibrium point can be determined by using Routh-Hurwitz criteria [19] as follows:

i) $d_i > 0$ for $i = 0,1,2,3,4$,

ii) $d_4d_3d_2 > d_2^2 + d_4^2d_1$,

iii) $(d_4d_1 - d_0)(d_4d_3d_2 - d_2^2 - d_4^2d_1) > d_0(d_4d_3 - d_2)^2 + d_4d_0^2$.

After we check the three conditions above by MATHEMATICA, we found that the endemic equilibrium point is locally stable for $B_A > 1$. The quantity $B_A' = \sqrt{B_A}$ is the basic reproductive number the disease, it gives the average number of secondary patients that one patient can produce if introduced into a susceptible human. So we can reduce the outbreak of dengue disease in the endemic region when the basic reproductive number (B_A') is greater than one.

B. Numerical Results

We are interested in the transmission of disease in pregnancy and non-pregnancy. The values of the parameter used in this study are as follows: $\mu_H = 1/(365\times 60)$ per day corresponds to a life expectancy of 60 years in human. The mean life of mosquito is 14 days; $\mu_V = (1/14)$ per day, $b = 1/3$; one bite provides blood meal for 3 days. The recovery rate equals to 1/3 per day. We assume that the number of the non-pregnancy is greater than the number of the pregnancy and there is no alternative host. Thus the ratio α and ℓ should be less than one. The other parameters are arbitrarily chosen. Numerical solutions of (3) are shown in the following figures.

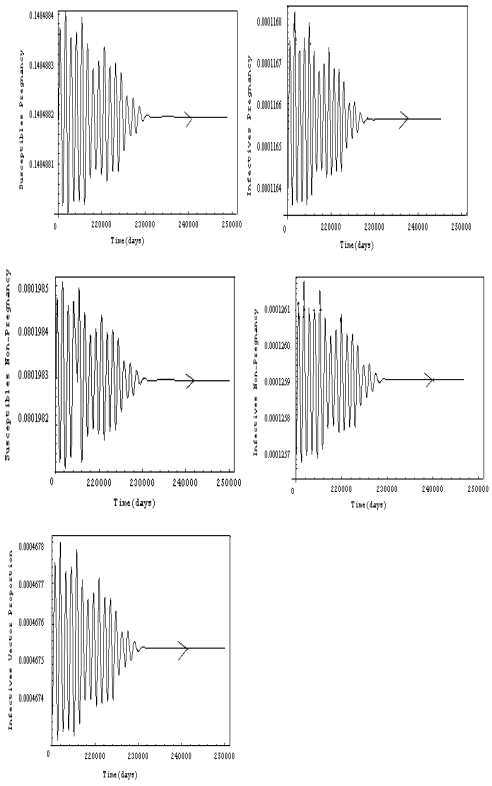

Fig.1 Numerical solutions of the system (3) demonstrate the times series of S_P, I_P, S_n, I_n, I_V, respectively, for $B_A > 1$ with $\mu_H = 1/(365\times 60)$ day^{-1}, $b = 1/3$ day^{-1} $\mu_V = (1/14)$ day^{-1}, $\beta_{H_P} = 0.4$, $\beta_{H_n} = 0.8$, $\alpha = 0.5$, $\gamma_H = 1/3$ day^{-1}, $\beta_{v_p} = 0.4$, $\beta_{v_n} = 0.8$, $\ell = 0.5$, $A = 30000$, $B_A = 12.4482$ $B_A^{'} = 3.5282$. The fractions of populations oscillate to the endemic disease equilibrium point.

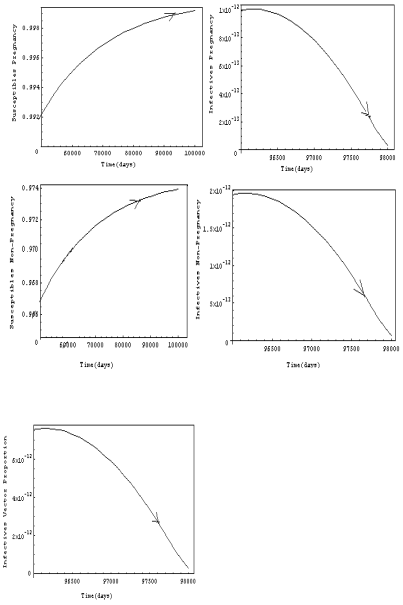

Fig.2 Numerical solutions of the system (3)demonstrate the times series of S_P, I_P, S_n, I_n, I_V, respectively, for $B_A < 1$with $\mu_H = 1/(365\times 60)$ day^{-1}, $b = 1/3$ day^{-1} $\mu_V = (1/14)$ day^{-1}, $\beta_{H_P} = 0.4$, $\beta_{H_n} = 0.8$, $\alpha = 0.5$, $\gamma_H = 1/3$ day^{-1}, $\beta_{v_p} = 0.4$, $\beta_{v_n} = 0.8$, $\ell = 0.5$, $A = 2000$, $N_H = 100000$, $N_{H_n} = 99000$, $N_{H_P} = 1000$ $B_A = 0.829881$ $B_A^{'} = 0.910978$. The fractions of populations approach to the disease free state.

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IV. CONCLUSION

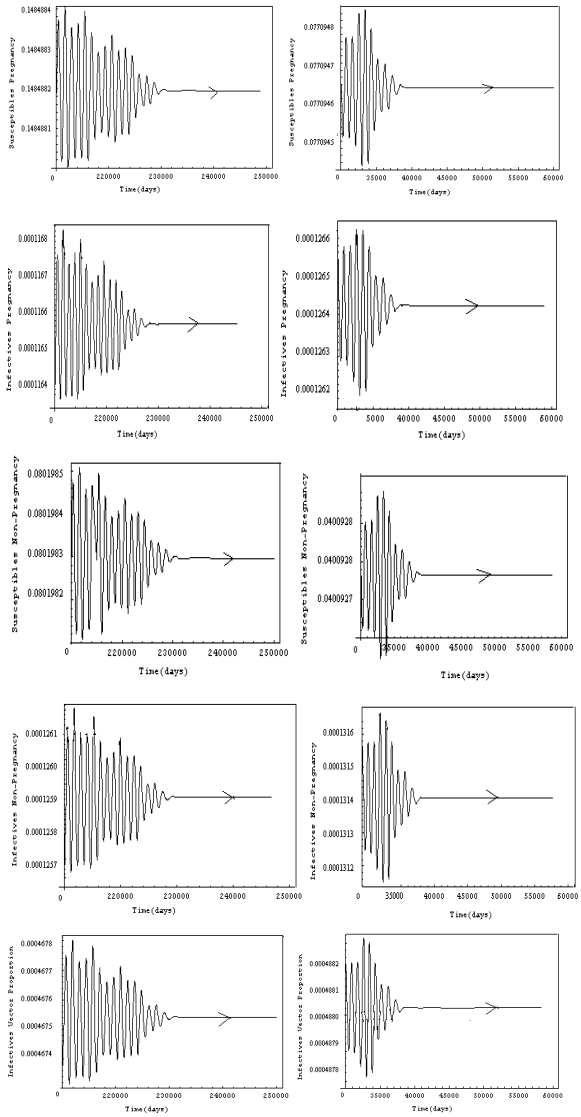
The mathematical model which we analyze in this study, the pregnancy, the non-pregnancy and the vector population are assumed to be constant size. The quantity $B_A^{'} = \sqrt{B_A}$ is the basic reproductive number of the disease where

$$B_A = \frac{\beta_2(M_2 + \alpha M_1)}{\beta_1 \alpha M_3} = \frac{\beta_{H_n}\beta_{V_n} b^2 (N_{H_n} + \alpha \ell N_{H_P})(A/\mu_V)}{\mu_V (N_H + m)^2 (\mu_H + \gamma_H)} = \frac{\beta_{H_n}\beta_{V_n} b^2 N_{H_n}(A/\mu_V)}{\mu_V (N_H + m)^2 (\mu_H + \gamma_H)} + \frac{\beta_{H_P}\beta_{V_P} b^2 N_{H_P}(A/\mu_V)}{\mu_V (N_H + m)^2 (\mu_H + \gamma_H)}, \quad (18)$$

it represents the average number of secondary cases that one case can produce if introduced into a susceptible population [16]. The first and second terms indicate the number of secondary non-pregnant and pregnant cases, respectively. Consider the second term, The infective pregnancy introduced into the susceptible pregnancy is bitten by $(\frac{b(A/\mu_V)}{(N_H + m)})(\frac{1}{\mu_H + \gamma_H})$

mosquitoes, a proportion, $\beta_{V_p}(\frac{b(A/\mu_V)}{(N_H+m)})(\frac{1}{\mu_H+\gamma_H})$, of these mosquitoes becomes infectious. One of these infectious mosquitoes; $\frac{b}{\mu_V}\left(\frac{N_{H_p}}{N_H+m}\right)$ will in turn bite. Multiplying this number by β_{H_p}, we get the number of infected pregnancy. The multiplication between the number of infected pregnancy and the number of infected mosquitoes during the life time of the infectious pregnancy obtains the second term of B_A. The summation of the average number of secondary pregnant cases and non-pregnant cases produces the value of B_A. Therefore, the geometric mean of these quantities, which is equal to $B_A^{'}$, gives the number of secondary infections.

Moreover, we consider the time series of human and vector populations when the basic reproductive numbers are difference. We show in fig.3.



(3a) (3b)

Fig.3 Numerical solutions of the system (3)demonstrate the times series of S_P, I_P, S_n, I_n, I_V , respectively, for $B_A > 1$
(3a) $B_A = 12.4482$, The fractions of populations oscillate to the endemic disease equilibrium point (0.1484882, 0.00011655, 0.0801983, 0.0001259, 0.00046752)
(3b) $B_A = 24.8964$, The fractions of populations oscillate to the endemic disease equilibrium point (0.07709464, 0.0001264, 0.04009275, 0.0001314, 0.00048802).

We compare the transmission of this disease for the different basic reproductive number. The basic reproductive number of the disease for fig.(3a) and fig.(3b) equals to 3.5282 and 4.98963, 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 3 years for fig.(3a) and 2 years for fig.(3b). If the basic reproductive rate is higher, this means that one case can produce the greater number of secondary cases, and then the period of oscillation is shorter. The endemic equilibrium point for the fractions of susceptible pregnant and non-pregnant humans decrease. The endemic equilibrium points for the fractions of infective pregnant, non-pregnant humans and infective vector increase. These subsequent behaviors occur since there are enough susceptible pregnancy and non-pregnancy to be infected from infectious vector. Application of an ultra low volume (ULV) amount of insecticides (the standard method used to control the spread of dengue disease and other arthropod-borne disease) could reduce the basic reproductive rate to below one. The value of the basic reproductive rate would return to the above one value once the application is stopped and since the endemic state is locally stable, the disease would return. Therefore the eradication program would have to be a continuing one. Thus, we can reduce the outbreak of the disease.

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