Stability research of dengue model with time delay

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Abstract: This paper studied the dengue fever model with time delay. This paper divided the time delay into four cases: (1) $\tau 1 = \tau 2 = 0$, (2) $\tau 1 = \tau$, $\tau 2 = 0$, (3) $\tau 1 = 0$, $\tau 2 = \tau$, (4) $\tau 1 = \tau$, $\tau 2 = \tau$, and studied the stability and Hopf bifurcation of the model on these three cases. At the end of this paper, we simulated the dengue model with time delay by using Matlab software, and gained the numerical condition of this model which appearing periodic solutions and Hopf bifurcation. On the first case $\tau 1 = \tau$, $\tau 2 = 0$, the time delay threshold is $\tau 0 = 0.6155$; on the second case $\tau 1 = 0$, $\tau 2 = \tau$, the time delay threshold is $\tau 0 = 0.0490$; on the third case $\tau 1 = \tau$, $\tau 2 = \tau$, the time delay threshold is $\tau 0 = 3.5454$.

1. Introduction

Dengue fever and dengue hemorrhagic fever is a kind of acute mosquito borne infectious diseases spread rapidly around the globe. It is one of the most serious diseases infecting humans[1]. It has the characteristics of spreading rapidly and high incidence which can cause large scale epidemic in an area. According to WHO estimates, it has the risk of 25 billion people suffering from the dengue fever, and 5000 million to 1 billion people infect dengue fever. There are 50 million dengue fever hospitalization, of which 2 million died from dengue fever and dengue shock syndrome. Dengue fever has been a threat to 1/3 of the world population's health and safety[2].

Dengue is transmitted to humans through the bite of infected Aedes aegypti and A. albopictus mosquitoes. It is understood that four closely related serotypes of DENV exist-DENV-1, DENV-2, DENV-3 and DENV-4 and these four serotypes cause infections of varying severity in humans [1]-[2]. The infected individual usually suffers from acute febrile illness called Dengue Fever (DF) which is cleared by a complex immune response in a short time of approximately 7 days after onset of fever. We note that, though there is a huge effort going on to develop an effective vaccine against dengue infections, commercial dengue vaccines are not yet available [3]-[7]. In this context, it is important to understand the biological mechanisms and dynamical processes involved during this infection. Also these complex non-linear biological processes lead to dynamic models that are interesting for their varied and rich dynamics. The epidemiology of dengue in different populations have been studied previously using improved or extended versions of the basic SIR model [8]-[16].

At present, people put forward various kinds of dengue fever model, for example, the SEIRS model considering the effects of using pesticides on dengue fever epidemic [17]; the population model only consider a virus and assume the number of susceptible people and patients is constant [18]; the model with the exponential growth population and constant infection rate [19]; the model with two kinds of serum virus and variable population [20].

So far, there is few research in dengue fever model considering time delay. The time delay plays a very important role on studying the dynamics of dengue virus transmission behavior. It explains the dynamics behavior of sick people "susceptible people" the carrying-virus aedes mosquitoes and the no-carryingvirus aedes mosquitoes from the angle of mathematics, which can help people understand the spread of dengue fever model law better, so as to control the spread of dengue virus better.

2. The dengue fever model with time delay

Let $S_1(t)$, $I_1(t)$, $R_1(t)$, $S_2(t)$, $I_2(t)$ represents the number of susceptible people, patient, remove people, no-viruses-carrying mosquitoes, viruses-carrying mosquitoes at time t. In reference[21],

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Tewa et.al study the following dengue fever model:

$$\begin{cases} S_1' = \mu_1 N_1 - \dfrac{b\beta_1}{N_1 + m} S_1 I_2 - \mu_1 S_1 \\ I_1' = \dfrac{b\beta_1}{N_1 + m} S_1 I_2 - (\mu_1 + \gamma_1) I_1 \\ R_1' = \gamma_1 I_1 - \mu_1 R_1 \\ S_2' = A - \dfrac{b\beta_1}{N_1 + m} S_2 I_1 - \mu_2 S_2 \\ I_2' = \dfrac{b\beta_1}{N_1 + m} S_2 I_1 - \mu_2 I_2 N_1 = S_1 + R_1 + I_1 \end{cases} \tag{1}$$

The definition of the parameters can refer to reference [1]. On the basis of equation(1), Ding Deqiong from Harbin Institute of Technology of China made some improvements, he study the following dengue fever model [22]:

$$\begin{cases} S_1' = \mu_1 N_1 - \beta_1 S_1 I_2 - \mu_1 S_1 \\ I_1' = \beta_1 S_1 I_2 - (\mu_1 + \gamma_1) I_1 \\ R_1' = \gamma_1 I_1 - \mu_1 R_1 \\ S_2' = A - \beta_2 S_2 I_1 - \mu_2 S_2 \\ I_2' = \beta_2 S_2 I_1 - \mu_2 I_2 N_1 = S_1 + R_1 + I_1 \end{cases} \tag{2}$$

In this paper, on the basis of equation(2), we make some improvements, we consider the effect of time delay to the dengue fever. The equations describing the model are given by:

$$\begin{cases} S_1' = \mu_1 N_1(t) - \beta_1 S_1(t) I_2(t) - \mu_1 S_1 \\ I_1' = \beta_1 S_1(t) I_2(t - \tau_1) - (\mu_1 + \gamma_1) I_1(t) \\ R_1' = \gamma_1 I_1(t) - \mu_1 R(t) \\ S_2' = A - \beta_2 S_2(t) I_1(t) - \mu_2 S_2(t) \\ I_2' = \beta_2 S_2(t) I_1(t - \tau_2) - \mu_2 I_2(t) \\ N_1(t) = S_1(t) + R(t) + I_1(t) \end{cases} \tag{3}$$

As we can see, in equation(3), the variable R is not explicit in the first two equations and the last two equations, so equation(3) can be simplified to be the following system

$$\begin{cases} S_1' = \mu_1 N_1(t) - \beta_1 S_1(t) I_2(t) - \mu_1 S_1 \\ I_1' = \beta_1 S_1(t) I_2(t - \tau_1) - (\mu_1 + \gamma_1) I_1(t) \\ S_2' = A - \beta_2 S_2(t) I_1(t) - \mu_2 S_2(t) \\ I_2' = \beta_2 S_2(t) I_1(t - \tau_2) - \mu_2 I_2(t) \\ N_1(t) = S_1(t) + I_1(t) \end{cases} \tag{4}$$

where μ1 and μ2 represents the death date of human and mosquito respectively, S1(t), I1(t), S2(t), I2(t) represents the number of susceptible patient, no-virusescarrying mosquitoes, viruses-carrying mosquitoes at time t. N1(t) is the total population at time t, β1 represents the infectious rate of the viruses-carrying mosquitoes to the susceptible, β2 represents the infectious rate of the patient to the no-viruses-carrying mosquitoes, γ1 represents the recovery rate of the patient, A represents the new rate of mosquitoes, τ1 and τ2 represent the rehabilitees immune period and incubation period to the diease respectively.

Define the basic reproductive ratio as follows:

$$R_0 = \frac{\beta_1 \beta_2 N_1 A}{\mu_2^2 (\mu_1 + \gamma_1)} \tag{5}$$

Define the closed region:

$$D=\left\{\left(S_1,I_1,S_2,I_2\right)\in R_+^4 : S_1\le N_1, S_1+I_1\le N_1, S_2\le\frac{A}{\mu_2}, S_2+I_2\le\frac{A}{\mu_2}\right\}$$

As the same as reference [1], region D is a positive invariant set. This paper will study the dynamical behavior of equation(4) on the closed region D. The dynamical behavior of R can be determined by the third equation of the system (3).

Obviously, for all non-negative parameters, equation(4) has no-disease equilibrium point $E_1^*\left(S_1,I_1,S_2,I_2\right)=\left(N_1,0,\frac{A}{\mu_2},0\right)$.

When the basic reproductive ratio $R_0 > 1$, the system has the only endemic equilibrium $E_1^*\left(S_1,I_1,S_2,I_2\right)$, where

$$\begin{cases}\bar{S}_1=\dfrac{\mu_1 N_1}{\mu_1 I_2+\mu_1}\\ \bar{I}_1=\dfrac{\mu_1\mu_2^2(R_0-1)}{\beta_2(\beta_1 A+\mu_1\mu_2)}\\ \bar{S}_2=\dfrac{A}{\beta_2\bar{I}_1+\mu_2}\\ \bar{S}_1=\dfrac{\beta_2\bar{S}_2\bar{I}_1}{\mu_2}\end{cases}\tag{6}$$

Do the transform as follow:

$$\begin{cases}U_1(t)=S_1(t)-\bar{S}_1\\ U_2(t)=I_1(t)-\bar{I}_1\\ V_1(t)=S_2(t)-\bar{S}_2\\ V_1(t)=I_1(t)-\bar{I}_2\end{cases}\tag{7}$$

then equation(4) comes to be:

$$\begin{cases}U_1'=-(\beta_1\bar{I}_2+\mu_1)U_1(t)-\beta_1\bar{S}_1V_2(t)-\beta_1U_1(t)V_2(t),\\ V_1'=-(\beta_2\bar{I}_1+\mu_2)V_1(t)-\beta_2\bar{S}_2U_2(t)-\beta_2V_1(t)U_2(t),\\ U_2'=\beta_1\bar{S}_1V_2(t-\tau_1)+\beta_1U_1(t)V_2(t-\tau_1)-\beta_1\bar{I}_2U_1(t)-(\mu_1+\gamma_1)U_2(t),\\ V_2'=\beta_2\bar{S}_2U_2(t-\tau_2)+\beta_2V_1(t)U_2(t-\tau_2)+\beta_2\bar{I}_1V_1(t)-\mu_2V_2(t),\end{cases}\tag{8}$$

Obviously, the origin point (0, 0, 0, 0) is the equilibrium point of equation(7),linearizing the system, we gain the following system:

$$\begin{cases}U_1'=-(\beta_1\bar{I}_2+\mu_1)U_1(t)-\beta_1\bar{S}_1V_2(t),\\ V_1'=-(\beta_2\bar{I}_1+\mu_2)V_1(t)-\beta_2\bar{S}_2U_2(t),\\ U_2'=\beta_1\bar{S}_1V_2(t-\tau_1)-\beta_1\bar{I}_2U_1(t)-(\mu_1+\gamma_1)U_2(t),\\ V_2'=\beta_2\bar{S}_2U_2(t-\tau_2)+\beta_2\bar{I}_1V_1(t)-\mu_2V_2(t),\end{cases}\tag{9}$$

Let

$$\begin{aligned}\mathbf{X}(t)&=(U_1(t),U_2(t),V_1(t),V_2(t))^{\mathrm{T}}\\ \mathbf{X}(t-\tau_1)&=(U_1(t-\tau_1),U_2(t-\tau_1),V_1(t-\tau_1),V_2(t-\tau_1))^{\mathrm{T}}\\ \mathbf{X}(t-\tau_2)&=(U_1(t-\tau_2),U_2(t-\tau_2),V_1(t-\tau_2),V_2(t-\tau_2))^{\mathrm{T}}\end{aligned}\tag{10}$$

And let

$$\dot{\mathbf{X}}(t)=(U_1',V_1',U_2',V_2')^{\mathrm{T}}\tag{11}$$

The coefficient matrix of linear equationis

$$A_0 = \begin{pmatrix} -(\beta_1 \bar{I}_2 + \mu_1) & 0 & 0 & \beta_1 \bar{S}_1 \\ \beta_1 \bar{I}_2 & -(\beta_1 + \gamma_1) & 0 & 0 \\ 0 & -\beta_2 \bar{S}_2 & -(\beta_2 \bar{I}_1 + \mu_2) & 0 \\ 0 & 0 & \beta_2 \bar{I}_1 & -\mu_2 \end{pmatrix}$$

$$A_1 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_1 \bar{S}_1 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$A_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \beta_2 \bar{S}_2 & 0 & 0 \end{pmatrix}$$

Then equation(11) becomes

$$\dot{\mathbf{X}}(t) = A_0 X(t) + A_1 X(t-\tau_1) + A_2 X(t-\tau_2) \tag{12}$$

The characteristic equation corresponding to equation (8) is:

$$P(\lambda, \tau_1, \tau_2) = \begin{vmatrix} -\lambda - (\beta_1 \bar{I}_2 + \mu_1) & 0 & 0 & \beta_1 \bar{S}_1 \\ \beta_2 \bar{I}_1 & -\lambda - (\mu_1 + \gamma_1) & 0 & \beta_1 \bar{S}_1 e^{-\tau_1 \lambda} \\ 0 & -\beta_2 \bar{S}_2 & -\lambda - (\beta_2 \bar{I}_1 + \mu_2) & 0 \\ 0 & \beta_2 \bar{S}_2 e^{-\tau_2 \lambda} & \beta_2 \bar{I}_1 & -\lambda - \mu_2 \end{vmatrix} = 0 \tag{13}$$

Expanding equation (13) in accordance to the first column, we have:

$$P(\lambda, \tau_1, \tau_2) = -\lambda^4 + L\lambda^3 + M\lambda^2 + N\lambda - Re^{-\tau_2 \lambda} - Se^{-(\tau_1 + \tau_2)\lambda} + T = 0 \tag{14}$$

3. The stability of the model

In this section, we discuss the time delay effect on the stability and the Hopf bifurcation of the dengue fever model in three cases.

3.1 τ1 = τ2 = 0

When τ1 = τ2 = 0, equation (14) becomes

$$P(\lambda) = -\lambda^4 + M_1 \lambda^2 - R - S + T = 0 \tag{15}$$

Let $x = \lambda^2$, then we have:

$$x^2 - M_1 x + R + S - T = 0.$$

In summary, we have the following theorem:

Theorem 1. When $M_1^2 \geq 4(R + S - T)$, equation (15) has four real roots, the no-disease equilibrium E_1^* and endemic equilibrium E_2^* of equation(2) is unstable. When $M_1^2 < 4(R+SST)$, equation (10) has four unequal and conjugate imaginary roots, the no-disease equilibrium E_1^* and endemic equilibrium E_2^* of equation(2) is stable.

3.2 τ1 = τ, τ2 = 0

When τ1=τ, τ2=0,equation(14) becomes to the following equation:

$$P(\lambda, \tau) = -\lambda^4 + L\lambda^3 + M_2 \lambda^2 - R - Se^{-\tau_1 \lambda} + T = 0 \tag{16}$$

In summary, we have the following theorem:

Theorem 2. When $\tau 1 = \tau$, $\tau 2 = 0$and $[M_2^2 + 2(R\text{-}T\text{-}LN_2)]^2 \geq 4[(R\text{-}T)^2\text{-}S^2]$, equation (16) has four real roots, the no-disease equilibrium E_1^* and endemic equilibrium $E_2^*$of system (2) is unstable. When $[M_2^2 + 2(R - T - LN_2)]^2 < 4[(R - T)^2\text{-}S^2]$, equation (10) has four unequal and conjugate imaginary roots, the no-disease equilibrium E∗ 1 and endemic equilibrium E∗ 2 of system (2) is stable.

3.3 $\tau 1 = 0, \tau 2 = \tau$

When $\tau 1 = 0$, $\tau 2 = \tau$, equation(14) becomes to the following equation:

$$P(\lambda, \tau) = -\lambda^4 + L\lambda^3 + M_3\lambda^2 + N_3\lambda - (R + S)e^{-\tau\lambda} + T \tag{17}$$

In summary, we have the following theorem: Theorem 3. When $\tau 1 = 0$, $\tau 2 = \tau$ and $(M_3^2 - 2LM_3)^2 \geq 4(R + S)^2$, equation (17) has four real roots, the no-disease equilibrium E_1^* and endemic equilibrium E_2^* of equation(2) is unstable. When $(M_3^2 - 2LM_3)^2 < 4(R + S)^2$, equation (11) has four unequal and conjugate imaginary roots, the no-disease equilibrium E_1^* and endemic equilibrium $E_2^*$of equation(2) is stable.

3.4 $\tau 1 = \tau 2 = \tau$

When $\tau 1 = \tau$, $\tau 2 = \tau$, equation(14) becomes to the following equation:

$$P(\lambda, \tau) = -\lambda^4 + L\lambda^3 + M_4\lambda^2 + N_4\lambda - \mathrm{Re}^{-\tau\lambda} - v\beta_1\beta_2\bar{S}_1\bar{S}_2e^{-2\tau\lambda} \tag{18}$$

4. Numerical simulation

For the analytical solution of equation (14) is too complex, so we solve its numerical solutions. When $\tau 1 = \tau 2 = 0$, set the value of the parameters are $\mu 1 = 0.6$, $\mu 1 = 0.6$, $\beta 1 = 0.01$, $\beta 2 = 0.04$, $\gamma 1 = 0.95$, N1 = 33, A = 38, we solve equation (14) and gain the characteristic root are:

$$\lambda_1 = 37.5675,$$

$$\lambda_2 = 0.2279,$$

$$\lambda_3 = —0.0828 + 0.1559i,$$

$$\lambda_4 = —0.0828 — 0.1559i.$$

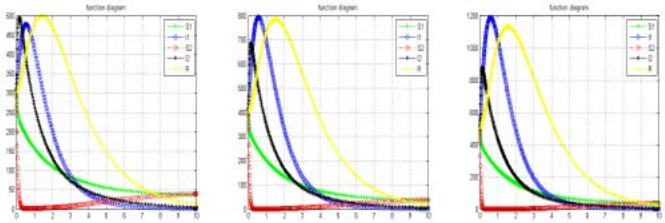
At this time, the basic reproductive ratio $R_0 = 0.3995 < 1$. The non-disease equilibrium point is $E_1^*(S_1, I_1, S_2, I_2) = (33, 0, 42.2222, 0)$, The endemic equilibrium point is $E_2^*(S_1, I_1, S_2, I_2) = (53.4864, 7.9302, 65.2035, 22.9813)$. Therefore, we have:

Theorem 4. When $\tau 1 = \tau 2 = 0$, the non-disease equilibrium point is $E_1^*$and the endemic equilibrium point E_2^* are stable.

We discuss the time delays affect on the number of susceptible and patients in three cases:(1)$\tau 1 = \tau$, $\tau 2 = 0$, (2)$\tau 1 = 0$, $\tau 2 = \tau$, (3)$\tau 1 = \tau 2 = \tau$.

4.1 $\tau 1 = \tau, \tau 2 = 0$

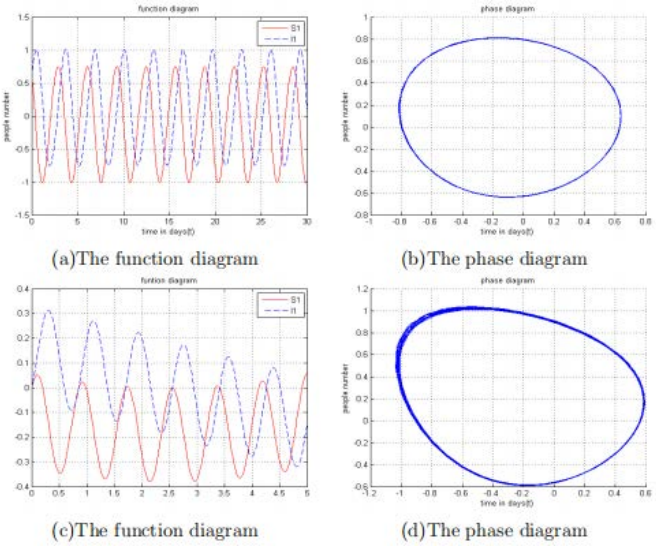
For the analytical solution of equation is too complex, so we solve its numerical solutions. When $\tau 1 = 3$, $\tau 2 = 0$, set the value of the parameters are $\mu 1 = 0.6$, $\mu 1 = 0.6$, $\beta 1 = 0.01$, $\beta 2 = 0.04$, $\gamma 1 = 0.95$, N1 = 33, A = 38, we gain the characteristic root is: $\omega = =0.2423 — 0.1502i$. At this time, the characteristic root is $\lambda 1 = i\omega = 0.1502 — 0.2423i$. The real positive root of equation is $\omega_0 = 1.3594$, and $\tau_0^* = 0.6155$.



(a) (b) (c)

Figure 1: The function diagram of different N1, where (a) is N1 = 300, (b) is N1 = 400, (c) is N1 = 500, the initial value A = 60, (S1, I1, S2, I2, R) = (40, 40, 40, 40, 40).

As the first case $\tau1=\tau$, $\tau2=0$, we choose different iteration step (IS), to simulate the phase diagram of the system, when the time delay $\tau2 > \tau0$, the system appears periodic solution and limit cycle, the result are showed as the following figures.



(a)The function diagram (b)The phase diagram

(c)The function diagram (d)The phase diagram

Figure 2: The function diagram and phase diagram of different iteration step, where (a) and (b) is the function diagram and phase diagram of IS=0.05, $\tau1\geq\tau0$, $\tau2=0$, (c) and (d) is the function diagram and phase diagram of IS=0.5, $\tau1\geq\tau0$, $\tau2=0$.

Theorem 5. When $\tau < \tau0$, the non-disease equilibrium point is E_1^* and the endemic equilibrium point E_2^* are asymptotically stable; when $\tau > \tau_0$, the endemic equilibrium point E_2^* is unstable, when τ is increasing and through $\tau0$, the endemic equilibrium point E_2^* branch into periodic small amplitude solutions.

4.2 $\tau1 = 0, \tau2 = \tau$

For the analytical solution is too complex, so we solve its numerical solutions.When $\tau1 = 0$, $\tau2 = 3$, set the value of the parameters are $\mu1 = 0.6$, $\mu1 = 0.6$, $\beta1 = 0.01$, $\beta2 = 0.04$, $\gamma1 = 0.95$, $N1 = 33$, $A = 38$, we gain the characteristic root is: $\omega = -0.0269 + 0.1885i$. At this time, the characteristic root is $\lambda_2 = i\omega = -0.1885 - 0.0269i$. The real positive root of equation(18) is $\omega0 = 0.7597$, and $\tau_0^* = 0.0490$.

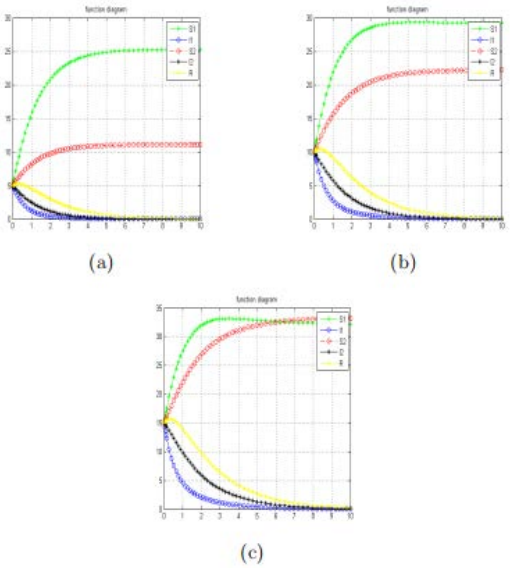


Figure 3: The function diagram of different N1 and A, and of different initial value S1, I1, S2, I2, where (a) is N1 = 30, A = 10, IV = 5, (S1, I1, S2, I2, R) = (5, 5, 5, 5, 5), (b) is N1 = 40, A = 20, IV = 10,(S1, I1, S2, I2, R) = (10, 10, 10, 10, 10), (c) is N1 = 50, A = 30, IV = 15, (S1, I1, S2, I2, R) = (15, 15, 15, 15, 15).

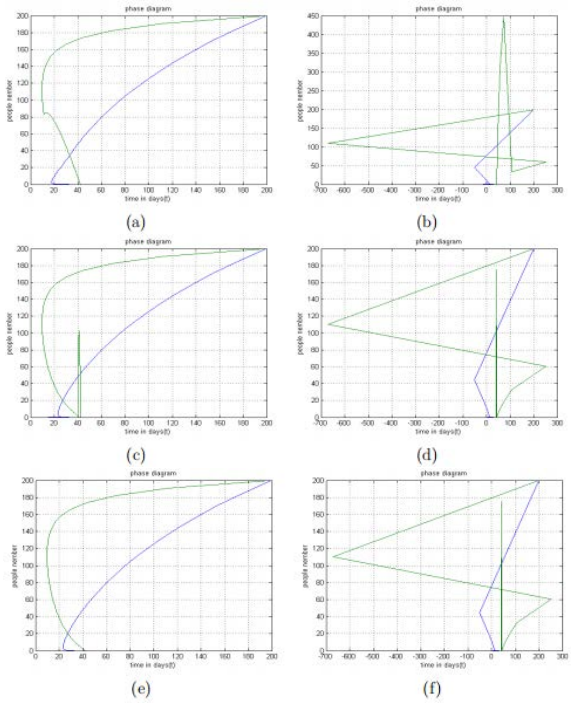


Figure 4: The phase diagram of different iteration step t, where (a) is IS = 0.05, τ1 = 0, τ2 = 1, (b) is IS = 0.5, τ1 = 0, τ2 = 1, (c) is IS = 0.05, τ1 = 0, τ2 = 5, (d) is IS = 0.5, τ1 = 0, τ2 = 5, (e) is IS = 0.05, τ1 = 0, τ2 = 50, (f) is IS = 0.5, τ1 = 0, τ2 = 50.

When the time delay τ2 > τ0,the system appear periodic solution and limit cycle, it is showed as the following figure.

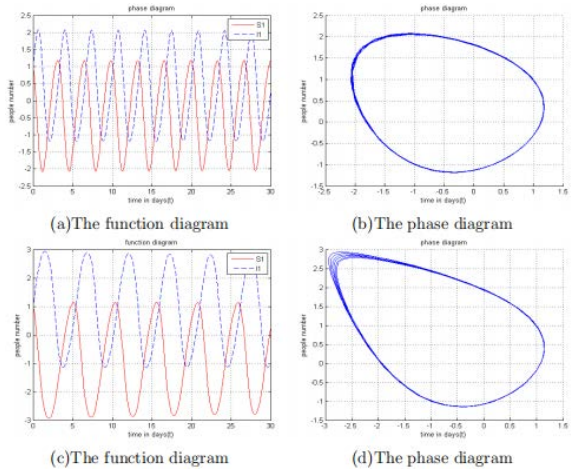


Figure 5: The function diagram and phase diagram of different iteration step, where (a) and (b) is the function diagram and phase diagram of IS = 0.05, $\tau 1 = 0$, $\tau 2 \geq \tau 0$, (c) and (d) is the function diagram and phase diagram of IS = 0.5, $\tau 1 = 0$, $\tau 2 \geq \tau 0$.

In summary, we have the following theorem:

Theorem 6. When $\tau < \tau_0^*$, the non-disease equilibrium point is E_1^* and the endemic equilibrium point E_2^* are asymptotically stable; when $\tau > \tau_0^*$, the endemic equilibrium point E_2^* is unstable, when τ is increasing and through τ_0^*, the endemic equilibrium point E_2^* branch into periodic small amplitude solutions.

4.3 $\tau 1 = \tau 2 = \tau$

Solving equation, we gain $\omega = -0.0147i$. At this time, the characteristic root is $\lambda_3 = i\omega = 0.0147$. When $\tau_1 = \tau_2 \geq 60$, By calculating, we gain $\omega_0 = 0.8861$, $\tau^{**} = 3.5454$.

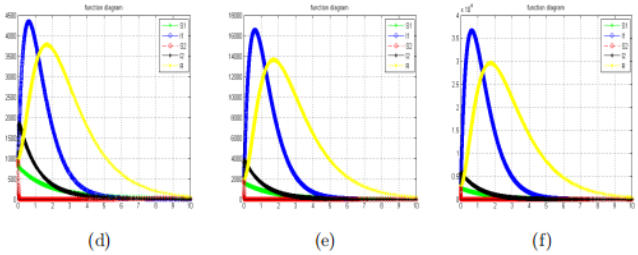


Figure 6: The function diagram of different N1, where (a) is N1 = 1000, (b) is N1 = 2000, (c) is N1 = 3000, the initial value A = 60, (S1, I1, S2, I2, R) = (40, 40, 40, 40, 40).

In order to simulate the phase of the system when $\tau 1 = \tau 2 = \tau$, we choose the different iteration step (IS) t, the result are showed as the following figures.

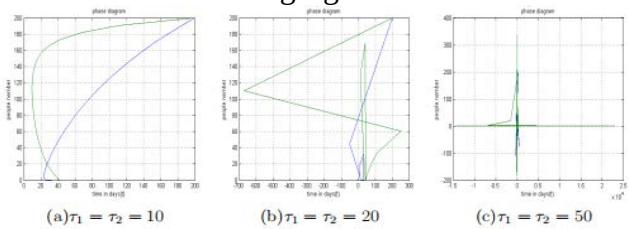


Figure 7: The phase diagram of the system when $\tau 1 = \tau 2 = \tau$, where (a) is the IS = 0.05, (b) is the IS = 0.5, (c) is IS = 1

In summary, we have the following theorem:

Theorem 7. When $(T - \omega_0^4 - M_4\omega_0^2)^2 + (L - N)^2\omega_0^2 \geq R^2 + S^2$ and for any $\tau_0^{**}$the non-disease equilibrium point is E_1^* and the endemic equilibrium point E_2^* are unstable.

5. Conclusion

Recently, dengue fever which is considered to be a tropical disease forms a major concern due to its severity and complexity in the world. It brings a serious threat to human health and life. Therefore, it is of great importance and meaning to study the infections of the dengue fever. There are many factors that can influence the spread of dengue fever, here we only consider the time delay.

In this paper, on the basis of the dengue model which was proposed by Tewa et.al. [21] and Dr. Ding Deqiong [22], we improve the model, present a model based on several features of dengue infection with time delay. We consider two time delays $\tau 1$ and $\tau 2$, where $\tau 1$ and $\tau 2$ represent the rehabilitees immune period and incubation period to the disease respectively. This paper analyzed the stability of the dengue model with time delay and gained the specific value of time delay threshold $\tau 0$ at which the dengue model appears periodic solutions and Hopf bifurcation.

In this paper, we discuss the effect of the time delay in three cases, that are (1)$\tau 1 = \tau$, $\tau 2 = 0$, (2)$\tau 1 = 0$, $\tau 2 = \tau$, (3)$\tau 1 = \tau 2 = \tau$. On each case, by calculating the model, we gained the time delay threshold $\tau 0$ at which the dengue model appears periodic solutions and Hopf bifurcation. On the first case, $\tau 1 = \tau$, $\tau 2 = 0$, $\tau 0 = 0.6155$; on the second case $\tau 1 = 0$, $\tau 2 = \tau$, $\tau 0 = 0.0490$; on the third case $\tau 1 = \tau 2 = \tau$, $\tau 0 = 3.5454$. From this, we can see that, all of the time delay $\tau 1$ and $\tau 2$ play importance role in the spreading of the dengue fever epidemic.

Our numerical results imply that in general the dengue fever epidemic latent in 7-14 days which is in agreement with clinical literature.

References

[1] World Health Organization. Dengue and severe dengue, 2013. http://www.who.int/mediacentre/factsheets/fs117/en/index.html. [accessed 22.09.13].

[2] World Health Organization. Health topics (dengue), 2013. http://www.who.int/topics/dengue/en/. [accessed 22.09.13]

[3] Hethcote, H.W., The Mathematics of Infectious Disease [J], SIAM Review, 42 (2000), 599-653.

[4] Freedman, H.I., Tang, M.X., Ruan, S.G., Uniform Persistence and Flows near a Closed Positively Invariant Set [J], Journal of Dynamics and Differential Equations, 6 (1994), 583-600.

[5] Li, M.Y., Graef, J.R., Wang, L., et al., Global Dynamics of a SEIR Model with Varying Total Population Size [J], Mathematical Biosciences, 160 (1999), 191-213.

[6] Feng, Z., Vealsco-Hernandez, V., Competitive Exclusion in a Vectorhost Model for the Dengue Fever [J], Journal of Mathematical Biology, 1997, 35:523-544.

[7] Naresh, R., Tripathi, A., Sharma, D., A Nonlinear AIDS Epidemic Model with Screening and Time Delay [J], Journal of the Mathematical Society of Japan, 2011, 217:4416-4426.

[8] McCluskey, C.C., Global Stability for an SIR Epidemic Model with Delay and Nonlinear Incidence [J], Nonlinear Analysis: Real World Applications, 2010, 11:3106-3109.

[9] Marsden, J.E., Sirovich, L., John, F., Nonlinear Oscillations Dynamical Systems, and Bifurcations of Vector Fields [M], Springer-Verlag, New York, 1997, 4-5.

[10] Kermack, W.O., McKendrick, A.G., Contributions to the Mathematical Theory of Epidemics [J], Proceedings of the Royal Society A, A115 (1927), 700-720.

[11] Kermack, W.O., McKendrick, A.G., Contributions to the Mathematical Theory of Epidemics [J], Proceedings of the Royal Society A, A138 (1932), 55-83.

[12] Hethcote, H.W., Li, Y., Jing, Z., Hopf Bifurcation in Models for Pertussis Epidemiology [J], Mathematical and Computer Modeling, 30 (1999), 29-45.

[13] Babar, A., Jamil, A., Amjad, A., Rehan, Z.P., Samar, H.K.T., Umar, N., Tariq, S., On the

modeling and analysis of the regulatory network of dengue virus pathogenesis and clearance [J], Computational Biology and Chemistry, 53 (2014), 277-291.

[14] Lung-Chang Chien, Hwa-Lung Yu, Impact of meteorological factors on the spatiotemporal patterns of dengue fever incidence, [J], Environment International, 73 (2014), 46-56.

[15] Tanvi, P., Gujarati, G.A., Virus antibody dynamics in primary and secondary dengue infections [J], Mathematical Biology, 69 (2014), 1773-1800.

[16] Garcia-Garaluz, E., Atencia, M., Joya, G., Garcia-Lagos, F., Sandoval, F., Hopfield networks for identification of delay differential equaltions with an application to dengue fever epidemics in Cuba [J], Neurocomputing, 74 (2011), 2691-2697.

[17] Newton, E.A., Reiter, A., A model of the Transmission of Dengue Fever with an Evaluation of the Impact of Ultra-low Volume (ULV) Insecticide Applications on Dengue Epidemics [J], American Journal of Tropical Medicine and Hygiene, 47 (1992), 709-720.

[18] Esteva, L., Vargas, C., Analysis of a Dengue Disease Transmission Model [J], Mathematical Biosciences, 150 (1998), 131-151.

[19] Esteva, L., Vargas, C., A Model for Dengue Disease with Variable Human Population [J], Journal of Mathematical Biology, 46 (1999), 220-240.

[20] Esteva, L., Vargas, C., Influence of Vertical and Mechanical Transmission on The Dynamics of Dengue Disease [J], Mathematical Biosciences, 167 (2000), 51-64.

[21] Tewa, J.J., Dimi, J.L., Bowong, S., Lyapunov Functions for a Dengue Disease Transmission Model [J], Chaos Solutions and Fractals, 39 (2009), 936-941.

[22] Ding, Deqiong, The numerical solution and global stability of two classes of infectious disease [D], Harbin Institute of Technology, 2011, 6.