Limit Cycle and Chaotic Behaviors for the Transmission Model of *Plasmodium Vivax* Malaria

P.Pongsumpun., and I.M.Tang

***Abstract*—** Malaria is an infectious disease caused by the bite of female *Anopheles* mosquitoes. There are four species, namely, *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium ovale* and *Plasmodium malariae* causing human malaria. The difference between *P.vivax* malaria and *P. falciparum* malaria is that a person suffering from *P. vivax* infection can suffer relapses of the disease. The effect of a time delay on the transmission of this disease is studied. The time delay is the period in which the *P.vivax* parasite develops inside the mosquito (vector) before the vector becomes infectious (i.e., pass on the infection). The model is analyzed by using standard dynamic modeling method. Two stable equilibrium states are found to be possible. It is found that the disease free equilibrium state is stable when a newly defined basic reproduction number L is less than one. If L is greater than one, the endemic equilibrium state is stable. The conditions for the endemic equilibrium state to be a stable spiral node are found. For realistic values of the parameters in the model, it is found that solutions in phase space are trajectories spiraling into the endemic equilibrium state. The bifurcation diagrams of our model are discussed. It is shown that the limit cycle and chaotic behaviors can occur with only unrealistic situations.

***Keywords*—**Limit cycle, local stability, *Plasmodium Vivax*, time delay.

I. Introduction

THE top six of the world's serious diseases in the world is Malaria. In each year, there are more than three hundred million cases due to this disease with between 1 and 1.5 million death annually (mostly in children). The evolutional biology [1] of the parasite *Plasmodium vivax* determines to a great extent the mathematical model needed to describe the transmission cycle of the human disease caused by this parasite. After being bitten by an infected mosquito, sporizoites (one of the stages of the malaria parasite) are introduced into the blood stream of the human. These then move to the liver of the human. Some of them transform themselves into merozoites, which then invade the blood cells and cause the illness. The remaining sporizoites are transformed into hypnozoites which then lay dormant in the liver. The relapses occur when some of the hypnozoites transform themselves into schizents and then into merozoites.

These new merozoites then reinvade the blood and cause the illness again. These relapses can occur up to three years after the initial infection. Only a small number of the *P. vivax* merozoites remain in the blood between the relapse episodes. The hypnozoite stage does not occur in the three other types of malaria, *P.falciparum*, *P. malariae* and *P. ovale*.

The absence of the hypnozoite stage in the malaria caused by the *P. falciparum* parasite makes the transmission models used to describe *P. falciparum* malaria invalid for describing the transmission of the malaria caused by the *P. vivax* parasite. The reasons for *P. falciparum* malaria to be studied more than P. vivax malaria are (1) most of the deaths due to malaria (2-3 million a year) occur in Africa [2] (2) 90% of the malaria cases in Africa is due to *P. falciparum* malaria and (3) P. falciparum malaria is a life threatening disease, whereas P .vivax malaria is not. It was commonly assumed that information about vivax could be extrapolated from the falciparum research. This assumption was challenged at a recent conference convened by the Multilateral Initiative on Malaria [3]. The transmission of malaria is usually described by the Ross-MacDonald (RM) model [4]. However, this model is only suitable for the transmission of the P. falciparum malaria since it does not contain the possibility of relapses of the illness. One of the present authors (IMT) has introduced a simple mathematical model [5] to describe the transmission of P. vivax malaria. The authors (PP & IMT) have presented the transmission model of P.vivax malaria with the effect of relapse but we did not consider the transmission of this disease when limit cycle and chaotic behaviors occur [6]. In the model, we included a dormant class in which there are no merozoites in the blood, only dormant hypnozoites in the liver. A person can be re-infected when the hypnozoites are re-activated.

Because there is no place for human experimentation to see what would happen if new therapies are adopted, mathematical model allows one to simulate what would occur.

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We introduce in Section 2, the modification of the model which would make it applicable to the transmission of P. vivax malaria In Section 3, we analyze our model to find the conditions for the local stability of each equilibrium point. The numerical simulations confirm the local stability of the endemic equilibrium point. Conditions for Hopf bifurcation are found. We found that limit cycle behavior and chaotic behavior can occur for the unrealistic parameter values.

II. TRANSMISSION MODEL

In 1911, Ross started the mathematical modeling of the epidemiology of malaria (*P. falciparum*) [7]. In the Ross model, an individual in the human population is separated to a non-infected and infected state. This gives rise to what is known as a SIS (susceptible-infected-susceptible) model. It has been suggested [8] that the human population should be divided into three states; non-infected, infected but without any acute clinical signs, infected with acute clinical sign, to better reflect the clinical status of the individual. Others believe that the population should be further divided into susceptible, infected but not infectious and infected and infectious.

The transmission of P. vivax is constructed by dividing the host (human) population into susceptible $(\bar{\bar{S}}_h)$, infected $(\bar{\bar{I}}_h)$, dormant $(\bar{\bar{D}}_h)$ and recovered $(\bar{\bar{R}}_h)$ classes. The last category, the recovered are susceptible to further infections and so they reenter into the $\bar{\bar{S}}_h$ class. λN_T humans are entering into the susceptible class through birth and $(1-\theta)r_1\bar{\bar{I}}_h$, $r_3\bar{\bar{D}}_h$ and $r_6\bar{\bar{R}}_h(t)$ through the recovery of members of the infected and dormant categories (with λ being the birth rate; N_T, the total human population; r_1, the recovery rate of a person in the infected category; r_3, the recovery rate of a member of the dormant population and θ being the percentage of infected people in whom some hypnozoites remain dormant in the liver). $(1-\theta)$ is the percentage of infected humans who recover and become susceptible again. The time rate of change of the number of susceptible members is equal to the number entering minus the number leaving. This gives us the following differential equation for the time rate of change of the susceptible population;

$$\frac{d}{dt}\bar{\bar{S}}_h(t) = \lambda N_T + r_3\bar{\bar{D}}_h(t) + (1-\theta)r_1\bar{\bar{I}}_h(t) - \gamma'_h\bar{\bar{I}}_v(t)\bar{\bar{S}}_h(t) - \mu_h\bar{\bar{S}}_h(t) + r_4\bar{\bar{R}}_h(t), \tag{1.1}$$

$$\frac{d}{dt}\bar{\bar{I}}_h(t) = \gamma'_h\bar{\bar{I}}_v(t)\bar{\bar{S}}_h(t) - (r_1+\mu_h)\bar{\bar{I}}_h(t) + r_2\bar{\bar{D}}_h(t) - r_5\bar{\bar{R}}_h(t), \tag{1.2}$$

$$\frac{d}{dt}\bar{\bar{D}}_h(t) = \alpha r_1\bar{\bar{I}}_h(t) - (r_2+r_3+\mu_h)\bar{\bar{D}}_h(t) \tag{1.3}$$

and

$$\frac{d}{dt}\bar{\bar{R}}_h(t) = r_5\bar{\bar{I}}_h(t) - (r_2+\mu_h)\bar{\bar{R}}_h(t) \tag{1.4}$$

where the parameters in the above equations are given by

- λ = the birth rate of human population,
- μ_h = the death rate of human population,
- N_T = the total number of human population,
- α = the percentage of infected human in whom some hypnozoites remain dormant in the liver,
- r_1 = the rate at which a person leaves the infected class by recovering or by entering into the dormant class,
- r_2 = the rate at which the dormant human relapses back to the infected human,
- r_3 = the recovery rate of the dormant human,
- r_4 = the rate at which the recovered human relapses back to the susceptible human, and
- r_5 = the rate at which the infected human recovers.

Because *P. vivax* infection is non lethal, the death rates will be the same for all human classes then we have

$$\bar{\bar{S}}_h + \bar{\bar{I}}_h + \bar{\bar{D}}_h + \bar{\bar{R}}_h = N_T$$

The term $\gamma'_h\bar{\bar{I}}_v(t)\bar{\bar{S}}_h(t)$ is contained in equation (1a). This term represents the lost of the susceptible person due to a bite of an infected mosquito. γ'_h is the rate at which the *P. vivax* parasite is transmitted from the mosquito to the human and is given by [9]

$$\gamma'_h = c\frac{\beta_h}{N_T + m} \tag{2}$$

where c is the specie-dependent biting rate of the mosquitoes; m is the population of other animals that the mosquitoes can feed on and β_h is the probability the parasite passed on by the mosquito will continue to thrive in the human. β_h depends partly on the immune response of the host to the infection. $\bar{\bar{I}}_V$ is the number of infected mosquitoes. The dynamics equations of the mosquitoes are given by

$$\frac{d}{dt}\bar{\bar{S}}_v(t) = V - \gamma'_v\bar{\bar{S}}_v(t)\bar{I}_h(t) - \mu_v\bar{\bar{S}}_v(t) \tag{3.1}$$

$$\frac{d}{dt}\bar{\bar{I}}_v(t) = \gamma'_v\bar{\bar{S}}_v(t-\tau)\bar{\bar{I}}_h(t-\tau)e^{-\mu_v\tau} - \mu_v\bar{\bar{I}}_v(t) \tag{3.2}$$

In this study, we are interested in the time rate of change of the infectious vector at time t and τ is the number of days for the infected vector to become infectious. We consider the number of susceptible vector who bit an infected human at time $t-\tau$ not at time t. Fraction of the infected mosquito would have died between the time t and $t-\tau$.

The total number of female mosquitoes at the equilibrium state will be V/μ_V. V is the rate at which the mosquitoes are recruited and μ_V is the death rate for the mosquitoes. It should be observed that a mosquito can not be infected through a bite of a human belonging to the dormant class. γ'_v is the rate at which the mosquitoes become infected with the Plasmodium

Vivax parasite once the mosquito has bitten an infected human. γ'_v is defined by [9]

$$\gamma'_v = c\frac{\beta_v}{N_T + m}$$

where m is the number of other animals that the mosquitoes can bite and β_v is the probability the parasite passed to the mosquito by biting human. We also assume $N_V = \bar{\bar{S}}_v + \bar{\bar{I}}_v$. The working equations of the model are obtained by dividing (1.1), (1.2), (1.3) and (1.4) by N_T and (3.1) and (3.2) by V/μ_v. This would give us six equations expressed in terms of the renormalized variables;

$$S_h = \bar{\bar{S}}_h/N_T,\ I_h = \bar{\bar{I}}_h/N_T,\ R_h = \bar{\bar{R}}_h/N_T,\ S_v = \bar{\bar{S}}_v/(V/\mu_v)$$

and $I_v = \bar{\bar{I}}_v/(V/\mu_v)$.

The conditions $S_h + I_h + D_h + R_h = 1$ and $S_v + I_v = 1$, leads to only four of these equations being needed. We pick the four equations to be

$$\frac{d}{dt}S_h(t) = \mu_h + r_3 D_h(t) + (1-\alpha)r_1 I_h(t) - \gamma_h I_v(t) S_h(t) - \mu_h \bar{S}_h(t) + r_4(1 - S_h(t) - I_h(t) - D_h(t)) \quad (4.1)$$

$$\frac{d}{dt}I_h(t) = \gamma_h I_v(t) S_h(t) - (r_1 + \mu_h) I_h(t) + r_2 D_h(t) - r_5 I_h(t) \quad (4.2)$$

$$\frac{d}{dt}D_h(t) = \alpha r_1 I_h(t) - (r_2 + r_3 + \mu_h) D_h(t)) \quad (4.3)$$

and

$$\frac{d}{dt}I_v(t) = \gamma_v(1 - I_v(t-\tau))\, I(t-\tau) e^{-\mu_v \tau} - \mu_v I_v(t) \quad (4.4)$$

where the new transmission rates are $\gamma_h = \gamma'_h(V/\mu_v)$ and $\gamma_v = \gamma'_v N_T$. The domain of solutions is

$$\Omega = \{(S_h, I_h, D_h, R_h, S_v, Iv) \mid 0 \le S_h + I_h + D_h + R_h \le 1,\ 0 \le S_v + I_v \le 1\}$$

We have substituted $I(t-\tau)$ by $I(t)$ since the density of infectious human is not anticipated to vary much over the period τ which is much less than the life expectancy of human.

III. ANALYSIS OF THE MATHEMATICAL MODEL

A. Analytical Results

The equilibrium states are found by setting the RHS's of (4.1) to (4.4) to zero. We obtain two equilibrium states: the disease free equilibrium state $E_O = (1, 0, 0, 0)$ and the endemic equilibrium state $E_1 = (S_h^*, I_h^*, D_h^*, I_v^*)$ where

$$S_h^* = \frac{I_h r_1(1-\theta) + D_h^* r_3 + r_4(1 - D_h^* - I_h^*) + \mu_h}{\gamma_h I_v^* + r_4 + \mu_h},$$

$$I_h^* = \frac{I_v^* \mu_v}{e^{-\mu_v \tau}\gamma_v(1 - I_v^*)},$$

$$D_h^* = \frac{\alpha r_1 I_h^*}{r_2 + r_3 + \mu_h},$$

$$I_v^* = \frac{L_0 e^{-\mu_v \tau} - 1}{\gamma + \beta e^{\mu_v \tau}} \quad (5)$$

with

$$L_0 = \frac{(\mu_h + r_2 + r_3)\gamma_h \gamma_v}{\mu_v(\mu_h^2 + \mu_h r_{1235} + r_1((1-\alpha)r_2 + r_3) + (r_2 + r_3)r_5)}$$

$$\beta = \gamma_h \mu_v \left(\mu_h + r_2 + r_3 + \alpha r_1 + \frac{(\mu_h + r_2 + r_3)r_5}{\mu_h + r_4}\right)$$

$$\gamma = \gamma_h \gamma_v (\mu_h + r_2 + r_3)$$

$$r_{1235} = r_1 + r_2 + r_3 + r_5 \quad (6)$$

Endemic equilibrium state exists for $e^{-\mu_v \tau} L_0$ greater than 1 or τ must be in the interval $0 < \tau < (\ln L_0)/\mu_v$. Let $L = e^{-\mu_v \tau} L_0$ then L is the basic reproduction number. It denotes the number of secondary infections resulting from a primary infection. The local stability of each equilibrium state is determined by the sign of all eigenvalues. If all eigenvalues have negative real parts, then that equilibrium state is locally stable. Eigenvalues for each equilibrium state are obtained by solving the characteristic equation

$$\det(J - \lambda I) = 0 \quad (7)$$

where J is the Jacobian matrix calculated at the equilibrium state.

The correspondent eigenvalues for each equilibrium state are found by solving the characteristic equation; which is in the form

$$E(\lambda, \tau) + \frac{F(\lambda, \tau)}{e^{\mu_v \tau}} = 0 \quad (8)$$

where

$$E(\lambda, \tau) = \lambda^4 + x_3(\tau)\lambda^3 + x_2(\tau)\lambda^2 + x_1(\tau)\lambda + x_0(\tau) \quad (9)$$

$$F(\lambda, \tau) = y_3(\tau)\lambda^3 + y_2(\tau)\lambda^2 + y_1(\tau)\lambda - y_0(\tau) \quad (10)$$

and

$x_3, x_2, x_1, x_0, y_3, y_2, y_1, y_0$ are functions of the time delay (τ).

For $\tau = 0$, the correspondent eigenvalues for each equilibrium state are found by solving the characteristic equation; which is in the form

$$x_0(0) + x_1(0)\lambda + x_2(0)\lambda^2 + x_3(0)\lambda^3 + \lambda^4.$$

The coefficients $x_3(0), x_2(0), x_1(0), x_0(0)$ are constants in this case. We let

$$z_3 = x_3(0), z_2 = x_2(0), z_1 = x_1(0), z_0 = x_0(0)$$

By Routh-Hurwitz criteria [10], each equilibrium point is locally stable when the following conditions are satisfied;

I) $z_3 > 0$, (11)

II) $z_1 > 0$, (12)

III) $z_0 > 0$, (13)

IV) $z_3 z_2 z_1 > z_1^2 + z_3^2 z_0$ (14)

The above conditions are checked by using MATHEMATICA (Wolfram Research, Champaign, IL), then we found that for $L_0 < 1$, E_0 will be locally stable and for $L_0 > 1$, E_1 will be locally stable.

B. Bifurcation Conditions for the Endemic State

Ruan, Wei [11], Klan and Greenhalgh [12] obtained the characteristic equations for their models:

$$\lambda^3 + p\lambda^2 + q\lambda + r = se^{-\lambda\tau} \ , \quad (15)$$

while the characteristic equation studied by Tam [12] has the form

$$\lambda^3 + p\lambda^2 + (q + re^{-\lambda\tau})\lambda + s = ue^{-\lambda\tau} \quad (16)$$

The constants values p, q, r, s and u in (15) and (16) are defined in the respective references. The important thing to note is that these constant do not depend on τ.

To determine the conditions for Hopf bifurcation, we apply the techniques used in [11],[12] and [14].Substituting $\lambda = r + si$ (where r and s are real numbers and may be functions of τ) into (8) and separating the real and imaginary parts, we obtain

$$\begin{aligned} &r(\tau)^4 - 6r(\tau)^2 s(\tau)^2 + s(\tau)^4 + x_0(\tau) + r(\tau)x_1(\tau) \\ &+ r(\tau)^2 x_2(\tau) - s(\tau)^2 x_2(\tau) + r(\tau)^3 x_3(\tau) \\ &- 3c(\tau)s(\tau)^2 x_3(\tau) + e^{-c(\tau)\tau}\cos(s(\tau)\tau)(r(\tau)y_1(\tau) \\ &- y_0(\tau) + r(\tau)^2 y_2(\tau) - s(\tau)^2 y_2(\tau) + r(\tau)^3 y_3(\tau) \\ &- 3r(\tau)s(\tau)^2 y_3(\tau)) + e^{-r(\tau)\tau}\sin(s(\tau)\tau)(s(\tau)y_1(\tau) \\ &+ 2r(\tau)s(\tau)y_2(\tau) + 3r(\tau)^2 d(\tau)y_3(\tau) - s(\tau)^3 y_3(\tau)) = 0 \end{aligned} \quad (17)$$

and

$$\begin{aligned} &4r(\tau)^3 s(\tau) - 4r(\tau)s(\tau)^4 x_1(\tau) + 2r(\tau)s(\tau)x_2(\tau) \\ &+ 3r(\tau)^2 s(\tau)x_3(\tau) - s(\tau)^3 x_3(\tau) \\ &+ e^{-c(\tau)\tau}\cos(s(\tau)\tau)(s(\tau)y_1(\tau) + \\ &2r(\tau)s(\tau)y_2(\tau) + 3r(\tau)^2 s(\tau)y_3(\tau) - s(\tau)^3 y_3(\tau) \\ &+ e^{-c(\tau)\tau}\sin(s(\tau)\tau)(y_0(\tau) - r(\tau)y_1(\tau) - r(\tau)^2 y_2(\tau) \\ &+ s(\tau)^2 y_2(\tau) - r(\tau)^3 y_3(\tau) + 3r(\tau)s(\tau)^2 y_3(\tau) = 0 \end{aligned} \quad (18)$$

We now let $\tau = \tau_c$. At this point, $r(\tau_c) = 0$ .We denote $s(\tau_c)$ as $\tilde{s}$, (17) and (18) become

$$\begin{aligned} &\tilde{s}^4 + x_0(\tau_c) - \tilde{s}^2 x_2(\tau_c) \\ &= (y_0(\tau_c) + \tilde{s}^2 y_2(\tau_c))\cos(\tilde{s}\tau_c) \\ &- s(y_1(\tau_c) - s^2 y_3(\tau_c))\sin(\tilde{s}\tau_c) \end{aligned} \quad (19)$$

$$\begin{aligned} &\tilde{s}^3 x_3(\tau_c) \\ &= \tilde{s}(y_1(\tau_c) - \tilde{s}^2 y_3(\tau_c))\cos(\tilde{s}\tau_c) \\ &+ (y_0(\tau_c) + \tilde{s}^2 y_2(\tau_c))\sin(\tilde{s}\tau_c) \end{aligned} \quad (20)$$

Squaring (19) and (20) and adding them together, we obtain

$$f(\delta) = \delta^4 + h_3(\tau_r)\delta^3 + h_2(\tau_r)\delta^2 + h_1(\tau_r)\delta + h_0(\tau_r) = 0 \quad (21)$$

where $\delta = \tilde{s}^2$ and

$$\begin{aligned} h_3(\tau_r) &= -2x_2(\tau_r) + x_3(\tau_r)^2 - y_3(\tau_r)^2 \\ h_2(\tau_r) &= 2x_0(\tau_r) + x_2(\tau_r)^2 - y_2(\tau_r)^2 + 2y_1(\tau_r)y_3(\tau_r) \\ h_1(\tau_r) &= -(2x_0(\tau_r)x_2(\tau_r) + y_1(\tau_r)^2 + 2y_0(\tau_r)y_2(\tau_r) \\ h_0(\tau_r) &= x_0(\tau_r)^2 - y_0(\tau_r)^2 \end{aligned} \quad (22)$$

We note that $h_3(\tau_r), h_2(\tau_r), h_1(\tau_r), h_0(\tau_r)$ are real. Critical point value τ_c is always determined from the requirement that $c(\tau_r) = 0$. In the technique used here, the critical point is determined from the condition that at least one root of (21) be real and positive, otherwise $d = \sqrt{\delta_0}$ (δ_0 is the root of the equation) would be imaginary. The existence of an imaginary part of the eigenvalue depends on whether equation (21) has a positive real root.

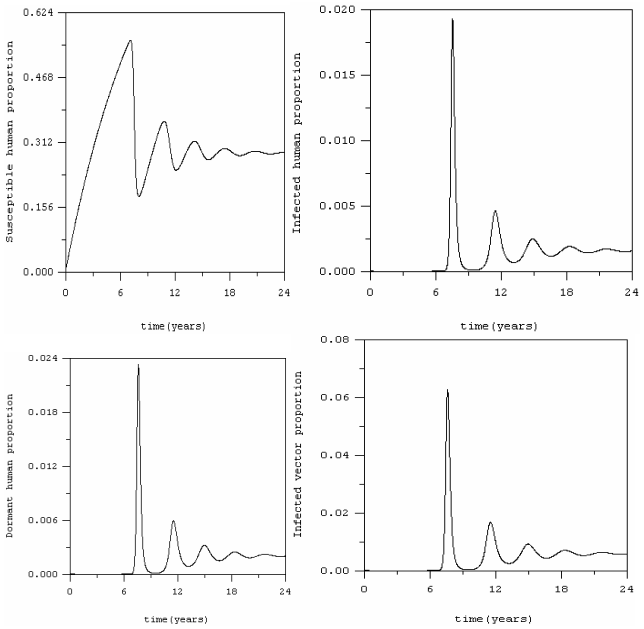
We use MATHEMATICA (Wolfram Research, Champaign, IL) to check whether equation (21) has a positive real root.

C. Numerical Results

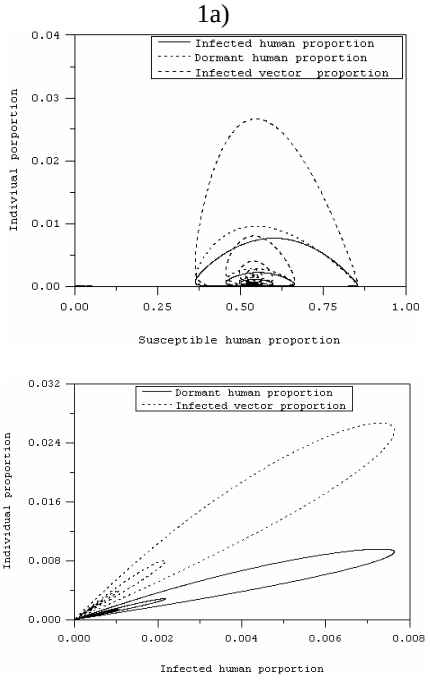
C.I. Realistic Parameter Values

The numerical simulations of the endemic equilibrium state are displayed in each case, in this section. The parameters are determined by real life observations. $\mu_h = 0.0000421^{-1}$ day corresponds to the real life expectancy of 65 years for human. $r_1 = 1/14^{-1}$ day corresponds to the 14 days of a person leaves the infected class by recovering or by entering into the dormant class. $r_2 = 1/(365*3)^{-1}$day corresponds to the 3 years of the relapse of the human. $r_3 = 1/25^{-1}$day corresponds to the 25 days of the recovery of the dormant human, $r_4 = 1/(365*10)^{-1}$day satisfy 10 years of the recovered human relapses back to the susceptible human. $r_5 = 1/3^{-1}$day satisfy 3 days of the recovery of the infected human. $\mu_v = 0.04^{-1}$ day corresponds to the mean life expectancy of 25 days for vector. $\theta, \gamma_h, \gamma_v$ are arbitrarily constants. We choose $\theta, \gamma_h, \gamma_v$ equal 0.55, 0.22, 0.16, respectively.

Case 1; $\tau=0$



1a)



1b)

Fig 1. 1a) Time series of S_h, I_h, D_h and I_v when there is no time delay and L_0 = 3.5.

1b) Stable spiral trajectories and the parameters are similar to fig.1a).

The period of oscillation is approximated 3 years. We observe that the trajectories in the $I_h - S_h$, $D_h - S_h$, $I_v - S_h$, $D_h - I_h$ and $I_v - I_h$ phase planes spiral into the endemic equilibrium state. There is not clearly evident for the trajectory $I_v - D_h$ phase plane, but this phase plane also spirals in.

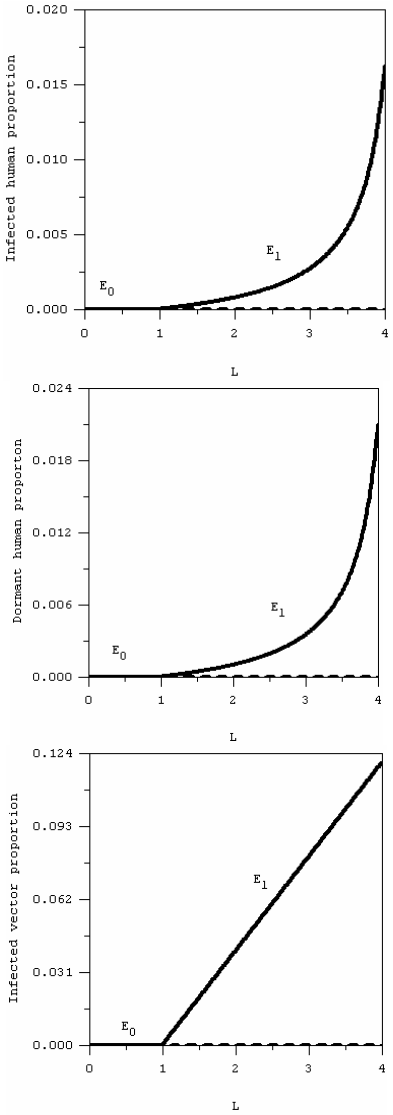
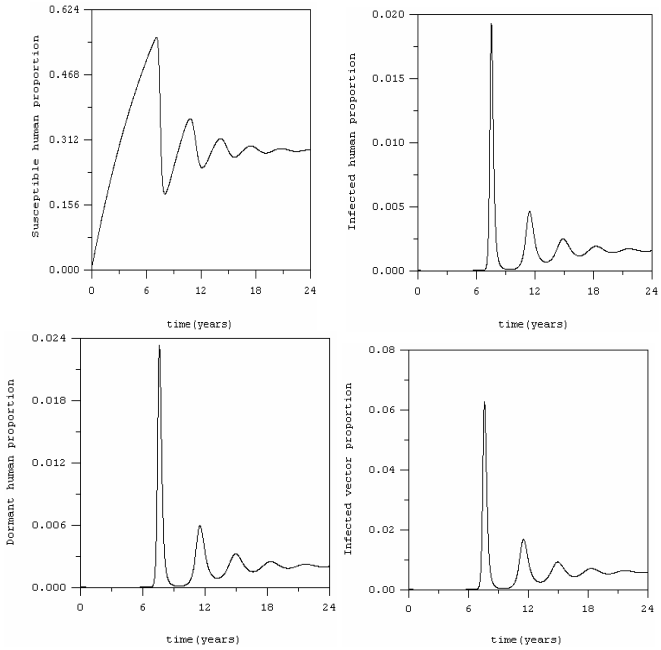


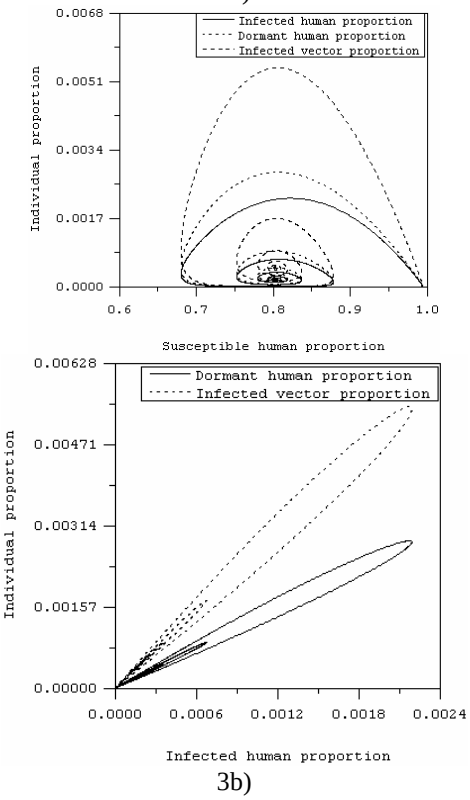
Fig.2. Bifurcation diagrams of equations (4a)-(4d), demonstrate the equilibrium solutions of infected, dormant human and infected vector populations (for $\tau = 0$), respectively. — represents the stable solutions and --- represents the unstable solutions. For $L < 1, E_0$ will be stable. For $L > 1$, E_1 will be stable.

case 2; $\tau \neq 0$

In this case, τ must be in the interval $0 < \tau < (\ln L_0)/\mu_v$. According to our parameters, τ must belong to this interval: (0, 31.5). We choose τ = 15.



3a)



3b)

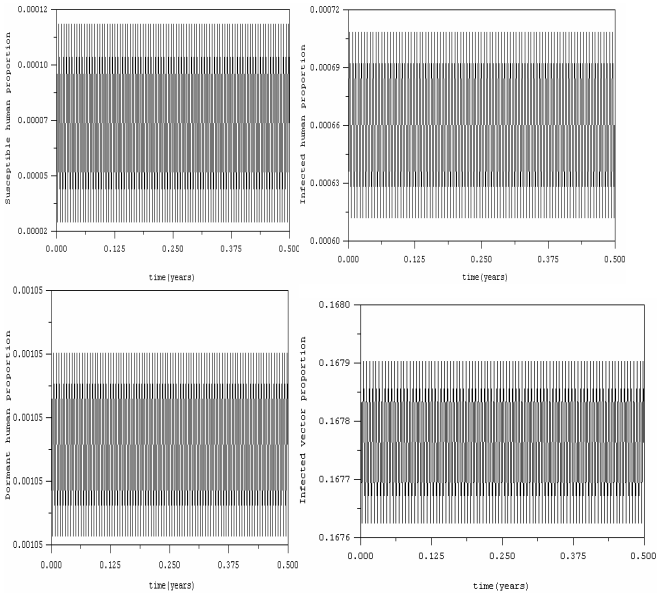
Fig.3. 3a) Time series of I_h, D_h and I_v when $\tau = 15$.
3b) Stable spiral trajectories and the parameters are similar to fig.3a).

The period of oscillation is 5.5 years. As we see, the trajectories in the two phase planes spiral into the endemic equilibrium state. We observe that the period of oscillations in this case is higher than when there is no time delay.

The time delay (τ) must be in the range $0 \le \tau < (\ln L_0)/\mu_v$. If τ is not in this interval then the endemic equilibrium point will be negative. This is meaningless.

C.II Unrealistic Parameter Values

To find the parameters such that a Hopf bifurcation is possible, we have chosen a set of parameter values: $\mu_h = 0.0000391^{-1}$ day, $r_1 = 1/14^{-1}$ day, $r_2 = 1/(365*5)^{-1}$day, $r_3 = 1/30^{-1}$day, $r_4 = 1/(365*15)^{-1}$day , $r_5 = 1/3^{-1}$day, $\mu_v = 0.04^{-1}$ day, $\alpha = 0.75$, $\tau = 20$, $\gamma_h = 24, \gamma_v = 24$. The numerical solutions are shown in fig.5.



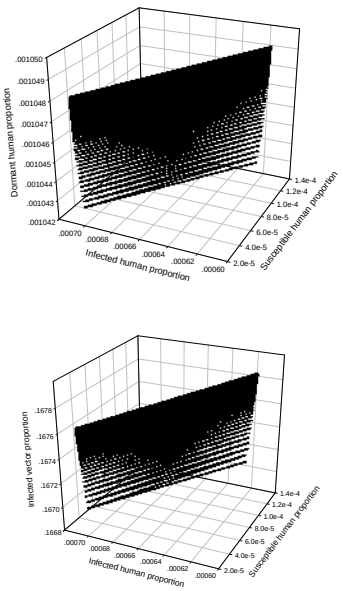
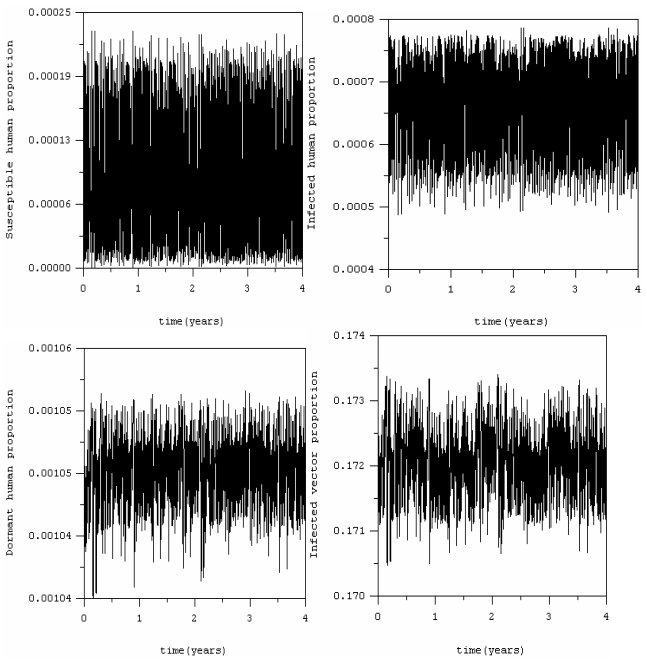


Fig.4. Behaviors of our model in two and three dimensions when limit cycle occurs.

Fig. 5 shows the behaviors of our model when we simulate the another set of parameters: $\mu_h = 0.0000421^{-1}$ day, $r_1 = 1/14^{-1}$ day, $r_2 = 1/(365*5)^{-1}$day, $r_3 = 1/30^{-1}$day, $r_4 = 1/(365*15)^{-1}$day , $r_5 = 1/7^{-1}$day, $\mu_v = 0.04^{-1}$ day, $\theta = 0.75$, $\tau = 20$, $\gamma_h = 27, \gamma_v = 27$.



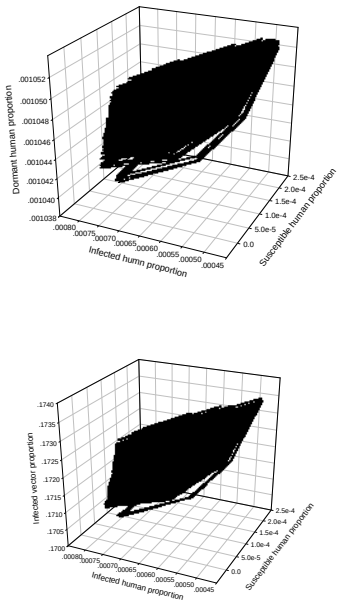


Fig.5. Behavior of our model in two and three dimensions when chaotic behavior occurs.

The parameters values in fig.4 and fig.5 give L = 16,019 and 20,274, respectively. This means than one primary case need to produce 16,019 and 20,274 secondary cases, respectively. These numbers are too much for a primary case can produce. This is impossible in the real life.

IV. CONCLUSION

In this study, the mathematical model of *P.Vivax* is analyzed. The time delay is included to the model. We establish the condition for local stability of endemic equilibrium point. We show the numerical simulations to confirm these results. The conditions for Hopf bifurcation are shown. The numerical simulations show that limit cycle and chaotic behaviors can occur only for unrealistic parameter values. The possibility for the occurrence of Hopf bifurcation can happen only in unreal situations. When we did not include time delay into our model, the basic reproductive number for the endemic equilibrium state will prevail if and only if the basic reproductive number exceeds one (see fig.1). The disease free equilibrium state exists and is local stability if the basic reproductive number is less than one and become unstable when the basic reproductive number is more than one (see fig.2). The numerical simulations are used to confirm results in the previous section. The behavior of solutions can be described in terms of the basic reproductive number; if this number is less than or equal to one, so an infective replace itself with less than one new infective, the disease die out. Moreover, if the basic reproductive number is greater than one, the normalized infectious human, dormant human populations increase. These behaviors occur because there are enough susceptible human to be infected from infectious vector.

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