Transmission Model for *Plasmodium Vivax* Malaria: Conditions for Bifurcation

P. Pongsumpun and I.M. Tang

Abstract—*Plasmodium vivax* malaria differs from *P. falciparum* malaria in that a person suffering from *P. vivax* infection can suffer relapses of the disease. This is due the parasite being able to remain dormant in the liver of the patients where it is able to re-infect the patient after a passage of time. During this stage, the patient is classified as being in the dormant class. The model to describe the transmission of *P. vivax* malaria consists of a human population divided into four classes, the susceptible, the infected, the dormant and the recovered. 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). We analyze our model by using standard dynamic modeling method. Two stable equilibrium states, a disease free state E_0 and an endemic state E_1, are found to be possible. It is found that the E_0 state is stable when a newly defined basic reproduction number G is less than one. If G is greater than one the endemic state E_1 is stable. The conditions for the endemic equilibrium state E_1 to be a stable spiral node are established. For realistic values of the parameters in the model, it is found that solutions in phase space are trajectories spiraling into the endemic state. It is shown that the limit cycle and chaotic behaviors can only be achieved with unrealistic parameter values.

Keywords—Equilibrium states, Hopf bifurcation, limit cycle behavior, local stability, *Plasmodium Vivax*, time delay.

I. INTRODUCTION

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, Plasmodium falciparum, Plasmodium malariae and Plasmodium 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. 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.

We wish to look at the model again. In the present state of concern for medical safety, there is no place for human experimentation to see what would happen if new therapies are adopted. Mathematical modeling allows one to simulate what would occur. 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 The implication of the insights obtained from the simulations is given.

It is estimated that about 50% of the malaria cases outside of Africa and 10% in Africa are due to P. vivax. Part of the urgency for doing research on P. vivax malaria is due to the fact that P. vivax malaria is becoming an emerging public health problem in many parts of the world where the percentage the cases due to are due to P. vivax is increasing.

II. TRANSMISSION MODEL

The mathematical modeling of the epidemiology of malaria (*P. falciparum*) was started by Ross [6] in 1911and improved on by MacDonald [7]. In the Ross model, an individual in the human population is classified as being in a non-infected or infected state. This gives rise to what is known as a SIS (susceptible-infected-susceptible) model. It has been suggested [7] 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.
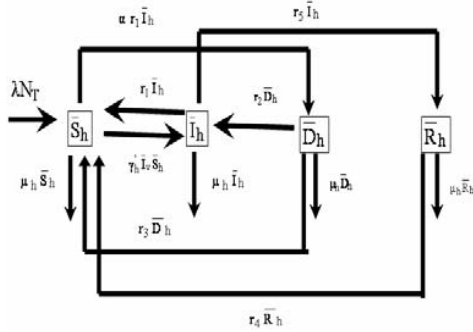

Fig. 1 Flow chart of the model

In our model for the transmission of P. vivax, we divide the host (human) population into susceptible $(\bar{S}_h)$, infected $(\bar{I}_h)$, dormant $(\bar{D}_h)$ and recovered $(\bar{R}_h)$ classes. The last category, the recovered are susceptible to further infections and so they reenter into the $\bar{S}_h$ class. In Figure 1, we show the flow chart describing what is occurring in the human population. As we see, λN_T humans are entering into the susceptible class through birth and $(1-\alpha)r_1\bar{I}_h$, $r_3\bar{D}_h$ and $r_6\bar{R}_h(t)$ 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 α being the percentage of infected people in whom some hypnozoites remain dormant in the liver). $(1-\alpha)$ 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;

$$\frac{d}{dt}\bar{S}_h(t) = \lambda N_T + r_3\bar{D}_h(t) + (1-\alpha)r_1\bar{I}_h(t)$$

$$- \gamma'_h\bar{I}_v(t)\bar{S}_h(t) - \mu_h\bar{S}_h(t) + r_4\bar{R}_h(t)$$

(1a)

Applying similar considerations to the other population classes, we obtain

$$\frac{d}{dt}\bar{I}_h(t) = \gamma'_h\bar{I}_v(t)\bar{S}_h(t) - (r_1 + \mu_h)\bar{I}_h(t)$$

$$+ r_2\bar{D}_h(t) - r_5\bar{I}_h(t),$$

(1b)

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

(1c)

and

$$\frac{d}{dt}\bar{R}_h(t) = r_5\bar{I}_h(t) - (r_4 + \mu_h)\bar{R}_h(t)$$

(1d)

where the parameters in the above equations are defined as

λ is the birth rate of human population,

μ_h is the death rate of human population,

N_T is the total number of human population,

α is the percentage of infected human in whom some hypnozoites remain dormant in the liver,

r_1 is the rate at which a person leaves the infected class by recovering or by entering into the dormant class,

r_2 is the rate at which the dormant human relapses back to the infected human,

r_3 is the recovery rate of the dormant human,

r_4 is the rate at which the recovered human relapses back to the susceptible human, and

r_5 is the rate at which the infected human recovers, since *P. vivax* infection is non lethal, the death rates will be the same for all human classes and we will have $N_T = \bar{S}_h + \bar{I}_h + \bar{D}_h + \bar{R}_h$

Equation (1a) also contains the term $\gamma'_h\bar{I}_v(t)\bar{S}_h(t)$. 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 [8]

$$\gamma'_h = b\frac{\beta_h}{N_T + m}$$

(2)

where b 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. $\bar{I}_v$ is the number of infected mosquitoes. The dynamics of the mosquito's populations are given by

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

(3a)

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

(3b)

Here, 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 - τ not at time t. Fraction of the infected mosquito would have died between the time t and t - τ.

At equilibrium, the total number of female mosquitoes will be A/μ_v. A is the rate at which the mosquitoes are recruited and μ_v is the death rate for the mosquitoes. It should be noted 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 [8]

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

where b is the specie-dependent biting rate of the mosquitoes; m is the population of other animals that the mosquitoes can feed on and β_v is the probability the parasite passed to the mosquito by biting human. We also assume $N_V = \bar{S}_V + \bar{I}_V$.

The working equations of the model are obtained by dividing (1a), (1b), (1c) and (1d) by N_T and (3a) and (3b) by A/μ_v. This would give us six equations expressed in terms of the renormalized variables;

$$S_h = \bar{S}_h/N_T, I_h = \bar{I}_h/N_T, R_h = \bar{R}_h/N_T,$$
$$S_v = \bar{S}_v/(A/\mu_v) \text{ and } I_v = \bar{I}_v/(A/\mu_v).$$

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

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

(4a)

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

(4b)

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

(4c)

and

$$\frac{d}{dt}I_v(t) = \gamma_v(1 - I_v(t-\tau)) I(t-\tau)e^{-\mu_v\tau} - \mu_v I_v(t)$$ (4d)

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

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

In (4d), we have replaced $I(t-\tau)$ by $I(t)$ because 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

To find the equilibrium points, we set the RHS's of (4a) to (4d) to zero. Doing this, we get
  i) the disease free equilibrium state $E_O = (1, 0, 0, 0)$
  ii) the endemic equilibrium state

$$E_1 = (S_h^*, I_h^*, D_h^*, I_v^*)$$

where

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

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

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

$$I_v^* = \frac{-1 + e^{-\mu_v\tau}G_0}{\beta e^{\mu_v\tau} + \gamma}$$

(5)

with

$$G_0 = \frac{\gamma_h\gamma_v(\mu_h + r_2 + r_3)}{\mu_v(\mu_h^2 + \mu_h r_{1235} + r_1((1-\alpha)r_2 + r_3) + (r_2 + r_3)r_5)}$$

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

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

$$r_{1235} = r_1 + r_2 + r_3 + r_5$$

(6)

We observe that endemic equilibrium point exists when $e^{-\mu_v\tau}G_0 > 1$ or τ must lie in the range $0 < \tau < (\ln G_0)/\mu_v$.

Let $G = e^{-\mu_v\tau}G_0$ then G is the basic reproduction number. It represents the number of secondary infections resulting from a primary infection. The local stability of each equilibrium point is determined by the sign of all eigenvalues. If all eigenvalues have negative real parts, then that equilibrium point is locally stable. Eigenvalues for each equilibrium point are obtained by setting

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

where J is the gradient matrix evaluated at the equilibrium point.

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

$$A(\lambda, \tau) + B(\lambda, \tau)e^{-\mu_v\tau} = 0$$ (8)

where

$$A(\lambda, \tau) = \lambda^4 + u_3(\tau)\lambda^3 + u_2(\tau)\lambda^2 + u_1(\tau)\lambda + u_0(\tau)$$ (9)

$$B(\lambda, \tau) = v_3(\tau)\lambda^3 + v_2(\tau)\lambda^2 + v_1(\tau)\lambda - v_0(\tau)$$ (10)

and
$u_3, u_2, u_1, u_0, v_3, v_2, v_1, v_0$ are functions of the time delay (τ).

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

$$\lambda^4 + u_3(0)\lambda^3 + u_2(0)\lambda^2 + u_1(0)\lambda + u_0(0).$$

In this case, the coefficients $u_3(0), u_2(0), u_1(0), u_0(0)$ are constants. We let

$$s_3 = u_3(0),$$
$$s_2 = u_2(0),$$
$$s_1 = u_1(0),$$
$$s_0 = u_0(0)$$

By using Routh-Hurwitz criteria [9], each equilibrium point is locally stable if the following conditions are satisfied;

i) $s_3 > 0$, (11)

ii) $s_1 > 0$, (12)

iii) $s_0 > 0$, (13)

iv) $s_3 s_2 s_1 > s_1^2 + s_3^2 s_0$ (14)

We check the above conditions by using MATHEMATICA (Wolfram Research, Champaign, IL), then we found that E_0 is locally stable for $G_0 < 1$ and E_1 is locally stable for $G_0 > 1$.

B. Bifurcation Conditions for the Endemic State.

The characteristic equations obtained by Ruan and Wei [10] and by Klan and Greenhalgh [11] for their models are of form

$$\lambda^3 + a\lambda^2 + b\lambda + c = de^{-\lambda\tau}, \qquad (15)$$

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

$$\lambda^3 + a\lambda^2 + (b + ce^{-\lambda\tau})\lambda + d = fe^{-\lambda\tau} \qquad (16)$$

The constants (a, b, c, d, f) 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 [10] and [11]. Substituting $\lambda = c + di$ (where c and d are real numbers and may be functions of τ) into (8) and separating the real and imaginary parts, we obtain

$$
\begin{aligned}
&c(\tau)^4 - 6c(\tau)^2 d(\tau)^2 + d(\tau)^4 + u_0(\tau) + c(\tau)u_1(\tau) \\
&+ c(\tau)^2 u_2(\tau) - d(\tau)^2 u_2(\tau) + c(\tau)^3 u_3(\tau) \\
&- 3c(\tau)d(\tau)^2 u_3(\tau) + e^{-c(\tau)\tau} \cos(d(\tau)\tau)(c(\tau)v_1(\tau) \\
&- v_0(\tau) + c(\tau)^2 v_2(\tau) - d(\tau)^2 v_2(\tau) + c(\tau)^3 v_3(\tau) \\
&- 3c(\tau)d(\tau)^2 v_3(\tau)) + e^{-c(\tau)\tau} \sin(d(\tau)\tau)(d(\tau)v_1(\tau) \\
&+ 2c(\tau)d(\tau)v_2(\tau) + 3c(\tau)^2 d(\tau)v_3(\tau) - d(\tau)^3 v_3(\tau)) = 0
\end{aligned}
\qquad (17)
$$

and

$$
\begin{aligned}
&4c(\tau)^3 d(\tau) - 4c(\tau)d(\tau)^4 u_1(\tau) + 2c(\tau)d(\tau)u_2(\tau) \\
&+ 3c(\tau)^2 d(\tau)u_3(\tau) - d(\tau)^3 u_3(\tau) \\
&+ e^{-c(\tau)\tau} \cos(d(\tau)\tau)(d(\tau)v_1(\tau) + \\
&2c(\tau)d(\tau)v_2(\tau) + 3c(\tau)^2 d(\tau)v_3(\tau) - d(\tau)^3 v_3(\tau) \\
&+ e^{-c(\tau)\tau} \sin(d(\tau)\tau)(v_0(\tau) - c(\tau)v_1(\tau) - c(\tau)^2 v_2(\tau) \\
&+ d(\tau)^2 v_2(\tau) - c(\tau)^3 v_3(\tau) + 3c(\tau)d(\tau)^2 v_3(\tau) = 0
\end{aligned}
\qquad (18)
$$

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

$$
\begin{aligned}
&\tilde{d}^4 + u_0(\tau_c) - \tilde{d}^2 u_2(\tau_c) \\
&= (v_0(\tau_c) + \tilde{d}^2 v_2(\tau_c))\cos(\tilde{d}\tau_c) \\
&- \tilde{d}(v_1(\tau_c) - \tilde{d}^2 v_3(\tau_c))\sin(\tilde{d}\tau_c)
\end{aligned}
\qquad (19)
$$

$$
\begin{aligned}
&\tilde{d}^3 u_3(\tau_c) \\
&= \tilde{d}(v_1(\tau_c) - \tilde{d}^2 v_3(\tau_c))\cos(\tilde{d}\tau_c) \\
&+ (v_0(\tau_c) + \tilde{d}^2 v_2(\tau_c))\sin(\tilde{d}\tau_c)
\end{aligned}
\qquad (20)
$$

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

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

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

$$
\begin{aligned}
&h_3(\tau_c) = -2u_2(\tau_c) + u_3(\tau_c)^2 - v_3(\tau_c)^2 \\
&h_2(\tau_c) = 2u_0(\tau_c) + u_2(\tau_c)^2 - v_2(\tau_c)^2 + 2v_1(\tau_c)v_3(\tau_c) \\
&h_1(\tau_c) = -(2u_0(\tau_c)u_2(\tau_c) + v_1(\tau_c)^2 + 2v_0(\tau_c)v_2(\tau_c) \\
&h_0(\tau_c) = u_0(\tau_c)^2 - v_0(\tau_c)^2
\end{aligned}
\qquad (22)
$$

We note that $h_3(\tau_c), h_2(\tau_c), h_1(\tau_c), h_0(\tau_c)$ are real. Critical point value τ_c is always determined from the requirement that $c(\tau_c) = 0$. 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.1 Realistic Parameter Values

In this section, the numerical simulations of the endemic equilibrium state are shown in each case. The parameters are determined by real life observations. $\mu_h = 0.0000391^{-1}$ day corresponds to the real life expectancy of 70 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. $\alpha, \gamma_h, \gamma_v$ equal 0.75, 0.2, 0.15, respectively. These values are arbitrarily constants.

Case 1; $\tau = 0$

2a)
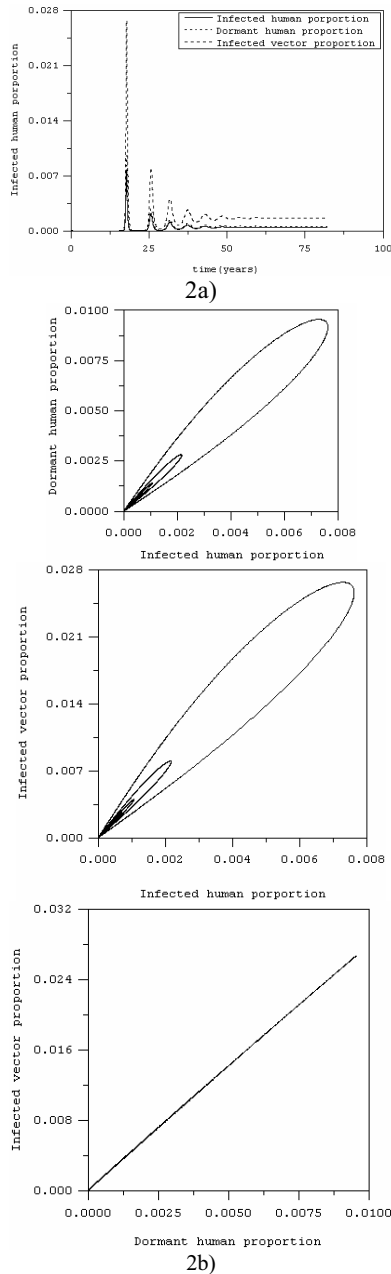
2b)

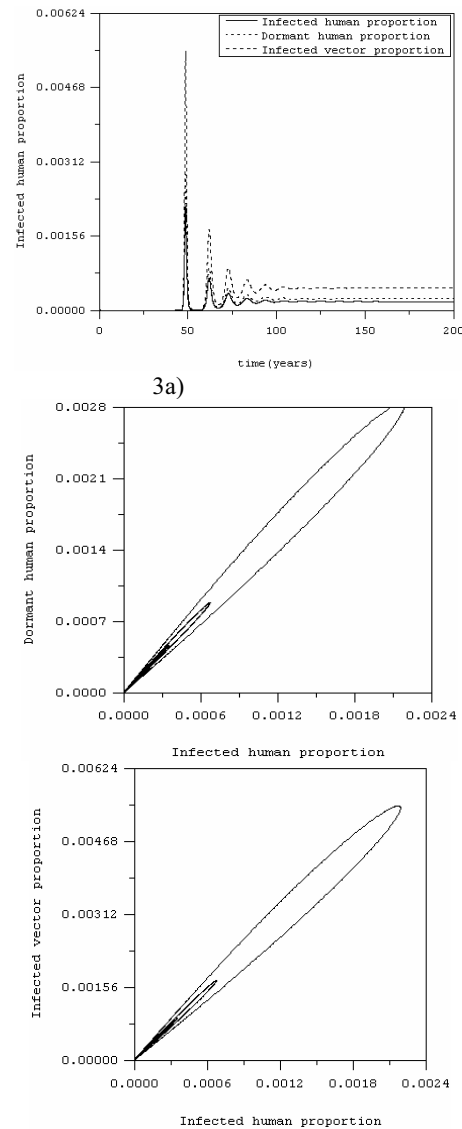Fig. 2   2a) Time series of I_h, D_h and I_v when there is no time delay and $G_0 = 1.85$.

2b) Stable spiral trajectories and the parameters are similar to fig.2a).

The period of oscillation is approximated 5.5 years. As we see, the trajectories in the $D_h - I_h$ 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.

Case 2; $\tau \neq 0$

In this case, τ must be in the range $0 < \tau < (\ln G_0)/\mu_v$. According to our parameters, τ must belong to this interval: (0, 15.5). We choose $\tau = 10$.
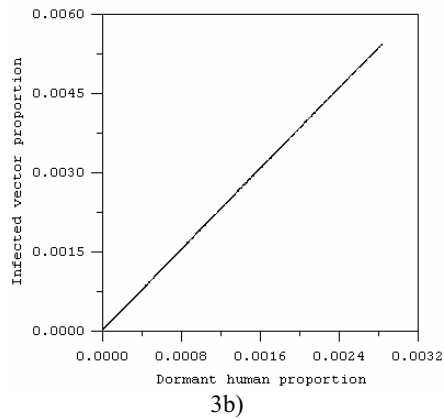

3a)

3b)

Fig. 3   3a) Time series of I_h, D_h and I_v when $\tau = 10$.

   3b) Stable spiral trajectories and the parameters are similar to fig.3a).

The period of oscillation is 10 years. As we see, the trajectories in the $D_h - I_h$ 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. 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 $0 \le \tau < (\ln G_0)/\mu_v$. If τ is not in this interval then the endemic equilibrium point will be negative which is meaningless.

B.2 Unrealistic Parameter Value

To determine whether it is possible that there are parameter values 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, $\alpha = 0.75$, $\tau = 18$, $\gamma_h = 25, \gamma_v = 25$. The numerical solution is shown in fig.4.
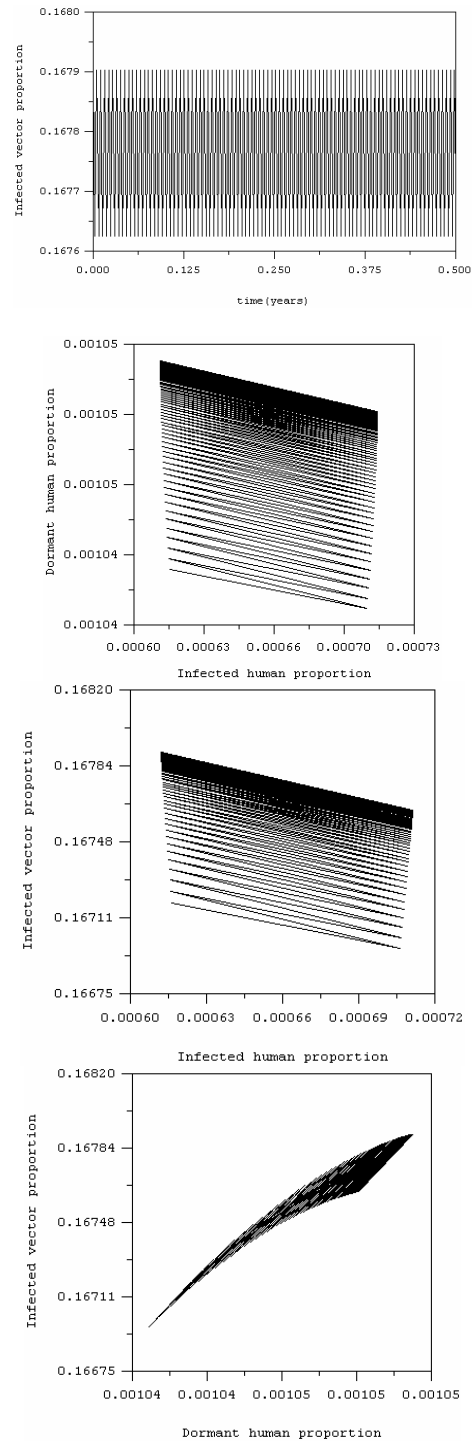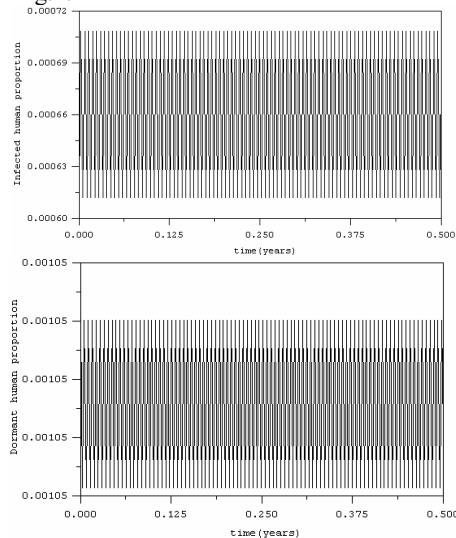

Fig. 4   Behavior of our model when limit cycle occurs.

Next, we simulate the another 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, $\alpha = 0.75$, $\tau = 18$, $\gamma_h = 26, \gamma_v = 26$. The numerical solution is shown in fig.5.
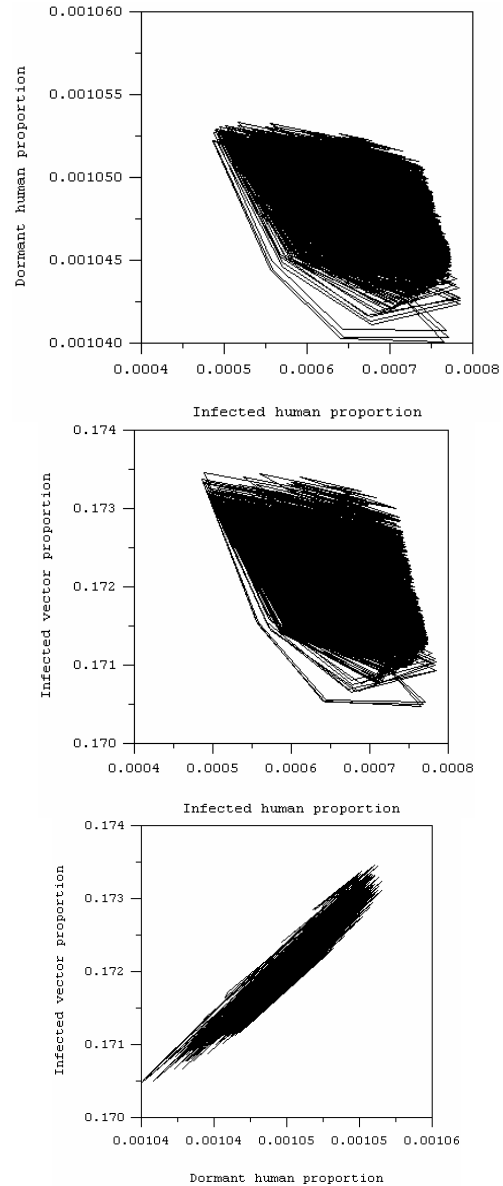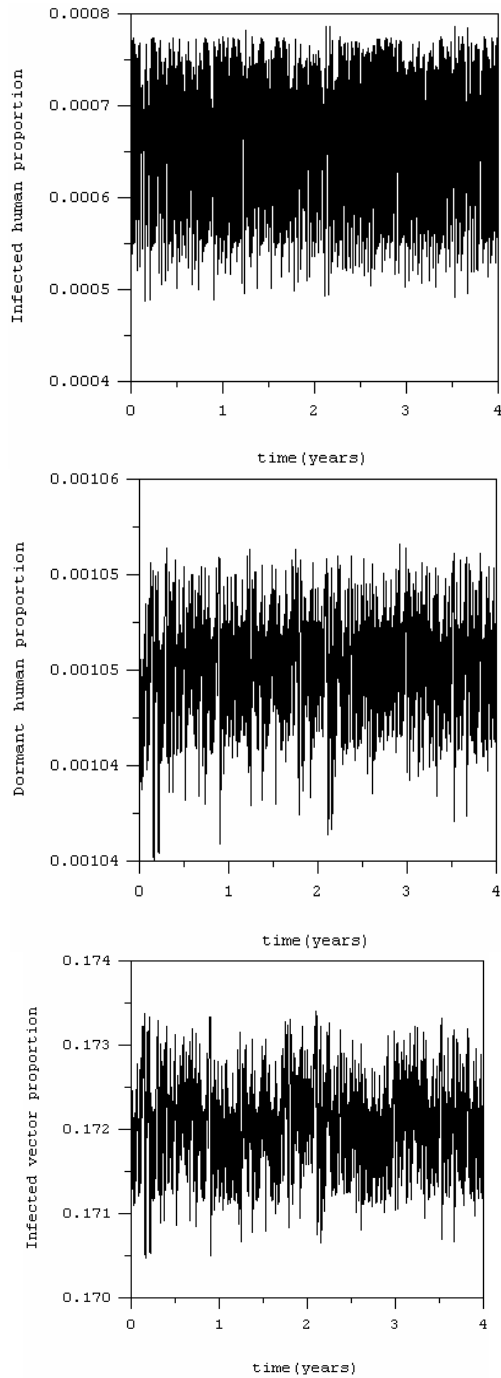

Fig. 5 Behavior of our model when chaotic behavior occurs.

Consider the parameters values in fig.4 and fig.5, the parameter values in fig.4 and fig.5 gives G = 18,829 and 20,365, respectively. This means than one primary case need to produce 18,829 and 20,365 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, we analyze the mathematical model of P.Vivax. The time delay is included to the model. The condition for local stability of endemic equilibrium point is established. The numerical simulations are shown to confirm these results. We show the conditions for Hopf bifurcation.

The numerical simulations show that limit cycle and chaotic behaviors can occur only for unrealistic parameter values.

REFERENCES

[1] PCC. Garnhan., *Malaria parasites of man: life-cycles and morphology (excluding unltrastructure)* IN W.H. Wernsdorfer and I. McGregor (Eds), *Malaria,* Churchill Livingstone, Edinburg, 1988.

[2] WHO, *World Malaria Situation in 1994: Weekly Epidemiological Record,* Geneva, 1997.

[3] B. Sina, "Focus on Plasmodium Vivax," *Trends in Parasitology.*, vol.18, pp.287-289, 2002.

[4] R.M. Anderson, and R.M. May, *Infectious Disease of Humans., Dynamics and Control.*Oxford U. Press, Oxford, 1991.

[5] A. Kammanee, N. Kanyamee, and I.M. Tang , "Basic Reproduction Number for the Transmission of *Plasmodium Vivax* Malaria," *Southeast Asian Journal of Tropical Medicine and Public Health* ., vol. 32, pp.702-706, 2001.

[6] R. Ross, *The preventation of Malaria*, 2th ed. Murray, London, 1911.

[7] G. MacDonald, *The epidemiology and control of malaria.* Oxford University Press, London, 1957.

[8] L. Esteva, and C. Vargus, "Analysis of a dengue disease trasmission model," *Mathematical Bioscience.,* vol. 150, pp.131-151, 1998.

[9] J.E. Marsden, and M. McCracken, *The Hopf Bifurcation and its application.* Springer-Verlag: New York, 1976.

[10] S.Ruan, and J.Wei, "On the zeros of a third degree exponential polynomial with application to a delay model for control of testosterone secretion," *IMA J. Math. Appl. Med. Biol.*, vol. 18, pp. 41-52, 2001.

[11] Q.L.A. Khan, and D. Greenhalgh, "Hopf bifurcation in epidemic models with a time delay in vaccination. IMA," J. Math. Appl. Med. Bio., vol. 16, pp. 113-142, 1998.

[12] J. Tam, "Delay effect in a model for virus replication.," IMA J. Math. Appl. Med. Biol., vol. 16, no. 29, 1999.