Mathematical model of *Plasmodium Vivax* and *Plasmodium Falciparum* Malaria

P.Pongsumpun and I.M.Tang

Abstract**—**Malaria is transmitted to the person by the biting of infectious *Anopheles* mosquitoes. This infectious disease caused by the parasite genus *Plasmodium*. Four species of this parasite cause human malaria, namely, *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium ovale* and *Plasmodium malariae.* The difference between *P.vivax* and *P. falciparum* is 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 cases where it is able to re-infect the case after a passage of time. During this stage, the case is classified as being in the dormant class. The model to describe the transmission between falciparum and viv*ax* malaria consists of a human population divided into four classes, the susceptible, the infectious, the dormant and the recovered classes. The vector population is separated into two classes, the susceptible and infectious classes. 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 basic reproductive number R_0 is less than one. If R_0 is greater than one, the endemic state E_1 is stable. The conditions for the local stability of each equilibrium state are established. The numerical simulations are shown to confirm the results.

Keywords**—**Basic reproductive number, Equilibrium states, local stability, *Plasmodium Falciparum, Plasmodium Vivax,*

I. Introduction

MALARIA (bad air) is recorded in writing from ancient times. References to the fever, the well-known warning sign of malaria, are made in papyri of ancient Egypt [1]. The malaria disease is caused by the parasite of the genus *Plasmodium* Malaria parasite can be found in birds, mammals and lizards. There are four types of malaria parasites: *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium malariae* and *Plasmodium ovale*. There are more than 3 hundred million cases of malaria per year; with between 1 and 1.5 million deaths annually (mostly in children) [2]. Malaria is a major public health problem in Thailand. It has not been eradicated for many reasons. First, Thailand has the physical features of the land that is suitable for mosquitoes' to breed in. Parts of the population are also at a higher risk, for example the migrant worker and people who work in the forest. Finally, malaria is developing resistance to the malaria drugs. Malaria in Thailand is found along the border with Burma, Cambodia, and Malaysia [3,4]. The infection arising from *Plasmodium falciparum*, *Plasmodium vivax* and *Plasmodium malariae* were found to be 50-60%; 40-50% and less than 1%, respectively. *Plasmodium ovale* is not found in Thailand. *Plasmodium vivax* malaria has become a gargantuan problem. In 1994, 45,123 from 109,321 cases of malaria reported in Thailand were due to *Plasmodium vivax* [5]. Malaria is still transmitted in the jungle areas of many provinces, such as Chiang Rai, Lampoon, Petchaboon, Tak, Kanchanaburi, Yala and Chantaburi [5]. The malarial parasite has a complicated double life cycle: a sexual reproductive cycle while it lives in the mosquito and an asexual reproductive cycle while in the human host. While it was in its asexual, free-swimming stage, when it is known as a sporozoite, the malarial parasite is injected into the human bloodstream by a mosquito, passing through the skin along with the latter's saliva. The sporozoite eventually enters a red blood cell of its human host, where it goes through ring-shaped and amoeba-like forms before fissioning (dividing) into smaller forms called merozoites. The red blood cell containing these merozoites then ruptures, releases them into the bloodstream (and also causes the chills and fever that are typical symptoms of the disease). The merozoites can then infect other red blood cells and their cycles of development are repeated. The World Health Organization estimates that there are over one million child deaths per year in sub-Saharan Africa and there are 300-500 million cases of malaria per year. More than two billion people or total 41% of the world's population throughout the world (e.g., part of Africa, Asia, the Middle East, Central and South America, Hispania and Oceania) live in areas where malaria is transmitted regularly and there are approximately 1.5-2.7 million people who die from malaria each year [6].

The progression of *Plasmodium vivax* malaria differs from *Plasmodium falciparum* in that a patient can breathe his last breath if he has *Plasmodium falciparum* malaria but will not pass away from *Plasmodium vivax* infection. A person who suffers from *Plasmodium falciparum* will recover from his bad health (if he does not die). An ill patient with a *Plasmodium vivax* infection will not die but will suffer relapses. As a consequence, the mathematical model of *Plasmodium vivax* malaria is different from that of *Plasmodium falciparum* [7]. The transmission of malaria is usually described by the Ross-MacDonald (RM) model [7]. Nevertheless, this model is only suitable for the transmission

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of the P. falciparum malaria because it does not contain the possibility of relapses of the illness.

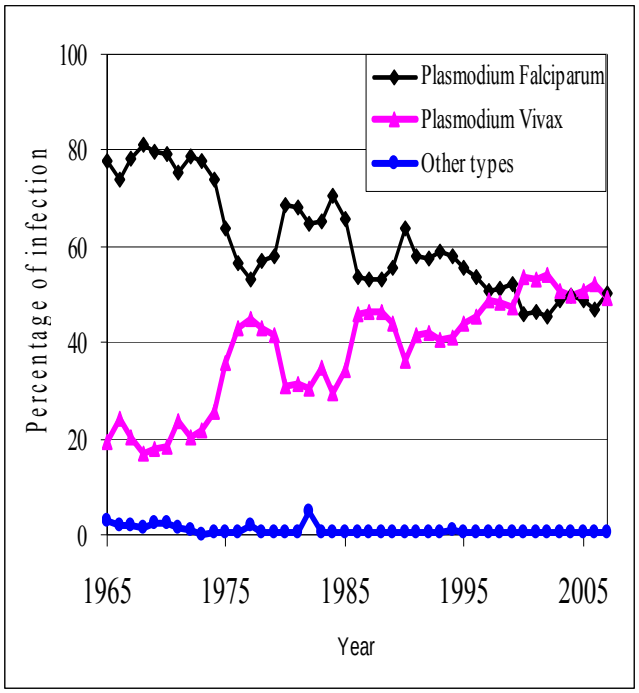


Fig 1. Situation of Malaria in Thailand classified by species of Plasmodium.

The data of Malaria in Thailand during 1965 to 2007 are shown in figure 1. We will see that the most cases of Malaria are due to *P.falciparum* and *P.vivax*. The authors (PP & IMT) have presented the transmission model of P.vivax malaria but we did not consider the effect of P.falciparum malaria [8].

The model for the transmission of Malaria is considered again. In this study, we consider the transmission of two species: *P.falciparum* and *P.vivax*. We introduce in Section 2, the modification of the model which would make it applicable to the transmission of *P.falciparum* and *P.vivax* malaria. In Section 3, we analyze our model to find the conditions for the local stability of each equilibrium state. The numerical simulations confirm the local stability of each equilibrium state.

II. TRANSMISSION MODEL

To study the transmission between two species: *P.falciparum* and *P.vivax*. The population is separated into human and vector classes. Human population is subdivided into susceptible, infectious, dormant and recovered subclasses. The vector population is subdivided into susceptible and infectious subclasses. The variables are defined as follows:

$S_h(t)$ is the number of susceptible human,

$I_h(t)$ is the number of infectious human,

$D_h(t)$ is the number of dormant human,

$R_h(t)$ is the number of recovered human,

$S_v(t)$ is the number of susceptible vector,

$I_v(t)$ is the number of infectious vector.

An infectious human can recover and re-enter the susceptible class. Only the recovered humans who were infected with P. vivax are susceptible to further infections. However, an infected mosquito cannot recover. In Figure 2, we show a flow chart describing what is occurring in the human population and vector populations. λN_T is the number of Thais entering the susceptible class through birth and $r_{5_F} I_h(t)$ and $r_{5_v} I_h(t)$ as, respectively, the numbers of infected human who were infected with *P. falciparum* or *P. vivax* malaria but have recovered. The rate at which susceptible human are lost by becoming infected with *P. falciparum* is $\gamma'_{h_F h} I'_v(t) S'_h(t)$ and by becoming infected with *P. vivax* is $\gamma'_{h_v h} I'_v(t) S'_h(t)$. A susceptible human will be infected by the *P. falciparum* (*P. vivax*) parasite if bitten by a mosquito carrying the particular parasite. To take this into account, the infection rates, $\gamma'_{h_F h}$ and $\gamma'_{h_v h}$ should be proportional to the fraction of the infected mosquitoes with the particular type of parasite. Additional increases in the number of people infected with *P. vivax* malaria occur when the members of the dormant class relapse.

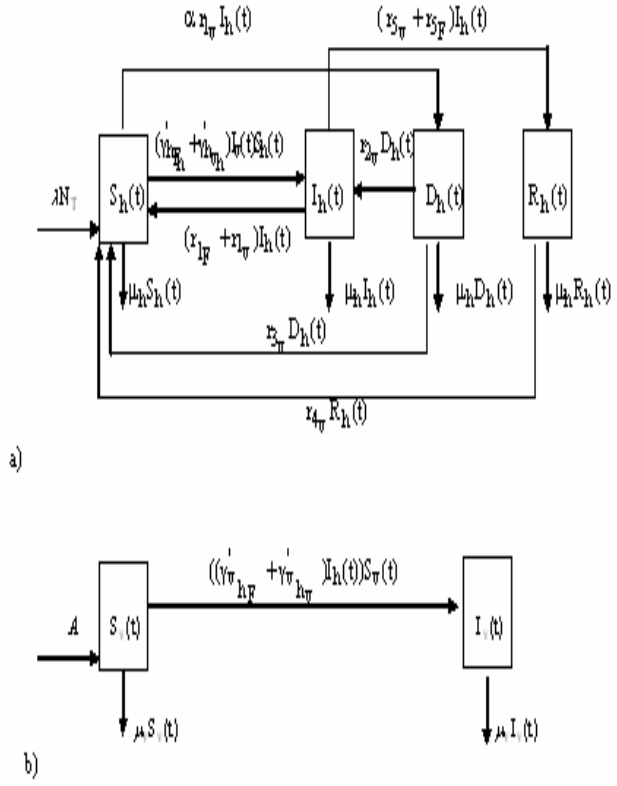


Fig.2 Flow chart of the model.
2a) For the human population
2b) For the vector population.

The 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 rate of change of the susceptible human population:

$$\frac{d}{dt}S_h(t)=\lambda N_h-\alpha r_{l_v}I_h(t)-\mu_h S_h(t)-(\gamma'_{h_{F_h}}+\gamma'_{h_{v_h}})I_v(t)S_h(t) \\ +(r_{l_F}+r_{l_v})I_h(t)+r_{3_v}D_h(t)+r_{4_v}R_h(t) \quad (1)$$

For the other population classes, we obtain

$$\frac{d}{dt}I_h(t)=(\gamma'_{h_{F_h}}+\gamma'_{h_{v_h}})I_v(t)S_h(t)-(r_{l_F}+r_{l_v})I_h(t) \\ -\mu_h I_h(t)-(r_{5_F}+r_{5_v})I_h(t)+r_{2_v}D_h(t) \quad (2)$$

$$\frac{d}{dt}D_h(t)=\alpha\, r_{l_v}I_h(t)-(r_{2_v}+r_{3_v}+\mu_h)D_h(t) \quad (3)$$

$$\frac{d}{dt}R_h(t)=(r_{5_F}+r_{5_v})I_h(t)-(r_{4_v}+\mu_h)R_h(t) \quad (4)$$

$$\frac{d}{dt}S_v(t)=A-\mu_v S_v(t)-(\gamma'_{v_{h_F}}+\gamma'_{v_{h_v}})I_h(t)S_v(t) \quad (5)$$

$$\frac{d}{dt}I_v(t)=(\gamma'_{v_{h_F}}+\gamma'_{v_{h_v}})I_h(t)S_v(t)-\mu_v I_v(t) \quad (6)$$

with the conditions

$$N_h=S_h+I_h+D_h+R_h \text{ and } N_v=S_v+I_v \quad (7)$$

where

μ_h is the death rate of human population,

$\gamma'_{h_{F_h}}$ and $\gamma'_{h_{v_h}}$ are the rates at which the *P.falciparum*(F) and *P.vivax* (V) parasites are transmitted from the mosquito to the human

$\gamma'_{v_{h_F}}$ and $\gamma'_{v_{h_v}}$ are the rates at which the *P.falciparum*(F) and *P.vivax* (V) parasites are transmitted from the human to the mosquito

λ is the birth rate of human population,

N_h is the total number of human population,

N_v is the total number of vector population,

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

r_{l_F} is the rate at which a person who infected with P.falciparum leaves the infected class,

r_{l_v} is the rate at which a person who infected with P.Vivax leaves the infected class,

r_{2_v} is the rate at which the dormant human relapses back to the infected human due to P.Vivax,

r_{3_v} is the recovery rate of the dormant human due to P.Vivax,

r_{4_v} is the rate at which the recovered human due to P.Vivax relapses back to the susceptible human, and

r_{5_F} is the rate at which the infected human due to P.Falciparum recovers,

r_{5_v} is the rate at which the infected human due to P.Vivax recovers,

The total human and vector populations are constant, thus the rate of change for both populations equal to zero. Then

$$\frac{d}{dt}N_h=0 \quad \text{and} \quad \frac{d}{dt}N_v=0 \quad (8)$$

From (8), we obtain $\lambda=\mu_h$ for human population and

$N_V=\frac{A}{\mu_v}$ for vector population.

We normalize equations (1)-(6) by letting

$$s_h=\frac{S_h}{N_h},\ i_h=\frac{I_h}{N_h},\ d_h=\frac{D_h}{N_h},$$

$$r_h=\frac{R_h}{N_h},\ s_v=\frac{S_v}{(A/\mu_v)},\ i_v=\frac{I_v}{(A/\mu_v)}$$

then the reduced equations become

$$\frac{d}{dt}s_h(t)=\mu_h(1-s_h(t))-\alpha r_{l_v}i_h(t)-(\gamma_{h_{F_h}}+\gamma_{h_{v_h}})i_v(t)s_h(t) \\ +(r_{l_F}+r_{l_v})i_h(t)+r_{3_v}d_h(t)+r_{4_v}(1-(s_h(t)+i_h(t)+d_h(t)) \quad (9)$$

$$\frac{d}{dt}i_h(t)=(\gamma_{h_{F_h}}+\gamma_{h_{v_h}})i_v(t)s_h(t)-(r_{l_F}+r_{l_v})i_h(t)-\mu_h i_h(t) \\ -(r_{5_F}+r_{5_v})i_h(t)+r_{2_v}d_h(t) \quad (10)$$

$$\frac{d}{dt}d_h(t)=\alpha\, r_{l_v}i_h(t)-(r_{2_v}+r_{3_v}+\mu_h)d_h(t) \quad (11)$$

$$\frac{d}{dt}i_v(t)=(\gamma_{v_{h_F}}+\gamma_{v_{h_v}})i_h(t)(1-iv(t))-\mu_v i_v(t) \quad (12)$$

with the conditions

$$s_h+i_h+d_h+r_h=1 \quad \text{and} \quad s_v+i_v=1$$

and

$$\gamma_{h_{F_h}}=\gamma'_{h_{F_h}}\left(\frac{A}{\mu_v}\right),\gamma_{h_{v_h}}=\gamma'_{h_{v_h}}\left(\frac{A}{\mu_v}\right),$$

$$\gamma_{v_{h_F}}=\gamma'_{v_{h_F}}N_h,\gamma_{v_{h_v}}=\gamma'_{v_{h_v}}N_h \quad (13)$$

III. ANALYSIS OF THE MODEL

A. Analytical Results

Finding equilibrium states by setting the right hand side of all equations (9)-(12) equal to zero, and then we obtain two equilibrium states:

i) Disease free equilibrium state: $E_o = (1,0,0,0)$ (14)

ii) Endemic equilibrium state: $E_1 = (s_h^*, i_h^*, d_h^*, i_v^*)$ (15)

where

$$s_h^* = \frac{\mu_h + d_h^* r_{3v} + i_h^*(r_{1F} + r_{1v}(1-\alpha) - r_{4v}) + r_{4v}(1-d_h^*)}{(\gamma_{hF_h} + \gamma_{hv_h})i_v^* + \mu_h + r_{4v}}, \quad (16)$$

$$i_h^* = \frac{d_h^* r_{2v} + (\gamma_{hF_h} + \gamma_{hv_h})i_v^* s_h^*}{\mu_h + r_{1F} + r_{1v} + r_{5F} + r_{5v}}, \quad (17)$$

$$d_h^* = \frac{\alpha i_h^* r_{1v}}{\mu_h + r_{2v} + r_{3v}}, \quad (18)$$

and

$$i_v^* = \frac{\mu_v(R_0 - 1)(\mu_h + r_{4v})(\mu_h(\mu_h + r_{123}) + r_{1F}(r_{2v} + r_{3v}) + r_{1v}(r_{2v}(1-\alpha) + r_{3v}) + (r_{2v} + r_{3v})(r_{5F} + r_{5v}))}{(\gamma_{hF_h} + \gamma_{hv_h})((\gamma_{vhF} + \gamma_{vhv})(\mu_h + r_{2v} + r_{3v})(\mu_h + r_{4v}) + \mu_v(\mu_h(\alpha_{12345} + \mu_h) + \alpha r_{1v} r_{4v} + (r_{2v} + r_{3v})(r_{4v} + r_{5F} + r_{5v})))} \quad (19)$$

with

$$r_{123} = r_{1F} + r_{1v} + r_{2v} + r_{3v} + r_{5F} + r_{5v},$$

$$\alpha_{12345} = \alpha r_{1v} + r_{2v} + r_{3v} + r_{4v} + r_{5F} + r_{5v}$$

and

$$R_0 = \frac{(\gamma_{hF_h} + \gamma_{hv_h})(\gamma_{vhF} + \gamma_{vhv})(\mu_h + r_{2v} + r_{3v})}{(\mu_v(\mu_h(\mu_h + r_{123}) + (r_{2v} + r_{3v})(r_{1F} + r_{5F} + r_{5v}) + r_{1v}(r_{2v}(1-\alpha) + r_{3v}))} \quad (20)$$

The local stability for each equilibrium state can be determined by the sign of all eigenvalues. If all eigenvalues have negative real part, then that equilibrium state is local stability. The eigenvalues for each equilibrium state are calculated by setting

$$\det(J - \lambda I) = 0 \quad (21)$$

where J is the Jacobian matrix of the right hand side of equations (9)-(12) evaluated at the equilibrium state.
For the disease free equilibrium state E_0, the characteristic equation is

$$(\lambda + \mu_h + r_{4v})(\lambda^3 + t_2\lambda^2 + t_1\lambda + t_0) = 0 \quad (22)$$

where

$$t_2 = 2\mu_h + \mu_v + r_{123},$$

$$t_1 = (\gamma_{hF_h} + \gamma_{hv_h})(\gamma_{vhF} + \gamma_{vhv}) + \mu_h^2 + \mu_v r_{123} + \mu_h(2\mu_v + r_{123}) + r_{1F}r_{2v} + r_{1v}r_{2v}(1-\alpha) + r_{2v}(r_{5F} + r_{5v}) + r_{3v}(r_{1F} + r_{1v} + r_{5F} + r_{5v}),$$

$$t_0 = \mu_v(1 - R_0)(\mu_h^2 + \mu_h r_{123} + r_{1v}r_{2v}(1-\alpha) + r_{1v}r_{3v} + (r_{2v} + r_{3v})(r_{1F} + r_{5F} + r_{5v})) \quad (23)$$

There are four eigenvalues corresponding to (22). We represent these four eigenvalues by $\lambda_1, \lambda_2, \lambda_3, \lambda_4$.

$\lambda_1 = -\mu_h - r_{4v}$ has negative real part. The other three eigenvalues can be obtained by solving

$$\lambda^3 + t_2\lambda^2 + t_1\lambda + t_0 \quad (24)$$

These three eigenvalues have negative real part if they satisfy the Routh-Hurwitz criteria [9, 10]:

$$t_2 > 0 \quad (25)$$

$$t_0 > 0 \quad (26)$$

$$t_2 t_1 - t_0 > 0 \quad (27)$$

It can be easily seen that coefficients t_2, t_1 and t_0 satisfy (25) and (26) for $R_0 < 1$. Calculating

$$t_2 t_1 - t_0 = (2\mu_h + \mu_v + r_{123})((\gamma_{hF_h} + \gamma_{hv_h})(\gamma_{vhF} + \gamma_{vhv}) + \mu_h(\mu_h + 2\mu_v) + (\mu_h + \mu_v)r_{123} + (r_{2v} + r_{3v})(r_{1F} + r_{5F} + r_{5v}) + r_{1v}(r_{2v}(1-\alpha) + r_{3v})) + \mu_v(1 - R_0)(\mu_h^2 + \mu_h r_{123} + (1-\alpha)r_{1v}r_{2v} + r_{1v}r_{3v} + (r_{2v} + r_{3v})(r_{1F} + r_{5F} + r_{5v})) \quad (28)$$

$t_2 t_1 - t_0$ is positive for $R_0 < 1$. Therefore the disease free equilibrium state is local stability for $R_0 < 1$.

For the endemic equilibrium state E_1, the characteristic equation is

$$\lambda^4 + w_3\lambda^3 + w_2\lambda^2 + w_1\lambda + w_0 = 0 \quad (29)$$

where

$$w_3 = (\gamma_{vhF} + \gamma_{vhv})i_h^* + (\gamma_{hF_h} + \gamma_{hv_h})i_v^* + 3\mu_h + \mu_v + r_{123} + 4r_{4v},$$

$$w_2 = 3\mu_h(\mu_h + \mu_v) + (2\mu_h + \mu_v + r_{1F} + (1-\alpha)r_{1v})r_{2v} + (r_{1F} + r_{1v})r_{3v} + (r_{1F} + r_{1v} + r_{2v} + r_{3v})r_{4v} + (\gamma_{vhF} + \gamma_{vhv})i_h^*(3\mu_h + r_{123} + r_{4v}) + (r_{2v} + r_{3v} + r_{4v})r_{5F} + (2\mu_h + \mu_v)(r_{1F} + r_{1v} + r_{3v} + r_{4v} + r_{5F}) + (2\mu_h + \mu_v + r_{2v} + r_{3v} + r_{4v})r_{5v} + (\gamma_{hF_h} + \gamma_{hv_h})(i_v^*(\alpha_{12345} + (\gamma_{vhF} + \gamma_{vhv})i_h^* + 2\mu_h + \mu_v) + (\gamma_{vhF} + \gamma_{vhv})s_v^* s_h^*,$$

$$w_1 = \mu_h(\mu_h^2 + 2\mu_v(r_{1F} + r_{1v}) + \mu_h(3\mu_v + r_{1F} + r_{1v}) + \gamma_{vhv}i_h^*(3\mu_h + 2(r_{1F} + r_{1v}))) + \mu_h(2\gamma_{vhv}i_h^* + \mu_h + 2\mu_v) + (\gamma_{vhv}i_h^* + \mu_h + \mu_v)r_{1F})r_{2v} + (1-\alpha)((\gamma_{vhF} + \gamma_{vhv})i_h^* + \mu_h + \mu_v)r_{1v}r_{2v} + \gamma_{vhF}i_h^*(3\mu_h^2 + r_{1F}r_{2v} + 2\mu_h(r_{1F} + r_{1v} + r_{2v})) + (\mu_h^2 + \mu_v(r_{1F} + r_{1v}) + \mu_h(2\mu_v + r_{1F} + r_{1v}))r_{3v} + (\mu_h^2 + (r_{1F} + r_{1v}(1-\alpha))r_{2v} + r_{1F} + r_{1v})r_{3v} + \mu_v(r_{1F} + r_{1v} + r_{2v} + r_{3v}) + \mu_h(2\mu_v + r_{1F} + r_{1v} + r_{2v} + r_{3v}))r_{4v}$$

$$w_0 = ((\gamma_{v_{h_F}} + \gamma_{v_{h_v}})i_h^* + \mu_v)(\mu_h + r_{4_v})(\mu_h^2 + r_{1_v}r_{2_v}(1-\alpha) + r_{1_v}r_{3_v} + (r_{2_v} + r_{3_v})(r_{1_F} + r_{5_F} + r_{5_v}) + \mu_h(r_{1_F} + r_{1_v} + r_{2_v} + r_{3_v} + r_{5_F} + r_{5_v})) + (\gamma_{h_{F_h}} + \gamma_{h_{v_h}})(i_v^*((\gamma_{v_{h_F}} + \gamma_{v_{h_v}})i_h^* + \mu_v)(\mu_h^2 + \alpha r_{1_v}r_{4_v} + (r_{2_v} + r_{3_v})(r_{4_v} + r_{5_F} + r_{5_v}) + \mu_h(\alpha r_{1_v} + r_{2_v} + r_{3_v} + r_{4_v} + r_{5_F} + r_{5_v})) + (\gamma_{v_{h_F}} + \gamma_{v_{h_v}})s_v^*(\mu_h + r_{2_v} + r_{3_v})(\mu_h + r_{4_v})s_h^*) \quad (30)$$

These four eigenvalues have negative real part if they satisfy the Routh-Hurwitz criteria [9-12] :

$$w_3 > 0 \quad (31)$$

$$w_1 > 0 \quad (32)$$

$$w_0 > 0 \quad (33)$$

$$w_1 w_2 w_3 > w_1^2 + w_3^2 w_0 \quad (34)$$

From our evaluations, we have found that coefficients w_3, w_2 w_1 and w_0 satisfy (31), (32), (33) and (34) when $R_0 > 1$.

Thus, the endemic equilibrium state is local stability for $R_0 > 1$.

B. Numerical Results

In this section, we present the results of our numerical simulations. The values of the parameters are taken from real life observations. We have set $\mu_h = 0.0000421$ per day which corresponds to the real life expectancy of 65 years for human, and $\mu_v = 1/30$, which corresponds to the life expectancy of 30 days for the Anopheline mosquito. The values r_{1_F} =1/20 per day, r_{1_v} =1/14 per day correspond to the time it takes people who are infected with *P. falciparum* and *P. vivax* to leave the infected class and become susceptible again, i.e., 20 days for *P. falciparum* and 14 days for *P. vivax*. The values r_{2_v} =1/365 per day, r_{3_v} =1/(2*365) per day correspond to the time it takes people who are infected with *P. vivax* to leave the dormant class, i.e., 1 year to enter the infected class and 2 years to enter the susceptible class. The value r_{4_v} =1/(3*365) per day corresponds to 3 years for the people who are infected with *P. vivax* to relapse. The values r_{5_F} =1/30 per day, r_{5_v} =1/25 per day correspond to the time it takes people who are infected with *P. falciparum* and *P. vivax* to recover, i.e., 30 days for *P. falciparum* and 25 days for *P. vivax*. $\alpha = 0.65$ To have the disease free equilibrium state as the local stable equilibrium state, we let $\gamma'_{h_{F_h}}, \gamma'_{h_{v_h}}, \gamma'_{v_{h_F}}, \gamma'_{v_{h_v}}$ equal to 0.025, 0.024, 0.03 and 0.02, respectively. To have the stable equilibrium state as the endemic equilibrium state, we set $\gamma'_{h_{F_h}}, \gamma'_{h_{v_h}}, \gamma'_{v_{h_F}}, \gamma'_{v_{h_v}}$ are equal to 0.14, 0.1, 0.15 and 0.1, respectively.

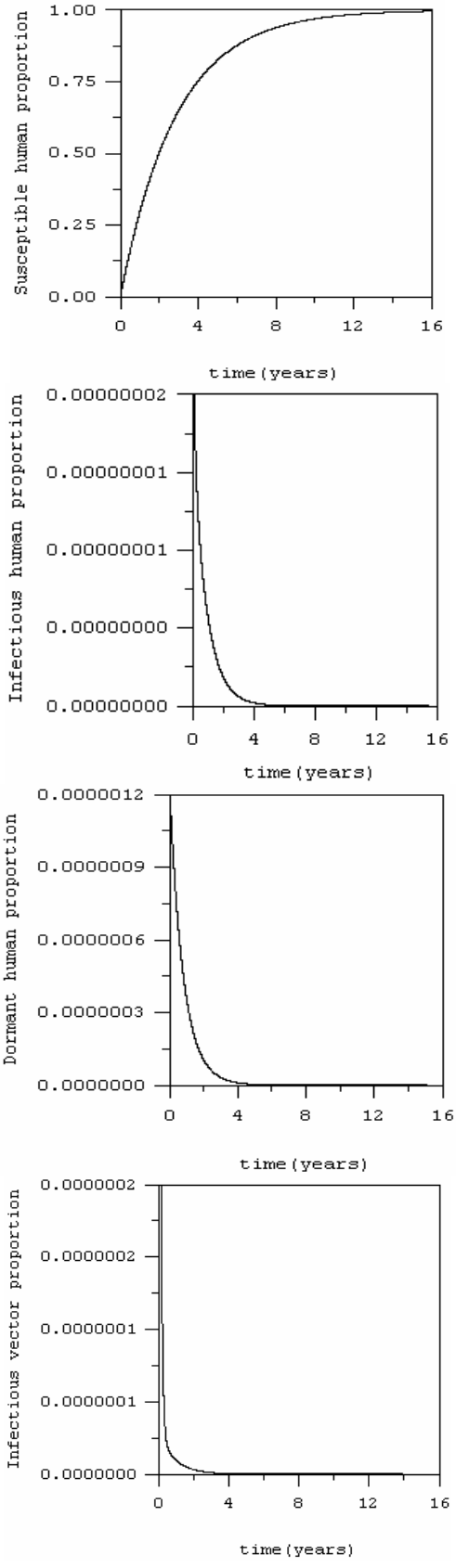
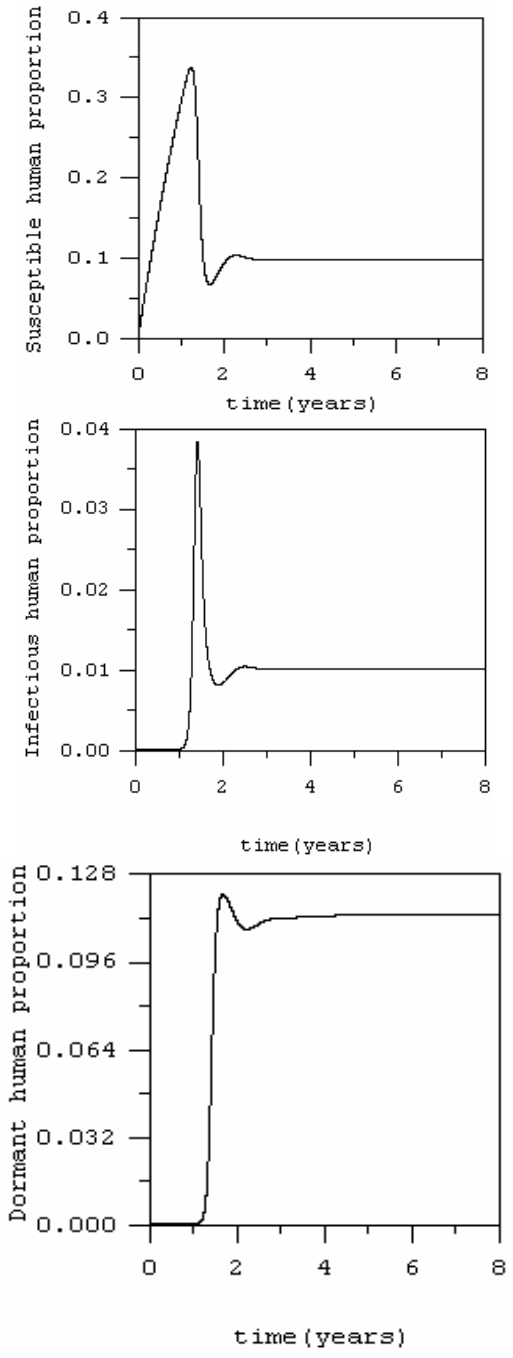


Fig. 3. Time series of susceptible human, infectious human, dormant human and infectious vector proportions. The values of parameters are in the text and R_0 =0.5



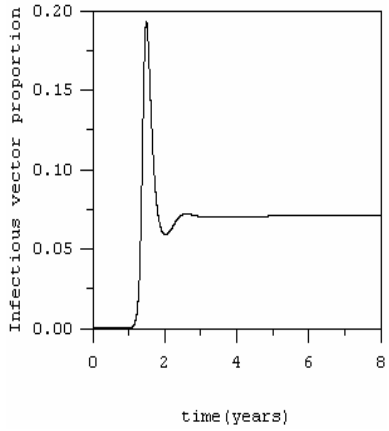


Fig.4. Time series of susceptible human, infectious human, dormant human and infectious vector proportions. The values of parameters are in the text and R_0 =10.96.

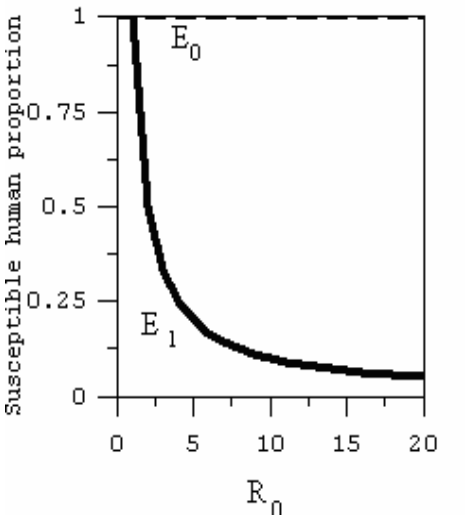
Fig.3 and fig.4 show time development of human and vector classes. Fig.3 shows numerical solutions for $R_0 < 1$. Fig.4 shows numerical solutions for $R_0 > 1$. The solutions converge to the disease free equilibrium state as shown in fig. 3. Fig. 4, the solutions oscillate to the endemic equilibrium state (0.0981393,0.0101401,0.113396,0.0706756).

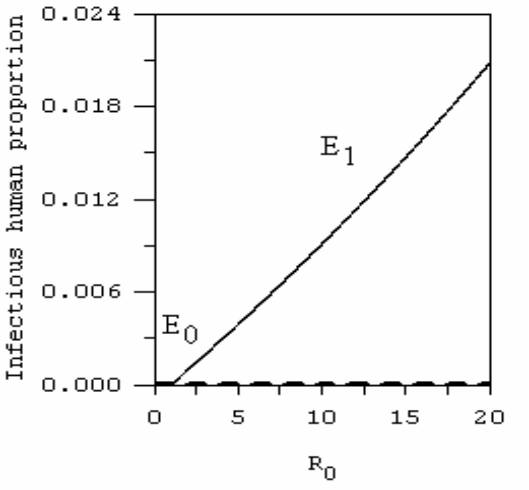
IV. DISCUSSION AND CONCLUSION

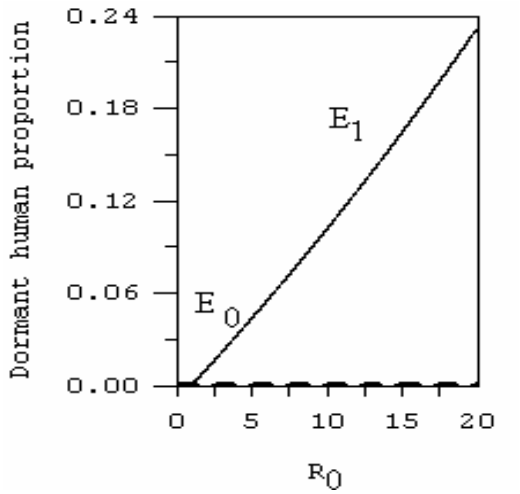
We formulate the transmission model of Malaria by considering the effect of two species: *Plasmodium Falciparum* and *Plasmodium Vivax*. The basic reproductive number is $R' = \sqrt{R_0}$ where

$$R_0 = \frac{(\gamma_{hFh} + \gamma_{hvh})(\gamma_{vhF} + \gamma_{vhv})(\mu_h + r_{2v} + r_{3v})}{(\mu_v(\mu_h(\mu_h + r_{123}) + (r_{2v} + r_{3v})(r_{lF} + r_{5F} + r_{5v}) + r_{lv}(r_{2v}(1-\alpha) + r_{3v}))} \quad (35)$$

R' represents the number of secondary cases that one case can produce if introduced into a susceptible person. R_0 is the threshold condition. The threshold condition and the stability of the solutions are shown in fig.5.







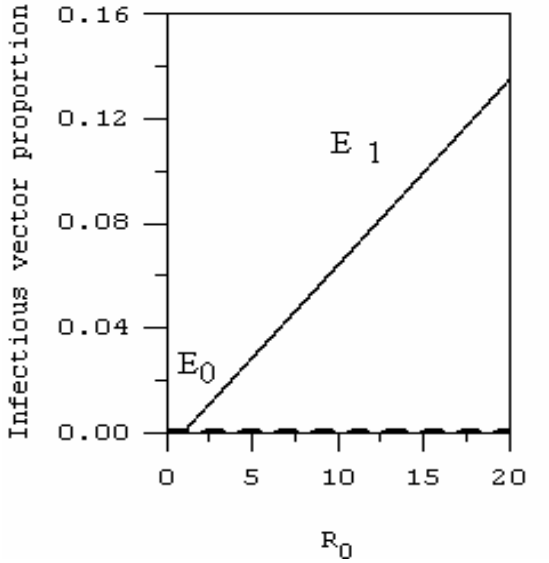


Fig.5. Bifurcation diagrams of equations (9)-(12), demonstrate the equilibrium solutions of susceptible, infectious, dormant human and infectious vector populations, respectively. — represents the stable solutions and --- represents the unstable solutions. For $R_0 < 1, E_0$ will be stable. For $R_0 > 1$, E_1 will be stable.

The basic reproductive number for the endemic equilibrium state will prevail if and only if the basic reproductive number exceeds one. 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. 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, therefore an infective replace itself with less than one new infective, the disease die out. Furthermore, the susceptible fraction approaches one since everyone is susceptible when the disease has vanished. If the basic reproductive number is greater than one, the normalized susceptible human decreases. The normalized infectious human, dormant human populations increase. These subsequent behaviors occur because there are enough susceptible human to be infected from infectious vector.

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