The Transmission Model of *P.falciparum* and *P.Vivax* Malaria between Thai and Burmese

P.Pongsumpun., and I.M.Tang

Abstract**—** The transmission of *Plasmodium falciparum* and *Plasmodium vivax* malaria of Thais and Burmese is studied through a mathematical model. The population is separated into two groups, Thai and Burmese. Each population is divided into susceptible and infectious subclasses. The loss of immunity by individuals in the infectious class causes them to move back into the susceptible class. Standard dynamical method is used to analyze the behavior of the model. Two stable equilibrium states, a disease free state and an epidemic state are found to be possible in each population. A disease free equilibrium state in the Thai population occurs when there are no infected Burmese entering into the community. When there are infected Burmese enters into the Thai community, the epidemic state can occur. It is found that the disease free state is stable when the threshold number R_0 is less than one. The epidemic state is stable when R_{E_T} and R_{E_B} (where these threshold numbers are for the individual populations) are greater than one. The numerical simulations of our model illustrate what the results would be for our theoretical model.

Keywords**—**Transmission models, *Plasmodium Vivax* malaria, equilibrium states, local stability, basic reproduction number.

I. INTRODUCTION

MALARIA is ranked among the top six of the world's serious diseases in the world by the World Health Organization (WHO).There are more than 3 hundred million cases of malaria each year; with between 1 and 1.5 million death annually (mostly in children). Malaria is a mosquito-borne disease caused by the protozoan parasites of the genus *Plasmodium* (phylum Apicomplexa) a parasite. In humans, malaria is caused by four species, *P. falciparum*, *P. vivax, P. malariae* and *P. ovale*. The two most common causes are the first two. *P. falciparum* causes 90% of the malaria in Africa and is the cause of over 2-3 million (mostly children) people in the world (mainly Africa) [1,2]. *P. vivax* is the cause of 50% of the malaria outside of Africa. 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.

Due to the differences in the economic conditions between Thailand and Myanmar, temporary migration of Burmese into Thailand occurs every year. The heaviest economic migrations occur at the beginning of the rainy season (May-June) and a lesser amount at the end of the year at harvest time. More than 60% of the Burmese in some groups (in Mae Sot and Bo Basi, (two provinces in Thailand along the border)) are infected with mefloquine-resistant malaria [3]. These economic migrations from neighboring countries into Thailand have caused problems to the malaria control program in Thailand [4]. Especially troubling is the problem of multi-drug resistance malaria whose presence is now seen in the high transmission areas around the market centers along the migratory routes. The first cases of malaria resistance were found along Thai-Kampuchean border, another border where the economic conditions on the two sides of the border are quite different. It is believed that the areas where the parasites having the highest drug resistance are along this latter border. From the medical data on Malaria cases in Thailand between 2003 and 2006 [5], most of the malaria infections in Thailand was due to *P. falciparum* and *P. vivax* (See Figure 1). The nationality of the malaria patients during these years are indicated on Figure 2.

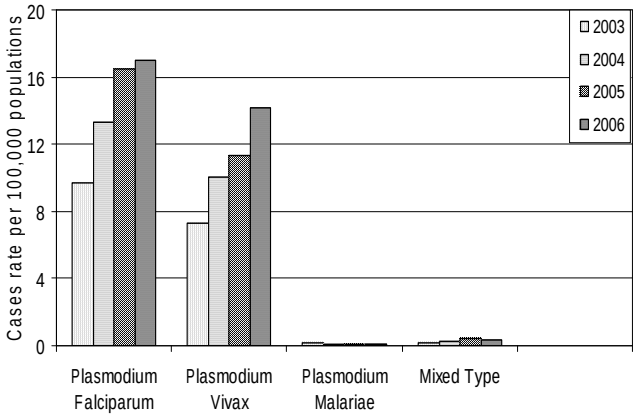

Fig.1 Situation of Malaria in Thailand classified by type of Malaria.

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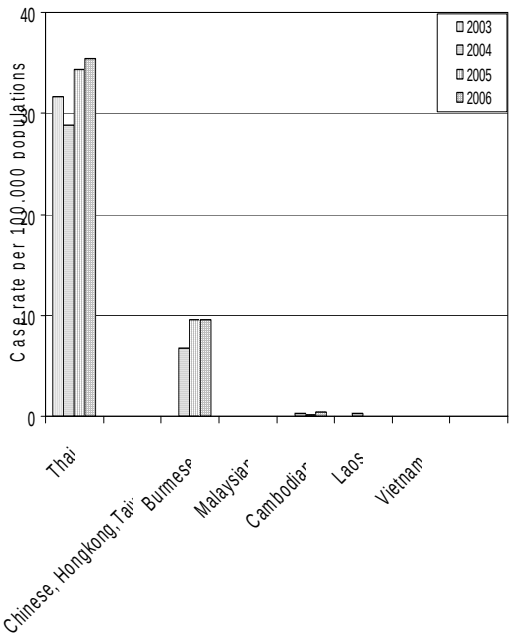


Fig.2 Situation of Malaria in Thailand classified by nationality of patients.

To better understand the problems facing the public health officials in Thailand, a new mathematical model must be introduced to understand the situations when two different forms of malaria are in co circulation in a population [6]. The transmission of malaria is usually described by the Ross-MacDonald (RM) model [7]. One of the present authors (IMT) has introduced a simple mathematical model [8] to describe the transmission of *P. vivax* malaria. In that model, we did not consider the effect of the Malaria transmission between Thai and Burmese.

In this study, we formulate a model in which different mathematical models are used to describe separately the transmission of *P. falciparum* and of *P. vivax.* In the present state of concern for medical safety, there is no place for human experimentation to see what would happen if new therapies were adopted. Mathematical modeling allows one to simulate what would occur. Since we are interested in applying the model to the situation along the Thai-Myanmar border (and to a lesser extent the Thai-Kampuchean border), we include rates which depend on whether the infecting malaria is *P. falciparum* (denoted by 'F') or *P. vivax* (denoted by 'v') or the person is a Thai (denoted by T) or a Burmese (denoted by B). We introduce in Section 2, the modification of the model which would make it applicable to the transmission of *P. falciparum* and *P. vivax* between Thai and Burmese. In Section 3, we analyze our model to find the conditions for the local stability of each equilibrium point. The numerical simulations are shown to confirm the local stability of the endemic equilibrium point.

II. Mathematical Model

The mathematical modeling of the epidemiology of malaria (P. falciparum) was started by Ross [9] in 1911 and improved on by MacDonald [10]. 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 [10] that the human population should instead 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 divided into susceptible, infected but not infectious and infectious.

In our model, we consider the transmission cycle between human in the two populations and in the vector populations. Both human populations (Thai and Burmese) and the vector populations are separated into susceptible and infectious subclasses. We let

$S_T^{'}(t)$ is the number of susceptible Thai human,

$S_B^{'}(t)$ is the number of susceptible Burmese human,

$I_T^{'}(t)$ is the number of infectious Thai human,

$I_B^{'}(t)$ is the number of infectious Burmese 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 reenter into susceptible class. However, an infected mosquito can not recover. In Figure 3, we show the flow chart describing what is occurring in the human population and vector populations. It is easy to interpret λNT as the number of Thais entering into the susceptible class through birth and $r_F I_T^{'}(t)$ and $r_v I_T^{'}(t)$ as the numbers of infected Thais who were infected with P. falciparum malaria or with P. vivax malaria, respectively, who have recovered. The rate at which susceptible Thais are lost by becoming infected with P. falciparum is $\gamma_{h_{FT}}^{'} I_v^{'}(t) S_T^{'}(t)$ and by becoming infected with P. vivax is $\gamma_{h_{vT}}^{'} I_v^{'}(t) S_T^{'}(t)$. A susceptible Thais will only be infected by the P. falciparum (P. vivax) parasite if it is bitten by a mosquito carrying the particular parasite. To take this into account, the infection rates, $\gamma_{h_{FT}}^{'}$ and $\gamma_{hv_T}^{'}$, should be proportional to the fraction of the infected mosquitoes with the particular type of parasite.

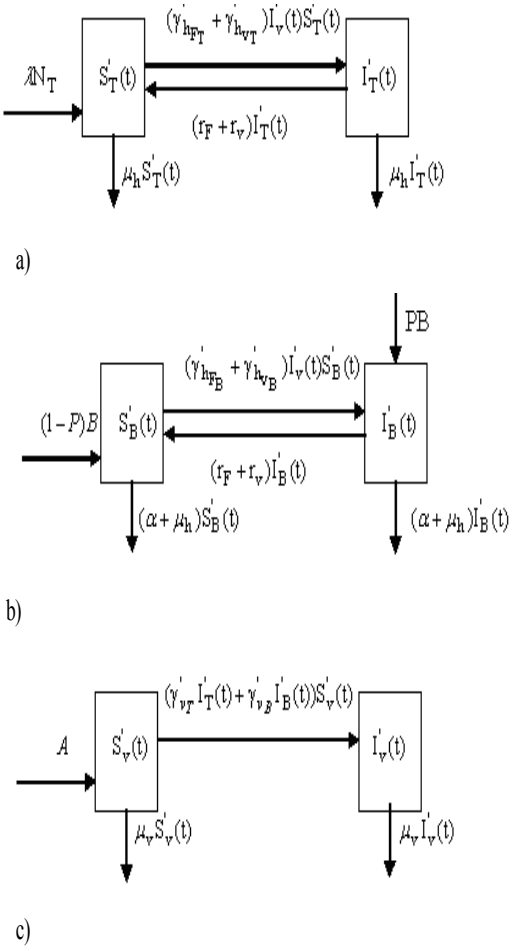


Fig.3 Flow chart of the model.
3a) For the Thai human population
3b) For the Burmese human population
3c) For the vector population.

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 Thai human;

$$\frac{d}{dt}S'_T(t) = \lambda N_T - \mu_h S'_T - (\gamma'_{h_{F_T}} + \gamma'_{h_{v_T}})I'_v(t)S'_T(t) + (r_F + r_v)I'_T(t) \quad (1)$$

Applying similar considerations to the other population classes, we obtain

$$\frac{d}{dt}I'_T(t) = (\gamma'_{h_{F_T}} + \gamma'_{h_{v_T}})I'_v(t)S'_T(t) - (r_F + r_v)I'_T(t) - \mu_h I'_T(t) \quad (2)$$

$$\frac{d}{dt}S'_B(t) = B - PB - (\gamma'_{h_{F_B}} + \gamma'_{h_{v_B}})I'_v(t)S'_B(t) - (r_F + r_v)I'_B(t) - (\mu_h + \alpha)S'_B(t) \quad (3)$$

$$\frac{d}{dt}I'_B(t) = PB - (r_F + r_v)I'_B(t) + (\gamma'_{h_{F_B}} + \gamma'_{h_{v_B}})I'_v(t)S'_B(t) - (\mu_h + \alpha)I'_B(t) \quad (4)$$

where the others parameters in the above equations are defined as
μ_h is the death rate of human population,
α is the rate that Burmese moves out the country,
P is the percentage of Burmese who are infectious when they are entering the community,
B is the constant recruitment rate of Burmese.
We assume that P. falciparum and P. vivax infections are non lethality, so the death rates will be the same for all human classes and we will have $N_T = S'_T + I'_T$ and $N_B = S'_B + I'_B$
The dynamics of the mosquito populations are given by

$$\frac{d}{dt}S'_v(t) = A - \mu_v S'_v(t) - (\gamma'_{v_T} I'_T(t) + \gamma'_{v_B} I'_B(t))S'_v(t) \quad (5)$$

$$\frac{d}{dt}I'_v(t) = (\gamma'_{v_T} I'_T(t) + \gamma'_{v_B} I'_B(t))S'_v(t) - \mu_v I'_v(t) \quad (6)$$

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. $\gamma'_{v_T}, \gamma'_{v_B}$ are the rate at which the mosquitoes become infected with the parasites once the mosquito has bitten an infected human (Thai and Burmese). We also assume $N_V = S'_v + I'_v$. The working equations of the model are obtained by dividing eqns. (1) and (2) by N_T, eqns. (3) and (4) by N_B and eqns. (5) and (6) by $A/\mu v$. This would give us six equations expressed in terms of the renormalized variables;

$$S_T = S'_T/N_T,\ I_T = I'_T/N_T,\ S_B = S'_B/N_B,\ I_B = I'_B/N_B$$
$$S_v = S'_v/N_v,\ I_v = I'_v/N_v$$

where

$$N_B = \frac{B}{\mu_h + \alpha},\quad N_v = \frac{A}{\mu_v}.$$

The conditions
$S_T + I_T = 1,$
$= 1\ S_T + I_T = 1,\ S_B + I_B = 1$
and $S_v + I_v = 1$, leads to only three of them being independent. We pick the three equations to be

$$\frac{d}{dt}I_T(t) = (\gamma_{h_{F_T}} + \gamma_{h_{v_T}})I_v(1 - I_T) - (r_F + r_v + \mu_h)I_T(t) \quad (7)$$

$$\frac{d}{dt}I_B(t) = P(\mu_h + \alpha) - (r_F + r_v)I_B + (\gamma_{h_{F_B}} + \gamma_{h_{v_B}})I_v(t)(1 - I_B(t)) - (\mu_h + \alpha)I_B(t) \quad (8)$$

and

$$\frac{d}{dt}I_v(t) = (\gamma_{v_T} I_T(t) + \gamma_{v_B} I_B(t))(1 - I_v(t)) - \mu_v I_v(t) \quad (9)$$

where the new transmission rates are

$$\gamma_{h_{F_T}} = \gamma'_{h_{F_T}}(A/\mu_v),\ \gamma_{h_{V_T}} = \gamma'_{h_{V_T}}(A/\mu_v),$$
$$\gamma_{h_{F_B}} = \gamma'_{h_{F_B}}(A/\mu_v), \gamma_{h_{V_B}} = \gamma'_{h_{V_B}}(A/\mu_v),$$
$$\gamma_{v_T} = \gamma'_{v_T} N_T, \gamma_{v_B} = \gamma'_{v_B} \frac{B}{\mu_h + \alpha} \tag{10}$$

The domain of solutions is

$$\Omega = \{(I_T, I_B, I_V) \mid 0 \le S_T + I_T \le 1,\ 0 \le S_B + I_B \le 1, 0 \le S_v + I_v \le 1\} \tag{11}$$

III. ANALYSIS OF THE MATHEMATICAL MODEL

A. Analytical Results

To find the equilibrium points, we set the RHS's of (7) to (9.) to zero. Doing this, we get equilibrium state (I_T^*, I_B^*, I_v^*) where

$$I_T^* = \frac{(\gamma_{h_{F_T}} + \gamma_{h_{v_T}})I_v^*}{(\gamma_{h_{F_T}} + \gamma_{h_{v_T}})I_v^* + (r_F + r_v + \mu_h)}, \tag{12}$$

$$I_B^* = \frac{P(\mu_h + \alpha) + (\gamma_{h_{F_B}} + \gamma_{h_{v_B}})I_v^*}{(\gamma_{h_{F_B}} + \gamma_{h_{v_B}})I_v^* + (\mu_h + \alpha + r_F + r_v)} \tag{13}$$

and I_v^* being the solutions of

$$I_v^{*3}(t) + a_1 I_v^{*2}(t) + a_2 I_v^*(t) + a_3 = 0 \tag{14}$$

where

$$a_1 = \left(\frac{1}{(\gamma_{hF_B} + \gamma_{hv_B})(\gamma_{hF_T} + \gamma_{hv_T})(\gamma_{vB} + \gamma_{v_T} + \mu_v)}\right)$$
$$[((\gamma_{hF_T} + \gamma_{hv_T})(\mu_h\mu_v + \gamma_{vB}\mu_h P + \alpha(\gamma_{v_T} + \mu_v + \gamma_{vB}P)$$
$$+ \mu_v(r_F + r_v) + \gamma_{v_T}(\mu_h + r_F + r_v))$$
$$-(\gamma_{hF_B} + \gamma_{hv_B})((\gamma_{hF_T} + \gamma_{hv_T})(\gamma_{vB} + \gamma_{v_T})$$
$$-(\gamma_{vB} + \mu_v)(\mu_h + r_F + r_v)))] \tag{15}$$

$$a_2 = \left(\frac{1}{(\gamma_{hF_B} + \gamma_{hv_B})(\gamma_{hF_T} + \gamma_{hv_T})(\gamma_{vB} + \gamma_{v_T} + \mu_v)}\right)$$
$$[-((\gamma_{hF_B} + \gamma_{hv_B})\gamma_{vB}\mu_h + \gamma_{v_T}\mu_h(\gamma_{hF_T} + \gamma_{hv_T})$$
$$-\mu_h^2\mu_v + \gamma_{vB}\mu_h P(\gamma_{hF_T} + \gamma_{hv_T}) - \gamma_{vB}\mu_h^2 P$$
$$+\gamma_{vB}r_F(\gamma_{hF_B} + \gamma_{hv_B}) + \gamma_{v_T}r_F(\gamma_{hF_T} + \gamma_{hv_T})$$
$$-2\mu_h\mu_v r_F - \gamma_{vB}\mu_h P r_F - \mu_v r_F^2 + ((\gamma_{hF_T} + \gamma_{hv_T})\gamma_{v_T}$$
$$+\gamma_{vB}((\gamma_{hF_B} + \gamma_{hv_B} - \mu_h P)$$
$$-2\mu_v(\mu_h + r_F))r_v - \mu_v r_v^2 + \alpha((\gamma_{hF_T} + \gamma_{hv_T})(\gamma_{v_T} + \gamma_{vB}P)$$
$$-(\mu_v + \gamma_{vB}P) - (\mu_v + \gamma_{vB}P)(\mu_h + r_F + r_v))) \tag{16}$$

and

$$a_3 = -\frac{\gamma_{v_B}(\alpha + \mu_h)P(\mu_h + r_F + r_v)}{(\gamma_{hF_B} + \gamma_{hv_B})(\gamma_{hF_T} + \gamma_{hv_T})(\gamma_{vB} + \gamma_{v_T} + \mu_v)} \tag{17}$$

The solution to eqn. (14) will be physically meaningless if the solutions are negative since the normalized infectious mosquito population must be non-negative real number. So we need to find all possible conditions for I_v^* to be real and positive. P is the percentage of Burmese who are infectious when they are entering the community, so this parameter is in the range [0, 1]. We consider two cases: P = 0 and $0 < P \le 1$.

For P = 0, a_3 is zero and one of the solutions of eqn. (14) is $I_v^* = 0$. The other solutions are the solutions of a quadratic equation. The numerical values of these two solutions will depend on the numerical values of the parameters in the model. These are often unknown. Using standard dynamical analysis (based on the Hopf Bifurcation Theory [11,12,13]), we can establish the conditions for the stability of the disease free state. We find the condition is

$$R_0 < 1 \quad \text{where} \quad R_0 = R_T + R_B \tag{18}$$

with

$$R_T = \frac{(\gamma_{h_{F_T}} + \gamma_{h_{v_T}})\gamma_{v_T}}{\mu_v(\mu_h + r_F + r_v)} \quad \text{and}$$
$$R_B = \frac{(\gamma_{h_{F_B}} + \gamma_{h_{v_B}})\gamma_{v_B}}{\mu_v(\alpha + \mu_h + r_F + r_v)}. \tag{19}$$

Determining whether the numerical values of the parameters satisfy eqn. (18), is not of direct concern to us in this paper. The important thing to remember is that the disease free state is one of the equilibrium state. This means that in the absence of any infectious Burmese entering into Thailand, malaria will not become epidemic in Thailand.

For $0 < P \le 1$, the equilibrium state will not be the disease free state since $a_3 \ne 0$ unless for some unknown reasons, the values of the parameters are such that $a_3 = 0$. For this case, the equilibrium state will be the epidemic state $E_1 = (I_T^*, I_B^*, I_v^*)$. It remains to be determined if this stable is stable. Performing an analysis similar to the one used

to establish the conditions disease free state to be stable, we find that the epidemic state will be stable if

$$R_{E_T} > 1, R_{E_B} > 1 \text{ and } R_{E_v} > 1. \tag{20}$$

where

$$R_{E_T} = \frac{I_v^* \mu_v + \gamma_{v_T}(I_T^* + I_v^*)}{\gamma_{v_T}}, \tag{21}$$

$$R_{E_B} = \frac{I_v^*(\gamma_{v_T} I_T^* + \mu_v) + \gamma_{v_B}(I_B^* + I_v^*)}{\gamma_{v_B}} \tag{22}$$

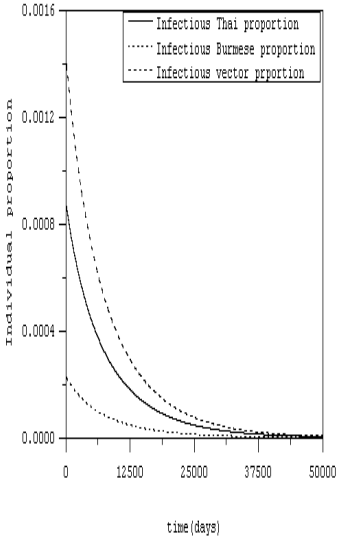
and

$$R_{E_v} = \frac{I_B^* + (\alpha + r_v) I_B^* + (\gamma_{h_{F_B}} + \gamma_{h_{v_B}})(I_B^* + I_v^*)}{(\gamma_{h_{F_B}} + \gamma_{h_{v_B}})} \tag{23}$$

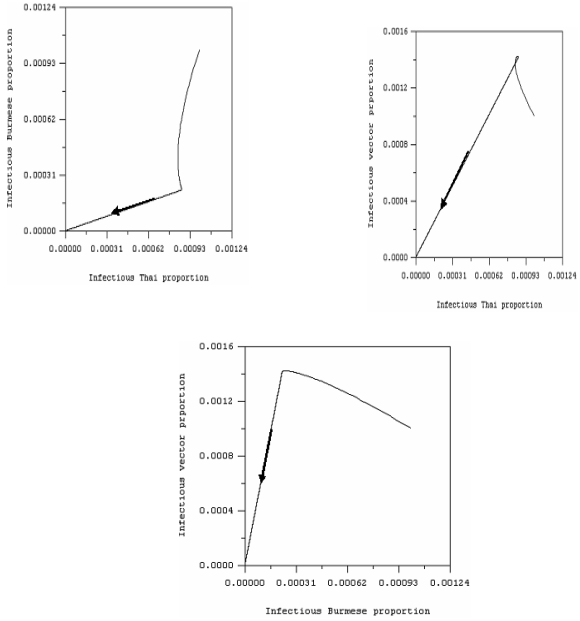
The numerical values of the equilibrium epidemic will again depend on the numerical values of the parameters. The stability analysis of the eigenvalues of dynamical systems will place limits on the values of the parameters which would lead the epidemic state to be stable. Again, what these values are is of no direct concern in this paper. What is known is that the equilibrium state will not be the disease free state but will instead be an epidemic state. Without infectious Burmese entering into the community, there will be no infected population.

B. Numerical Results

In this section, we present the results of our numerical simulations for the case of P = 0 in Figure 4(a). The values of the parameters are taken from real life observations. We have set $\mu_h = 0.0000391$ per day which corresponds to the real life expectancy of 70 years for human and $\mu_v = 1/30$ which corresponds to the life expectancy of 30 days for the Anopheline mosquitoes. The values $r_F = 1/30$ per day, $r_v = 1/25$ per day correspond to the time it takes people who are infected with *P. falciparum* and *P. vivax* to loss their illness, i.e., 30 days for *P. falciparum* and 25 days for *P. vivax*. 1/α is the average time, a Burmese stays in Thailand and we take this to be $\alpha = 0.000183$ per day. $\gamma_{h_{F_T}}, \gamma_{h_{v_T}}, \gamma_{h_{F_B}}, \gamma_{h_{v_B}}, \gamma_{v_T}, \gamma_{v_B}$ are arbitrarily chosen. To have the disease free state as the stable equilibrium state, we set P = 0. To have the stable equilibrium state to be the epidemic state, we set P = 0.6 [3].



a)



(b)

Fig. 4 4a) Time series of I_T, I_B and I_v.

The parameters for the transmission rate are as follows: $\gamma_{h_{F_T}} = 0.04, \gamma_{h_{v_T}} = 0.005,$

$\gamma_{h_{F_B}} = 0.008, \gamma_{h_{v_B}} = 0.004,$

$\gamma_{v_T} = 0.045, \gamma_{v_B} = 0.035.$

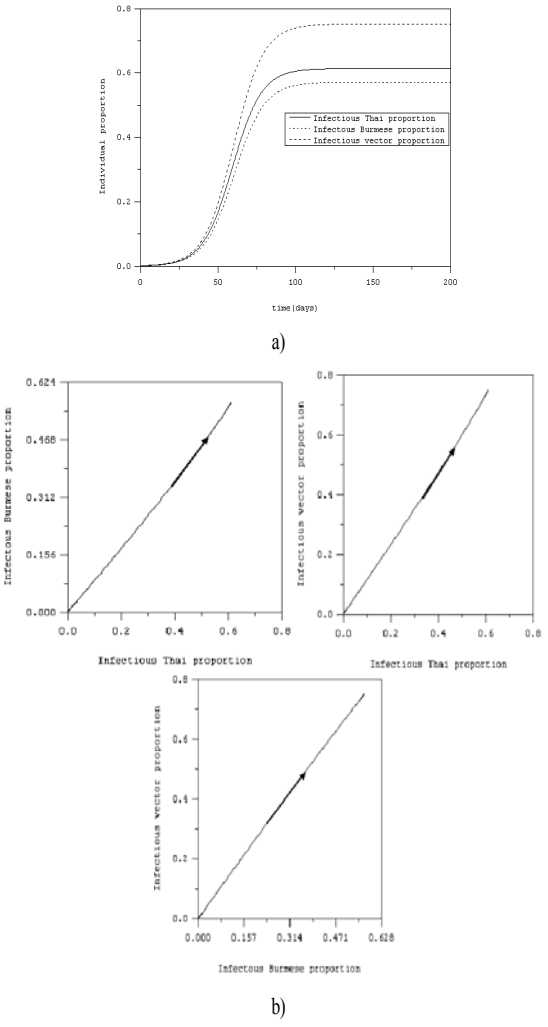
The other parameters are on the text and $R_0 = 0.9$.

4b) The solution trajectories of our model.

The parameters are similar to fig.4a).

As we see in Figure 4(a), the three infectious populations ($I_T(t)$, $I_B(t)$ and $I_v(t)$) go to zero as $t \to \infty$, meaning that the equilibrium state is the disease free state. The numerical

values of the parameters lead to a threshold number $R_0 = 0.9$. The trajectories of the solutions in the 2D $I_B - I_T$ plane, the 2D $I_v - I_T$ plane and the 2D $I_v - I_B$ plane are shown in Figure 4b. The arrows in these planes show the directions of the trajectories as $t \to \infty$, which are towards the disease free state. The numerical simulation is therefore in agreement with the behavior predicted when $R_0 < 1$.



a)

b)

Fig. 5 5a) Time series of I_T, I_B and I_v.

The parameters for the transmission rate are as follows: $\gamma_{h_{F_T}} = 0.08, \gamma_{h_{v_T}} = 0.075,$

$\gamma_{h_{F_B}} = 0.07, \gamma_{h_{v_B}} = 0.06,$

$\gamma_{v_T} = 0.09, \gamma_{v_B} = 0.08$. The other parameters are on the text and $R_{E_T} = 1.64376$, $R_{E_B} = 2.15522$ and $R_{E_v} = 5.89$.

5b) The solution trajectories of our model. The parameters are similar to fig.5 a).

We now change the values of the parameters and set P = 0.6. The values are given in the figure caption of Figure 5. These values give $R_{E_T} = 1.644$, $R_{E_B} = 2.155$ and $R_v = 5.89$. These are the conditions for the epidemic state $E_1 = (I_T^*, I_B^*, I_v^*)$ to be the stable equilibrium state. This is indeed seen in Figure 5a. The trajectories of the solutions in the 2D $I_B - I_T$ plane, the 2D $I_v - I_T$ plane and the 2D $I_v - I_B$ plane are shown in Figure 5b. As $t \to \infty$, the trajectories tend to the limiting values indicated on Figure 5a.

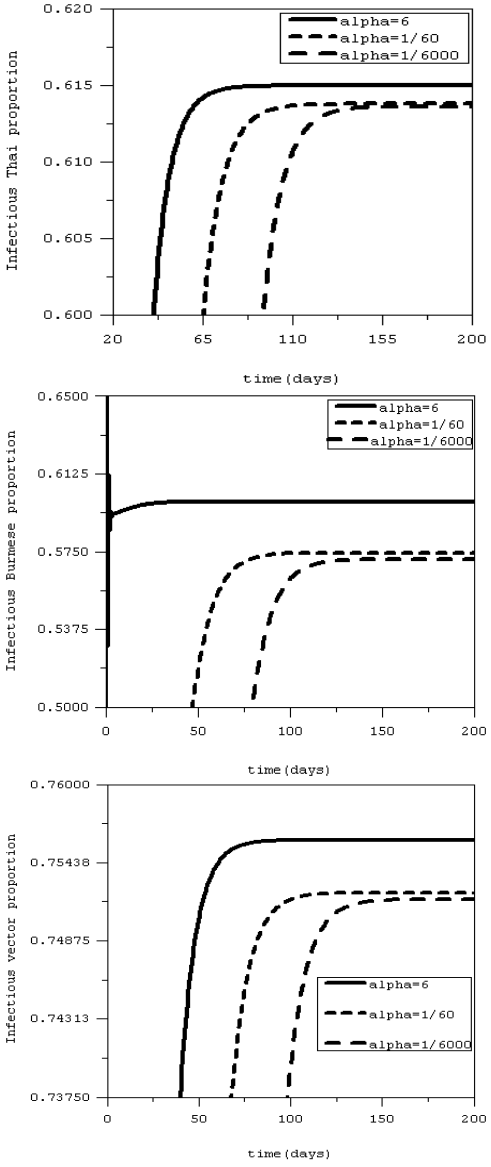


Fig. 6. Time series of I_T, I_B and I_v for the different values of α. The values of parameters are $\mu_h = 0.0000391$, $\mu_v = 1/30$, $r_F = 1/30$, $r_v = 1/25$, $\gamma_{h_{F_T}} = 0.008,$

$\gamma_{h_{v_T}} = 0.075,\ \gamma_{h_{F_B}} = 0.07,\ \gamma_{h_{v_B}} = 0.06,$

$\gamma_{v_T} = 0.09, \gamma_{v_B} = 0.08$, $\mu_v = 1/30$.

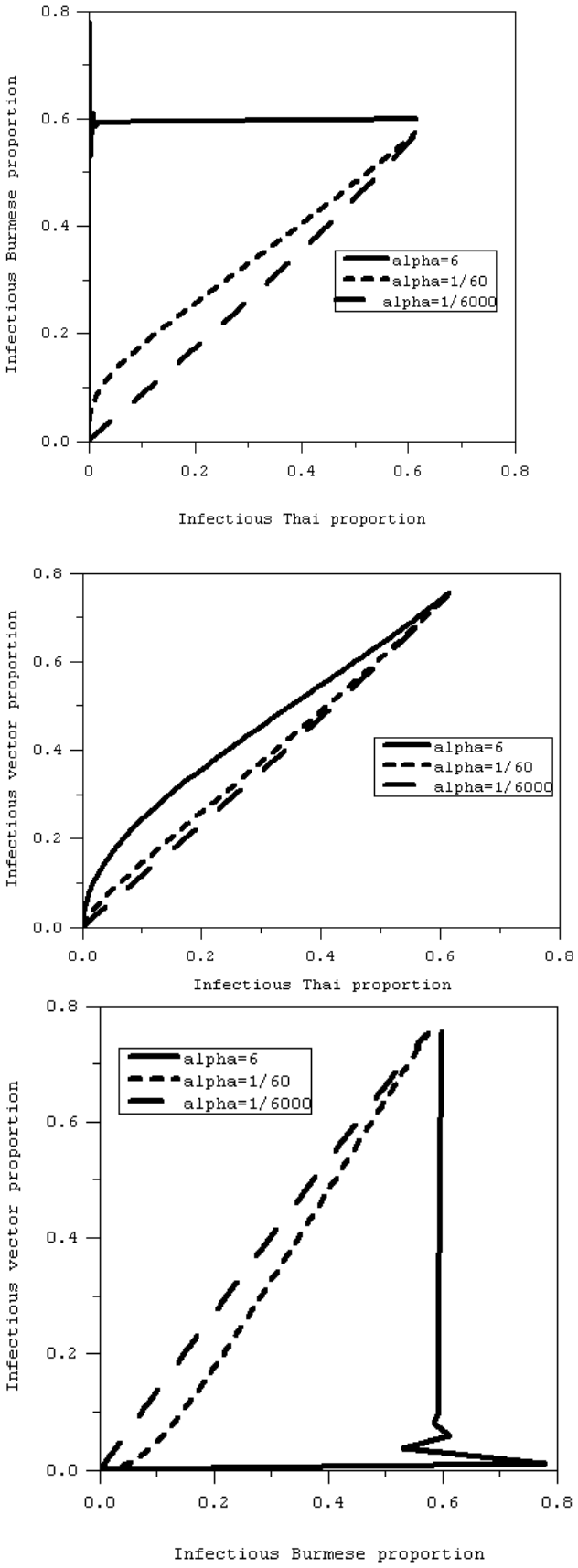


Fig. 7. The solution trajectories of our model for the different values of α. The parameters are similar to fig.6.

On Figure 6 and figure 7, we plot the time evolutions of the three infected populations ($I_T(t)$, $I_B(t)$ and $I_v(t)$) and the trajectories of the solutions in the 2D $I_B - I_T$ plane, the 2D $I_v - I_T$ plane and the 2D $I_v - I_B$ plane, respectively for different values of α, the reciprocal of the time that the Burmese stay in Thailand before they return to Myanmar. As $t \to \infty$, the trajectories tend to the limiting values indicated on Figure 6. The time evolutions of the three populations shown in Figure 5a are those when the Burmese stay a long time. The present behaviors are for the case when the Burmese stay 1/6 day, 60 days and 6000 days. Figure 6 shows that a higher number of Thais will be infectious if the Burmese stay in Thailand for shorter periods. If the Burmese stay for longer periods, the number of Thais infectious at a given time will be lower. The reason for this is that initially, the Burmese have a higher incident rate of active malaria infection. They would be able to pass to the illness to the Thai at the beginning. If they stay longer, they would develop the same incidence rate as the Thais and less likely to pass on the malaria

IV. CONCLUSION

In this study, we have analyzed a mathematical model of Malaria which describes the situation along the Thai-Myanmar border. Along this border, there are two types of malaria in circulation, *P .Falciparum* and *P. Vivax*. There is a seasonal migration of Burmese into Thailand. We find that there are two equilibrium states, a disease free state and an epidemic state. We establish the threshold conditions needed for each of the equilibrium states to exist. The numerical results confirm our analytical results (see figure 4 and 5). When R_0 is less than one, the normalized individual populations tend to the disease free state. The normalized individual population tend to the epidemic state when R_{E_T}, R_{E_B} and R_{E_v} are greater than one.

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