

Mathematical Model for the Transmission of Two Plasmodium Malaria

P. Pongsumpun*

Abstract—Malaria is transmitted to the human by biting of infected Anopheles mosquitoes. This disease is a serious, acute and chronic relapsing infection to humans. Fever, nausea, vomiting, back pain, increased sweating anemia and splenomegaly (enlargement of the spleen) are the symptoms of the patients who infected with this disease. It is caused by the multiplication of protozoa parasite of the genus Plasmodium. Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae and Plasmodium ovale are the four types of Plasmodium malaria. A mathematical model for the transmission of Plasmodium Malaria is developed in which the human and vector population are divided into two classes, the susceptible and the infectious classes. In this paper, we formulate the dynamical model of Plasmodium falciparum and Plasmodium vivax malaria. The standard dynamical analysis is used for analyzing the behavior for the transmission of this disease. The Threshold condition is found and numerical results are shown to confirm the analytical results.

Keywords—Dynamical analysis, Malaria, mathematical model, threshold condition.

I. INTRODUCTION

MALARIA disease is a major public health problem in the world. This disease continues to afflict the poor nations.

It is one of the top ten killer diseases in the world. In each year, there are estimated between 300 to 500 million clinical episodes of malaria and 1.5 to 2.7 million deaths worldwide, 90% of which occur in tropical Sahara. Outside Africa, some two-thirds of the remaining cases occur in just three countries; Brazil, India and Sri Lanka. However, it exists in some 100 countries [1]. It is an infectious disease caused by the parasite genus Plasmodium. There are four species of this parasite causing Malaria, namely, Plasmodium vivax, Plasmodium falciparum, Plasmodium ovale and Plasmodium malariae [2]. Malaria is transmitted to the human by biting of the female Anopheles mosquito. 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. Plasmodium falciparum is the commonest species spreading on the tropic and subtropics such as Africa, South America and Asia. Plasmodium vivax is found in the widest area. It can be found in many temperature zones, subtropics and tropic such as China, Turkey, Latin America and Asia. Plasmodium malariae is found in the same breadth as Plasmodium falciparum but is much less common in areas such as Central America. Plasmodium ovale is found predominantly in tropic Africa, but many occur in the West Pacific [3]. The Plasmodium genus of protozoa parasites has a life cycle which is split between a vertebrate host and an insect vector. The Plasmodium species, with the exception of Plasmodium malariae (which may affect the higher primates) are exclusively parasites of man. The sporozoites from the mosquito salivary gland are injected into the human as the mosquito must infect anticoagulant saliva to ensure an even flowing meal. Once in the human bloodstream, the sporozoites arrive in the liver and penetrate hepatocytes, where they remain for 9-16 days, multiplying within the cells. On release, they return to the blood and penetrate red blood cells in which they produce either merozoites or micro and macrogametocytes, which have no further activity within the human host. Another mosquito arriving to feed on the blood may suck up these gametocytes into its gut, where exflagellation of microgametocytes occurs, and the macrogametocytes are fertilized. The resulting ookinete penetrates the wall of a cell in the midgut, where it develops into an oocyst. Sporogony within the oocyst produces many sporozoites and, when the oocyst ruptures, the sporozoites migrate to the salivary gland, for injection into another host [4,5]. The transmission of malaria is usually described by the Ross-MacDonald (RM) model [6]. Nevertheless, this model is only suitable for the transmission of the P. falciparum malaria. Following the data of Malaria in Thailand during 1965 to 2007[7], the most Malaria cases in Thailand are due to Plasmodium Falciparum and Plasmodium Vivax.

II. TRANSMISSION MODEL

The human and vector populations are considered in this transmission model. The human and vector populations are separated into susceptible and infectious classes. We assume

P. Pongsumpun is with the Department of Mathematics, Faculty of Science, King Mongkut's Institute of Technology Ladkrabang, Chalokkrung road, Ladkrabang, Bangkok 10520, Thailand (corresponding author to provide phone: 662-737-3000 ext. 6196; fax: 662-326-4344 ext. 284 ; e-mail: kppuntan@kmitl.ac.th)*

that there are two different species of Plasmodium Malaria (Plasmodium falciparum and Plasmodium Vivax) and the human and vector populations are constant. The diagram for the transmission of this disease is described by the following diagram:

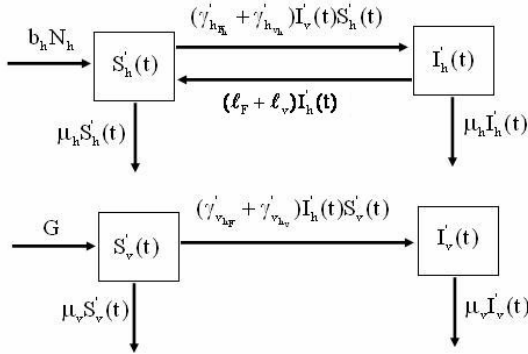


Fig. 1 The transmission diagram of the model.

Let $S'_h(t)$ is the number of susceptible human,

$I'_h(t)$ is the number of infectious human,

$S'_v(t)$ is the number of susceptible vector,

$I'_v(t)$ is the number of infectious vector.

The dynamical equations for the human population are

$$\frac{dS'_h(t)}{dt} = b_h N_h + (\ell_F + \ell_v) I'_h(t) - ((\gamma'_{h_{hF}} + \gamma'_{h_{hV}}) I'_v(t) S'_h(t) + \mu_h) S'_h(t) \quad (1)$$

$$\frac{dI'_h(t)}{dt} = (\gamma'_{h_{hF}} + \gamma'_{h_{hV}}) I'_v(t) S'_h(t) - (\ell_F + \ell_v + \mu_h) I'_h(t) \quad (2)$$

$$\frac{dS'_v(t)}{dt} = G - ((\gamma'_{v_{hF}} + \gamma'_{v_{hV}}) I'_h(t) S'_v(t) + \mu_v) S'_v(t) \quad (3)$$

$$\frac{dI'_v(t)}{dt} = (\gamma'_{v_{hF}} + \gamma'_{v_{hV}}) I'_h(t) S'_v(t) - \mu_v I'_v(t) \quad (4)$$

with two conditions $S'_h + I'_h = N_h$ and $S'_v + I'_v = N_v$

where

μ_h is the death rate of human population,

μ_v is the death rate of vector population,

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

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

b_h 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,

ℓ_F is the rate at which a person who infected with *P.falciparum* leaves the infectious class,

ℓ_v is the rate at which a person who infected with *P.vivax* leaves the infectious class,

G is the constant recruitment rate of vector population.

We normalize the equations (1)-(4) by defining new variables:

$$S_h(t) = S'_h(t) / N_h, \quad I_h(t) = I'_h(t) / N_h, \quad S_v(t) = S'_v(t) / N_v,$$

$$I_v(t) = I'_v(t) / N_v.$$

The total human and vector populations are constant, this gives $b_h = \mu_h$ and G/μ_v . We obtain the equations as follows:

$$\frac{dI_h(t)}{dt} = (\gamma'_{h_{hF}} + \gamma'_{h_{hV}})(G/\mu_v)I_v(t)(1 - I_h(t)) - (\ell_F + \ell_v + \mu_h)I_h(t) \quad (5)$$

$$\frac{dI_v(t)}{dt} = (\gamma'_{v_{hF}} + \gamma'_{v_{hV}})N_h I_h(t)(1 - I_v(t)) - \mu_v I_v(t) \quad (6)$$

with the new two conditions

$$S_h(t) + I_h(t) = 1, \text{ and } S_v(t) + I_v(t) = 1.$$

III. ANALYSIS MODEL

A. Equilibrium Point

Two equilibrium points are found by setting the right hand side of (5)-(6) equal to zero. This gives

1) the disease free equilibrium point

$$E_0 = (0, 0) \text{ and}$$

2) the endemic disease equilibrium point

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

where

$$I_h^* = \frac{I_v^*}{M_1 + I_v^*} \quad (7)$$

$$I_v^* = \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hV}})N_h - \mu_v M_1}{(\gamma'_{v_{hF}} + \gamma'_{v_{hV}})N_h + \mu_v} \quad (8)$$

$$\text{with } M_1 = \frac{\ell_F + \ell_v + \mu_h}{(\gamma'_{h_{hF}} + \gamma'_{h_{hV}})(G/\mu_v)}. \quad (9)$$

A.1 Local Asymptotical Stability

The local stability of an equilibrium point is determined from the Jacobian matrix of the right hand side of the above set of differential equations evaluated at the equilibrium point.

A.1.1 Disease free equilibrium point

For the equations defined by (5)-(6), the Jacobian matrix evaluated at E_0 is the 2×2 matrix given by

$$\begin{bmatrix} -M_2 & M_2/M_1 \\ (\gamma'_{v_{hF}} + \gamma'_{v_{hV}})N_h & -\mu_v \end{bmatrix} \quad (10)$$

The eigenvalues are obtained by solving the matrix equation, $\det(J - \lambda I_2) = 0$. To evaluate the determinant, we obtained the following characteristic equation

$$\lambda^2 + a_1 \lambda + a_0 = 0 \quad (11)$$

where

$$a_1 = M_2 + \mu_v, \quad (12)$$

$$a_0 = M_2 \left(\frac{M_1 \mu_v - (\gamma'_{v_{hF}} + \gamma'_{v_{hv}}) N_h}{M_1} \right) \quad (13)$$

with $M_2 = \ell_F + \ell_v + \mu_h$.

From (11), the above two conditions of Routh-Hurwitz criteria for local asymptotical stability in second order characteristic polynomial equation are

- i) $a_1 > 0$,
- ii) $a_0 > 0$.

We will see that a_1 is always positive. a_0 is positive when

$$\frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}}) N_h}{M_1 \mu_v} < 1.$$

Therefore the disease free equilibrium point is locally stable

for $P_0 = \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}}) N_h}{M_1 \mu_v} < 1$.

A.1.2 Disease endemic equilibrium point

The Jacobian for this equilibrium point is

$$\begin{bmatrix} -(M_2/M_1)I_v^* - M_2 & (M_2/M_1)(1 - I_h^*) \\ (\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h(1 - I_v^*) & -(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h I_h^* - \mu_v \end{bmatrix} \quad (14)$$

where I_h^* and I_v^* are given by equations (7)-(9).

The characteristic equation for the Jacobian matrix evaluated at the second equilibrium state, given by (5)-(6), is

$$\lambda^2 + b_1 \lambda + b_0 = 0 \quad (15)$$

with

$$b_1 = \mu_v + (\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h I_h^* + M_2 \left(1 + \frac{I_v^*}{M_1} \right), \quad (16)$$

$$b_0 = \frac{M_2}{M_1} (\mu_v (M_1 + I_v^*) + (\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h (1 + (1 + M_1)I_h^* + I_v^*)) \quad (17)$$

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

- i) $b_1 > 0$
- ii) $b_0 > 0$

We can see that b_1 is always positive.

$$b_0 = \frac{M_2}{M_1} (\mu_v (M_1 + I_v^*) + (\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h (1 + (1 + M_1)I_h^* + I_v^*))$$

is positive for $I_v^* = \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h - \mu_v M_1}{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h + \mu_v}$ or

$$(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h > \mu_v M_1 \text{ or } \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h}{\mu_v M_1} > 1.$$

So, E_1 is locally stable for $P_0 > 1$.

B. Numerical Simulation

In this paper, we are interested in the transmission of disease in human with season. The values of the parameters used in this study are as follows:

$\mu_h = 1/(365 \times 65)$ per day corresponds to a life expectancy of 65 years in human. The mean life of vectors is 30 days; $\mu_v = (1/30)$ per day. The other parameters are arbitrarily chosen.

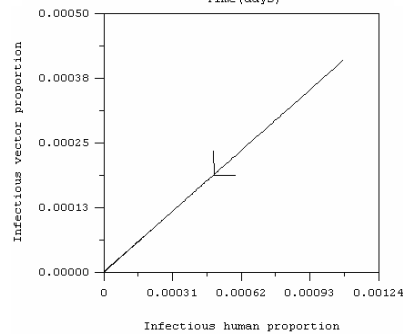
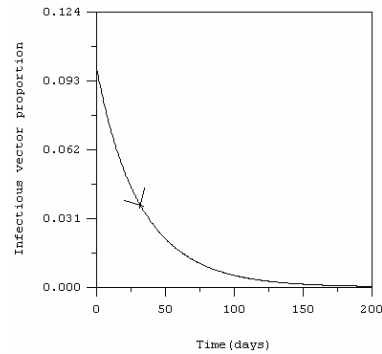
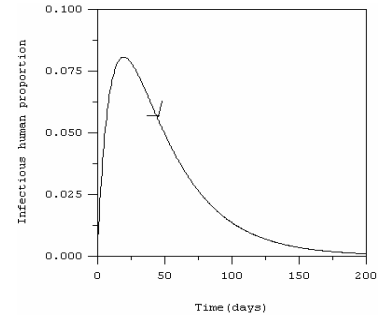


Fig. 2 Numerical solutions of (5)-(6), demonstrate the time series of I_h , I_v and trajectory solutions, respectively, for $R_0 < 1$, with $\mu_h = 1/(365 \times 65) \text{ day}^{-1}$, $\mu_v = (1/30) \text{ day}^{-1}$, $G = 200$, $N_h = 100$, $\gamma'_{h_{hF}} = 0.00001$, $\gamma'_{h_{hv}} = 0.00001$, $\gamma'_{v_{hF}} = 0.00001$ and $\gamma'_{v_{hv}} = 0.00001$, $P_0 = 0.0981254$. The fractions of populations converge to the free state $(0, 0)$.

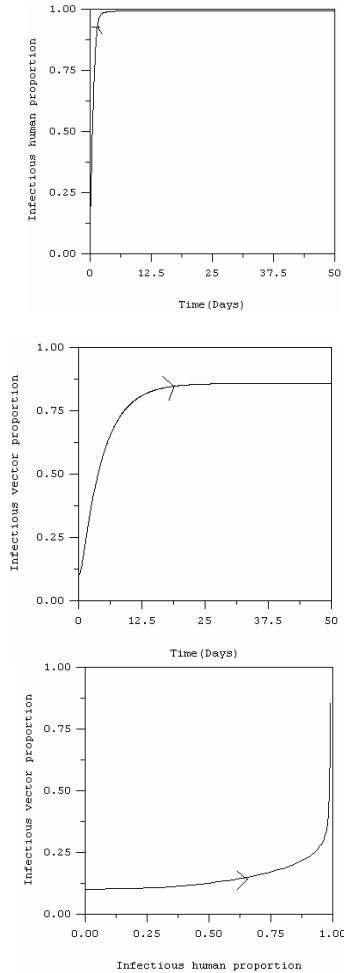


Fig. 3 Numerical solutions of (5)-(6) , demonstrate the times series of I_h and I_v respectively, for $R_0 > 1$, with $\mu_h = 1/(365 \times 65) \text{ day}^{-1}$, $\mu_v = (1/30) \text{ day}^{-1}$, $G = 20,000, N_h = 10,000, \gamma'_{h_{Fh}} = 0.00001, \gamma'_{h_{vh}} = 0.00001, \gamma'_{v_{hF}} = 0.00001$ and $\gamma'_{v_{hv}} = 0.00001$, $P_0 = 981.254$. The fractions of populations oscillate to the endemic state (0.99291,0.856269).

IV. DISCUSSION AND CONCLUSION

The threshold number for our model is

$$P_0 = \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})N_h}{M_1\mu_v} = \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})(\gamma'_{h_{Fh}} + \gamma'_{h_{vh}})(G/\mu_v)N_h}{(\ell_F + \ell_v + \mu_h)\mu_v}$$

$$= \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})\gamma'_{h_{Fh}}GN_h}{(\ell_F + \ell_v + \mu_h)\mu_v^2} + \frac{(\gamma'_{v_{hF}} + \gamma'_{v_{hv}})\gamma'_{h_{vh}}GN_h}{(\ell_F + \ell_v + \mu_h)\mu_v^2} \quad (18)$$

The quantity $P_0 = \sqrt{P_0}$ is the basic reproductive number of the disease. We compare the proportion of all populations when the basic reproductive number are difference, we show in the following figures:

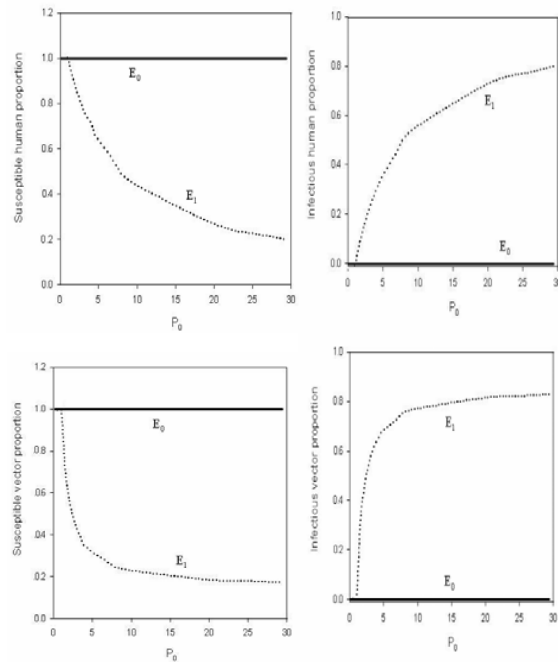


Fig. 4 Bifurcation diagram of (5)-(6), demonstrate the equilibrium solutions of S_h, I_h, S_v and I_v respectively, with $\mu_h = 1/(365 \times 65) \text{ day}^{-1}, \mu_v = (1/30) \text{ day}^{-1}, N_h = 10,000, \gamma'_{h_{Fh}} = 0.00001, \gamma'_{h_{vh}} = 0.00001, \gamma'_{v_{hF}} = 0.00001$ and $\gamma'_{v_{hv}} = 0.00001, \ell_F = 1/30, \ell_v = 1/25$.

From Fig. 2 and Fig. 3, we will see that the proportions of populations converge to the disease free equilibrium state for P_0 less than 1. For P_0 greater than 1, the proportions of populations oscillate to the endemic disease equilibrium point. The threshold numbers are used to be the alternative way for controlling the diseases [8]-[11]. The behavior of the solutions in the mathematical model can be described in terms of the threshold number: If this number is less than or equal to one, thus an infective replace itself with less than one new infective, the disease die out. Moreover, the susceptible fraction approaches one since everyone is susceptible when the disease has vanished. If this number is greater than one, the normalized susceptible classes decrease. The normalized infectious classes increase. This subsequent behavior occurs because there are enough susceptible human to be infected from infectious vector.

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