International Journal of Engineering, Mathematical and Physical Sciences
ISSN: 2517-9934
Vol:2, No:8, 2008

Plasmodium Vivax Malaria Transmission in a Network of Villages

P. Pongsumpun, and I. M. Tang

Abstract**—**Malaria is a serious, acute and chronic relapsing infection to humans. It is characterized by periodic attacks of chills, fever, nausea, vomiting, back pain, increased sweating anemia, splenomegaly (enlargement of the spleen) and often-fatal complications.The malaria disease is caused by the multiplication of protozoa parasite of the genus Plasmodium. Malaria in humans is due to 4 types of malaria parasites such that Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae and Plasmodium ovale. P.vivax malaria differs from P. falciparum malaria in that a person suffering from P. vivax malaria can experience relapses of the disease. Between the relapses, the malaria parasite will remain dormant in the liver of the patient, leading to the patient being classified as being in the dormant class. A mathematical model for the transmission of P. vivax is developed in which the human population is divided into four classes, the susceptible, the infected, the dormant and the recovered. In this paper, we formulate the dynamical model of P. vivax malaria to see the distribution of this disease at the district level.

Keywords**—**Dynamical model, household, local level, *Plasmodium Vivax* Malaria.

I. Introduction

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 [1]. The developmental biology [2] 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. The sporizoites (one of the stages of the malaria parasite) are introduced into the blood stream of the human by the bite of infected mosquitoes. These then move to the liver of the human. Here 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 *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 the *P. vivax* parasite. The reasons for *P. falciparum* malaria to be studied more than *P. vivax* malaria are (1) 90% of the malaria cases in Africa is due to *P. falciparum* malaria, (2) most of the deaths due to malaria (2-3 million a year) occur in Africa [3] and (3) *P. falciparum* malaria is a life threatening disease, while *P vivax* malaria is not.

Data on the incidences of Malaria due to the different types of malaria between during 2003 to 2006 in Thailand indicates that the rate of increase of the incidence of *P. vivax* is the highest among the four types. This can be seen in Fig. 1.

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

I. M. Tang is with the Department of Physics, Faculty of Science, Mahidol University, Bangkok 10520, Thailand.

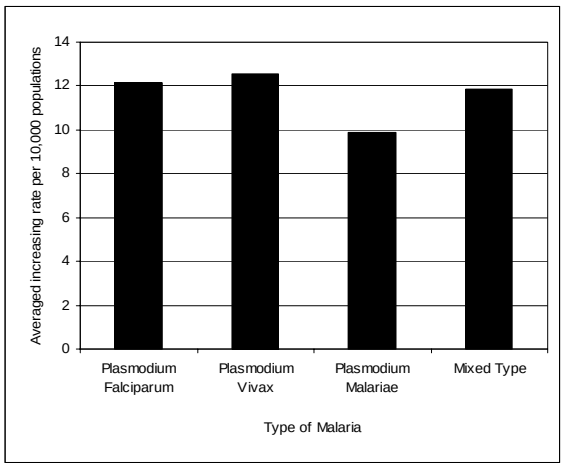

Fig. 1 The average increasing rate per 10,000 populations of Malaria in Thailand during 2003-2006[4]

It was commonly assumed that information about *vivax* could be extrapolated from the *falciparum* research. This assumption was challenged at a conference convened by the Multilateral Initiative on Malaria [5]. The transmission of malaria is usually described by the Ross-MacDonald (RM) model [6]. However, this model is only suitable for the transmission of the *P. falciparum* malaria since it does not contain a role of possible relapses of the illness. One of the present authors (IMT) has introduced a simple mathematical model [7] 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 reinfected when the hypnozoites are activated.

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. In this study, the dynamics transmission of *P. vivax* between village is considered. We introduce in Section II, the modification of the RM model which would make it applicable to the transmission of *P. vivax* malaria in the district. In Section III, we simulate the consequences of changing the number of villages, number of houses in each village, contact rates and relapse rates. We discuss in Section IV, the implication of the insights obtained from the simulations. 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. It is estimated that about 50% of the malaria cases outside of Africa and 10% in Africa are due to P. *vivax* and that the percentages are soaring.

II. Transmission Model

The mathematical modeling of the epidemiology of malaria (*P. falciparum*) was started by Ross [8] in 1911 and improved on by MacDonald [9]. 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 [9] 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 infected and infectious.

In this study, the epidemics of *P. vivax* at the district level are considered, the movement of the human and the mosquitoes are satisfied the following assumptions:

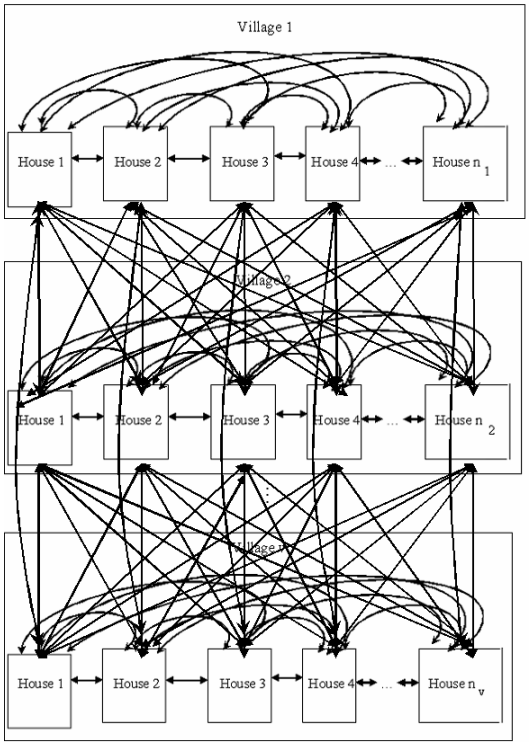

Fig. 2 The diagram shows the movement of the population between houses in several villages for one district

Suppose that there are M persons, v villages in each district, n_i is the number of house in village i^{th}; i = 1,…, v. The persons move to any houses in the same village or the different village by random process. This process is done by random the 1^{st} person to the M^{th} person (with uniformly distribution) go to each house of the same village or the different village everyday. Each day, one person can go only one time in one house of his/her village or one house of the others villages. The probability for each person to visit each house of his/her village or the others villages is equal. The persons who stay in any house at the beginning time will come back to their villages and their houses at the ending time. We assume that at the first day, there is only one infected human in one house and there is no infected human for the other houses in the district.

In our model for the transmission of *P. vivax* at the district level, we divide the host (human) population into susceptible, infected, dormant and recovered classes. The last category, the recovered are susceptible to further infections and so they

reenter into the susceptible class. The vector population is separated into susceptible and infected classes.

The variables in our model are defined as follows:

$Sh_{t,i}$ is the number of susceptible persons in house i^{th} after visited at day t , i = 1,2,3,…, n_j; j = 1,2,3,...,v,

$Ih_{t,i}$ is the number of infected persons in house i^{th} after visited at day t, i = 1,2,3,…, n_j; j = 1,2,3,...,v,

$Dh_{t,i}$ is the number of dormant persons in house i^{th} after visited at day t, i = 1,2,3,…, n_j; j = 1,2,3,...,v,

$Rh_{t,i}$ is the number of recovered persons in house i^{th} after visited at day t, i = 1,2,3,…, n_j; j = 1,2,3,...,v,

$Sv_{t,i}$ is the number of susceptible mosquitoes in house i^{th} after visited at day t, i = 1,2,3,…, n_j; j = 1,2,3,...,v,

$Iv_{t,i}$ is the number of infected mosquitoes in house i^{th} after visited at day t, i = 1,2,3,…, n_j; j = 1,2,3,...,v,

M is the total number of persons,

c is the ending time.

The dynamics of human and vector populations in one district are given by

$$\begin{aligned}
\Delta Sh_{t,i} &= -\gamma Iv_{t,i} Sh_{t,i} + r_3 Dh_{t,i} + (1-\alpha) r_1 Ih_{t,i} + r_4 Rh_{t,i} \\
\Delta Ih_{t,i} &= \gamma Iv_{t,i} Sh_{t,i} - r_1 Ih_{t,i} + r_2 Dh_{t,i} - r_5 Ih_{t,i} \\
\Delta Dh_{t,i} &= \alpha r_1 Ih_{t,i} - (r_2 + r_3) Dh_{t,i} \qquad (1) \\
\Delta Rh_{t,i} &= r_5 Ih_{t,i} - r_4 Rh_{t,i} \\
\Delta Sv_{t,i} &= K_{t,i} - \beta Sv_{t,i} Ih_{t,i} - \mu_v Sv_{t,i} \\
\Delta Iv_{t,i} &= \beta Sv_{t,i} Ih_{t,i} - \mu_v Iv_{t,i}
\end{aligned}$$

for i = 1,2,3,…, n_j; j = 1,2,3,...,v.

n_i is the number of house in village i^{th}; i = 1,…, v,

v is the number of villages in each district,

$K_{t,i} = \mu_v(Sv_{t,i}+Iv_{t,i})$ = Basic recruitment number of the mosquitoes in house n_i at day t,

β is the transmission rate of dengue virus from infectious person to susceptible vector,

μ_v is the death rate of the mosquitoes,

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

(1-α) is the percentage of infected humans who recover and become susceptible again,

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,

γ is the rate at which the *P. vivax* parasite is transmitted from the mosquito to the human and is given by [10]

$$\gamma = b\frac{\beta_h}{N_T + m} \qquad (2)$$

where b is the specie-dependent biting rate of the mosquitoes; m is the population of other animals that the mosquitoes can feed on; N_T is the total human populations and β_h is the probability the parasite passed on by the mosquito will continue to thrive in the human. β is the rate at which the mosquitoes become infected with the Plasmodium *vivax* parasite once the mosquito has bitten an infected human. β is defined by [10]

$$\beta = b\frac{\beta_v}{N_T + m}$$

where β_v is the probability the parasite passed to the mosquito by biting human.

III. Numerical Solutions

The simulations of the model are calculated to see the time distributions of this disease. The time distributions of infected and dormant humans for the different situations are shown in the following figures:

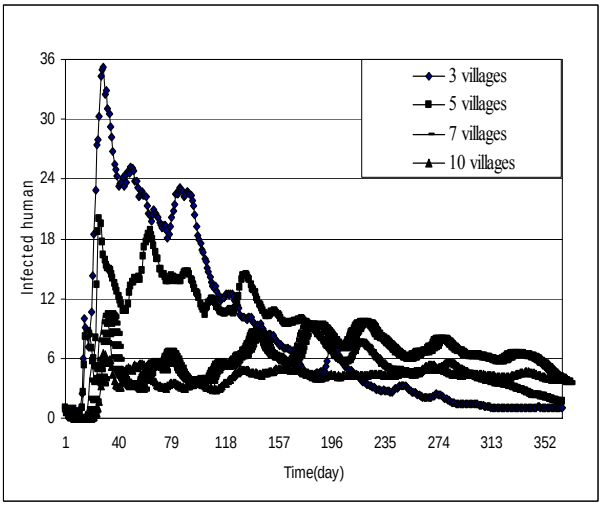

Fig. 3 Model outputs display the time distribution of infected human for the different number of villages in each district. The parameters in our model are $r_1 = 1/14$, $r_2 = 1/(365*3)$, $r_3 = 1/(25)$, $r_4 = 1/(365*10)$, $r_5 = 1/3$, γ = 0.55, β = 0.45, μ_v = 0.04, M = 1,000, c = 365

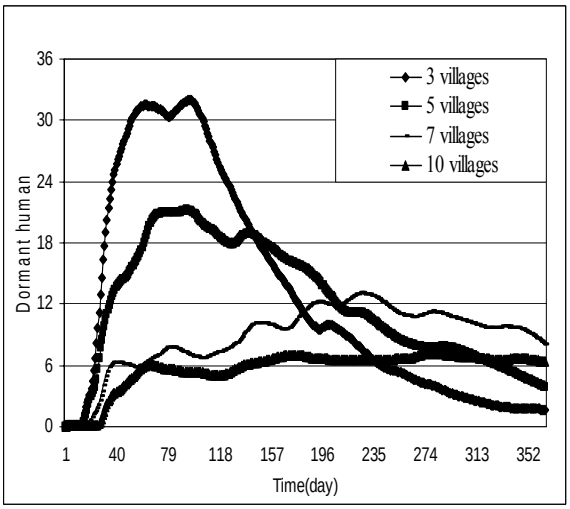

Fig. 4 Model outputs display the time distribution of dormant human for the different number of villages in each district. The parameters are same as Fig. 3

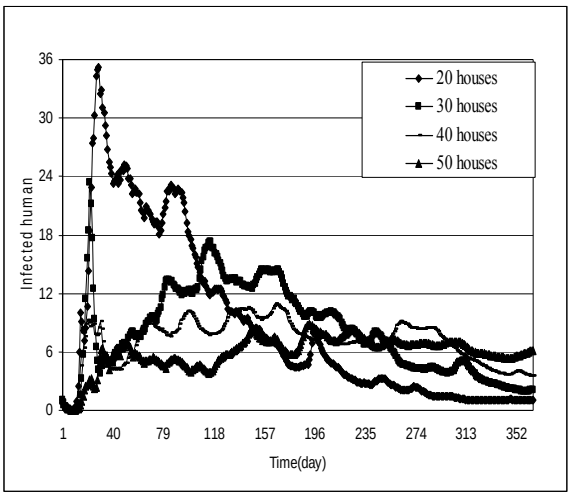

Fig. 5 Model outputs display the time distribution of infected human for each district. There are 3 villages in each district. The different numbers of houses in each village are compared. The other parameters are same as Fig. 3

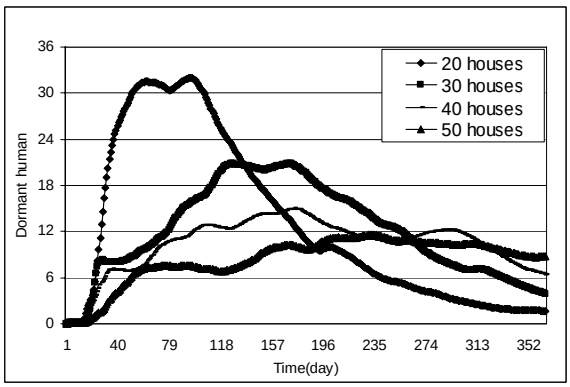

Fig. 6 Model outputs display the time distribution of dormant human for each district. The parameters are same as Fig. 5

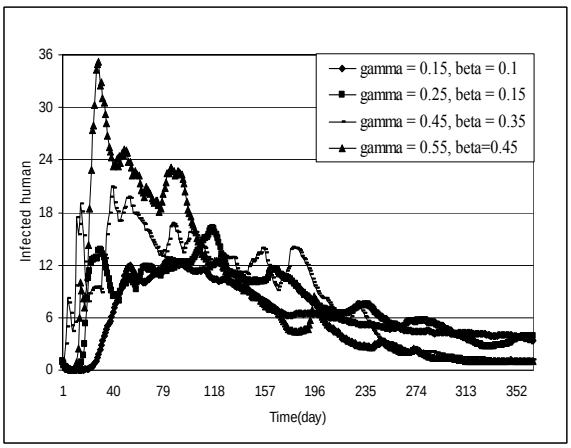

Fig. 7 Model outputs display the time distribution of infected human for the different contact rates in each district. There are 3 villages in each district; 20 houses in each village. The other parameters are same as Fig. 3 except the contact rates

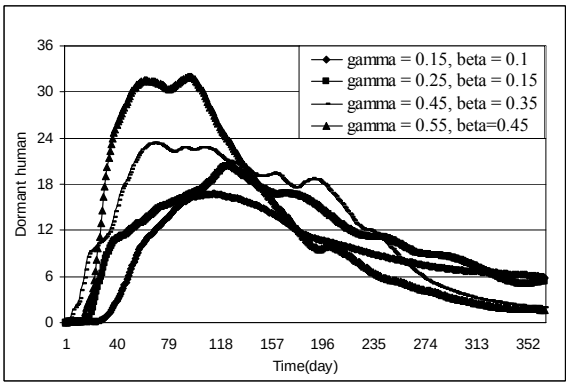

Fig. 8 Model outputs display the time distribution of dormant human for the different contact rates in each district. The parameters are same as Fig. 7

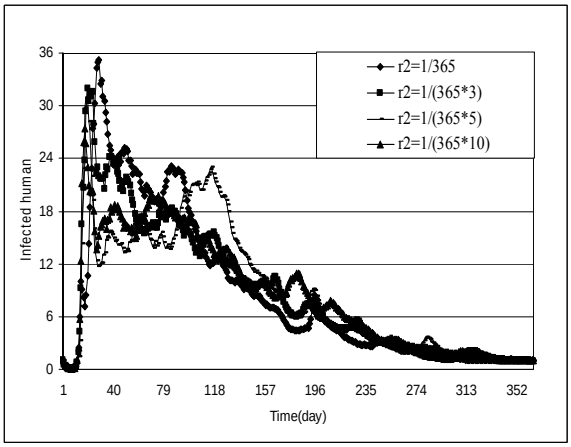

Fig. 9 Model outputs display the time distribution of infected human for the different rate at which the dormant human relapses back to the infected human in each district. There are 3 villages in each district; 20 houses in each village. The other parameters are same as Fig. 3 except r_2

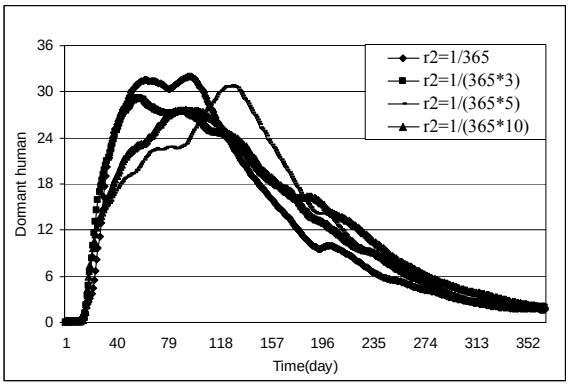

Fig. 10 Model outputs display the time distribution of dormant human for the different rate at which the dormant human relapses back to the infected human in each village. The parameters are same as Fig. 9

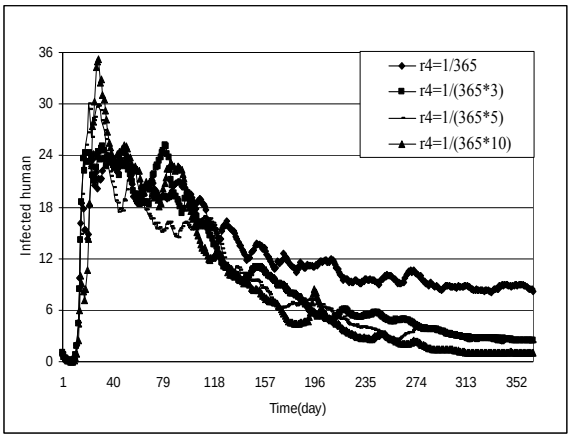

Fig. 11 Model outputs display the time distribution of infected human for the different rate at which the recovered human relapses back to the susceptible human in each district. There are 3 villages in each district; 20 houses in each village. The other parameters are same as Fig. 3 except r_4

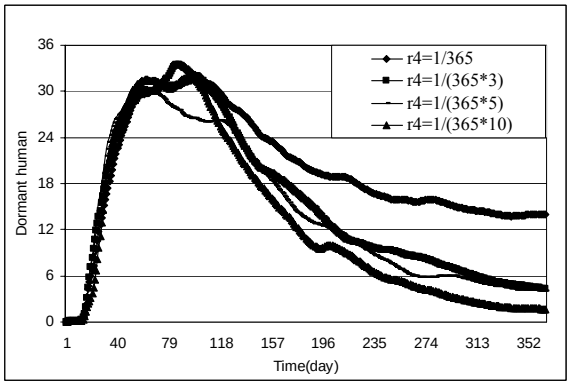

Fig. 12 Model outputs display the time distribution of dormant human for the different rate at which the recovered human relapses back to the susceptible human in each district. The other parameters are same as Fig. 11

IV. Conclusion

In this study, we have compared the results of the simulation when different values of several parameters are used. Figs. 3 to 4 show the time distributions of infected and dormant human when the number of villages in each district is difference. Figs. 5 to 6 show the time distributions of infected and dormant human when the number of house in each village for each district is difference but the number of villages is equal. Figs. 7 to 8 show the time distributions of infected and dormant human when the contact rates in each district are difference. Figs. 9 to 10 show the time distributions of infected and dormant human when the rate at which the dormant human relapses back to the infected human in each district is difference. Figs. 11 to 12 show the time distributions of infected and dormant human when the rate at which the recovered human relapses back to the susceptible human in each district are difference. We will see that the epidemic sizes are higher when the smaller number of households, the smaller number of villages and the higher contact rates. But when the rate at which the dormant human relapses back to the infected human and the rate at which the recovered human relapses back to the susceptible human are higher, the epidemic sizes are smaller.

Acknowledgment

This work is supported by Commission on Higher Education and the Thailand Research Fund according to contract number MRG5080078. The authors would like to thank Dr. Sopon Iamsirithaworn, Ministry of Public Health, Thailand for supporting the data.

References

[1] WHO World malaria situation in 1994. Weekly Epidemiological Record. WHO 1997, vol.72, no.38, 269-276.
[2] 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.
[3] WHO, World Malaria Situation in 1994: Weekly Epidemiological Record, Geneva, 1997.
[4] Annual Epidemiological Surveillance Report, 2003-2006, Division of Epidemiology, Ministry of Public Health, Royal Thai Government.
[5] B.Sina, "Focus on Plasmodium Vivax," Trends in Parasitology, vol.18, pp.287-289, 2002.
[6] R.M.Anderson and R.M.May, Infectious Disease of Humans, Dynamics and Control.Oxford U. Press, Oxford, 1991.
[7] 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.
[8] R.Ross, The preventation of Malaria, 2th ed. Murray, London, 1911.
[9] G.MacDonald, The epidemiology and control of malaria. Oxford University Press, London, 1957.
[10] L.Esteva and C.Vargas, "Analysis of a dengue disease transmission model," Mathematical Bioscience, vol.150, pp.131-151, 1998.