Deterministic Malaria Transmission Model with Acquired Immunity

J. Labadin, C. Kon M. L. and S. F. S. Juan

***Abstract*— This paper focuses on the development of a deterministic Malaria transmission model by considering the recovered population with and without immunity. A transmission model is found to be useful in providing a better understanding on the disease and the impact towards the human population. In this research, two possibilities were taken into account where one possibility is that infectious humans do not gain immunity while another possibility is that infectious humans will gain temporary immunity. The mathematical model is developed based on the SEIR model which has susceptible S_H, exposed E_H, infectious I_H and recovered R_H classes. The system of equations which were obtained were solved numerically and results were simulated and analyzed. The analysis includes the impact of the different values of the average duration to build effective immunity on the infectious humans. We observed that when the value of q, per capita rate of building effective immunity is increased, the maximum number of infected humans decreased. Hence, if an effective immunity can be build in a short period of time for those who recover from the disease, the number of cases could be reduced.**

***Index Terms*—mathematical modeling, malaria, transmission model, differential equations, immunity**

I. Introduction

Malaria is one of the most common infections in the world today. It is commonly caused by four species of protozoan parasites of the genus *Plasmodium : P.falciparum, P.vivax, P.ovale* and *P.malariae* [1]. Malaria is transmitted through the vectors, *Anopheles* mosquitoes and not directly from human to human. The disease infects humans of all ages and can be lethal. According to the World Health Organization (WHO) in year 2007, about 40% of the world's population, mostly those living in the poorest countries, are at risk of malaria. Of the 2.5 billion people at risk, more than 500 million become severely ill with malaria every year and more than 1 million die from the effects of the disease.

At present, malaria is endemic in most tropical countries including America, Asia and Africa. It remains a public health concern in many countries in South East Asia. Apart from the four common species mentioned above, simian malaria, *P.inui, P.cynomolgi,* and *P.knowlesi* are also known to cause the disease in humans [2]. Cases of malaria in the Kapit division Sarawak has been detected to be caused by *P.knowlesi* [3]. This malaria parasite which infects long-tailed and pig-tailed macaque monkeys in nature had accounted for half of the cases studied in the Kapit division [3].

Mathematical models for transmission dynamics of malaria are useful in providing a better knowledge of the disease, to plan for the future and consider appropriate control measures. Models have played great roles in the development of the epidemiology of the disease. The study on malaria using mathematical modeling originated from the works of Ross [3]. According to Ross, if the mosquito population can be reduced to below a certain threshold then malaria can be eradicated. MacDonald did some modification to the model and included superinfection [4]. He showed that reducing the number of mosquitoes have little effect on epidemiology of malaria in areas of intense transmission. Dietz et al [5] added two classes of humans in their mathematical model, namely those with low recovery rate (more infections, greater susceptibility) and high recovery rate (less infections, less susceptibility). Compartmental models of malaria and differential equations are constructed to model the disease [7,8,13,14,20]. Chitnis et al [13] did a bifurcation analysis of a malaria model. Malaria transmission model which incorporate immunity in the human population had been studied [7,8,14]. Epidemiological models on the spread of anti-malarial resistance were also constructed [15].

In this paper, we present the malaria transmission model in Section II, where we took into account two possibilities: one is where infectious humans do not gain any immunity and the other, who have temporary immunity. After which, the model is simulated and the impact of changing the rate to build effective immunity and other parameters are studied numerically in Section III. We based our work on Malaria cases in general and not specifically on particular parasite genus. Finally, concluding remarks are discussed in Section IV.

II. Model Formulation

A malaria transmission model has been produced based on the epidemiology aspects of the disease. The compartmental model is as shown in Fig. 1. The human population is divided into the SEIR compartmental model which consists of four

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classes: susceptible S_H, exposed E_H, infectious I_H and recovered R_H. Blood meal taken by an infectious female Anopheline mosquito on a susceptible individual will cause sporozoites to be injected into the human bloodstream and will be carried to the liver. The individual will then move to the exposed class E_H. This will decrease the susceptible population S_H. Exposed humans are those who have parasites in them and the parasites are in asexual stages. They are without gametocytes and are not capable of transmitting the disease to susceptible mosquitoes. After the latent period, humans who are exposed will be transferred to the infectious class as they are with gametocytes in their blood stream making them infectious to female *Anopheles* mosquitoes. The infectious humans will recover after some time, gain immunity and move to the recovered with temporary immunity class or they can be susceptible again. This is because continuous exposure is necessary to ensure immunity is built [7]. Those who have recovered have immunity against the disease for a certain period. Acquired immunity exists but the mechanisms are yet to be fully apprehended [22]. As the immunity is temporary, it will fade off after a period of time [7]. Thus, the recovered humans will return to the susceptible class. Every class of human population is decreased by density-dependent and independent death and emigration except for the infectious class which has disease-induced death as an addition.

For the vector mosquitoes, the three compartments represent susceptible S_M, exposed E_M, and infectious I_M. There is no recovered class for the vector as mosquitoes never recover [13]. They are regulated by mortality [8]. Susceptible mosquitoes that feed on infectious or recovered human would have taken gametocytes in blood meal but do not have sporozoites in their salivary glands yet, thus this means they are entering into the exposed class. After fertilization, sporozoites are produced and migrate to the salivary glands ready to infect any susceptible host, the vector is then considered as infectious. The three compartments for the vector mosquitoes are reduced by density-dependent and independent death and emigration.

In the malaria model which has been constructed here, the total number of mosquito bites is restricted by total mosquito population whereas in [13] the total bites on humans is dependent on both human and mosquito population. In our model, the mosquito-human interaction follows the classic model as mentioned in Hethcote's review [9]. The differential equations which describe the dynamics of malaria in human and mosquito populations are formulated based from the compartmental diagram described in Figure 1. The descriptions for the variables used in the model are shown in Table 1 and parameters in Table 2.

The following assumptions are made to characterize the model:

(i) All newborns are susceptible to the disease.
(ii) The infectious period of mosquitoes ends when they die.
(iii) The lifespan of the mosquitoes does not depend on infection.
(iv) Human hosts recover from infection (without immunity) and move right back to susceptible state OR gain temporary immunity before losing it and returning to the susceptible class.
(v) Duration of building effective immunity is right after the duration of recovery from the disease.
(vi) Recovered humans are still able to transmit the disease but at a lower rate.
(vii) Duration of latent period and immune period are constant.
(viii) Human and mosquito populations are not constant.

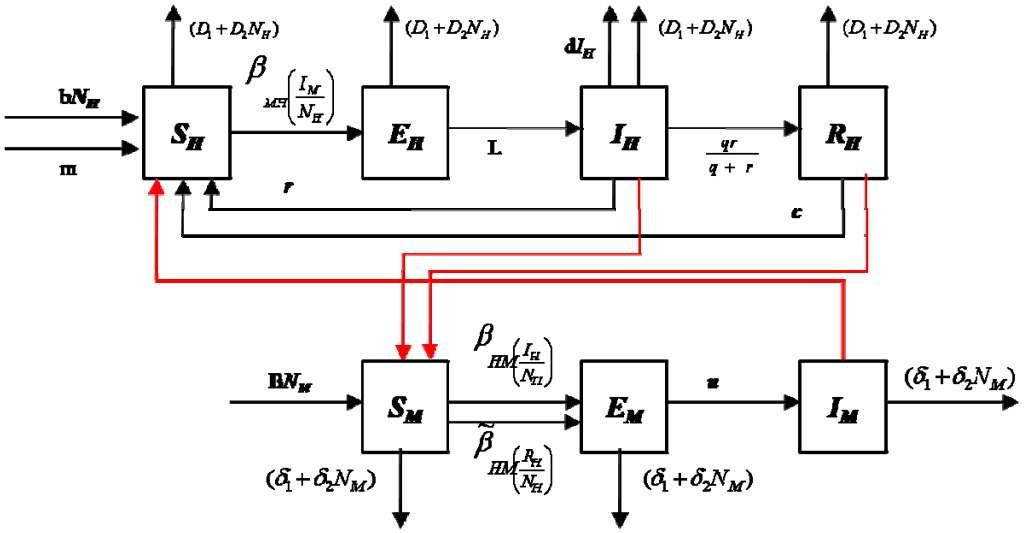

Figure 1: The compartmental model of the Malaria transmission of host human and vector mosquito

Following the compartmental model in Figure 1 and according to the Balance Law, the differential equations describing the transmission of the disease are as follows:

$$\frac{dS_H}{dt} = m + bN_H + cR_H - \beta_{MH}\left(\frac{I_M}{N_H}\right)S_H + rI_H - (D_1 + D_2N_H)S_H \tag{1}$$

$$\frac{dE_H}{dt} = \beta_{MH}\left(\frac{I_M}{N_H}\right)S_H - LE_H - (D_1 + D_2N_H)E_H \tag{2}$$