

# MATHEMATICAL ANALYSIS OF MALARIA TRANSMISSION MODEL WITH NONLINEAR INCIDENCES

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## ABSTRACT

In this paper, an epidemic model with nonlinear incidences is proposed to describe the dynamics of malaria transmission. The stability of the system can be controlled by the threshold number  $\mathfrak{R}_0$  which governs the existence and stability of the endemic equilibrium. It is found that the disease-free equilibrium point is locally asymptotically stable when  $\mathfrak{R}_0 \leq 1$ . For  $\mathfrak{R}_0 > 1$ , the disease-free equilibrium becomes unstable and the endemic equilibrium is locally asymptotically stable using the general theory of competitive system and compound matrices. Numerical results are shown that the contribution of the nonlinear saturating incidence provides important guidelines for accessing control of malaria diseases.

Keywords—Malaria, Nonlinear incidence, Basic reproduction number, Stability

## 1. INTRODUCTION

Malaria remains a major cause of mortality and morbidity in the tropical and subtropical areas of the world. According to WHO (2003), around 36% of the global population is at constant risk of infection, with sub-Saharan Africa being the worst affected region. Each year, an estimated 300 to 500 million clinical cases of malaria occur, making it one of the common infectious diseases worldwide (Bloland, 2001). Malaria infection is caused by the protozoan *Plasmodium*, and transmitted to humans by biting female *Anopheles* mosquitoes. Four species of the parasite (*P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*) infect humans. These species differ in geographical distribution, microscopic appearance, clinical features. Of the four species, *P. falciparum* is the most virulent, and potentially lethal to humans.

Mathematical models describing the population dynamics of infectious diseases have been playing an important role in a better understanding of epidemiological patterns and disease control for a long time. Since the mathematical models of malaria transmission have recently been reviewed and discussed elsewhere (Khasnis, 2005; Tumwiine, et al., 2007; Chiyaka, 2008; Tumwiine, et al., 2008). These researches assumed that the disease incidence rate is directly proportional to the densities of the susceptible host (human) and infected vector populations. However, actual

data and evidences observed for many diseases show that dynamics of disease transmission are not always as simple as shown in these models. Thus, it has been suggested by several authors that the disease transmission process may have a nonlinear incidence rate. After studying the cholera epidemic spread in Bari in 1973, Capasso and Serio (1978) introduced a saturated incidence rate  $g(I)S$  into epidemic models, where  $g(I)$  tends to a saturation level when  $I$  gets large, i.e.,  $g(I) = \beta I / (1 + \alpha I)$ , where  $\beta I$  measures the infection force of the disease and  $1/(1 + \alpha I)$  measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. This incidence rate seems more reasonable than the bilinear incidence rate  $g(I)S = \beta IS$ , because it includes the behavioral change and crowding effect of the infective individuals and prevents the unbounded of the contact rate by choosing suitable parameters.

In this paper, the model proposed by Tumwiine, et al., (2007) and Cai and Li (2010) is modified incorporating the effect of nonlinear incidences to gain some insights into the transmission dynamics of malaria in the population. The paper is organized as follows. The model is formulated in Section 2. In Section 3, conditions for the existence of equilibria are derived and the stability of the model is analyzed by using the general theory of competitive system and compound matrices. Numerical simulations are reported in Section 4.

## 2. MODEL FORMULATION

In this paper, a model for malaria is formulated in the human and mosquito population. The total human population at time  $t$ , denoted by  $N_H(t)$ , is divided into three sub-populations of susceptible humans  $S_H(t)$ , infectious humans  $I_H(t)$  and recovered humans  $R_H(t)$ , so that  $N_H(t) = S_H(t) + I_H(t) + R_H(t)$ . Similarly, the total vector population at time  $t$ , denoted by  $N_V(t)$ , is split into susceptible mosquito vectors  $S_V(t)$ , infectious mosquito vectors  $I_V(t)$ , so that  $N_V(t) = S_V(t) + I_V(t)$ . All parameters are assumed positive. Thus, the model for transmission dynamics of malaria is formulated by the following system of differential equations:

$$S'_H = \Pi_H - \frac{\beta_H I_V S_H}{1 + \alpha_H I_V} + \nu I_H + \gamma R_H - \mu_H S_H, \quad (1)$$

$$I'_H = \frac{\beta_H I_V S_H}{1 + \alpha_H I_V} - (\nu + m + \mu_H) I_H, \quad (2)$$

$$R'_H = m I_H - (\gamma + \mu_H) R_H, \quad (3)$$

$$S'_V = \Pi_V - \frac{\beta_V I_H S_V}{1 + \alpha_V I_H} - \mu_V S_V, \quad (4)$$

$$I'_V = \frac{\beta_V I_H S_V}{1 + \alpha_V I_H} - \mu_V I_V, \quad (5)$$

where  $\Pi_H$  is the recruitment of humans into the population,  $\beta_H$  be the transmission rate from vector to human and  $\mu_H$  is the natural death rate of humans. Infectious humans recover at a rate  $m$  and recover from the disease at the rate  $\nu$ . It is assumed that recovered individuals loss immunity and move into the  $S_H$  class at the rate  $\gamma$ . The susceptible vector population is generated by birth at a rate  $\Pi_V$ . This population is reduced by infection, following effective contact with infectious humans, at the rate  $\beta_V$  and natural death at a rate  $\mu_V$ . Infectious vectors die at a rate  $\mu_V$ . The incidence terms for human and vector populations are given, respectively,  $\beta_H I_V S_H / (1 + \alpha_H I_V)$  and  $\beta_V I_H S_V / (1 + \alpha_V I_H)$  where  $\alpha_H$  and  $\alpha_V$  determine the level at with the force of infection saturates. The incidence function forms reflect a saturating effect of diseases transmission. Both are generalizations the contact rates of the Ross-Macdonald model for Malaria (Ross, 1911).

### 3. STABILITIES OF THE EQUILIBRIUM POINTS

#### 3.1 Local stability of disease-free equilibrium

The existence of equilibria of the system can be investigated by setting the right-hand side of (1)-(5) to zero. The model has disease-free equilibrium given by

$$E_0 = (\Pi_H / \mu_H, 0, 0, \Pi_V / \mu_V, 0).$$

The linear stability of  $E_0$  is determined by using the next generation matrix method (Driessche, et al., 2002) on system (1)-(5). The matrices,  $F$  (for the new infection terms) and  $V$  (for the transition terms) are giving by

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_H \Pi_H}{\mu_H} \\ 0 & 0 & 0 \\ \frac{\beta_V \Pi_V}{\mu_V} & 0 & 0 \end{bmatrix} \text{ and } V^{-1} = \begin{bmatrix} Q & 0 & 0 \\ -m & \gamma + \mu_H & 0 \\ 0 & 0 & \mu_V \end{bmatrix}.$$

It follows that the basic reproductive number, denoted by  $\mathfrak{R}_0$ , is given by

$$\mathfrak{R}_0 = \rho(FV^{-1}) = \sqrt{\beta_H \Pi_H \beta_V \Pi_V / (\mu_H \mu_V^2 Q)},$$

where  $Q = \nu + m + \mu_H$  and  $\rho$  is the spectral radius (dominant eigenvalue is magnitude) of the next generation matrix  $FV^{-1}$ . Hence, using Theorem 2 of (Driessche, et al., 2002), the following result is established:

**Lemma 1.** The disease-free equilibrium,  $E_0$ , is locally asymptotically stable if  $\mathfrak{R}_0 \leq 1$  and unstable if  $\mathfrak{R}_0 > 1$ .

### 3.2 Endemic equilibrium

#### 3.3.1 Existence of endemic equilibrium

The existence of endemic equilibrium of the model (1)-(5) is explored as follow.

$$\text{Let } \lambda_H^* = \beta_H I_V^* / (1 + \alpha_H I_V^*) \text{ and } \lambda_V^* = \beta_V I_H^* / (1 + \alpha_V I_H^*)$$

where  $*$  represents the component of the endemic equilibrium at steady state. Then the state variables of the model (1)-(5), can be expressed at an arbitrary endemic equilibrium, denoted by  $E^* = (S_H^*, I_H^*, R_H^*, S_V^*, I_V^*)$ .

Solving (1)-(4) gives the state variables of the model as:

$$S_H^* = \frac{\Pi_H Q P}{\lambda_H (QP - \nu P - \gamma m) + \mu_H Q P}, I_H^* = \frac{\lambda_H \Pi_H P}{\lambda_H (QP - \nu P - \gamma m) + \mu_H Q P}, \quad (6)$$

$$R_H^* = \frac{m \lambda_H \Pi_H}{\lambda_H (QP - \nu P - \gamma m) + \mu_H Q P}, S_V^* = \frac{\Pi_V}{\lambda_V + \mu_V}, I_V^* = \frac{\lambda_V \Pi_V}{\mu_V (\lambda_V + \mu_V)}$$

when  $P = \gamma + \mu_H$ .

Substituting the equations (6) into the expression for  $\lambda_H^*$  above, we consider only the case where  $\lambda_H^* \neq 0$ . It can be simplified to  $a_0 \lambda_H - a_1 = 0$  where  $a_0 = \mu_V \beta_H \Pi_H P + \mu_V^2 \omega + \alpha_V \Pi_H \beta_V \Pi_V P$  and  $a_1 = \mathfrak{R}_0^2 - 1$ . Thus, the linear equation has a unique positive solution when  $\mathfrak{R}_0 > 1$  (i.e.,  $\lambda_H^* > 0$  iff  $\mathfrak{R}_0 > 1$ ). These results are summarized below.

**Theorem 1.** The model (1)-(5), has a unique endemic equilibrium,  $E^*$ , if  $\mathfrak{R}_0 > 1$ , and no  $E^*$  if  $\mathfrak{R}_0 \leq 1$ .

#### 3.3.2 Stability of endemic equilibrium

In order to establish the local stability of  $E^*$ , adding equations (1)-(3) and (4)-(5) give the total population of human and vector, respectively. Thus,  $N_H(t) \rightarrow \Pi_H / \mu_H$  and  $N_V(t) \rightarrow \Pi_V / \mu_V$  as  $t \rightarrow \infty$ . Assuming that the size of human and vector populations have reached their limiting value, then eliminate  $R_H$  and  $S_V$  from the equations in (1) and (5) leads to the reduced three-dimensional model:

$$S'_H = \Pi_H - \frac{\beta_H I_V S_H}{1 + \alpha_H I_V} + \nu I_H + \gamma \left( \frac{\Pi_H}{\mu_H} - S_H - I_H \right) - \mu_H S_H, \quad (7)$$

$$I'_H = \frac{\beta_H I_V S_H}{1 + \alpha_H I_V} - (\nu + m + \mu_H) I_H, I'_V = \frac{\beta_V I_H}{1 + \alpha_V I_H} \left( \frac{\Pi_V}{\mu_V} - I_V \right) - \mu_V I_V.$$

The following lemma is used to prove the local stability of the endemic equilibrium  $E^*$  of the model (7).

**Lemma 2.** Let  $M$  be a  $3 \times 3$  real matrix and its second additive compound matrix is  $M^{[2]}$ . If  $\text{tr}(M)$ ,  $\det(M)$  and  $\det(M^{[2]})$ , are all negative, then all eigenvalues of  $M$  have negative real part.

**Theorem 2.** The unique endemic equilibrium  $E^*$  of the model (7)-(9) is locally asymptotically stable if  $\mathfrak{R}_0 > 1$ .

**Proof.** From the Jacobian matrix  $J(E^*)$ , it demonstrates that

$$\text{tr}(J(E^*)) = -[\sigma^* + Q + \delta^*] < 0 \text{ and}$$

$$\det(J(E^*)) = - \left[ \sigma^* \beta_H^* S_H^* \beta_V^* \Pi_V^* \left( 1 - \frac{\beta_H^* \beta_V^*}{\beta_H \beta_V} \right) + I_V^* \frac{(\beta_H^*)^3 (\beta_V^*)^2 S_H^* \Pi_V^*}{\beta_H \beta_V} \right] - [\sigma^* Q \beta_V^* I_H^* + \beta_H^* I_V^* (\gamma - \nu) \delta^*].$$

Similarly, the determinant of second additive compound matrix  $J^{[2]}(E^*)$  (Tumwiine, et al., 2007) is

$$\det(J^{(2)}(E^*)) = -(\sigma^* + Q) \left[ (\delta^* + Q)(\sigma^* + \beta_H^* I_H^*) + (\gamma - \nu) \beta_H^* I_V^* \right] - (\delta^* + Q + 1) \beta_H^* S_H^* \beta_V^* \Pi_V^* \left( 1 - \frac{\beta_H^* \beta_V^*}{\beta_H \beta_V} \right) - \mu_V \delta^* \sigma^* - Q P \mu_V$$

with  $\beta_H^* = \frac{\beta_H}{1 + \alpha_H I_H^*}$ ,  $\beta_V^* = \frac{\beta_V}{1 + \alpha_V I_V^*}$ ,  $\Pi_V^* = \frac{\Pi_V}{\mu_V} - I_V^*$ ,  $\delta^* = \beta_V^* I_H^* + \mu_V$ ,  $\sigma^* = \beta_H^* I_V^* + P$ .

It is seen that  $\det(J(E^*)) < 0$  and  $\det(J^{(2)}(E^*)) < 0$  when  $\gamma > \nu$ . Hence, by lemma 2 and the Routh–Hurwitz criterion (Willems, 1970), all eigenvalues of  $J(E^*)$  have negative real parts whenever  $\mathfrak{R}_0 > 1$ . Thus  $E^*$  is locally asymptotically stable whenever it exists.

#### 4. NUMERICAL SIMULATIONS

To see the dynamical behavior of the model system, the system (1)–(5) is integrated numerically by fourth-order Runge–Kutta method using the parameters:  $\Pi_H = 2.5$ ,  $\beta_H = 0.01$ ,  $\mu_H = 4.089 \times 10^{-5}$ ,  $m = 0.14286$ ,  $\nu = 0.0038$ ,  $\gamma = 0.0146$ ,  $\Pi_V = 5000$ ,  $\beta_V = 0.02$ ,  $\mu_V = 0.14$ ,  $\alpha_H = 0.1$ ,  $\alpha_V = 0.2$  with initial values  $S_H(0) = 70000$ ,  $I_H(0) = 110$ ,  $R_H(0) = 0$ ,  $S_V(0) = 1000$ ,  $I_V(0) = 50$ .

The simulation results are shown in Figs. 1-4. Fig. 1 shows that  $S_H$  and  $S_V$  approach to its steady-state value while  $I_H$ ,  $R_H$  and  $I_V$  approach zero as time progresses, the disease dies out when  $\mathfrak{R}_0 < 1$ . Fig. 2 shows the stability of endemic equilibrium  $E^*$ , the disease becomes endemic when  $\mathfrak{R}_0 > 1$ . These verify that the asymptotic behavior of the model is determined by its basic reproduction number,  $\mathfrak{R}_0$ . It is also verified that, from Fig. 2,  $\mathfrak{R}_0$  is proportional to the total number of the human population available as blood sources for the mosquitoes and the carrying capacity of the mosquito population, respectively. This means that in the urban centers, where alternative hosts are scarce, the probability of disease contact in the human population is higher than in the rural regions. Therefore, it is not surprising that mosquitoes that are adapted to urban environments such as the *Aedes* mosquitoes have become a major public health problem.

The significance of  $\alpha_H$  and  $\alpha_V$  in the nonlinear incidence terms is investigated using various values of  $\alpha_H$  and  $\alpha_V$  respectively. The results are depicted in Figs. 3-4. It is observed that although the stability results do not depend directly on the parameters  $\alpha_H$  and  $\alpha_V$ , a decrease in  $\alpha_H$  and  $\alpha_V$  produces an increase in the equilibrium level of the infectious humans (see Fig. 3) and infectious vectors (see Fig. 4), respectively. Thus, this is expected that  $\alpha_H$  and  $\alpha_V$  are variable that control the magnitude of the infectious individuals.

#### CONCLUSION AND DISCUSSION

In this paper, a deterministic mathematical model for the transmission dynamics of malaria is qualitatively and quantitatively studied. The local stability analysis of the model reveals that when  $\mathfrak{R}_0 < 1$ , the disease-free equilibrium is locally asymptotically stable. When  $\mathfrak{R}_0 > 1$ ,

the endemic equilibrium exists and is locally asymptotically stable. Though  $\mathfrak{R}_0$  does not depend on the force of infection saturates,  $\alpha_H$  and  $\alpha_V$ , numerical simulations indicate that when the disease is endemic, the steady state values of the infectious humans and infectious vectors decrease as  $\alpha_H$  and  $\alpha_V$  increases. This implies that if the social consciousness about the disease decreases among the susceptible, it might encourage to increase the infection rate and to spread the disease rapidly.

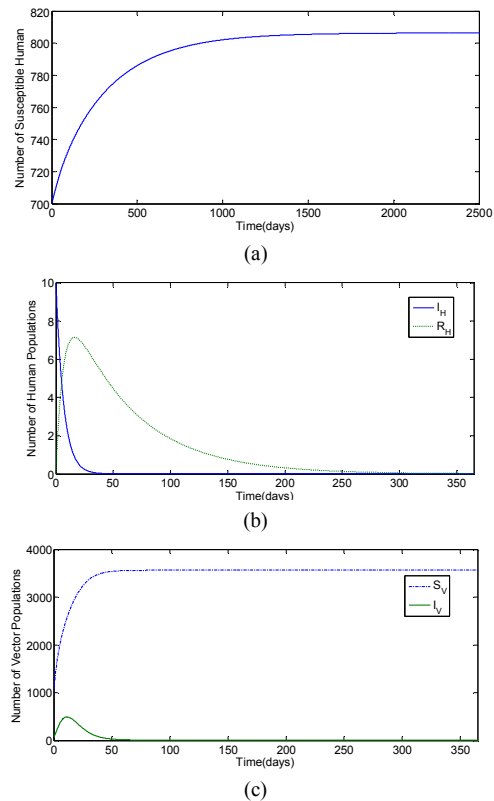


Fig. 1 Numerical solution of model (1)-(5) when  $\mathfrak{R}_0 < 1$ , for  $\beta_H = 4.9897 \times 10^{-7}$ .

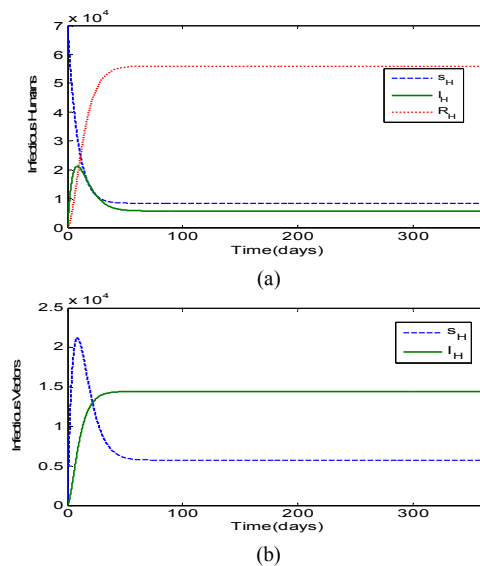
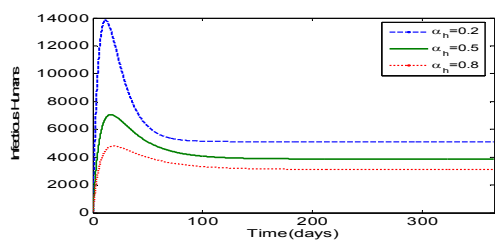
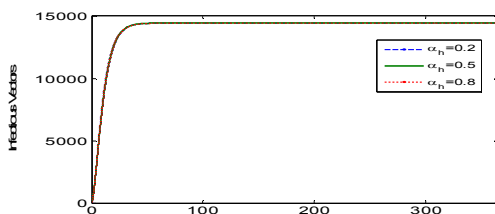


Fig. 2 Numerical solution of model (1)-(5), when  $\mathfrak{R}_0 > 1$ .

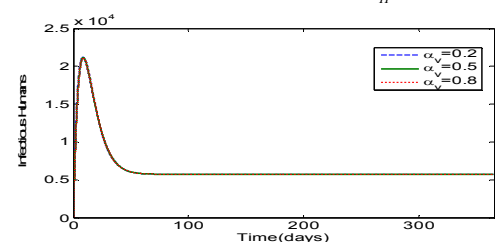


(a)

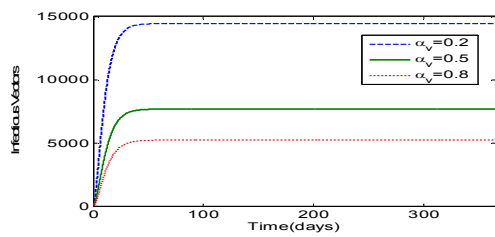


(b)

Fig. 3 Variation of infected human population with time for different values of  $\alpha_H$ .



(a)



(b)

Fig. 4 Variation of infected human population with time for different values of  $\alpha_V$ .

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