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# MATHEMATICAL MODEL FOR MALARIA TRANSMISSION DYNAMICS IN HUMAN AND MOSQUITO POPULATIONS WITH NONLINEAR FORCES OF INFECTION

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**Abstract:** This paper presents a seven-dimensional ordinary differential equation modelling the transmission of *Plasmodium falciparum* malaria between humans and mosquitoes with non-linear forces of infection in form of saturated incidence rates. These incidence rates produce antibodies in response to the presence of parasite-causing malaria in both human and mosquito populations.The existence of region where the model is epidemiologically feasible is established. Stability analysis of the disease-free equilibrium is investigated via the threshold parameter (reproduction number $R_0$) obtained using the next generation matrix technique. The model results show that the disease-free equilibrium is asymptotically stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. The existence of the unique endemic equilibrium is also determined under certain conditions. Numerical simulations are carried out to confirm the analytic results and explore the possible behavior of the formulated model.

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## 1. Introduction

Malaria is one of the deadliest infectious diseases that has claimed million of lives around the world. Globally, 3.3 billion people or half of the world's population in 104 countries are at the risk of getting infected by malaria disease[1] and [2]. It has been estimated that between 300 and 500 million individuals of all ages are infected annually and between 1.5 and 2.7 million people die of malaria every year[3].

Malaria is widely spread in tropical and subtropical regions, including Africa, Asia, Latin America, the middle East and some parts of Europe. The most cases and deaths occur in sub-Saharan Africa. In particular, thirty countries in sub-Saharan Africa account for 90% of global malaria deaths[1]. Shockingly, the disease kills an african child every 30 seconds and over 2,000 young lives are lost daily across the globe[4] and [5]. For example, malaria accounts for 60% of outpatient visits and 30% of hospitalizations among children under five years of age in Nigeria[6].

The disease, malaria, which remains one of the most prevalent and lethal human infection world wide, is caused by infection with single-celled (protozoan) parasites of genus *Plasmodium* and is characterized by paroxysms of chills, fever, headache, pain and vomiting. The parasites are transmitted to humans through the bites of infected female *Anopheles* mosquitoes (vectors). Of the five parasite species (*Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, Plasmodium malariae* and *Plasmodium knowlesi*) that cause malaria in humans, *Plasmodium falciparum* is the most deadly form and it predominates in Africa[2]. The parasite is responsible for the greatest number of deaths and clinical cases in the tropics. Its infection can lead to serious complications affecting brain, lungs, kidneys and other organs[4].

Mathematical models for transmission dynamics of malaria are useful in providing better insights into the behavior of the disease.The models have played great roles in influencing the decision making processes regarding intervention strategies for preventing and controlling the insurgence of malaria. The study on malaria using mathematical modelling began in 1911 with Ronald Ross[7]. He introduced the first deterministic two-dimensional model with one variable representing humans and the other representing mosquitoes where it was shown that reduction of mosquito population below a certain threshold was sufficient to eradicate malaria. In [8], the Ross's model was modified by considering the latency period of the parasites in mosquitoes and their survival during that period. However, in this case, it was shown that reducing the number of mosquitoes is an inefficient control strategy that would have little effect on the

epidemiology of malaria in areas of intense transmission. Further extension was described by Anderson and May [9], where the latency of infection in humans was introduced by making the additional exposed class in humans. This modification further reduces the long term prevalence of both the infected humans and mosquitoes.

Thus, all other models that exist for malaria dynamics are developed from the three basic models explained earlier by incorporating different factors to make them biologically more realistic in explaining disease prevalence and prediction[10]. For examples, a number of epidemiological studies [11], [12] and [13] considered the inclusion of the recovered class which incorporates a time dependent immunity developed on recovery from infection in humans. Further work on acquired immunity in malaria has been conducted by Aron [14] and Bailey [15]. Their models take into account that acquired immunity to malaria depends on continuous exposure to reinfection. Moreover, some models have integrated other factors such as: environmental effects [16], [17] and [18]; mosquito's resistance to insecticides and resistance of some parasite strains to anti-malaria drugs [5]

Incidence rate is of utmost importance in the transmission dynamics of the disease, as the qualitative behavior of the disease depends on it. The incidence of malaria infection is referred to as the number of new infected individuals (humans or mosquitoes) yielding in unit time [19]. The most commonly used incidence rates in the formulation of models for malaria transmission are simple mass-action and standard incidences. For other forms of incidence functions that arise in epidemiological models, see [20] and [21].

The model presented in this work consists of four compartments in humans and three compartment in mosquitoes, with inclusions of nonlinear forces of infection in form of saturated incidence rates in both the host and vector populations. The disease-induced death rates for humans and mosquitoes are also incorporated into the the model. We study the effects of these inclusions on the behavior of the formulated model.

The rest of this work is organized as follows: we give a full description of the model and show a domain where the model is epidemiologically well-posed in Section 2. Section 3 provides the existence of equilibria including a derivation of the basic reproduction number and stability analysis of the equilibria. In Section 4, we perform numerical simulations of the model with graphical illustrations and give concluding remarks in Section 5.

## 2. Model Formulation

To study the transmission and spread of malaria in two interacting population of humans (the host) and mosquitoes (the vector), we formulate a model which subdivides the total human population size at time $t$, denoted by $N_h(t)$, into susceptible humans $S_h(t)$, exposed humans $E_h(t)$, infectious humans $I_h(t)$ and recovered humans $R_h(t)$. Hence, we have

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t).$$

Unlike human population, we divide the mosquito population into three subclasses: susceptible mosquitoes $S_m(t)$, exposed mosquitoes $E_m(t)$ and infectious mosquitoes $I_m(t)$. The mosquitoes remain infectious for life and have no recovered class [22] and [23]. Thus, the total size of the mosquito population at any time $t$ is denoted by

$$N_m(t) = S_m(t) + E_m(t) + I_m(t).$$

Susceptible individuals are recruited into the human population by input rate $\Lambda_h$. Using the ideas in Ibezim and Odo [24], an infectious female *anopheles* mosquito, $I_m$, usually attacks susceptible human $S_h(t)$ by piercing and sucking using its proboscis, with which it introduces an enzyme from the saliva into human's bloodstream in order to inhibit blood clotting while sucking. In the process, the parasite (in the form of sporozoites) is injected into the blood and the susceptible human moves to the exposed class $E_h(t)$. 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 the susceptible mosquitoes [25].

From the blood of the exposed human, the parasite goes into the liver for cell division and multiplication before being released into the blood again as merozoites. At this stage, the exposed human becomes infectious $I_h(t)$. After some time, the infectious human recovers and moves to the recovered class $R_h(t)$. However, the recovered human has some immunity to the disease for some period of time and later loses the immunity to become susceptible again. Every class of human population is decreased by natural death or through emigration except for the infectious class which has a per capital disease-induced death rate $\delta_h$ as an addition.

In a similar manner, when a susceptible mosquito $S_m(t)$ bites an infectious human, the parasite (in the form of a gametocytes) enters the mosquito with some probability, $\beta_m$, and the mosquito moves from the susceptible to the

exposed class $E_m(t)$. The exposed mosquito then becomes infectious and enters the class $I_m(t)$ after a given time. Mosquitoes leave the population through natural death rate $\mu_m$ or through a disease-induced death rate $\delta_m$.

The compartmental model which shows the mode of transmission of malaria between the two interacting populations is depicted in the figure below: The

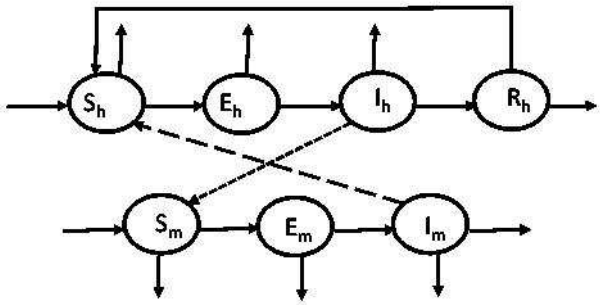


Figure 1: The compartmental model of malaria parasite transmission between humans and mosquitoes.

state variables and parameters used for the transmission model are described in the following tables:

| | |
|---|---|
| $S_h(t)$ | Number of host humans susceptible to malaria infection at time $t$ |
| $E_h(t)$ | Number of host humans exposed to malaria infection at time $t$ |
| $I_h(t)$ | Number of infectious host humans at time $t$ |
| $R_h(t)$ | Number of recovered host humans at time $t$ |
| $S_m(t)$ | Number of susceptible mosquitoes at time $t$ |
| $E_m(t)$ | Number of exposed mosquitoes at time $t$ |
| $I_m(t)$ | Number of infectious mosquitoes at time $t$ |

Table 1: Description of state variables.

In what follows, we obtain a seven-dimensional system of ordinary differential equations which describe the progress of the disease as:

$$\frac{dS_h}{dt} = \Lambda_h - \frac{b\beta_h S_h(t) I_m(t)}{1 + \nu_h I_m(t)} - \mu_h S_h(t) + \omega R_h(t) \tag{2.1}$$

$$\frac{dE_h}{dt} = \frac{b\beta_h S_h(t) I_m(t)}{1 + \nu_h I_m(t)} - (\alpha_h + \mu_h) E_h(t) \tag{2.2}$$

| | |
|---|---|
| $\Lambda_h$ | Recruitment term of the susceptible humans |
| $\Lambda_m$ | Recruitment term of the susceptible mosquitoes |
| $b$ | Bitting rate of the mosquito |
| $\beta_h$ | Probability that a bite by an infectious mosquito results in transmission of disease to human |
| $\beta_m$ | Probability that a bite results in transmission of parasite to a susceptible mosquito |
| $\mu_h$ | Per capital death rate of human |
| $\mu_m$ | Per capital death rate of mosquito |
| $\delta_h$ | Disease-induced death rate of human |
| $\delta_m$ | Disease-induced death rate of human |
| $\alpha_h$ | Per capital rate of progression of humans from the exposed state to the infectious state |
| $\alpha_m$ | Per capital rate of progression of mosquitoes from the exposed state to the infectious state |
| $r$ | Per capital recovery rate for humans from the infectious state to the recovered state |
| $\omega$ | Per capital rate of loss of immunity in humans |
| $\nu_h$ | Proportion of an antibody produced by human in response to the incidence of infection caused by mosquito |
| $\nu_m$ | Proportion of an antibody produced by mosquito in response to the incidence of infection caused by human |

Table 2: Description of model parameters.

$$\frac{dI_h}{dt} = \alpha_h E_h(t) - (r + \mu_h + \delta_h) I_h(t) \tag{2.3}$$

$$\frac{dR_h}{dt} = r I_h(t) - (\mu_h + \omega) R_h(t) \tag{2.4}$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{b\beta_m S_m(t) I_h(t)}{1 + \nu_m I_h(t)} - \mu_m S_m(t) \tag{2.5}$$

$$\frac{dE_m}{dt} = \frac{b\beta_m S_m(t) I_h(t)}{1 + \nu_m I_h(t)} - (\alpha_m + \mu_m) E_m(t) \tag{2.6}$$

$$\frac{dI_m}{dt} = \alpha_m E_m(t) - (\mu_m + \delta_m) I_h(t) \tag{2.7}$$

together with the initial conditions:

$$\left.\begin{array}{l} S_h(0) = S_{0h},\ E_h(0) = E_{0h},\ I_h(0) = I_{0h}, R_h(0) = R_{0h} \\ S_m(0) = S_{0m},\ E_m(0) = E_{0m},\ I_m(0) = I_{0m} \end{array}\right\} \tag{2.8}$$

The term $b\beta_h S_h(t)I_m(t)$, a bilinear incidence used in [23], refers to the rate at which the susceptible human $S_h(t)$ gets infected by infectious mosquitoes $I_m(t)$. In this work, we use a saturated incidence of the form $\frac{b\beta_h S_h(t)I_m(t)}{1+\nu_h I_m(t)}$, where $\frac{1}{1+\nu_h I_m(t)}$ denotes a saturating feature that inhibits the force of infection from infectious mosquitoes to susceptible humans. In other words, it produces antibodies at $\nu_h \in [0,1]$ in response to the presence of antigens (in form of parasites) produced by infectious *anopheles* mosquitoes.

Moreover, $b\beta_m S_m(t)I_h(t)$ refers to the rate at which the susceptible mosquitoes $S_m(t)$ are infected by the infectious humans $I_h(t)$. Since mosquitoes have DNA like humans [26], they also develop antibodies against the malaria parasites [27]. Thus, we have a saturated force of infection of the form $\frac{b\beta_m I_h(t)}{1+\nu_m I_h(t)}$, where $\nu_m \in [0,1]$ is the rate at which antibodies are produced against the antigens contacted from infectious humans.

### 2.1. Existence and Positivity of Solutions

Here, we provide the following results which guarantee that the malaria model governed by system (2) is epidemiologically and mathematically well-posed in a feasible region $\mathcal{D}$ given by:

$$\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_m \subset \mathbb{R}_+^4 \times \mathbb{R}_+^3,$$

where

$$\mathcal{D}_h = \{(S_h, E_h, I_h, R_h) \in \mathbb{R}_+^4 : N_h \leq \frac{\Lambda_h}{\mu_h}\}$$

and

$$\mathcal{D}_m = \{(S_m, E_m, I_m) \in \mathbb{R}_+^3 : N_h \leq \frac{\Lambda_m}{\mu_m}\}.$$

**Theorem 2.1.** *There exists a domain $\mathcal{D}$ in which the solution set $\{S_h, E_h, I_h, R_h, S_m, E_m, I_m\}$ is contained and bounded.*

*Proof.* Given the solution set $\{S_h, E_h, I_h, R_h, S_m, E_m, I_m\}$ with positive initial conditions (2.8), we let

$$V_1(S_h, E_h, I_h, R_h) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$$

and

$$V_2(S_m, E_m, I_m) = S_m(t) + E_m(t) + I_m(t).$$

Then the time derivatives $V_1'$ and $V_2'$, along solutions of system (2) for humans and mosquitoes respectively, are obtained by

$$V_1' = \frac{\partial V_1}{\partial S_h}\frac{dS_h}{dt} + \frac{\partial V_1}{\partial E_h}\frac{dE_h}{dt} + \frac{\partial V_1}{\partial I_h}\frac{dI_h}{dt} + \frac{\partial V_1}{\partial R_h}\frac{dR_h}{dt}$$

and

$$V_2' = \frac{\partial V_2}{\partial S_m}\frac{dS_m}{dt} + \frac{\partial V_2}{\partial E_m}\frac{dE_m}{dt} + \frac{\partial V_2}{\partial I_m}\frac{dI_m}{dt}$$

It follows that $V_1' \leq \Lambda_h - \mu_h V_1$ and $V_2' \leq \Lambda_m - \mu_m V_2$. Solving the differential inequalities yields

$$V_1 \leq \frac{\Lambda_h}{\mu_h}(1 - exp(-\mu_h t)) + V_1(S_{0h}, E_{0h}, I_{0h}, R_{0h})exp(-\mu_h t)$$

and

$$V_2 \leq \frac{\Lambda_m}{\mu_m}(1 - exp(-\mu_m t)) + V_2(S_{0m}, E_{0m}, I_{0m})exp(-\mu_m t).$$

Consequently, taking the limits as $t \to \infty$ gives $V_1 \leq \frac{\Lambda_h}{\mu_h}$ and $V_2 \leq \frac{\Lambda_m}{\mu_m}$. Thus, all solutions of the humans population only are confined in the feasible region $\mathcal{D}_h$ and all solutions of the mosquitoes population are confined in $\mathcal{D}_m$. Showing that the feasible region for the formulated model (2) exists and is given by

$$\mathcal{D} = \left\{(S_h, E_h, I_h, R_h, S_m, E_m, I_m) \in \mathbb{R}_+^7 : N_h \leq \frac{\Lambda_h}{\mu_h}; N_m \leq \frac{\Lambda_m}{\mu_m}\right\}. \qquad \square$$

It remains to show that the solutions of system (2) are nonnegative in $\mathcal{D}$ for any time $t > 0$ since the model represents humans and mosquitoes populations.

**Theorem 2.2.** *The solutions $S_h, E_h, I_h, R_h, S_m, E_m, I_m$ of the malaria model (2) with nonnegative initial data (2.8) in the feasible domain $\mathcal{D}$, remain nonnegative in $\mathcal{D}$ for all $t > 0$.*

*Proof.* We will carry out the proof following ideas by [23] and [28]. It is easy to see that $S_h(t) > 0$ for all $t \geq 0$. If not, let there exists $t_* > 0$ such that $S_h(t_*) = 0, S_h'(t_*) \leq 0$ and $S_h, E_h, I_h, R_h, S_m, E_m, I_m > 0$ for $0 < t < t_*$. Then from (2.1) of the system (2), we have

$$\begin{aligned} S_h'(t_*) &= \Lambda_h - \tfrac{b\beta_h S_h(t_*) I_m(t_*)}{1+\nu_h I_m(t_*)} - \mu_h S_h(t_*) + \omega R_h(t_*) \\ &= \Lambda_h + \omega R_h(t_*) > 0, \end{aligned} \tag{2.9}$$

which is a contradiction. Hence $S_h(t) > 0$.

Assume that there exists $t_* = sup\{t > 0 : S_h, E_h, I_h, R_h, S_m, E_m, I_m > 0\}$. Then from (2.2) of the system (2) we have that

$$\frac{d}{dt}(E_h exp(\alpha_h + \mu_h)t) = \frac{b\beta_h S_h(t) I_m(t)}{1 + \nu_h I_m(t)} exp(\alpha_h + \mu_h)t. \tag{2.10}$$

Integrating (2.10) from 0 to $t_*$ we get

$$E_h(t_*)exp(\alpha_h + \mu_h)t_* - E_h(0) = \int_0^{t_*} \frac{b\beta_h S_h(\theta) I_m(\theta)}{1 + \nu_h I_m(\theta)} exp(\alpha_h + \mu_h)\theta d\theta, \tag{2.11}$$

multiplying both sides of (2.11) by $exp\{-(\alpha_h + \mu_h)t_*\}$, gives

$$\begin{array}{c} E_h(t_*) = E_h(0)exp\{-(\alpha_h + \mu_h)t_*\} + exp\{-(\alpha_h + \mu_h)t_*\} \\ \times \int_0^{t_*} \frac{b\beta_h S_h(\theta) I_m(\theta)}{1+\nu_h I_m(\theta)} exp(\alpha_h + \mu_h)\theta d\theta > 0. \end{array}$$

Hence $E_h(t) > 0$.

Next for $I_h(t)$, suppose $t_* > 0 \ni I_h(t_*) = 0$, and $I_h(t) > 0$ where $t \in [0, t_*)$. Then from (2.3) of system (2) we have

$$\frac{d}{dt}(I_h exp(r + \mu_h + \delta_h)t) = \alpha_h E_h(t) exp(r + \mu_h + \delta_h)t. \tag{2.12}$$

Integrating (2.12) from 0 to $t_*$ we obtain

$$\begin{array}{c} I_h(t_*) = I_h(0)exp\{-(r + \mu_h + \delta_h)t_*\} + exp\{-(r + \mu_h + \delta_h)t_*\} \\ \times \int_0^{t_*} \alpha_h E_h(\theta) exp(r + \mu_h + \delta_h)\theta d\theta > 0, \end{array},$$

which contradicts $I_h(t_*) = 0$. Similarly for $R_h(t)$, assume that there is some $t_* > 0$ such that $I_h(t_*) = 0$ and $I_h(t) > 0$. Then integrating (2.4) of the system (2) from 0 to $t_*$, we see that

$$\begin{array}{c} R_h(t_*) = R_h(0)exp\{-(\mu_h + \omega)t_*\} + exp\{-(\mu_h + \omega)t_*\} \\ \times \int_0^{t_*} r I_h(\theta) d\theta > 0, \end{array}$$

which contradicts $R_h(t_*) = 0$.

Assuming further that $S_m(t_*)$ is non-increasing and other variables are positive with $S_m(t) > 0$ for $t \in [0, t_*)$. It follows from (2.5) that

$$S'_m(t_*) = \Lambda_m - \frac{b\beta_m S_m(t_*) I_h(t_*)}{1 + \nu_m I_h(t_*)} - \mu_m S_m(t_*) > 0,$$

which is a contradiction. Hence there is no such time $t_*$ for which $S_m(t_*) = 0$.

Likewise for $E_m(t)$, one sees from (2.6) that

$$\frac{d}{dt}(E_m(t)exp(\alpha_m+\mu_m)t) = \frac{b\beta_m S_m(t)I_h(t)}{1+\nu_m I_h(t)}exp(\alpha_m+\mu_m)t. \tag{2.13}$$

Integrating (2.13) from 0 to $t_*$ for some $t_* > 0$ where $t \in [0, t_*)$ and such that $E_m(t_*) = 0$, we get

$$\begin{aligned} E_m(t_*) = E_m(0)exp\{-(\alpha_m+\mu_m)t_*\} + exp\{-(\alpha_m+\mu_m)t_*\} \\ \times \int_0^{t_*} \frac{b\beta_m S_m(\theta)I_h(\theta)}{1+\nu_m I_h(\theta)}exp(\alpha_m+\mu_m)\theta d\theta > 0, \end{aligned}$$

showing that $E_m(t) > 0$.

Finally, it is easy to see from (2.7) of the system (2) that

$$\frac{dI_m}{dt} \geq -\mu_m I_m(t)$$

and

$$I_m(t) \geq I_m(0)exp(-\mu_m t) \geq 0.$$

This completes the proof. □

## 3. Existence and Stability of Equilibrium Points

The points at which the differential equations of the system (2) equal to zero are referred to as equilibrium points or steady-state solutions. As shown in Theorem 2.2, it is important to note that there is no trivial equilibrium points as long as the recruitment terms $\Lambda_h$ and $\Lambda_m$ are not zero. This implies that $(S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*) \neq (0,0,0,0,0,0,0)$ and the populations will not be extinct.

### 3.1. Existence of Disease-free Equilibrium Point

Disease-free equilibrium points are steady-state solutions where there is no malaria infection. Thus, the disease-free equilibrium point, $E_0$, for the malaria model (2) implies that $E_h^* = 0, I_h^* = 0, R_h^* = 0, E_m^* = 0, I_m^* = 0$ and solving (2.1) and (2.5) yields $S_h^* = \frac{\Lambda_h}{\mu_h}$ and $S_m^* = \frac{\Lambda_m}{\mu_m}$ respectively. Thus we obtain

$$E_0 = (\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0).$$

### 3.2. Reproduction Number

An important notion in epidemiological models is the basic reproduction number, usually denoted by $R_0$. This number can be understood as the average number of secondary infectious infected by an infective individual during whose whole cause of disease in the case that all members of the population are susceptible [19]. It is an important parameter that gives us whether an infection will spread through the population or not.

To obtain $R_0$ for model (2), we use the next-generation matrix technique described in [29] and [30]. Let $x = (E_h, I_h, E_m, I_m, S_h, R_h, S_m)^T$. Then the model (2) can be written as
$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x)$, where

$$\mathcal{F}(x) = \begin{pmatrix} \frac{b\beta_h S_h I_m}{1+\nu_h I_m} \\ 0 \\ \frac{b\beta_m S_m I_h}{1+\nu_m I_h} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad and \quad \mathcal{V}(x) = \begin{pmatrix} (\alpha_h + \mu_h)E_h \\ (r + \delta_h + \mu_h)I_h - \alpha_h E_h \\ (\alpha_m + \mu_m)E_m \\ (\delta_m + \mu_m)I_m - \alpha_m E_m \\ \mu_h S_h - \Lambda_h - \omega R_h \\ (\mu_h + \omega)R_h - rI_h \\ \mu_m S_m - \Lambda_m \end{pmatrix}.$$

Finding the derivatives of $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium point $E_0$ gives $\mathbf{F}$ and $\mathbf{V}$ respectively. Where

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & 0 & b\beta_h \frac{\Lambda_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & b\beta_m \frac{\Lambda_m}{\mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$\mathbf{V} = \begin{pmatrix} \alpha_h + \mu_h & 0 & 0 & 0 \\ -\alpha_h & r + \mu_h + \delta_h & 0 & 0 \\ 0 & 0 & \alpha_m + \mu_m & 0 \\ 0 & 0 & -\alpha_m & \delta_m + \mu_m \end{pmatrix}$$

So that,

$$\mathbf{V^{-1}} = \begin{pmatrix} 0 & 0 & \frac{b\alpha_m\beta_h\Lambda_h}{\mu_h(\delta_m+\mu_m)(\alpha_m+\mu_m)} & \frac{b\beta_h\Lambda_h}{\mu_h(\delta_m+\mu_m)} \\ 0 & 0 & 0 & 0 \\ \frac{b\alpha_h\beta_m\Lambda_m}{\mu_m(r+\delta_h+\mu_h)(\alpha_h+\mu_h)} & \frac{b\beta_m\Lambda_m}{\mu_m(r+\delta_h+\mu_h)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number, $R_0 = \rho(\mathbf{FV^{-1}})$, is the spectral radius of the product $\mathbf{FV^{-1}}$. Hence, for the model (2), we arrive at

$$R_0 = \sqrt{\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{\mu_h(\alpha_h+\mu_h)(r+\delta_h+\mu_h)\mu_m(\delta_m+\mu_m)(\alpha_m+\mu_m)}}. \tag{3.1}$$

In (3.1), $\frac{\alpha_h}{(\alpha_h+\mu_h)}$ is the probability that a human will survive the exposed state to become infectious; $\frac{1}{(r+\delta_h+\mu_h)}$ is the average duration of the infectious period of the human; $\frac{\alpha_m}{(\alpha_m+\mu_m)}$ is the probability that a mosquito will survive the exposed state to become infectious and $\frac{1}{(\delta_m+\mu_m)}$ is state to become infectious; $\frac{1}{(r+\delta_h+\mu_h)}$ is the average duration of the infectious period of the mosquito.

Let the basic reproduction number,$R_0$, be written as

$$R_0 = \sqrt{R_h R_m}, \tag{3.2}$$

where $R_h = \frac{b\alpha_h\beta_h\Lambda_h}{\mu_h(\alpha_h+\mu_h)(r+\delta_h+\mu_h)}$ and $R_m = \frac{b\alpha_m\beta_m\Lambda_m}{\mu_m(\alpha_m+\mu_m)(r+\delta_m+\mu_m)}$. Here, $R_h$ describes the number of humans that one infectious mosquito infects over its expected infection period in a completely susceptible humans population, while $R_m$ is the number of mosquitoes infected by one infectious human during the period of infectiousness in a completely susceptible mosquitoes population.

### 3.3. Stability of the Disease-free Equilibrium Point

Using the basic reproduction number obtained for the model (2), we analyze the stability of the equilibrium point in the following result.

**Theorem 3.1.** *The disease-free state, $E_0$, is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

*Proof.* The Jacobian matrix of the system (2) evaluated at the disease-free equilibrium point $E_0$, is obtained as

$$J(E_0) = \begin{pmatrix} J_{11} & 0 & 0 & J_{14} & 0 & 0 & J_{17} \\ 0 & J_{22} & 0 & 0 & 0 & 0 & J_{27} \\ 0 & J_{32} & J_{33} & 0 & 0 & 0 & 0 \\ 0 & 0 & J_{43} & J_{44} & 0 & 0 & 0 \\ 0 & 0 & J_{53} & 0 & J_{55} & 0 & 0 \\ 0 & 0 & J_{63} & 0 & 0 & J_{66} & 0 \\ 0 & 0 & 0 & 0 & 0 & J_{76} & J_{77} \end{pmatrix},$$

where $J_{11} = -\mu_h, J_{17} = -\frac{b\beta_h\Lambda_h}{\mu_h}, J_{22} = -(\alpha_h+\mu_h), J_{27} = \frac{b\beta_h\Lambda_h}{\mu_h}, J_{32} = \alpha_h,$
$J_{33} = -(r+\delta_h+\mu_h), J_{43} = r, J_{44} = -(\mu_h+\omega), J_{53} = -\frac{b\beta_m\Lambda_m}{\mu_m}, J_{55} = -\mu_m, J_{63} =$

$\frac{b\beta_m\Lambda_m}{\mu_m}$,
$J_{66} = -(\alpha_m + \mu_m), J_{76} = \alpha_m$, and $J_{77} = -(\mu_m + \delta_m)$.
We need to show that all the eigenvalues of $J(E_0)$ are negative. As the first and fifth columns contain only the diagonal terms which form the two negative eigenvalues, $-\mu_h$ and $-\mu_m$, the other five eigenvalues can be obtained from the sub-matrix, $J_1(E_0)$, formed by excluding the first and fifth rows and columns of $J(E_0)$. Hence we have

$$J_1(E_0) = \begin{pmatrix} -(\alpha_h+\mu_h) & 0 & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h} \\ \alpha_h & -(r+\delta_h+\mu_h) & 0 & 0 & 0 \\ 0 & r & -(\mu_h+\omega) & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & 0 & -(\alpha_m+\mu_m) & 0 \\ 0 & 0 & 0 & \alpha_m & -(\mu_m+\delta_m) \end{pmatrix}.$$

In the same way, the third column of $J_1(E_0)$ contains only the diagonal term which forms a negative eigenvalue, $-(\mu_h + \omega)$. The remaining four eigenvalues are obtained from the sub-matrix

$$J_2(E_0) = \begin{pmatrix} -(\alpha_h+\mu_h) & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h} \\ \alpha_h & -(r+\delta_h+\mu_h) & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & -(\alpha_m+\mu_m) & 0 \\ 0 & 0 & \alpha_m & -(\mu_m+\delta_m) \end{pmatrix}.$$

The eigenvalues of the matrix $J_2(E_0)$ are the roots of the characteristic equation

$$(\lambda+\alpha_h+\mu_h)(\lambda+r+\delta_h+\mu_h)(\lambda+\alpha_m+\mu_m)(\lambda+\mu_m+\delta_m) - \frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{\mu_h\mu_m} = 0. \tag{3.3}$$

If we let $B_1 = \alpha_h + \mu_h, B_2 = r + \delta_h + \mu_h, B_3 = \alpha_m + \mu_m$, and $B_4 = \mu_m + \delta_m$, then (3.3) becomes

$$A_4\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 = 0, \tag{3.4}$$

where

$$\left.\begin{aligned} A_4 &= 1 \\ A_3 &= B_1 + B_2 + B_3 + B_4 \\ A_2 &= (B_1 + B_2)(B_3 + B_4) + B_1B_2 + B_3B_4 \\ A_1 &= (B_1 + B_2)B_3B_4 + (B_3 + B_4)B_1B_2 \\ A_0 &= B_1B_2B_3B_4 - \frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{\mu_h\mu_m} \end{aligned}\right\}. \tag{3.5}$$

Further manipulation of $A_0$ in terms of the reproduction number, $R_0$, yields

$$A_0 = B_1B_2B_3B_4(1 - R_0^2). \tag{3.6}$$

We employ the Routh-Hurwitz criterion, see [31] and [32], which states that all roots of the polynomial (3.4) have negative real parts if and only if the coefficients $A_i$ are positive and matrices $H_i > 0$, for $i = 0, 1, 2, 3, 4$. From (3.5), it is easy to see that $A_1 > 0, A_2 > 0, A_3 > 0, A_4 > 0$, since all $B_i's$ are positive. Moreover, if $R_0 < 1$, it follows from (3.6) that $A_0 > 0$. Also the Hurwitz matrices for the polynomial (3.4) are found to be positive. That is, $H_1 = A_3 > 0, H_2 = \begin{vmatrix} A_3 & A_4 \\ A_1 & A_2 \end{vmatrix} > 0, H_3 = \begin{vmatrix} A_3 & A_4 & 0 \\ A_1 & A_2 & A_3 \\ 0 & A_0 & A_1 \end{vmatrix} > 0$, and $H_4 = \begin{vmatrix} A_3 & A_4 & 0 & 0 \\ A_1 & A_2 & A_3 & A_4 \\ 0 & A_0 & A_1 & A_2 \\ 0 & 0 & 0 & A_0 \end{vmatrix} > 0.$

Therefore, all the eigenvalues of the Jacobian matrix $J(E_0)$ have negative real parts when $R_0 < 1$ and the disease-free equilibrium point is locally asymptotically stable.

However, when $R_0 > 1$, we see that $A_0 < 0$ and by Descartes'rule of signs [32], there is exactly one sign change in the sequence, $A_4, A_3, A_2, A_1, A_0$, of coefficients of the polynomial (3.4). So, there is one eigenvalue with positive real part and the disease-free equilibrium point is unstable. □

### 3.4. Existence of Endemic Equilibrium Point

Besides the disease-free equilibrium point, we shall show that the formulated model (2) has an endemic equilibrium point, $E_e$. The endemic equilibrium point is a positive steady state solution where the disease persists in the population.

**Theorem 3.2.** *The malaria model (2) has no endemic equilibrium when $R_0 < 1$ and a unique endemic equilibrium exists when $R_0 > 1$.*

*Proof.* Let $E_e = (S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, S_m^{**}, E_m^{**}, I_m^{**})$ be a nontrivial equilibrium of the model (2); i.e., all components of $E_e$ are positive. If we set all the

differential equations in (2) to zero, we get

$$\left.\begin{array}{rcl} S_h^{**} &=& \frac{((\alpha_m+\mu_m)(\mu_m+\delta_m)b\beta_m+\nu_h R_m)\Lambda_h I_h^{**}+\Lambda_h}{\mu_h R_0^2}, \\ E_h^{**} &=& \frac{(r+\delta_h+\mu_h)I_h^{**}}{\alpha_h}, \\ R_h^{**} &=& \frac{rI_h^{**}}{\mu_h+\omega}, \\ S_m^{**} &=& \frac{\Lambda_m}{\frac{b\beta_m I_h^{**}}{1+\nu_m I_h^{**}}+\mu_m}, \\ E_m^{**} &=& \frac{b\beta_m S_m^{**} I_h^{**}}{(1+\nu_m I_h^{**})(\alpha_m+\mu_m)}, \\ I_m^{**} &=& \frac{R_m I_h^{**}}{1+((\alpha_m+\mu_m)(\mu_m+\delta_m)b\beta_m+\nu_m)I_h^{**}}, \end{array}\right\} \tag{3.7}$$

and $I_h^{**}$ is a positive solution of a equation given by

$$C_1(I_h^{**})^2 + C_2 I_h^{**} + C_3 = 0, \tag{3.8}$$

with

$$\begin{array}{rcl} C_1 &=& \Lambda_h\Phi(\mu_h+\omega)(b\beta_h R_m + \mu_h\Phi - \omega r\mu_h R_0^2\Phi), \\ C_2 &=& \Lambda_h(\mu_h+\omega)(2\mu_h\Phi + b\beta_h R_m - \mu_h\Phi R_0^2) - \omega r\mu_h R_0^2\Phi, \\ C_3 &=& \Lambda_h\mu_h(\mu_h+\omega)(1-R_0^2), \end{array}$$

where $\Phi = (\alpha_m+\mu_m)(\mu_m+\delta_m)b\beta_m + \nu_m + \nu_h R_m$. For $C_1 > 0$ and $C_2 > 0$, when $R_0 < 1$, one sees that $C_3 > 0$. Then model (2) has no positive solution. However, when $R_0 > 1$, then $C_3 < 0$ and endemic equilibrium exists. This completes the proof. □

Numerical simulations in the next section also confirm the existence of an endemic equilibrium and its stability when $R_0 > 1$.

## 4. Numerical Results and Discussion

Here, we study numerically the behavior of the system (2) using some of the parameter values compatible with malaria [5], [23] and [33] as given in Table 3 and by considering initial conditions, $S_h(0) = 100$, $E_h(0) = 20$, $I_h(0) = 10$, $R_h(0) = 0$, $S_m(0) = 1000$, $E_m(0) = 20$, $I_m(0) = 30$.

The numerical simulations are conducted using Maple and the results are given in Figures 2-16 to illustrate the system's behavior for different values of model's parameters. Figures 2, 3, 4 and 5 show the varrying effects of the proportion of antibody on human population while keeping the remaining parameters in Table 3 unchanged and the reproduction number is less than unity. In particular, Figure 2 shows the behavior of the susceptible human population

| Parameters | Values |
|---|---|
| $\Lambda_h$ | 0.000215 |
| $\Lambda_m$ | 0.07 |
| $b$ | 0.12 |
| $\beta_h$ | 0.1 |
| $\beta_m$ | 0.09 |
| $\mu_h$ | 0.0000548 |
| $\mu_m$ | 1/15 |
| $\delta_h$ | 0.001 |
| $\delta_m$ | 0.01 |
| $\alpha_h$ | 1/17 |
| $\alpha_m$ | 1/18 |
| $r$ | 0.05 |
| $\omega$ | 1/730 |

Table 3: Model parameters and values used in simulation.

as the antibody, $\nu_h$, increases in proportion. We observe that the susceptible human population drops as a result of infection by infectious mosquito and later stabilizes when the human develops an antibody against the parasite-causing malaria. An increase in the proportion of the antibody reduces the sharp decrease in the susceptible human population. The magnitudes of the exposed and infectious human populations in Figures 3 and 4 respectively, decrease with increased presence of antibody. Thus, the decreased number of infectious human population contributes to the increase in the number of recovered human in Figure 5, which in turn influences the reduction in the sharp decrease experienced by susceptible human population.

Similarly, Figures 6, 7 and 8 depict the varring effects of the proportion of antibody, $\nu_m$, produced against parasite, on mosquito populations. As seen in Figure 6, the number of susceptible mosquito decreases with time since there is no recovered class for mosquito population. However, increasing the proportion of antibody, $\nu_m$, inhibits the reduction in the number of susceptible mosquito. Also, in Figures 7 and 8, the number of the exposed and infectious mosquito populations decreases due to increase in resistance to the malaria parasite. Figures 9-12 assess the impact of the antibody produced by susceptible human in response to the presence of the parasite, when the reproduction number is greater than unity with an increased number of mosquito's biting rate, $b$. We see that increasing the proportion of the antibody with the biting rate, has lower effect in reducing the burden of the endemic malaria infection when compared

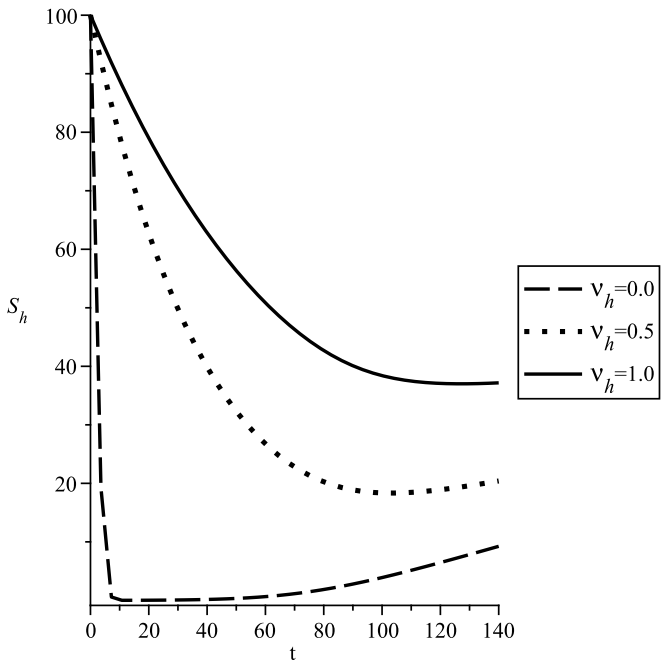


Figure 2: The behavior of susceptible human for different values of $\nu_h$ when $R_0 < 1$ and $b = 0.12$.

with Figures 2 to 5.

Figure 13 illustrates the effect of the mosquito's resistance to the endemic disease on infectious mosquito population when the human-mosquito contact rate, $b$, is increased. We notice that the number of infectious mosquito decreases slowly with increased propportion of antibody, $\nu_m$. This observation can be seen clearly when compared with Figure 8, as the number of infectious mosquito decreases faster with increased propportion of antibody, $\nu_m$ in Figure 8.

We further investigate the impact of the disease-induced death rate of the infectious mosquito, $\delta_m$, on susceptible human, infectious human and infectious mosquito poppulations in Figures 14, 15 and 16 respectively. It is observed that increasing the disease-induced death rate of mosquito, inhibits the reduction in the number of susceptible human population and reduces the number of the infectious human population. This explains that when mosquito lacks the capacity to resist the parasite-causing malaria on biting an infectious human, its life expectancy is shortened as seen in Figure 16, making it impossible to cause further infection.

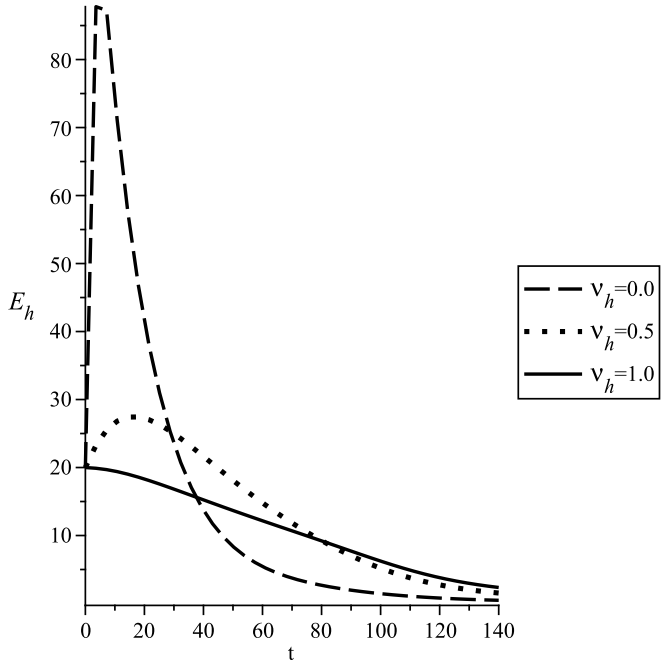


Figure 3: The behavior of exposed human for different values of $\nu_h$ when $R_0 < 1$ and $b = 0.12$.

## 5. Concluding Remarks

In this paper, we have formulated and analysed a compartmental model for malaria transmission in human and mosquito populations with non-linear forces of infection. The human population was divided into four compartments: susceptible, exposed, infectious and recovered, while the mosquito population was divided into three compartments: susceptible, exposed and infectious; since mosquitoes remain infectious for life and have no recovered class. The non-linear forces of infection in the formulated model present saturated incidence rates unlike the most commonly used simple mass-action and standard incidence rates. We established a region where the model is epidemiologically feasible and mathematically well-posed. The existence of a disease-free equilibrium point was shown.

Moreover, we employed the next generation matrix technique to obtain an explicit formula for a reproduction number, $R_0$, which is the expected number of secondary cases (in humans or mosquitoes) produced by a typical infectious

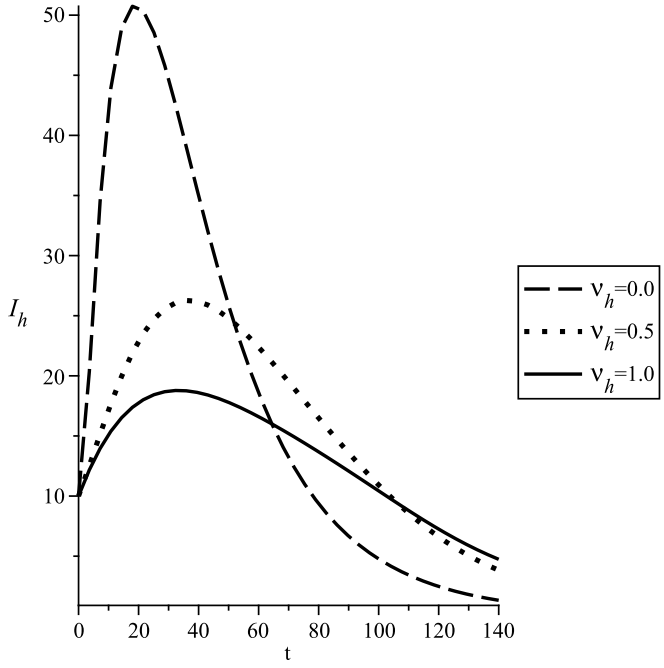


Figure 4: The behavior of infectious human for different values of $\nu_h$ when $R_0 < 1$ and $b = 0.12$.

individual (mosquito or human) during its entire period of infectiousness in a completely susceptible population. This number, $R_0$, represents a threshold parameter for the model. We proved that the disease-free equilibrium is asymptotically stable at threshold parameter less than unity and unstable at threshold parameter greater than unity. We further showed that a unique endemic equilibrium point exists when $R_0 > 1$.

When an antigen, a foreign substance in form of malaria parasite, invades the body, several types of cells work together to recognize and respond to it. These cells produce antibodies; which are a primary form of immune response in resistance to parasite-causing malaria, and act by attaching themselves to the parasite in order to weaken or destroy it. We showed that both human and mosquito develop antibodies through saturated incidence rates. The numerical simulations were performed to see the effects of the proportions of antibodies produced by both populations and other key parameters on the spread of the disease. Our results showed that increasing the proportions of antibodies has significant effect in reducing the transmission of the malaria infection. However,

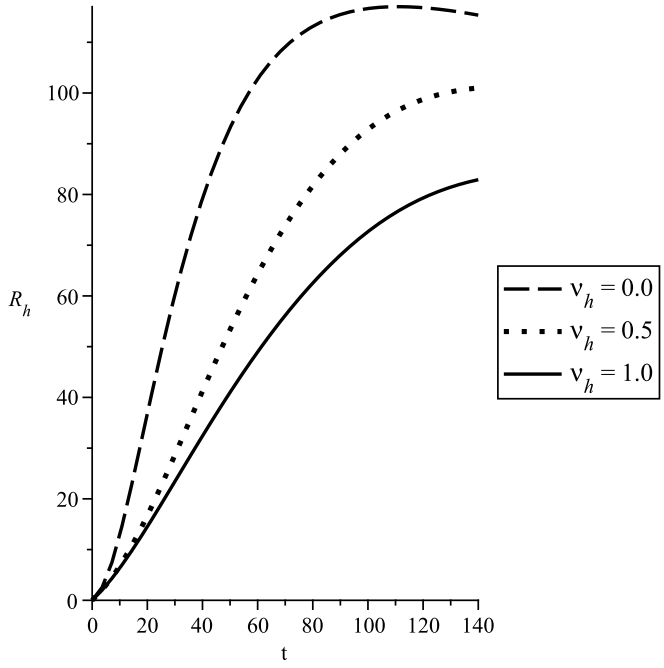


Figure 5: The behavior of recovered human for different values of $\nu_h$ when $R_0 < 1$ and $b = 0.12$.

with increased mosquito biting rate, increasing the proportions of antibodies has lower effect in reducing the burden of malaria. We also revealed that an increase in the number of disease-induced death rate of the mosquito reduces the number of infectious human.

In view of the above, humans need to boost their antibodies production to be able to subdue the invasion of parasites in the bloodstream. The immunity state of the individual, that is, the general health and nutritional status of the infected individual, is a factor for preventing or aiding the occurrence of malaria. Thus, leading a healthy lifestyle and eating right foods can help boost the level of antibodies in humans. It is also important to note that reducing human-mosquito contact rate plays a big role in inhibiting the prevalence of malaria. Hence, we can achieve a malaria-free state by scaling down mosquito biting rate through; the regular indoor residual spraying with insecticides, the use of insecticide-treated bednets, closing of doors and windows against mosquitoes, clearing of stagnant water and drainages, and the use of mosquito repellant lotions, which are all regarded as vector control measures.

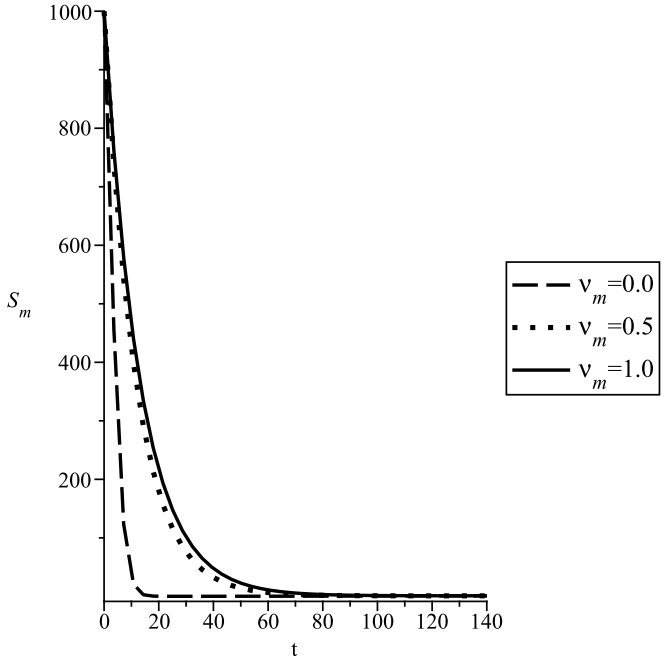


Figure 6: The behavior of susceptible mosquito for some values of $\nu_m$ when $R_0 < 1$ and $b = 0.12$.

Finally, Prophylaxis and chemotherapy have also been found to be very useful in preventing the occurrence of malaria and treating malaria attack respectively by the use of drugs. However, efforts should be intensified in developing malaria vaccine as this would facilitate the stimulation of the immune system in producing antibodies against malaria.

## 6. Acknowledgments

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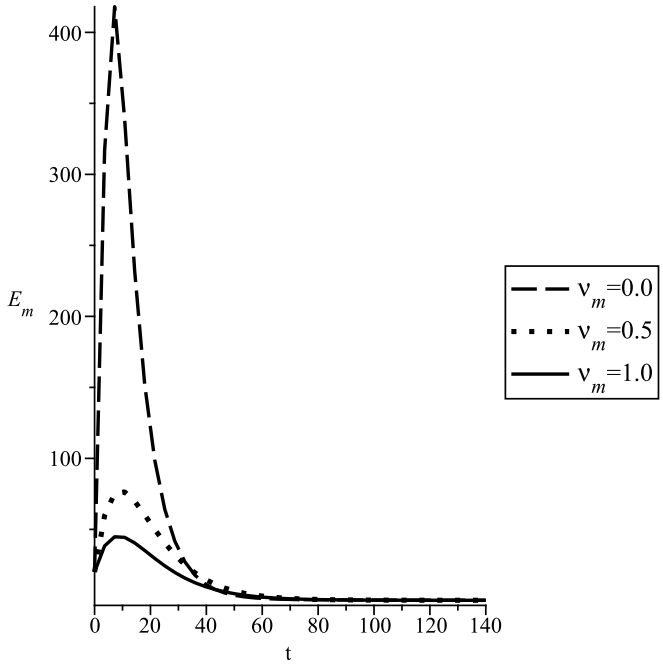


Figure 7: The behavior of exposed mosquito for different values of $\nu_m$ when $R_0 < 1$ and $b = 0.12$.

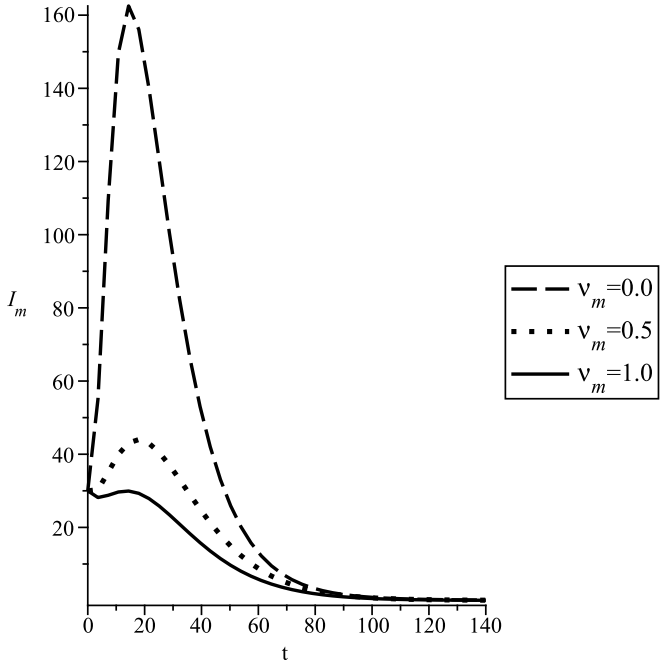


Figure 8: The behavior of infectious mosquito for different values of $\nu_m$ when $R_0 < 1$ and $b = 0.12$.

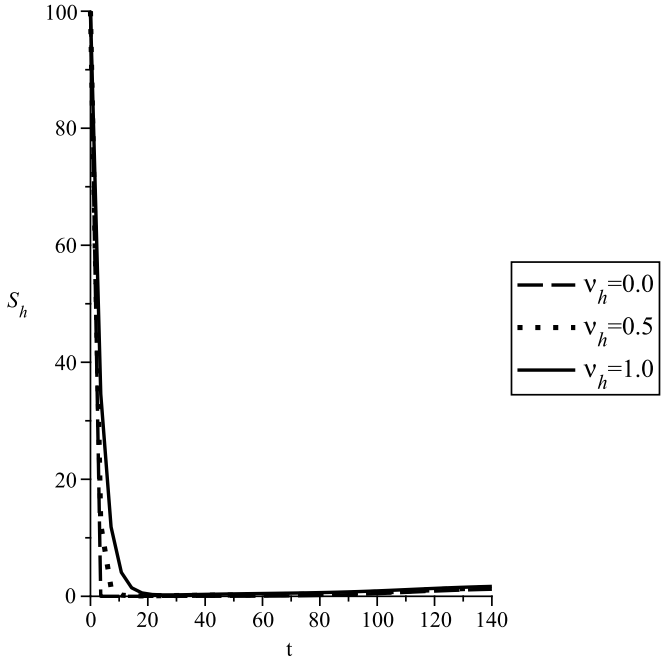


Figure 9: The behavior of susceptible human for different values of $\nu_h$ when $R_0 > 1$ and $b = 3$.

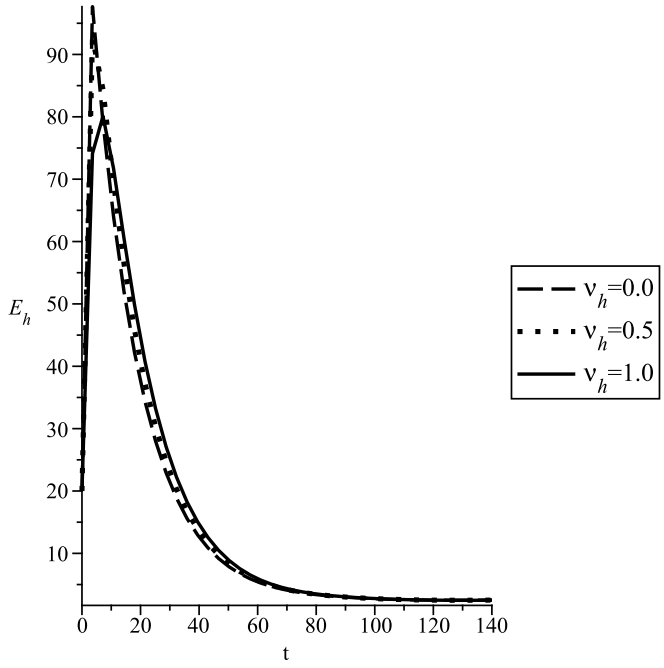


Figure 10: The behavior of exposed human for different values of $\nu_h$ when $R_0 > 1$ and $b = 3$.

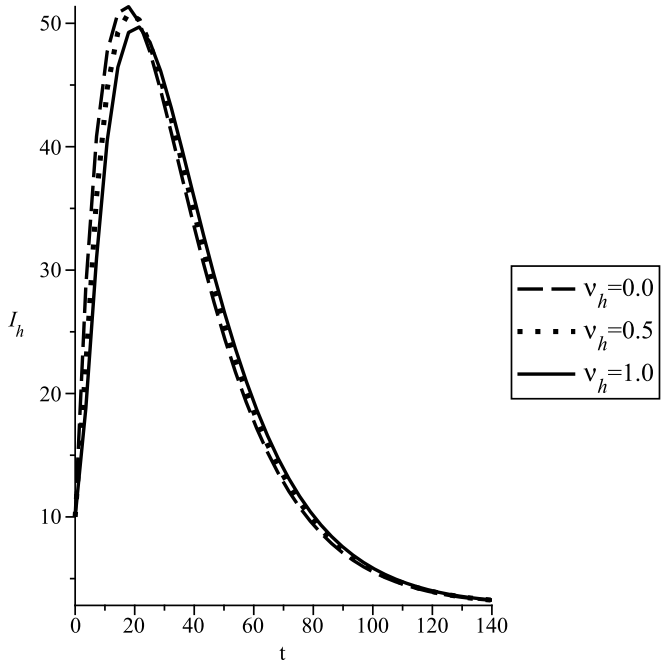


Figure 11: The behavior of infectious human for different values of $\nu_h$ when $R_0 > 1$ and $b = 3$.

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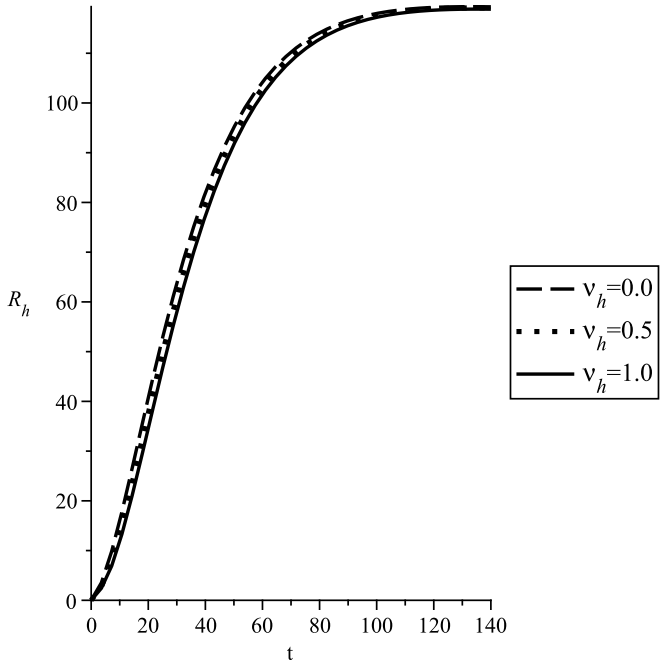


Figure 12: The behavior of recovered human for different values of $\nu_h$ when $R_0 > 1$ and $b = 3$.

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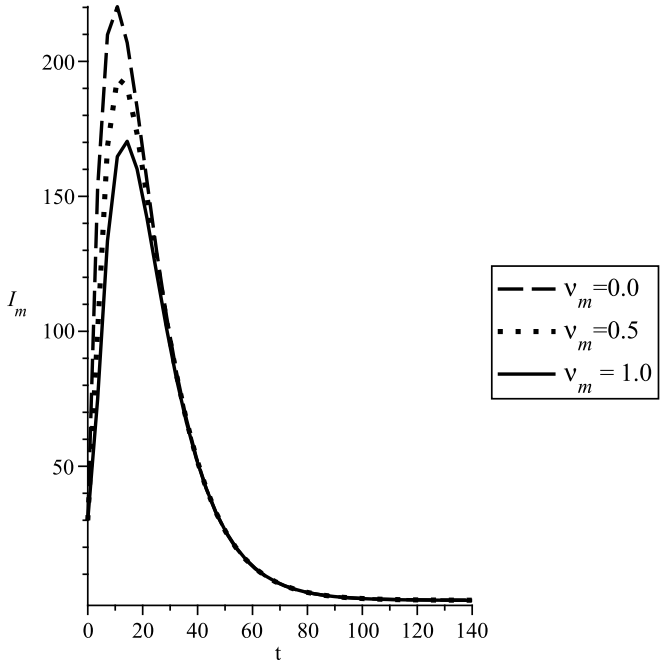


Figure 13: The behavior of infectious mosquito for different values of $\nu_m$ when $R_0 > 1$ and $b = 3$.

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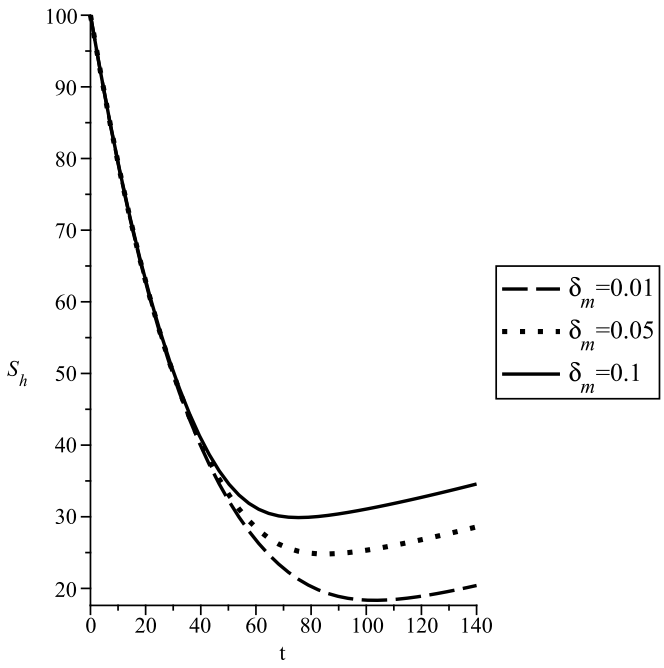


Figure 14: The behavior of susceptible human for different values of $\delta_m$ when $\nu_h = 0.5$ and $R_0 < 1$.

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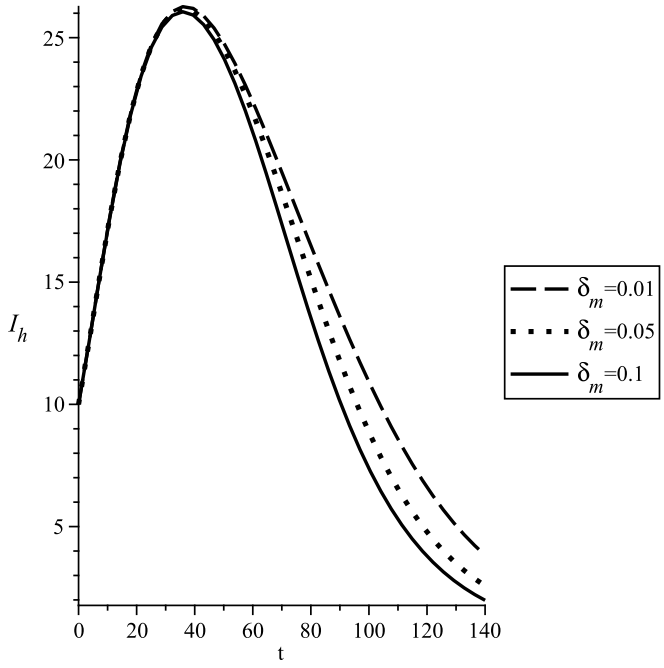


Figure 15: The behavior of infectious human for different values of $\delta_m$ when $\nu_h = 0.5$ and $R_0 < 1$.

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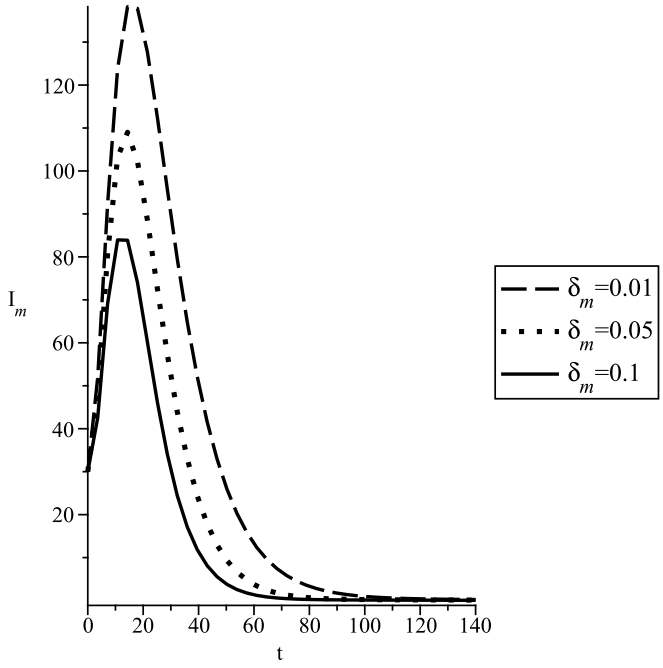


Figure 16: The behavior of infectious mosquito for some values of $\delta_m$ when $\nu_h = 0.5$ and $R_0 < 1$.

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