Qualitative Analysis of Malaria Dynamics with Nonlinear Incidence Function

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Abstract
A compartmental model for the transmission dynamics of malaria with nonlinear incidence function is presented and rigorously analysed. An explicit formula for the threshold parameter, known as the basic reproduction number, is used to determine the stability of the disease-free and endemic equilibria of the model. Using center manifold theory, the model is shown to exhibit a phenomenon of subcritical bifurcation whenever the threshold parameter crosses unity. Under specific conditions on the model parameters, the global dynamics of the model around the equilibria are explored using Lyapunov functions. For a threshold parameter less than unity, a globally asymptotically stable disease-free equilibrium is established while the endemic equilibrium is shown to be globally asymptotically stable at threshold parameter greater than unity. A sensitivity analysis is further carried out to investigate the impact of the model parameters on the transmission and spread of the disease.

Mathematics Subject Classification: 37N25, 92B05, 93A30, 93D20
Keywords: Malaria, Global stability, Lyapunov function, Bifurcation, Sensitivity analysis

1 Introduction

Among the known infectious diseases that are transmitted by vectors, malaria remains the most widespread and life-threatening disease in the world. In 2012, an estimated 3.4 billion people, about half of the world’s population, were at risk of this ancient foe called malaria and approximately 90% of the global malaria deaths occurred in the sub-Saharan African countries [23].

Malaria which is caused by infection with parasites of genus *Plasmodium* is transmitted to humans by female anopheles mosquitoes’ bites. Thus, to describe the malaria transmission dynamics using mathematical tools, two interacting populations of human and mosquito are needed. After the pioneering work of Ross [18], several mathematical models have been developed and analysed by incorporating more realistic features [1,5,11,13,14,15,24].

The import of the incidence of a disease in mathematical epidemiology cannot be overemphasized as the qualitative behaviour of the disease dynamics depends on it. This incidence refers to the number of new cases per unit time and depends on the product of the densities of the susceptibles and infectives [9]. The most commonly used incidence functions in the formulation of epidemiological models are the mass action (bilinear) incidence, $\beta SI$, and the standard (proportional) incidence, $\beta SI/N$. Where $\beta$ is the disease transmission probability and the numbers of susceptibles and infectives are denoted by $S$ and $I$ respectively.

However, transmission dynamics of some diseases are not always as simple as described by both mass action and standard incidence functions, because there are diverse biological mechanisms which may result in nonlinearities in the transmission rates [17]. Following [3], a number of authors (see, for instance, [2], [16], [17]) have employed a nonlinear incidence (saturated) function, $\frac{\beta SI}{1+\alpha I}$, to describe the transmission and spread of vector-borne diseases. The need for the saturated incidence in malaria model is borne out of the fact that the number of effective contact between susceptible and infectious individuals may saturate at high infective level due to the crowding of the infectious individuals (mosquitoes and humans) in the population or due to the precautionary measures (behavioural changes) exhibited by the susceptible individuals against the insurgence of the disease. One of such precautionary measures by the susceptible, as shown in [16], is the ability to develop resistance to the parasite by producing antibody in response to the manifestation of the disease.

In mathematical epidemiology, establishing global stability of dynamical systems around the equilibria using Lyapunov function is one of the most
taxing and vigorous research activities. The qualitative analysis carried out in this paper extends that in [16]. Apart from exploring the global dynamical behaviour of the malaria model, we first investigate the phenomenon of a backward bifurcation using the center manifold theory. In addition, we perform the sensitivity analysis of the model to determine how the parameters contribute to the transmission and spread of the disease with respect to the basic reproduction number of the model.

2 Malaria Model

In this section we consider the seven-dimensional system of ordinary differential equations modelling the interaction of human and mosquito populations with nonlinear incidence functions studied in [16] as follows:

\[
\begin{align*}
\frac{dS_h}{dt} &= \Lambda_h - b\beta_h S_h I_m \frac{1}{1 + \nu_h I_m} - \mu_h S_h + \omega R_h \\
\frac{dE_h}{dt} &= b\beta_h S_h I_m \frac{1}{1 + \nu_h I_m} - (\alpha_h + \mu_h) E_h \\
\frac{dI_h}{dt} &= \alpha_h E_h - (r + \mu_h + \delta_h) I_h \\
\frac{dR_h}{dt} &= r I_h - (\mu_h + \omega) R_h \\
\frac{dS_m}{dt} &= \Lambda_m - b\beta_m S_m I_h \frac{1}{1 + \nu_m I_h} - \mu_m S_m \\
\frac{dE_m}{dt} &= b\beta_m S_m I_h \frac{1}{1 + \nu_m I_h} - (\alpha_m + \mu_m) E_m \\
\frac{dI_m}{dt} &= \alpha_m E_m - (\mu_m + \delta_m) I_m
\end{align*}
\]

(1)

To avoid re-inventing the wheel, the variables of the model have been shown to be nonnegative, noting that the model parameters are all positive except \( \delta_h \) and \( \delta_m \) that are nonnegative. Hence, the model (1) will be further analysed in the positively-invariant region defined by

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

where \( N_h \) and \( N_m \) are the total human population and total mosquito population respectively. The description of the variables and the model parameters are shown in the following table.
Table 1: The model variables and parameters

<table>
<thead>
<tr>
<th>Variable</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$S_h(t)$</td>
<td>Number of host humans susceptible to malaria infection at time $t$</td>
</tr>
<tr>
<td>$E_h(t)$</td>
<td>Number of host humans exposed to malaria infection at time $t$</td>
</tr>
<tr>
<td>$I_h(t)$</td>
<td>Number of infectious host humans at time $t$</td>
</tr>
<tr>
<td>$R_h(t)$</td>
<td>Number of recovered host humans at time $t$</td>
</tr>
<tr>
<td>$S_m(t)$</td>
<td>Number of susceptible mosquitoes at time $t$</td>
</tr>
<tr>
<td>$E_m(t)$</td>
<td>Number of exposed mosquitoes at time $t$</td>
</tr>
<tr>
<td>$I_m(t)$</td>
<td>Number of infectious mosquitoes at time $t$</td>
</tr>
<tr>
<td>$\Lambda_h$</td>
<td>Recruitment term of the susceptible humans</td>
</tr>
<tr>
<td>$\Lambda_m$</td>
<td>Recruitment term of the susceptible mosquitoes</td>
</tr>
<tr>
<td>$b$</td>
<td>Biting rate of the mosquito</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>Probability that a bite by an infectious mosquito results in transmission of disease to human</td>
</tr>
<tr>
<td>$\beta_m$</td>
<td>Probability that a bite results in transmission of parasite to a susceptible mosquito</td>
</tr>
<tr>
<td>$\mu_h$</td>
<td>Per capita death rate of human</td>
</tr>
<tr>
<td>$\mu_m$</td>
<td>Per capita death rate of mosquito</td>
</tr>
<tr>
<td>$\delta_h$</td>
<td>Disease-induced death rate of human</td>
</tr>
<tr>
<td>$\delta_m$</td>
<td>Disease-induced death rate of human</td>
</tr>
<tr>
<td>$\alpha_h$</td>
<td>Per capita rate of progression of humans from the exposed state to the infectious state</td>
</tr>
<tr>
<td>$\alpha_m$</td>
<td>Per capita rate of progression of mosquitoes from the exposed state to the infectious state</td>
</tr>
<tr>
<td>$r$</td>
<td>Per capita recovery rate for humans from the infectious state to the recovered state</td>
</tr>
<tr>
<td>$\omega$</td>
<td>Per capita rate of loss of immunity in humans</td>
</tr>
<tr>
<td>$\nu_h$</td>
<td>Proportion of an antibody produced by human in response to the incidence of infection caused by mosquito</td>
</tr>
<tr>
<td>$\nu_m$</td>
<td>Proportion of an antibody produced by mosquito in response to the incidence of infection caused by human</td>
</tr>
</tbody>
</table>

2.1 Basic Reproduction Number

The basic reproduction number, $R_0$, defined as the average number of secondary infections caused by a typical infectious individual in a completely susceptible population, will be used to determine the overall dynamical behaviour of the model (1). Using the next generation operator method [22], $R_0$ can be obtained as

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

(2)

where

$$R_h = \frac{b \alpha_h \beta_h \Lambda_h}{\mu_h (\alpha_h + \mu_h)(r + \delta_h + \mu_h)}$$
Qualitative analysis of malaria dynamics

and

\[ R_m = \frac{b\alpha_m \beta_m \Lambda_m}{\mu_m (\alpha_m + \mu_m) (r + \delta_m + \mu_m)}. \]

With \( R_h \) describing the number of humans that one infectious mosquito infects over its expected infection period in a completely susceptible humans population, and \( R_m \) is the number of mosquitoes infected by one infectious human during the period of infectiousness in a completely susceptible mosquitoes population [16].

2.1.1 Disease-Free and Endemic Equilibria

The disease-free equilibrium of the model (1) exists and is given by

\[ E_0 = \left( \frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0 \right). \]

The following local stability result is established:

Lemma 2.1. The disease-free equilibrium point, \( E_0 \), is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

The implication of the above result is that the malaria disease can be eradicated from the population if the invasion by infectious individual is small enough so that \( R_0 < 1 \).

Further, in terms of \( I_h^* \), the endemic equilibrium of the model (1) exists and is given by

\[
\begin{align*}
S_h^* &= \frac{\Lambda_h [\mu_m + (b\beta_m + \mu_m \nu_m + \nu_h \mu_m R_m) I_h^*]}{\mu_h \mu_m R_0^2} \\
E_h^* &= \frac{(r + \mu_h + \delta_h) I_h^*}{\alpha_h} \\
R_h^* &= \frac{r I_h^*}{\mu_h + \omega} \\
S_m^* &= \frac{b\beta_m S_m I_h^*}{\nu_m + \mu_m I_h^* + \mu_m} \\
E_m^* &= \frac{(1 + \nu_m I_h^*) (\alpha_m + \mu_m)}{R_m \mu_m I_h^*} \\
I_m^* &= \frac{\mu_m + (b\beta_m + \mu_m \nu_m) I_h^*}{\mu_m + (b\beta_m + \mu_m \nu_m) I_h^*}
\end{align*}
\]

where

\[ I_h^* = \frac{\mu_h \mu_m \Lambda_h (\mu_h + \omega) (R_0^2 - 1)}{C} \]

and

\[ C = (\mu_h + \omega) [b\beta_h \Lambda_h \mu_m R_m + \Lambda_h \mu_h (b\beta_m + \mu_m \nu_m + \nu_h \mu_m R_m)] - \mu_h \mu_m \omega R_0^2. \]
Clearly in (5), no positive solution (endemic equilibrium) exists when $R_0 < 1$ and $C > 0$. Whereas, with $\omega = 0$; $\mathcal{C} > 0$, a unique endemic equilibrium exists when $R_0 > 1$.

It is noteworthy to remark that positive solution exists for the model (1) in a case where $\mathcal{C} < 0$ and $R_0 < 1$. This implies that the disease-free equilibrium co-exists with the endemic equilibrium state when $R_0$ crosses unity. This results into a phenomenon of subcritical (backward) bifurcation which is shown in the next section.

3 Bifurcation Analysis

Here we investigate the possibility of the co-existence of the equilibria of model (1) at $R_0$ slightly less than unity due to the transient immunity acquired by the recovered human, i.e. $\omega \neq 0$. To do this, the Center Manifold Theory of bifurcation analysis [4] which has been applied in some epidemic models [1,12,20] shall be used as follows.

Let the malaria model (1) be written in the vector form $\frac{dX}{dt} = G(X)$, where $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$ and $G = (g_1, g_2, g_3, g_4, g_5, g_6, g_7)^T$ so that $S_h = x_1, E_h = x_2, I_h = x_3, R_h = x_4, S_m = x_5, E_m = x_6$, and $I_m = x_7$. Then the malaria model (1) becomes

$$\begin{align*}
\frac{dx_1}{dt} &= \Lambda_h - \frac{b \beta_h x_1 x_7}{1 + \nu h x_7} - \mu_h x_1 + \omega x_4 := g_1 \\
\frac{dx_2}{dt} &= \frac{b \beta_h x_1 x_7}{1 + \nu h x_7} - (\alpha_h + \mu_h) x_2 := g_2 \\
\frac{dx_3}{dt} &= \alpha_h x_2 - (r + \mu_h + \delta_h) x_3 := g_3 \\
\frac{dx_4}{dt} &= r x_3 - (\mu_h + \omega) x_4 := g_4 \\
\frac{dx_5}{dt} &= \Lambda_m - \frac{b \beta_m x_5 x_3}{1 + \nu m x_3} - \mu_m x_5 := g_5 \\
\frac{dx_6}{dt} &= \frac{b \beta_m x_5 x_3}{1 + \nu m x_3} - (\alpha_m + \mu_m) x_6 := g_6 \\
\frac{dx_7}{dt} &= \alpha_m x_6 - (\mu_m + \delta_m) x_7 := g_7
\end{align*}$$

Let the bifurcation parameter $\beta_h$ be chosen so that at $R_0 = 1$ in (2), we obtain

$$\beta_h^* = \frac{\mu_h \mu_m (\alpha_h + \mu_h)(r + \mu_h + \delta_h)(\mu_m + \delta_m)(\alpha_m + \mu_m)}{b^2 \alpha_h \Lambda_h \alpha_m \beta_m \Lambda_m}.$$
The linearized matrix of the model (1) around the disease-free equilibrium \( E_0 \) and evaluated at \( \beta_h^* \) is given by

\[
J(E_0, \beta_h^*) = \begin{pmatrix}
-\mu_h & 0 & 0 & \omega & 0 & 0 & A \\
0 & B & 0 & 0 & 0 & 0 & C \\
0 & \alpha_h & D & 0 & 0 & 0 & 0 \\
0 & 0 & r & E & 0 & 0 & 0 \\
0 & 0 & G & 0 & -\mu_m & 0 & 0 \\
0 & 0 & K & 0 & 0 & L & 0 \\
0 & 0 & 0 & 0 & 0 & \alpha_m & M
\end{pmatrix}, \tag{8}
\]

where \( A = -\frac{b\beta_h^*\Lambda_h}{\mu_h}, \ B = -(\alpha_h + \mu_h), \ C = \frac{b\beta_h^*\Lambda_h}{\mu_h}, \ D = -(r + \delta_h + \mu_h), \ E = -(\mu_h + \omega), \ G = -\frac{b\beta_m\Lambda_m}{\mu_m}, \ K = \frac{b\beta_m\Lambda_m}{\mu_m}, \ L = -(\alpha_m + \mu_m), \ M = -(\mu_m + \delta_m). \)

The eigenvalues of \( J(E_0, \beta_h^*) \) are the roots of the characteristic equation given by

\[
(\lambda + \mu_h)(\lambda + \mu_m)(\lambda + \mu_h + \omega)P(\lambda) = 0, \tag{9}
\]

where \( P(\lambda) \) is a polynomial of degree four whose roots are all negative except one zero eigenvalue. Let the right eigenvector corresponding to this simple zero eigenvalue be denoted by \( w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T \) so that \( J(E_0, \beta_h^*)w = 0 \). Then we have

\[
\begin{align*}
w_1 &= -Pw_6, \quad w_2 = \frac{(\mu_h + \omega)}{b\beta_m\lambda_m\alpha_h}w_6, \\
w_3 &= \frac{(\mu_m)}{b\beta_m\lambda_m}w_6, \quad w_4 = r\frac{(\mu_m)}{b\beta_m\lambda_m(\mu_h + \omega)}w_6, \\
w_5 &= -\frac{(\mu_m)}{\mu_m}w_6, \quad w_6 = w_6, \quad w_7 = \frac{\alpha_m}{\mu_m + \delta_m}w_6.
\end{align*} \tag{10}
\]

where \( P = \frac{(\alpha_m + \mu_m)\mu_m[r\mu_h(\alpha_h + \mu_h + \omega) + (\delta_h + \mu_h)(\alpha_h + \mu_h)(r + \delta_h + \mu_h)]}{b\beta_m\lambda_m\alpha_h\mu_h(\mu_h + \omega)} \).

Further, the left eigenvector, \( v = (v_1, v_2, \ldots, v_7) \), corresponding to the simple zero eigenvalue of \( J(E_0, \beta_h^*) \) is obtained as

\[
\begin{align*}
v_1 &= 0, \quad v_2 = \frac{b\beta_m\alpha_m\alpha_h\lambda_m}{(\alpha_h + \mu_h)(r + \mu_h + \delta_h)(\alpha_m + \mu_m)}v_7, \\
v_3 &= \frac{b\beta_m\alpha_m\lambda_m}{(r + \mu_h + \delta_h)(\alpha_m + \mu_m)}v_7, \quad v_4 = 0, \quad v_5 = 0 \\
v_6 &= \frac{\alpha_m}{\alpha_m + \mu_m}v_7, \quad v_7 = v_7.
\end{align*} \tag{11}
\]

It is easy to see from (6) that all the second-order partial derivatives at \( E_0 \) and
\[ \beta_h^* \] are zero except the following:

\[
\frac{\partial^2 g_1}{\partial x_1 \partial x_7} = \frac{\partial^2 g_1}{\partial x_7 \partial x_1} = -b\beta_h^*, \quad \frac{\partial^2 g_2}{\partial x_1 \partial x_7} = \frac{\partial^2 g_2}{\partial x_7 \partial x_1} = b\beta_h^*
\]

\[
\frac{\partial^2 g_5}{\partial x_3 \partial x_5} = \frac{\partial^2 g_5}{\partial x_5 \partial x_3} = -b\beta_m, \quad \frac{\partial^2 g_6}{\partial x_3 \partial x_5} = \frac{\partial^2 g_6}{\partial x_5 \partial x_3} = b\beta_m
\]

and

\[
\frac{\partial^2 g_1}{\partial x_7 \partial \beta_h} = \frac{\partial^2 g_1}{\partial \beta_h \partial x_7} = -b\frac{\Lambda_h}{\mu_h}, \quad \frac{\partial^2 g_2}{\partial x_7 \partial \beta_h} = \frac{\partial^2 g_2}{\partial \beta_h \partial x_7} = b\frac{\Lambda_h}{\mu_h}.
\]

The nature of the bifurcation at \( R_0 = 1 \) is determined by the signs of the bifurcation coefficients \( a \) and \( b \), obtained from the above partial derivatives, given, respectively, by

\[
a = \sum_{k,i,j=1}^7 v_kw_iw_j \frac{\partial^2 g_k}{\partial x_i \partial x_j}(E_0, \beta_h^*)
\]

\[
= \frac{2v_7\alpha_m w_6}{\Lambda_m} \left[ P\mu_m\mu_h\Lambda_m + \alpha_m + \mu_m \right] \tag{12}
\]

and

\[
b = \sum_{k,i=1}^7 v_kw_i \frac{\partial^2 g_k}{\partial x_i \partial \beta_h}(E_0, \beta_h^*)
\]

\[
= \frac{2b^2\alpha_h\alpha_m^2 \beta_m \Lambda_m v_7 w_6}{(\mu_m + \delta_m)(\alpha_h + \mu_h)(\mu + \mu_h + \delta_h)(\alpha_m + \mu_m)\mu_h} \tag{13}
\]

Since \( a > 0 \) and \( b > 0 \), it follows that the malaria model (1) exhibits a backward bifurcation whenever the threshold parameter \( R_0 \) crosses unity. This shows the co-existence of disease-free and the endemic equilibrium at \( R_0 \) slightly less than unity. Thus we have established the following result:

**Theorem 3.1.** The malaria model given by (1) undergoes a phenomenon of backward bifurcation at \( E_0 \) and \( R_0 = 1 \).

The implication of the above result is that reduction of \( R_0 \) below unity alone is not sufficient, though necessary, to eliminate the disease. To rule out this occurrence, the global dynamics of the equilibria is investigated in the next section for the special case with \( \omega = 0 \).

## 4 Global Stability Analysis

**Theorem 4.1.** The disease-free equilibrium, \( E_0 \), of the model (1), is globally asymptotically stable in \( D \) if \( R_0|_{\omega=0} \leq 1 \).
Proof. Consider the linear Lyapunov function $\mathcal{L} : \{(S_h, E_h, I_h, R_h, S_m, E_m, I_m) \in \mathcal{D} : S_h > 0, E_h \geq 0, I_h \geq 0, R_h \geq 0, S_m > 0, E_m \geq 0, I_m \geq 0, \} \text{ defined by}$

$$
\mathcal{L} = c_1 E_h + c_2 I_h + c_3 E_m + c_4 I_m
$$

where

$$
c_1 = \frac{\alpha_h}{\mu_m(\alpha_h + \mu_h)(r + \delta_h + \mu_h)}, \quad c_2 = \frac{1}{\mu_m(r + \delta_h + \mu_h)}, \quad c_3 = \frac{1}{b\beta_m \Lambda_m}, \quad c_4 = \frac{\alpha_m + \mu_m}{b\beta_m \alpha_m \Lambda_m}.
$$

The time derivative of $\mathcal{L}$ given by (14) along the solutions of the model (1) yields

$$
\dot{\mathcal{L}} = \frac{\alpha_h}{\mu_m(\alpha_h + \mu_h)(r + \delta_h + \mu_h)} \left[ \frac{b\alpha_h \beta_h S_h I_m}{1 + \nu_m I_m} - (\alpha_h + \mu_h) E_h \right] + \frac{1}{\mu_m(r + \delta_h + \mu_h)} \left[ (\alpha_h E_h - (r + \delta_h + \mu_h) I_h \right]

+ \frac{1}{b\beta_m \Lambda_m} \left[ \frac{b\beta_m S_m I_h}{1 + \nu_m I_m} - (\alpha_m + \mu_m) E_m \right] + \frac{\alpha_m + \mu_m}{b\beta_m \alpha_m \Lambda_m} \left[ (\alpha_m E_m - (\mu_m + \delta_h) I_m \right]

= \frac{b\alpha_h \beta_h S_h I_m}{\mu_m(\alpha_h + \mu_h)(r + \delta_h + \mu_h)} - \frac{I_h}{\mu_m} + \frac{S_m I_h}{\Lambda_m(1 + \nu_m I_h)} - \frac{(\alpha_m + \mu_m)(\mu_m + \delta_h) I_m}{b\beta_m \alpha_m \Lambda_m}

\leq \frac{b\alpha_h \beta_h \Lambda_h I_m}{\mu_m(\alpha_h + \mu_h)(r + \delta_h + \mu_h)} - \frac{(\alpha_m + \mu_m)(\mu_m + \delta_h) I_m}{b\beta_m \alpha_m \Lambda_m}

= \frac{(\alpha_m + \mu_m)(\mu_m + \delta_h)}{b\beta_m \alpha_m \Lambda_m} \left[ \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)} - 1 \right] I_m

= \frac{(\alpha_m + \mu_m)(\mu_m + \delta_h)}{b\beta_m \alpha_m \Lambda_m} [R_0^2 - 1] I_m.
$$

Therefore $\dot{\mathcal{L}} \leq 0$ for $R_0 \leq 1$ and $\dot{\mathcal{L}} = 0$ if and only if $I_m = 0$. Further, one sees that $(S_h, E_h, I_h, R_h, S_m, E_m) \to \left( \frac{\Delta h}{\mu_h}, 0, 0, 0, \frac{\Delta m}{\mu_m}, 0 \right)$ as $t \to \infty$ since $I_m \to 0$ as $t \to \infty$. Consequently, the largest compact invariant set in $\{(S_h, E_h, I_h, R_h, S_m, E_m, I_m) \in \mathcal{D} : \dot{\mathcal{L}} = 0 \}$ is the singleton $\{E_0\}$ and by Lasalle’s invariance principle [10], $E_0$ is globally asymptotically stable in $\mathcal{D}$ if $R_0 \leq 1$. \square

Apart from the fact that the result above rules out the co-existence of disease-free and endemic equilibria at $R_0 < 1$, further epidemiological implication of the result is that the disease elimination is possible irrespective of the initial sizes of the sub-populations of the model whenever the threshold parameter, $R_0$, is less than unity.

The global asymptotic stability analysis of the endemic equilibrium is next explored for the special case with $\omega = 0$ since a unique endemic equilibrium exits for this case. To achieve this, we use the nonlinear Lyapunov function.
of Goh-Volterra type which has been found to be very successful. See, for instance, [7,8,14,19,21] for a successful application of this Lyapunov function.

**Theorem 4.2.** The endemic equilibrium of the model (1) is globally asymptotically stable whenever $\mathcal{R}_0|\omega=0 > 1$.

**Proof.** Let the endemic equilibrium of the model (1) be denoted by $\mathcal{E}_e = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*)$ and let $\mathcal{R}_0|\omega=0 > 1$ so that $\mathcal{E}_e$ exists. Consider the following nonlinear Lyapunov function:

$$
\mathcal{L} = S_h - S_h^* - S_h^* \ln \left(\frac{S_h}{S_h^*}\right) + E_h - E_h^* - E_h^* \ln \left(\frac{E_h}{E_h^*}\right)
$$

$$
+ \frac{\alpha_h + \mu_h}{\alpha_h} \left[ I_h^* - I_h - I_h^* \ln \left(\frac{I_h}{I_h^*}\right) \right] + S_m - S_m^* - S_m^* \ln \left(\frac{S_m}{S_m^*}\right)
$$

$$
+ E_m - E_m^* - E_m^* \ln \left(\frac{E_m}{E_m^*}\right) + \frac{\alpha_m + \mu_m}{\alpha_m} \left[ I_m - I_m^* - I_m^* \ln \left(\frac{I_m}{I_m^*}\right) \right],
$$

(15)

with the Lyapunov derivative given by

$$
\dot{\mathcal{L}} = \dot{S}_h - \frac{S_h^*}{S_h} \dot{S}_h + \dot{E}_h - \frac{E_h^*}{E_h} \dot{E}_h + \frac{\alpha_h + \mu_h}{\alpha_h} \left( \dot{I}_h - \frac{I_h^*}{I_h} \dot{I}_h \right)
$$

$$
+ \dot{S}_m - \frac{S_m^*}{S_m} \dot{S}_m + \dot{E}_m - \frac{E_m^*}{E_m} \dot{E}_m + \frac{\alpha_m + \mu_m}{\alpha_m} \left( \dot{I}_m - \frac{I_m^*}{I_m} \dot{I}_m \right)
$$

(16)

where upper dot represents the differentiation with respect to time $t$. Putting the appropriate equations of model (1) in (16), we have

$$
\dot{\mathcal{L}} = \Lambda_h - \frac{b\beta_h S_h I_m}{1+\nu_h I_m} - \mu_h S_h - \frac{S_h^*}{S_h} \left( \Lambda_h - \frac{b\beta_h S_h I_m}{1+\nu_h I_m} - \mu_h S_h \right)
$$

$$
- \left[ \alpha_h + \mu_h \right] E_h - \frac{E_h^*}{E_h} \left( \frac{b\beta_h S_h I_m}{1+\nu_h I_m} - \left[ \alpha_h + \mu_h \right] E_h \right) + \frac{\alpha_h + \mu_h}{\alpha_h}
$$

$$
\times \left[ \alpha_h E_h - \left[ r + \mu_h + \delta_h \right] I_h - \frac{I_h^*}{I_h} \left( \alpha_h E_h - \left[ r + \mu_h + \delta_h \right] I_h \right) \right]
$$

$$
+ \Lambda_m - \frac{b\beta_m S_m I_h}{1+\nu_m I_h} - \mu_m S_m - \frac{S_m^*}{S_m} \left( \Lambda_m - \frac{b\beta_m S_m I_h}{1+\nu_m I_h} - \mu_m S_m \right)
$$

$$
+ \frac{b\beta_m S_m I_h}{1+\nu_m I_h} - \left[ \alpha_m + \mu_m \right] E_m - \frac{E_m^*}{E_m} \left( \frac{b\beta_m S_m I_h}{1+\nu_m I_h} - \left[ \alpha_m + \mu_m \right] E_m \right)
$$

$$
+ \frac{\alpha_m + \mu_m}{\alpha_m} \left[ \alpha_m E_m - \left[ \mu_m + \delta_m \right] I_m - \frac{I_m^*}{I_m} \left( \alpha_m E_m - \left[ \mu_m + \delta_m \right] I_m \right) \right],
$$

(17)
For convenience, let \( f(I_m) = \frac{I_m}{1 + \nu_m I_m} \) and \( f(I_h) = \frac{I_h}{1 + \nu_h I_h} \) then further simplification yields

\[
\dot{\xi} = \lambda_h \left( 1 - \frac{S^*_h}{S_h} \right) - \mu_h S_h \left( 1 - \frac{S^*_h}{S_h} \right) + b\beta_h S^*_h f(I_m) - \frac{E_h^* b\beta_h S_h f(I_m)}{E_h} + (\alpha_h + \mu_h) E_h^* - \frac{(\alpha_h + \mu_h)(r + \mu_h + \delta_h) I_h}{\alpha_h} - \frac{(\alpha_h + \mu_h) I_h^* E_h}{I_h} + b\beta_m S^*_m f(I_h) - \frac{E_m^* b\beta_m S_m f(I_h)}{E_m} + (\alpha_m + \mu_m) E_m^* - \frac{(\alpha_m + \mu_m)(\mu_m + \delta_m) I_m}{\alpha_m} - \frac{(\alpha_m + \mu_m) I_m^* E_m}{I_m}.
\]

Using the equilibrium relations obtained from (1) at the endemic steady state, then (17) becomes

\[
\dot{\xi} = \mu_h S^*_h \left( 2 - \frac{S^*_h}{S_h} - \frac{S_h}{S^*_h} \right) + b\beta_h S^*_h f(I_m) - \frac{b\beta_h(S^*_h)^2 f(I_m)}{S_h} + b\beta_h S^*_h f(I_m)
\]

\[
- \frac{E_h^* b\beta_h S_h f(I_m)}{E_h} + b\beta_h S^*_h f(I_m) - \frac{b\beta_h S_h f(I_m)}{I_h} - \frac{b\beta_h E_h f(I_m)}{E_h} + b\beta_m S_m f(I_h)
\]

\[
- \frac{b\beta_m(S_m^2 f(I_m))}{S_m} + b\beta_m S_m f(I_h) - \frac{E_m^* b\beta_m S_m f(I_h)}{E_m} + b\beta_m S_m f(I_h)
\]

\[
- \frac{b\beta_m S_m f(I_h)}{I_m} - \frac{b\beta_m S_m f(I_h)}{E_m} + \frac{b\beta_m f(I_h)}{I_m}.
\]

Moreover, if we add and subtract \( b\beta_h S^*_h f(I_m), \frac{b\beta_h S^*_h f(I_m)}{I_m f(I_m)} \) and \( b\beta_m S^*_m f(I_h) \) and \( \frac{b\beta_m S^*_m f(I_h)}{I_m f(I_m)} \) in (18) systematically, we obtain

\[
\dot{\xi} = \mu_h S^*_h \left( 2 - \frac{S^*_h}{S_h} - \frac{S_h}{S^*_h} \right) + b\beta_h S^*_h f(I_m) \left[ 4 - \frac{S^*_h}{S_h} - \frac{E_h S_h f(I_m)}{S_h} - \frac{E^*_h}{E_h} - \frac{I_m f(I_m)}{I_m} \right]
\]

\[
+ b\beta_h S^*_h f(I_m) - \frac{b\beta_h S_h f(I_m)}{I_h} + \frac{b\beta_h S_h f(I_m)}{I_h f(I_m)} - b\beta_h S^*_h f(I_m)
\]

\[
+ \mu_m S^*_m \left( 2 - \frac{S^*_m}{S_m} - \frac{S_m}{S^*_m} \right) + b\beta_m S^*_m f(I_h) \left[ 4 - \frac{S^*_m}{S_m} - \frac{E_m S_m f(I_h)}{S_m} - \frac{E^*_m}{E_m} - \frac{I_m f(I_h)}{I_m} \right]
\]

\[
+ b\beta_m S^*_m f(I_h) - \frac{b\beta_m S_m f(I_h)}{I_m} + \frac{b\beta_m S_m f(I_h)}{I_m f(I_h)} - b\beta_m S^*_m f(I_h).
\]

Qualitative analysis of malaria dynamics 3899
Further algebraic manipulation on (19) yields

\[
\dot{\mathbf{L}} = -\mathbf{L}_1 + \mathbf{L}_2 + b\beta_h S_h f(I_m) I_h \left[ \frac{f(I_m)}{I_h} - \frac{I_h f(I_m)}{I_h} \right] - 1 \\
- \mathbf{L}_3 + \mathbf{L}_4 - b\beta_h S_h f(I_m) I_h \left[ \frac{f(I_m)}{I_h} - \frac{f(I_m)}{I_h} \right] \left[ 1 - \frac{f(I_m)}{I_h} \right]
\]

where

\[
\mathbf{L}_1 = \mu_h S_h \left( \frac{S_h}{S_h} + \frac{S_h}{S_h} - 2 \right), \\
\mathbf{L}_2 = b\beta_h S_h f(I_m) \left[ \frac{S_h}{S_h} + \frac{E_h S_h f(I_m)}{E_h S_h f(I_m)} + \frac{I_h E_h}{I_h E_h} + \frac{I_h f(I_m)}{I_h f(I_m)} - 4 \right], \\
\mathbf{L}_3 = \mu_m S_m \left( \frac{S_m}{S_m} + \frac{S_m}{S_m} - 2 \right), \text{ and} \\
\mathbf{L}_4 = b\beta_m S_m f(I_h) \left[ \frac{S_m}{S_m} + \frac{E_m S_m f(I_h)}{E_m S_m f(I_h)} + \frac{I_m E_m}{I_m E_m} + \frac{I_m f(I_h)}{I_m f(I_h)} - 4 \right].
\]

Using AM – GM inequality: (arithmetic mean is greater or equal to the geometric mean), and since \( f(I_h) \) and \( f(I_m) \) are increasing functions, it follows from (20) that \( \mathbf{L} \leq 0 \) with \( \mathbf{L} = 0 \) if and only if \( S_h = S_h^*, E_h = E_h^*, I_h = I_h^*, S_m = S_m^*, E_m = E_m^*, I_m = I_m^* \). Hence, the largest compact invariant subset of the set where \( \dot{\mathbf{L}} = 0 \) is the singleton \( \{(S_h, E_h, I_h, S_m, E_m, I_m) = (S_h^*, E_h^*, I_h^*, S_m^*, E_m^*, I_m^*)\} \).

From this, it is easy to show that \( R_h \to \frac{R^*_h}{\mu_h} = R^*_h \) as \( t \to \infty \). By Lasalle’s invariance principle [10], it follows that every solution in \( \mathcal{D} \) approaches \( \mathcal{E}_e \) for \( R_0|_{\omega = 0} > 1 \) as \( t \to \infty \). This ends the proof.

\[ \square \]

5 Sensitivity Analysis

To determine the parameters most responsible for the transmission and spread of the malaria disease, a sensitivity analysis of the model (1) is carried out in the sense of [6,15].

**Definition 5.1.** The normalized forward-sensitivity index of a variable, \( v \), that depends differentiably on a parameter, \( p \), is defined as:

\[
\Upsilon_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v}.
\]
In particular, sensitivity indices of the basic reproduction number, $R_0$, with respect to the model parameters are computed as follows:

\[
\begin{align*}
\Upsilon_{R_0 b} &= \frac{\partial R_0}{\partial b} \times \frac{b}{R_0} = 1, \\
\Upsilon_{R_0 \alpha_h} &= \frac{\partial R_0}{\partial \alpha_h} \times \frac{\alpha_h}{R_0} = \frac{\mu_h}{2(\alpha_h + \mu_h)}, \\
\Upsilon_{R_0 \beta_h} &= \frac{\partial R_0}{\partial \beta_h} \times \frac{\beta_h}{R_0} = \frac{1}{2}, \\
\Upsilon_{R_0 \beta_m} &= \frac{\partial R_0}{\partial \beta_m} \times \frac{\beta_m}{R_0} = \frac{1}{2}, \\
\Upsilon_{R_0 r} &= \frac{\partial R_0}{\partial r} \times \frac{r}{R_0} = -\frac{r}{2(r + \delta_h + \mu_h)}.
\end{align*}
\]

(22)

In a similar manner, we can compute the sensitivity index (S.I) of $R_0$ to other parameters of the model (1). The signs of S.I are shown in the table below.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>S.I</th>
<th>Parameter</th>
<th>S.I</th>
</tr>
</thead>
<tbody>
<tr>
<td>$b$</td>
<td>+ve</td>
<td>$\delta_h$</td>
<td>-ve</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>+ve</td>
<td>$\delta_m$</td>
<td>-ve</td>
</tr>
<tr>
<td>$\alpha_h$</td>
<td>+ve</td>
<td>$\mu_h$</td>
<td>-ve</td>
</tr>
<tr>
<td>$\beta_m$</td>
<td>+ve</td>
<td>$\mu_m$</td>
<td>-ve</td>
</tr>
<tr>
<td>$\alpha_m$</td>
<td>+ve</td>
<td>$r$</td>
<td>-ve</td>
</tr>
<tr>
<td>$\Lambda_h$</td>
<td>+ve</td>
<td>$\Lambda_m$</td>
<td>+ve</td>
</tr>
</tbody>
</table>

The positive sign of S.I of the basic reproduction number to the model parameters indicates that an increase (or decrease) in the value of each of the parameter in this category will lead to an increase (or decrease) in the basic reproduction number of the disease. For example, $\Upsilon_{R_0 b} = 1$ suggests that increasing (or decreasing) the mosquito biting rate by 10% increases (or decreases) the basic reproduction number, $R_0$, by 10%. On the other hand, the negative sign of S.I of the basic reproduction number to the model parameters implies that an increase (or decrease) in the value of each of the parameter in this category leads to a corresponding decrease (or increase) in the basic reproduction number of the disease. Thus, sensitivity analysis of the malaria model (1) provides a very good insight into the transmission dynamics of the disease. In particular, it helps the public health authorities in focusing on an appropriate intervention strategy for preventing and controlling the spread of the disease.
6 Conclusion

This paper presented a qualitative analysis of a malaria dynamics with nonlinear incidence function. The results of the analysis are highlighted as follows.

(i) The model has a disease-free equilibrium which co-exists with the endemic equilibrium at $R_0 < 1$ due to the temporary immunity acquired by the recovered individual. This behaviour is termed backward (sub-critical) bifurcation.

(ii) The disease-free equilibrium of the model is globally asymptotically stable when $R_0 < 1$ with no loss of immunity acquired by the recovered individual. Showing that the malaria-free population is possible.

(iii) The model has a globally asymptotically stable endemic equilibrium if the threshold parameter, $R_0$, is greater than unity.

(iv) Sensitivity analysis reveals that mosquito biting rate among other parameters contributes most significantly to the transmission and spread of the malaria disease. Hence the intervention strategy that inhibits the human-mosquito contact should be encouraged in order to achieve a malaria-free population.

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References


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