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MATHEMATICAL ANALYSIS OF THE TRANSMISSION DYNAMICS OF MALARIA IN THE PRESENCE OF MOSQUITO TRAP

A. B. Okrinya[1], Arrow B. Dibolouembele[2] and I. C. Eli[3]

[1]Department of Mathematics, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.
[2]Department of Mathematics, Niger Delta University, Wilberforce Island, Bayelsa State, Nigeria.
[3]Department of Mathematics and Statistics, Federal University Otuoke, Bayelsa State, Nigeria.

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ABSTRACT

Here we model the transmission dynamics of malaria in the presence of mosquito trap. The model based on some relevant assumptions, is a simple system of five ordinary differential equations, devoid of the exposed classes of both human and mosquito populations and including a compartment of trapped mosquitoes. We analyze the system considering its mathematical and biological relevance by considering its positivity, existence and uniqueness conditions. The basic reproduction, R_0, was derived and a simple sensitivity analysis was carried out to determine the most sensitive parameter. The results reveal the 'Trap effectiveness' as the most important parameter. We carried out numerical simulations using MATLAB ode45 Routine Solver and both analytical and numerical results affirm the local and global stability of the disease free equilibrium point based on the threshold parameter, R_0, showing stability for $R_0 < 1$ and instability for $R_0 > 1$.

1.1 Introduction

Every day, malaria is becoming an alarming issue as it poses a very serious public health challenge. Malaria is a disease caused by the protozoan parasite of the genus Plasmodium. Pregnant mothers, infants and immune-compromised individuals are at high risk to the disease. The causative species are Plasmodium falciparum, *P. Vivax*, *P. Ovale*, *P. Malariae* and *P. Knowlesi.*

Malaria contributes significantly to maternal and fetal morbidity and mortality in affected areas especially, in the tropics and subtropics. Areas of high transmission are in developing countries with lots of stagnant water, which form a breeding site for the disease vector such as the anopheles mosquito. Malaria burden has been quite alarming as reported in [1], and there was an estimated 241million cases and 627000 death in 2020 [2]. Plasmodium falciparum is pre-dominant among other species and it is the most serious and difficult form of the disease and can be very fetal in the absence of recognition and medication. *Plasmodium falciparum* is the most common cause of infection in Africa and southeast Asia, and it is responsible for *80%* of all malaria cases and *90%* of death caused

*Corresponding author: Okrinya A.B.
E-mail address: okrinya@ndu.edu.ng, Tel: +2348022039490
https:xxxxxxxxxxxxxxxxxxxxxxxx

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[3]. The pattern of infection may change due to climatic factors which may cause a drift from lower latitude to regions where population have not developed immunity to the disease.
Malaria has huge financial effect on the global economy as can be found in an estimate accounting both for government and out of pocket spending – amounting to $4.3 billion (95% of UI 4.1-4.4) in 2016, representing 8.6% (95% UI 8.1-8.9) yearly increase over malaria spending in 2000 [4].
Poor people mostly suffer from the disease due to financial instability, which deprives them from gaining access to medical care or preventive measures for combating the disease. This results in more malaria deaths being prevalent at home.
Asymptomatic malaria poses a lot of dangers to malaria transmission. Resistance of parasite to anti-malaria agent continue to be a threat to controlling and elimination efforts globally [5]. The anopheles is the most wide spread in Africa and the most difficult to control [6]. Malaria is very endemic in Nigeria with a very high population at risk with estimated 68 million cases and 194,000 deaths due the disease in 2021 and accounts for about 27% of the word malaria burden [7]. Nigeria suffers the world's greatest malaria burden, with approximately 51 million cases and 207,000 death reported annually, being approximately *30%* of the total malaria burden in Africa [8]. According to the federal ministry of health (2012), the financial loss due to malaria annually is estimated as 132 billion naira in form of treatment cost, prevention, etc. In recent decades, both governmental and non-governmental organizations have been established to wage the fight against malaria in Africa and beyond. This has led to the improvement in methods used to monitor, control, prevent, evaluate and even manage malaria in endemic areas. These are focused on research and information, education and communication [9].
There is high level of mosquito abundance especially in the tropical regions of the world. Areas with stagnant water are breeding sites for mosquitos. Mosquitoes prefer to stay indoors at night where the temperature is warm to feed on human hosts. The inherent high growth rate of the population of mosquitoes has serious implication in malaria transmission. Most times malaria cases are resistant to some chemotherapeutic drugs which constitute threat to life and loss of scarce resources that could be put into alternative use. Since Ronald Rose's idea of reducing the population of mosquitoes below a threshold value emerged, a lot of efforts and strategies have been employed at various times to reduce the population of mosquitoes and also to reduce the contact between mosquitoes and humans.
Here we propose a simple deterministic model represented by a system of differential equations that describes the transmission dynamics of malaria in humans and vector (mosquito) populations incorporating a trapping system of mosquitoes with the intent of reducing the infection rate of humans and mosquitoes. Section 1 entails a brief introduction, whereas the formulation of the model is given in section 2. The model analysis is presented in section 3, followed by the numerical simulation in section 4. The paper was rounded up in section 5 with a brief discussion and conclusion.

2.1. The Formulation of the Model

We divide the human population into compartments of susceptible and infectious humans. Whereas the mosquito population is divided into the susceptible, infectious and trapped mosquitoes. State variables in the model are given in Table 1 and the movement between compartments is summarized in figure 1, the individual pathways to be discussed below.

Table 1: The state variables in the model

State variables	Description
S_h	Susceptible humans
S_m	Susceptible mosquitoes
I_h	Infectious humans
I_m	Infectious mosquitoes
T_m	Trapped mosquitoes
N_h	Total humans
N_m	Total mosquitoes

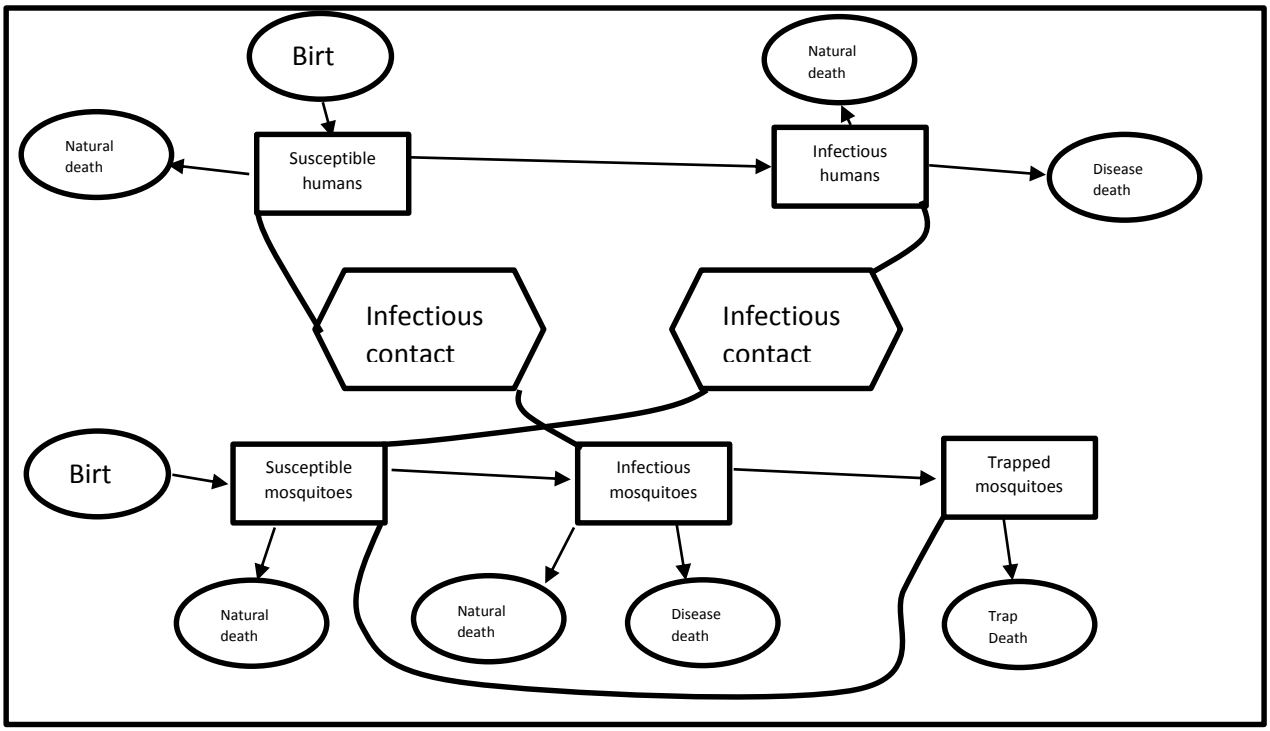


Fig 1. Schematic representation of the human and mosquito interaction

The entire human population is described by the equation defined as

$N_h = S_h + I_h$

While the mosquito population is describe by

$N_m = S_m + I_m + T_m$

Susceptible humans get infected at a rate $\beta_h S_h \frac{I_m}{N_m}$, where β_h is the probability that susceptible humans get infected after being bitten by an infectious mosquito. Susceptible humans die naturally at a rate $\mu_h S_h$. Infected humans die naturally at a rate $\alpha_h I_h$ and naturally at a rate $\mu_h I_h$.

Susceptible mosquitoes get infection after blood meal from infectious humans at a rate $\beta_m S_m \frac{I_h}{N_h}$ where β_m is the probability that a susceptible mosquito gets infected after biting an infectious human. Susceptible mosquitoes are caught in the trap at a rate ηS_m and die naturally at a rate $\mu_m S_m$.

Infectious mosquitoes are caught in the trap at a rate, ηI_m, while some die due to the disease at a rate α_m and naturally, at a rate $\mu_m I_m$. Here, we have assumed the same trap attraction rate constant, η, for both susceptible and infectious mosquitoes, which appears to be quite reasonable since we are not considering transmission probability but the ability of the trap to attract mosquitoes due to its biochemical components. All trapped mosquitoes from both susceptible and infectious mosquito compartments are recruited into the trap compartment, where, every trapped mosquito is expected to be removed or die naturally at a rate $\mu_m T_m$

From fig 1 and with the above assumptions lead to the following system of equations.

$$\frac{dS_h}{dt} = \lambda_h N_h - \beta_h S_h \frac{I_m}{N_m} - \mu_h S_h \quad (1)$$

$$\frac{dI_h}{dt} = \beta_h S_h \frac{I_m}{N_m} - \alpha_h I_h - \mu_h I_h \quad (2)$$

$$\frac{dS_m}{dt} = \lambda_m N_m - \beta_m S_m \frac{I_h}{N_h} - \eta_1 S_m - \mu_m S_m \quad (3)$$

$$\frac{dI_m}{dt} = \beta_m S_m \frac{I_h}{N_h} - \eta_1 I_m - \alpha_m I_m - \mu_m I_m \quad (4)$$

$$\frac{dT_m}{dt} = \eta_1 (S_m + I_m) - \mu_m T_m \quad (5)$$

$$\frac{dN_h}{dt} = \lambda_h N_h - \alpha_h I_h - \mu_h N_h \quad (6)$$

$$\frac{dN_m}{dt} = \lambda_m N_m - \alpha_m I_m - \mu_m N_m \quad (7)$$

Where $N_h = S_h + I_h \;;\quad N_h = S_h + I_h + T_m$

and *(6)* is derived from adding *(1), (2)* and *(3)* while *(7)* is derived from adding *(3) – (4).* The system is to be solved subject to the following initial conditions:

$N_h(0) = N_{h_0}$, $N_m(0) = N_{m_0}$, $S_h(0) = S_{h_0}$, $I_h(0) = 0$, $S_m(0) = S_{m_0}$, $I_m(0) = N_{m_0} - S_{m_0}$, $T_m(0) = 0$

2.2. Parameter values

All parameters for the construction of the model are listed in *table 3.1* together with values taken from various sources. However assumptions are made on some other parameters that do not seem to have well defined values.

Table 2: Model parameters and their dimensions. Values marked with (*) are assumed values and the rest are obtained from data

rameters	Description	Values	Units	Source
ι	Natural death rate of humans	0.0000548	Day^{-1}	[8]
λ_h	Birth rate of humans	0.000104	Day^{-1}	[9]
$_m$	Birth rate of mosquitoes	0.4216*	Day^{-1}	varied
ι_h	Disease related death rate of humans	0.01244*	Day^{-1}	varied
ι_m	Disease related death rate of mosquitoes	0.0279*	Day^{-1}	varied
ι,	The probability that a bite by an infectious mosquito- infects a susceptible human	0.086	Day^{-1}	[9]
n	Natural death rate of mosquitoes	0.125	Day^{-1}	[9]
1	Trap rate of mosquitoes	0.8276*	Day^{-1}	varied
m	The probability that a bite by an infectious mosquito- infects a susceptible human	0.320	Day^{-1}	[10]

2.3 Nondimensionalisation

Is will be convenient to re-express the compartments values as a population fractions since the total population is the sum of the relevant compartments for both populations.

Using the population fraction

$\frac{S_h}{N_h} = \hat{S}_h \quad \Rightarrow \quad S_h = \hat{S}_h N_h, \qquad \frac{I_h}{N_h} = \hat{I}_h \;\Rightarrow\; I_h = \hat{I}_h N_h$

$\frac{S_m}{N_m} = \hat{S}_m \Rightarrow S_m = \hat{S}_m N_m$, $\frac{I_m}{N_m} = \hat{I}_m \Rightarrow I_m = \hat{I}_m N_m$, $\frac{T_m}{N_m} = \hat{T}_m \Rightarrow \quad T_m = \hat{T}_m N_m$

$\frac{t}{t_o} = \hat{t} \qquad \Rightarrow \qquad t = t_o \hat{t}$

So that

$\hat{S}_h + \hat{I}_h = 1$ and $\hat{S}_m + \hat{I}_m + \hat{T}_m = 1$

The effectiveness of the trap is targeted at controlling the population of mosquitoes, therefore we scale time with the mosquito birth parameter λ_m , and write

$$t = \frac{\hat{t}}{\lambda_m}$$

Assuming that $N_{h,o}$ and $N_{m,o}$ are the initial population of humans and mosquitoes, we write

$N_h = \widehat{N}_h N_{h_0}$ and $N_m = \widehat{N}_m N_{m_0}$

By defining the following non-dimensional parameters:

$$a = \frac{\lambda_h}{\lambda_m},\ c = \frac{\mu_h}{\lambda_m},\ g = \frac{\mu_m}{\lambda_m},\ b = \frac{\beta_h}{\lambda_m},\ f = \frac{\beta_m}{\lambda_m},\ q = \frac{\alpha_h}{\lambda_m},\ h = \frac{\alpha_m}{\lambda_m},\ \eta = \frac{\eta_1}{\lambda_m}$$

And by substituting these new parameters into *(2.1)* - *(2.7)* and dropping the hats for clarification we get

$$\frac{dS_h}{dt} = a(1 - S_h) - bS_hI_m + qS_hI_h \quad (8)$$

$$\frac{dI_h}{dt} = bS_hI_m - (q + a)I_h + qI_h^{\,2} \quad (9)$$

$$\frac{dS_m}{d\hat{t}} = 1 - (1 + \eta)S_m - fS_mI_h + hS_mI_m \quad (10)$$

$$\frac{dI_m}{d\hat{t}} = fS_mI_h - (1 + \eta + h)I_m + hI_m^{\,2} \quad (11)$$

$$\frac{dT_m}{dt} = \eta(S_m + I_m) - T_m + hI_mT_m \quad (12)$$

$$\frac{dN_h}{dt} = [a - qI_h - c]N_h \quad (13)$$

$$\frac{dN_m}{dt} = [1 - hI_m - g]N_m \quad (14)$$

Subject to the initial conditions

$$N_h(0) = N_m(0) = S_h(0) = 1,\ I_h(0) = T_m(0) = 0,\ S_m(0) = 1,\ I_m(0) = 1 - S_m(0).$$

3. Model Analysis

3.1. Establishing the disease free equilibrium point

We look at the disease free equilibrium of the model. The infection becomes stable at the space of point given as

$$E_0 = \{(S_h^*, I_h^*, S_m^*, I_m^*, T_m^*)\varepsilon\ \mathbb{R}^5 : S_h^*, I_h^*, S_m^*, I_m^*, T_m^*\ \varepsilon\ \mathbb{R}^1\ and\ 0 \le S_h^* \le 1, 0 \le I_h^* \le 1, 0 \le S_m^* \le 1, 0 \le I_m^* \le 1, 0 \le T_m^* \le 1\}$$

And $S_h^*, I_h^*, S_m^*, I_m^*, T_m^*$ are obtained when in the model

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dS_m}{dt} = \frac{dI_m}{dt} = \frac{dT_m}{dt} = \frac{dN_h}{dt} = I_h = I_m = 0$$

Substituting the above conditions into *(8)* - *(14)* and solving we obtain the disease free equilibrium point;

$$E_0 = \left\{\left(1, 0, \frac{1}{(1+\eta)}, 0, \frac{\eta}{(1+\eta)}\right)\varepsilon\ \mathbb{R}^5 : S_h^*, I_h^*, S_m^*, I_m^*, T_m^*\ \varepsilon\ \mathbb{R}^1\ and\ 0 \le S_h^* \le 1, 0 \le I_h^* \le 1, 0 \le S_m^* \le 1, 0 \le I_m^* \le 1, 0 \le T_m^* \le 1\right\}$$

3.2. Basic Reproduction Number R_0

The basic reproduction number, denoted by R_0 is the number of secondary infectious cases that would arise from the introduction of a single primary case into a fully susceptible human population *[9]*. The method of the next generation matrix in *[9]* in determining the basic reproduction is used to find R_0. The linearized system is investigated and expressed in the form.

$$X' = FX - VX, \quad (15)$$

Where

$$X' = \frac{dX}{dt},\ X = \begin{bmatrix} I_h \\ I_m \end{bmatrix},\ F = \begin{bmatrix} 0 & b \\ \frac{f}{1+\eta} & 0 \end{bmatrix}, V = \begin{bmatrix} (q + a) & 0 \\ 0 & (1 + \eta + h) \end{bmatrix} \quad (16)$$

Here, F represents the immergence of new infection, V is the transition of infection between compartments and X is the reservoir of infection. This method assumes a non-negative matrix $G = FV^{-1}$ that guarantees a unique, positive real Eigen-value strictly greater than all others. Further expansion leads to

$$G = \frac{1}{(q+a)(1+\eta+h)}\begin{bmatrix} 0 & b(q+a) \\ \frac{f(1+\eta+h)}{1+\eta} & 0 \end{bmatrix} \quad (18)$$

Hence the characteristic equation of *(18)* in terms of the largest eigenvalue λ is given as $\frac{bf}{(1+\eta)(q+a)(1+\eta+h)}$.

Thus the basic reproduction number is expressed as

$$R_0 = \frac{bf}{(1+\eta)(q+a)(1+\eta+h)} \quad (19)$$

3.3. Positivity, Existence and Uniqueness of Solution

We consider the situation in which the solution is positive, exists and unique i.e., test for positivity, existence and uniqueness for the solution to the model.

The model is described in the domain

$\Phi = \{(S_h,\ I_h,\ S_m,\ I_m,\ T_m,\ N_h,\ N_m)\ \varepsilon\ \mathbb{R}^7 : S_h,\ I_h,\ S_m,\ I_m,\ T_m \geq 0\ and\ N_h,\ N_m > 0, S_h + I_h = 1,\ S_m + I_m + T_m = 1\ \}$

Suppose that at point t = 0 all variables become non-negative, then $S_h(0) + I_h(0) = 1$ and $S_m(0) + I_m(0) + T_m(0) = 1$. If $S_h = 0$, all other variables are in Φ, then $\frac{dS_h}{dt} \geq 0$. This remains the case for every other variable in *(9)- (12)*. Note that from (9) and (12), $I_h = I_m = 0$ implies that $\frac{dI_h}{dt} = \frac{dI_m}{dt} = 0$ meaning, $I_h = I_m = 0$ at all times and there will be no disease transmission in the absence of the disease. If $N_h = 0 = N_m$, then $\frac{dN_h}{dt} = 0 = \frac{dN_m}{dt}$. But if $N_h,\ N_m > 0$ and assuming a > c and g < 1, then with appropriate initial conditions, $\frac{dI_h}{dt}, \frac{dI_m}{dt} > 0$ for all values of t > 0. Noting that the right-hand side of *(8)* - *(14)* is continuous with continuous partial derivatives, so solutions exist and are unique. The model is therefore mathematically and biologically well posed with solutions in Φ for all $t\ \varepsilon\ [0, \infty)$.

3.4. Stability Analysis

Here, we investigate the local stability of the disease free equilibrium state *[10]*. It has been shown in the model that the disease free equilibrium point is $E_0 = (S_h, I_h, S_m, I_m, T_m) = (1, 0, \frac{1}{1+\eta}, 0, \frac{\eta}{1+\eta})$. The Jacobian matrix is obtained by linearizing the system *(8)- (14)* about the disease free equilibrium point.

$$J(E_0) = \begin{bmatrix} -a & q & 0 & -b & 0 \\ 0 & -(q+a) & 0 & b & 0 \\ 0 & \frac{-f}{1+\eta} & -(1+\eta) & \frac{h}{1+\eta} & 0 \\ 0 & \frac{f}{1+\eta} & 0 & -(1+\eta+h) & 0 \\ 0 & 0 & \eta & \left(\eta + \frac{h\eta}{1+\eta}\right) & -1 \end{bmatrix} \quad (20)$$

Lemma 3.1 The disease free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

The characteristic polynomial of (20) with eigenvalue (κ) is

$(\kappa + a)(\kappa + 1 + \eta)(\kappa + 1)[\kappa^2 + B_1\kappa + B_2] = 0$ *(21)*

Where $B_1 = 1 + \eta + h + q + a$ and $B_2 = (q + a)(1 + \eta + h)(1 - R_0)$

We observe that the linear factorization in *(21)* yields negative real eigenvalue but no such deduction can immediately be made from the quadratic equation. It follows from the Routh-Hurwitz criterion [12] that $B_1,\ B_2$ is a necessary and sufficient condition that all the eigenvalues of the characteristic equation have negative real parts. This proves part of ***Lemma 3.1***, which says that the disease free equilibrium is locally asymptomatically stable if $R_0 < 1$. But if $R_0 > 1$, then the quadratic equation in *(21)* will have atleast one

sign change, in which case the disease free equilibrium is will be locally unstable. We note that the point $R_0 = 1$ is a bifurcation point in the parameter space.

Lemma 3.2

The disease-free equilibrium point of the model equations is globally asymptotically stable if $R_0 < 1$ and in addition, if H_1 and H_2 holds.

Proof

We show that:

(H_1) For $\frac{dA}{dt} = F(A, 0)$ and

(H_2) $\widehat{G}(A, B) = QB - G(A, B) \geq 0 \forall (A, B) \in E_0$ holds as used in *[12, 13, 14]*

The model equations can be expressed as follows:

$$\frac{dA}{dt} = F(A, B) = \begin{bmatrix} \lambda(1 - U) - \beta DUX + \alpha UV \\ a(1 - W) - VW + BWX \\ \theta \mathrm{VW} - \mathrm{aY} + bXY \end{bmatrix}$$

$$\frac{dB}{dt} = G(A, B) = \begin{bmatrix} \beta UX - (\alpha + \lambda)V + \alpha V^2 \\ (1 - \theta)V - (a + b)X + bX^2 \end{bmatrix}$$

Where $A = (U, W, Y)$ and $B = (V, X)$,with the components of $A \in R^3$ which represent the non-infectious class, and the components of $B \in R^2$, which represent the infectious class.

Now, the equilibrium point of the model $(U, W, X, Y, V) = (1,1,0,0,0)$.

$$F(A, 0) = \begin{bmatrix} \lambda(1 - U) \\ a(1 - W) \\ 0 \end{bmatrix} \quad (22)$$

From *(1);*

$$\frac{dU}{dt} = \lambda(1 - U)$$

$$\frac{dU}{dt} + \lambda U = \lambda \quad (23)$$

Solving *(23)* using the principle of integrating factor, we have;

$$U(t) = 1 + ce^{-\lambda t}$$

As $t \to \infty$, $U(t) = 1$

Similarly, $W(t) = 1,\ as\ t \to \infty$.

Hence, H_1 holds.

$$\frac{dB}{dt} = G(A, B) = \begin{bmatrix} \beta UX - (\alpha + \lambda)V + \alpha V^2 \\ (1 - \theta)VW - (a + b)X + bX^2 \end{bmatrix}$$

$$Q = \begin{bmatrix} -(\alpha + \lambda) & \beta U \\ (1 - \theta)W & -(a + b) \end{bmatrix}$$

$$QB = \begin{bmatrix} -(\alpha + \lambda) & \beta U \\ (1 - \theta)W & -(a + b) \end{bmatrix} \begin{bmatrix} V \\ X \end{bmatrix}$$

$$= \begin{bmatrix} \beta UX - (\alpha + \lambda)V \\ (1 - \theta)VW - (a + b)X \end{bmatrix}$$

$$\hat{G}(X, Y) = QB - G(A, B)$$

$$= \begin{bmatrix} \beta UX - (\alpha + \lambda)V \\ (1 - \theta)VW - (a + b)X \end{bmatrix} - \begin{bmatrix} \beta UX - (\alpha + \lambda)V + \alpha V^2 \\ (1 - \theta)VW - (a + b)X + bX^2 \end{bmatrix}$$

$$\therefore \hat{G}(X, Y) = \begin{bmatrix} -\alpha V^2 \\ -bX^2 \end{bmatrix}$$

$$\to \hat{G}(A, B) \geq 0\ \forall (A, B) \in E_0\ iff\ \alpha = b = 0$$

Thus, H_2 holds.

Hence, the equilibrium point is globally asymptotically stable.

3.5 Sensitivity Analysis

It is important to know the different parameters (factors) in the basic reproduction number that are responsible for the transmission of the disease, that is, we want to know the effect of the parameters on the dependent variable. Here we calculate the sensitivity index of each of the parameters in the model. The sensitivity index shows how effective each parameter is to disease transmission. According to Shah et. al *[15]*, the normalized forward sensitivity index of a variable, *u*, which depends continuously on a parameter, *p*, is defined as

$$\gamma_p^u = \frac{\partial u}{\partial p} \cdot \frac{p}{u}$$

In our work, *u* is represented by the threshold parameter, R_0 and each of the parameters determining R_0, plays the role of *p*. Table 3 below gives the sensitivity index of the parameters responsible for the transmission of the disease. The plus sign indicates a direct relationship between the parameter and R_0 whereas the reverse is the case for the negative sign.

Table 3: Sensitivity indices of R_0 to the parameters for the malaria model

rameter	Sign	Value
b	+	1.0000
f	+	1.0000
q	-	1.0127
h	-	0.3828
η	-	1.4401

The results in the table suggest that the most sensitive parameter to the basic reproduction number, R_0, is the trap effectiveness.

4. Numerical Simulations

The numerical simulations were done using *Matlab ODE 45* Routine solver. The parameters used for the simulations as defined in section 2 are a = 0.0201, b = 16.5895, q =83.295, f = 61.7284, h = 5.6611, c = 0.0106, g = 24.1127 and η = 8.12886. We used the following initial conditions for the state variable $I_h = 0, S_m = 0.7, I_m = 0.3,\ T_m = 0,\ N_h = 1, N_m = 1.$

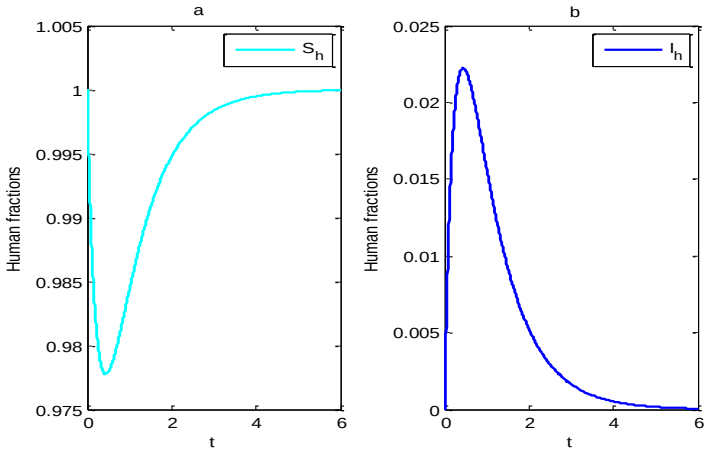


Figure 2. Results showing the effect of the disease on the human compartments. The initial conditions used are $S_h = 1,\ I_h = 0,\ S_m = 0.9,\ I_m = 0.1,\ T_m = 0,\ N_h = 1, N_m = 1$ *and the parameter values are*

$a = 3.0201,$ $b = 1.5895,$ $c = 2.0106,$ $f = 6.7284, g = 0.9997$, h $= 5.6611,$ $q = 0.0295,$ $\eta = 1.9662886.$

Figures 2a and *2b* show an entirely susceptible population in the beginning where there was no human malaria infection, but when infectious mosquitoes are introduced into the human population, the susceptible population starts dropping from 1 and then grows back to a steady state as infectious contact begins to reduce, which could be due to the killing of mosquitoes by the trap. In *figure 2b*, the number of infectious humans initially grows to a pick level and later drops to an infection-free steady state. The result shows that both susceptible and infectious populations have inverse growth relationship.

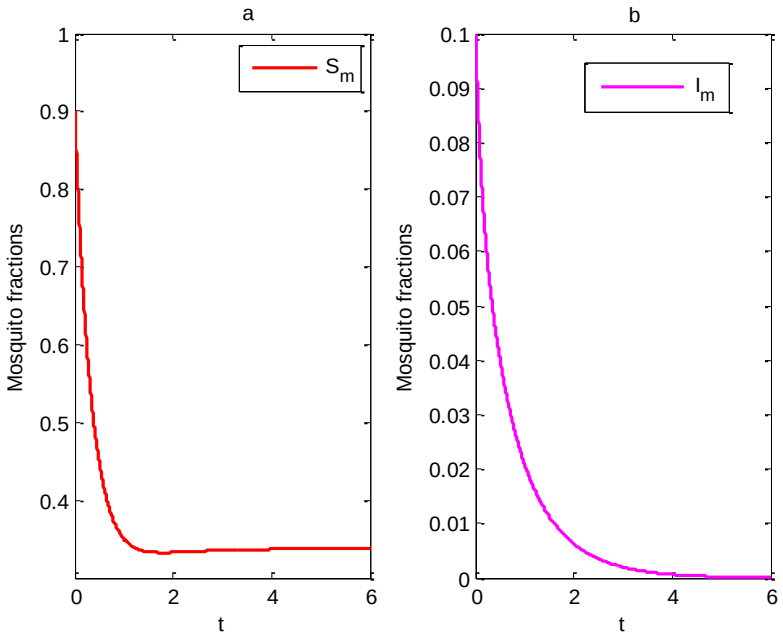


Figure 3: Results showing the effect of the disease on the mosquito compartments. The initial conditions and parameter values are the same as those in Figure 2.

Figures 3a and *3b* are the susceptible and the infectious mosquito compartments. We use the same initial conditions as those in *Figure 2* and as the disease establishes itself in *Figure 3a* due to introduction of some infectious mosquitoes, the number of susceptible mosquito population falls from the initial condition to a steady state as a result of the effectiveness of the trap. There is a corresponding effect on the number of infectious mosquitoes in *Figure 3b* as the trap acts as an attractor of all classes of mosquitoes. Consequently, the disease dies out as the number of infectious mosquitoes reduces to zero, culminating in a general reduction of the entire mosquito population as can be observed in *Figure 4b*. We note from *Figure 3a* and *Figure 4a* that at the disease free equilibrium point, the total dimensionless value of the number of susceptible mosquitoes, S_m and the number of the trapped mosquitoes, T_m is expressed as, $\frac{1}{1+\eta} + \frac{\eta}{1+\eta} = 1$. From our assumption, the mosquitoes in the trap cannot escape from the trap. Thus their buid-up in the trap does not pose any threat to disease transmission. Thus this compartment only play the role of the recovered or removed in the original *SIR* model. Using the values of our model parameters, we obtain $S_{m=0.33712}$ and $T_{m=}0.66288$. The disease free-state is a situation where the basic reproduction number is less than unity and in this case $R_{0=}0.39077$, precisely.

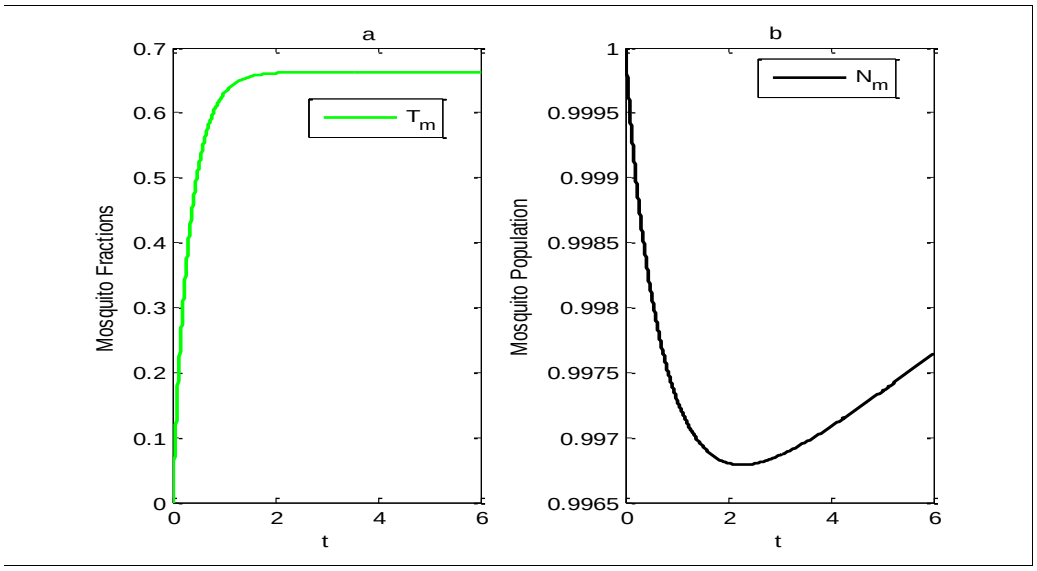
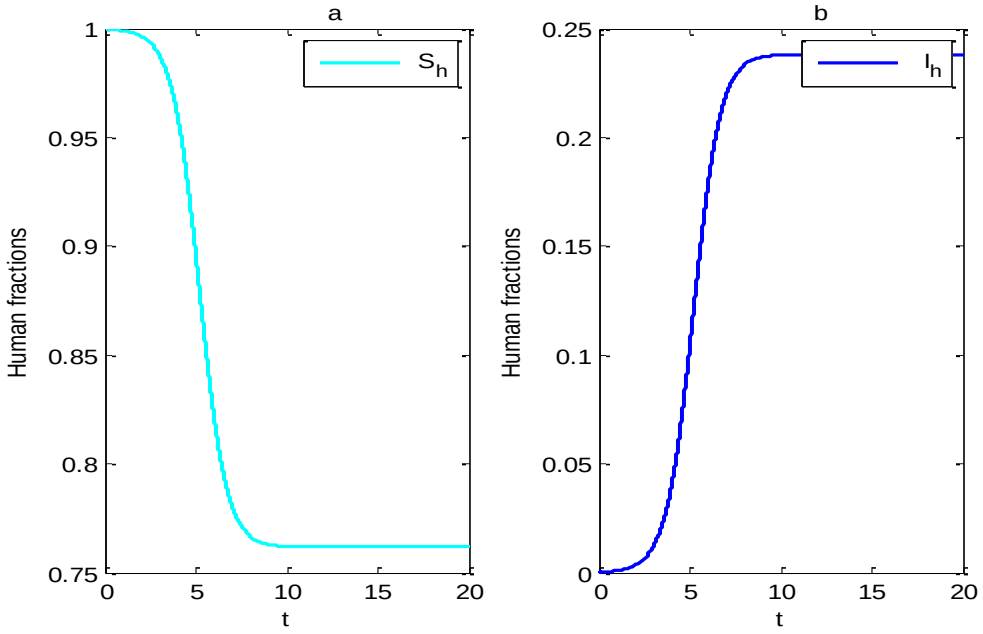


Figure 4. Results showing the effect of the mosquito trap on the entire mosquito population. The initial conditions and parameter values are the same as those in Figure 3.

In the case of the basic reproduction number being greater than unity, the trap effect, $\eta < 0.84$ and there is a fall in the population of susceptible humans as the disease continues to grow causing the infectious humans population to grow. Figure 4 describes the endemic state of the disease for both human and mosquito fractions, where $R_0 > 1$. In *Figure 5a,b,* the number of susceptible humans declines as more humans get infected. While Figure *5c,d,* describe that of the human population. Here, we have used $\eta = 0.012886$ with values of the other parameters to obtain $R_0 = 3.2088$.



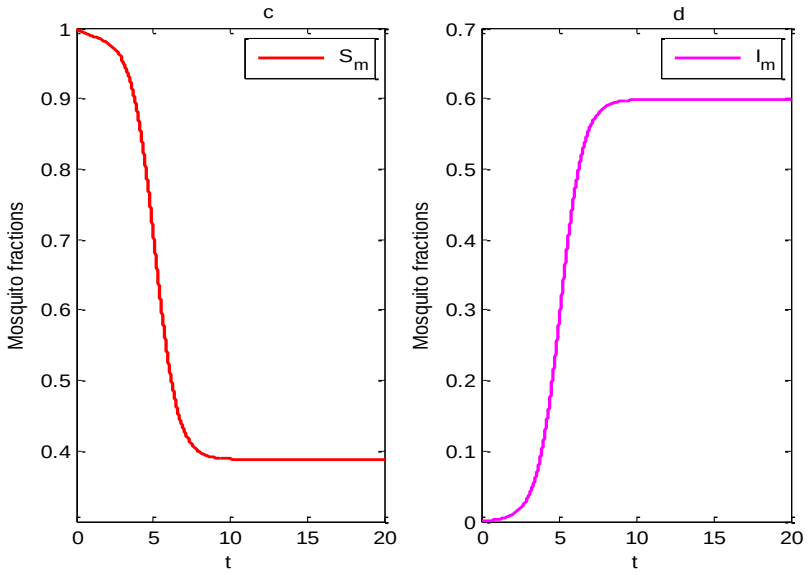


Figure 5: Results showing the endemic state in the human and mosquito compartments by reducing the trap effectiveness. The initial conditions used are $S_h = 1,\ I_h = 0,\ S_m = 0.999,\ I_m = 0.001, T_m = 0,\ N_h = 1, N_m = 1$ *and the parameter values are the same as those in Figure 3 except that we have used* $\eta = 0.012886$.

5. Discussion and Conclusion

Over the years, methods and procedures have been formulated by varies mathematicians and scientists across the globe to tackle the problem of malaria transmission. Vector based control methods used so far, including the use of treated mosquito bed-nets are not effective enough to control mosquito population. Mosquitoes sometimes sneak into the net through tiny/small holes within the net and can still infect susceptible humans or be infected by infectious humans. A treated mosquito net can lose its concentration after a period of time. Therefore, mosquito nets are not *100%* effective tackling the problem of malaria.

Controlling mosquitoes with very toxic and concentrated chemicals (insecticides) either in liquid or gaseous form may be very harmful to humans and it is capable of causing health challenges like catarrh, sneezing, asthma and other complications. Therefore, chemicals are not medically advisable to be used.

Some inventors and scientists have gone ahead to create electrical devices that eventually become useless in the absence of electricity.

Vaccines are not always made available for individuals that are not financially buoyant especially those in rural areas. Therefore the trapping system introduced in this work could be safe and reliable for the control and/or even elimination of malaria.

In the model, the graphs of the susceptible humans and infectious humans are always inversely related. As the disease dies out, the number of susceptible humans grows to a limit even as the number of infectious humans reduces to zero. The trap is made to catch both susceptible and infectious mosquitoes. This keeps the susceptible mosquitoes to a particular level, given in the disease free equilibrium state as the number of infectious mosquitoes reduces to zero. In *Figure 4a*, the number of trapped mosquitoes grows to a steady state as given in the disease free equilibrium. The total mosquito population initially drops due to the immergence of the disease and grows to a steady state as the disease dies out. The initial decline in the total population of mosquitoes indicates a reduction in contact between mosquitoes and humans.

It can be easily observed that mosquitoes sense the presence of the human host at night, and also in dark places in the day. We propose that the human body may be radiating some biochemical substances in the form of smell that make them very attractive to mosquitoes. We recommend that scientists and health organizations across the globe, take up the task of finding the chemical substance within the human body

responsible for giving away human identity to the mosquitoes. This will enable them to produce another substance of higher concentration that would be more attractive to mosquitoes than the one found in humans, so that an effective mosquito trap could be produced. The trap should be made in such a way that would attract mosquito preference for the trap instead of the human host.

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