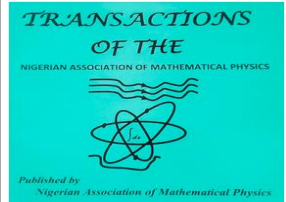


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THE ROLE OF MOSQUITO TRAP IN MALARIA CONTROL- A MATHEMATICAL APPROACH)

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ABSTRACT

We propose a simple mathematical model of malaria transmission involving a system of five ordinary differential equations with only susceptible and infectious classes of both humans and mosquitoes and a new class of trapped mosquitoes. The motivation is to use mathematical approach to analyze the role of mosquito trap in malaria control. We obtain the basic reproduction number, R_0 and found that the trap effectiveness is a key parameter that drives the dynamics of the disease. The analytical results show that, for $R_0 < 1$, the disease-free equilibrium point is locally asymptotically stable and globally asymptotically stable in the absence of disease related death, and unstable for $R_0 > 1$. We found from the numerical solution that with the given parameter values in the absence of mosquito trap, malaria infection may be as high as 80% within six months of introduction of few infected mosquitoes into an entirely susceptible population. Although, other parameters like the infection rates of both humans and mosquitoes can cause the disease to invade the population when the level of trap effectiveness is low but trap effectiveness very close to 1 may likely lead to disease eradication.

1. Introduction

Malaria pathogenesis has been an age-long disease that has inflicted mankind especially those living in tropical regions of the world for so many years. The plasmodium parasite, which is the cause of malaria is first introduced into the human body by a blood feeding female anopheles mosquito vector. The parasite survives in different stages causes flue-like symptoms that sometime degenerates into life threatening complications causing more deaths in vulnerable pregnant women and children between age zero and five years.

The World Health Organization estimates in the *2021 World malaria report* that, there were **241 million malaria cases** and **627,000 malaria deaths** worldwide in 2020 [1], following a trend from about 14 million

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additional cases with 69 thousands more deaths in 2020 compared to 2019 figure. Various health researchers and organizations have carried out studies on understanding the biology of malaria pathogenesis including factors determining the parasite load, vectorial capacity, human host configuration, etc., in order to control, eliminate and eradicate the disease. Some measures have been taken to reduce the parasite load within the human host through the administration of antimalarial drugs like quinine, chloroquine, Sulfadoxine-Pyrimethamine, mefloquine, artemisinin, etc.[2]. Host–vector contact is a critical parameter that integrates many factors driving disease transmission [3]. Thus any malaria control strategy that discourages or reduces human- mosquito contact is a lead way to malaria eradication.

Various mathematical models have been used to explain the dynamics of malaria transmission since Ronald Ross's pioneering work on mosquito threshold phenomenon and its impact on malaria elimination. These models that have been reviewed in [4], among others have assisted in giving an insight into the interaction between the host and vector population, the dynamics of malaria, how to control malaria transmission, and eventually how to eradicate it [5]. A human-mosquito interaction model was analyzed in [6] and their findings suggest that malaria could be controlled by reducing the contact rate between human and mosquito, through the use of insecticides and mosquito treated nets, and also the use of active malaria drugs which can help to reduce mosquito population and malaria transmission respectively. Various attempts have been made to eliminate malaria through these strategies and unfortunately these efforts have been reduced to mere control with no sure way in sight in the near future to eliminating the disease. The discovery of malaria vaccine would have been a key to disease elimination but despite the discovery of malaria vaccine people still fall sick after use and it is still only 30% effective against death. Hence, the recommendation of its use in combination with other therapeutic or preventive drugs [7]. Due to some intervening variables with inherent refractory characteristics usually described as parasite resistance to some or all of these control measures ever introduced, the global effort or the road map to malaria elimination seems to be a vicious circle. Antimalarial drug resistance has been reviewed in [8] and mathematical models on mosquito resistance have been proposed and analyzed, some of which are given in [8-11]. In the work of [10], two mathematical models on human antimalarial drug resistance and mosquitoes insecticides resistance together with human–mosquitoes population mobility in patches were analyzed, and their numerical solution confirmed the theoretical results of existence of a forward bifurcation and the global stability of a disease–free equilibrium. Although the use of bed-nets is believed to be effective because it drastically reduces the contact rate between human and mosquito, it is still worrisome that the decay of bed-nets poses danger to the advantage derived from their use. Thus the work of [12] analyzes the impact of decay in bed-nets efficacy on malaria transmission contending that the potential impact of Insecticide Treated Nets on reducing malaria transmission is limited due to inconsistent or improper use, as well as physical decay in effectiveness. They therefore suggest the provision of bed nets with longer life span and increase in bed-net coverage.

The war against malaria is a two way process namely, chemotherapy (fighting the parasite in the human body to reduce or eliminate disease burden) and fighting the vector (mosquito) and preventing it from transmitting the parasite to humans through the use of bed nets, insecticides, repellent devices in form of cream and otherwise. Fighting the vector seems to be a better way in that this will prevent humans from contacting the disease and there would be no need for chemotherapy which involves introducing some chemicals into the human system that could be detrimental to health even as care should be taken in the use of some chemicals that may be detrimental to human health in trying to fight the vector. The use of

ineffective mosquito killer lamps or Led Light Trap lamps has been introduced in the public domain with a lot of them being advertised online.

To the best of our knowledge there no mathematical model that has incorporated this idea. We therefore propose a mathematical model with a mosquito trap. In section 1 we present the introduction and the general background. We present the model formulation in section 2 and the analysis in section 3. The numerical solution is given in section 4 and the paper is rounded up with discussion and conclusion in section 5.

2. Model Formulation

We propose a human-mosquito interaction model comprising five compartments. The human and mosquito populations are represented by N_h and N_m respectively, and their compartmental descriptions are given by number of susceptible humans (S_h), number of infectious humans (I_h), number of susceptible mosquitoes (S_m), number of infectious mosquitoes (I_m) and number of trapped mosquitoes (T_m). The human and mosquito populations are described by the equations

$$N_h = S_h + I_h \quad (1)$$

$$N_m = S_m + I_m + T_m \quad (2)$$

Susceptible humans become infected through an infectious bite by a female Anopheles mosquito at a rate, $\beta_1 S_h \frac{I_m}{N_m}$ where, β_1 is a rate constant. The fraction, $\frac{I_m}{N_m}$ is the contact probability between infectious mosquitoes. Susceptible humans are recruited into the population through a constant birth rate, λ_h and the human population dies naturally at a per capita rate, μ_h while some individuals in the infectious class die at an additional rate, α_h from the disease. We also assume that infectious humans also infect susceptible mosquitoes as susceptible mosquitoes feed on them. Thus, the transition of susceptible mosquitoes into the infectious class is expressed by the rate, $\beta_2 S_m \frac{I_h}{N_h}$ in which the fraction, $\frac{I_h}{N_h}$ indicates the probability of contact between infectious humans and susceptible mosquitoes and β_2 representing the infectious rate constant. Mosquitoes get attracted to the mosquito trap at a rate, $\beta_2 \theta S_m \frac{I_h}{N_h}$. Out of the total infectious mosquitoes, a fraction, $1 - \theta$ is attracted to humans and a fraction, θ , where $0 \leq \theta \leq 1$ is attracted to the mosquito trap. We note that θ is the mosquito trap effectiveness and β_1 is the contact rate, where the effect of the contact rate is controlled by θ . We assume that, all mosquitoes die naturally at a rate, $\mu_m N_m$ and also die at a rate $\alpha_m I_m$ as a result of carrying the plasmodium parasite. The proposed model consistent with the above assumptions is given by:

$$\frac{dS_h}{dt} = \lambda_h N_h - \beta_1 S_h \frac{I_m}{N_m} - \mu_h S_h \quad (3)$$

$$\frac{dI_h}{dt} = \beta_1 S_h \frac{I_m}{N_m} - \alpha_h I_h - \mu_h I_h \quad (4)$$

$$\frac{dS_m}{dt} = \lambda_m N_m - \beta_2 S_m \frac{I_h}{N_h} - \mu_m S_m \quad (5)$$

$$\frac{dI_m}{dt} = \beta_2(1 - \theta)S_m \frac{I_h}{N_h} - \alpha_m I_m - \mu_m I_m \quad (6)$$

$$\frac{dT_m}{dt} = \beta_2 \theta S_m \frac{I_h}{N_h} - \mu_m T_m \quad (7)$$

$$\frac{dN_h}{dt} = \lambda_h N_h - \alpha_h I_h - \mu_h N_h \quad (8)$$

$$\frac{dN_m}{dt} = \lambda_m N_m - \alpha_m I_m - \mu_m N_m \quad (9)$$

Equation (8) is obtained by adding equations (3)-(4) and equation (9) is obtained by adding equations (5)-(7).

2.1 Parameter Values and Nondimensionalisation

The model parameters are listed in Table 1 below together with values taken from relevant sources.

Table 1. List of model parameters.

Symbols	Description	Value	Units	Source
λ_h	Constant recruitment rate of humans into the susceptible class	0.0000433	Day^{-1}	[4,6,8]
θ	Effectiveness of mosquito attractor	0.00000123	Dimensionless	[4,6,8]
β_1	Infection rate of susceptible humans by infectious mosquitoes	0.0987	Day^{-1}	[4,6,8]
k_1	Killing rate of trapped mosquitoes	0.00024	Day^{-1}	[4,6,8]
μ_h	Per capita death rate of humans	0.0000357	Day^{-1}	[4,6,8]
μ_m	Per capita death rate of mosquitoes	0.035	Day^{-1}	[4,6,8]
β_2	Infection rate of susceptible mosquitoes by infectious humans	0.854	Day^{-1}	[4,8,14]
α_h	Disease related death rate of infectious humans	0.05	Day^{-1}	[15]
α_m	Malaria related death rate of infected mosquitoes	0.03152	Day^{-1}	[16]

We rescale the variables using the following definitions:

$$U = \frac{S_h}{N_h}, V = \frac{I_h}{N_h}, W = \frac{S_m}{N_m}, X = \frac{I_m}{N_m}, Y = \frac{T_m}{N_m}, H = \frac{N_h}{H_0}, M = \frac{N_m}{M_0}, \hat{t} = \frac{t}{t_0} \quad (10)$$

to obtain

$$U + V = 1 \quad (11)$$

$$W + X + Y = 1 \quad (12)$$

The time derivatives for the variables will become, using the variable S_h as an example,

$$\frac{dUN_h}{dt} = N_h \frac{dU}{dt} + U \frac{dN_h}{dt} = N_h \frac{dU}{dt} + \left(\lambda_h - \alpha_h \frac{V}{N_h} - \mu_h \right) UN_h. \quad (13)$$

We rescale time with the Infection rate of susceptible mosquitoes by infectious humans and by substituting (10) in (3) – (9),

and after performing some algebraic simplifications we define the following dimensionless parameters.

$$t_0 = \frac{1}{\beta_2}, \lambda = \frac{\lambda_h}{\beta_2}, \beta = \frac{\beta_1}{\beta_2}, \alpha = \frac{\alpha_h}{\beta_2}, a = \frac{\lambda_h}{\beta_2}, b = \frac{\alpha_m}{\beta_2}, \mu = \frac{\mu_h}{\beta_2}, d = \frac{\mu_m}{\beta_2}, \quad (14)$$

and after dropping the hats for notational simplicity, we have the following nondimensional system;

$$\frac{dU}{dt} = \lambda(1 - U) - \beta UX + \alpha UV \quad (15)$$

$$\frac{dV}{dt} = \beta UX - (\alpha + \lambda)V + \alpha V^2 \quad (16)$$

$$\frac{dW}{dt} = a(1 - W) - VW + bWX, \quad (17)$$

$$\frac{dX}{dt} = (1 - \theta)WV - (a + b)X + bX^2, \quad (18)$$

$$\frac{dY}{dt} = \theta WV - aY + bXY, \quad (19)$$

$$\frac{dH}{dt} = (\lambda - \mu)H - \alpha VH, \quad (20)$$

$$\frac{dM}{dt} = (a - d)M - bXM, \quad (21)$$

System (15) – (21) is to be analysed with (11) and (12), subject to the initial conditions,

$U(0) = 1, V(0) = 0, W(0) = w_0, X(0) = 1 - w_0, Y(0) = 0$, where we have considered introduction of few infectious mosquitoes in an entirely susceptible human population.

3.0 Analysis of the Model

3.1 Establishing the Disease Determining Threshold Parameter, R_0

Here we apply the next generation matrix method used in [12, 13, 14], by considering the equation

$$C' = \frac{dC}{dt}, \text{ where} \quad (22)$$

$$C' = DC - EC$$

$$D = \begin{bmatrix} 0 & \beta & 0 \\ 1 - \theta & 0 & 0 \\ \theta & 0 & 0 \end{bmatrix}, E = \begin{bmatrix} \alpha + \lambda & 0 & 0 \\ 0 & a + b & 0 \\ 0 & 0 & a \end{bmatrix}, C = \begin{bmatrix} V \\ X \\ Y \end{bmatrix}$$

Here, DC represents the emergence of new infections, EC is the distribution of these infections among compartments and

C , the infection carrying matrix.

The largest eigenvalue of $G = DE^{-1}$ given by

$$G = \frac{1}{g_0} \begin{bmatrix} 0 & \beta(\alpha + \lambda)(a) & 0 \\ a(1 - \theta)(a + b) & 0 & 0 \\ \theta a(a + b) & 0 & 0 \end{bmatrix}, \quad (23)$$

gives the basic reproduction number, R_0 , where

$$R_0 = \frac{\beta(1-\theta)}{(a+b)(\alpha+\lambda)} \quad (24)$$

Here we have used square of the spectral radius of G to represent its largest eigenvalue instead of the bigger of its square root. This is an assumption used by the original work using the new generation matrix method [16].

3.2 Positivity, Existence and Uniqueness of Solution

The model is described in the domain

$$\Omega \in \mathbb{R}^8 = \{Q, L, S, A, R, V, P, N: Q \geq 0, L \geq 0, S \geq 0, A \geq 0, R \geq 0, V \geq 0, P \geq 0, N > 0, Q + L + S + A + V + R = 1\} \quad (25)$$

Assuming all variables are positive at $t = 0$, then $Q(0) + L(0) + S(0) + A(0) + V(0) + R(0) = 1$. If $L = 0$, and all other variables are in Ω , then, $\frac{dL}{dt} \geq 0$, this is also the case for variables in (2.15) - (2.19). If $N = 0$, then, $\frac{dN}{dt} = 0$. But if $N > 0$ and assuming $\lambda > \mu$, then with suitable initial conditions, $\frac{dN}{dt} > 0 \forall t > 0$. We observe that the right-hand side of (2.15) - (2.20) is continuous with continuous partial derivatives. Thus, solutions exist and are unique and the model has mathematically and biologically relevant solutions in the domain $\Omega \forall t \in [0, \infty)$.

3.3 Local Stability Analysis of the Disease Free State

The disease free equilibrium point is given by $(U, V, W, X, Y) = (1, 0, 1, 0, 0)$. We will derive sufficient conditions for the stability of the disease-free state.

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

Proof:

Considering the following Jacobian matrix of the system of equations (15) – (19).

$$J_{cob} = \begin{bmatrix} -\lambda & a & 0 & -\beta & 0 \\ 0 & -h_1 & 0 & \beta & 0 \\ 0 & -1 & -a & b & k \\ 0 & r_1 & 0 & -h_2 & 0 \\ 0 & \theta & 0 & 0 & -h_3 \end{bmatrix}, \quad (26)$$

Its characteristic equation in terms of the eigenvector δ , is given by

$$(\delta + \lambda)(\delta^4 + L_1\delta^3 + L_2\delta^2 + L_3\delta + L_4) = 0, \quad (27)$$

Where the h_i , s and L_i 's are defined below in terms of the model parameters;

$$h_1 = a + \lambda, \quad h_2 = a + b, \quad h_3 = a + k, \quad r_1 = 1 - \theta, \quad h_4 = h_1 + h_2 + h_3, \quad h_5 = h_1h_2, \quad h_6 = h_2h_3, \quad h_7 = h_1h_3, \quad h_8 = h_3h_5,$$

$$h_9 = h_6 + h_7, \quad h_{10} = (h_1 + h_2)(a + h_3)h_5(1 - R_0)^2, \quad h_{11} = h_5(h_1 + h_2)(a + h_3)(a^2 + a(h_1 + h_2) + h_9 + h_3^2)(1 - R_0),$$

$$h_{12} = ah_3h_4(h_1 + h_2)(a + h_3)(a + h_1 + h_2), \quad L_1 = a + h_4, \quad L_2 = ah_4 + h_9 + h_5(1 - R_0), \quad L_3 = ah_9 + h_5(a + h_3)(1 - R_0),$$

$$L_4 = ah_8(1 - R_0), \quad (28)$$

and R_0 is as defined in (24) above. We note that one of the eigenvalues of (27) is strictly negative and if $R_0 < 1$, then the coefficients of the quartic polynomial are all positive and non-zero; and by the Descartes' rule of signs there is no positive real eigenvalue. This means there are 4 negative real eigenvalues or 2 negative real eigenvalues and a

complex conjugate pair, or two pairs of complex conjugate eigenvalues. Hence, the

Routh Hurwitz stability criterion for a quartic polynomial as stated in

[17] and expressed in our case as $\rho = L_1L_2L_3 - (L_3^2 + L_1^2L_4) > 0$ is satisfied.

Some algebraic simplifications yields

$$\rho = h_{14}(1 - R_0)^2 + h_{16}(1 - R_0) + h_{17},$$

Where

$$h_{13} = (h_1 + h_2)(a + h_3), \quad h_{14} = h_5^2h_{10}, \quad h_{15} = a^2 + a(h_1 + h_2) + h_9 + h_3^2, \quad h_{16} = h_5h_{13}h_{15}, \quad h_{17} = ah_3h_4h_{13}(a + h_1 + h_2).$$

But if $R_0 > 1$, then there will be at least one sign change or one positive root meaning the solution may change its stability status. Thus the point $R_0 = 1$ is a bifurcation point in the parameter space $(\lambda, \beta, \alpha, \theta, a, b, \mu, d, k)$.

3.4 Global Stability Analysis of the Disease Free Equilibrium (E_0)

For easier usage, we explore the method used in [14, 18-20] to show the global stability of the disease-free equilibrium point.

Lemma

The disease-free equilibrium point of the model equations is globally asymptotically stable if $R_0 < 1$ and the additional conditions, H_1 and H_2 are satisfied. These are;

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

$$H_2: \widehat{G}(A, B) = QB - G(A, B) \geq 0 \forall (A, B) \in E_0$$

Proof

The model equations can be expressed as follows:

$$\begin{aligned} \frac{dA}{dt} = F(A, B) &= \begin{bmatrix} \lambda(1 - U) - \beta DUX + \alpha UV \\ a(1 - W) - VW + BWX + KVW \\ \theta VW - aY + bXY \end{bmatrix} \\ \frac{dB}{dt} = G(A, B) &= \begin{bmatrix} \beta UX - (\alpha + \lambda)V + \alpha V^2 \\ (1 - \theta)V - (a + b)X + bX^2 \end{bmatrix} \end{aligned}$$

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

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

$$F(A, 0) = \begin{bmatrix} \lambda(1 - U) \\ a(1 - W) \\ 0 \end{bmatrix} \tag{6}$$

From (1);

$$\frac{dU}{dt} = \lambda(1 - U) \frac{dU}{dt} + \lambda U = \lambda \tag{7}$$

Solving (7) using the method of integrating factor, we have;

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

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

Similarly, $W(t) = 1, \text{ as } t \rightarrow \infty.$

Hence, H_1 is satisfied.

$$\begin{aligned} \frac{dB}{dt} = G(A, B) &= \begin{bmatrix} \beta UX - (\alpha + \lambda)V + \alpha V^2 \\ (1 - \theta)VW - (a + b)X + bX^2 \end{bmatrix} \\ Q &= \begin{bmatrix} -(\alpha + \lambda) & \beta U \\ (1 - \theta)W & -(a + b) \end{bmatrix} \\ QB &= \begin{bmatrix} -(\alpha + \lambda) & \beta U \\ (1 - \theta)W & -(a + b) \end{bmatrix} \begin{bmatrix} V \\ X \end{bmatrix} \\ &= \begin{bmatrix} \beta UX - (\alpha + \lambda)V \\ (1 - \theta)VW - (a + b)X \end{bmatrix} \\ \widehat{G}(X, Y) = QB - G(A, B) & \\ = \begin{bmatrix} \beta UX - (\alpha + \lambda)V \\ (1 - \theta)VW - (a + b)X \end{bmatrix} - \begin{bmatrix} \beta UX - (\alpha + \lambda)V + \alpha V^2 \\ (1 - \theta)VW - (a + b)X + bX^2 \end{bmatrix} & \\ \therefore \widehat{G}(X, Y) = \begin{bmatrix} -\alpha V^2 \\ -bX^2 \end{bmatrix} & \\ \rightarrow \widehat{G}(A, B) \geq 0 \forall (A, B) \in E_0 \text{ iff } \alpha = b = 0. & \end{aligned}$$

This satisfies condition, H_2 . Thus, the equilibrium point is globally asymptotically stable.

3.6 Numerical Solution

Numerical simulations are carried out using MATLAB's ODE15s, with the following values of the dimensionless parameters:

$\lambda = 0.0439$, $\beta = 0.23$, $\alpha = 0.025$, $a = 0.157$, $b = 0.1485$, $k = 0.136$, $\mu = 0.0253$, $\theta = 0.2$, $\gamma = 0.0067$, $d = 0.0403$, with initial conditions, $U = 1$, $V = 0$, $W = 0.999$, $X = 0.001$, $Y = 0$, $N = 1$, $M = 1$.

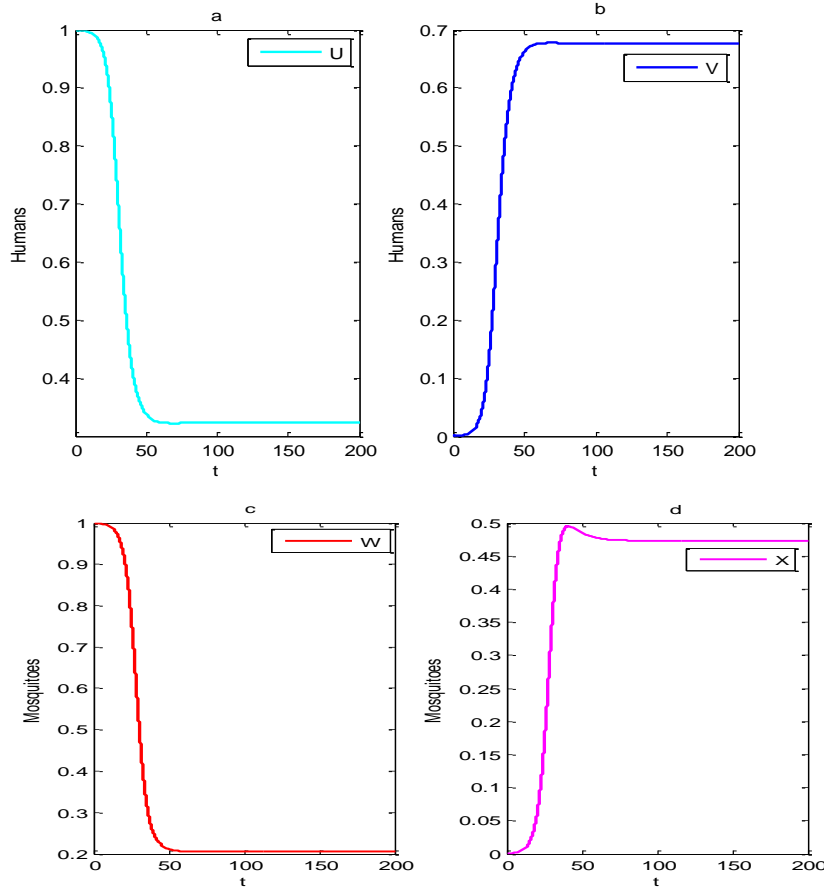
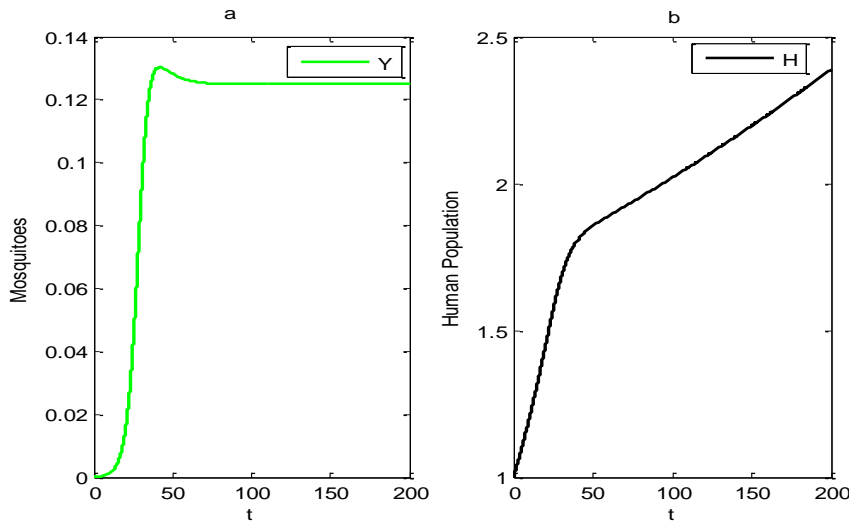


Figure 1. Results showing the effect of the disease on the human and mosquito compartments, where $t = 1$, represents approximately 1.2 days in real time. The initial conditions used are $U = 1$, $V = 0$, $W = 0.999$, $X = 0.001$, $Y = 0$, $N = 1$, $M = 1$ and the parameter values are $\lambda = 0.0439$, $\beta = 0.23$, $\alpha = 0.025$, $a = 0.157$, $b = 0.1485$, $k = 0.136$, $\mu = 0.0253$, $\theta = 0.2$, $\gamma = 0.0067$, $d = 0.0403$. $R_0 = 8.74$



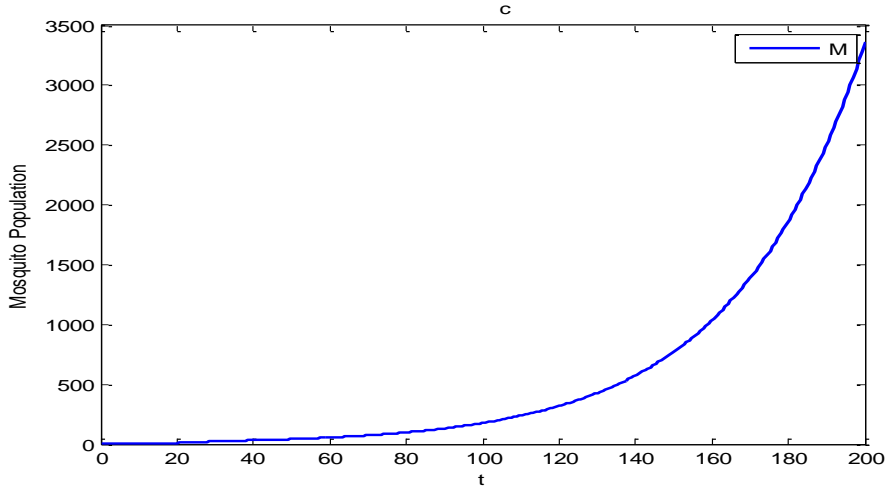


Figure.2. Results showing the effect of the disease on trapped mosquitoes, human population and mosquito population, where $t = 1$, represents approximately 1.2 days in real time. The initial conditions and parameter values used are the same as those in Figure 1.

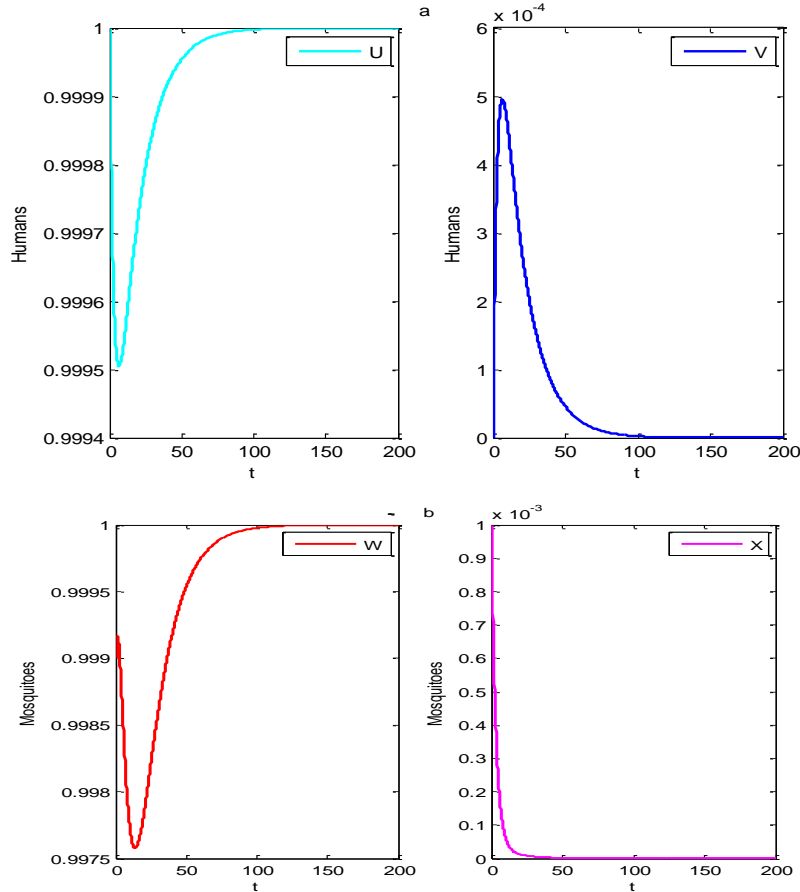


Figure 3. Results showing the effect of mosquito trap on mosquito infectiousness and susceptibility. The initial conditions and parameter values used are the same as those in Figure 1 except that we have used $\theta = 0.9$ to beef up the effectiveness of the trap. Here, $R_0 = 0.109$.

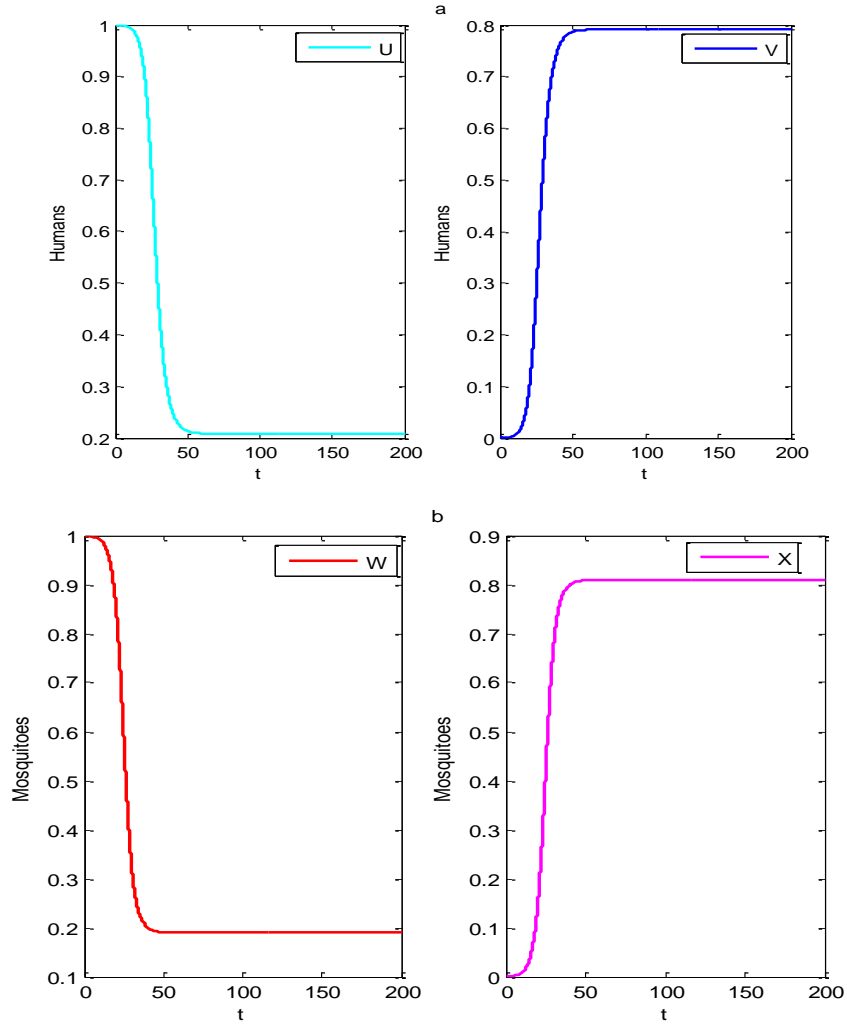


Figure.2. Results showing disease transmission in the absence of mosquito trap or when the trap is completely ineffective. The initial conditions and parameter values used are the same as those in Figure 1 except that we have used $\theta = 0$.

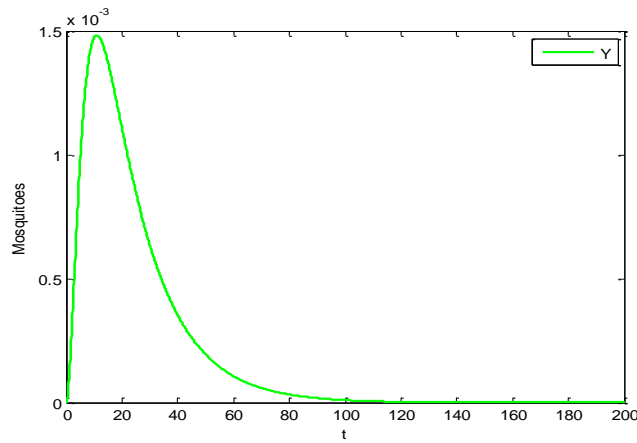


Figure 4. Results showing the impact of mosquito trap effectiveness on trapped mosquitoes. The initial conditions and parameter values used are the same as those in Figure 3.

3.5 Discussions

In this model, we describe the transmission dynamics of malaria in an entirely susceptible human population due to the introduction of a single case and the effect of mosquito trap on Disease control. Even though malaria transmission data may vary regionally, the use of mosquito trap in malaria control may have some global effect. However, we have used some available global data in conjunction with mosquito trap to demonstrate the possibility of malaria eradication. Our analyses shows that the effectiveness of the trap appears to have reasonable impact on the Basic Reproduction Number, R_0 , which suggests a likelihood of the disease decaying in the presence of mosquito trap as seen in figure.3a,b,c,d and figure 4. In Figure 3a and 3b, the number of infectious humans and infectious mosquitoes die out as the number of susceptible humans and mosquitoes recover. This is due to a boost or an improvement in the trap effectiveness, θ , from 0.2 to 0.9. A comparison of the number of mosquitoes in the trap as shown in Figure 1d and Figure 4, shows that an effective trap could eliminate mosquitoes in the trap, which hitherto could be reserved by an ineffective mosquito trap. Figure 2a and Figure 2b depict a situation where there is complete ineffectiveness or absence of mosquito trap and the disease dynamics is only driven by the parameters β , a , b , α and λ . With the given values of these parameters in the absence of mosquito trap, the results show an endemic scenario where more than 80% of mosquitoes and a little below 80% of humans may likely be infected within about six months of introducing few infectious mosquitoes in an entirely susceptible human population.

These results demonstrate the likely positive contribution of mosquito trap to possible disease eradication. The analysis shows that the disease free-state is globally asymptotically stable in the absence of disease related death of both humans and mosquitoes. We note from the basic reproduction number that even in the presence mosquito trap, malaria infection may continue to increase if the trap is not very effective. Although the trap effectiveness is a hypothetical parameter, it is a measure to provide guidance to mosquito trap manufacturers to effectively consider the biochemical substances that make humans attractive to mosquitoes and make the trap more attractive so that mosquitoes would have preference for the trap instead of humans. This could be interpreted from the model as the trap effectiveness, θ , being as close as possible to unity.

4. Conclusion

In this work, we presented a mathematical model on the role of mosquito trap in malaria control. The model describes the effect of mosquito trap in malaria transmission and control in a totally susceptible population due to the introduction of few infectious mosquitoes. Analysis of the model shows that with the use of effective mosquito trap the disease may likely die out. Recent times the manufacturing of ineffective mosquito killer lamps or Led Light Trap lamps is very alarming with a lot of them being advertised online. A lot of people fall victim to purchasing these lamps that could not attract a single mosquito, a practical experience of the one of the authors of this article, which took him time to get refund of part of the money. Mosquito trap or killer lamp manufactures should engage the services of professional researchers to unravel the biochemical component of mosquito-human attractor, which will serve as a basis of an effective mosquito trap.

References

- [1] WHO, World Malaria Report, 2021. [online]. [Viewed 10/06/2022] <https://www.who.int/teams/global-malaria-programme/reports/world-malaria-report-2021>
- [2] Institute of Medicine (US) Committee on the Economics of Antimalarial Drugs; Arrow K. J., Panosian, C., Gelband, H., editors. Saving Lives, Buying Time: Economics of Malaria Drugs in an Age of Resistance. Washington (DC): National Academies Press (US); 2004. 5, A Brief History of Malaria.

- [3] Thongsripong, P., Hyman, J. M., Kapan, D. D., Bennett, A. N. Human–Mosquito Contact: A Missing Link in Our Understanding of Mosquito-Borne Disease Transmission Dynamics, *Annals of the Entomological Society of America*, Volume 114, Issue 4, July 2021, Pages 397–414, <https://doi.org/10.1093/aesa/saab011>
- [4] Mandal, A., Sarkar, R. R., Sinha, S. Mathematical models of malaria - A review *Malaria Journal*. 2011;10(1):202: DOI:10.1186/1475-2875-10-202.
- [5] Cobremeskel, A. A., Krogstad, H. E. Mathematical modeling of endemic transmission. *American journal of Applied Mathematics*. 2015;3(2):36-46.
- [6] Mojeeb A., EL-Nor, I., Isaac, K. A. Simple Mathematical Model on Malaria Transmission. *Journal of Advances in Mathematics and Computer Science*. 2017; 25(6):1-24. DOI: 10.9734/JAMCS/2017/37843
- [7] First Malaria Vaccine – A Major Milestone Despite Huddles Ahead. WebMD. WebMD, 2 December 2021. Web. 30 May 2022.
- [8] Agosto F. B., “Malaria drug resistance: The impact of human movement and spatial heterogeneity”, *Bull. Math. Biol.*, 76 (2014), No. 7, 1607–1641. doi: 10.1007/s11538-014-9970-6 [Links]
- [9] Tchuente J. M., Chiyaka C., Chan D., Matthews A. and Mayer G., “A mathematical model for antimalarial drug resistance”, *Math. Med. Biol.*, 28 (2011), No. 4, 335–355. doi: 10.1093/imammb/dqq017 [Links]
- [10] Montoya, C., Romero–Leiton, J. P., Mathematical modelling for malaria under resistance and population movement. *Integración - UIS* [online]. 2020, vol.38, n.2, pp.133-163. Epub June 30, 2020. ISSN 0120-419X. <https://doi.org/10.18273/revint.v38n2-2020006>.
- [11] Aneke S., “Mathematical modelling of drug resistant malaria parasites and vector populations”, *Math. Methods Appl. Sci.*, 25 (2002), No. 4, 335–346. doi: 10.1002/mma.291 [Links]
- [12] Ngonghala, C. N., Del Valle, S. Y., Zao, R., Mohammed- Awel, J., Quantifying the impact of decay in bed-net efficacy on malaria transmission. 2014; *J of Theor Biol.* 363, 247-261. Doi: 10.1016/j.jtbi.2014.08.018
- [13] Okrynya, A. B., Consul, J. L., Logistic mathematical model of Ebola virus disease with convalescence. *International Journal of Applied Scientific and Research*. 2(6) (2019), 1-14.
- [14] Okrynya, A. B., Timinibife C. N., Global Stability Analysis of a Mathematical Model on the Transmission Dynamics of Covid-19 with Vaccination. *International Journal of Mathematics and Computer Research*. 10 (12) (2022), 3039-3049. DOI: 10.47191/ijmcr/v10i12.06
- [15] Chitnis, N., Cushing, J. M. Hyman, J. M., Bifurcation analysis of a mathematical model or malaria transmission. *SIAM J. Appl. Math.* 67 (2006), 24-45.
- [16] Okrynya, A. B., Mathematical modelling of Malaria Transmission and Pathogenesis, PhD Thesis, Loughborough University, 2015.
- [17] Allen, L. J. S. (2007). *An Introduction to Mathematical Biology*, Prentice Hall, Upper Saddle River, N. J.
- [18] M. O. Onuorah, and N. I. Akinwande, (2016). Sensitivity analysis of Lassa fever model. *European Centre for Research Training and Development UK*. (www.eajournals.org).14(1)

- [19] B. M. Ndiaye, and L. S. Tendeng, (2020). Analysis of the COVID-19 pandemic by SIR model and machine learning techniques for forecasting. Available <https://doi.org/10.48550/arXiv.2004.01574>
- [20]BT. Chen, J. Rui, Q. Wang, Z. Zhao, J. Cui, L. Yin, (2020). A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. *Infectious Diseases of Poverty*. 9(24). Available:<https://doi.org/10.1186/s40249-020-00640-3>.