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On the Transmission Dynamics of Malaria with Three Control Strategies

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In this paper, we proposed a non-linear model with three strategies for the control of malaria transmission between the human and the vectors. The model incorporated insecticide treated bed nets, treatment of infected human and the indoor residual spray as the control strategies. The disease-free equilibrium of the model and stability conditions were obtained. We derived the basic reproduction number for the model using next generation matrix approach. The Routh Hurwitz criterion has been applied where we carried out the local and global stabilities of the disease-free equilibrium. However, the results show that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. We carried out numerical simulations using Runge-Kutta of order four and the results revealed that the disease could be curtailed when the three control strategies are adequately implemented.

Keywords: Strategies, Malaria, Transmission, Equilibria, Basic reproduction number

1. Introduction

Malaria is one of the deadliest infectious diseases that have claimed millions of lives around the globe. Malaria in human beings is caused by five species of parasites belonging to the genus *Plasmodium*. Four of these species, *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium malariae* and *Plasmodium ovale*, affect human beings and are spread from one person to another via the bite of female mosquitoes of the genus *Anopheles*. There are about 400 different species of *Anopheles* mosquitoes, but only 30 of these are major vectors. Recently, human infections of malaria due to *Plasmodium knowlesi* have been recorded – these species of malaria are usually found among monkeys in certain forested areas of South-East Asia. Current information suggests that *Plasmodium knowlesi* malaria is not spread from person to person, but rather occurs in people when an *Anopheles* mosquito bites an infected monkey and transmits it to humans (zoonotic transmission) WHO [16]. They also reported that about 3.2 billion people or almost half of the world's population remain at risk of infection by the malaria parasite.

Tumwiine, Mugisha and Luboobi [13] proposed a model that tracks the dynamics of malaria in the human host and mosquito vector. The model incorporates some infected humans that recover from infection and immune humans after loss of immunity to the disease to join the susceptible class again. All the new borne are susceptible to the infection and there is no vertical transmission. The stability of the system is analyzed for the existence of the disease-free and endemic equilibria points. Chitnis, Cushing and Hyman [4] performed sensitivity analyses on a mathematical model of malaria transmission to determine the relative importance of model parameters to disease transmission and prevalence. They compiled two sets of baseline parameter values: one for areas of high transmission and one for low transmission. They computed sensitivity indices of the reproductive number (which measures initial disease transmission) and the endemic equilibrium point (which measures disease prevalence) to the parameters at the baseline values. Olaniyi and Obabiyi [9] presented a seven-dimensional ordinary differential equation, modelling the transmission of *Plasmodium falciparum* malaria between humans and mosquitoes with non-linear forces of infection in form of saturated incidence

rates. Mwangi, Haario and Nannyonga [7] studied the robustness of optimal control solutions under such parameter uncertainty. For the given model simulation, they created data so that a plausible variability of the epidemiological dynamics was covered. Lutambi, Penny, Smith and Chitnis[6] developed and simulated a discrete-space continuous-time mathematical model to investigate the impact of dispersal and heterogeneous distribution of resources on the distribution and dynamics of mosquito populations. They build an ordinary differential equation model of the mosquito life cycle and replicate it across a hexagonal grid (multi-patchsystem) that represents two-dimensional space. Malarial infection could be controlled or prevented through drug treatment of malaria infected patients which would then reduce transmission of the disease, use of insecticide-treated nets (ITNs), indoor residual spraying and, in specific settings, larval control, World Health Organization (WHO) [15]. Otieno, Koske and Mutiso [10] studied a deterministic model for malaria transmission was studied and incorporated the intervention strategies for the most at risk groups (pregnant women and children under five years of age). Analyses of the model for cost effectiveness of the control strategies were undertaken. Silva and Torres [12] studied a Mathematical model for the effects of insecticide treated nets (ITNs) on the transmission dynamics of malaria infection which took into account human behavior and introduced a supervision control, representing information, education, communication (IEC) campaigns for improving the ITN usage. They proposed an optimization model whose aim was to minimize the number of infected human beings while keeping the cost low. They found that an effective and optimal use of preventive measure without the use of larvacide is not possible if total elimination is the objective Ozair *et al.* [11]. Ngwa and Shu [8] analyses a deterministic differential equation model for endemic malaria involving variable human end mosquito populations.

We considered the model of Alhassan, Momoh, Kadzai and Abdullahi [1] which was only used for optimal control and cost effectiveness analysis in controlling the transmission of malaria. So we carried out the stability analysis of the model using Routh-Hurwitz criterion as described in Flores [5] and the numerical simulation using Runge-Kutta of order four.

2. Model Formulation

The model formulation was explained in Alhassan, Momoh, Kadzai and Abdullahi [1].

2.1 Model equations

$$\dot{S}_h(t) = \Lambda_h - [1 - u_1(t)] \frac{p_1 \beta I_v(t)}{N_h(t)} S_h(t) + [\gamma_h + \sigma_h u_2(t)] I_h(t) - \mu_h S_h(t) \quad (1)$$

$$\dot{E}_h(t) = [1 - u_1(t)] \frac{p_1 \beta I_v(t)}{N_h(t)} S_h(t) - \varepsilon_h E_h(t) - \mu_h E_h(t) \quad (2)$$

$$\dot{I}_h(t) = \varepsilon_h E_h(t) - [\gamma_h + \sigma_h u_2(t)] I_h(t) - (\mu_h + \delta_h) I_h(t) \quad (3)$$

$$\dot{S}_v(t) = \Lambda_v - [1 - u_1(t)] \frac{p_2 \beta I_h(t)}{N_h(t)} S_v(t) - (\theta u_3 + \mu_{vb}) S_v(t) \quad (4)$$

$$\dot{E}_v(t) = [1 - u_1(t)] \frac{p_2 \beta I_h(t)}{N_h(t)} S_v(t) - \varepsilon_v E_v(t) - (\theta u_3 + \mu_{vb}) E_v(t) \quad (5)$$

$$\dot{I}_v(t) = \varepsilon_v E_v(t) - (\theta u_3 + \mu_{vb}) I_v(t) \quad (6)$$

The rate of change of the total populations for human and mosquito are given by

$$\dot{N}_h(t) = \Lambda_h - \mu_h N_h(t) - \delta_h I_h(t) \quad (7)$$

$$\dot{N}_v(t) = \Lambda_v - (\theta u_3 + \mu_{vb}) N_v(t) \quad (8)$$

Table 1: Parameters and variables descriptions and values used in the model

Symbols	Description	Estimated Values	References
Λ_h	Recruitment rate in humans	$10^3 / (70 \times 365)$	Silva and Torres [12]
Λ_v	Recruitment rate in mosquitoes	$10^4 / 21$	Silva and Torres [12]
μ_h	Natural mortality rate in humans	$1 / (70 \times 365)$	Silva and Torres [12]
u_1	Control measure for the use of ITNs	0 - 1	Assumed
u_2	Control measure for the use of treatment in humans	0 - 1	Assumed
u_3	Control measure for the use of indoor spray	0 - 1	Assumed
μ_{vb}	Natural mortality rate of mosquitoes	$1 / 21$	Silva and Torres [12]
δ_h	Disease induced mortality rate in humans	10^{-3}	Silva and Torres [12]
γ_h	Spontaneous recovery for humans	0.005	Assumed
p_1	Probability of disease transmission from mosquito to human	1	Silva and Torres [12]
p_2	Probability of disease transmission from human to mosquito	1	Silva and Torres [12]
σ_h	Weight constant for the use of treatment in humans	$1/4$	Silva and Torres [12]
θ	Weight constant for the use of indoor spray	2.5	Assumed
ϵ_h	Breakthrough from the exposed humans to infected humans	$1/17$	Assumed
ϵ_v	Breakthrough from the total population of mosquitoes	$1/18$	Assumed
β	Biting rate of mosquito	0.3	Agusto <i>et al.</i> , [2]
$S_h(0)$	Susceptible humans initial value	800	Silva and Torres [12]
$E_h(0)$	Exposed humans initial value	20	Assumed
$I_h(0)$	Infected humans initial value	0	Assumed
$S_v(0)$	Susceptible mosquitoes initial value	9500	Assumed
$E_v(0)$	Exposed mosquitoes initial value	20	Assumed
$I_v(0)$	Infected mosquitoes initial value	30	Assumed

3. Mathematical Analysis

In this paper, we analyze the local stability of the model using the diseases free equilibrium and the basic reproduction (10) as obtained in Alhassan, Momoh, Kadzai and Abdullahi [1].

3.1 Equilibrium state of the model

The diseases free equilibrium is given as:

$$M_0 = (S_h^*, E_h^*, I_h^*, S_v^*, E_v^*, I_v^*) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{(\theta u_3 + \mu_{vb})}, 0, 0 \right) \tag{9}$$

3.2 Basic reproduction number of the model

The basic reproduction number can be defined as the average number of secondary infectious individual in a completely susceptible population. The next generation matrix method of computing

R_0 was used to compute the reproduction number as described by Van den Driessche and Watmough [14] on the model (1) to (6). Thus, the basic reproduction number (10) obtained in Alhassan, Momoh, Kadzai and Abdullahi [1] is given below;

$$R_0 = \sqrt{\frac{(1-u_1)^2 \Lambda_h p_1 p_2 \beta^2 \epsilon_h \epsilon_v \mu_h}{\Lambda_h (\epsilon_h + \mu_h) (\gamma_h + \sigma_h u_2 + \mu_h + \delta_h) (\epsilon_v + \theta u_3 + \mu_{vb}) (\theta u_3 + \mu_{vb}) (\theta u_3 + \mu_{vb})}} \tag{10}$$

3.3 Local stability of the disease-free equilibrium

The basic reproduction number of the model (1) – (6) that was obtained in Alhassan, Momoh, Kadzai and Abdullahi [1] is used to analyze the local stability of the disease free equilibrium point of the model.

The disease free equilibrium state M_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

The Jacobian matrix of the system (1) – (6) is evaluated at the disease free equilibrium point M_0 as obtained below:

$$J(M_0) = \begin{bmatrix} -\mu & 0 & (\gamma_h + \sigma_h \mu_2) & 0 & 0 & (u_1 - 1)p_1 \beta \\ 0 & -(\varepsilon_h + \mu_h) & 0 & 0 & 0 & (1 - u_1)p_1 \beta \\ 0 & \varepsilon_h & -(\gamma_h + \sigma_h \mu_2 + \mu_h + \delta_h) & 0 & 0 & 0 \\ 0 & 0 & \frac{(u_1 - 1)\Lambda_h \mu_h p_2 \beta}{\Lambda_h (\theta u_3 + \mu_{vb})} & -(\theta u_3 + \mu_{vb}) & 0 & 0 \\ 0 & 0 & \frac{(1 - u_1)\Lambda_h \mu_h p_2 \beta}{\Lambda_h (\theta u_3 + \mu_{vb})} & 0 & -(\varepsilon_v + \theta u_3 + \mu_{vb}) & 0 \\ 0 & 0 & 0 & 0 & \varepsilon_v & -(\theta u_3 + \mu_{vb}) \end{bmatrix} \quad (11)$$

We reduce (11) in order to show all the eigenvalues of $J(M_0)$ are negatives. From (11), the first row and the fourth column contain the diagonal terms which formed two negative eigenvalues $-\mu_h$ and $-(\theta u_3 + \mu_{vb})$. Therefore, the other four (4) eigenvalues will be obtained after reducing the (11) to get the sub-matrix (12) below;

$$\begin{bmatrix} -(\varepsilon_h + \mu_h + \lambda) & 0 & 0 & (1 - u_1)p_1 \beta \\ \varepsilon_h & -(\gamma_h + \sigma_h \mu_2 + \mu_h + \delta_h + \lambda) & 0 & 0 \\ 0 & \frac{(1 - u_1)\Lambda_h \mu_h p_2 \beta}{\Lambda_h (\theta u_3 + \mu_{vb})} & -(\varepsilon_v + \theta u_3 + \mu_{vb} + \lambda) & 0 \\ 0 & 0 & \varepsilon_v & -(\theta u_3 + \mu_{vb} + \lambda) \end{bmatrix} = 0 \quad (12)$$

The other eigenvalues are obtained from the roots of the characteristics equation (13) below;

$$(\varepsilon_h + \mu_h + \lambda)(\gamma_h + \sigma_h \mu_2 + \mu_h + \delta_h + \lambda)(\varepsilon_v + \theta u_3 + \mu_{vb} + \lambda)(\theta u_3 + \mu_{vb} + \lambda) - \frac{(1 - u_1)^2 p_1 p_2 \beta^2 \varepsilon_h \varepsilon_v \mu_h \Lambda_v}{\Lambda_h (\theta u_3 + \mu_{vb})} = 0 \quad (13)$$

We let $C_1 = \varepsilon_h + \mu_h$, $C_2 = \gamma_h + \sigma_h \mu_2 + \mu_h + \delta_h$, $C_3 = \varepsilon_v + \theta u_3 + \mu_{vb}$ and $C_4 = \theta u_3 + \mu_{vb}$ and substitute them from (13) to get

$$(C_1 + \lambda)(C_2 + \lambda)(C_3 + \lambda)(C_4 + \lambda) - \frac{(1 - u_1)^2 p_1 p_2 \beta^2 \varepsilon_h \varepsilon_v \mu_h \Lambda_v}{\Lambda_h (\theta u_3 + \mu_{vb})} = 0 \quad (14)$$

Now after evaluating (14), we obtain

$$D_4 \lambda^4 + D_3 \lambda^3 + D_2 \lambda^2 + D_1 \lambda + D_0 = 0 \quad (15)$$

where

$$D_4 = 1, D_3 = C_1 + C_2 + C_3 + C_4, D_2 = (C_1 + C_2)(C_3 + C_4) + C_1 C_2 + C_3 C_4 \quad \text{and}$$

$$D_0 = C_1 C_2 C_3 C_4 - \frac{(1 - u_1)^2 p_1 p_2 \beta^2 \varepsilon_h \varepsilon_v \mu_h \Lambda_v}{\Lambda_h (\theta u_3 + \mu_{vb})}$$

Evaluating D_0 in terms of reproduction number R_0

$$R_0^2 = \frac{(1 - u_1) p_1 p_2 \beta^2 \varepsilon_h \varepsilon_v \mu_h \Lambda_v}{\Lambda_h (\varepsilon_h + \mu_h) (\gamma_h + \sigma_h \mu_2 + \mu_h + \delta_h) (\varepsilon_v + \theta u_3 + \mu_{vb}) (\theta u_3 + \mu_{vb})}$$

$$R_0^2 = \frac{(1 - u_1) p_1 p_2 \beta^2 \varepsilon_h \varepsilon_v \mu_h \Lambda_v}{\Lambda_h C_1 C_2 C_3 C_4}$$

Gives

$$D_0 = C_1 C_2 C_3 C_4 (1 - R_0) \quad (16)$$

Using Routh-Hurwitz criterion in Flores [5] for the polynomial (15), which states that all the determinants of the Routh-Hurwitz matrices H_i where $(i = 1, 2, 3, 4)$ are positive. In the polynomial (15) shows that $H_i > 0$, $(i = 1, 2, 3, 4)$.

$$H_4 = \begin{vmatrix} D_3 & D_1 & 0 & 0 \\ D_4 & D_2 & D_0 & 0 \\ 0 & D_3 & D_1 & 0 \\ 0 & D_4 & D_2 & D_0 \end{vmatrix} > 0,$$

$$H_3 = \begin{vmatrix} D_3 & D_1 & 0 \\ D_4 & D_2 & D_1 \\ 0 & D_3 & D_1 \end{vmatrix} > 0, H_2 = \begin{vmatrix} D_3 & D_1 \\ D_4 & D_2 \end{vmatrix} > 0 \quad \text{and}$$

$$H_1 = |D_1| > 0$$

Therefore, all the eigenvalues of the Jacobian have negative real parts, implying that the disease free equilibrium is locally asymptotically stable if $R_0 < 0$ and unstable if $R_0 > 0$.

3.4 Global stability of the disease-free equilibrium

We employ the method of Castillo-Chavez, et al [3] for analyzing global stability of DFE. The GAS is achieved if the two given conditions below are met. The model equations (1) – (6) are rewritten in the form:

$$\frac{dX}{dt} = F(X, Z) \tag{17}$$

$$\frac{dZ}{dt} = G(X, Z); G(X, 0) = 0 \tag{18}$$

where $X \in \mathbb{R}^2$ denotes the uninfected individuals and $Z \in \mathbb{R}^4$ denotes the infected individuals which includes the latent and the infectious. The disease free equilibrium of the model as shown in (9) is represented below as:

$$M_0 = (S_h^*, E_h^*, I_h^*, S_v^*, E_v^*, I_v^*) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{(\theta u_3 + \mu_{vb})}, 0, 0 \right)$$

The two conditions to be met for the global stability are;

(i) $\frac{dX}{dt} = F(X, 0)$, X^* is globally asymptotically stable (19)

(ii) $G(X, Z) = AZ - \hat{G}(X, Z)$, where $\hat{G}(X, Z) \geq 0$ for $(X, Z) \in \Pi$ (20)

where $A = D_Z G(X, 0)$ is an M -matrix (the off diagonal elements of A are non-negative) and Π is the biological feasible region. If the two conditions given above are satisfied by the model equations, then following theorem holds, Castillo-Chavez, *et al* [5].

Theorem

The equilibrium point

$$M_0 = F(X^*, 0) = \left(\frac{\Lambda_h}{\mu_h}, \frac{\Lambda_v}{(\theta u_3 + \mu_{vb})}, 0 \right)$$

of the model equations is globally asymptotically stable provided $R_0 < 1$ (locally asymptotically stable) and both the two conditions are satisfied.

Proof

To apply the above theorem on the model equations we proceed as follows;

For condition (i)

$$\frac{dX}{dt} = F(X, 0) = \left(\begin{matrix} \Lambda_h - \mu_h S_h \\ \Lambda_v - (\theta u_3 + \mu_{vb}) S_v \end{matrix} \right) \tag{21}$$

We solve the linear differential (21) and got

$$S_h(t) = \left(1 - e^{-\mu_h t} \right) \frac{\Lambda_h}{\mu_h} + S_h^*(0) e^{-\mu_h t} \tag{22}$$

$$S_v(t) = \left(1 - e^{-(\theta u_3 + \mu_{vb}) t} \right) \frac{\Lambda_v}{\theta u_3 + \mu_{vb}} + S_v^*(0) e^{-(\theta u_3 + \mu_{vb}) t} \tag{23}$$

From (9), $S_h^*(t) + S_v^*(t) \rightarrow N^*(t)$ as $t \rightarrow \infty$ Irrespective of the values of $S_h^*(0)$ and $S_v^*(0)$.

Therefore, $X^* = \left[\frac{\Lambda_h}{\mu_h}, \frac{\Lambda_v}{\theta u_3 + \mu_{vb}} \right]$ is globally asymptotically stable.

For condition (ii)

$$G(X, Z) = AZ - \hat{G}(X, Z)$$

$$A = \begin{pmatrix} -(\varepsilon_h + \mu_h) & 0 & 0 & \frac{(1-u_1)p_1\beta S_h}{N_h^*} \\ \varepsilon_h & -(\gamma_h + \sigma_h u_2 + \mu_h + \delta_h) & 0 & 0 \\ 0 & \frac{(u_1-1)p_2\beta S_v}{N_h^*} & -(\varepsilon_v + \theta u_3 + \mu_{vb}) & 0 \\ 0 & 0 & \varepsilon_v & (\theta u_3 + \mu_{vb}) \end{pmatrix}$$

and

$$\hat{G}(X, Z) = \begin{pmatrix} \frac{(1-u_1)p_1\beta I_v S_h}{N_h} - \varepsilon_h E_h - \mu_h I_h \\ \varepsilon_h E_h - (\gamma_h + \sigma_h u_2) I_h - (\mu_h + \delta_h) I_h \\ \frac{(1-u_1)p_2\beta I_h S_v}{N_h} - \varepsilon_v E_v - (\theta u_3 + \mu_{vb}) E_v \\ \varepsilon_v E_v - (\theta u_3 + \mu_{vb}) I_v \end{pmatrix}$$

Thus, $G(X, Z) = AZ - \hat{G}(X, Z) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$, i.e.

$G(X, Z) = [0 \ 0 \ 0 \ 0]^T$. This shows that $G(X, Z) = 0$. Hence, the model is globally asymptotically stable

4. Numerical simulation

In this section we performed the numerical simulation of the model equations using the inbuilt Range - Kutta of order four in MATLAB. We explore the model with preventive and treatment as control parameters to study the

effects of control on the transmission of malaria. We used one control parameter at a time, combination of two control parameters at a time and the combination of three control parameters at a time. For the figures presented, we used table 1 for parameters and variables values to illustrate the impact of the control strategies on the transmission of malaria in a population.

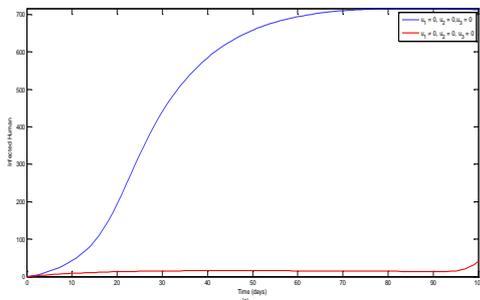


Figure 1(a). Graphical solution for infected human when treated bednets is used as the only control strategy

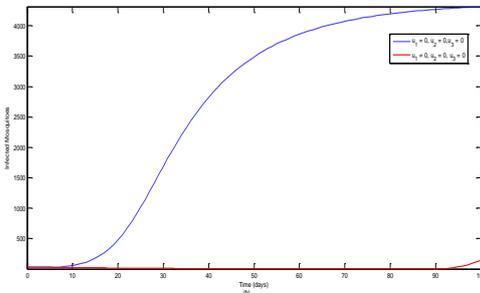


Figure 1(b). Graphical solution for infected mosquitoes when treated bednets is used as the only control strategy

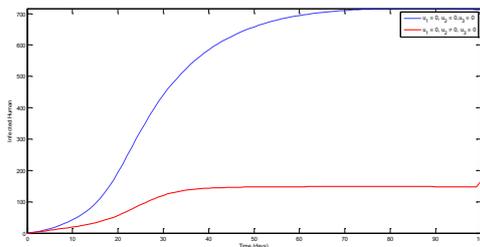


Figure 2(a). Graphical solution for infected human when treatment is used as the only control strategy

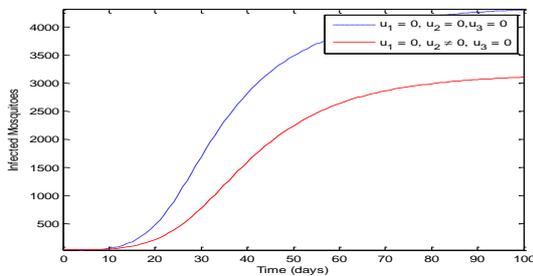


Figure 2(b). Graphical solution for infected mosquitoes when treatment is used as the only control strategy

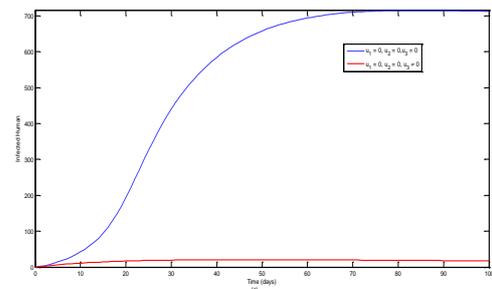


Figure 3(a). Graphical solution for infected humans when indoor spray is used as the only control strategy.

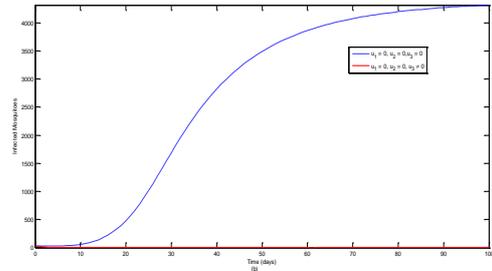


Figure 3(b). Graphical solution for infected mosquitoes when indoor spray is used as the only control strategy

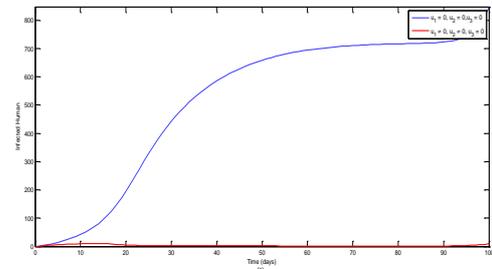


Figure 4(a). Graphical solution for infected humans when treated bednet and treatment are used as the control strategies

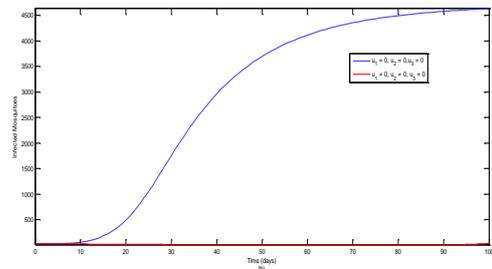


Figure 4(b). Graphical solution for infected mosquitoes when treated bednet and treatment are used as the control strategies.

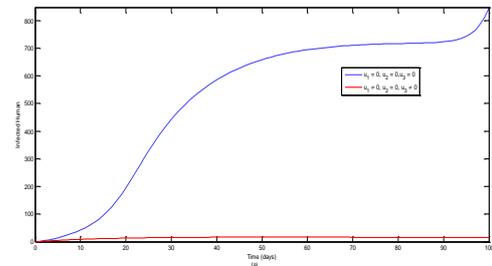


Figure 5(a). Graphical solution for infected humans when treated bednet and indoor spray are used as the control strategies

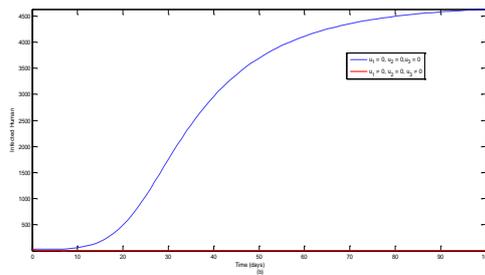


Figure 5(b). Graphical solution for infected mosquitoes when treated bednet and indoor spray are used as the control strategies

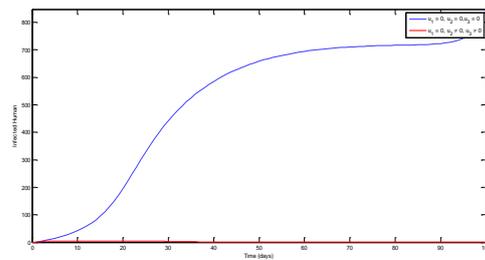


Figure 6(a). Graphical solution for infected humans when treatment and indoor spray are used as the control strategies.

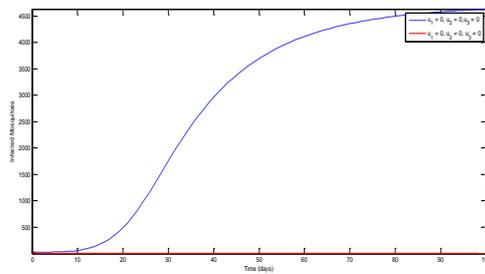


Figure 6(b). Graphical solution for infected mosquitoes when treatment and indoorspray are used as the control strategies

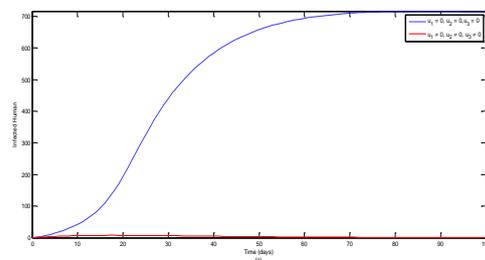


Figure 7(a). Graphical solution for infected humans when treated bednet, treatment and indoor spray are used as the control strategies

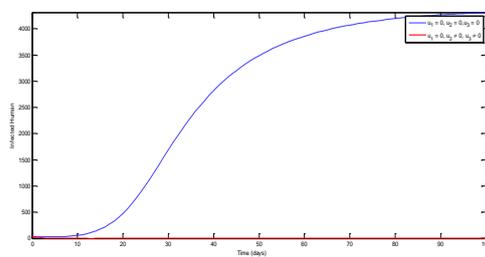


Figure 7(b). Graphical solution for infected mosquitoes when treated bednet, treatment and indoor spray are used as the control strategies

Figures 1(a), 2(a) and 3(a) show significant differences in the infected humans I_h and the

infected mosquitoes I_v when a single control measure is applied to control the transmission dynamics of malaria. We observed in figures 1(a), 2(a) and 3(a) that due to the use of a control measure, the number of infected humans I_h decreased significantly. Similarly, figures 1(b), 2(b) and 3(b) show a decrease in infected mosquitoes I_v .

The use of two (2) control measures at a time also reduced the number of infected human (I_h) as shown in figures 4(a), 5(a) and 6(a) as against the increase when there is no control is used. Similarly it was observed that the number of infected mosquitoes I_v has decrease significantly when a combination of two (2) control measures is applied in reducing malaria transmission, while the number of infected mosquitoes I_v increase when there was no control measure is applied.

Figures 7(a) and 7(b) illustrate how the number of infected humans I_h and the number of infected mosquitoes I_v decrease when all the three (3) controls measures are applied in controlling the transmission dynamics as compared to the case without control.

5. Conclusion

This paper provides stability analysis to the model proposed by Alhassan *et al.*, [1]. The model established that the disease-free equilibrium point is globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. This means that malaria will die out if one infected individual is introduced into a population. The global stability of the model signifies that the model is independent of the size of the initial population. It was also observed that the disease could be curtailed when the three control strategies are adequately implemented. We recommend that government at all levels should ensure that all the three strategies are implemented to help in eradicating malaria.

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Conflict of interest

The authors declare no conflict of interest.

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