Modified Mathematical Model for Malaria Control

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ABSTRACT

In this paper, mathematical model of malaria transmission is developed by modifying Ngwa and Shu's model. Modification was made to the susceptible, exposed, infectious, recovered and total human population, as well as the susceptible and infectious mosquitoes. The mathematical model was first realized using analytical method based on malaria transmission parameters. Then ODE45 solver was applied to demonstrate the effects of varying the transmission parameters in order to suggest control of malaria infection level to a tolerable limit. The simulation of the model was done using Matlab software. The numerical results obtained guarantee that malaria infection can be reduced to a tolerable limit.

Keywords: Malaria; drug effectiveness; mosquitoes, equilibrium state; dynamic equation

1. INTRODUCTION

Malaria is a vector borne infectious disease that has affected the human race since earliest times and an estimated 40% of the world's population lives in malaria endemic area [1]. Malaria is one of the most common infectious diseases and enormous public health problem. The yearly mortality from malaria is about 9 times that of HIV/Aids. Malaria ranked among the major infectious disease causing deaths, after pneumococcal acute respiratory infection designated pneumonia and tuberculosis [2]. Malaria is a parasitic disease caused mainly by plasmodium falciparum. (p. falciparum) and three other malaria parasites that cause milder diseases in human (p.vivax, p.ovale, and p. malariae) are transmitted by more than a dozen species of Anopheles mosquitoes widely [3]. The disease kills about 1 to 3 million people yearly, 75% of whom are African children [4]

The incidence of malaria has been on the increase recently owing to parasite-drug-resistance and mosquito-insecticide-resistance [5]. And this has lowered the productivity of the economy of the endemic zone [6]. Therefore, it is crucial to understand the important transmission parameters of the disease and develop efficient and effective solution strategies for its prevention and control.

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Mathematical modeling of malaria began in 1911 with Ross Model, and major extensions are described in Macdonald's 1957 book. Because of the simplified nature of Macdonald's model, malaria eradication/prediction of his model for total eradication of malaria from the world was not achieved as he failed or did not adequately advertise the long term consequences of the simplifications made to his model [4].

The first models were two-dimensional with one variable representing humans and the other representing the mosquitoes. In the same vein, malaria models on acquired immunity were proposed by Dietz, Molineaux and Thomas. Mosquito host choice and the epidemiology of malaria was studied by Kingsolver [7]. Recently, works on the spread of resistance to drugs [5] and the economics of drug resistance [8] were published.

In this paper, we modify or extend Ngwa and Shu's model for the spread of malaria with a Susceptible-Exposed-Infectious-Recovered-Susceptible (SEIRS) pattern for human and a Susceptible-Exposed-Infectious (SEI) pattern for mosquitoes. In [4], the authors analyzed a similar model for malaria transmission. In the model, the human population was divided into four classes: susceptible, exposed, infections and recovered human populations. The vector population is divided into three classes: susceptible, exposed and infectious and only female mosquitoes (because only female mosquitoes feed on human blood) were considered. The modified Ngwa and Shu's model in this paper includes human recruitment rate, it does not restrict the total number of bites on humans only to the number of mosquitoes as the total number of bites depend on both the human and mosquito population size in our model. Likewise, we included not only the birth of susceptible mosquitoes offsprings but also infectious offsprings of infected mosquitoes, the probability that a recovering individual become infected by one infected mosquito per contact unit time and effectiveness of drugs are included. But the model excludes direct human recovery from the infections to the susceptible class.

In this work, the transmission of malaria using mathematical and epidemiological model is studied. The modified model is presented in section 2. The analytical and numerical results are presented in section 3. Finally, the conclusion is presented in section 4.

Table 1:: The state variables for the malaria model

Symbol	Description
S_h	Population of susceptible humans
E_h	Population of exposed humans
I_h	Population of infectious humans
R_h	Population of recovered (immune and asymptomatic, but slightly infectious) humans
S_v	Population of susceptible mosquitoes
E_v	Population of exposed mosquitoes
I_v	Population of infectious mosquitoes
N_h	Total human population
N_v	Total mosquito population

2. MATHEMATICAL MODEL

In this section, a modified mathematical model of Ngwa and Shu's model for the evolution of the malaria in a population is formulated. Following the basic ideas and structure of mathematical modeling in epidemiology, the model for the malaria disease will be developed under the following terms in tables 1 and 2.

The dynamical equations of both human and mosquito population are given as

$$\frac{dS_h}{dt} = \Lambda_h + \beta_h R_h + \lambda_h N_h - \alpha_1 S_h - f_h(N_h) S_h \quad (1)$$

$$\frac{dE_h}{dt} = \alpha_1 S_h - (V_h + f_h(N_h)) E_h \quad (2)$$

$$\frac{dI_h}{dt} = V_h E_h - (\gamma_h + \alpha_2 A + f_h(N_h)) I_h + \alpha_3 I_v R_h \quad (3)$$

$$\frac{dR_h}{dt} = \alpha_2 A I_h - (\beta_h + \alpha_3 I_v + f_h(N_h)) R_h \quad (4)$$

$$\frac{dS_v}{dt} = N_v \lambda_v + (1-\theta)\lambda_v I_v - \alpha_4 S_v - f_v(N_v) S_v \quad (5)$$

$$\frac{dE_v}{dt} = \alpha_4 S_v - (V_v + f_v(N_v)) E_v \quad (6)$$

$$\frac{dI_v}{dt} = V_v E_v + \theta \lambda_v I_v - f_v(N_v) I_v \quad (7)$$

$$\frac{dN_h}{dt} = \Lambda_h + \lambda_h N_h - \gamma_h I_h - f_h(N_h) N_h \quad (8)$$

Table 2:: The parameters for the malaria model

Symbol	Description
Λ_h	The recruitment rat
T	Time
C_{hv}	Probability of transmission of infection from an infectious mosquito to a susceptible human, when there is contact between the two
C_{vh}	Probability of transmission of infection from an infectious human to a susceptible mosquito, when there is contact between the two
$\tilde{C}_{vh}$	Probability of transmission of infection from recovered (asymptomatic carrier) human to a susceptible mosquito, when the two are in contact
β_h	Rate at which a human looses his immunity
λ_h	Per capita human birth rate
λ_v	Per capita mosquito birth rate
α_1	The probability that a susceptible individual become infected by one infected mosquito per unit contact time
V_h	Per Capita rate of progression of humans from the exposed state to the infectious state
V_v	Per Capita rate of progression of mosquitoes from the exposed state to the infectious state
γ_h	Infected humans who die from the disease
α_2	The recovery rate of the infected human
A	The measure of the effectiveness of antimalaria drugs
α_3	The probability that a recovering individual become infected by one infected mosquito per contact unit time
α_4	The probability that a susceptible mosquito becomes infected
θ	Proportion of the offspring of the infected mosquito that are infected
$(1-\theta)$	Proportion of the offspring of the infected mosquito that are susceptible
$f_h(N_h)$	Per capita human death rate
$f_v(N_v)$	Per capita mosquito death rate
a_h	The human biting rate of the mosquito
a_v	Average number of mosquito bites per unit time
μ_{1h}	Density-independent part of the death (and emigration) rate for humans
μ_{2h}	Density-dependent part of the death (and emigration) rate for humans
μ_{1v}	Density-independent part of the death (and emigration) rate for mosquitoes
μ_{2v}	Density-dependent part of the death (and emigration) rate for mosquitoes

$$\frac{dN_v}{dt} = \lambda_v N_v - f_v(N_v)N_v \tag{9}$$

where, $f_h(N_h) = \mu_{1h} + \mu_{2h}N_h$ which is the per capita density-dependent death and emigration rate for humans and $f_v(N_v) = \mu_{1v} + \mu_{2v}N_v$ which is the per capita density-dependent death and emigration rate for mosquitoes [4]. The infection rates of human and mosquitoes per unit time as given in [1] are modified to:

$$\Big(\frac{C_{hv}a_vI_v}{N_h}\Big)S_h = \alpha_1 S_h \tag{10}$$

$$\Big(\frac{C_{vh}a_hI_h}{N_h}\Big)S_v + \Big(\frac{\tilde{C}_{vh}a_hR_h}{N_h}\Big)S_v = \alpha_4 S_v \tag{11}$$

In order to simplify the calculations, we make use of fractional qualities of the variables by rescaling the critical population of each class by total species population. Hence

$$u = \frac{S_h}{N_h}; g = \frac{E_h}{N_h}; w = \frac{I_h}{N_h}; R = \frac{R_h}{N_h}; x = \frac{S_v}{N_v}; \\ y = \frac{E_v}{N_v}; z = \frac{I_v}{N_v} \tag{12}$$

In [1], a rescaling of t was introduced with the quantity of $^1/_{\mu_v}$ by setting $\tau = \mu_v t$ and the following dimensionless variables were further introduced:

$$\tau = \mu_v t; \lambda = \frac{\lambda_h}{\mu_v}; \beta = \frac{\beta_h}{\mu_v}; \gamma = \frac{\gamma_h}{\mu_v}; \nu = \frac{v_h}{\mu_v}; a = \frac{\lambda_v}{\mu_v}; \\ b = \frac{C_{hv}a_v}{\mu_v}; C = \frac{C_{vh}a_h}{\mu_v}; d = \frac{\tilde{C}_{vh}a_h}{\mu_v}; \Lambda = \frac{\Lambda_h}{\mu_v}; e = \frac{V_v}{\mu_v}; \tag{13}$$

Using the definitions in (10) (13) and bifurcation analysis,(1) (7) can be represented as follows:

$$\frac{du}{d\tau} = \frac{\Lambda}{N_h} + \beta R + \lambda - \Big(\alpha_1 + \lambda + \frac{\Lambda}{N_h}\Big)u + \gamma w u \tag{14}$$

$$\frac{dg}{d\tau} = \alpha_1 u - \Big(v + \lambda + \frac{\Lambda}{N_h}\Big)g + \gamma w g \tag{15}$$

$$\frac{dw}{d\tau} = vg - \Big(\gamma + \alpha_2 A + \lambda + \frac{\Lambda}{N_h}\Big)w + \gamma w^2 + \alpha_3 z N_v R \tag{16}$$

$$\frac{dR}{d\tau} = \alpha_2 A w - \Big(\beta + \alpha_3 z N_v + \lambda + \frac{\Lambda}{N_h}\Big)R + \gamma w R \tag{17}$$

$$\frac{dx}{d\tau} = a\Big(1 + \big(1-\theta\big)\Big)z - \big(\alpha_4 + a\big)x \tag{18}$$

$$\frac{dy}{d\tau} = \alpha_4 x - \big(a + e\big)y \tag{19}$$

$$\frac{dz}{d\tau} = ey + \big(\theta a - a\big)z \tag{20}$$

The equation for the total population of human and mosquitoes is now represented as:

$$\frac{dN_h}{d\tau} = \Lambda + \lambda N_h - (\mu_{1h} + \mu_{2h}N_h)N_h - \gamma w N_h \tag{21}$$

$$\frac{dN_v}{d\tau} = aN_v - (\mu_{1v} + \mu_{2v}N_v)N_v \tag{22}$$

All parameters in the model are assumed positive for ease of analysis, then $N_h > 0$ with $0 \leq$ g, w, R ≤ 1; $N_v > 0$ with $0 \leq$ y, z ≤ 1. At t = 0 when g = w = R = 0 and y = z = 0, then $g', w', R' \geq 0$ and $y', z' > 0$. If $N_h = 0$ and $N_v = 0$, then $N_h > 0$ and $N_v = 0$. If $N_h > 0$, then $N_h^{'} > 0$. Lastly, if g + w + R = 1, then $g' + w' + R' < 0$ and if y + z = 1, then $y' + z' < 0$ at t > 0. Thus, there exist unique solutions satisfying the initial conditions for all t ≥ 0. Hence, the system is epidemically and mathematically well posed.

An interesting note is that since the equations are with respect to d, this means that the unit time measurement is with respect to the life span of a mosquito which is about 21 days [1].

2.1 Disease Free Equilibrium Point and Threshold Parameter

2.1.1 Disease Free Equilibrium Point

The disease-free equilibrium points are steady-state solutions where there is no disease i.e. $g = w = R = y = z = \gamma w = 0$ In the absence of disease, the positive equilibrium of human and mosquito population values for (21) and (22) are

$$N_h^* = \frac{(\lambda - \mu_{1h}) + \sqrt{(\lambda - \mu_{1h})^2 + 4\mu_{2h}\Lambda}}{2\mu_{2h}} \tag{23}$$

$$N_v^* = \frac{a - \mu_{1v}}{\mu_{2v}} \tag{24}$$

Hence, the malaria model has exactly one equilibrium point E_0: (0, 0, 0, N_h^*, 0, 0, N_v^*) and no other equilibrium points on the entire population. This is same with that realized in [4].

2.1.2 Threshold Parameter

To demonstrate the existence of a steady state in this model, we define the threshold parameter R_0. Threshold parameter is also called basic reproductive number in other papers [1, 9]. Here, R0 is defined by using the next generation operator approach as described in [4], as the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone else is susceptible. The next generation operator, K, gives the number of secondary infections

in human and mosquitoes caused by one generation of infectious humans and mosquitoes, as

$$K = \begin{bmatrix} 0 & K_{hv} \\ K_{vh} & 0 \end{bmatrix} \tag{25}$$

where K_{hv}: The number of humans that one mosquito infects through its infectious life, assuming all humans are susceptible.

K_{vh}: The number of mosquitoes that one human infects through the duration of the infectious period, assuming all mosquitoes are susceptible.

Using the terms defined in (13) and some other parameters, we modify the result in [4], to obtain

$$\left.\begin{array}{c} K_{hv} = \left(\frac{e}{e+f_v(N_v)}\right) \times b \times \left(\frac{1}{f_v(N_v)}\right) \\ K_{vh} = \left(\frac{v}{v+f_h(N_h)}\right) \times c \times \left(\frac{1}{\alpha_2 A+\gamma+f_h(N_h)}\right) \times \\ \left(\frac{v}{v+\mu_{1h}+f_h(N_h)} \times \frac{\alpha_2 A}{\alpha_2 A+\gamma+f_h(N_h)}\right) \times d \times \left(\frac{1}{\beta_h+f_h(N_h)}\right) \end{array}\right\} \tag{26}$$

Thus,

$$R_0 = \sqrt{K_{vh}K_{hv}} \tag{27}$$

This definition of R_0 is equivalent to the reproductive number in [4].

2.2 Stability of the System

Using the same procedure as in [3], the local stability of a system is determined from the signs of eigenvalues of the Jacobian Matrix of the right hand side of (15)-(17) and (19)-(20) [3].

2.2.1 Disease Free State

The Jacobian Matrix evaluated at $E_0(J_{E_0})$ for (15) (20) is given by

$$J_{E_0} = \begin{bmatrix} -(v+\lambda+\frac{\Lambda}{N_h^*}) & 0 & 0 & 0 & b \\ v & -(v+\alpha_2 A+\lambda+\frac{\Lambda}{N_h^*}) & 0 & 0 & 0 \\ 0 & \alpha_2 A & -(\beta+\alpha_3 z A N_v^*+\lambda+\frac{\Lambda}{N_h^*}) & 0 & 0 \\ 0 & c & d & -(a+e) & 0 \\ 0 & 0 & 0 & e & -(\theta a+a) \end{bmatrix}$$

Equations (14) and (18) are not used because our concern is on the populations of human and mosquito that have malaria parasite in them. Also, we assume that exposed individuals can travel and spread malaria.

The eigenvalues are obtained by solving the characteristic equation, $\det\left(J_{E_0} - sI_5\right)$ where I_5 is the identity matrix of dimension 5×5. If all eigenvalues for each equilibrium state have negative real part, then that equilibrium state is locally stable. The characteristics equation for the disease free state is represented by

$$\begin{aligned} &\left(-v-\lambda-\tfrac{\Lambda}{N_h^*}-s\right)\left(-\gamma-\alpha_2 A-\lambda-\tfrac{\Lambda}{N_h^*}-s\right) \\ &\left(-\beta-\alpha_3 z N_v^*-\lambda-\tfrac{\Lambda}{N_h^*}-s\right)\left(-a-e-s\right)\left(-\theta a \right. \\ &\left.-a-s\right)=0 \end{aligned} \tag{28}$$

From the characteristic equation (28), eigenvalues are $s_1 = -v-\lambda-\frac{\Lambda}{N_h^*}; s_2 = -\gamma-\alpha_2 A-\lambda-\frac{\Lambda}{N_h^*}; s_3 = -\beta-\lambda-\frac{\Lambda}{N_h^*}; s_4 = -a-e; and\ s_5 = -\theta a-a.$

The proof of stability in free disease state follows closely the method in [4] and is omitted here. However, the system is locally asymptotically stable when $R_0 < 1$ otherwise unstable.

2.2.2 Endemic Disease State

The endemic disease states, $E_1 : (g^*, w^*, r^*, N_h^*, y^*, z^*, N_v^*)$, are steady-state solutions where the diseases persists in the population (all state variables are positive). The steady-states are found by setting the left hand sides of (15)-(17) and (19)-(21) (because all transients disappear with sufficient passage of time) and then solving the resultant equations for the solutions, the steady states are

$$\begin{aligned} g^* = &\frac{\left(\left[T_1(\beta+T_2+\lambda)+\Lambda\right]\left[T_1(\gamma+\alpha_2 A+\lambda)+\Lambda\right]-\alpha_2 A T_1 T_2\right)w}{T_1 v(\beta+T_1+\lambda-\gamma w)} \\ &-\frac{\left[\left(T_1\gamma(\beta+T_2+\lambda)+T_1(\gamma+\alpha_2 A+\lambda)+\Lambda\right)+\gamma\Lambda\right]w^2+T_1\gamma^2 w^2}{T_1 v(\beta+T_1+\lambda-\gamma w)} \end{aligned} \tag{29}$$

$$R^* = \frac{\alpha_2 A T_1 w}{\left[T_1(\beta+T_2+\lambda-\gamma w)+\Lambda\right]} \tag{30}$$

$$y^* = \frac{(a-\theta a)\alpha_4}{\left[(a-\theta a)(a+e+\alpha_4)+\alpha_4 e\right]} \tag{31}$$

$$z^* = \frac{\alpha_4 e}{\left[(a-\theta a)(a+e+\alpha_4)+\alpha_4 e\right]} \tag{32}$$

$$N_h^* = \frac{(\lambda-\mu_{1h}-\gamma w)+\sqrt{(\lambda-\mu_{1h}-\gamma w)^2+4\mu_{2h}\Lambda}}{2\mu_{2h}} \tag{33}$$

$$N_v^* = \frac{a-\mu_{1v}}{\mu_{2v}} \tag{34}$$

Equations (29) - (34) are the steady-state variable solutions for the equations. Substituting the values of g^*, R^* and z^* from (28), (29) and (31) into (16) results in w^* which is a fifth order equation of the form

$$q_5 w^{*5} + q_4 w^{*4} + q_3 w^{*3} + q_2 w^{*2} + q_1 w^* + q_0 = 0 \tag{35}$$

$T_1 = N_h^* = \frac{(\lambda-\mu_{1h}-\gamma w)+\sqrt{(\lambda-\mu_{1h}-\gamma w)^2+4\mu_{2h}\Lambda}}{2\mu_{2h}}$;
$T_2 = \frac{\alpha_4 e(a-\mu_{1v})}{\mu_{2v}[(a-\theta a)(a+e+\alpha_4)+\alpha_4 e]}$;
$L_1 = [T_1(\beta+T_2+\lambda)+\Lambda)(T_1(\beta+T_2+\lambda)+\Lambda)$;
$L_2 = [T_1(\beta+T_2+\lambda)+\Lambda]+[T_1(v+\lambda+\alpha_1)+\Lambda]T_1\gamma$;
$L_3 = [T_1(\beta+T_2+\lambda)+\Lambda)(T_1(\gamma+\alpha_2 A+\lambda)+\Lambda-\alpha_2 A T_1 T_2]$;
$L_4 = [T_1\gamma(\beta+T_2+\lambda)+T_1(\gamma+\alpha_2 A+\lambda)+\Lambda)+\gamma\Lambda]$.

,where

$$q_5 = T_1^3\gamma^4 \tag{36}$$

$$q_4 = [L_2 T_1\gamma^2 + L_4(\gamma T_1)^2] \tag{37}$$

$$q_3 = [L_1 T_1\gamma^2 + L_2 L_4 + L_3(\gamma T_1)^2 + T_1^3\gamma^2 v] \tag{38}$$

$$q_2 = [L_1 L_4 + L_2 L_3 + T_1^2\alpha_1\gamma v(T_1(\beta+T_2+\lambda)+\Lambda)+T_1(\beta+T_1+\lambda)-\alpha_1 T_1^3 v\gamma(\alpha_2 A+\gamma)] \tag{39}$$

$$q_1 = [\alpha_1 T_1^3 v(\alpha_2 A+\gamma)(\beta+T_1+\lambda)+T_1 v\gamma(T_1(\beta+T_2+\lambda+\Lambda)-L_1 L_3 - T_1^2\alpha_1 v(T_1(\beta+T_2+\lambda)+\Lambda)(\beta+T_1+\lambda)] \tag{40}$$

$$q_0 = [T_1(\beta+T_2+\lambda)+\Lambda](\beta+T_1+\lambda)T_1 v \tag{41}$$

with

The eigenvalues are found by solving (35). The signs of the eigenvalues are negatives when they satisfy the Routh-Hurwitz criteria. Using the same procedure as in [3],

$$q_5 > 0 \tag{42}$$

$$q_5 q_2 - q_4 q_3 > 0 \tag{43}$$

$$q_5(q_3 q_2 - q_4 q_1) - (q_5^2 q_0 - q_4 q_3^2) > 0 \tag{44}$$

$$q_1[q_4 q_3 q_2 - (q_4^2 q_1 + q_5 q_2^2)] - q_0[q_3(q_4 q_3 - q_5 q_2) - (2q_5 q_4 q_1 - q_5^2 q_0)] > 0 \tag{45}$$

$$q_1[q_5 q_0(q_3 q_2 + q_4 q_1) + q_4 q_1(q_3 q_2 + q_5 q_0)] - [q_5(q_2^2 q_1 + q_5 q_0^2) + q_4(q_3^2 q_0 + q_4 q_1^2)] > 0 \tag{46}$$

The inequality (42) satisfies Routh-Hurwitz criteria because all terms in q_5 have positive value. Thus, the endemic equilibrium state is locally stable for $R_0 > 1$ when inequalities (43)−(46) satisfy Routh-Hurwitz.

3. NUMERICAL SIMULATION

The simulations that will be discussed in this section are based on the solution to the system of differential equations for the malaria transmission (14)−(22), when the system is solved in the mathematical software, MATLAB, using Ode45 differential equations solver.

Note that in all the simulations run, the Ode45 function was implemented using a time period of 50 days and initial conditions of $S_h = 900, E_h = 30, I_h = 50, R_h = 0, S_v = 1000, E_v = 100, I_v = 30, N_h = 980, N_v = 1130$.

In testing the malaria model, the following parameters taken from [1], [4] and [10] were used.

3.1 Simulations for dynamicity of human and mosquitos populations

When the parameter values in Table 3 are used and the population trends for $S_h, E_h, I_h, R_h, S_v, E_v, I_v$ observed over a period of 50 days are plotted in the fig 1. From Fig 1, it was seen that the expected population dynamics is observed in all classes. The susceptible populations (i.e. S_h & S_v) decrease with time. Hence, indicating movement into the exposed

Table 3:: The parameter values used for the simulation

Symbol	Values	Symbol	Values
β_h	0.01	μ_{2h}	0.0000001
γ_h	5.02×10^{-6}	μ_{1v}	0.1429
C_{vh}	0.8333	μ_{2v}	0.0002279
$\tilde{C}_{vh}$	0.08333	α_4	6.0036
C_{hv}	0.0200	α_1	0.5
a_h	18	α_2	0.1
a_v	0.600	α_3	0
μ_{1h}	0.00004212	Λ	0.03285
Θ	0.425	A	0.95
V_h	2.9888	a	0.071
V_V	0.8	$f_h(N_h)$	0.000047
λ_h	0.0184	$f_v(N_v)$	0.0071

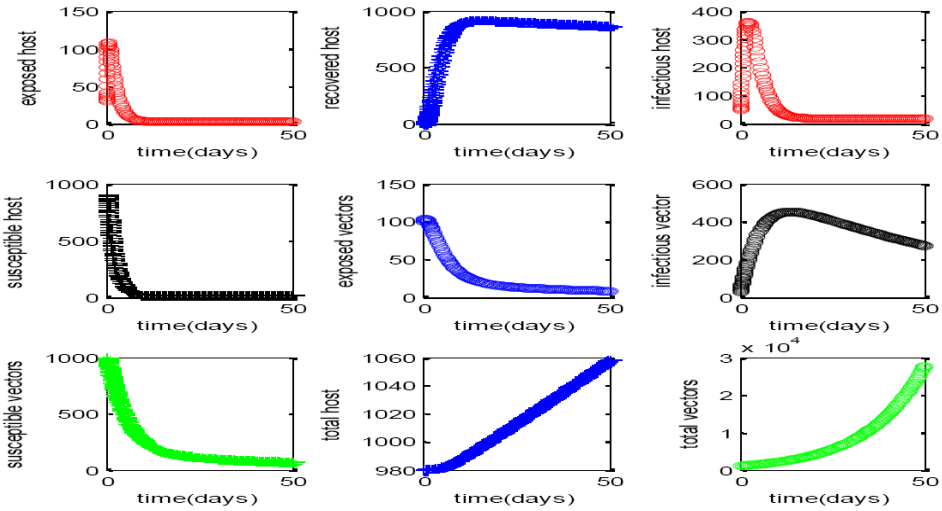

Fig.1:: The dynamic solutions of the populations trend

class. The exposed population for both humans and mosquitoes (i. e. E_h and E_v) peak and subsequently fall. The peak(s) indicates movement of respective individual from the susceptible class into the exposed class. While the fall indicates that individual concerned eventually leaves the exposed class. The infected class for both humans and mosquitoes peaks and falls. However, the fall is gradual in vector class than the host's. The peak shows movement of the respective individual into the infected class from the exposed class while the fall indicates that individual eventually leaves the infected class. The recovered class peak and falls, although in a more gradual but similar fashion to the infected class, therefore, showing movement into the recovered class and subsequent movement back into the susceptible class respectively. It was observed that the total human and mosquito population increase overtime, indicating that the infection is endemic.

3.2 3D Plots of Equilibrium Solutions

The next sets of simulations that were run analyzed the model given by (14)−(17). The trajectories of the solutions at initial conditions of $S_h = 900, E_h = 30, I_h = 50, R_h = 0, S_v = 1000, E_v = 100, I_v = 30, N_h = 980, N_v = 1130$, when the parameter values (in table 3) will lead to the endemic equilibrium state and at initial conditions $S_h = E_h = I_h = R_h = S_v = E_v = I_v = 0, N_h = 980, N_v = 1130$, when the parameter values will lead to a disease free equilibrium state are shown in Fig 2. From Fig 2, numerical solutions demonstrate the solution trajectories, projected into 3D-space $(S_h, I_h, R_h), (S_h, I_h, E_h), (S_h, E_h, R_h))$ respectively. The phase plots of the model are shown in Fig 3.

3.3 Treatment and Drug Effectiveness

The effects of treatment on patients and effectiveness of drugs (drugs not resisted by malaria parasite) were analyzed. Recall that α_2 is the rate of recovery of the infected human. It is expected that this probability will be higher for human when the effectiveness of drug (A) is very high. Current data show that the value of this probability is 0.5. To see if the human population as a whole is affected by changing α_2 value, the dynamics of the malaria model was investigated for range of α_2 values between 0.0 and 0.5 as shown in Fig 4.

From Fig 4, it is observed that the smaller the values of α_2, the fewer the recovered human host but the larger the infected human host. However, the reverse is the case when the value of α_2 is increased. This shows that larger α_2 values are important for possibly reducing the infection level.

3.4 The Transition from Susceptible State to Exposed State

The effect of different values of α_1 (the rate at which a susceptible human becomes infected by one infected mosquito) ranging from 0.0 - 0.5 is presented in Fig 5. It can be observed that the smaller the values of α_1, the fewer the exposed human and consequently the infected human. It shows that smaller α_1 values are important for reducing infection level among human possibly even for mosquitoes.

3.5 The Rate at which Uninfected Mosquito get Infected

The effect of α_4 (i.e. the probability of transmitting the parasite from an infected human to an uninfected mosquito), when varied from 0.0 to 0.5 as presented in Fig 6.

This result is significant because it shows that reducing α_4 is a good target for intervention in the

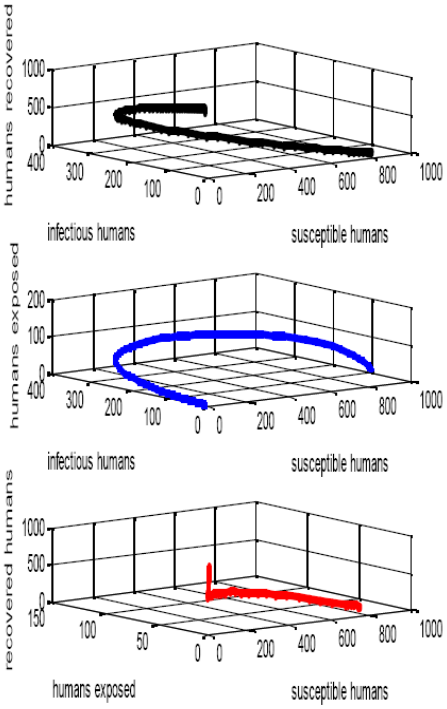

Fig 2a: Endemic Equilibrium

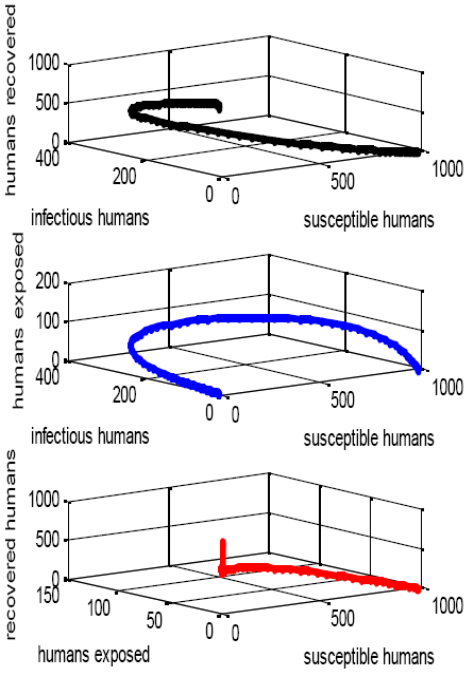

Fig 2b: Free Equilibrium State

Fig.2:: 3D plots of equilibrium solutions

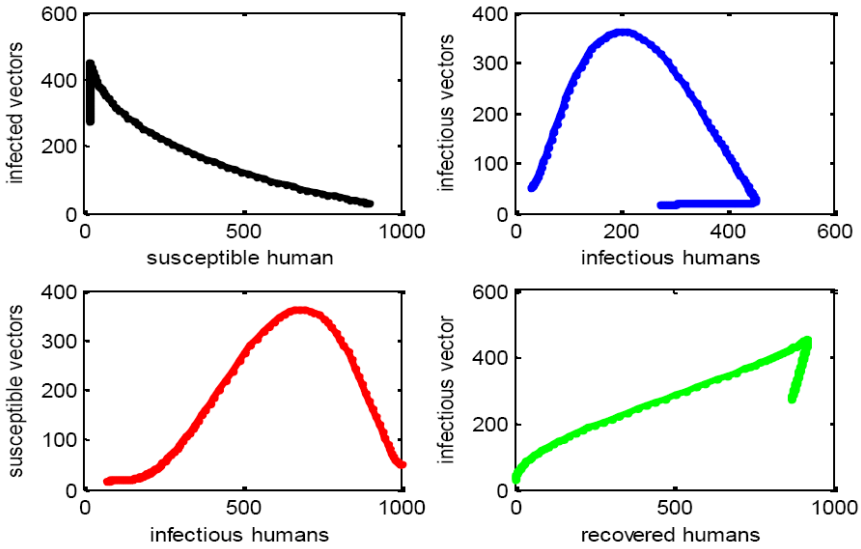

Fig.3:: Phase plots of the numerical solutions

spread of malaria. Meaning, if the probability of transmitting the disease from human to mosquitoes is reduced, malaria incidence can be reduced.

As can be observed from section C5, efforts are concentrated in the control of infection rate of both human and mosquitoes. This can be achieved by increasing human resistance against the parasite using anti-malaria drugs and also increasing the resistance of mosquitoes to infection from humans (note the mosquito resistance is for human benefit) by the use of bed nets and other physical means of prevention apart from anti-malaria drugs.

3.6 The Relationship between α_1 and α_4

Here, the effects of α_1 and α_4 on the infection rate of both humans and mosquitoes are determined. The model was run using various values of α_1 and α_4 between 0.0 and 0.5, respectively. The results are shown in Fig 7.

For humans, it is observed that the lowest infection level occurs at the smallest α_1 values of 0.0 and the largest α_4 value of 0.5. For the mosquito popu-

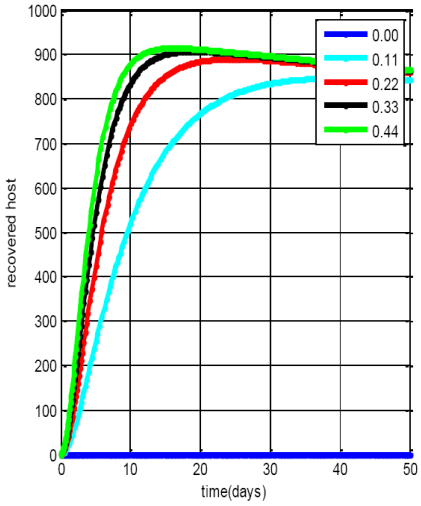

Fig 4a: Recovered hosts versus time

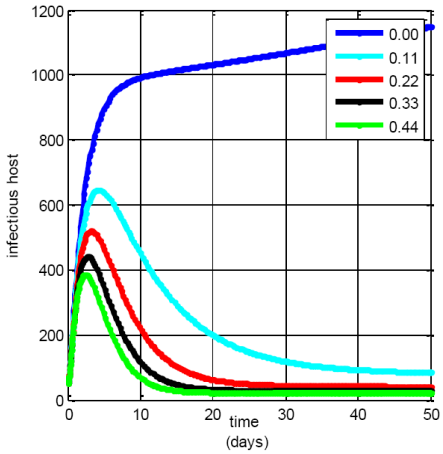

Fig 4b: Infectious host versus time

Fig.4:: Dynamics of malaria model for various values of α_2

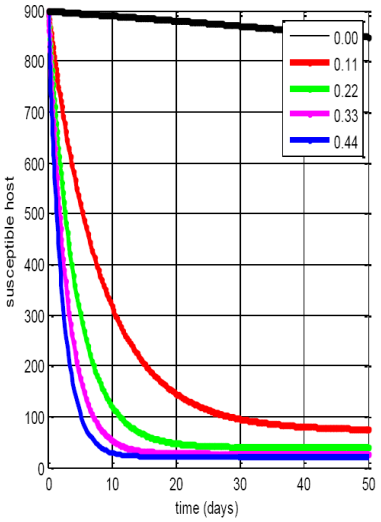

Fig 5a: susceptible hosts versus time

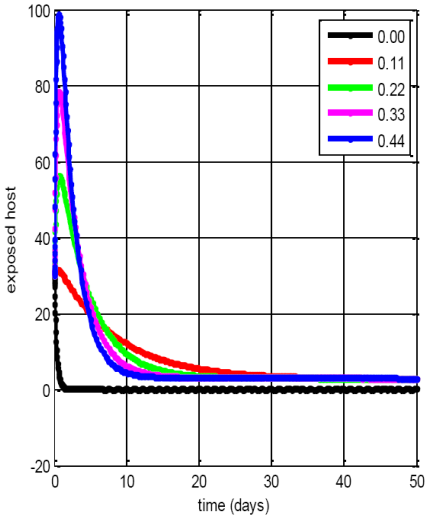

Fig 5b: exposed hosts versus time

Fig.5:: Effect of various values of α_1

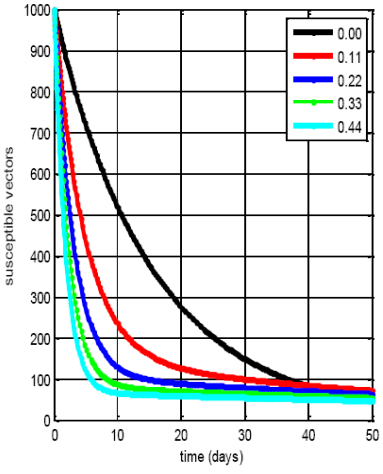

Fig 6a: susceptible vectors versus time

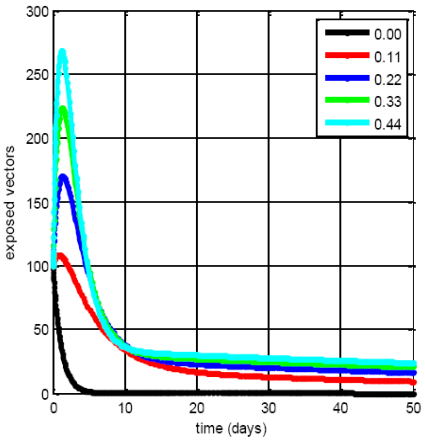

Fig 6b: exposed vectors versus time

Fig.6:: susceptible and exposed vectors against time.

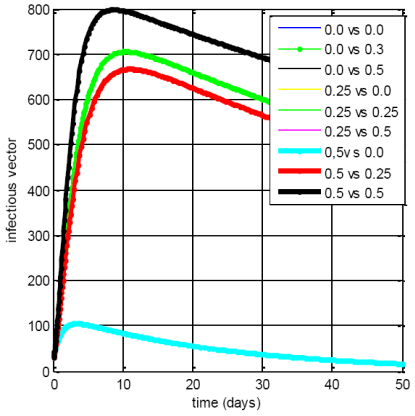

Fig 7a: Infectious vectors versus time

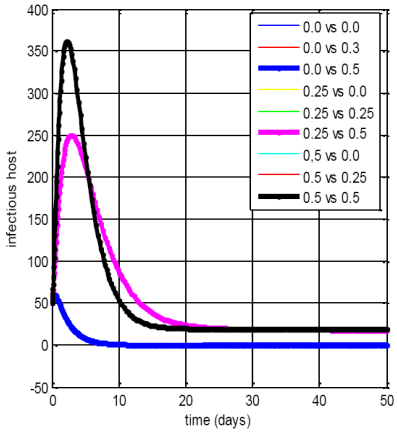

Fig 7b: Infectious hosts versus time

Fig.7:: α_1 versus α_4 effect on infectious vectors and hosts

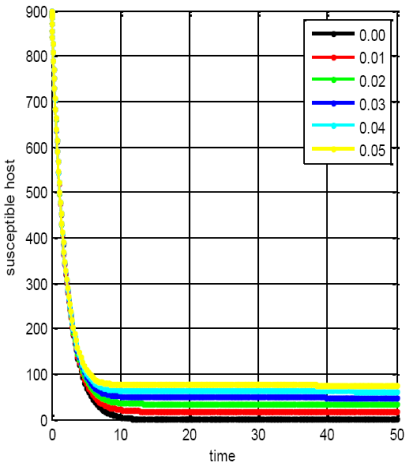

Fig 8a: Susceptible host versus time

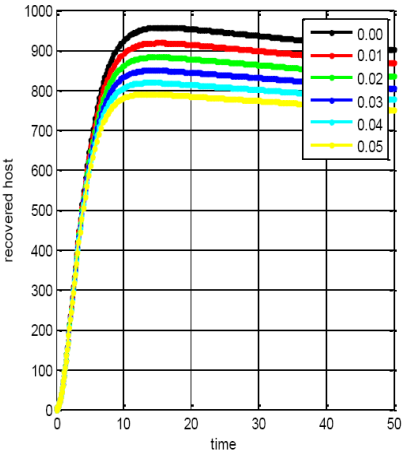

Fig 8b: Recovered host versus time

Fig.8:: Effect of different values of on susceptible and recovered host

lation, on the other hand, the opposite is observed. In mosquitoes, the lowest infection level (presented in Fig 7a) occurs at the highest α_1 value of 0.5 and the lowest α_4 of 0.0. Recall that α_1 measures the probability with which the infection is transmitted from mosquito to human and α_4 is the probability with which the infection is transmitted from human to mosquitoes. This means the human population will have a low level of infection if the ability of humans to be infected is kept at a minimum. Similarly, the mosquito population will have a low level of infection if the capability for humans to infect mosquito is also kept at low. Also it is observed that in all the two infected populations humans and mosquitoes, the highest infection level is observed when α_1 and α_4 are both high. Also, decreasing levels of infection are observed when both α_1 and α_4 are low simultaneously.

The effect of varying β (i.e. rate a human loses his immunity) on recovered human and susceptible human is illustrated in Fig 8. β is in the range of 0.0 0.05. It is observed that when β increases, the recovered host population falls while the susceptible host population rises. Hence efforts should be made to reduce the rate at which human loses immunity/-tolerance to malaria.

4. DISCUSSION

In this paper, we demonstrated numerically and analyzed the dynamics of the modified Ngwa and Shu malaria model in order to grasp the epidemiology of malaria and determine whether the infection is endemic. Fig 2 shows that the solutions move towards its equilibrium state. As observed, the trajectory is spiraling into the endemic equilibrium state in Fig 2a, while in Fig 2b the trajectory approaches the disease free equilibrium states when each one is stable. Also, the effect of treatment on patients and the effectiveness of drugs is captured as analyzed in Fig 4. The effect of α_1 helps in reduction of infections in humans and even mosquitoes as shown in Fig 5. The analysis of the rate at which susceptible mosquitoes get infected as analyzed is presented in Fig 6. This analysis helps suggest control of malaria using anti-malaria drugs and increasing mosquito resistance to

infection from humans by using mosquito treated nets and other means for resisting the vectors. If the positive effects of these parameters variations are taken into account, the number of infected human population would be favourably reduced. Hence, any community will be healthy for living.

5. CONCLUSION

In this paper, a modified Ngwa and Shu's malaria model was analyzed, the simulation of the ordinary differential equations model was carried out and the effects of varying malaria parameters on malaria transmission were studied and this can be applied to any given community. This was done in order to have an environment where malaria transmission is reduced to a tolerable level. It was observed that reducing the probability that a susceptible mosquito becomes infected is a good target for intervention in the spread of malaria. Also, the next generation operator R_0 is stable for disease free state when its value is less than one, while for an endemic equilibrium state the value is less than one to ensure stability.

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