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The Mathematical Analysis of Malaria Transmission: The effect of Sanitation

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Abstract: ***A deterministic mathematical model for malaria is considered under different level of sanitation strategy. The malaria disease free equilibrium point is locally asymptotically stable when the reproduction number is less than unity and unstable when reproduction number is greater than unity. The Comparison theorem is used to establish the global stability of the disease free equilibrium. A bifurcation analysis of the model was performed by applying the Centre manifold theory. Sensitivity indices of the basic reproductive number 'R_m' to the parameters in the model was calculated. The study reveals that increase in sanitation level results in decrease in number of mosquito bites and transmission rate of malaria. To illustrate the analytical results, numerical simulations using a set parameter values were provided.***

Keywords: Sanitation, Malaria, Mass action Incidence, Bifurcation, Reproduction number

1. Introduction

Malaria is one of the most deadly diseases of our time. Malaria was first discovered centuries ago by the Chinese in 2700 BC [1]. However it was in the 1800's when Ross made his ground breaking discoveries that led to our understanding of the mechanism behind malaria infections. The parasitic disease malaria is transmitted to the Human through a biting from an infected female Anopheles mosquito [2]. Malaria is caused by the protozoan parasites called plasmodium. There are four species of the plasmodium parasites, namely plasmodium falciparum, plasmodium ovale, plasmodium vivax and plasmodium malariae, of the four species, plasmodium falciparum is the most virulent, lethal and responsible for the majority of morbidity and mortality due to malaria [3].

The most common first symptoms of malaria are headache, aching muscles, stomach ache and weak or lack of energy. After a day or so the body temperature may rise (up to 40℃) and the patient may have fever, shivers, severe headache, diarrhoea, loss of appetite, nausea, vomiting, back pain and increased sweating [4]. The individuals most vulnerable to malaria are children under the age of 5 years. This is attributed to their weaker immunity. Aside from children, pregnant women are also heavily affected, with resultant effects on maternal health and birth outcomes [5]

Li-Ming Cai *et al* [6], replaced the standard incidence with the mass action incidence in their study of malaria model with partial immunity to reinfection and indeed shows that their model exhibits a backward bifurcation. Buonomo et al [7] further gives a deep insight to backward bifurcation in their study. The main reason why it is important to investigate the occurrence of bifurcations is that they play a vital and relevant role in disease control and eradication. Recent studies by [8] shows that a necessary condition for disease eradication is that the *basic reproductive number* R_0 must be less than unity. However, when a backward bifurcation occurs, an endemic equilibrium may also co-exist for $R_0 < 1$. This means that the occurrence of a backward bifurcation have important public health implications. It is not sufficient to reduce R_0 below 1 to eliminate the disease but R_0 must be further reduced below a certain critical value R_c to guarantee total elimination and avoid endemic situations. The bifurcation analysis is based on the use of centre manifold theory [9, 10, 11].

In [12], the analysis did not consider sanitation as a way of reducing the transmission of malaria infection. We think such a feature is worth to be investigated. For this reason, in this paper we aim to incorporate the sanitation function and also derive conditions for which the system exhibit forward or backward bifurcation through the bifurcation method introduced in [9].

The paper is organized as follows: in Section 2 we give the model description and model formulation. In Section 3, we obtain the equilibrium points and provide the local and global stabilities of the disease free. In Section 4, the bifurcation analysis is performed. In Section 5 the bifurcation analysis was studied. In Section 6 the effect of sanitation on malaria transmission was discussed and the Sensitivity analysis is performed in section 7. Section 8, present numerical verification which were not consider in [1]. Discussion of result and concluding remarks are presented in Section 8.

2. Mathematical Formulation and Model Description

Oluyo and Adeniyi [12], studied the following model in (1) on malaria-pneumonia co-infection with mass action incidence following the modification of the work of Lawi et al [13].

The model in [12] subdivides the total human population of interest into sub population depending on the malaria or pneumonia status of individuals. The classes consist of Susceptibles (S) representing the number of individuals who are at risk of acquiring malaria or pneumonia or both diseases, Infectives (I) representing infectious individuals

with malaria or pneumonia or both infections capable of transmitting infection to susceptible mosquitoes.

$$\left.\begin{aligned} S_h'(t) &= \Lambda_h - \alpha\beta_m I_v S_h - \beta_p c(I_p + kI_{mp})S_h - \mu_h S_h + \pi I_m + \tau I_p + \phi I_{mp} \\ I_m'(t) &= \alpha\beta_m I_v S_h - \vartheta\beta_p c(I_p + kI_{mp})I_m - (\sigma_m + \pi + \mu_h)I_m \\ I_p'(t) &= \beta_p c(I_p + kI_{mp})S_h - \varepsilon\alpha\beta_m I_v I_p - (\sigma_p + \tau + \mu_h)I_p \\ I_{mp}'(t) &= \varepsilon\alpha\beta_m I_v I_p + \vartheta\beta_p c(I_p + kI_{mp})I_m - (\sigma_m + \sigma_p + \sigma_{mp} + \phi + \mu_h)I_{mp} \\ S_v'(t) &= \Lambda_v - \alpha\beta_v(I_m + \delta I_{mp})S_v - \mu_v S_v \\ I_v'(t) &= \alpha\beta_v(I_m + \delta I_{mp})S_v - \mu_v I_v \end{aligned}\right\} \quad (1)$$

In the present work, great attention would be focused on malaria transmission dynamics. Thus, if

If $I_p(t) = I_{mp}(t) = 0$ in (1), then

$$\left.\begin{aligned} S_h'(t) &= \Lambda_h - \alpha\beta_m I_v S_h - \mu_h S_h + \pi I_m \\ I_m'(t) &= \alpha\beta_m I_v S_h - (\sigma_m + \pi + \mu_h)I_m \\ S_v'(t) &= \Lambda_v - \alpha\beta_v I_m S_v - \mu_v S_v \\ I_v'(t) &= \alpha\beta_v I_m S_v - \mu_v I_v \end{aligned}\right\} (2)$$

One intervention strategy recommended controlling malaria transmission, transmission in [12] was One intervention strategy recommended controlling for malaria is either through treatment using anti-malaria drugs or using preventive measures like sleeping under the Bed Treated Nets, Insecticides spray. Sanitation function was introduced in [14] and how these functions affect the endemicity of infection. Based on the above submission, the model in (1) is further extended to include the exposed classes for malaria and the sanitation function for malaria. Thus, the improved model for malaria only transmission now reads:

$$\left.\begin{aligned} S_h(t) &= \Lambda_h - \alpha\beta(H)I_v S_h - \mu_h S_h + \pi I_m + \pi E_m \\ E_m'(t) &= \alpha\beta(H)I_v S_h - (k_m + \pi + \mu_h)E_m \\ I_m'(t) &= k_m E_m - (\sigma_m + \pi + \mu_h)I_m \\ S_v'(t) &= \Lambda_v - \alpha\beta_v I_m S_v - \mu_v(H)S_v \\ E_v'(t) &= \alpha\beta_v I_m S_v - (k_v + \mu_v(H))E_v \\ I_v'(t) &= k_v E_v - \mu_v(H)I_v \end{aligned}\right\} (3)$$

Where:

S_h: Susceptible class of human population
I_m: Human population infectious with malaria
S_v: Susceptible class of vector population
I_v: Infectious class of the vector population
Λ_h: Constant per capita recruitment rate into susceptible human population
Λ_v :Constant per capita recruitment rate into susceptible vector population
μ_h : Natural death rate of human population
μ_v : Natural death rate of vector population
σ_m :Malaria induced mortality rate
π: Rate of recovery from malaria to the susceptible Class
α : Number of bites per human per mosquito
β_m : Transmission rate of malaria in human
β_v : Rate at which a mosquito become infected with malaria from any infected human

2.1 Model Assumptions

The assumptions for our model follow from [13] with this addition: this model assumes a homogeneous mixing of individuals in the population where all individuals have equal likelihood of catching the infection if they come into effective contact with infectious mosquitoes and that transmission of the infection occurs with a mass action incidence rate.

2.2 Model Analysis

2.2.1 The Dynamics of the Human and Vector Population

The total human population at any time t is denoted by $N_h(t)$. The total human population is subdivided into sub-population namely; Susceptible $S_h(t)$ who are not yet infected but can be infected by malaria, individuals Exposed $E_m(t)$to malaria and Infectious $I_m(t)$ with malaria.

Thus,

$$N_h(t) = S_h(t) + E_m(t) + I_m(t) \quad 3a)$$

By differentiating equation $(3a)$ with respect to time t to get

$$N_h'(t) = \Lambda_h - \mu_h N_h(t) - \sigma_m I_m \quad (3b)$$

Now, if we assume that the malaria induce death is small or negligible i.e $\sigma_m \cong 0$ in equation $(3b)$ gives

$$N_h'(t) = \Lambda_h - \mu_h N_h(t) \quad (3c)$$

The solution of (3c) is

$$N_h(t) = \frac{\Lambda_h}{\mu_h} + c_1 e^{-\mu_h t}$$

c_1 is a constant of integration. In the course of time i.e as $t \to \infty$, the carrying capacity of the human population will reach a constant value

$$N_h(t) = \frac{\Lambda_h}{\mu_h} = 1 \quad \text{(say)} \quad (3d)$$

Similar reasoning gives

$$N_v(t) = \frac{\Lambda_v}{\mu_v} + c_2 e^{-\mu_v t}$$

c_2 is a constant of integration. In the course of time i.e as $t \to \infty$, the carrying capacity of the vector population will reach a constant value

$$N_v(t) = \frac{\Lambda_v}{\mu_v} = 1 \quad \text{(say)} \quad (3e)$$

2.2.2 Positivity and Boundedness of Solutions

For the system of equations (3) to be epidemiologically meaningful, it is important to prove that all soltion with non-negative initial conditions will remain non-negative. We prove by the following theorem:

Theorem 1: If $S_h(0)$, $E_m(0)$,$I_m(0)$, $S_v(0)$, $E_v(0)$ and $I_v(0)$ are non-negative, then so are $S_h(t)$, $E_m(t)$,$I_m(t)$, $S_v(t)$, $E_v(t)$ and $I_v(t)$ for all $t > 0$. Moreover,

$\lim_{t\to\infty} N_h(t) \le \frac{\Lambda_h}{\mu_h}$, $\lim_{t\to\infty} N_v(t) \le \frac{\Lambda_v}{\mu_v}$ (4a)

Furthermore, if

$N_h(0) \le \frac{\Lambda_h}{\mu_h}$ then $N_h(t) \le \frac{\Lambda_h}{\mu_h}$ and $N_v(0) \le \frac{\Lambda_v}{\mu_v}$,

Then

$N_v(t) \le \frac{\Lambda_v}{\mu_v}$, then the region $\Gamma = \Gamma_{hv} \subset \mathcal{R}_+^{\,6}$ with

$$\Gamma_{hv} = \left\{(S_h, E_m, I_m, S_v, E_v, I_v) \in \mathcal{R}_+^{\,6} : S_h + E_m + I_m \le \frac{\Lambda_h}{\mu_h}, S_v + E_v + I_v \le \frac{\Lambda_v}{\mu_v}\right\}$$

is positively invariant.

It follows from theorem 1 that it is sufficient to consider the dynamics of system (3) in Γ. In this region the model system (3) can be considered to be epidemiologically well-posed [15].

3. The Equilibria

The equilibriums of system (3) are determined by solving the resulting equation obtained by equating the derivatives of system (3) to zero and setting

$E_m = E_m{}^*, I_m = I_m{}^*, I_v = I_v{}^*, , S_v = S_v{}^*, \ S_h = S_h{}^*, E_v = E_v{}^*$ (4b)

Thus we have

$$AI_m{}^{*3} + BI_m{}^{*2} + CI_m{}^* = 0 \quad (5a)$$

Where

$A = k_v\alpha^3\beta(H)\beta_v{}^2\Lambda_v\pi(\pi + \sigma_m + \mu_h + k_m) - (\pi + k_m + \mu_h)(\pi + \sigma_m + \mu_h)\alpha^3\beta(H)\beta_v{}^2k_v\Lambda_v - (\pi + k_m + \mu_h)(\pi + \sigma_m + \mu_h)\mu_h\mu_v(k_v + \mu_v)\alpha^2\beta_v{}^2$

$B = k_mk_v\alpha^3\beta(H)\beta_v{}^2\Lambda_v\Lambda_h + k_v\alpha^2\beta(H)\beta_v\Lambda_v\mu_v\pi(\pi + \sigma_m + \mu_h + k_m) - \mu_v(\pi + k_m + \mu_h)(\pi + \sigma_m + \mu_h)\alpha^2\beta(H)\beta_vk_v - 2(\pi + k_m + \mu_h)(\pi + \sigma_m + \mu_h)\mu_h\mu_v{}^2\alpha\beta_v(k_v + \mu_v)$

$C = k_mk_v\alpha^2\beta(H)\beta_v\Lambda_v\Lambda_h\mu_v - \mu_v{}^3(\pi + k_m + \mu_h)(\pi + \sigma m + \mu h\mu hkv + \mu v$

Equation (5a) admits

$I_m{}^* = 0$ or $AI_m{}^{*2} + BI_m{}^* + C = 0$

$I_m{}^* = 0$ admits the disease free equilibrium denoted by M_0 i.e.

$M_0 = (S_{h0}{}^*, E_{m0}{}^*, I_{m0}{}^*, S_{v0}{}^*, E_{v0}{}^*, I_{v0}{}^*) = (1,0,0,1,0,0)$

The malaria endemic equilibrium is obtained by considering the real positive solution of the equation

$$AI_m{}^{*2} + BI_m{}^* + C = 0 \quad (5b)$$

There is an observation that the constant C in equation (5b) above is always negative then equation (5b) will have two positive real roots $I_{m1}{}^*$ and $I_{m2}{}^*$ if $A < 0$ and $B > 0$ by Descartes' rule of signs. Therefore, the endemic equilibrium points namely:

$M_1{}^* = (S_{h1}{}^*, E_{m1}{}^*, I_{m1}{}^*, S_{v1}{}^*, E_{v1}{}^*, I_{v1}{}^*)$ and $M_2{}^* = (S_{h2}{}^*, E_{m2}{}^*, I_{m2}{}^*, S_{v2}{}^*, E_{v2}{}^*, I_{v2}{}^*)$ emerges.

3.1 The Basic Reproduction Number R_0

Diekmann et al. [16] defined the basic reproduction number denoted by $\boldsymbol{R_0}$ as the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness.

The basic reproduction number $\boldsymbol{R_0}$ is computed using the next generation matrix approach as described by Heffernan et al [17]. We introduce the matrices

$$F = \begin{pmatrix} 0 & 0 & 0 & \alpha\beta(H) \\ 0 & 0 & 0 & 0 \\ 0 & \alpha\beta_v & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix};$$

$$V = \begin{pmatrix} (k_m + \pi + \mu_h) & 0 & 0 & 0 \\ 0 & (\sigma_m + \pi + \mu_h) & 0 & 0 \\ 0 & 0 & (k_v + \mu_v) & 0 \\ 0 & 0 & -k_v & -\mu_v \end{pmatrix}$$

Where F and V are matrix of newly created infections and matrix of transferred of infections respectively. Then the next generation matrix denoted by G is defined thus

$$G = FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\alpha\beta(H)k_v}{\mu_v(k_v+\mu_v)} & \frac{\alpha\beta(H)}{\mu_v} \\ 0 & 0 & 0 & 0 \\ \frac{\alpha\beta_vk_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)} & \frac{\alpha\beta_v}{(\sigma_m+\pi+\mu_h)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

hus, the basic reproduction number for malaria denoted by R_m is

$$R_m = \sqrt{\frac{\alpha^2\beta(H)\beta_vk_mk_v}{\mu_v(k_v+\mu_v)(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}}$$

3.2 Local Stability of Malaria Free Equilibrium Only

Theorem 2: The malaria free equilibrium M_0 of system (3) is locally asymptotically stable if $R_m < 1$ and unstable if $R_m > 1$.

Proof:

The Jacobian matrix of system (3) evaluated at M_0 is

$$J(M_0) = \begin{pmatrix} -\mu_h & \pi & \pi & 0 & 0 & -\alpha\beta(H) \\ 0 & -(k_m+\pi+\mu_h) & 0 & 0 & 0 & \alpha\beta(H) \\ 0 & k_m & -(\sigma_m+\pi+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & -\alpha\beta_v & -\mu_v & 0 & 0 \\ 0 & 0 & \alpha\beta_v & 0 & -(k_v+\mu_v) & 0 \\ 0 & 0 & 0 & 0 & k_v & -\mu_v \end{pmatrix} \quad (6)$$

The characteristic equation of equation (6) is

$$(-\mu_h - \lambda)(-\mu_v - \lambda)g(\lambda) = 0$$

Where

$g(\lambda) = \lambda^4 + g_0\lambda^3 + g_1\lambda^2 + g_2\lambda + g_3$

$g_0 = (k_m + 2\pi + 2\mu_h + \sigma_m + 2\mu_v + k_v)$,

$g_1 = \big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu_h + \sigma mkv + \mu v + g0\mu v$,

$g_2 = \big(\mu_v\big((k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h) + (k_m + 2\pi + 2\mu h + \sigma mkv + \mu v + km + \pi + \mu h\sigma m + \pi + \mu hkv + \mu v$,

$g_3 = \mu_v(k_m + \pi + \mu_h)(\sigma_m + \pi + \mu_h)(k_v + \mu_v) - \alpha^2\beta(H)\beta_vk_mk_v$

Clearly, $\lambda_1 = -\mu_h$, $\lambda_2 = -\mu_v$, while λ_3 , λ_4 , λ_5 and λ_6 are obtained from $g(\lambda) = 0$ (7)

Equation (7) will have four negative real roots if (By Descartes rule of signs)

$$\frac{\alpha^2\beta(H)\beta_vk_mk_v}{\mu_v(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)(k_v+\mu_v)} < 1$$

$\therefore R_m < 1$

Hence, M_0 is locally asymptotically stable if $R_m < 1$. The result follows immediately that M_0 is unstable if $R_m > 1$.

3.3 Global Stability of Malaria Free Equilibrium

Theorem 3: The malaria free equilibrium M_0 of system (3) is globally asymptotically stable if $R_m < 1$ and unstable if $R_m > 1$.

Proof:
The Comparison Theorem as implemented in [18] and [19] is employed here. The rate of change of the infected components of system (3) can be written as

$$\begin{pmatrix} \frac{dE_m}{dt} \\ \frac{dI_m}{dt} \\ \frac{dE_v}{dt} \\ \frac{dI_v}{dt} \end{pmatrix} = (F-V)\begin{pmatrix} E_m \\ \mathrm{I}_m \\ E_v \\ I_v \end{pmatrix} - F_i \begin{pmatrix} E_m \\ \mathrm{I}_m \\ E_v \\ I_v \end{pmatrix}$$

Where F, V and F_i are as defined in section (3.1)
Since at the disease free $I_m = I_v = 0 \rightarrow (0,0)$ as $t \rightarrow \infty$. Thus,

$$\begin{pmatrix} \frac{dE_m}{dt} \\ \frac{dI_m}{dt} \\ \frac{dE_v}{dt} \\ \frac{dI_v}{dt} \end{pmatrix} \leq \begin{pmatrix} -(k_m+\pi+\mu_h) & 0 & 0 & \alpha\beta(H) \\ -k_m & -(\sigma_m+\pi+\mu_h) & 0 & 0 \\ 0 & \alpha\beta_v & -(k_v+\mu_v) & 0 \\ 0 & 0 & k_v & -\mu_v \end{pmatrix} \begin{pmatrix} E_m \\ \mathrm{I}_m \\ E_v \\ I_v \end{pmatrix}$$

$$= (F-V)\begin{pmatrix} E_m \\ \mathrm{I}_m \\ E_v \\ I_v \end{pmatrix} \quad (8)$$

According to [8] and [9], all eigenvalues of the matrix $(F-V)$ have negative real parts i.e

$$\lambda^4 + \big((k_m+\pi+\mu_h) + (\sigma_m+\pi+\mu_h) + \mu_v + kv+\mu v\lambda 3+km+\pi+\mu h\sigma m+\pi+\mu h+\mu vkv+\mu v+km+\pi+\mu h +\mu v\sigma m+\pi+\mu h+\mu vkm+\pi+\mu h\lambda 2+kv+\mu vkm+\pi+\mu h+\mu v \sigma m+\pi+\mu h+\mu vkm+\pi+\mu h\sigma m+\pi+\mu h\lambda+\mu vkv+\mu vkm+\pi +\mu h\sigma m+\pi+\mu h1-Rm2=0 \quad (9)$$

Equation (9) will have four negative roots if $R_m < 1$ (By Descartes rule). It follows that the linearized differential inequality (8) is stable whenever $R_m < 1$. Consequently, $(E_m, I_m, E_v, I_v) \rightarrow (0,0,0,0)$ as $t \rightarrow \infty$. Evaluating system (3) at $E_m = I_m = E_v = I_v = 0$ gives $S_h \rightarrow 1$, $S_v \rightarrow 1$ for $R_m < 1$.

Hence, the diseases free point M_0 is globally asymptotically stable if $R_m < 1$. The result also follows that diseases free point M_0 is unstable if $R_m > 1$

4. Local Asymptotic Stability of Malaria Endemic Equilibrium

The Centre Manifold Theorem as used in [7] and [20] was used to analyse the local stability of malaria endemic equilibrium as stated below:

The Centre Manifold Theorem [7]
Consider a general system of ODEs with a parameter $\boldsymbol{\phi}$

$$\dot{x} = f(x,\phi);\ f: R^n \times R \rightarrow R^n;\ f \in C^2(R^n \times R) \quad (10)$$

Without loss of generality, assume that $x = 0$ is equilibrium for

Theorem 4:: Assume:
(i) $A = D_x f(0,0)$ is the linearization matrix of system (14) around the equilibrium $x = 0$ with ϕ evaluated at O. Zero is a simple eigenvalue of (10) and all other eigenvalues of (10) have negative real parts.
(ii) Matrix A has a (nonnegative) right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue. Let f_m denote the mth complements of f and $a = \sum_{m,j,i=1}^{n} v_m w_i w_j \frac{\partial^2 f_m(0,0)}{\partial x_i \partial x_j}$; $b = \sum_{m,j,i=1}^{n} v_m w_i \frac{\partial^2 f_m(0,0)}{\partial x_i \partial \phi}$

Then the local dynamics of system (10) around $x = 0$ are totally determined by a and b
(a), $b < 0$. When $\phi < 0$, with $|\phi| \ll 1$, $x = 0$ is locally asymptotically stable and there exist a positive unstable equilibrium; when $0 < \phi \ll 1$, $x = 0$ is unstable and there exist a negative and locally asymptotically stable equilibrium
(b), $b < 0$. When $\phi < 0$, with $|\phi| \ll 1$, $x = 0$ is unstable; when $0 < \phi \ll 1$, $x = 0$ is locally asymptotically stable and there exist a positive unstable equilibrium.
(c), $b < 0$. When $\phi < 0$, with $|\phi| \ll 1$, $x = 0$ is unstable and there exist a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, $x = 0$ is stable and a positive unstable equilibrium appears
(d), $b < 0$. When ϕ changes from negative to positive, $x = 0$ changes its stability from stable to unstable. Correspondingly, an unstable equilibrium becomes positive and locally asymptotically stable.

The proof of theorem 4 can be found in [22].

The theorem A in appendix A is now applied to determine if the model system (3) exhibit a backward or forward bifurcation at $R_m = 1$.

Recall that

$$R_m = \sqrt{\frac{\alpha^2\beta(H)\beta_v k_m k_v}{\mu_v(k_v+\mu_v)(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}}$$

Let $\alpha = \alpha^*$ be a bifurcation parameter and if we consider the case $R_m = 1$ and solving for $\alpha = \alpha^*$, then

$$\alpha = \alpha^* = \sqrt{\frac{\mu_v(k_v+\mu_v)(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}{\beta(H)\beta_v k_m k_v}} \quad (11)$$

It follows that the disease free equilibrium point M_0 is locally stable when $\alpha < \alpha^*$, whereas it loses its stability when $\alpha > \alpha^*$,

So, the critical value $\alpha = \alpha^*$, is a bifurcation value.

The nature of the bifurcation involving the disease-free equilibrium M_0 at $\alpha = \alpha^*$ (or equivalently at $R_m = 1$) is investigated as follows:

Firstly, the Jacobian matrix of system (3) at point (M_0, α^*) is determined i.e.

$$J(M_0,\alpha^*) = \begin{pmatrix} -\mu_h & \pi & \pi & 0 & 0 & -\alpha^*\beta(H) \\ 0 & -(k_m+\pi+\mu_h) & 0 & 0 & 0 & \alpha^*\beta(H) \\ 0 & k_m & -(\sigma_m+\pi+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & -\alpha^*\beta_v & -\mu_v & 0 & 0 \\ 0 & 0 & \alpha^*\beta_v & 0 & -(k_v+\mu_v) & 0 \\ 0 & 0 & 0 & 0 & k_v & -\mu_v \end{pmatrix}$$

The characteristic equation of (12) has a simple zero eigenvalue i.e.

$\lambda(-\mu_h - \lambda)(-\mu_v - \lambda)B(\lambda) = 0$ (13)

Where

$B(\lambda) = \lambda^3 + B_0\lambda^2 + B_1\lambda + B_2$

$B_0 = (\sigma_m + 2\pi + k_v + k_m + 4\mu_v),$

$B_1 = (\pi^2 + \pi k_m + 2\pi k_v + 6\pi\pi^2 + \pi\sigma_m + k_m k_v + 3k_m\mu_v + k_m\sigma_m + 3k_v\mu_v + k_v\sigma_m + 6{\mu_v}^2 + 3\mu_v\sigma_m)$

$B_2 = \pi^2 k_v + 2\pi^2\mu_v + \pi k_m k_v + 2\pi k_m\mu_v + 4\pi k_v\mu_v + \pi k_v\sigma_m + 6\pi{\mu_v}^2 + 2\pi\mu_v\sigma_m + 2k_m k_v\mu_v + k_m k_v\sigma_m + 3k_m{\mu_v}^2 + 2k_m\mu_v\sigma_m + 3k_v{\mu_v}^2 + 2k_v\mu_v\sigma_m + 4{\mu_v}^3 + 3{\mu_v}^2\sigma_m$

has the following eigenvalues

$\lambda_1 = -\mu_h, \lambda_2 = -\mu_v, \lambda_3 = 0$

while λ_4 , λ_5 , and λ_6 are obtained from

$B(\lambda) = 0$ (14)

Equation (14) will have three negative real roots since there are no changes in signs in $B(\lambda)$ (By Descartes rule of signs).

Thus, $\lambda_3 = 0$ is a simple zero eigenvalue and the other eigenvalues are real and negative, then the assumptions of theorem 4 is then verified.

Furthermore, we obtain the right eigenvector associated with the zero eigenvalue $\lambda_3 = 0$ given by

$w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$, it follows that

$$\begin{pmatrix} -\mu_h & \pi & \pi & 0 & 0 & -\alpha^*\beta(H) \\ 0 & -(k_m+\pi+\mu_h) & 0 & 0 & 0 & \alpha^*\beta(H) \\ 0 & k_m & -(\sigma_m+\pi+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & -\alpha^*\beta_v & -\mu_v & 0 & 0 \\ 0 & 0 & \alpha^*\beta_v & 0 & -(k_v+\mu_v) & 0 \\ 0 & 0 & 0 & 0 & k_v & -\mu_v \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (15)$$

By solving the matrix equations (15) gives

$$w = \left(\frac{\left(\frac{\alpha^{*2}\beta(H)\beta_v k_m k_v - \pi(k_v+\mu_v)(\sigma_m+\pi+\mu_h+k_m)}{\mu_v k_m \alpha^*\beta_v}\right) w_5}{\frac{(k_v+\mu_v)(\sigma_m+\pi+\mu_h)}{k_m\alpha^*\beta_v} w_5, \frac{(k_v+\mu_v)}{\alpha^*\beta_v} w_5, -\frac{(k_v+\mu_v)}{\mu_v} w_5, w_5, \frac{k_v}{\mu_v} w_5} \right)^T$$

where $w_5 > 0$ is a free right eigenvector.

Similarly, the left eigenvector associated with the zero eigenvalue $\lambda_3 = 0$ given by $\bar{v} = (\bar{v}_1, \bar{v}_2, \bar{v}_3, \bar{v}_4, \bar{v}_5, \bar{v}_6)$, then (12)

$$(\bar{v}_1, \bar{v}_2, \bar{v}_3, \bar{v}_4, \bar{v}_5, \bar{v}_6) \begin{pmatrix} -\mu_h & \pi & \pi & 0 & 0 & -\alpha^*\beta(H) \\ 0 & -(k_m+\pi+\mu_h) & 0 & 0 & 0 & \alpha^*\beta(H) \\ 0 & k_m & -(\sigma_m+\pi+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & -\alpha^*\beta_v & -\mu_v & 0 & 0 \\ 0 & 0 & \alpha^*\beta_v & 0 & -(k_v+\mu_v) & 0 \\ 0 & 0 & 0 & 0 & k_v & -\mu_v \end{pmatrix}$$

$$\bar{v} = \left(0, \frac{k_m\alpha^*\beta_v}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\bar{v}_5, \frac{\alpha^*\beta_v}{(\sigma_m+\pi+\mu_h)}\bar{v}_5, 0, \bar{v}_5, \frac{(k_v+\mu_v)}{k_v}\bar{v}_5\right)$$

where $\bar{v}_5 > 0$ is a free left eigenvector.

The Computation of the Coefficient a and b

The coefficients

$$a = \sum_{m,i,j=1}^{6} \bar{v}_m w_i w_j \frac{\partial^2 f_m(M_0,\alpha^*)}{\partial x_i \partial x_j},$$

$$b = \sum_{m,i,j=1}^{6} \bar{v}_m w_i \frac{\partial^2 f_m(M_0,\alpha^*)}{\partial x_i \partial \varphi}$$

may be explicitly computed taking into account of system (3) and considering only the nonzero components of the left eigenvector $\bar{v}$; it follows that

Let $S_h = x_1$, $E_m = x_2$, $I_m = x_3$, $S_v = x_4$, $E_v = x_5$, $I_v = x_6$

With $x_1 + x_2 + x_3 = N_h(t) = 1$ and $x_4 + x_5 + x_6 = N_v(t) = 1$

Furthermore, we introduce the vector

$X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$, and then the model in system (3) can now be written in the form

$\frac{dX}{dt} = F(x)$, where $F = (f_1, f_2, f_3, f_4)^T$

It implies that system (3) can be written in term of the New variables as

$$\left.\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda_h - \alpha\beta_m x_6 x_1 - \mu_h x_1 + \pi x_2 + \pi x_3 \\
\frac{dx_2}{dt} &= f_2 = \alpha\beta_m x_4 x_1 - (k_m + \pi + \mu_h)x_2 \\
\frac{dx_3}{dt} &= f_3 = k_m x_2 - (\sigma_m + \pi + \mu_h)x_3 \\
\frac{dx_4}{dt} &= f_4 = \Lambda_v - \alpha\beta_v x_3 x_4 - \mu_v x_4 \\
\frac{dx_5}{dt} &= f_5 = \alpha\beta_v x_3 x_4 - (k_v + \mu_v)x_5 \\
\frac{dx_6}{dt} &= f_6 = k_v x_5 - \mu_v x_6
\end{aligned}\right\} \quad (17)$$

Where

$$a_0 = \left(\frac{\mu_v(k_v+\mu_v)(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h) + \pi(\sigma_m+\pi+\mu_h+k_m k_v)k_v\,\alpha^*\beta(H)}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right)$$

Thus, the following cases arise:

(i) The coefficient a is positive if $R_m > \sqrt{a_0}$

(ii) The coefficient a is negative if $R_m < \sqrt{a_0}$

$$b = \bar{v}_2 w_6 \frac{\partial^2 f_2}{\partial x_6 \partial \alpha^*} + \bar{v}_5 w_3 \frac{\partial^2 f_5}{\partial x_3 \partial \alpha^*},$$

$$b = \bar{v}_2 w_6 \beta(H) + \bar{v}_5 w_3 \beta_v,$$

$$b = \frac{(k_v+\mu_v)}{\alpha^*}\bar{v}_5 w_5 \left[{R_m}^2 + 1\right]$$

Thus, the coefficient b is always positive.

According to theorem A, it is the sign of the coefficient a that decides the local dynamics around the disease free equilibrium for $\alpha = \alpha^*$.

We conclude by applying theorem A that:

(i) If $a > 0$ and $b > 0$, a backward bifurcation occurs (ii) If $a < 0$ and $b > 0$, a forward bifurcation occurs

5. The Effect of Sanitation on Malaria Disease Transmission Dynamics

The aim, objective and ultimate goal of a public health strategy is to change the transmission dynamics of a disease such that if an infected individual enters a community he/she will not trigger an epidemic in the community.

Mathematically, it is reasonable to assume that if $R_m < 1$,

$$\text{then } \frac{dI_m}{dt} < 0 \quad (18)$$

For malaria, intervention can be in the following ways:

(i) Treatment using anti-malaria drugs

(ii) Reducing the rate of contact between susceptible individuals and infected mosquitoes and vice-versa.

The latter can be achieved through sleeping under mosquitoes treated nets, clean environment (i.e maintaining high level of sanitation) etc.

Model system (3) introduces the function $\beta(H)$ which defines the relationship between the transmission rate of malaria and sanitation is presented in three alternative forms as follows:

$$\beta(H) = \beta_{max} - \gamma_1 H \quad (19)$$

$$\beta(H) = \beta_{max}\, e^{-\gamma_2 H} \quad (20)$$

$$a = 2\bar{v}_2 w_1 w_6 \frac{\partial^2 f_2}{\partial x_1 \partial x_6} + 2\bar{v}_5 w_3 w_4 \frac{\partial^2 f_5}{\partial x_3 \partial x_4}$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \alpha^*\beta(H), \frac{\partial^2 f_4}{\partial x_2 \partial x_3} = \alpha^*\beta_v$$

$$a = 2\bar{v}_2 w_1 w_6\, \alpha^*\beta(H) + 2\bar{v}_5 w_3 w_4\, \alpha^*\beta_v\,,$$

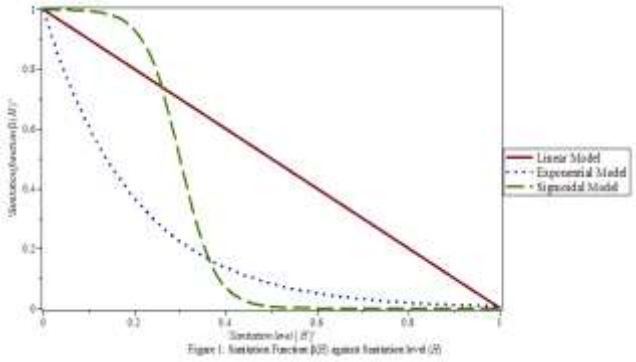
$$a = \frac{2(k_v+\mu_v)\bar{v}_5 {w_5}^2}{\mu_v}\left[{R_m}^2 - a_0\right]$$

$$= \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (16)$$

$$\beta(H) = \frac{\beta_{max}}{1+e^{\gamma_3(H-H_0)}} \quad (21)$$

Figure 1 gives a graphical interpretations of $\beta(H)$. The linear function in (19) predicts that malaria transmission is reduced proportionally to the improvement of sanitation conditions. The exponential function (20) predicts that a small improvement in hygiene or sanitation causes great impact on malaria transmission. Equation (21) is a sigmoidal curve predicts that low sanitation level has small effect on malaria transmission and high level of sanitation has great effect on malaria transmission, where γ's represent the expected reduction / increase in transmission rate of malaria as sanitation level H increase / decrease.

H is the sanitation level of the community and is defined to be $H \in [0,1]$, so that if $H = 0$, then there is maximum transmission of malaria in the community and if $H = 1$ means there is access to maximum sanitation facilities in the community hence, minimum transmission rate of malaria is achieved.



Using the functions defined in (19), (20) and (21), the required level of sanitation to prevent the outbreak and low transmission of malaria is defined.

Thus if $R_m < 1$ then the required level of sanitation to prevent the outbreak and low transmission of malaria using the linear, exponential and sigmoidal functions respectively are:

$$H > \frac{1}{\gamma_1}\left[\beta_{max} - \frac{\mu_v(k_v+\mu_v)(\sigma_m+\pi+\mu_h)(k_m+\pi+\mu_h)}{\alpha^2\beta_v k_m k_v}\right] \quad (22)$$

$$H > \frac{1}{\gamma_2} + \ln\left(\frac{\alpha^2\beta_{max}\,\beta_v k_m k_v}{\mu_v(k_v+\mu_v)(\sigma_m+\pi+\mu_h)(k_m+\pi+\mu_h)}\right) \quad (23)$$

$$H > H_0 + \frac{1}{\gamma_3}\ln\left(\frac{\alpha^2\beta_{max}\,\beta_v k_m k_v}{\mu_v(k_v+\mu_v)(\sigma_m+\pi+\mu_h)(k_m+\pi+\mu_h)}\right) \quad (24)$$

Where H_0 is a critical threshold

6. Sensitivity Analysis

The sensitivity analysis of the parameters is investigated in order to determine the relation of model parameters to disease transmission. The sensitivity analysis is carried out by computing sensitivity indices of basic reproduction number R_m which measures initial disease transmission using the approach of Arriola and Hyman [21]. The forward sensitivity index with respect to each of the parameter used in model (3) is presented below using the following formula

$$r_q^{R_m} = \frac{\partial R_m}{\partial q} \times \frac{q}{R_m},$$

where q and R_m represent the parameter and the reproduction number. Thus, the forward sensitivity index to key parameter under different level of sanitation functions is:

Linear Function

$\frac{\alpha}{R_m}\cdot\frac{\partial R_m}{\partial \alpha} = 1, \frac{\beta_{max}}{R_m}\cdot\frac{\partial R_m}{\partial \beta_{max}} = \frac{1}{2}\left(\frac{\beta_{max}}{\beta_{max}-\gamma_1 H}\right) < 1,$

$\frac{\gamma_1}{R_m}\cdot\frac{\partial R_m}{\partial \gamma_1} = -\frac{1}{2}\left(\frac{\gamma_1 H}{\beta_{max}-\gamma_1 H}\right) < 1$

$,\frac{\mu_v}{R_m}\cdot\frac{\partial R_m}{\partial \mu_v} = -\frac{1}{2}\left(\frac{2\mu_v+k_v}{k_v+\mu_v}\right) < 1$

$\frac{H}{R_m}\cdot\frac{\partial R_m}{\partial H} = -\frac{1}{2}\left(\frac{\gamma_1 H}{\beta_{max}-\gamma_1 H}\right) < 1, \frac{\beta_v}{R_m}\cdot\frac{\partial R_m}{\partial \beta_v} = \frac{1}{2} < 1, \frac{k_m}{R_m}\cdot\frac{\partial R_m}{\partial k_m} = \frac{1}{2}\left(\frac{\pi+\mu_h}{k_m+\pi+\mu_h}\right) < 1,$

$\frac{\sigma_m}{R_m}\cdot\frac{\partial R_m}{\partial \sigma_m} = -\frac{1}{2}\left(\frac{\sigma_m}{\sigma_m+\pi+\mu_h}\right) < 1$

$\frac{k_v}{R_m}\cdot\frac{\partial R_m}{\partial k_v} = \frac{1}{2}\left(\frac{\mu_v}{k_v+\mu_v}\right) < 1,$

$\frac{\pi}{R_m}\cdot\frac{\partial R_m}{\partial \pi} = -\frac{\pi}{2}\left(\frac{\sigma_m+2\pi+2\mu_h+k_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right) < 1,$

$\frac{\mu_h}{R_m}\cdot\frac{\partial R_m}{\partial \mu_h} = -\frac{\mu_h}{2}\left(\frac{\sigma_m+2\pi+2\mu_h+k_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right) < 1,$

Exponential Function

$\frac{\alpha}{R_m}\cdot\frac{\partial R_m}{\partial \alpha} = 1, \quad \frac{\beta_{max}}{R_m}\cdot\frac{\partial R_m}{\partial \beta_{max}} = \frac{1}{2} < 1,$

$\frac{\gamma_1}{R_m}\cdot\frac{\partial R_m}{\partial \gamma_1} = -\frac{1}{2}\gamma_2 H < 1, \frac{H}{R_m}\cdot\frac{\partial R_m}{\partial H} = -\frac{1}{2}\gamma_2 H < 1$

$\frac{\beta_v}{R_m}\cdot\frac{\partial R_m}{\partial \beta_v} = \frac{1}{2} < 1, \frac{k_m}{R_m}\cdot\frac{\partial R_m}{\partial k_m} = \frac{1}{2}\left(\frac{\pi+\mu_h}{k_m+\pi+\mu_h}\right) < 1$

$\frac{k_v}{R_m}\cdot\frac{\partial R_m}{\partial k_v} = \frac{1}{2}\left(\frac{\mu_v}{k_v+\mu_v}\right) < 1, \frac{\mu_v}{R_m}\cdot\frac{\partial R_m}{\partial \mu_v} = -\frac{1}{2}\left(\frac{2\mu_v+k_v}{k_v+\mu_v}\right) < 1$

$\frac{\pi}{R_m}\cdot\frac{\partial R_m}{\partial \pi} = -\frac{\pi}{2}\left(\frac{\sigma_m+2\pi+2\mu_h+k_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right) < 1,$

$\frac{\mu_h}{R_m}\cdot\frac{\partial R_m}{\partial \mu_h} = -\frac{\mu_h}{2}\left(\frac{\sigma_m+2\pi+2\mu_h+k_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right) < 1,$

$\frac{\sigma_m}{R_m}\cdot\frac{\partial R_m}{\partial \sigma_m} = -\frac{1}{2}\left(\frac{\sigma_m}{\sigma_m+\pi+\mu_h}\right) < 1$

Sigmoidal Function

$\frac{\alpha}{R_m}\cdot\frac{\partial R_m}{\partial \alpha} = 1, \quad \frac{\beta_{max}}{R_m}\cdot\frac{\partial R_m}{\partial \beta_{max}} = \frac{1}{2} < 1,$

$\frac{\gamma_1}{R_m}\cdot\frac{\partial R_m}{\partial \gamma_1} = -\frac{1}{2}\left(\frac{\gamma_3(H-H_0)e^{\gamma_3(H-H_0)}}{1+e^{\gamma_3(H-H_0)}}\right) < 1,$

$\frac{H}{R_m}\cdot\frac{\partial R_m}{\partial H} = -\frac{1}{2}\left(\frac{H\gamma_3 e^{\gamma_3(H-H_0)}}{1+e^{\gamma_3(H-H_0)}}\right) < 1 \quad , \frac{\beta_v}{R_m}\cdot\frac{\partial R_m}{\partial \beta_v} = \frac{1}{2} < 1,$

$\frac{k_m}{R_m}\cdot\frac{\partial R_m}{\partial k_m} = \frac{1}{2}\left(\frac{\pi+\mu_h}{k_m+\pi+\mu_h}\right) < 1,$

$\frac{k_v}{R_m}\cdot\frac{\partial R_m}{\partial k_v} = \frac{1}{2}\left(\frac{\mu_v}{k_v+\mu_v}\right) < 1,$

$\frac{\pi}{R_m}\cdot\frac{\partial R_m}{\partial \pi} = -\frac{\pi}{2}\left(\frac{\sigma_m+2\pi+2\mu_h+k_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right) < 1$, $\frac{\mu_v}{R_m}\cdot\frac{\partial R_m}{\partial \mu_v} = -\frac{1}{2}\left(\frac{2\mu_v+k_v}{k_v+\mu_v}\right) < 1,$

$\frac{\mu_h}{R_m}\cdot\frac{\partial R_m}{\partial \mu_h} = -\frac{\mu_h}{2}\left(\frac{\sigma_m+2\pi+2\mu_h+k_m}{(k_m+\pi+\mu_h)(\sigma_m+\pi+\mu_h)}\right) < 1$, $\frac{\sigma_m}{R_m}\cdot\frac{\partial R_m}{\partial \sigma_m} = -\frac{1}{2}\left(\frac{\sigma_m}{\sigma_m+\pi+\mu_h}\right) < 1$

The calculations of the sensitivity analysis using the linear, exponential and sigmoidal functions respectively shows that α is the most sensitive to R_m. The result also reveals that $\pi, \gamma_1, \gamma_2, \gamma_3$ and H have an inverse proportional relationship with R_m. Although, sensitivity analysis of π have an inverse proportional relationship with R_m, this will only reduce malaria prevalence in the population but cannot guarantee total eradication of malaria disease for some individuals will not have adequate or no treatment, hence, we suggest that more effort should be concentrated by the public health workers and health policy makers at reducing α the biting rates of the mosquitoes which can be achieved through improved sanitation and hygiene conditions, provision of mosquito treated nets and so on

7. Numerical Simulations and Discussion of Results

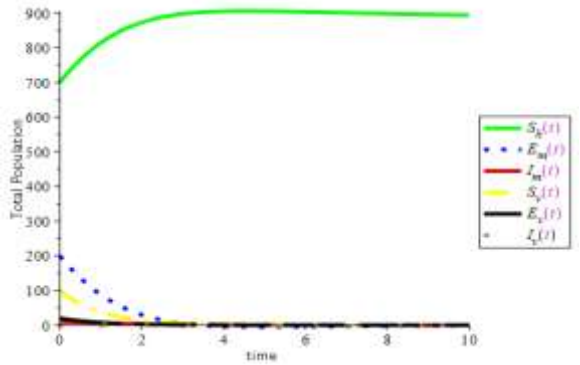


Figure 2: The graph of the total human population at parameter values $\Lambda_h = 0.1, \gamma = 1, H = 0.5, \mu_{vn} = 0.5, \mu_{max} = 0.9, \alpha = 0.1, \sigma_m = 0.001, \beta_{max} = 0.2, k_m = 0.001, k_v = 0.1, \mu_h = 0.002, \pi = 0.5, \Lambda_v = 0.1, \beta_v = 0.019$ and $R_0 < 1$

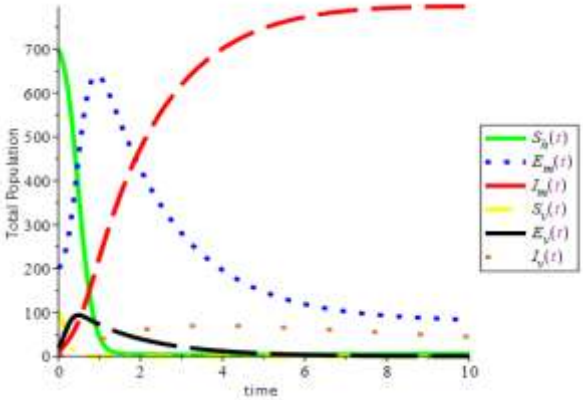


Figure 3: The graph of the total human population at parameter values $\Lambda_h = 0.1, \gamma = 1, H = 0.001, \mu_{vn} = 0.1, \mu_{max} = 0.05, \alpha = 0.8, \sigma_m = 0.001, \beta_{max} = 0.2, k_m =$

$0.5, k_v = 0.5, \mu_h = 0.002, \pi = 0.05, \Lambda_v = 0.1, \beta_v = 0.2$ and $R_0 > 1$

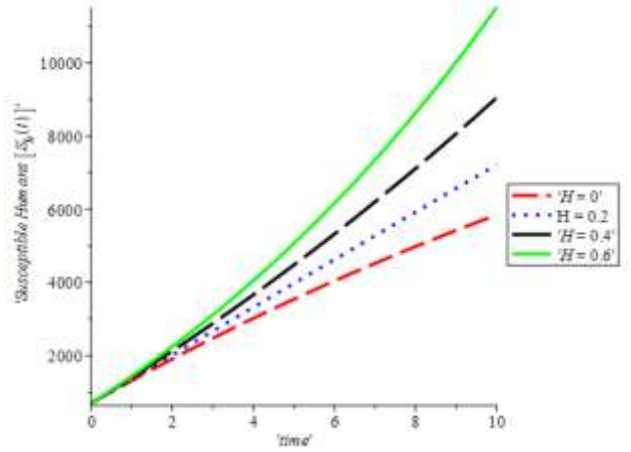


Figure 4: The graph of the Susceptible human population against time at varying values of sanitation level H with other parameter values fixed at $\Lambda_h = 0.1, \gamma = 1, H = 0.5, \mu_{vn} = 0.5, \mu_{max} = 0.9, \alpha = 0.1, \sigma_m = 0.001, \beta_{max} = 0.2, k_m = 0.001, k_v = 0.1, \mu_h = 0.002, \pi = 0.5, \Lambda_v = 0.1, \beta_v = 0.019$

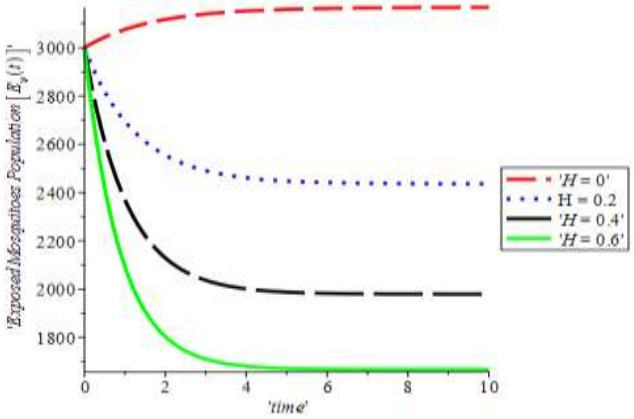


Figure 5: The graph of the Exposed mosquito population against time at varying values of sanitation level H with other parameter values fixed at $\Lambda_h = 0.1, \gamma = 1, H = 0.5, \mu_{vn} = 0.5, \mu_{max} = 0.9, \alpha = 0.1, \sigma_m = 0.001, \beta_{max} = 0.2, k_m = 0.001, k_v = 0.1, \mu_h = 0.002, \pi = 0.5, \Lambda_v = 0.1, \beta_v = 0.019$

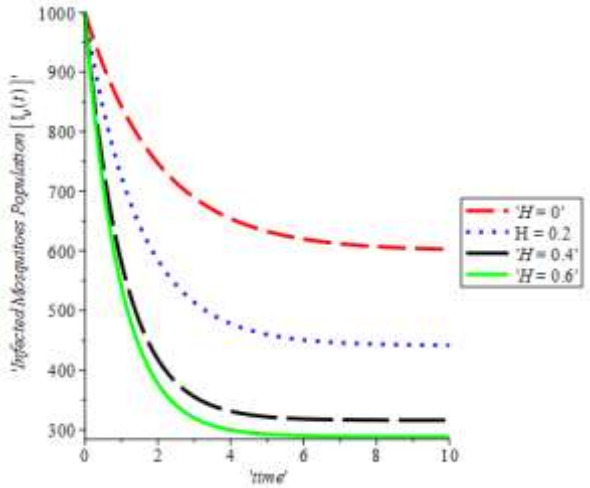


Figure 6: The graph of the Infected mosquito population against time at varying values of sanitation level H with other parameter values fixed at $\Lambda_h = 0.1, \gamma = 1, H = 0.5, \mu_{vn} = 0.5, \mu_{max} = 0.9, \alpha = 0.1, \sigma_m = 0.001, \beta_{max} = 0.2, k_m = 0.001, k_v = 0.1, \mu_h = 0.002, \pi = 0.5, \Lambda_v = 0.1, \beta_v = 0.019$

Figure 2 support our theoretical result that the total human population is stable if $R_0 < 1$ under a high level of sanitation H and become unstable if $R_0 > 1$ when poor sanitation or no sanitation as depicted in **figure 3**. Increase in sanitation level H bring about low level of transmission which result in the increase in number of susceptible individual as shown in **figure 4**. Furthermore, increase in the sanitation level result in significant reduction in the number of exposed and infected mosquitoes as seen in **figure 5** and **figure 6** respectively.

8. Conclusion

A rigorous mathematical analysis on the effect of sanitation on malaria transmission model has been carried out in this work. The local and global stabilities of the model investigated. The result revealed that the malaria model exhibit a backward bifurcation which suggests that malaria eradication does not only depend on reducing $R_m < 1$ but other factors such as sanitation should be considered and sustained. The result of the sensitivity analysis gives a clear indication to public health workers and health policy makers that more effort should be concentrated at reducing biting rates of the mosquitoes α through sanitation among other measures for a clean environment.

References

[1] Centre for Disease Control (CDC)., (2007): World Wide Web http:www.cdc.gov/malaria/facts.htm Retrieved on 22nd August 2011
[2] Allman E.S. and Rhodes J.A., (2004):Mathematical models in Biology an introduction. New York: Cambridge University press.
[3] Burke A.C.,(2010):Infectious diseases in Critical care Medicine. New York: Informa Health care USA. Inc.
[4] Bupa U.K., (2009): Malaria, symptoms, causes and treatment of Disease. http:hcd2.bupa.co.uk/fact_sheets/html/malaria.htmt Accessed on 20th August2011
[5] Abu-Raddad L.J., Patnaik P. and Kublin J.G., (2006): Dual infection with HIV and malaria fuels the spread of both diseases in Sub-Saharan Africa. Section 314(5805) ,1603-1606
[6] Li-Ming Cai, Abid Ali Lashari, II Hyo Jung, Kazeem Oare Okosun and Young II Seo., (2013): "Mathematical Analysis of Malaria Model with Partial Immunity to Re-infection" Journal of Abstract and Applied Analysis, Hindawi Publishing Corporation.
[7] Buonomo B. and Lacitignola D., (2011): "On the backward bifurcation of a vaccination model with nonlinear incidence". Nonlinear Analysis: Modeling and Control, Vol. 16, No. 1, pp 30-46
[8] Vanden Driessche, P. and Watmough, J., (2002):" Reproduction numbers and Sub-threshold endemic equilibria for compartmental models of disease Transmission" Mathematical Bio-sciences, 180:29-48.

[9] Castillo – Chavez and Song, B.,(2004):"Dynamical Models of Tuberculosis and their Applications". Mathematical Biosciences and Engineering,1(2): p p 361- 404.
[10] J. Carr., (1981): Applications or Centre Manifold Theory, Springer, New York.
[11] J. Guckenheimer, P. Holmes., (1983): Nonlinear Oscillations, Dynamical Systems and Bifurcations of Vector Fields, Springer – Veriag, Berlin.
[12] Oluyo T.O. and Adeniyi M.O. (2014): Mathematical Analysis of Malaria-Pneumonia Model with Mass Action. International Journal of Applied Mathematics, ISSN: 2051-5227, Vol.29, Issue. 2
[13] Lawi G.O, Mugisha J.Y.T and Omolo-Ongati N., (2013): " Modeling Co-infection of Paediatric Malaria and Pneumonia ". Int. Journal of Math. Analysis, Vol. 7, no. 9, pp 413-424
[14] Guimaraens M.A and Codeco C.T: Experiment with mathematical model to simulate hepatitis A population dynamics under different levels of endemicity. Cad. Saude Publica, Rio de Janeiro, 21(5): 1531-1539, set out, 2005
[15] Hethcote H.W., (2000): "The mathematics of Infectious Diseases". SIAM Rev., 42, pp 599-653
[16] Diekmann O, J.A.P Heesterbeek., (2000): "Mathematical Epidemiology of Infectious Diseases" John Wiley 171 & Son, Ltd
[17] Heffernan, J. M., Smith, R. J. and Wahl, L. M., (2005): "Perspectives on the basic reproductive Ratio" J. R. Soc. Interface 2, 281– 293H.
[18] Lakshmkantham V, Leela S. And Martynyuk A.A., (1989): "Stability Analysis of Nonlinear Systems, Marcel Dekker, New York." ISBN 0-8247-8067-1. Pure and Applied Mathematics: A series of Monographs and Textbooks, Vol. 125
[19] Mushayabasa S., Tchuenche J.M.,Bhunu C.P., Ngarakana-Gwasira E., (2011): "Modeling gonorrhoea and HIV co-interaction" Journal of Biological and Information Processing Sciences 103:27-37
[20] A.B. Gumel, S.M. Moghadas., (2003): A qualitative studyof a vaccination model with non- linear incidence, App. Math. Comput., 143, pp. 409 –419.
[21] Arriola L., Hyman J., (2005): Lecture notes, forward and adjoint sensitivity analysis: with application in Dynamical Systems, Linear Algebra and Optimization Mathematical and Theoretical Biology Institute, Summer.
[22] D. Xiao, S. Ruan.,(2007): Global analysis of an epidemic model with non-monotone incidence rate, Math. Biosci., 208, pp. 419-429.