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The role of treatment and counseling in an HIV/AIDS, Malaria and Tuberculosis model: an analysis of HIV/AIDS and Malaria

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Abstract: *HIV/AIDS remains one of the leading causes of death in the world with its effects most devastating in Sub Saharan Africa due to its dual infection with opportunistic infections especially malaria and tuberculosis. This study presents a co infection deterministic model defined by a system of ordinary differential equations for HIV/AIDS, malaria and tuberculosis. The HIV/AIDS malaria co infection sub model is analyzed to determine the conditions for the stability of the equilibria points and assess the role of treatment and counseling in controlling the spread of the infections. This study shows that treatment of malaria a lone even in the absence of HIV/AIDS, may not eliminate malaria from the community therefore strategies for the reduction of malaria infections in humans should not only target malaria treatment but also the reduction of mosquito biting rate. The study showed that counseling is the most sensitive parameter in the spread of HIV/AIDS - malaria co infections, therefore effective counseling strategy is very useful in controlling the spread of the HIV/AIDS and malaria co infections. The study further showed that ARV treatment and counseling for HIV/AIDS infectives have no effect on the spread of malaria. Finally the HIV/AIDS malaria model undergoes backward bifurcation which is favoured by the occurrence of high mosquito biting rate.*

Keywords: *Bifurcation, Counseling, HIV/AIDS - TB and Malaria, Stability, Treatment.*

I. Introduction

Infectious diseases, alongside cardiovascular diseases and cancer, have been the main threat to human health. Acute and chronic respiratory diseases, especially pulmonary tuberculosis, malaria and HIV/AIDS are responsible for a large portion of mortality especially in developing countries [16].

Globally HIV/AIDS has killed more than 35 million people since it was first discovered in 1981 and almost 70 million people have been infected with the HIV/AIDS virus making it one of the most destructive epidemics in recorded history [22]. It remains one of the leading causes of death in the world with its effects most devastating in sub saharan Africa. One of the key factors that fuels the high incidence of HIV/AIDS in Sub Saharan Africa is its dual infection with malaria and tuberculosis [16].

World Health Organization statistics show that tuberculosis (TB) is the most common illness and the leading cause of death among people living with HIV/AIDS, accounting for one in four HIV/AIDS related deaths and at least one-third of the 34 million people living with HIV/AIDS worldwide are infected with latent TB. Persons co infected with TB and HIV/AIDS are 21-34 times more likely to develop active TB disease than persons without HIV/AIDS. In 2011, there were an estimated 1.1 million HIV/AIDS positive new TB cases globally and about 79 percent of these people live in Sub-Saharan Africa [22].

According to the World Health Organization report of April 2008, malaria increases the viral load in HIV/AIDS patients. Conversely HIV/AIDS increases the risk of malaria infection and accelerate the development of clinical symptoms of malaria with the greatest impact on the immune suppressed persons [22]. Ever since the co infections were recorded, malaria has seen a 28 percent increase in its prevalence and malaria related death rates have also nearly doubled for those with co infections [7]. The co infection between malaria and HIV-1 is the commonest in Sub-Saharan Africa and, to a lesser extent, in other developing countries. It is estimated that 22 million Africans are infected with HIV-1, and around 500 million are suffering from malaria annually [22].

Hohman and Kami [10], discovered that HIV/AIDS and malaria have similar global distributions. The discovery motivated a study on the impact of HIV/AIDS and malaria co infection and established that globally, 500 million people are infected with malaria annually resulting in one million deaths yearly. Thirty three million people get infected with HIV/AIDS and 2 million die from it every year. The study further showed that those with HIV/AIDS have more frequent episodes of symptomatic malaria and that malaria increases HIV/AIDS plasma viral load and decrease $CD4^+$ cells. During episodes of parasitemia, HIV/AIDS infected people have an increase in viremia leading to potential increase in risk of HIV/AIDS transmission. A comparison of the geographical distributions of HIV/AIDS, TB and malaria especially in Africa, reveal that these three diseases have similar geographical distributions suggesting a possible existence of HIV/AIDS, TB and malaria co infection. This may be due to shared risk factors and/or the presence of opportunistic infections.

Audu et al. [4] investigated the possible impact of co infections of tuberculosis and malaria on the $CD4^+$ cell counts of HIV/AIDS patients and established the following: The healthy control group recorded a median $CD4^+$ cell counts of 789 cells/ µl (789 cells per mm^3 of blood); subjects infected with HIV/AIDS only recorded a median $CD4^+$ cell counts of 386 cell/ µl; subjects co infected with HIV/AIDS and TB recorded a median $CD4^+$ cell counts of 268 cell/ µl; subjects co infected with HIV/AIDS and malaria recorded a median $CD4^+$ cell counts of 211 cell/µl and those co infected with HIV/AIDS, malaria and TB recorded the lowest median $CD4^+$ cell counts of 182 cell/µl.

Motivated by these findings, a deterministic model exploring the joint dynamics of the simultaneous co infections of HIV/AIDS, TB and malaria incorporating treatment and counseling is presented. It represents the first deterministic mathematical model incorporating HIV/AIDS, TB and Malaria co infections within a single model to gain insights into their combined transmission dynamics and determine effective control strategies.

II. Model Formulation And Description

To study the dynamics of HIV/AIDS, malaria and TB co infection, a deterministic model is formulated described by a system of ordinary differential equations. The model sub-divide the human population into the following epidemiological classes: $S_H(t)$ - Susceptible population at time t, $I_M(t)$ - Malaria infectives at time t, $I_H(t)$ - HIV cases at time t, $I_A(t)$ - AIDS cases at time t, $I_T(t)$ - TB cases at time t. $I_{HM}(t)$ - Those co infected with malaria and HIV at time t, $I_{AM}(t)$ - Those co infected with malaria and AIDS at time t, $I_{MT}(t)$ - Those co infected with malaria and TB at time t, $I_{HT}(t)$ - Those co infected with HIV and TB at time t, $I_{AT}(t)$ - Those co infected with AIDS and TB at time t, $I_{HMT}(t)$ - Those co infected with HIV, Malaria and TB at time t, $I_{AMT}(t)$ - Those co infected with AIDS, Malaria and TB at time t. The total human population ($N_H(t)$) is therefore denoted by: $N_H(t) = S_H(t) + I_M(t) + I_H(t) + I_A(t) + I_T(t) + I_{HM}(t) + I_{AM}(t) + I_{MT}(t) + I_{HT}(t) + I_{AT}(t) + I_{HMT}(t) + I_{AMT}(t)$.

The vector (mosquito) population at time t denoted by $N_V(t)$ is sub-divided into the following classes: $S_V(t)$ - Vector susceptibles at time t, $I_V(t)$ – Vector infectives at time t. The total vector population $N_V(t)$ is given by $N_V(t) = S_V(t) + I_V(t)$.

2.1 Definition Of Parameters

It is assumed that susceptible humans are recruited into the population at a constant rate either by birth or recovery from malaria and TB. They acquire infection with either HIV/AIDS, malaria or TB and move to the infectious classes. Susceptible mosquitoes are recruited into the mosquito population at a constant rate. They acquire malaria infection following a blood meal feeding on infected malaria humans, becomes infectious and move to the infectious class.

The recruitment rate of humans into the susceptible population is denoted by Λ_H while that of vectors (mosquitoes) is denoted by Λ_V and are both assumed to be constant. The natural death rate of humans is given by d_n while that of vectors is given by d_v. The death rates due to AIDS, malaria and TB in humans are da, d_m and d_t respectively. The parameters d_{am}, d_{mt}, d_{at} and d_{amt} account for the combined death rates in the I_{AM}, I_{MT}, I_{AT} and I_{AMT} classes respectively. The parameters r_m and r_t are the recovery rates from malaria and TB respectively due to effective treatment. It is assumed that the recovered individuals do not acquire temporary immunity to either or both diseases thus become susceptible again. The model assumes that susceptible humans cannot simultaneously get infected with malaria, HIV/AIDS and TB since the transmission mechanics are completely different for the three diseases. The model further assumes that humans acquire HIV/AIDS through sexual contacts between an infective and a susceptible. The average force of infection for HIV/AIDS denoted λ_{ah} is given by

$$\lambda_{ah} = \frac{\beta_a(1-\delta)c_1(I_H + I_{HM} + I_{HT})}{N_H} \qquad (2.1.1)$$

Where βa is the average transmission probability of HIV/AIDS between an infective and a susceptible per sexual contact and c_1 is the per capita number of sexual contacts of susceptible humans with HIV/AIDS infected individuals per unit time. The parameter δ measures the effectiveness of counseling through condom use and a reduction in the number of sexual partners, where $0 \leq \delta \leq 1$. Effective counseling reduces the value of the parameter c_1. The model assumes that the classes I_{HMT}, I_A, I_{AM}, I_{AT} and I_{AMT} do not transmit the virus due to acute ill health and noticeable AIDS symptoms.

Define α_1 as the number of bites per human per mosquito (biting rate of mosquitoes), β_m as the transmission probability of malaria in humans per bite thus the force of infection with malaria for humans, denoted λ_{mh} is given by

$$\lambda_{mh} = \frac{\alpha_1 \beta_m I_V}{N_H} \qquad (2.1.2)$$

whereas the average force of infection with malaria for vectors, denoted λ_{mv} is given by

$$\lambda_{mv} = \frac{\alpha_1 \beta_v (I_M + I_{HM} + I_{MT} + I_{AM} + I_{HMT} + I_{AMT})}{N_H} \qquad (2.1.3)$$

where β_v is the transmission probability of malaria in vectors from any infected human. Finally the average force of infection for TB denoted λ_{th} is given by

$$\lambda_{th} = \frac{\beta_t c_2 (I_T + I_{HT} + I_{MT} + I_{HMT} + I_{AMT} + I_{AT})}{N_H} \qquad (2.1.4)$$

Where β_t is the transmission probability of TB in humans and c_2 is the average per capita contact rate of susceptible humans with TB infected individuals. The rate of progression from HIV to AIDS for the untreated HIV cases is p. The parameters $\theta_1 p$, $\theta_2 p$ and $\theta_3 p$ account for increased rates of progression to AIDS for individuals co infected with HIV - TB, HIV - malaria and HIV malaria - TB respectively where $\theta_1 < \theta_2 < \theta_3$.

Define α as the proportion of the HIV/AIDS infectives receiving effective treatment. This involves the administration of ARV'S that keeps the HIV patients from progressing to AIDS while transferring the AIDS patients back to the HIV classes. The modification parameters e^h_m, e^h_t and e^h_{mt} account for the reduced susceptibility to infection with HIV for individuals in the I_M, I_T and the I_{MT} classes respectively due to reduced sexual activity as a result of ill health where $e^h_m < 1$, $e^h_t < 1$, $e^h_m < 1$, $e^h_{mt} << 1$. The parameters e^m_a, e^m_h, e^m_{ht}, e^m_{at}, account for the increased susceptibility to infection with malaria for individuals already infected with AIDS, HIV, HIV - TB and AIDS - TB respectively due to suppressed immune system where $e^m_a > 1$, $e^m_h > 1$, $e^m_{ht} > 1$, $e^m_{at} > 1$. It is also clear that $e^m_a < e^m_{at}$ and $e^m_h < e^m_{ht}$. The parameters e^t_h, e^t_a, e^t_{mh} and e^t_{am} account for the increased susceptibility to infection with TB for individuals already infected with HIV, AIDS, HIV - malaria and AIDS - malaria respectively due to suppressed immune system where $e^t_h > 1$, $e^t_a > 1$, $e^t_{hm} > 1$, $e^t_{am} > 1$. Again $e^t_h < e^t_{hm}$ and $e^t_a < e^t_{am}$. Malaria and TB does not lead to the depletion of the $CD4^+$ cell counts, however their association with HIV/AIDS leads to a significant reduction in the $CD4^+$ cell counts within an individual leading to faster progression to AIDS. Combining all the aforementioned assumptions and definitions, the model for the transmission dynamics of HIV/AIDS, TB and malaria is given by the following system of differential equations.

2.2 The Model Equations

$$\begin{aligned}
\frac{dS_H(t)}{dt} &= \Lambda_H + r_m I_M(t) + r_t I_T(t) - \lambda_{ah} S_H(t) \qquad (2.2.1)\\
&\quad -\lambda_{mh} S_H(t) - \lambda_{th} S_H(t) - d_n S_H(t)\\
\frac{dI_M(t)}{dt} &= \lambda_{mh} S_H(t) + r_t I_{MT}(t) - r_m I_M(t) - e^h_m \lambda_{ah} I_M(t)\\
&\quad -\lambda_{th} I_M(t) - d_n I_M(t) - d_m I_M(t).\\
\frac{dI_H(t)}{dt} &= \lambda_{ah} S_H(t) + r_m I_{HM}(t) + r_t I_{HT}(t) - (1-\alpha) p I_H(t)\\
&\quad -e^m_h \lambda_{mh} I_H(t) - e^t_h \lambda_{th} I_H(t) - d_n I_H(t) + \alpha I_A(t).
\end{aligned}$$

$$
\begin{aligned}
\frac{dI_A(t)}{dt} &= (1-\alpha)pI_H(t) + r_m I_{AM}(t) + r_t I_{AT}(t) - e_a^m \lambda_{mh} I_A(t) \\
&\quad -e_a^t \lambda_{th} I_A(t) - d_a I_A(t) - d_n I_A(t) - \alpha I_A(t) \\
\frac{dI_T(t)}{dt} &= \lambda_{th} S_H(t) + r_m I_{MT}(t) - e_t^a \lambda_{ah} I_T(t) - \lambda_{mh} I_T(t) \\
&\quad -d_n I_T(t) - d_t I_T(t) - r_t I_T(t) \\
\frac{dI_{MH}(t)}{dt} &= e_h^m \lambda_{mh} I_H(t) + e_m^a \lambda_{ah} I_M(t) + r_t I_{HMT}(t) - r_m I_{HM}(t) - e_{hm}^t \lambda_{th} I_{HM}(t) + \\
&\quad \alpha I_{AM}(t) - d_m I_{HM}(t) - (1-\alpha)\theta_2 p I_{HM}(t) - d_n I_{HM}(t) \\
\frac{dI_{AM}(t)}{dt} &= (1-\alpha)\theta_2 p I_{HM}(t) + e_a^m \lambda_{mh} I_A(t) - r_m I_{AM}(t) - d_m I_{AM}(t) - \alpha I_{AM}(t) \\
&\quad +r_t I_{AMT}(t) - e_{am}^t \lambda_t I_{AM}(t) - d_n I_{AM}(t) - d_a I_{AM}(t) - d_{am} I_{AM}(t). \\
\frac{dI_{MT}(t)}{dt} &= \lambda_{th} I_M(t) + \lambda_{mh} I_T(t) - r_m I_{MT}(t) - e_{mt}^a \lambda_{ah} I_{MT}(t) - r_t I_{MT}(t) \\
&\quad -d_m I_{MT}(t) - d_n I_{MT}(t) - d_t I_{MT}(t) - d_{mt} I_{MT}. \\
\frac{dI_{HT}(t)}{dt} &= e_t^a \lambda_{ah} I_T(t) + r_m I_{HMT}(t) + e_h^t \lambda_{th} I_H(t) - e_{ht}^m \lambda_{mh} I_{HT}(t) - (1-\alpha)\theta_1 p I_{HT}(t) \\
&\quad -d_n I_{HT}(t) - d_t I_{HT}(t) - r_t I_{HT}(t) + \alpha I_{AT}(t) \\
\frac{dI_{AT}(t)}{dt} &= e_a^t \lambda_t I_A(t) + r_m I_{AMT}(t) + (1-\alpha)\theta_1 p I_{HT}(t) - \alpha I_{AT}(t) \\
&\quad -e_{at}^m \lambda_{mh} I_{AT}(t) - d_n I_A(t) - d_a I_{AT}(t) - d_t I_{AT}(t) - r_t I_{AT}(t) - d_{at} I_{AT} \\
\frac{dI_{HMT}(t)}{dt} &= e_{ht}^m \lambda_m I_{HT}(t) + e_{hm}^t \lambda_{th} I_{HM}(t) + e_{mt}^a \lambda_{ah} I_{MT}(t) + \alpha I_{AMT}(t) \\
&\quad -r_m I_{HMT}(t) - d_m I_{HMT}(t) - d_n I_{HMT}(t) \\
&\quad -(1-\alpha)\theta_3 p I_{HMT}(t) - d_t I_{HMT}(t) - r_t I_{HMT}(t) - d_{mt} I_{HMT} \\
\frac{dI_{AMT}(t)}{dt} &= e_{at}^m \lambda_{mh} I_{AT}(t) + e_{am}^t \lambda_{th} I_{AM}(t) + (1-\alpha)\theta_3 p I_{HMT}(t) \\
&\quad -r_m I_{AMT}(t) - d_m I_{AMT}(t) - d_a I_{AMT}(t) - \alpha I_{AMT}(t) \\
&\quad -d_n I_{AMT}(t) - d_t I_{AMT}(t) - r_t I_{AMT}(t) - d_{amt} I_{AMT} \\
\frac{dS_V(t)}{dt} &= \Lambda_V - \lambda_{mv} S_V(t) - d_v S_V(t) \\
\frac{dI_V(t)}{dt} &= \lambda_{mv} S_V(t) - d_v I_V(t).
\end{aligned}
$$

2.3 POSITIVITY AND BOUNDEDNESS OF SOLUTIONS

The model system 2.2.1 describes living populations therefore the associated state variables are non-negative for all time t > 0. The solutions of this model with positive initial data therefore remain positive for all time t > 0.

Lemma 2.1. *Let the initial data set be* $\{(S_H(0), S_V(0) > 0), (I_M(0), I_H(0), I_A(0), I_T(0), I_{HM}(0), I_{AM}(0), I_{MT}(0), I_{HT}(0), I_{AT}(0), I_{HMT}(0), I_{AMT}(0), I_V(0))\} \in \Psi$. *Then the solution set* $\{(S_H, S_V, I_M, I_H, I_A, I_T\ I_{HM}, I_{AM}, I_{MT}, I_{HT}, I_{AT}, I_{HMT}, I_{AMT}, I_V\}(t)$ *is positive for all time* $t > 0$.

Proof. Consider the first equation of 2.2.1 at time t

$$\frac{dS_H}{dt} = \Lambda_H + r_m I_M + r_t I_T - \lambda_{ah} S_H - \lambda_{mh} S_H - \lambda_{th} S_H - d_n S_H$$

then

$$\frac{dS_H}{dt} \geq -(\lambda_{ah} + \lambda_{mh} + \lambda_{th} + d_n) S_H$$

$$\int \frac{dS_H}{S_H} \geq -\int (\lambda_{ah} + \lambda_{mh} + \lambda_{th} + d_n) d(t)$$

$$S_H(t) \geq S_H(0) e^{-\int (\lambda_{ah} + \lambda_{mh} + \lambda_{th} + d_n) d(t)} \geq 0$$

From the second equation of 2.2.1 at time t

$$\frac{dI_M}{dt} = \lambda_{mh} S_H + r_t I_{TM} - r_m I_M - e_m^a \lambda_{ah} I_M - \lambda_{th} I_M - d_n I_M - d_m I_M.$$

then

$$\frac{dI_M}{dt} \geq -(r_m + e_m^a \lambda_{ah} + \lambda_{th} + d_n + d_m) I_M.$$

$$\frac{dI_M}{I_M} \geq -\int (r_m + e_m^a \lambda_{ah} + \lambda_{th} + d_n + d_m) dt.$$

$$I_M(t) \geq I_M(0) e^{-\int (r_m + e_m^a \lambda_{ah} + \lambda_{th} + d_n + d_m) dt} \geq 0.$$

We can proceed in a similar manner and show that all the state variables are positive for all time t.

Lemma 2.2. *The solutions of the model 2.2.1 are uniformly bounded in a proper subset* $\Psi = \Psi_H \times \Psi_V$

Proof. Let $\{(S_H, I_M, I_H, I_A, I_T\, I_{HM}, I_{AM}, I_{MT}, I_{HT}, I_{AT}, I_{HMT}, I_{AMT}\}(t) \in \mathbb{R}_+^{12}$, be any solution with non-negative initial conditions. The rate of change of the total human population with time is given by:

$$\frac{dN_H}{dt} = \Lambda_H - d_n N_H - (I_M + I_{HM}(t) + I_{AM} + I_{MT} + I_{HMT} + I_{AMT}) d_m -$$

$$(I_T + I_{MT} + I_{HT} + I_{AT} + I_{HMT} + I_{AMT}) d_t - (I_A + I_{AM} + I_{AT} + I_{AMT}) d_a$$

$$- d_{am} I_{AM} - d_{mt}(I_{MT} + I_{HMT}) - d_{at} I_{AT} - d_{amt} I_{AMT}$$

The model system 2.2.1 has a varying human population size $\frac{dN_H}{dt} \neq 0$ and therefore a trivial equilibrium is not feasible. Whenever $N_H > \frac{\Lambda_H}{d_n}$, then $\frac{dN_H}{dt} < 0$. Since $\frac{dN_H}{dt}$ is bounded by $\Lambda_H - d_n N_H$, a standard comparison theorem by (Birkoff and Rota, 1989) shows that $0 \leq N_H(t) \leq N_H(0) e^{-d_n} + \frac{\Lambda_H}{d_n}(1 - e^{-d_n})$, where $N_H(0)$ represents the value of $N_H(t)$ evaluated at the initial values of the respective variables. Thus as $t \to \infty$, we have, $0 \leq N_H(t) \leq \frac{\Lambda_H}{d_n}$. In particular, $N_V(t) \leq \frac{\Lambda_H}{d_n}$, if $N_0 \leq \frac{\Lambda_H}{d_n}$. This shows that N_H is bounded and all the feasible solutions of the human only component of model 2.2.1 starting in the region Ψ_H approach, enter or stay in the region, where:

$$\Psi_H = \{(S_H, I_M, I_H, I_A, I_T, I_{MH}, I_{MA}, I_{MT}, I_{HT}, I_{TA}, I_{MHT}, I_{MAT}) : N(t) \leq \tfrac{\Lambda_H}{d_n}\}.$$

Similarly let $\{(S_V, I_V\}(t) \in \mathbb{R}_+^2$, be any solution with non-negative initial conditions. The rate of change of the total vector population with time is given by:

$\frac{dN_V}{dt} = \Lambda_V - (S_V(t) - I_V(t)) d_v$. $\frac{dN_V}{dt} \neq 0$ and therefore a trivial equilibrium is not feasible. Whenever $N_V > \frac{\Lambda_V}{d_v}$, then $\frac{dN_V}{dt} < 0$. Since $\frac{dN_V}{dt}$ is bounded by $\Lambda_V - d_v N_V$, a standard comparison theorem by Birkoff and Rota (1989), shows that $0 \leq N_V(t) \leq N_V(0) e^{-d_v} + \frac{\Lambda_V}{d_v}(1 - e^{-d_v})$, where $N_V(0)$ represents the value of $N_V(t)$ evaluated at the initial values of the respective variables. Thus as $t \to \infty$, $0 \leq N_V(t) \leq \frac{\Lambda_V}{d_v}$. In particular, $N(t) \leq \frac{\Lambda_V}{d_v}$, if $N_0 \leq \frac{\Lambda_V}{d_v}$. This shows that N_V is bounded and all the feasible solutions of the vector only component of model 2.2.1 starting in the region Ψ_V approach, enter or stay in the region, where: $\Psi_V = \{(S_V, I_V) : N_V \leq \frac{\Lambda_V}{d_v}\}$. □.

III. Hiv/Aids-Malaria Model

The model of HIV/AIDS-malaria co infection model is obtained by setting $I_T = I_{MT} = I_{HT} = I_{AT} = I_{HMT} = I_{AMT} = 0$. The total human population is given by $N_H = S_H + I_M + I_H + I_A + I_{HM} + I_{AM}$ and the total vector population $N_V = S_V + I_V$. The model equations are given by:

$$\begin{aligned}
\frac{dS_H(t)}{dt} &= \Lambda_H + r_m I_M(t) - \lambda_{ah} S_H(t) - \lambda_{mh} S_H(t) - d_n S_H(t) \qquad (3.0.1)\\
\frac{dI_M(t)}{dt} &= \lambda_{mh} S_H(t) - r_m I_M(t) - e_m^h \lambda_{ah} I_M(t) - d_n I_M(t) - d_m I_M(t).\\
\frac{dI_H(t)}{dt} &= \lambda_{ah} S_H(t) + r_m I_{HM}(t) - (1-\alpha) p I_H(t) - e_h^m \lambda_{mh} I_H(t) - d_n I_H(t) + \alpha I_A(t).\\
\frac{dI_A(t)}{dt} &= (1-\alpha) p I_H(t) + r_m I_{AM}(t) - e_a^m \lambda_{mh} I_A(t) - d_a I_A(t) - d_n I_A(t) - \alpha I_A(t)\\
\frac{dI_{MH}(t)}{dt} &= e_h^m \lambda_{mh} I_H(t) + e_m^a \lambda_{ah} I_M(t) - r_m I_{HM}(t) + \alpha I_{AM}(t)\\
&\quad -d_m I_{HM}(t) - (1-\alpha)\theta_2 p I_{HM}(t) - d_n I_{HM}(t)\\
\frac{dI_{AM}(t)}{dt} &= (1-\alpha)\theta_2 p I_{HM}(t) + e_a^m \lambda_{mh} I_A(t) - r_m I_{AM}(t) - d_m I_{AM}(t) - \alpha I_{AM}(t)\\
&\quad -d_n I_{AM}(t) - d_a I_{AM}(t) - d_{ma} I_{AM}(t).\\
\frac{dS_V(t)}{dt} &= \Lambda_V - \lambda_{mv} S_V(t) - d_v S_V(t)\\
\frac{dI_V(t)}{dt} &= \lambda_{mv} S_V(t) - d_v I_V(t).
\end{aligned}$$

$\frac{dN_H}{dt} = \Lambda_H - d_n N_H - d_m(A_{AM} + d_a B_{AM}) - d_{am} I_{AM}$
$A_{AM} = (I_{HM} + I_M + I_{AM})$, and $B_{AM} = (I_A + I_{AM})$, $\frac{dN_V}{dt} = \Lambda_V - d_v N_V$.

The forces of infection are given by:

$$\lambda_{ah} = \frac{\beta_a(1-\delta)c_1(I_H+I_{MH})}{N_H}, \quad \lambda_{mh} = \frac{\alpha_1\beta_m I_V}{N_H} \quad \text{and} \quad \lambda_{mv} = \frac{\alpha_1\beta_v(I_M+I_{MH}+I_{MA})}{N_H}$$

For this model (3.0.1), it can be shown that the solutions are uniformly bounded in a proper subset $\Psi_{H1} = \Psi_{H2} \times \Psi_{V2}$, which is positively-invariant and attracting thus model 3.0.1 is mathematically well posed and its dynamics can be considered in Ψ_{H1}. Where:

$$\Psi_{H2} = \{(S_H, I_M, I_H, I_A, I_{MH}, I_{MA}) : N(t) \leq \tfrac{\Lambda_H}{d_n}\}$$

and

$$\Psi_{V2} = \{(S_V, I_V) : N_V \leq \tfrac{\Lambda_V}{d_v}\}$$

Scaling the sub-populations using the following set of new variables, $s_H = \frac{S_H}{N_H}, i_H = \frac{I_H}{N_H}$, $i_A = \frac{I_A}{N_H}, i_M = \frac{I_M}{N_H}, i_{HM} = \frac{I_{HM}}{N_H}, i_{AM} = \frac{I_{AM}}{N_H}$, yield the following model.

$$\begin{aligned}
\frac{ds_H(t)}{dt} &= \frac{\Lambda_H}{N_H} + r_m i_M(t) - \lambda_{ah} s_H(t) - \lambda_{mh} s_H(t) \qquad (3.0.2)\\
&\quad -s_H[\frac{\Lambda_H}{N_H} - d_n - (d_m A_{AM}(t) + d_a B_{AM}(t) + d_{ma} i_{AM}(t)]\\
\frac{di_M(t)}{dt} &= \lambda_{mh} s_H(t) - r_m i_M(t) - e_m^h \lambda_{ah} i_M(t)\\
&\quad -i_M[\frac{\Lambda_H}{N_H} - d_n - (d_m A_{AM}(t) + d_a B_{AM}(t) + d_{am} i_{AM}(t)]\\
\frac{di_H(t)}{dt} &= \lambda_{ah} s_H(t) + r_m i_{HM}(t) - (1-\alpha) p i_H(t) - e_h^m \lambda_{mh} i_H(t) + \alpha i_A(t)\\
&\quad -i_H[\frac{\Lambda_H}{N_H} - d_n - (d_m A_{AM}(t) + d_a B_{AM}(t) + d_{am} i_{AM}(t)]\\
\frac{di_A(t)}{dt} &= (1-\alpha) p i_H(t) + r_m i_{AM}(t) - e_a^m \lambda_{mh} i_A(t) - \alpha i_A(t)\\
&\quad -i_A[\frac{\Lambda_H}{N_H} - d_n - (d_m A_{AM}(t) + d_a B_{AM}(t) + d_{am} i_{AM}(t)]\\
\frac{di_{MH}(t)}{dt} &= e_h^m \lambda_{mh} i_H(t) + e_m^a \lambda_{ah} i_M(t) - r_m i_{HM}(t) + \alpha i_{AM}(t) - (1-\alpha)\theta_2 p i_{HM}(t)\\
&\quad -i_{HM}[\frac{\Lambda_H}{N_H} - d_n - (d_m A_{AM}(t) + d_a B_{AM}(t) + d_{am} i_{AM}(t)]\\
\frac{di_{AM}(t)}{dt} &= (1-\alpha)\theta_2 p i_{HM}(t) + e_a^m \lambda_m i_A(t) - r_m i_{AM}(t) - d_m i_{AM}(t) - \alpha i_{AM}(t)\\
&\quad -i_{MA}[\frac{\Lambda_H}{N_H} - d_n - (d_m A_{AM}(t) + d_a B_{AM}(t) + d_{am} i_{AM}(t)]\\
\frac{ds_V(t)}{dt} &= \frac{\Lambda_V}{N_V}(t) - \lambda_{mv} s_V(t) - s_V \frac{\Lambda_V}{N_V}(t)\\
\frac{di_V(t)}{dt} &= \lambda_{mv} s_V(t) - i_V \frac{\Lambda_V}{N_V}(t).
\end{aligned}$$

The feasible region Γ, (i.e where the model makes biological sense) is given by $\Gamma = \{s_H, i_M, i_H, i_A, i_{HM}, i_{AM}, s_V, i_V \in \mathbf{R}^8_+ : 0 \leq s_H + i_M + i_H + i_A + i_{MH} + i_{AM} \leq 1; 0 \leq s_V + i_V \leq 1\}$. It can be shown that the above region is positively invariant with respect to the system 3.0.2, where $\mathbf{R}^8_+$ denotes the non-negative cone of R^8 including its lower dimensional faces. The boundary and the interior of Γ is denoted by ∂Γ and Γ, respectively.

3.1 Disease-Free Equilibrium Point Of The Model

In the absence of infection by either or both diseases, the model 3.0.2, has a steady-state solution called the disease-free equilibrium (DFE) $E_0 = s_H, i_M, i_H, i_A, i_{MH}, i_{AM}, s_V, i_V = (1, 0, 0, 0, 0, 0, 1, 0)$. To study the stability of the disease-free equilibrium, the basic reproduction number R_{HM} is first obtained. Define F_i as the rate of appearance of new infections in the class or compartment i and $V_i = (V_i^- - V_i^+)$, where V_i^- is the rate of transfer of individuals out of compartment i, and V_i^+ is the rate of transfer of individuals into compartment i by all other means. Therefore: The Jacobian of F_i and V_i at the disease-free equilibrium denoted by F and V respectively is given by:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \alpha_1\beta_m \\ 0 & \beta_a(1-\delta)c_1 & 0 & \beta_a(1-\delta)c_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha_1\beta_v & 0 & 0 & \alpha_1\beta_v & \alpha_1\beta_v & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} h1 & 0 & 0 & 0 & 0 & 0 \\ 0 & h2 & -\alpha & -r_m & 0 & 0 \\ 0 & -(1-\alpha)p & h3 & 0 & -r_m & 0 \\ 0 & 0 & 0 & h4 & -\alpha & 0 \\ 0 & 0 & 0 & -(1-\alpha)\theta_2 p & h5 & 0 \\ 0 & 0 & 0 & 0 & 0 & d_v \end{pmatrix}$$

where $h_1 = (r_m + d_m + d_n)$, $h2 = (1 - \alpha)p + d_n$, $h3 = d_a + \alpha + d_n$, $h4 = r_m + d_m + (1 - \alpha)\theta_2 p + d_n$, $h5 = r_m + d_m + \alpha + d_a + d_n$. The basic reproduction number $R_0 = R_{HM}$ is by definition the maximum value of the spectral radius of the matrix FV^{-1} and is given by: $R_{HM} = \max\{R_M, R_H\}$. Where

$$R_M = \frac{\alpha_1\sqrt{\beta_m\beta_v}}{\sqrt{d_m d_v + d_n d_v + d_v r_m}} \qquad (3.1.1)$$

$$R_H = \frac{\beta_a(1-\delta)c_1 h3\{(\alpha-1)\alpha p\theta_2 + h5h4\}}{(1-\alpha)p\theta_2 D + E(h5-\alpha)h4} \qquad (3.1.2)$$

$$D = -(\alpha^2 d_n + \alpha d_a d_n + \alpha d_n + \alpha d_a p + \alpha d_n p) + \alpha^2 d_a p + \alpha^2 d_n p.$$
$$E = \alpha d_n + d_a d_n + d_n^2 + d_a p - \alpha d_a p + d_n p - \alpha d_n p.$$

Lemma 3.1. *The DFE of the HIV/AIDS-Malaria model is locally asymptotically stable (LAS) if $R_{HM} < 1$, and unstable otherwise.*

Lemma 3.1 follows from Theorem two by Van, P. and Watmough, J. (2002).

2.2. Parameter Values For The Hiv/Aids Malaria Model

Symbol	Parameter	Value (day^{-1})	Source
Λ_H	Recruitment rate of humans	4.38356×10^{4}	Kenya demographics profile (2014)
d_n	Natural death rate of humans	4.56630×10^{-5}	Kenya demographics profile (2014)
d_a	HIV/AIDS-induced death rate	1.09589×10^{-3}	WHO report (2014)
p	Progresion rate from HIV to AIDS (untreated)	2.73972×10^{-3}	Baryama, F. and Mugisha, T. (2007)
α	Proportion of the HIV/AIDS victims treated	1.64384×10^{-3}	Kenya NACC report (2014)
β_a	Transmission probability of HIV/AIDS	0.019	Baryama, F. and Mugisha, T. (2007)
c_1	Per capita number of sexual contacts	2.46575×10^{-2}	Kenya NACC report (2014)
δ	Effectiveness of counseling	Variable	
r_m	Proportion of malaria victims treated	1.86301×10^{-3}	WHO report (2013)
d_m	Death rate due to malaria	0.00714	WHO report (2013)
α_1	Mosquito biting rate	0.7	Lawi *et al* (2011)
β_m	Transmission probability of malaria in humans	0.8333	Lawi *et al* (2011)
β_v	Transmission probability of malaria in vectors	(0 - 1)	Chiyaka and Dube (2007)
θ_2	Increased Progresion rate from HIV to AIDS due to malaria	1.5	Estimated
Λ_V	Recruitment rate of vectors	6	Chiyaka and Dube (2007)
d_v	Death rate of mosquitoes	0.1429	Lawi *et al* (2011)

3.3 The Role Of Treatment And Counseling

The equation 3.1.1 represents the total number of secondary malaria infections in humans caused by one infected mosquito. Numerical simulation of the reproduction number (R_M) against malaria treatment (r_m) is depicted in figure 1using the set of parameters in table 3.2. This Figure shows that malaria treatment a lone, without strategies to reduce the mosquito biting rate α_1 may not eliminate malaria from the community therefore strategies for the reduction of malaria infections in humans should not only target malaria treatment but also the reduction of mosquito biting rate α_1 by encouraging the use of insecticide treated nets, vector elimination or reduction (spraying) and draining stagnant water (breeding grounds)

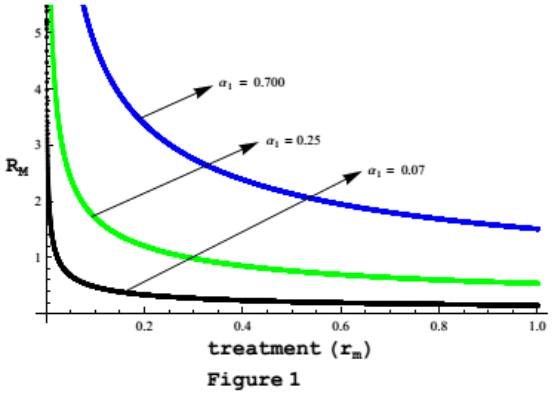

Figure 1

From equation 3.1.2, it is noted that ARV treatment and counseling for HIV/AIDS infectives have no effect on the spread of malaria. Assuming that $R_H > R_M$, implying that $R_H = R_{HM}$, then the graph of R_{HM} against ARV treatment with and without counseling is shown in figure 2.

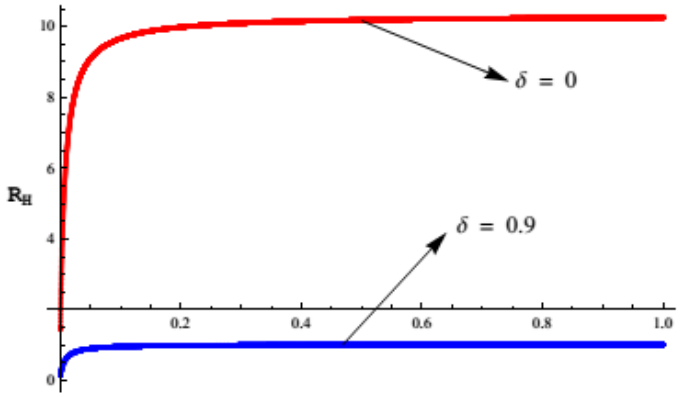

Figure 2

This figure shows that, an effective HIV/AIDS counseling strategy is very effective in controlling the spread of the HIV/AIDS- malaria co infections. The sensitivity indices of R_{HM} with respect to ARV treatment and counseling are given as 0.538433 and −9, respectively. This reveals that counseling (δ) is the most sensitive parameter in reducing/controlling the spread of HIV/AIDS and malaria co infections. The sensitivity index of (R_{HM}) with respect to malaria treatment yields 0 .071791, suggesting that malaria treatment could fuel the spread of the co infections, however figure 3 shows that the effects of malaria treatment is not significant in reducing the value of R_{HM}.

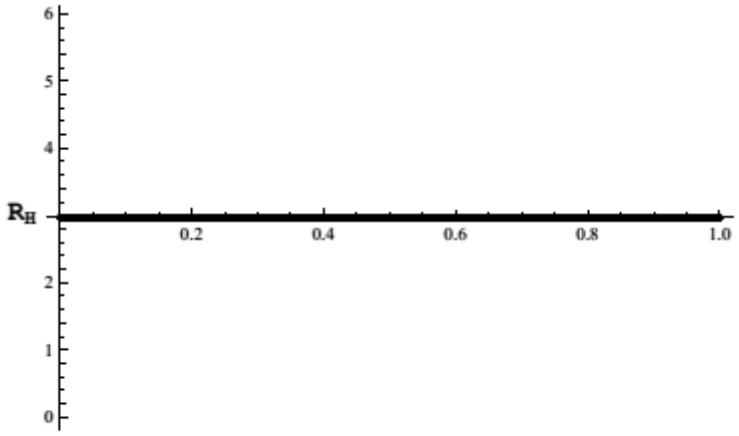

Figure 3

Figure 4 shows the HIV/AIDS incidence in the absence and presence of malaria. This figure shows that co infections of HIV/AIDS and malaria reduces the number of HIV/AIDS cases in the population. This could be due to the fact that malaria increases the rate of progression from HIV to AIDS leading to more HIV/AIDS deaths reducing the number of the HIV/AIDS cases.

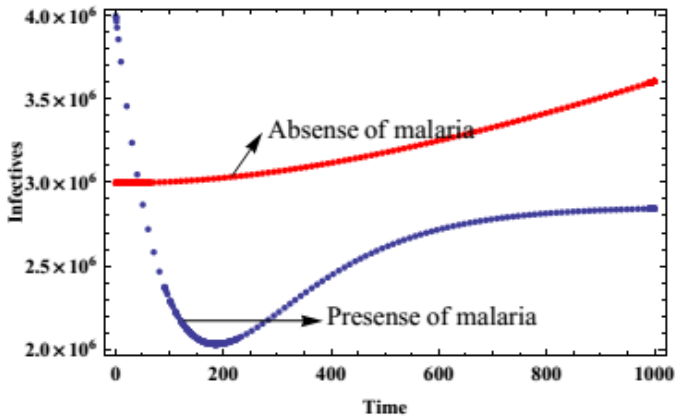

Figure 4

Figure 5 shows the malaria incidence in the absence and presence of HIV/AIDS and indicates that co infections of malaria and HIV/AIDS increases the malaria cases in the population due to the compromised immune system of HIV/AIDS victims as a result of the co infection.

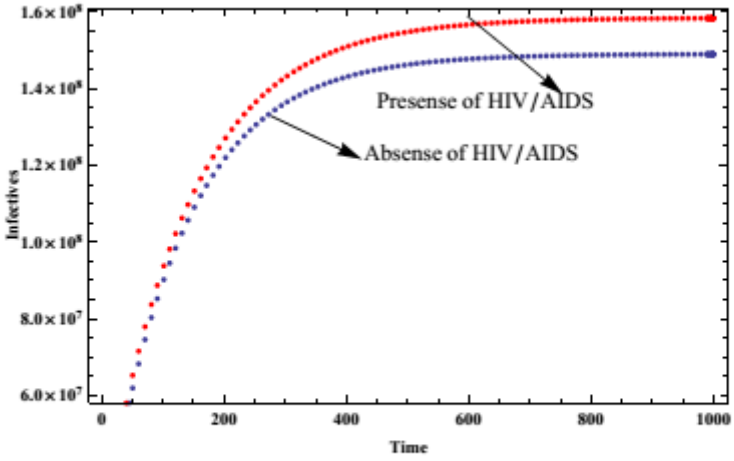

Figure 5

Biologically, lemma 3.1 implies that HIV/AIDS can be eliminated from the community (when $R_{HM} < 1$) if the initial sizes of the sub-populations of the model are in the basin of attraction of E_0. To ensure that elimination of the virus is independent of the initial sizes of the sub-populations, it is necessary to show that the DFE is globally asymptotically stable.

3.3.1 Global Stability Of Disease-Free Equilibrium (Dfe

The global asymptotic stability (GAS) of the disease-free state of the model is investigated using the theorem by Castillo-Chavez et al. (2002). The model is re written as follows:

$$\frac{dX}{dt} = H(X, Z) \qquad (3.3.1)$$

$$\frac{dZ}{dt} = G(X, Z), \quad G(X, 0) = 0 \qquad (3.3.2)$$

where the components of the column-vector $X \in R^m$ denote the uninfected population and the components of $Z \in R^n$ denote the infected population. $E_0 = (X^*, 0)$ denotes the disease-free equilibrium of this system. The fixed point $E_0 = (X^*, 0)$ is a globally asymptotically stable equilibrium for this system provided that $R_0 < 1$ (locally asymptotically stable) and the following two conditions satisfied:

(H1) For $\frac{dX}{dt} = H(X,0), X^*$ is globally asymptotically stable
(H2) $G(X,Z) = PZ - \widehat{G}(X,Z), \quad \widehat{G}(X,Z) \geq 0$ for $(X,Z) \in \Omega_H$,

where $P = D_Z G(X^*, 0)$ is an M-matrix (the off diagonal elements of P are non negative) and Ω_H is the region where the model makes biological sense. The disease-free equilibrium is now denoted as

$$E^0 = (X^*, 0), \quad X^* = (\tfrac{\Lambda_H}{d_n}, \tfrac{\Lambda_V}{d_v})$$

Theorem 3.2. *The fixed point $E^0 = (X^*, 0)$ is a globally asymptotically stable equilibrium of system 3.0.2 provided that $R_{HM} < 1$ and the assumptions H1 and H2 are satisfied.*

Proof. From the system 3.0.2

$$H(X,0) = \begin{pmatrix} \Lambda_H - d_n \\ \Lambda_V - d_v \end{pmatrix}$$

$$G(X,Z) = PZ - \widehat{G}(X,Z)$$

$$P = \begin{pmatrix} -h1 & 0 & 0 & 0 & 0 & \alpha_1\beta_m \\ 0 & -h2 & \alpha & \beta_a(1-\delta)c_1 + r_m & 0 & 0 \\ 0 & (1-\alpha)p & -h3 & 0 & r_m & 0 \\ 0 & 0 & 0 & -h4 & \alpha & 0 \\ 0 & 0 & 0 & (1-\alpha)\theta_2 p & -h5 & 0 \\ \alpha_1\beta_v & 0 & 0 & \alpha_1\beta_v & \alpha_1\beta_v & -d_v \end{pmatrix}$$

where: $h1 = r_m + d_n + d_m$, $h2 = \beta_a(1-\delta)c_1 - (1-\alpha)p + d_n$, $h3 = da + dn + \alpha$, $h4 = r_m + d_m + (1-\alpha)p\theta_2 - dn$, $h5 = rm + dm + \alpha + dn + da$

and

$$\widehat{G} = \begin{pmatrix} G_1(X,Z) \\ G_2(X,Z) \\ G_3(X,Z) \\ G_4(X,Z) \\ G_5(X,) \\ G_6(X,Z) \end{pmatrix} = \begin{pmatrix} \lambda_{mh}(1 - \frac{S_H}{N_H}) + e_m^h \lambda_{ah} I_M \\ \lambda_{ah}(1 - \frac{S_H}{N_H}) + e_h^m \lambda_{mh} I_H \\ e_a^m \lambda_{mh} I_A \\ -(e_h^m \lambda_{mh} I_H + e_m^h \lambda_{ah} I_M) \\ -(e_a^m \lambda_{mh} I_A) \\ \lambda_{mv}(1 - \frac{S_V}{N_V}) \end{pmatrix}$$

Notice that $\widehat{G_4}(X,Z) < 0, \widehat{G_5}(X,Z) < 0$ and so the conditions of **H1** and **H2** are not met so E^0 may not be globally asymptotically stable when $R_{HM} < 1$. □

This implies that there is the possibility of future disease outbreaks when the conditions favouring the outbreaks are prevailing.

3.4 Backward Bifurcation And Stability Of The Endemic Equilibrium

A bifurcation point is a point in parameter space where the number of equilibrium points, or their stability properties, or both, change. As noted earlier, an infectious disease does not invade a population of susceptibles when the basic reproduction number is less than unity. The epidemiological implication of backward bifurcation is that reducing the basic reproduction number to less than unity is not sufficient to control an epidemic. When the basic reproduction number is unity each infectious individual causes one new infection therefore, whether a disease invades with the basic reproduction number equal to unity will be determined by whether the basic reproduction number increases or decreases as the disease increases along the centre manifold.

When backward bifurcation occurs, the diseases-free equilibrium may not be globally asymptotically stable even if the basic reproduction number is less than unity and thus a stable endemic state co-exists with the diseases-free equilibrium. We employ the theorem by Castillo-Chavez and Song (2004), to investigate the possible occurrence of backward bifurcation. For purpose of convenience, we reproduce the theorem below.

Theorem 3.3. *Consider the following general system of ordinary differential equations with a parameter* ϕ.
$\frac{dx}{dt} = f(x,\phi)$, $f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n$ *and* $f \in C^2(\mathbb{R}^n \times \mathbb{R})$ *where 0 is an equilibrium point of the system (i.e)* $f(0,\phi) = 0$ *for all* ϕ).

and

1. $A = D_x f(0,0) = \frac{\partial f_i}{\partial x_j}(0,0)$ *is the linearization matrix of the system around the equilibrium point 0 with f evaluated at 0;*

2. *Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;*

3. *Matrix A has a right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.*

Let f_k *be the* k^{th} *component of f and* $a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$
$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_m^*}(0,0)$, *(let* $\beta_m^* = \phi$*) then the local dynamics of the system around the equilibrium point 0 is totally determined by the signs of a and b. Particularly,*

(i) $a > 0, b > 0$, *when* $\beta_m^* < 0$ *with* $|\beta_m^*| \ll 1, (0,0)$ *is locally asymptotically stable and there exists a positive unstable equilibrium; when* $0 < \beta_m^* \ll 1, (0,0)$ *is unstable and there exists a negative and locally asymptotically stable equilibrium.*

(ii) $a < 0, b < 0$, *when* $\beta_m^* < 0$ *with* $|\beta_m^*| \ll 1, (0,0)$ *is unstable; when* $0 < \beta_m^* \ll 1, (0,0)$ *is asymptotically stable and there exists a positive unstable equilibrium.*

(iii) $a > 0, b < 0$, *when* $\beta_m^* < 0$ *with* $|\beta_m^*| \ll 1, (0,0)$ *is unstable, and there exists a negative and locally asymptotically stable equilibrium; when* $0 < \beta_m^* \ll 1, (0,0)$ *is stable and there exists a positive unstable equilibrium.*

(iv) $a < 0, b > 0$, *when* $\beta_m^* < 0$ *changes from negative to positive, (0, 0) changes its stability from stable to unstable. Correspondingly a negative equilibrium becomes positive and locally asymptotically stable. Particularly, if* $a > 0$ *and* $b > 0$, *then a backward bifurcation occurs at* $\beta_m^* < 0 = 0$.

To apply this theorem we make the following change of variables. Let $S_H = x_1$, $I_M = x_2$, $I_H = x_3$, $I_A = x_4$, $I_{MH} = x_5$, $I_{MA} = x_6$, $S_V = x_7$, $I_V = x_8$ $N_H = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$ and $N_V = x_7 + x_8$. The model 3.0.2 is given by:

$$\frac{dx_1}{dt} = f_1 = \Lambda_H + r_m x_2 - \lambda_{ah} x_1 - \lambda_{mh} x_1(t) - d_n x_1(t) \qquad (3.4.1)$$

$$\frac{dx_2}{dt} = f_2 = \lambda_{mh} x_1 - r_m x_2 - e_m^h \lambda_{ah} x_2 - d_n x_2 - d_m x_2$$

$$\begin{aligned}
\frac{dx_3}{dt} &= f_3 = \lambda_{ah}x_1 + r_m x_5 - (1-\alpha)px_3 - e_h^m\lambda_{mh}x_3 - d_n x_3 + \alpha x_4\\
\frac{dx_4}{dt} &= f_4 = (1-\alpha)px_3 + r_m x_6 - e_a^m\lambda_{mh}x_4 - d_a x_4 - d_n x_4 - \alpha x_4\\
\frac{dx_5}{dt} &= f_5 = e_h^m\lambda_{mh}x_3 + e_m^h\lambda_{ah}x_2 - r_m x_5 + \alpha x_6\\
&\quad -d_m x_5 - (1-\alpha)\theta_2 px_5 - d_n x_5\\
\frac{dx_6}{dt} &= f_6 = (1-\alpha)\theta_2 px_5 + e_a^m\lambda_{mh}x_4 - r_m x_6 - d_m x_6 - \alpha x_6\\
&\quad -d_n x_6 - d_a x_6 - d_{am}x_6.\\
\frac{dx_7}{dt} &= f_7 = \Lambda_V - \lambda_{mv}x_7 - d_v x_7\\
\frac{dx_8}{dt} &= f_8 = \lambda_{mv}x_7 - d_v x_8.
\end{aligned}$$

Where $\lambda_{mh} = \frac{\alpha_1\beta_m x_8}{N_H}$ $\lambda_{ah} = \frac{\beta_a(1-\delta)c_1(x_3+x_5)}{N_H}$, $\lambda_{mv} = \frac{\alpha_1\beta_v(x_2+x_5+x_6)}{N_H}$

The Jacobian of 3.4.1 at the DFE E^0 is given by:

$$J(E^0) = \begin{pmatrix}
-d_n & r_m & -K6 & 0 & -K6 & 0 & 0 & -\alpha_1\beta_m\\
0 & -K1 & 0 & 0 & 0 & 0 & 0 & \alpha_1\beta_m\\
0 & 0 & -K2 & \alpha & K7 & 0 & 0 & 0\\
0 & 0 & (1-\alpha)p & -K3 & 0 & r_m & 0 & 0\\
0 & 0 & 0 & 0 & -r_m & \alpha & 0 & 0\\
0 & 0 & 0 & 0 & (1-\alpha)\theta_2 p & K4 & 0 & 0\\
0 & -\alpha_1\beta_v & 0 & 0 & -\alpha_1\beta_v & -\alpha_1\beta_v & -d_v & 0\\
0 & \alpha_1\beta_v & 0 & 0 & \alpha_1\beta_v & \alpha_1\beta_v & 0 & -d_v
\end{pmatrix}$$

where: $K_1 = r_m + d_n + d_m$, $K_2 = -\beta_a(1-\delta)c_1 + (1-\alpha)p + d_n$, $K_3 = d_a + d_n + \alpha$, $K_4 = r_m + d_m + (1-\alpha)\theta_2 p + d_n$, $K_5 = r_m + d_m + \alpha + d_n + d_a$, $K_6 = \beta_a(1-\delta)c_1$, $K_7 = \beta_a(1-\delta)c_1 + r_m$

To analyze the endemic dynamics of 3.4.1, the eigenvectors of its Jacobian are computed at $\beta_m = \beta^*_m$ where:

$$\beta^\star_m = \frac{d_m d_v + d_n d_v + d_v r_m}{\alpha_1^2\beta_v}, \text{ and } \beta_a(1-\delta)c_1 = \beta^\star_a(1-\delta^\star)c^\star_1 = \frac{(1-\alpha)p\theta_2 D + E(h5-\alpha)h4}{(\alpha-1)\alpha p\theta_2 + h5h4}$$

It is clear that 0 is a simple eigenvalue of $J(E^0)$. A right eigenvector associated with the 0 eigenvalue is denoted by $W = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8)^T$ and is given by: $w_1 = -\frac{d_m d_v + d_n d_v}{\alpha_1\beta_v d_n}$, $w_2 = \frac{d_v}{\alpha_1\beta_v}$ $w_3 = w_4 = w_5 = w_6 = 0$, $w_7 = -1$, $w_8 = 1$. The left eigenvector associated with the 0 eigenvalue is defined by $V = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8)^T$ and is given by:

$$v_1 = 0, \quad v_2 = \frac{\alpha_1\beta_v d_v}{d_m d_v + d_n d_v + d_v r_m},$$

$$v_3 = -\frac{K_3 v_4}{\alpha}, \quad v_4 = -\left(\frac{\alpha_1\beta_v + \alpha v_5 + K_4 v_6}{r_m}\right), \quad v_5 = \frac{-g_3}{g_5} + \frac{g_4 v_6}{g_5}, \quad v_6 = \frac{g_2}{g_1}, \quad v_7 = 0, \quad v_8 = 1.$$

Where $g_1 = \alpha(g_6+g_8)(-g_7 + g_8\theta_1 r_m) - K4(-g_6-g_8)(\alpha d_n K K_3 + g_9)$

$g_2 = \alpha(g_6+g_8)(g_3 - \alpha\alpha_1\beta_v d_n K K3) + \alpha_1\beta_v(g_6+g_8)(\alpha d_n K K3 + g_9)$

$g_3 = (\alpha_1\beta_v d_n K K3 - \alpha\alpha_1\beta_v d_n r_m), \quad g_4 = -(g_7 + g_8\theta_1 r_m), \quad g_5 = \alpha d_n K K3 + g_9$

$g_6 = d_n K2K3, \quad g_7 = d_n K K3K4, \quad g_8 = (-1+\alpha)\alpha d_n p, \quad g_9 = \alpha d_n r m^2.$

Computations of a and b

The associated non-zero partial derivatives of the system 3.4.1 at the DFE are given by:

$$\frac{\partial^2 f_8}{\partial x_1 \partial x_2} = -\alpha_1 \beta_v \frac{\Lambda_V}{d_v}\left(\frac{d_v}{\Lambda_H}\right)^2, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_8} = -\frac{\alpha_1 \beta_m d_n}{\Lambda_H}, \quad \frac{\partial^2 f_8}{\partial x_2 \partial x_1} = -\alpha_1 \beta_v \frac{\Lambda_V}{d_v}\left(\frac{d_n}{\Lambda_H}\right)^2$$
$$\frac{\partial^2 f_8}{\partial x_7 \partial x_2} = \alpha_1 \beta_v \frac{d_n}{\Lambda_H}, \quad \frac{\partial^2 f_2}{\partial x_8 \partial x_2} = -\alpha_1 \beta_m \frac{d_n}{\Lambda_H}, \quad \frac{\partial^2 f_2}{\partial \beta_m \partial x_8} = \alpha_1$$

$$a = -\frac{d_n}{\Lambda_H}\left(2w_1 w_2 \alpha_1 \beta_v \frac{\Lambda_V d_n}{d_v \Lambda_H} + 2v_2 w_2 \alpha_1 \beta_m + w_2 \beta_v\right)$$

$b = v_2\alpha_1 = \frac{\alpha_1^2 \beta_v d_v}{d_m d_v + d_n d_v + d_v r_m} > 0.$ Since $b > 0$, then theorem 3.4 follows.

Theorem 3.4. *The unique endemic equilibrium of the model 3.4.1 is locally asymptotically stable when $R_{HM} < 1$ and unstable when $R_{HM} > 1$. If $a > 0$, then the model , undergoes backward bifurcation at $R_0 = R_{HM} = 1$*

Since $w_1 < 0$, then $a > 0$ whenever $(2w_1 w_2 \beta_v \frac{\Lambda_V d_n}{d_v \Lambda_H}) > (2v_2 w_2 \beta_m + \frac{w_2 \beta_v}{\alpha_1})$, therefore the occurrence of backward bifurcation is favoured by a high mosquito biting rate α_1.

IV. Conclusion

In summery, treatment of malaria alone, may not eliminate malaria from the community therefore strategies for the reduction of malaria infections in humans should not only target malaria treatment but also the reduction of mosquito biting rate. Counseling is the most sensitive parameter in the spread of HIV/AIDS and malaria co infections, therefore effective counseling strategy is very useful in controlling the spread of the HIV/AIDS- malaria co infections. Finally the HIV/AIDS malaria model undergoes backward bifurcation which is favoured by the occurrence of high mosquito biting rate.

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