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Mathematical Analysis of Sensitive Parameters on the Dynamical Transmission of HIV-Malaria Co-infection

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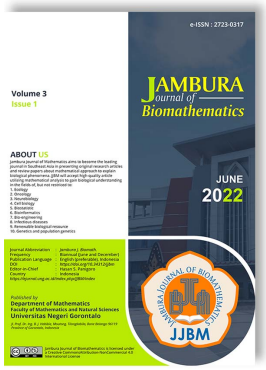

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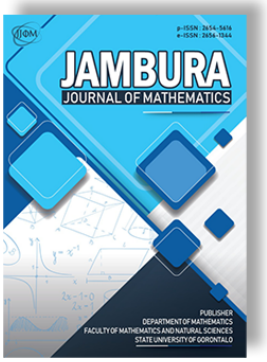

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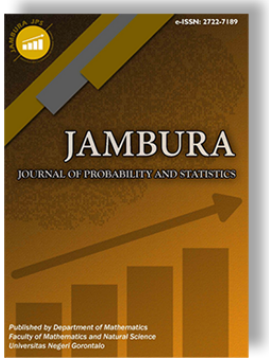

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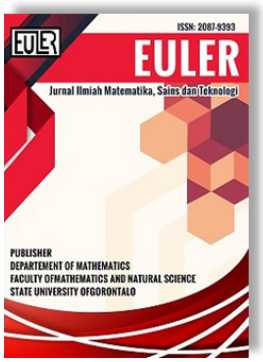

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Research Article

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Mathematical Analysis of Sensitive Parameters on the Dynamical Transmission of HIV-Malaria Co-infection

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ABSTRACT. *Malaria disease increases the mortality rate of HIV patients. In this work, a mathematical model incorporating an infected, undetected, and treated set of people was developed. The analysis showed that the model is well-posed, the disease-free equilibrium for the model was obtained, and the basic reproduction number of the HIV-malaria co-infection model was calculated. The 14 compartmental models were analyzed for stability, and it was established that the disease-free equilibrium of each model and their co-infections were locally and globally asymptotically stable whenever the basic reproduction number was less than unity or endemic otherwise. Based on the sensitivity analysis, the parameter that has the greatest impact is the contact rate; therefore, it is recommended for public health policies aimed at reducing the burden of these diseases in co-endemic regions.*

1. Introduction

The use of mathematical models enables us to address various physical phenomena, including crises in biology and epidemiology. Some of the applications found way back are in [1] and [2]. Till date, researchers continue the rigorous applications of the models, and some recent studies like [3–6] are examples. Co-infection with HIV and malaria is common, particularly in developing countries where malaria is already endemic. Malaria caused about 438,000 deaths in 2015, and the HIV virus claimed 11,310 lives [7]. The literature and development of mathematical epidemiology are well documented and can be found in [8]. Two of the prevailing infections in sub-Saharan Africa are malaria and HIV. Important results on the transmission dynamics of malaria have only been revealed in the last decade; for instance, see [9]. In their paper, a mathematical model is formulated using a system of differential equations to understand the co-dynamics of two diseases: HIV/AIDS and malaria. The entire human population (ages 16–45) is divided into six compartments, and the mosquito population into two. The model is analyzed, and steady-state conditions are derived. It is shown that the disease-free equilibrium is stable if the basic reproduction number, R_0, is less than unity. Sensitivity analysis and simulation results prove that malaria makes people move faster from HIV to AIDS and reduces their lifespan [10]. They proposed and investigated a deterministic model for the co-infection of HIV and malaria in a community.

The available literature reviewed did not consider the HIV infected undetected group of people and the treated group. Hence, a new fourteen 14-compartmental model that incorporated these classes was formulated to gain insight into the transmission dynamics of the spread of HIV-malaria co-infections. To effectively control and stop the spread of HIV and malaria, it is imperative to understand the dynamics of co-infection [11]. Researchers can use mathematical models to examine the impacts of various parameters on disease transmission and to forecast the consequences of interventions like vaccination or treatment plans. In the instance of HIV-malaria co-infection, identifying sensitive variables that significantly influence disease transmission might direct the creation of focused interventions to lower the burden of disease. As a result, this research on the mathematical analysis of delicate parameters on the dynamical transmission of HIV-malaria co-infection is very important for public health and has the ability to influence decisions about measures for disease control and prevention [12, 13]. To gain a better knowledge of the disease's transmission dynamics and control, many models have been developed and studied using various methodologies. These studies include the following [14–25].

The model of HIV-malaria co-infection was analyzed for the positivity and boundedness of solutions, to determine if the model was well-posed. The disease-free equilibria for the models were obtained. Also, the basic reproduction numbers for the models were computed using the next-generation matrix method. Moreover, the stability of the disease-free equilibria was determined, and bifurcation analysis of the sub-models was carried out using center manifold theory. Sensitivity analysis of the basic reproduction numbers of HIV, malaria, and the full model was examined, and optimal control analysis was carried out to identify the best strategies for the control of the disease. Nu-

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merical simulation was carried out using Maple 17 software.

2. Methods

2.1. Model description and Formulation

In order to model the dynamics, the total homogeneous mixing population at time t, denoted by $N(t)$, is divided into fourteen compartments: susceptible $(S(t))$ individuals, exposed $(E_H(t))$ individuals, HIV-infected undetected $(I_U(t))$ individuals, HIV-infected detected $(I_D(t))$ individuals, treated $(T_H(t))$ individuals, recovered $(R_M(t))$ individuals, latently HIV and malaria $(L_H(t))$ individuals, active HIV and malaria $(A_{HM}(t))$ So that

$$\begin{aligned} N(t) = S_H(t) + L_H(t) + I_U(t) + I_D(t) + T_H(t) \\ + E_M(t) + I_M(t) + T_M(t) + R_M(t) \\ + E_{HM}(t) + A_{HM}(t). \end{aligned} \tag{1}$$

The total vector (mosquito) population at time t, denoted by $N_V(t)$ is subdivided into susceptible mosquitoes $(S_V(t))$, exposed mosquitoes $(E_V(t))$, and infected mosquitoes $(I_V(t))$ so that

$$N_V(t) = S_V(t) + E_V(t) + I_V(t). \tag{2}$$

The susceptible humans are recruited into the population at the constant rate π_H. Susceptible individuals aquire HIV infection following effective contact with HIV-infected individuals (at a rate λ_H) and acquire infection with malaria following effective with infected mosquitoes (at a rate λ_M) and also aquire HIV-malaria co-infection following effective contact with HIV-infected individuals and infected mosquitoes (at a rate λ_{HM}). The population increases by recovered individuals who loss immunity (at a rate ϕ_1) and the natural death occurs in all human sub-population (at a rate μ) decreases the population. The force of infection associated with HIV-infection, denoted by λ_H is given by;

$$\lambda_H = \frac{\beta_H(L_H + \eta_U I_U + \eta_D I_D + T_H)}{N_H} \tag{3}$$

In eq. (3) above β_H represents the effective contact rate (contact sufficient to result in HIV infection), η_U is a modification parameter comparing the individual transmissibility of undetected infected individuals in relationship to latently infected. Also, η_D is a modification parameter comparing transmissibility of infected detected.

The rate of change of susceptible population is given by

$$\begin{aligned} \frac{dS_H}{dt} = \pi_H \lambda_H S_H - \lambda_M S_H - \mu S_H \\ + \varphi_1 R_M - \lambda_{HM} S_H. \end{aligned} \tag{4}$$

A fraction ε_1 of the newly infected individuals are assumed to show no disease symptoms initially. These individuals (known as "slow progressor") are moved to latently HIV class (L_H). The remaining fraction $(1-\varepsilon)$ move to infected undetected class (fast progressor) I_U. The population of latently infected class is further increased by the individuals who are successfully treated (at the rate ϕ_2) and by fraction of individuals who are treated for active HIV-malaria (at a rate $(1-\ell)\phi_3$), since malaria can only be cured. The population decreases by progression to HIV-detected class (at rate κ_H) and natural death (at the rate μ).

$$\begin{aligned} \frac{dL_H}{dt} = \varepsilon_1 \lambda_H S_H - (\kappa_H + \mu) L_H + \varphi_2 T_H \\ + (1-\ell)\phi_3 A_{HM}. \end{aligned} \tag{5}$$

The population of undetected infected individuals is increased by the fraction of the newly infected individuals low immunity (at the rate $(1-\varepsilon_1)\lambda_H$) and those that develop symptoms by latently infected individual at the rate $(1-\omega_1)\kappa_H$ where ω_1 is the fraction of latently infected individuals who are not detected, $\omega_1\kappa_H$. Furthermore, it decreases by the detection of the infection (at the rate γ_{UH}), natural death (at the μ) and disease induced death (at a rate δ_{UH}).

$$\begin{aligned} \frac{dI_U}{dt} = (1-\varepsilon_1)\lambda_H S_H + \omega_1 \kappa_H L_H \\ - (\gamma_{UH} + \mu + \delta_{UH}) I_U. \end{aligned} \tag{6}$$

The population of detected infected individuals is increased by a fraction latently infected individuals who are detected upon showing symptoms (at the rate $(1-\omega_1)\kappa_H$) also, the population increases due to HIV exposed malaria individuals that are treated (at the rate τ_1), and by the detection rate of undetected individuals (at the rate γ_{UH}). The population is decreased by treatment (at the rate τ_1), natural death(at the rate μ) and disease induced death (at a rate δ_{DH}). Hence;

$$\begin{aligned} \frac{dI_D}{dt} = (1-\omega_1)\kappa_H L_H - (\tau_1 + \mu + \delta_{dH}) I_D \\ + \gamma_{UH} I_U + \tau_4 E_{HT}. \end{aligned} \tag{7}$$

The population of treated HIV individuals is increased by those that have recovered treatment from HIV detected infected individual at the rate (τ_1) this population reduces by fraction of treated individual that moved back to latently HIV individuals at the rate (ϕ_2) since treatment does not completely clears the bacteria and finally reduced by natural death rate (μ). Hence,

$$\frac{dT_H}{dt} = \tau_1 I_D - (\varphi_2 + \mu) T_H \tag{8}$$

the population of latent malaria and HIV is increased by infection, which can acquired following effective contact with infectious individuals in the latent malaria and HIV (L_{TH}), Active induced HIV $(\eta_U A_{HM})$ or recovered malaria induced HIV

$$\lambda_{HM} = \frac{\beta_{HM}(E_{HM} + \eta_{AHM} A_{HM})}{N_{HM}} \tag{9}$$

Where β_{HM} represents the effective contact rate.

The population of HIV exposed malaria is generated by fraction ε_3 of the newly infected individuals with low immunity who moved to HIV exposed malaria class. The remaining fraction are moved to Active HIV-malaria class. The population decreases due to progression to active HIV-malaria (at the rate κ_{HM}) treatment of the population (at the rate τ_4), natural death (at the rate μ) and death due to the disease (at the rate δ_{EHM}).

$$\begin{aligned} \frac{dE_{HM}}{dt} = \varepsilon_3 \lambda_{HM} S_H - (K_{HM} + \mu + \delta_{EHM}) E_{HM} \\ - \tau_4 E_{HM}, \end{aligned} \tag{10}$$

the population of active HIV-malaria class contains the remaining individuals with low immunity (at the rate $1-\varepsilon_3$) and those that progresses from HIV exposed malaria class (at the rate κ_{HM}). The decreases by those that are successfully treated (at the rate ϕ_3), natural death (at the rate μ) and death due to disease (at the rate δ_{AHM}), hence

$$\begin{aligned}\frac{dA_{HM}}{dt} &= (1-\varepsilon_3)\lambda_{HM}S_H + K_{HM}E_{HM} \\ &\quad -(\varphi_3+\delta_{AHM}+\mu)A_{HM},\end{aligned} \tag{11}$$

the population of exposed HIV and malaria is increased by infection, which can be acquired following effective contact with infectious individuals in expose HIV and malaria (E_{HM}), or active HIV induced malaria ($\eta_U A_{HM}$) categories at a rate λ given by

$$\lambda_{HM}=\frac{\beta_{HM}(L_{HT}+\eta_U A_{HM})}{N_{HM}}, \tag{12}$$

where β_{HM} represents the effective contact rate.The population reduced by progression firm exposed stage to active stage at the rate (κ_{HM}) and by natural death rate.

A fraction ε_2 of new infected individuals with low immunity move to exposed class (E_M) and the remaining fraction $(1-\varepsilon_2)$ move to the infected class (I_M). The exposed population decreases when individuals become infected (at the rate $\rho\phi_3$). The exposed class decreases by progression to infected individuals (at rate κ_M), them who are treated at the rate τ_2), natural death (at the rate μ). Hence, we have,

$$\frac{dE_M}{dt} = \varepsilon_3\lambda_M S_H - (K_M+\mu)E_M - \tau_4 E_M + \ell\phi_3 A_{HM}. \tag{13}$$

The population of individual infected with malaria is generated by a fraction of the new infected individuals with low immunity (at the rate $1-\varepsilon_2$) and progression to infected individual from the exposed class. The population decreases by treatment of the infected individuals (at the rate τ_3), those who are successfully treated recovered (at the rate r), natural death (at the rate μ) and disease induced death (at the rate δ_{IM}). Therefore,

$$\begin{aligned}\frac{dI_M}{dt} &= (1-\varepsilon_2)\lambda_M S_H + K_M E_M \\ &\quad -(\tau_3+\delta I_M+\mu)I_M\end{aligned} \tag{14}$$

The population of treated class increases by treatment of detected individuals (at the rate τ_1), since, HIV has no cure, treated individuals will move to latently infected class (at the rate ϕ_2). Furthermore, the population also decreases by natural death (at the rate μ).

$$\frac{dT_M}{dt}=\tau_3-(r+\mu)T_M. \tag{15}$$

The recovered population is generated by treatment (at the rate τ_2 and τ_3), of the exposed and infected class respectively and those who are successfully treated and recovered (at the rate μ) and those that loss immunity (at the rate ϕ_1) hence

$$\frac{dR}{dt}=\tau_2 E_M + rT_M - (\varphi_1+\mu)R_M \tag{16}$$

Susceptible mosquitoes (S_V) are generated at a constant rate (recruitment π_r) and acquire malaria infection following effective contacts with human infected with malaria at a rate λ_V, where the force of infection λ_V is given

$$\lambda_V=\frac{\beta_V b(I_M+\eta_{HM}E_{HM}+\eta_{AHM}A_{HM})}{N_H}, \tag{17}$$

where η_{HM} and η_{AHM} are the modification parameters number of human bites one mosquito has per unit time, β_V is the transmission probability from human to mosquito. Newly infected mosquitoes move to exposed class and they are assumed to suffer death (at the rate μ_V). Hence,

$$\frac{ds_V}{dt}=\pi_V-\lambda_V S_V-\mu_V S_V \tag{18}$$

The expose mosquitoes consist of newly infected mosquitoes and their population diminishes by progression into infected class (at the rate σ_V) and death of the mosquitoes (at the rate μ_V) therefore.

$$\frac{dE_V}{dt}=\lambda_V S_V-(\sigma_V+\mu_V)E_V \tag{19}$$

The infected mosquitoes have those that progress from exposed class and diminish by the death of the mosquitoes (at the rate μ_V), hence

$$\frac{dI_V}{dt}=\sigma_V E_V-\mu_V I_V \tag{20}$$

In summary, the above formulations and assumptions together give the following system of differential equations. By following [10], we designed a new deterministic compartmental model as follows.

$$\begin{aligned}
\frac{dS_H}{dt} &= \pi_H-\lambda_H S_H-\lambda_M S_H-\mu S_H \\
&\quad +\phi_1 R_M-\lambda_{HM}S_H, \\
\frac{dL_H}{dt} &= \varepsilon_1\lambda_H S_H-(K_H+\mu)L_H+\phi_2 T_H \\
&\quad +(1-\ell)\phi_3 A_{HM}, \\
\frac{dI_U}{dt} &= (1-\varepsilon_1)\lambda_H S_H+\omega_1 K_H L_H \\
&\quad -(\gamma_{UH}+\mu+\delta_{UH})I_U, \\
\frac{dI_D}{dt} &= (1-\omega_1)K_H L_H-(\tau_1+\mu+\delta_{dH})I_D \\
&\quad +\gamma_{UH}I_U+\tau_4 E_{HT}, \\
\frac{dT_H}{dt} &= \tau_1 I_D-(\phi_2+\mu)T_H, \\
\frac{dE_M}{dt} &= \varepsilon_2\lambda_M S_H-(K_M+\mu)E_M \\
&\quad -\tau_4 E_M+\ell\phi_3 A_{HM}, \\
\frac{dI_M}{dt} &= (1-\varepsilon_2)\lambda_M S_H+K_M E_M \\
&\quad -(\tau_3+\delta I_M+\mu)I_M, \\
\frac{dT_M}{dt} &= \tau_3 I_M-(r+\mu)T_M, \\
\frac{dR_M}{dt} &= \tau_2 E_M+rT_M-(\phi_1+\mu)R_M, \\
\frac{dE_{HM}}{dt} &= \varepsilon_3\lambda_{HM}S_H-(K_{HM}+\mu+\delta_{EHM})E_{HM} \\
&\quad -\tau_4 E_{HM},
\end{aligned} \tag{21}$$

$$\begin{aligned}
\frac{dA_{HM}}{dt} &= (1-\varepsilon_3)\lambda_{HM}S_H + K_{HM}E_{HM} \\
&\quad -(\phi_3+\delta_{AHM}+\mu)A_{HM}, \\
\frac{dS_V}{dt} &= \pi_V - \lambda_V S_V - \mu_V S_V, \\
\frac{dE_V}{dt} &= \lambda_V S_V - (\sigma_V+\mu_V)E_V, \\
\frac{dI_V}{dt} &= \sigma_V E_V - \mu_V I_V,
\end{aligned}$$

where

$$\begin{aligned}
\lambda_E &= \beta_H\left(\frac{L_H+\eta_D I_D+\eta_U I_U+\eta_T T_H}{N_H}\right), \\
\lambda_{HM} &= \beta_{HM}\frac{(E_{HM}+\eta_{EM}A_{HM})}{N_H}, \\
\lambda_M &= \frac{\beta_M b I_V}{N_V}, \\
\lambda_V &= \frac{\beta_V b(I_M+\eta_{HM}E_{HM}+\eta_{AHM}A_{HM})}{N_H}.
\end{aligned}$$

2.2. Boundedness Solutions of the model

For the system (21) to be epidemiological meaningful, it is important to prove that all solutions with non-negative initial data will remain non-negative i.e. $t \geq 0$.

Theorem 1. *If $S_H(0)$, $L_H(0)$, $I_U(0)$, $I_D(0)$, $T_H(0)$, $E_M(0)$, $I_M(0)$, $T_M(0)$, $R_M(0)$, $E_{HM}(0)$, $I_{HM}(0)$, $S_V(0)$, $E_V(0)$, and $I_V(0)$ be non-negative, then the solutions S_H, L_H, I_U, I_D, T_H, E_M, I_M, T_M, R_M, E_{HM}, A_{HM}, S_V, E_V, and I_V are non-negative for all $t>0$.*

Proof. Consider the biologically- feasible region $\Omega = \Omega_H \times \Omega_V \subset \mathbb{R}^{14}_+$ with

$$\begin{aligned}
\Omega_H &= \{(S_H, L_H, I_U, I_D, T_H, E_M, I_M, T_M, \\
&\qquad E_{HM}, A_{HM}, R_M) \in \mathbb{R}^{11}_+ : N_H \leq \frac{\pi_H}{\mu}\}, \text{ and} \\
\Omega_V &= \left\{(S_V, E_V, I_V) \in \mathbb{R}^3_+ : N_V \leq \frac{\pi_V}{\mu_V}\right\},
\end{aligned}$$

is positively invariant. From this theorem, this can be concluded that it is sufficient to consider the dynamics of (21) in Ω. In this region, the model can be considered as being epidemiological well-posed [26]. □

The total human population N_H is calculated as:

$$\begin{aligned}
N_H &= S_H + L_E + I_U + I_D + T_H + E_M + I_M \\
&\quad + T_M + E_{HM} + A_{HM} + R_M
\end{aligned}$$

Therefore upon simplifications obtain the following

$$\begin{aligned}
\frac{dN_H}{dt} &= \pi_H - \mu\,(S_H + L_H + I_U + I_D + T_H \\
&\quad + E_M + I_M + T_M + E_{HM} + A_{HM} + R_M) \\
&\quad - (\delta_{UH}I_U + \delta_{DH}I_D + \delta_T T_H + \delta_{IM}I_M \\
&\quad + \delta_{HM}E_{HM} + \delta_{AHM}I_{HM})\,.
\end{aligned} \tag{22}$$

If there is no death from HIV and malaria infections, eq. (22) become

$$\frac{dN_H}{dt} \leq \pi_H - \mu N_H. \tag{23}$$

After evaluating eq. (23) as time approaches infinity, obtain

$$N_H(t) \leq N_H(0)e^{-\mu t} + \frac{\pi_H}{\mu},\left(1-e^{-\mu t}\right) \tag{24}$$

where $N_H(0)$ represents the value of total population of human evaluated at the initial values of the respective variables.

Similarly, the rate of change of the total population of vectors (mosquitoes) N_V is calculated as:

$$N_V = S_V + E_V + I_V$$

Thus obtain

$$\frac{dN_V}{dt} = \pi_V - \mu_V\left(S_V + E_V + I_V\right). \tag{25}$$

Then,

$$\frac{dN_V}{dt} \leq \pi_V - \mu_V N_V. \tag{26}$$

After, solving eq. (25) and evaluating it as time tends to infinity, obtain

$$N_V(t) \leq N_V(0)e^{-\mu_V t} + \frac{\pi_V}{\mu_V}\left(1-e^{-\mu_V t}\right), \tag{27}$$

where $N_V(0)$ represents the value of total population of the vectors (mosquitoes) evaluated at the initial values of the respective variables. Thus as $t \to \infty$, equations (27) become $\lim_{t\to\infty} N_H(t) \leq \frac{\pi_H}{\mu}$ and $\lim_{t\to\infty} N_V(t) \leq \frac{\pi_V}{\mu_V}$ if $N_H(0) \leq \frac{\pi_H}{\mu}$ and $N_V(0) \leq \frac{\pi_V}{\mu_V}$. Therefore, all the solution set of (21) is bounded in Ω.

2.3. Equilibrium analysis of HIV-Malaria co-infection model

The disease free equilibrium of eq. (21) is obtained by equating all equations of the model to zero and then set $L_H = I_U = I_D = T_H = E_M = I_M = T_M = R_M = E_{HM} = A_{HM} = E_V = I_V = 0$. Then obtain: $E_0 = \left(\frac{\pi_H}{\mu}, 0,0,0,0,0,0,0,0,0,0, \frac{\pi_V}{\mu_V}, 0, 0\right)$.

2.4. The basic reproductive number of model of HIV-Malaria co-infection

Following [27] principle of next generation matrix, from the above model equation, the non-negative matrix F (new infection terms) and the non-singular matrix V (i.e other transferring terms) can be partition as follow

$$F = \begin{bmatrix} F_1 & F_2 \\ F_3 & F_4 \end{bmatrix}, \text{ and } V = \begin{bmatrix} V_1 & V_2 \\ V_3 & V_4 \end{bmatrix}. \tag{28}$$

where F_1, F_2, F_3, F_4 and V_1, V_2, V_3, V_4 are 6×6 matrices. Therefore, from the model equation the non-negative matrices F_1 to F_4 (new infection term rate) are as follows.

$$F_1 = \begin{bmatrix} 0 & F^1_{12} & F^1_{13} & F^1_{14} & F^1_{15} & 0 \\ 0 & F^1_{22} & F^1_{23} & F^1_{24} & F^1_{25} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

Table 1. Definitions Of Parameters And Variables Used In The Model Formulation

Parameters and variables	Definition
S_h	Susceptible individuals
L_H	HIV Latently infected individuals
I_U	Undetected HIV individuals
I_D	Detected HIV individual
T_H	Treated HIV individuals
T_M	Treated malaria individuals
E_M	Malaria exposed individuals
I_M	Malaria infected individuals
R_M	Malaria recovered individuals
E_{HM}	HIV exposed malaria individuals
A_{HM}	Active HIV-malaria individuals
S_V	Susceptible vectors (mosquitoes)
E_V	Exposed vectors (mosquitoes)
I_V	Infected vectors (mosquitoes)
$\pi_h,\ \pi_V$	Recruitment rate of human and vectors respectively
$\lambda_H,\ \lambda_{HM}$	Forces of infection in HIV and HIV- malaria individuals
μ	Human natural death rate
μ_V	Death rate of vectors (mosquitoes)
$\tau_1, \tau_2, \tau_3, \tau_4$	Treatment rate for malaria exposed, infected, HIV-detected and HIV exposed malaria individuals
$\varepsilon_1, \varepsilon_2, \varepsilon_3$	Fraction of individuals with low immunity, infected with HIV, malaria and HIV-malaria co-infection
γ_{UH}	Detection rate for undetected HIV
$\delta_{EM},\ \delta_{IM}$	Malaria induced death rate for classes E_M and I_M
$\delta_{UH},\ \delta_{dH}, \delta_j,\ \delta_{EHM}, \delta_{AHM}$	HIV induced death rate for classes H_U, H_D, J, E_{HM} and A_{HM} respectively
$\kappa_H, \kappa_M,\ \kappa_{HM}$	Progression rate for HIV, malaria and HIV-malaria
$\sigma_1,\ \sigma_2$	Isolation rate for classes L_H and H_D respectively
β_H, β_{HM}	Effective contact rate for HIV and HIV-malaria respectively
β_M, β_V	Transmission probability from mosquito to human and human to mosquito respectively
ω_1	Fraction of latently infected class that moves to HIV undetected class
σ_V	Progression rate of vectors (mosquitoes)
ρ	Fraction of active HIV-malaria that is treated which moves to malaria exposed class
ϕ_2	Progression rate HIV treated class to latent class
ϕ_3	Progression rate active HIV-malaria individual after treatment
R	Recovery rate of malaria
A	Number of mosquito bites per unit time
B	Number of human bitten by mosquito per unit time
λ_M, λ_V	Force of infection from mosquito to human and from human to mosquito respectively
ϕ	Rate of loss of immunity

$$F_2 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & F_{66}^2 \end{bmatrix},$$

$$F_3 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \text{ and}$$

$$F_4 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & F_{16}^4 \\ 0 & F_{22}^4 & F_{23}^4 & 0 & 0 & 0 \\ 0 & F_{32}^4 & F_{33}^4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

where

$$F_{12}^1 = \varepsilon_1\beta_H, \qquad F_{13}^1 = \varepsilon_1\beta_H\eta_U,$$
$$F_{14}^1 = \varepsilon_1\beta_H\eta_D, \qquad F_{15}^1 = \varepsilon_1\beta_H\eta_T,$$
$$F_{22}^1 = (1-\varepsilon_1)\beta_E, \qquad F_{23}^1 = (1-\varepsilon_1)\beta_H\eta_U,$$
$$F_{24}^1 = (1-\varepsilon_1)\beta_H\eta_D, \qquad F_{25}^1 = (1-\varepsilon_1)\beta_H\eta_T,$$
$$F_{66}^2 = \tfrac{\varepsilon_2\beta_M b\pi_H\mu_V}{\mu\pi_V}, \qquad F_{16}^4 = \tfrac{(1-\varepsilon_2)\beta_M b\pi_H\mu_V}{\mu\pi_V},$$
$$F_{22}^4 = \varepsilon_3\beta_{HM}, \qquad F_{23}^4 = \varepsilon_3\beta_{HM}\eta_{HM},$$
$$F_{32}^4 = (1-\varepsilon_3)\beta_{HM}, \qquad F_{33}^4 = (1-\varepsilon_3)\beta_{HM}\eta_{HM}.$$

Other transferring terms V_1 to V_4 are given as follows.

$$V_1 = \begin{bmatrix} V_{11}^1 & 0 & 0 & -\phi_2 & -V_{15}^1 & 0 \\ -\omega_1\kappa_H & V_{22}^1 & 0 & 0 & 0 & 0 \\ -V_{31}^1 & -\gamma_{UH} & V_{33}^1 & 0 & 0 & 0 \\ 0 & 0 & -\tau_1 & V_{44}^1 & 0 & 0 \\ -\sigma_1 & 0 & -\sigma_2 & 0 & V_{55}^1 & 0 \\ 0 & 0 & 0 & 0 & 0 & V_{66}^1 \end{bmatrix},$$

$$V_2 = \begin{bmatrix} 0 & 0 & -V_{13}^2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\tau_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\rho\phi_3 & 0 & 0 & 0 \end{bmatrix},$$

$$V_3 = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & -\kappa_M \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\tau_2 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \text{ and}$$

$$V_4 = \begin{bmatrix} V_{11}^4 & 0 & 0 & 0 & 0 & 0 \\ 0 & V_{22}^4 & 0 & 0 & 0 & 0 \\ 0 & -\kappa_{HM} & V_{33}^4 & 0 & 0 & 0 \\ -V_{41}^4 & 0 & 0 & V_{44}^4 & 0 & 0 \\ 0 & 0 & 0 & 0 & V_{55}^4 & 0 \\ 0 & 0 & 0 & 0 & -\sigma_V & \mu_V \end{bmatrix},$$

with

$$\begin{aligned} V_{11}^1 &= \kappa_H + \mu + \sigma_1, & V_{15}^1 &= (1-\alpha)\theta, \\ V_{13}^2 &= (1-\rho)\phi_3, & V_{22}^1 &= \gamma_{UH} + \mu + \delta_{UH}, \\ V_{31}^1 &= (1-\omega_1)\kappa_H, & V_{33}^1 &= \tau_1 + \mu + \delta_{DH} + \sigma_2, \\ V_{44}^1 &= \phi_2 + \mu, & V_{55}^1 &= \mu + \theta, \\ V_{66}^1 &= \kappa_M + \mu + \tau_2, & V_{11}^4 &= \tau_3 + r + \delta_{IM} + \mu, \\ V_{22}^4 &= \kappa_{HM} + \mu & V_{33}^4 &= \phi_3 + \delta_{AHM} + \mu, \\ &\quad + \delta_{AHM} + \tau_4, & V_{41}^4 &= \tau_3 + r, \\ V_{44}^4 &= \phi_1 + \mu, & V_{55}^4 &= \sigma_V + \mu_V. \end{aligned}$$

From the above matrix, V_2 and V_3 are singular matrices. Then $(F_1V_1^{-1})$ and $(F_4V_4^{-1})$ will be considered for the calculation of the reproduction number. Therefore, the reproduction number R_1 of the matrix F_1V_1 is

$$R_1 = \frac{\beta_H \begin{pmatrix} \varepsilon_1 a_1 a_7 a_8 \eta_D \gamma_{UH} + \varepsilon_1 a_1 a_7 a_8 \eta_T \gamma_{UH} \tau_1 \\ -\varepsilon_1 a_2 a_7 \eta_D \gamma_{UH} \sigma_1 - \varepsilon_1 a_2 \eta_T \gamma_{UH} \sigma_1 \tau_1 \\ +\varepsilon_1 \eta_T \gamma_{UH} \phi_2 \sigma_1 \tau_1 - a_4 a_5 a_7 a_8 \varepsilon_1 \eta_D \\ -a_4 a_5 a_7 \varepsilon_1 \eta_T \sigma_2 - a_4 a_5 a_8 \varepsilon_1 \eta_T \tau_1 \\ -a_4 a_6 a_7 \varepsilon_1 \eta_T \sigma_1 - a_1 a_7 a_8 \eta_D \gamma_{UH} \\ -a_1 a_7 \eta_T + \gamma_{UH} \sigma_2 - a_1 a_8 \eta_T \gamma_{UH} \tau_1 \\ +a_2 a_7 \eta_D \gamma_{UH} \sigma_1 + a_2 \eta_T \gamma_{UH} \sigma_1 \tau_1 \\ -\eta_J \gamma_{UH} \phi_2 \sigma_1 \tau_1 + \varepsilon_1 a_1 a_6 a_7 a_8 \\ -\varepsilon_1 a_2 + a_5 a_7 \sigma_2 - \varepsilon_1 a_2 a_6 a_7 \sigma_1 - \varepsilon_1 a_5 a_8 \phi_2 \tau_1 \\ -a_1 a_6 a_7 a_8 + a_2 a_5 a_7 \sigma_2 + a_2 a_5 a_7 \sigma_1 + a_5 a_8 \phi_2 \tau_1 \\ -\varepsilon_1 \omega_1 + \kappa_E a_6 a_7 a_8 - \varepsilon_1 a_7 a_8 \eta_D \gamma_{UH} \kappa_H \omega_1 \\ -\varepsilon_1 a_7 \eta_T \gamma_{UH} \kappa_H \omega_1 \sigma_2 - \varepsilon_1 a_8 \eta_T \gamma_{UH} \kappa_H \omega_1 \tau_1 \end{pmatrix}}{\begin{pmatrix} a_2 a_7 \gamma_{UH} \kappa_H \omega_1 \sigma_2 + a_8 \gamma_{UH} \kappa_H \omega_1 \phi_2 \tau_1 - a_1 a_4 a_6 a_7 a_8 \\ +a_2 a_4 a_5 a_7 \sigma_2 + a_2 a_4 a_6 a_7 \sigma_1 + a_4 a_5 a_8 \phi_2 \tau_1 \end{pmatrix}}$$

The associated reproduction number R_4 for $F_4V_4^{-1}$ given by $\rho(F_4V_4^{-1})$, where is the spectral radius of the dominant eigenvalue of the next generation matrix $(F_4V_4^{-1})$ is

$$R_4 = \frac{\beta_{HM}\left((\phi_3 + \delta_{AHM} + \mu)\varepsilon_3 + (\kappa_{HM} + \mu + \delta_{AHM} + \tau_4)\eta_{HM} + \varepsilon_3\eta_{HM}\kappa_{HM} - \varepsilon_3\eta_{HM}(\kappa_{HM} + \mu + \delta_{AHM} + \tau_4)\right)}{(\kappa_{HM} + \mu + \delta_{AHM} + \tau_4)(\phi_3 + \delta_{AHM} + \mu)}$$

So, that the basic reproduction number of the HIV- malaria co-infection model is obtained to be

$$R_{(HM)} = \max\{R_1, R_4\}.$$

2.5. *Sensitivity analysis of HIV-Malaria co-infection model*

Sensitivity analysis was carried out to determine the model's robustness to parameter values. This helps identify the parameters that have a high impact on the reproductive number. Moreover, sensitivity indices help in developing efficient and effective intervention strategies for the control of HIV-malaria co-infection in the community.

This was calculated using the normalized forward sensitivity method, which is defined as the ratio of the relative change in $R_{HM}$to the relative change in the parameter: $'P'$:

$$\mathrm{Z}_P^{R_{HM}} = \frac{\partial R_{HM}}{\partial P} \times \frac{P}{R_{HM}}$$

Table 2. Sensitivity values on the basic reproduction number R_{HM} of HIV-malaria co-infection model

Parameters	Sensitivity indices
β_{HM}	1.000000000
ε_3	0.9907058877
ϕ_3	0.5720364764
μ	0.4145191860
δ_{AHM}	0.02901634301
η_{HM}	0.01119409061
κ_{HM}	0.004653570032
τ_4	0.0002042987416

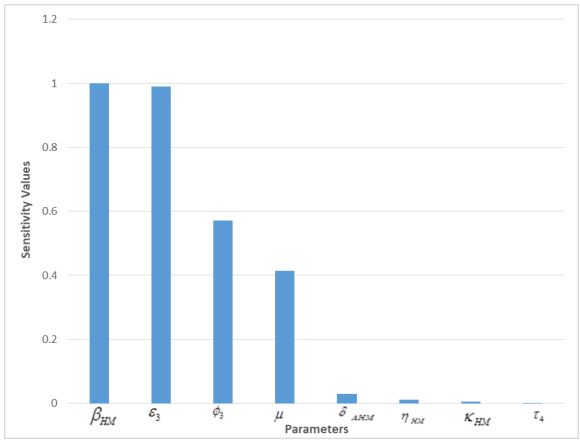

Figure 1. Chart on sensitivity indices of basic reproduction number of HIV-Malaria co-infection

3. Results and Discussion

3.1. *Results*

The analytical results of this study are illustrated by carrying out numerical simulations of the models using parameter values in Table 3 with initial values $S_H(0) = 14000$, $L_H(0) = 2000$, $I_U(0) = 200$, $I_D(0) = 300$, $T_H(0) = 350$, $E_{HM}(0) = 700$, $A_{HM}(0) = 100$, $E_M(0) = 2000$, $I_M(0) = 9000$, $T_M(0) = 180$, $R_M(0) = 7500$, $S_V(0) = 900$, $E_V(0) = 700$, and $I_V(0) = 500$. The simulations are carried out with the help of MAPLE 17 software and the results are given below

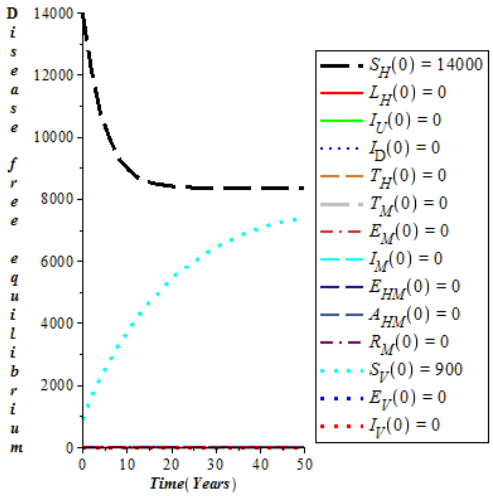

Figure 2. Graph showing disease free equilibrium point at different time

Table 3. Parameters values used for the numerical simulation

Parameters	Values	Sources
δ_{IM}	0.05	[28]
β_{HM}	0.8	Estimated
κ_{HM}	0.093	Estimated
κ_M	0.071	[29]
β_M	0.03	[30]
ε_3	0.69	Estimated
ε_2	0.6	Estimated
τ_4	0.0069	[31].
τ_3	0.0013	[32]
τ_2	0.0018	Estimated
π_V	400	Estimated
π_H	1800	Estimated
μ	0.2	Estimated
μ_V	0.05	[33]
τ_1	0.3143	[26]
ε_1	0.92	[34]
γ_{UH}	0.2	Estimated
δ_{HM}	0.093	[10]
κ_H	0.2	[35]
σ_2	0.6	[35]
σ_1	0.712	[35]
β_H	0.8	Estimated
β_V	0.09	[36]
ω_1	0.2	[35]
σ_V	0.1	[37]
ϕ_2	0.02	[35]
ϕ_3	0.28	[32]
r	0.02	[38]
b	0.4	[39]
θ	0.8	[35]
δ_{UH}	0.01	Estimated
δ_{DH}	0.008	Estimated
δ_{AHM}	0.014	[40]
$\eta_U, \eta_D, \eta_T, \eta_{HM}$	0.01	Estimated

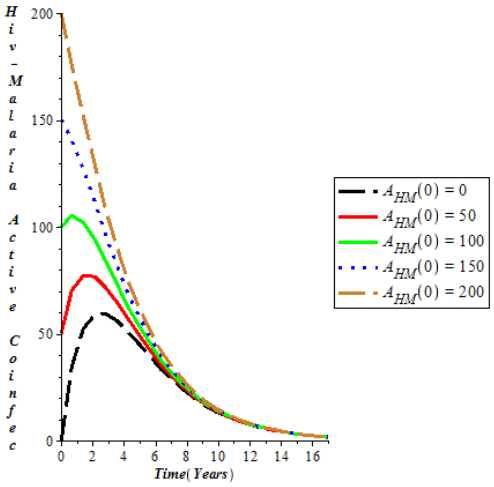

Figure 3. Graph shows the global stability of endemic point at different time

3.2. *Discussion*

Figure 2 shows the disease-free equilibrium point of HIV-malaria co-infection, as it shows that there is always someone susceptible in the population while infected individuals tend to zero. Also, Figure 3 shows the global stability of endemic, which indicates that whatever the initial values, the system will converge to the same point as time goes on. Figure 4 depicts the

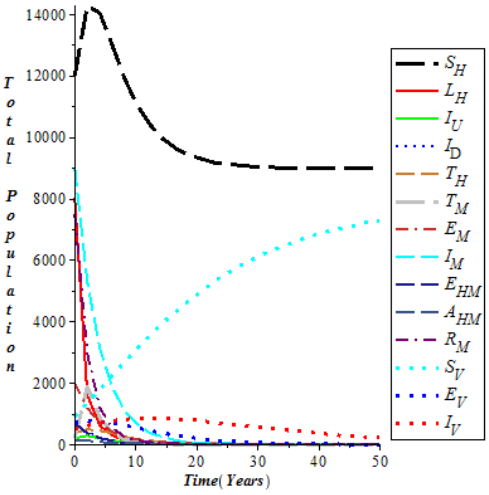

Figure 4. Graph of behavior of total population with initial value at different time

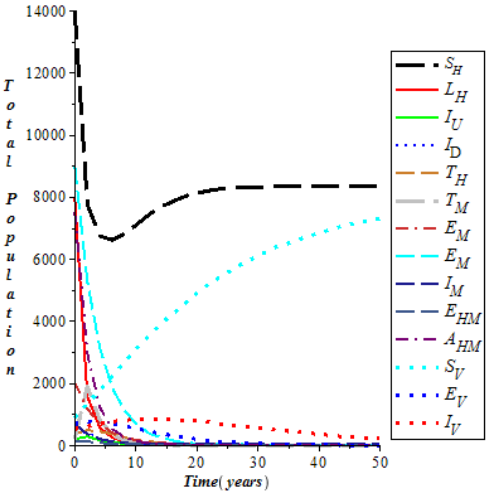

Figure 5. Graph of increasing the most positive sensitive value which is HIV-malaria contact rate on total population at different time

behavior of the HIV-malaria co-infection model, displaying the dynamics with different times of susceptible, HIV latently, infected undetected, infected detected, infected treated, isolated, exposed malaria human, infected malaria human, HIV-malaria exposed, HIV-malaria infected, susceptible vector, exposed vector, and infected vector population.

In Figure 5, the susceptible population decreases initially until the contact rate increases while the susceptible vector increases and the co-infection trajectories increase. Also, the dynamics of their trajectories remain the same. As it shows in Figure 6, when the contact rate was eliminated from the entire co-infection model, the susceptible population increased to its peak but later dropped due to malaria endemicity in the population, while the co-infection trajectories decreased and remained the same.

4. Conclusion

This work presents a comprehensive mathematical analysis of a model that incorporates the biological characteristics of

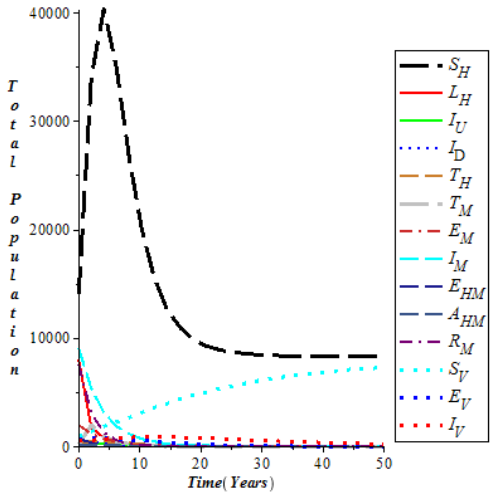

Figure 6. Graph of eliminating the most positive sensitive value which is HIV-malaria contact rate on total population at different time

HIV and malaria diseases. The analysis confirms that the model is well-posed, and the disease-free equilibrium for the model is obtained. The basic reproduction number of the HIV-malaria co-infection model is calculated, and the model stability is analyzed. It is shown that the disease-free equilibrium of each model and their co-infections are locally and globally asymptotically stable when the basic reproduction number is less than unity or endemic otherwise. The sensitivity analysis of the basic reproduction number in Table 3 and Figure 6 indicates that controlling the co-infection contact rate of HIV-malaria disease in the population is crucial in controlling HIV-malaria co-infection.

Author Contributions. **Asimiyu Olalekan Oladapo:** conceptualization, methodology, software, and validation. **Morufu Oyedunsi Olayiwola:** investigation, resources, and data curation. **Kamilu Adewale Adedokun:** writing—review and editing, and visualization. **Adedapo Ismaila Adedapo:** visualization, supervision, and project administration. **Joseph Adeleke Adedeji:** funding acquisition, software, and validation. **Kareem Oyeleye Kabiru:** writing—review and editing, visualization, and supervision. **Akeem Olanrewaju Yunus:** supervision, project administration, and funding acquisition. All authors have read and agreed to the published version of the manuscript.

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Data availability. Several parameter values are cited from some references. See Table 3.

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