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RESEARCH ARTICLE

Mathematical Modeling for the Transmission Dynamics Control of HIV and Malaria Coinfection in Nigeria towards Attaining Millenium Development Goal

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ABSTRACT

Human immune deficiency virus and malaria coinfection are among the greatest health problems globally and the understanding of how the two infections network could be imperative for the control of both diseases. Hence, this research formulated a mathematical model to study the coinfection of human immunodeficiency virus/acquired immunodeficiency syndrome and malaria in the presence of treatment as control measures. The basic reproduction number was obtained using the next generation matrix method. The simulations were carried out using MAPLE 18 software. The result shows that the malaria treatment does not have any effect on the total coinfected populations. Thus, every sick person should be tested to ascertain the source of their health challenges.

Key words: Basic reproduction number R_0, Disease free and endemic equilibria, Locally and globally asymptotically stable, Simulation

INTRODUCTION

Human immunodeficiency virus (HIV) is an infection which destroys the body resilient systems, upsurges the risk of pathologies which harms body organs such as; the brain, kidney, heart, and causes death. Malaria is a disease spread by infected mosquitoes; it was first revealed centuries ago by the Chinese in 2700BC. However, it was until the late 1800s when Ross made his ground breaking findings that led to our understanding of the mechanisms behind malaria infection and transmission.[1] *Plasmodium falciparum* reported for 99.7% of estimated malaria cases in the world. In Africa region, 50% of cases in the World Health Organization (WHO) South-East Asia region, 71% of cases in the Eastern Mediterranean and 65% in the Western Pacific. Malaria has also been found to cause 20% of deaths in children under the age of five.

As at 2016, the morbidity of HIV globally, tolls at about 36.7 million people, from which only 20.9 million people were having access to the anti-retroviral drugs. 1.18 million people became newly infected. Totally, about 78 million people have been infected with HIV since the discovery of epidemics and the mortality tolls at 35 million people. Recently known infected persons globally numbers at 2.1 million. In Nigeria, about 3.5 million persons are reported to be carrying the deadly virus. Mortality tolls at 180,000 individuals both old and young, with a predominant rate of 3.1% among adults aged 15 and above.[2,3] HIV/acquired immunodeficiency syndrome (AIDS) can be contracted through insecure sex with an infected person, breast milk of an infected woman, blood transfusion from an infected person, and sharing of sharp objects with infected persons.[4] Mathematical models have been well-thought-out to weigh the consequence of public sensitization programs,[5,6] the usage of anti-retroviral drugs, and provision of long-time forecasts regarding HIV/AIDS prevalence and control in various regions.

The spreading of HIV and malaria overlap globally. Hene, there is always a probability of co-infection. The effect of medication on HIV-Malaria coinfection has been analyzed and a

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mathematical model has been formulated[5,7] incorporating the treatment class. The disease-free equilibrium (DFE) of the HIV-only model is globally-asymptotically stable when the reproduction number is <1. However, it is shown that in the malaria-only model, there is a cooccurrence of stable DFE and stable endemic equilibrium, for a certain interval of the reproduction number less than unity. This indicates the existence of backward bifurcation. Numerical simulations of the full model are carried out to observe the impact of treatment at different levels. It is shown that malaria-only treatment strategy reduces more new cases of the mixed infection than the HIV-only treatment strategy. Moreover, mixed treatment strategy reduces the least number of new cases compared to single treatment strategies.

HIV-malaria coinfection may be responsible for growth seen in set-point viral load (spVL),[8] at the within-host level over time.[9,10] This rises because the coinfection varies significantly within the host.[11] Furthermore, the concentrations of coinfection in high-risk groups versus low-risk groups may affect how HIV blow-outs in the entire population.[7] To understand the paraphernalia responsible for these effects of coinfection, an immunoepidemiological model was formulated.[12] They found that populations with higher spVL lead to higher increases in viral load due to coinfection, whereas populations with lower spVL lead to reduction in viral load due to coinfection. This leads to a greater chance of coinfection increasing the occurrence of HIV in populations with high average spVL.[12] Therefore, the result of coinfection may be eased by sensing the viral factors that can shrink the spVL in the population.

HIV immunoepidemiologic models syndicate the immune-viral dynamics at the within-host immunological scale with the transmission dynamics at the between-host epidemiological scale to analyze HIV dynamics of a single strain infection, coinfection, super-infection, evolution, drug resistance, and treatment protocols in heterogeneous populations. Hence, this research commended previous works on HIV and malaria coinfection model and complemented their works by formulating an HIV-malaria coinfection model incorporating malaria recovery class.

MODEL FORMULATION FOR COINFECTION

The following assumptions are put into consideration in the model formulation:

i. It is assumed that every human who are coming into the population are susceptible to HIV and malaria
ii. A person gets infected with HIV through blood contact with HIV infected human and gets infected with malaria when exposed to infected mosquito bite
iii. An individual can only recover from malaria and then becomes susceptible after the waning period.

The model schematic diagram is as shown in Figure 1:

The susceptible individuals (S) are generated through constant recruitment of individuals into the population either by birth or immigration at the rate Λ. They decrease due to effective contact with I_H and I_M given by the force of infection.

$$\lambda_H+\lambda_M \tag{1}$$

Where

$$\lambda_H = \frac{\beta_H I_H}{N}, \lambda_M = \frac{\beta_M I_V}{N} \tag{2}$$

β_H and β_M are the effective contact rate of the susceptible individuals with the HIV-infected individuals and the infected mosquitoes, respectively. The susceptible population further decreases due to natural mortality at rate μ and increases due to waning rate ω of anti-malaria immunity.

The malaria-infected individuals I_m are generated through effective contact between the susceptible

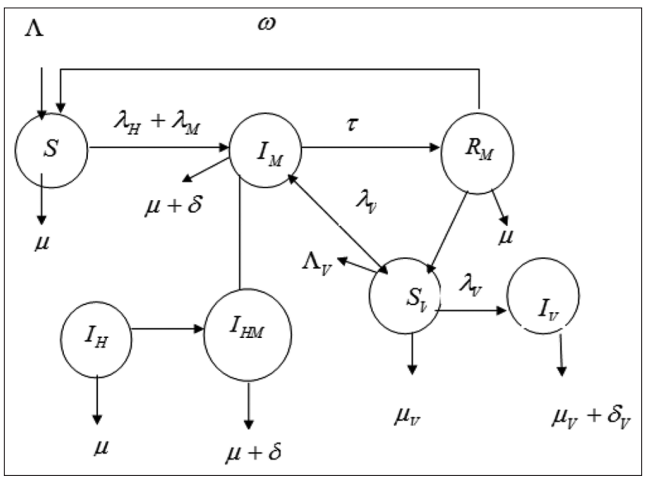

Figure 1: Schematic diagram of HIV/malaria coinfection model incorporating malaria recovery compartment

individuals and the infected mosquitoes with the force of infection $\frac{\beta_M I_V}{N}$. The malaria-infected population decreases due to the contact between λ_H and I_M, and progresses to I_{HM} at the rate σ_1; hence, the malaria-infected population further decreases due to natural mortality μ, malaria-induced death δ and treatment at the rate τ.

The HIV-infected individuals I_H are generated through effective contact between the susceptible individuals and the HIV-infected individuals with the force of infection $\frac{\beta_H I_H}{N}$. The HIV infected population decreases due to coinfection and progresses to HIV malaria coinfection compartment at the rate σ_2, the HIV-infected population further decreases due to natural mortality μ, and when it has an effective contact with carrier vector with infection force $\frac{\beta_M I_V}{N}$.

The HIV/malaria coinfection compartment is generated from $\sigma_1\lambda_H I_M$, $\lambda_M I_H$ and $\sigma_2 I_{HM}$ and decreases due to natural mortality μ and disease-induced death τ.

The malaria recovered class is generated due to the treatment of malaria-infected individuals at rate τ and decreases due to natural mortality μ and waning rate ω of anti-malaria immunity.

The susceptible vectors (S_v) are generated through constant recruitment of mosquitoes into the environment by hatching of new mosquitoes at rate Λ_v. It is reduced due to effective contact with I_M and I_{HM} at rate β_{MV} with the infection force

$$\lambda_V = \frac{\beta_{MV}(I_{HM} + \rho I_M)}{N} \tag{3}$$

The susceptible vectors are further reduced due to natural mortality μ_v

The infected vectors (I_v) are generated through effective contact with I_M and I_{HM} at rate β_{MV} with the susceptible vectors populations and it decreases due to natural mortality μ_v and disease-induced death δ_v.

The model equations are as shown:

$$\frac{dS}{dt} = \Lambda + \omega R_m - (\lambda_H + \lambda_M)S - \mu S \tag{4}$$

$$\frac{dI_M}{dt} = \lambda_M S - \sigma_1 \lambda_H \mathrm{I}_M - (\tau + \mu + \delta) I_M \tag{5}$$

$$\frac{dI_H}{dt} = \lambda_H S - \sigma_2 I_{HM} - \mu I_H - \lambda_M I_H \tag{6}$$

$$\frac{dI_{HM}}{dt} = \lambda_M I_H + \sigma_1 \lambda_H I_M + \sigma_2 I_{HM} - (\mu + \delta) I_{HM} \tag{7}$$

$$\frac{dR_M}{dt} = \tau I_M - (\mu + \omega)\mathrm{R}_M \tag{8}$$

$$\frac{dS_V}{dt} = \Lambda_V - \lambda_V S_V - \mu_V S_V \tag{9}$$

$$\frac{dI_V}{dt} = \lambda_V S_V - (\mu_V + \delta_V) I_V \tag{10}$$

$$N_H = S + I_M + I_H + I_{HM} + R_M \tag{11}$$

$$N_V = S_V + I_V \tag{12}$$

Since the model is dealing with populations, all the variables and parameters of the model are positive with the natural death rates positive, that is, *(*μ>0), thus considering the region Ω where:

$$(\mathrm{S}, \mathrm{I}_M, \mathrm{I}_H, \mathrm{I}_{HM}, \mathrm{R}_M, \mathrm{S}_V, \mathrm{I}_V) = (\mathrm{S}^*, \mathrm{I}_M^*, \mathrm{I}_H^*, \mathrm{I}_{HM}^*, \mathrm{R}_M^*, \mathrm{S}_V^*, \mathrm{I}_V^*) \tag{13}$$

It can be established that all solutions of the system starting in Ω remain in Ω for all t > 0. In this region, the usual existence, uniqueness, and continuation of results hold for the system.

EQUILIBRIUM POINTS OF THE MODEL

Equilibrium state is the point at which there is no external influence on the system. Thus, at equilibrium

$$\frac{dS}{dt} = \frac{dI_M}{dt} = \frac{dI_H}{dt} = \frac{dI_{HM}}{dt} = \frac{dR_M}{dt} = \frac{dS_V}{dt} = \frac{dI_V}{dt} = 0 \tag{14}$$

Let

$$(\mathrm{S}, \mathrm{I}_M, \mathrm{I}_H, \mathrm{I}_{HM}, \mathrm{R}_M, \mathrm{S}_V, \mathrm{I}_V) = (\mathrm{S}^*, \mathrm{I}_M^*, \mathrm{I}_H^*, \mathrm{I}_{HM}^*, \mathrm{R}_M^*, \mathrm{S}_V^*, \mathrm{I}_V^*) \tag{15}$$

At any arbitrary equilibrium point. Thus, the model equations become;

$$\Lambda + \omega R_M^* - \left(\frac{\beta_H I_H^*}{N} + \frac{\beta_M I_V^*}{N}\right) S^* - \mu S^* = 0 \tag{16}$$

$$\frac{\beta_M I_V^*}{N} S^* - A_1 I_M^* = 0 \quad (17)$$

$$\frac{\beta_H I_H^*}{N} S^* + \sigma_2 I_{HM}^* - \mu I_H^* - \frac{\beta_M I_V^*}{N} I_H^* = 0 \quad (18)$$

$$\frac{\beta_M I_V^*}{N} I_H^* - A_2 I_{HM}^* = 0 \quad (19)$$

$$\tau I_M^* - A_3 \mathrm{R}_M^* = 0 \quad (20)$$

$$\Lambda_V - \frac{\beta_{MV}(I_{HM}^* + \rho I_M^*)}{N} S_V^* - \mu_V S_V^* = 0 \quad (21)$$

$$\frac{\beta_{MV}(I_{HM}^* + \rho I_M^*)}{N} S_V^* - A_4 I_V^* = 0 \quad (22)$$

$$A_1 = (\tau + \mu + \delta), A_2 = (\mu + \delta),$$
$$A_3 = (\mu + \omega), A_4 = (\mu_V + \delta_V) \quad (23)$$

$$\left(\frac{\beta_H}{N} S^* + \sigma_2 \frac{(\beta_H I_M^* \sigma_1 + \beta_M I_V^*)}{A_2 N} - \mu - \frac{\beta_M I_V^*}{N} \right) = 0 \quad (24)$$

$$I_H^* = 0 \quad (25)$$

DFE POINT OF THE MODEL

DFE is the state at which the population is free from infections.

Lemma 1: The DFE of the model exists and is given by;

$$E^0 = \left(S^0, I_M^0, I_H^0, I_{HM}^0, R_M^0, S_V^0, I_V^0\right)$$
$$= \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \frac{\Lambda_V}{\mu_V}, 0\right) \quad (26)$$

Thus, let

$$E^o = \left(S, I_M, I_H, I_{HM}, R_M, S_V, I_V\right)$$
$$= \left(S^0, I_M^0, I_H^0, I_{HM}^0, R_M^0, S_V^0, I_V^0\right) \quad (27)$$

$$E^0 = \left(S^0, I_M^0, I_H^0, I_{HM}^0, R_M^0, S_V^0, I_V^0\right)$$
$$= \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \frac{\Lambda_V}{\mu_V}, 0\right) \quad (28)$$

BASIC REPRODUCTION NUMBER, R_0 OF COINFECTION MODEL

The basic reproduction number is the average number of secondary infections caused by a single infectious individual during his/her entire infectious life time. Applying next generation matrix operator to compute the basic reproduction number of the model as used by Cuadros and García-Ramos[8] and improved by Van den Driessche and Watmough.[13] The basic reproduction number is obtained by dividing the whole population into n compartments in which there are $m<n$ infected compartments. Let x_i, i=1,2,3,…,m be the number of infected individuals in the i^{th} infected compartment at time t. The largest eigenvalue or spectra radius of FV^{-1} is the basic reproduction number of the model.

$$FV^{-1} = \left[\frac{\partial F_i(E^0)}{\partial x_i}\right]\left[\frac{\partial V_i(E^0)}{\partial x_i}\right]^{-1} \quad (29)$$

Where F_i is the rate of appearance of new infection in compartment i to another and E^0 is the DFE.

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_M S^*}{N} \\ 0 & \frac{\beta_H S^*}{N} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta_{MV}\rho S_V^*}{N} & 0 & \frac{\beta_{MV}\rho S_V^*}{N} & 0 \end{pmatrix} \quad (30)$$

$$V = \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{pmatrix} = \begin{pmatrix} -\sigma_1 \frac{\beta_H I_H^*}{N} \mathrm{I}_M^* - A_1 I_M^* \\ -\mu I_H^* - \frac{\beta_M I_V^*}{N} I_H^* \\ -A_2 I_{HM}^* \\ -A_4 I_V^* \end{pmatrix} \quad (31)$$

$$V^{-1} = \begin{pmatrix} 0 & -\frac{\beta_M S^*}{N A_4 A_2 A_1} & 0 & 0 \\ 0 & \frac{\beta_H S^*}{N A_4 A_2 A_1} & 0 & 0 \\ 0 & 0 & \frac{-\sigma_2}{\mu A_1 A_4} & 0 \\ \frac{\beta_{MV}\rho S_V^*}{N\mu A_2 A_1} & 0 & \frac{-\beta_{MV} S_V^*}{N\mu A_4 A_1} & 0 \end{pmatrix} \quad (32)$$

$$\left| fv^{-1} - \lambda I \right| = 0 \quad (33)$$

$$R_0 = \frac{\beta_{MV}\rho S_V \beta_H S^2 \beta_M \sigma_2}{N^3 A_2^3 \mu^2 A_4^4 A_1^3} \quad (34)$$

Hence, the basic reproduction number of the model is given by (34) which is the average number of secondary infections caused by a single infectious individual during his/her entire infectious life time.

LOCAL STABILITY OF DFE FOR COINFECTION MODEL

According to Cuadros and García-Ramos[8] theorem, the DFE is locally asymptotically stable (LAS) if there exists R_C and $R_C<1$ or if all the Eigen values of the model Jacobian matrix are negative, otherwise it is unstable. We want to further justify the theorem using Jacobian techniques for stability.

Lemma 3.1: The disease free equilibrium of the model is LAS if all the Eigen values of the model Jacobian matrix are negative or $R_C<1$.

Proof:

The Jacobian matrix of equations (3.2) to (3.6) at DFE, E^0 is given by:

$$J=\begin{pmatrix} -\mu & 0 & \frac{-\beta_H S}{N} & 0 & \omega & 0 & \frac{-\beta_M S}{N} \\ 0 & -A_1 & 0 & 0 & 0 & 0 & \frac{\beta_M S}{N} \\ 0 & 0 & -\mu & -\sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_2+\sigma_2 & 0 & 0 & 0 \\ 0 & \tau & 0 & 0 & -A_3 & 0 & 0 \\ 0 & -\frac{\beta_{MV}\rho S_V}{N} & 0 & \frac{-\beta_{MV}\rho S_V}{N} & 0 & -\mu_V & 0 \\ 0 & \frac{\beta_{MV}\rho S_V}{N} & 0 & \frac{\beta_{MV} S_V}{N} & 0 & 0 & -A_4 \end{pmatrix} \tag{35}$$

Reducing to upper triangular matrix;

$$J=\begin{pmatrix} -\mu & 0 & \frac{-\beta_H S}{N} & 0 & \omega & 0 & \frac{-\beta_M S}{N} \\ 0 & -A_1 & 0 & 0 & 0 & 0 & \frac{\beta_M S}{N} \\ 0 & 0 & -\mu & -\sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_2+\sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_3 & 0 & \frac{\tau\beta_M S}{A_1 N} \\ 0 & 0 & 0 & 0 & 0 & -\mu_V & -\frac{\beta_{MV}\rho S_V \beta_M S}{A_1 N^2} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{MV}\rho\sigma_2 S_V \beta_M S - A_4^4 N^3 \mu^2 A_1^3 A_2^3}{A_1 N^2} \end{pmatrix} \tag{36}$$

$$|J-\lambda I|=0 \tag{37}$$

Implies,

$$\lambda_1<0,\ \lambda_2<0,\ \lambda_3<0,\ \lambda_4<0,\ \lambda_5<0,\ \lambda_6<0 \tag{38}$$

$$\lambda_7=\frac{\beta_{MV}\rho\sigma_2 S_V \beta_M S - A_4^4 N^3 \mu^2 A_1^3 A_2^3}{A_1 N^2} \tag{39}$$

$$\lambda_7<0 \text{ if } R_0<1 \tag{40}$$

$$\frac{\beta_{MV}\rho\sigma_2 S_V \beta_M S}{A_4^4 N^3 \mu^2 A_1^3 A_2^3} - 1 < 0 \quad (41)$$

$$R_0 < 1 \quad (42)$$

Hence, the disease free is LAS.

MALARIA ONLY MODEL

The model equations for the malaria only are given as:

$$\frac{dS}{dt} = \Lambda + \omega R_m - \frac{\beta_M I_V S}{N} - \mu S \quad (43)$$

$$\frac{dI_M}{dt} = \frac{\beta_M I_V S}{N} - A_1 I_M \quad (44)$$

$$\frac{dR_M}{dt} = \tau I_M - A_3 \mathrm{R}_M \quad (45)$$

$$\frac{dS_V}{dt} = \Lambda_V - \frac{\beta_{MV}\rho I_M S_V}{N} - \mu_V S_V \quad (46)$$

$$\frac{dI_V}{dt} = \frac{\beta_{MV}\rho I_M S_V}{N} - A_4 I_V \quad (47)$$

$$N_H = S + I_M + R_M \quad (48)$$

$$N_V = S_V + I_V \quad (49)$$

Existence of Equilibria of Malaria Only

Suppose at equilibria,

$$E^* = (S, I_M, R_M, S_V, I_V) = (S^*, I_M^*, R_M^*, S_V^*, I_V^*) \quad (50)$$

$$\left(\frac{\beta_{MV}\beta_M S^*}{N A_1 N} S_V^* - A_4\right) = 0 \quad (51)$$

Or

$$I_V^* = 0 \quad (52)$$

DFE for Malaria Only

Lemma 2: The DFE of the model exists and is given by;

$$E^o = (S, I_M, R_M, S_V, I_V) = (S^0, I_M^0, R_M^0, S_V^0, I_V^0)$$

$$= \left(\frac{\Lambda}{\mu}, 0, 0, \frac{\Lambda_V}{\mu_V}, 0\right) \quad (53)$$

Suppose at DFE,

$$E^o = (S, I_M, R_M, S_V, I_V) = (S^0, I_M^0, R_M^0, S_V^0, I_V^0) \quad (54)$$

Thus,

$$E^o = (S, I_M, R_M, S_V, I_V) = (S^0, I_M^0, R_M^0, S_V^0, I_V^0)$$

$$= \left(\frac{\Lambda}{\mu}, 0, 0, \frac{\Lambda_V}{\mu_V}, 0\right) \quad (55)$$

Basic reproduction number, R_0 of Malaria Model

$$F = \begin{pmatrix} 0 & \frac{\beta_M S}{N} \\ \frac{\beta_{MV} S_V}{N} & 0 \end{pmatrix} \quad (56)$$

$$V = \begin{pmatrix} -A_1 & 0 \\ 0 & -A_4 \end{pmatrix} \quad (57)$$

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta_M S}{A_4 N} \\ \frac{\beta_{MV} S_V}{A_1 N} & 0 \end{pmatrix} \quad (58)$$

$$R_0 = \frac{\beta_M \beta_{MV} S S_V}{A_1 A_4 N} \quad (59)$$

$$J = \begin{pmatrix} -\mu & 0 & \omega & 0 & -\frac{\beta_M S}{N} \\ 0 & -A_1 & 0 & 0 & \frac{\beta_M S}{N} \\ 0 & \tau & -A_3 & 0 & 0 \\ 0 & -\frac{\beta_{MV} S_V}{N} & 0 & -\mu_V & 0 \\ 0 & \frac{\beta_{MV} S_V}{N} & 0 & 0 & -A_4 \end{pmatrix} \quad (60)$$

Reducing to upper triangular matrix

$$J = \begin{pmatrix} -\mu & 0 & \omega & 0 & -\frac{\beta_M S}{N} \\ 0 & -A_1 & 0 & 0 & \frac{\beta_M S}{N} \\ 0 & 0 & -A_3 & 0 & \frac{\tau\beta_M S}{A_1 N} \\ 0 & 0 & 0 & -\mu_V & -\frac{\beta_{MV} S_V \beta_M S}{N^2 A_1} \\ 0 & 0 & 0 & 0 & \frac{\beta_{MV} S_V \beta_M S - A_4 A_1 N^2}{N^2 A_1} \end{pmatrix} \quad (61)$$

$$|J-\lambda I|=0 \quad (62)$$

Implies,

$$\lambda_1<0, \lambda_2<0, \lambda_3<0, \lambda_4<0 \quad (63)$$

$$\lambda_5 = \frac{\beta_{MV} S_V \beta_M S - A_4 A_1 N^2}{N^2 A_1} \quad (64)$$

$$R_0-1<0 \quad (65)$$

$$R_0<1 \quad (66)$$

Hence, the disease free is LAS.

RESULTS AND DISCUSSION

In this section, some numerical simulation for monitoring the dynamics of the full model was presented, to have the pictorial demonstration of the model dynamics using MAPLE 18 software. Table 1 shows the baseline values for variables of the HIV and malaria coinfection, while Table 2 shows the values for the parameters used in the model.

SIMULATIONS

In this section, some numerical simulations associated with different values of the effective reproduction number of the model were presented with the aid of graph [Figures 2-6] to further justify the analytical results.

This present Total Number of Infected Individuals with the same initial variable conditions at (I(0)=1000 (R_C=1.916554180>1). It could be noted that with the same initial population of the

Table 1: Baseline values for variables of the HIV and malaria coinfection in Nigeria

S/No	Variables	Values	References
1	N	206,139,589	[14]
2	S	206,135,589	Calculated
3	I_M	1000	Assumed
4	I_H	1000	Assumed
5	I_{HM}	1000	Assumed
6	R_M	1000	Assumed
7	S_V	1,000,000	[1]
8	I_V	629,576	Assumed

Table 2: Baseline values for the parameters used in the model

S/No	Variables	Values	References
1	Λ	3,710,513	Calculated
2	β_M	0.2	[14]
3	β_H	0.2	Assumed
4	β_{MV}	0.2	Assumed
5	ω	0.2	Assumed
6	σ_1	0.05	Assumed
7	σ_2	0.05	Assumed
8	μ	0.018	Assumed
9	δ	0.01	Assumed
10	τ	(0–1)	Assumed

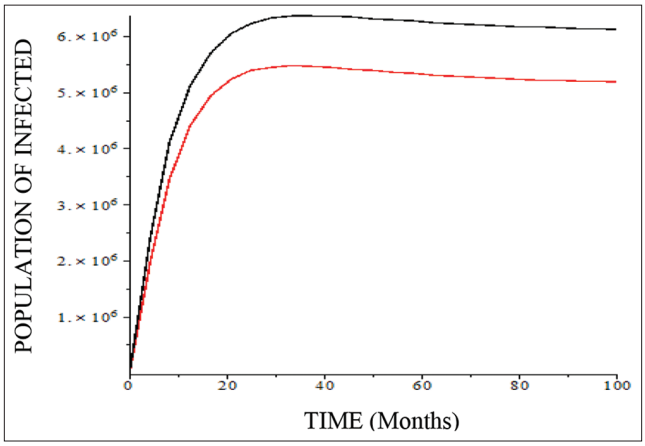

Figure 2: Graph of coinfection and malaria only

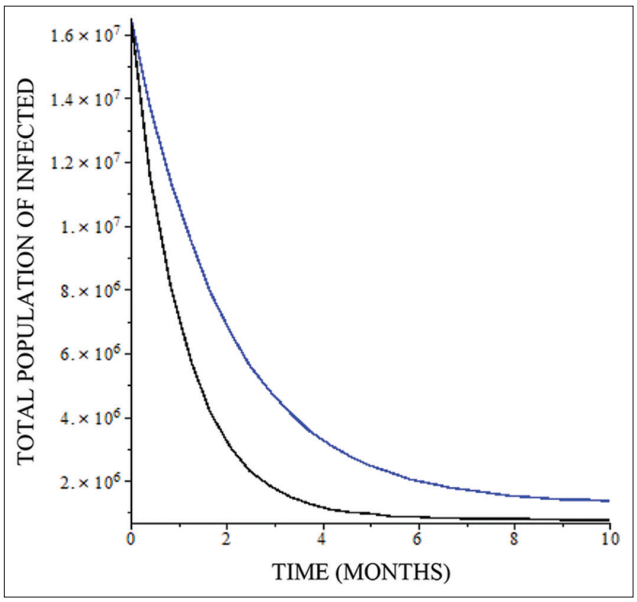

Figure 3: Graph of coinfection and single infection

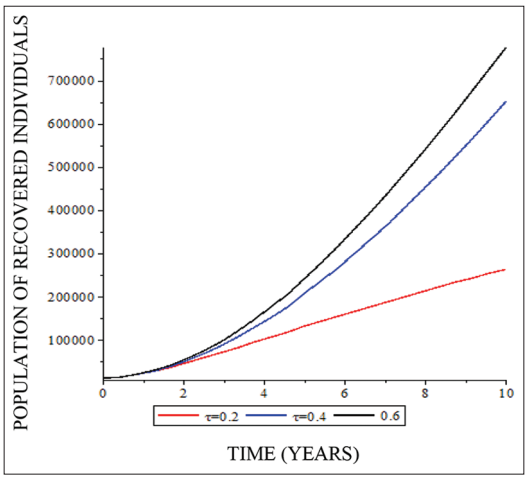

Figure 4: Effect of treatment on malaria recovery class

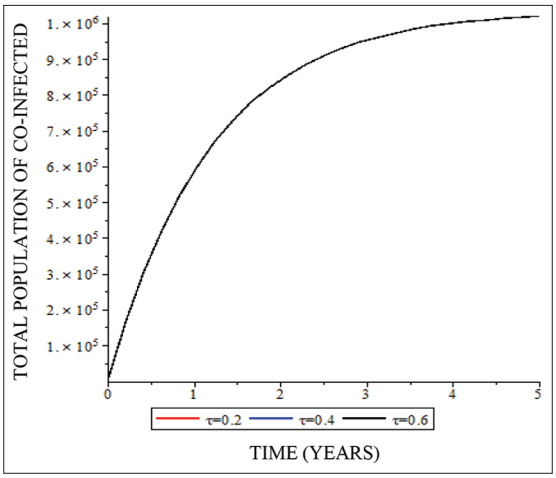

Figure 5: Effect of treatment on coinfected individuals

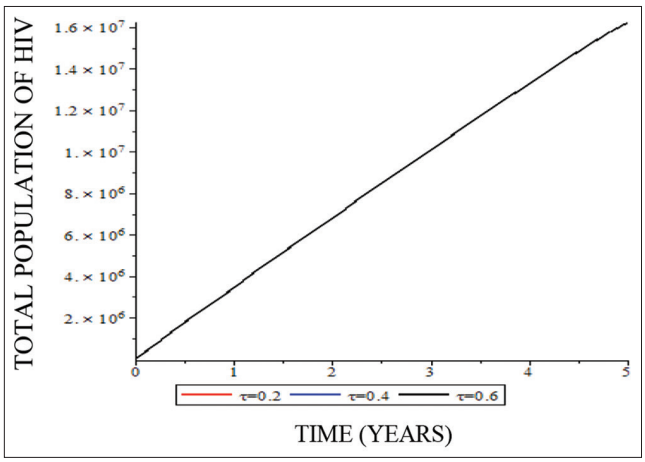

Figure 6: Effect of treatment on HIV-infected individuals

infected individuals, the infected population grows when R_C>1, although the growth for malaria only is not as rapid and high as that of coinfection. Hence, it is a justification of the stability analysis of the model.

Total number of infected individuals with the same initial variable conditions (i.e., I(0)=1,600,000 (R_C=0.74558<1). It could be noted that with the same initial population of the infected individuals, the infected population declines when R_C<1, although the drop for coinfection is not as rapid and low as that of single-infection. Hence, it is a justification of the stability analysis of the model.

It could be noted from the graph that the higher the treatment, the higher the malaria recovery population.

It could be noted from the graph that treatment rate does not have effect on the coinfected population.

It could be noted from the graph that malaria treatment rate does not have effect on the HIV-infected population.

CONCLUSION

This research work has revealed that 60% rate of treatment of malaria-infected individuals will reduce the malaria-infected population drastically, while increasing the recovered populations within 10 months. Since malaria treatment does not have effect on HIV and malaria coinfected individuals, it is advisable to run HIV test on every sick patient to know their status before treating them for malaria fever.

RECOMMENDATIONS

i. Effective anti-malaria treatment of malaria-infected individuals is highly recommended
ii. Since antimalaria will not have significant effect on coinfected individuals, HIV test should be carried out on every malaria patient to treat those who tested positive with anti-retroviral drugs first before administering anti-malaria treatments to them
iii. We also want to recommend to the WHO, CDC, and NAFDAC that the efficacy level of the produced condoms should be at 95% and above to reduce the risk of getting infected through sex. Since HIV transmission depends largely on effective contact rate
iv. One of the limitations of this study is the unavailability of records of HIV-malaria coinfection cases; therefore, health workers should make data available for researchers.

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