Local and global asymptotic behavior of malaria-filariasis coinfections in compliant and noncompliant susceptible pregnant women to antenatal medical program in the tropics

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Communicated by Valery Covachev

Abstract. In this paper, a mathematical nonlinear model system of equations describing the dynamics of the co-interaction between malaria and filariasis epidemic affecting the susceptible host population of pregnant women in the tropics is formulated. The basic reproduction number R_{mf} of the coepidemic model is obtained, and we investigated that it is the threshold parameter between the extinction and persistence of the coepidemic disease. If $R_{mf} < 1$, then the disease-free steady state is both locally and globally asymptotically stable resulting in the disease dying out of the host. Also, if $R_{mf} > 1$, the disease lingers on. The center manifold theory is used to show that the unique endemic equilibrium is locally asymptotically stable. However, variations in the parameter values involved in the model build up will bring about appropriate control measures to curtail the spread of the coepidemic disease. Numerical simulations are carried out to confirm the theoretical results.

Keywords. Malaria-filariasis, coinfection, reproduction number, center manifold, local stability.

2010 Mathematics Subject Classification. 92B05, 93D05, 34K12, 34K20.

1   Introduction

Coinfection is an infection of a single host simultaneously by a causative agent with multiple pathogens which possess various strains of the disease. Malaria - filariasis coinfection is an old parasitic disease that still poses a serious threat and a medical challenge to the global world population, where the endemic impact is most felt in the developing and third world countries in Africa and Asia. These two coendemic diseases account for the world largest mortality and morbidity respectively. According to the World Health Organization (WHO) fact sheet of 2018 [1], malaria killed an estimated 438, 000 individuals, where over 90 percent deaths occurred in Africa, while 10 and 2 percents deaths were recorded in Asia and the

Eastern Mediterranean Region respectively. Malaria is a deadly parasitic disease transmitted to the human body through the bites of infected mosquitos, mainly female Anopheles mosquito. The clinical signs associated with malaria after few days are tiredness, headache, high fever, chills, etc.

Lymphatic filariasis is another debilitating mosquito-borne disease caused by parasitic nematode of filaroidea family. There are three types of filaria parasites, namely, Wuchereria bancrofti, which is responsible for 90 percent of cases, Brugia timori, Brugia malayi. According to WHO fact sheet of 2018 [2], 856 million people in 52 countries remain threatened by filariasis and require preventive chemotherapy to curtail the spread of this parasitic disease. Infection happens when filarial parasites are deposited in human body through mosquito bite, thereby causing damage to the lymphatic system. The filaria worms can live in the lymphatic vessels for $6-8$ years, producing millions of microfilariae (immature larvae) that circulate in the blood. Infected humans exhibit clinical manifestation of lymphoedema, elephantiasis, body deformation, thereby leading to social stigma. Nigeria is one of the subtropical countries where this codisease thrives. A. G. Abdullahi et al. [8], reported the presence of lymphatic filariasis and malaria in rural communities in Nassarawa state of Nigeria, see also [3, 13]. In 2014, Nigeria launches Africa's first nationwide malaria and lymphatic filariasis elimination coimplementation plan to protect the citizens from this dreaded disease [5, 7].

In this paper, our focus is on analyzing the dynamics of the spread of coinfectious malaria - filariasis disease among susceptible pregnant women in Nigeria, who are compliant or noncompliant to antenatal medical program to access treatment and care to reduce cases of maternal and foetal mortality [4, 6, 9]. The works of $[10 - 12, 14 - 22, 24]$ have proved very useful in the understanding of coinfection dynamics of the spread of diseases. Having gone through all the cited literatures above, and motivated by the works of H.C. Slater [23] and $[12, 25 - 29]$, in this study, a mathematical coendemic deterministic model describing the qualitative dynamics of malaria - filariasis coinfections considering pregnant women only is considered. The rest of the paper is organized as follows: Section 2 presents the analytical discussion and formulation of malaria - filariasis coinfection transmission model with corresponding initial conditions. In Section 3, the basic reproduction number R_{mf} is obtained. Also, in Section 4, the time-independent steady-state solutions are found where the malaria - filariasis coinfection is present and absent in the host population, while the necessary and sufficient conditions for the asymptotic stability of the coinfections at disease-free steady state are investigated both locally and globally. Furthermore, the endemic steady state is analyzed by employing the center manifold theory. Section 5 deals with the numerical simulations and graphical illustrations of the behavior of the variables and parameters involved in the model. Also, conclusions and recommendations were made.

2   Mathematical model formulation

In this model, the total pregnant women population at time t denoted N_h is subdivided into disjoint sub-populations of individual pregnant women who are compliant to medical antenatal program denoted S_h; Individual susceptible women who are noncompliant to medical antenatal program as S_2; Individual pregnant women infected with malaria only I_m; Individual pregnant women infected with filariasis only I_f; Individual pregnant women infected with both malaria and filariasis I_{mf}; Individual pregnant women treated from malaria - filariasis coinfections as T; Thus, $N_h = S_h + S_2 + I_m + I_f + I_{mf} + T$. Since the spread of malaria - filariasis is being carried out by the same vector (Anopheles mosquito), the total vector population is denoted N_v; Susceptible mosquito as S_v; and exposed mosquito as E_v, and infected mosquito as I_v, such that $N_v = S_v + E_v + I_v$.

The population of the susceptible pregnant women compliant to medical antenatal program is recruited into the host population at constant per capita rate A_h, while π and τ are represented as the fraction of susceptible pregnant women that are compliant to antenatal medical program, and loss of immunity to the codisease respectively. This group is further reduced by natural death rate μ_h and δ as the progression rate from S_h to S_2 group and force of infection $K_{ma}S_h$ and $K_f S_h$ respectively. Hence the rate of change of the host population is thus given as

$$\dot{S}_h = \pi A_h - (K_{ma} + K_f)S_h - (\mu_h + \delta)S_h + \tau T. \qquad (1)$$

Also, S_2 is generated by $(1 - \pi)A_h$ individual susceptible pregnant women who are noncompliant to antenatal medical program, progression rate δ_1, and force of infection $(K_{ma} + K_f)S_h$. It is reduced by mortality rate due to S_2 group, natural death rate μ_h, and $\epsilon\sigma_h$, where ϵ is the modification parameter representing the assumption that filariasis infected individuals exposed to malaria develop malaria faster than those infected with filariasis. Also, σ_h is the rate of malaria symptoms by susceptible pregnant women noncompliant to antenatal medical program, while γ is the disease-induced death rate. Thus, the rate of change is given by

$$\dot{S}_2 = (1 - \pi)A_h + \delta_1 S_h + (K_{ma} + K_f)S_h - (\epsilon\sigma_h + \gamma + \mu_h)S_2. \qquad (2)$$

Furthermore, the rate of change of I_m class is increased by $\epsilon\sigma_h S_2$ and decreased by $\theta K_f I_m$, where θ takes into account the increased susceptibility to infection with filariasis for individuals infected with malaria. Also, it is further reduced by the quantity $(\psi + \phi_1 + \mu_h)I_m$, where ϕ_1, μ_h, ψ are the treatment rate, natural death rate, and progression rate respectively. Hence, it is given by

$$\dot{I}_m = \epsilon\sigma_h S_2 - \theta K_f I_m - (\psi + \phi_1 + \mu_h)I_m. \tag{3}$$

Also, I_f class is increased with progression rate ψI_m but decreased by $\rho K_{ma}I_f$, where ρ takes into account the decreased susceptibility to infection with malaria for individuals infected with filariasis, $\phi_2 I_f$ as the recovery rate from filariasis through treatment, natural death rate $\mu_h I_f$. Thus, the state equation is given by

$$\dot{I}_f = \psi I_m - \rho K_{ma}I_f - (\gamma + \phi_2 + \mu_h)I_f. \tag{4}$$

The rate of change of population of individual pregnant women infected with both malaria and filariasis is increased by $\theta K_f I_m$ and $\rho K_{ma}I_f$ but decreased by recovery rate $\phi_3 I_{mf}$, $\upsilon\phi I_{mf}$ as the treatment rate for the coinfection, $\eta\gamma I_{mf}$ is the codisease induced death rate and natural death rate $\mu_h I_{mf}$. Hence, it is given by

$$\dot{I}_{mf} = \theta K_f I_m + \rho K_{ma}I_f - (\phi_3 + \upsilon\phi + \eta\gamma + \mu_h)I_{mf}. \tag{5}$$

The rate of change of population of treated pregnant women infected with both malaria and filariasis is increased by $\upsilon\phi I_{mf}$, $\phi_1 I_m$, $\phi_2 I_f$, $\phi_3 I_{mf}$ but decreased by natural death rate $\mu_h T$ and loss of immunity rate τT. Then

$$\dot{T} = \upsilon\phi I_{mf} + (\phi_1 I_m + \phi_2 I_f + \phi_3 I_{mf}) - (\mu_h + \tau)T. \tag{6}$$

The rate of change of population of susceptible mosquito is generated by the recruitment of mosquitos A_v and decreased by the natural death rate $\mu_v S_v$ and contact rate $K_v S_v$ between susceptible and exposed class such that

$$\dot{S}_v = A_v - K_v S_v - \mu_v S_v. \tag{7}$$

The rate of change of population of exposed mosquito is generated by the force of infection K_v and decreased by progression rate of mosquito σ_v and natural death rate of mosquito μ_v. Thus, it is given by

$$\dot{E}_v = K_v S_v - \sigma_v E_v - \mu_v E_v. \tag{8}$$

Finally, the rate of change of population of infected mosquito class I_v is generated by

$$\dot{I}_v = \sigma_v E_v - \mu_v I_v. \tag{9}$$

The notation

$$K_{ma} = \frac{\alpha a I_v}{N_h}, \tag{10}$$

where α is the biting rate of mosquitos and a is the transmission probability of malaria in pregnant women per bite, is introduced. Also

$$K_f = \frac{\beta(I_m + I_{mf} + kI_f)}{N_h}, \tag{11}$$

where b is the probability of transmission in mosquitos from any infected pregnant women, β is the effective contact rate of infection between the infective classes, while

$$K_v = \frac{\alpha b(I_m + \delta I_{mf})}{N_h}. \tag{12}$$

Here, δ is the modification parameter that takes into account the increase in the likelihood of infection of mosquitos from susceptible pregnant women with coinfection of malaria - filariasis in relation to acquiring infection from pregnant women with malaria only. Also, k is the modification parameter that accounts for the relative infectiousness of asymptomatically infected individual with filariasis exposed to malaria.

Assumptions in the model build-up are:

- The total population of pregnant women in the tropics is considered alone.

- There are birth and death rates.

- There is no vertical transmission between the mother and the child.

- Treatment is carried out for both pregnant women that are infected with malaria or filariasis.

- After treatment, immunity is lost, thereby making the pregnant women to be susceptible to the codisease again.

Hence the full malaria - filariasis model is given by

$$\dot{S}_h = \pi A_h - K_{ma}S_h - K_f S_h - (\mu_h + \delta)S_h + \tau T,$$
$$\dot{S}_2 = (1 - \pi)A_h + \delta S_h + K_{ma}S_h + K_f S_h - (\epsilon\sigma_h + \gamma + \mu_h)S_2,$$
$$\dot{I}_m = \epsilon\sigma_h S_2 - \theta K_f I_m - (\psi + \phi_1 + \mu_h)I_m,$$
$$\dot{I}_f = \psi I_m - \rho K_{ma}I_f - (\gamma + \phi_2 + \mu_h)I_f,$$
$$\dot{I}_{mf} = \theta K_f I_m + \rho K_{ma}I_f - (\phi_3 + \upsilon\phi + \eta\gamma + \mu_h)I_{mf},$$
$$\dot{T} = \upsilon\phi I_{mf} + (\phi_1 I_m + \phi_2 I_f + \phi_3 I_{mf}) - \mu_h T - \tau T,$$
$$\dot{S}_v = A_v - K_v S_v - \mu_v S_v,$$
$$\dot{E}_v = K_v S_v - (\sigma_v + \mu_v)E_v,$$
$$\dot{I}_v = \sigma_v E_v - \mu_v I_v, \tag{13}$$

subject to initial conditions $S_h(0) = S_{ho}$, $S_2(0) = S_{2o}$, $I_m(0) = I_{mo}$, $I_f(0) = I_{fo}$, $I_{mf}(0) = I_{mfo}$, $T(0) = T_o$, $S_v(0) = S_{vo}$, $E_v(0) = E_{vo}$, $I_v(0) = I_{vo}$.

2.1   Positivity and boundedness of solutions

The solutions of the malaria - filariasis model with nonnegative initial data are nonnegative for all time $t > 0$ [17, 19].

Theorem 2.1. *If* $S_h(0), S_2(0), I_m(0), I_f(0), I_{mf}(0), T(0), S_v(0), E_v(0), I_v(0)$ *are nonnegative, then* $S_h(t), S_2(t), I_m(t), I_f(t), I_{mf}(t), T(t), S_v(t), E_v(t), I_v(t)$ *are defined for all* $t > 0$, *such that*

$$\lim_{t \to \infty} \sup N_h(t) \leq \frac{A_h}{\mu_h} \tag{14}$$

and

$$\lim_{t \to \infty} \sup N_v(t) \leq \frac{A_v}{\mu_v}, \tag{15}$$

which implies that

$$N_h(0) \leq \frac{A_h}{\mu_h}, \quad N_v(0) \leq \frac{A_v}{\mu_v}. \tag{16}$$

The feasible region for (13) *is given by*

$$\xi = (\xi_h)(\xi_v) \subset \Re_+^6 \times \Re_+^3, \tag{17}$$

where

$$\xi_h = \Big[(S_h, S_2, I_m, I_f, I_m f, T) \in \Re_+^6 :$$

$$S_h + S_2 + I_m + I_f + I_{mf} + T \leq \frac{A_h}{\mu_h} \Big] \tag{18}$$

and

$$\xi_v = \Big[(S_v, E_v, I_v) \in \Re_+^3 : S_v + E_v : \leq \frac{A_v}{\mu_h} \Big] \tag{19}$$

show that ξ *is a positively invariant region.*

Proof. Since

$$S_h(0), S_2(0), I_m(0), I_f(0), I_{mf}(0), T(0), S_v(0), E_v(0), I_v(0) \qquad (20)$$

are nonnegative, where $t_1 > 0$, such that

$$t_1 = \sup[t > 0 : S_h, S_2, I_m, I_f, I_{mf}, T, S_v, E_v, I_v \text{ are nonnegative on } [0, t]]. \qquad (21)$$

If $t_1 < \infty$, from the first equation of (13), we have

$$S_h(t_1) = Z(t_1, 0)S_h(0) + \int_0^{t_1} A\Omega(t_1, \varsigma)d\varsigma, \qquad (22)$$

where

$$Z(t_1, \varsigma) = e^{-\int_\varsigma^t (K_{ma} + K_f + \mu_h + \delta)(s)ds}. \qquad (23)$$

Furthermore, the sum of the last three state equations in (13) is given by

$$\dot{N}_v = A_v - (S_v + E_v + I_v)\mu_v = A_v - N_v\mu_v \qquad (24)$$

so that,

$$N_v(t) = N_v(0)e^{-\mu_v t} + \frac{A_v}{\mu_v}(1 - e^{-\mu_v t}) \qquad (25)$$

and

$$\limsup_{t \to \infty} N_v(t) = \frac{A_v}{\mu_v}. \qquad (26)$$

From (26), it follows that

$$N_v(0) \le \frac{A_v}{\mu_v} \qquad (27)$$

and

$$N_v(0) \le \frac{A_v}{\mu_v}. \qquad (28)$$

Moreover, the addition of the first six state equations in (13) yields

$$N_h = A_h - (S_h + S_2 + I_m + I_n + I_{mf} + T)\mu_h - \gamma S_2 - \gamma I_f - \eta\gamma I_{mf}, \qquad (29)$$

so that, in the absence of disease-induced mortality, (29) becomes

$$\dot{N}_h \le A_h - \mu_h N_h, \qquad (30)$$

then

$$A_h - (\mu_h + \gamma + \eta)N_h \leq \dot{N}_h \leq A_h - \mu_h N_h. \tag{31}$$

Applying the standard comparison theorem method yields

$$N_h(0)e^{(\mu_h+\gamma+\eta t)} + \frac{A_h}{\mu_h + \gamma + \eta}(1 - e^{(\mu_h+\gamma+\eta)t}) \leq N_h \leq N_h(0)e^{-\mu_h t},$$

so that

$$N_h(0) \leq \frac{A_h}{\mu_h} \tag{32}$$

and

$$N_h(t) \leq \frac{A_h}{\mu_h}. \tag{33}$$

Thus, the malaria - filariasis model is epidemiologically feasible and mathematically well - posed. $\qquad\square$

3   Basic reproduction number (R_{mf})

The basic reproduction number (R_{mf}) is a measure of potential for the malaria - filaria disease spread in a susceptible host population of pregnant women. If $R_{mf} < 1$, few infectives introduced into the population will fail to replace themselves leading to the eradication of the disease. However, if $R_{mf} > 1$, the total number of infectives will increase which in turn leads to disease spread and mortality until optimum strategies are considered in order to curtail the spread of the disease. Also, if $R_{mf} < 1$, the disease-free equilibrium is locally asymptotically stable. There exists the endemic equilibrium if $R_{mf} > 1$.

Theorem 3.1. [24] *Let $x = (x_1, x_2, ..., x_n)^T$ be the number of individuals in each compartment, where the first $m < n$ compartments contain infected individuals.*

$$\frac{dx_i}{dt} = F_i(x) - V_i(x), \quad i = 1, 2, ...m,$$

where $f_i(x)$ is the rate of appearance of new infections in compartment i. $V_i(x)$ is the rate of either transition between compartment i and other infected compartments, such that

$$F = \left[\frac{\partial f_1(x_o)}{\partial x_j}\right], \quad V = \left[\frac{\partial V_i(x_o)}{\partial x_j}\right], \quad 1 \leq i, \ j \leq m,$$

F is entrywise nonnegative and V is a nonsingular M-matrix. Also, V^{-1} is entrywise nonnegative. Thus, the matrix (FV^{-1}) has $[i, j]$ entry equal to the expected number of secondary infections in compartment i produced by an infected individual introduced in compartment j. FV^{-1} is the next generation matrix and $R_0 = \rho(FV^{-1})$, where ρ is the spectral radius.

Proof. We use the next generation matrix method [24]. Suppose that

$$
F = \begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \beta & \beta k & \beta & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \theta\beta & \theta\beta k & \theta\beta & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & K_v & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}, \tag{34}
$$

$$
V = \begin{pmatrix}
J_1 & 0 & 0 & 0 & 0 & \tau & 0 & 0 & 0 \\
\delta & J_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \epsilon\sigma_h & J_3 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \psi & J_4 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & J_5 & 0 & 0 & 0 & 0 \\
0 & 0 & \phi_1 & \phi_2 & v\phi + \phi_3 & J_6 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & J_7 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & J_8 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_v & J_9
\end{pmatrix}, \tag{35}
$$

where $J_1 = (\mu_h + \delta)$, $J_2 = (\epsilon\sigma_h + \gamma + \mu_h)$, $J_3 = (\psi + \phi_1 + \mu_h)$, $J_4 = (\gamma + \phi_2 + \mu_h)$, $J_5 = (\phi_3 + v\phi + n\gamma + \mu_h)$, $J_6 = (\mu_h + \tau)$, $J_7 = \mu_v$, $J_8 = (\sigma_v + \mu_v)$, $J_9 = \mu_v$,

$$
R_{mf}(FV^{-1}) = \frac{\epsilon\sigma_h (J_6 J_1(\beta k\psi - \beta J_4) + \tau\delta(\psi\theta\beta k - \theta\beta J_4))}{\tau\delta(\psi\phi_2 - J_4\phi_3)\epsilon\sigma_h + J_1 J_2 J_3 J_4 J_5 J_6}. \tag{36}
$$

$\square$

4   Stability analysis of the model

4.1   Local stability analysis of disease-free steady state

Theorem 4.1. [25] *The disease-free equilibrium is asymptotically stable if $R_{mf} \leq 1$ and unstable if $R_{mf} > 1$.*

Proof. Firstly, we analyze the malaria - filaria codisease qualitatively to investigate the steady states of the model. We make the model system static by equating the right-hand side of the model to zero, that is, the system does not depend on time, such that

$$\dot{S_h} = \dot{S} = \dot{I_m} = \dot{I_f} = \dot{I_{mf}} = \dot{T} = 0, \ \ \dot{S_v} = \dot{E_v} = \dot{I_v} = 0. \tag{37}$$

Therefore, the steady states are given by

$$\left(S_h = \frac{\pi A_h}{\mu_h}, S_2 = \frac{A_h}{\mu_h}, I_m = 0, I_f = 0, I_{mf} = 0, T = 0, \right),$$

$$\left(S_v = \frac{A_v}{\mu_v}, E_v = 0, I_v = 0 \right). \tag{38}$$

The local stability of the system is determined by the signs of the eigenvalues and it is further proved by linearizing to obtain its Jacobian at disease-free steady-state points so that

$$J_{dfe} = \begin{pmatrix} -J_1 & 0 & 0 & 0 & 0 & \tau & 0 & 0 & 0 \\ \delta & -J_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \epsilon\sigma_h & -J_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \psi & -J_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -J_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_1 & \phi_2 & v\phi+\phi_3 & -J_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -J_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -J_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_v & -J_9 \end{pmatrix}. \tag{39}$$

The eigenvalues of the system above satisfy $-J_9 < 0, -J_8 < 0, -J_7 < 0, -J_5 <$

0, and the remaining characteristics polynomial is given as

$$\lambda^5 - (J_1 + J_2 + J_3 + J_4 + J_6)\lambda^4$$
$$+ (J_1J_2 + J_1J_3 + J_1J_4 + J_1J_6 + J_2J_3 + J_2J_4$$
$$+ J_2J_6 + J_3J_6 + J_4J_6)\lambda^3 - (J_1J_2J_3 + J_1J_2J_4 + J_1J_2J_6 + J_1J_3J_4$$
$$+ J_1J_3J_6 + J_1J_4J_6 + J_2J_3J_4 + J_2J_3J_6 + J_2J_4J_6 + J_3J_4J_6)\lambda^2$$
$$+ (-\delta\tau\phi_1\epsilon\sigma_h + J_1J_2J_3J_4 + J_1J_2J_3J_6 + J_1J_2J_4J_6$$
$$+ J_1J_3J_4J_6 + J_2J_3J_4J_6)\lambda$$
$$- (\phi_2\psi\epsilon\sigma_h\delta\tau + \phi_1\epsilon\sigma_h\delta\tau J_4 - J_1J_2J_3J_4, J_6(1 - R_{mf})). \quad (40)$$

Clearly, in the trace-determinant plane, the trace of (39) is

$$-(J_9 + J_8 + J_7 + J_6 + J_5 + J_4 + J_3 + J_2 + J_1) < 0 \quad (41)$$

and the determinant of (39) is

$$J_9J_8J_7J_5(-\delta\tau\phi_1\epsilon\sigma_h - \delta\tau J_4\phi_1\epsilon\sigma_h + J_1 + J_2 + J_3 + J_4 + J_6) > 0. \quad (42)$$

Hence by the Routh-Hurwitz criteria [25], all the eigenvalues have negative real parts, which proves that the disease-free equilibrium is locally asymptotically stable when $R_{mf} < 1$. □

4.2 Global stability of disease-free steady state

Theorem 4.2. [27, 28] *The disease-free steady state of system* (13) *is globally asymptotically stable whenever $R_{mf} < 1$.*

Proof. From the theorem of Castillo - Chavez et al. [27], given that

$$\dot{X} = F(X, Z),$$
$$\dot{Z} = G(X, Z), \quad (43)$$

where $X = (S_h, S_2, T, S_v)$ represents the total numbers of compartments without infections, such that $X \in \Re^{+4}$ and $Z = (I_m, I_f, I_{mf}, I_v, E_v)$ represents the total number of compartments with infections. The disease-free equilibrium solutions is denoted as $E^0 = (X^*, Z^*) = (X^*, 0)$, where $X^* = \left(\frac{A_h}{\mu_h}, \frac{\pi A_h}{\mu_h}, \frac{A_v}{\mu_v}\right)$. The following conditions hold:

i $\dot{X} = F(X, 0)$, X^* is globally asymptotically stable,

ii $G(X, Z) = D_Z(X^*, 0)Z - \hat{G}(X, Z) \geq 0$ for all $(X, Z) \in \Delta$, where Δ is a region where the model system (13) makes an epidemiological sense,

where $D_Z G(X^*, 0)$ denotes the Metzler matrix with nonnegative off-diagonal elements.

Therefore,

$$F(X, 0) = \begin{pmatrix} \pi A_h - (\mu_h + \delta)S_h + \tau T \\ (1 - \pi)A_h + \delta S_h - (\epsilon \sigma_h + \gamma + \mu_h)S_2 \\ -(\mu_h + \tau)T \\ \lambda_v - \mu_v S_v \end{pmatrix}, \tag{44}$$

and

$$G(X, Z) = \begin{pmatrix} -\theta I_m K_f - (\psi + \phi_1 + \mu_h)I_m \\ \psi I_m - \rho K_m I_f - (\gamma + \phi_2 + \mu_h)I_f \\ \theta K_f I_m + \rho K_m I_f - (\phi_3 + \upsilon \phi + \eta \gamma + \mu_h)I_{mf} \\ -(\sigma_v + \mu_v)E_v \\ -\mu_v I_v \end{pmatrix}. \tag{45}$$

In the absence of disease, it is clear that $G(X, 0) = 0$. Also

$$D_Z G(X^*, 0) = \begin{pmatrix} -m_1 & 0 & 0 & 0 & 0 \\ \psi & -m_2 & 0 & 0 & 0 \\ 0 & 0 & -m_3 & 0 & 0 \\ 0 & 0 & 0 & -m_4 & 0 \\ 0 & 0 & 0 & \sigma_v & -m_5 \end{pmatrix}, \tag{46}$$

where $m_1 = (\psi + \phi_1 + \mu_h)$, $m_2 = (\gamma + \phi_2 + \mu_h)$, $m_3 = (\phi_3 + \upsilon \phi + \eta \gamma + \mu_h)$, $m_4 = (\sigma_v + \mu_v)$, $m_5 = \mu_v$.

so that

$$\hat{G}(X, Z) = \begin{pmatrix} -\theta(I_m^* - I_m)K_f - m_1(I_m^* - I_m) \\ \psi(I_m^* - I_m^*) - \rho K_m(I_f^* - I_f) - m_2(I_f^* - I_f) \\ \theta K_f(I_m^* - I_m) + \rho K_m(I_f^* - I_f) - m_4(I_{mf}^* - I_{mf}) \\ -m_4(E_v^* - E_v) \\ -m_5(I_v^* - I_v) \end{pmatrix}. \tag{47}$$

From the first condition in the stated proof above, $S_h(t) = \frac{A_h}{\mu_h} + \left(S_h(0) - \frac{A_h}{\mu_h}\right)e^{-\mu_h t} \rightarrow \frac{A_h}{\mu_h}$ as $t \rightarrow \infty$, $S_2(t) = \frac{\pi A_h}{\mu_h} + \left(S_2(0) - \frac{\pi A_h}{\mu_h}\right)e^{-\mu_h t} \rightarrow \frac{\pi A_h}{\mu_h}$

as $t \to \infty$ and $S_v(t) = \frac{A_v}{\mu_v} + \left(S_v(0) - \frac{A_v}{\mu_v}\right)e^{-\mu_v t} \to \frac{A_v}{\mu_v}$ as $t \to \infty$. Since $X^* = \left(\frac{A_h}{\mu_h}, \frac{\pi A_h}{\mu_h}, \frac{A_v}{\mu_v}\right)$, X^* is globally asymptotically stable.

Also, if $I_m^* = I_m, I_f^* = I_f, I_{mf}^* = I_{mf}, E_v^* = E_v, I_v^* = I_v$, then the disease-free steady-state points denoted as $I_m^*, I_f^*, I_{mf}^*, E_v^*, I_v^*$, are all zeros. Then, $\hat{G}(X, Z) \geq 0$. Therefore, from the second condition in the stated theorem that $G(X, Z) = D_Z(X^*, 0)Z - \hat{G}(X, Z) \geq 0$, the disease-free steady state $E^0 = (X^*, 0)$ is globally asymptotically stable. □

4.3 Local stability of endemic steady state

The possibility of the existence of a backward bifurcation is investigated to establish the local stability of the endemic equilibrium of the system. If $R_{mf} < 1$, there is the possibility of a backward bifurcation. Also, $R_{mf} > 1$, the disease persists and become endemic and $R_{mf} = 1$ is the bifurcation point where a forward bifurcation occurs.

Theorem 4.3. [27, 28] *If $R_{mf} = 1$, then $R_{mf} = 1$ is the bifurcation value where the stability of the system changes.*

Proof. The center manifold theorem is employed, where the following change in notations of variables was made. Let $S_h = x_1, S_2 = x_2, I_m = x_3, I_f = x_4, I_{mf} = x_5, T = x_6, S_v = x_7, E_v = x_8, I_v = x_9$, and $\beta k = \beta k^*$ denote the bifurcation value and $R_{mf} = 1$ as the bifurcation point. That is,

$$1 = \frac{\tau\delta(\psi\phi_2 - J_4\phi_3)\epsilon\sigma_h + J_1 J_2 J_3 J_4 J_5 J_6}{\epsilon\sigma_h(J_6 J_1(\beta k^*\psi - \beta J_4) + \tau\delta(\psi\theta\beta k^* - \theta\beta J_4))}, \tag{48}$$

where $\beta k^* = \frac{1}{R_{mf}}$. The model of system (13) is redefined as

$$\dot{x}_1 = \pi A_h - K_{ma}x_1 - K_f x_1 - \delta x_1 + \tau x_6,$$

$$\dot{x}_2 = (1 - \pi)A_h + \delta x_1 + K_{ma}x_1 + K_f x_1 - (\epsilon\sigma_h + \gamma + \mu_h)x_2,$$

$$\dot{x}_3 = \epsilon\sigma_h x_2 - \theta K_f x_3 - (\psi + \phi_1 + \mu_h)x_3,$$

$$\dot{x}_4 = \psi x_3 - \rho K_{ma}x_4 - (\gamma + \phi_2 + \mu_h)x_4,$$

$$\dot{x}_5 = \theta K_f x_3 + \rho K_{ma}x_4 - (\phi_3 + \upsilon\phi + \eta\gamma + \mu_h)x_5,$$

$$\dot{x}_6 = \upsilon\phi x_5 + (\phi_1 x_3 + \phi_2 x_4 + \phi_3 x_5) - \mu_h x_6 - \tau x_6,$$

$$\dot{x}_7 = A_v - K_v x_7 - \mu_v x_7,$$

$$\dot{x}_8 = K_v x_7 - (\sigma_v + \mu_v)x_8,$$

$$\dot{x}_9 = \sigma_v x_8 - \mu_v x_9. \tag{49}$$

The Jacobian of the system (13) is given by

$$
\begin{pmatrix}
-J_1 & 0 & 0 & 0 & 0 & \tau & 0 & 0 & 0 \\
\delta & -J_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \epsilon\sigma_h & -J_3 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \psi & -J_4 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -J_5 & 0 & 0 & 0 & 0 \\
0 & 0 & \phi_1 & \phi_2 & v\phi + \phi_3 & -J_6 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -J_7 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -J_8 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_v & -J_9
\end{pmatrix}, \qquad (50)
$$

where $J_1 = (\mu_h+\delta)$, $J_2 = (\epsilon\sigma_h+\gamma+\mu_h)$, $J_3 = (\psi+\phi_1+\mu_h)$, $J_4 = (\gamma+\phi_2+\mu_h)$, $J_5 = (\phi_3 + v\phi + n\gamma + \mu_h)$, $J_6 = (\mu_h + \tau)$, $J_7 = \mu_v$, $J_8 = (\sigma_v + \mu_v)$, $J_9 = \mu_v$. We now proceed to calculate the left and right eigenvectors associated with (50). Let $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)$ be the right eigenvector associated with the matrix (50), then, we obtain

$$
w_1 = \frac{\tau}{(\mu + \delta)} w_6, \quad w_2 = \frac{(\psi + \phi_1 + \mu_h)}{\epsilon\sigma_h} w_3,
$$

$$
w_3 = \frac{(\gamma + \phi_2 + \mu_h)}{\psi} w_4, \quad w_4 = w_4 > 0,
$$

$$
w_5 = w_5 > 0, \quad w_6 = \frac{\phi_1}{\mu_h + \tau} w_4,
$$

$$
w_7 = 0, \quad w_8 = w_8 > 0, \quad w_9 = w_9 > 0. \qquad (51)
$$

Also, for $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)^T$, we obtain

$$
v_1 = \frac{\delta}{J_1} v_2, \quad v_2 = \frac{\epsilon\sigma_h v_3}{J_2}, \quad v_3 = v_3 > 0, \quad v_4 = \frac{\phi_2}{J_4} v_6,
$$

$$
v_5 = \frac{v\phi + \phi_3}{J_5} v_6, \quad v_6 = \frac{J_3 v_3}{J_4(\psi\phi_2 + \phi_1)}, \quad v_7 = 0, v_8 = 0, \quad v_9 = 0. \qquad (52)
$$

□

Next, the following theorem is used.

Theorem 4.4. $[19, 28]$ *Consider the general system of differential equations with a parameter μ such that*

$$
\frac{dx}{dt} = f(x, \mu), f : \Re^n \times \Re^+ \to \Re^+,
$$

and $f \in C^2(\Re^n \times \Re)$, and 0 is an equilibrium point of the system for all μ and

H1 $B = D_x f(0,0) = \frac{\partial f_i(0,0)}{\partial x_j}$ is the linearization matrix of the system around the equilibrium 0 with θ evaluated at 0.

H2 Zero is a simple eigenvalue of B and all other eigenvalues of B have negative real parts.

H3 The matrix B has a right eigenvector w and left eigenvector v corresponding to the zero eigenvalue.

If f_k is the kth component of f, then

i $a_o = \sum_{k,i,j=1}^n w_k \, v_i \, v_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j}$,

ii $b_o = \sum_{k,i,j=1}^n w_k \, v_i \, v_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \beta k^*}$,

the local dynamics of the system (13) at the equilibrium solutions is determined by the signs of a_o and b_o, especially, if $a_o > 0$ and $b_o > 0$, then a subcritical (or backward) bifurcation occurs at $\mu = 0$.

iii $a_o > 0, b_o > 0$: when $\mu < 0$ with $|\mu| << 1, 0$ is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \mu << 1, 0$ is unstable and there exists a negative and locally asymptotically stable equilibrium.

iv $a_o < 0, b_o < 0$: when $\mu < 0$ with $|\mu| << 1, 0$ is unstable; when $0 < \mu << 1, 0$ is unstable and there exists a positive and unstable equilibrium.

v $a_o > 0, b_o < 0$: when $\mu < 0$ with $|\mu| << 1, 0$ is stable and there exists a locally asymptotically stable negative equilibrium; when $0 < \mu << 1, 0$ is unstable and there exists a positive and unstable equilibrium.

vi $a_o < 0, b_o > 0$: when $\mu < 0$, if μ changes from negative to positive and 0 changes from stable to unstable, there exists a positive and unstable equilibrium. Also, a negative unstable equilibrium becomes positive and locally asymptotically stable if $a_o > 0$ and $b_o > 0$, then a backward bifurcation occurs at $\mu = 0$.

Computations of a_o and b_o

To compute for a_o for the transformed system of (50), the associated nonzero partial derivatives of f (evaluated at the disease-free steady state) are given by

$$\dot{x}_1 = f_1(x_1, x_2, ..., x_9) = \pi A_h - K_{ma}x_1 - K_f x_1 - \delta x_1 + \tau x_6,$$
$$\dot{x}_2 = f_2(x_1, x_2, ..., x_9) = (1-\pi)A_h + \delta x_1 + K_{ma}x_1 + K_f x_1$$
$$- (\epsilon\sigma_h + \gamma + \mu_h)x_2,$$
$$\dot{x}_3 = f_3(x_1, x_2, ..., x_9) = \epsilon\sigma_h x_2 - \theta K_f x_3 - (\psi + \phi_1 + \mu_h)x_3,$$
$$\dot{x}_4 = f_4(x_1, x_2, ..., x_9) = \psi x_3 - \rho K_{ma}x_4 - (\gamma + \phi_2 + \mu_h)x_4,$$
$$\dot{x}_5 = f_5(x_1, x_2, ..., x_9) = \theta K_f x_3 + \rho K_{ma}x_4 - (\phi_3 + \upsilon\phi + \eta\gamma + \mu_h)x_5,$$
$$\dot{x}_6 = f_6(x_1, x_2, ..., x_9) = \upsilon\phi x_5 + (\phi_1 x_3 + \phi_2 x_4 + \phi_3 x_5) - \mu_h x_6 - \tau x_6,$$
$$\dot{x}_7 = f_7(x_1, x_2, ..., x_9) = A_v - K_v x_7 - \mu_v x_7,$$
$$\dot{x}_8 = f_8(x_1, x_2, ..., x_9) = K_v x_7 - (\sigma_v + \mu_v)x_8,$$
$$\dot{x}_9 = f_9(x_1, x_2, ..., x_9) = \sigma_v x_8 - \mu_v x_9, \tag{53}$$

where

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_9} = -\alpha a, \quad \frac{\partial^2 f_1}{\partial x_1 \partial x_3} = -\beta = \frac{\partial^2 f_1}{\partial x_5 \partial x_1}, \quad \frac{\partial^2 f_1}{\partial x_4 \partial x_1} = -\beta k,$$

$$\frac{\partial^2 f_2}{\partial x_4 \partial x_1} = \beta k, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_9} = \alpha a, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_3} = \beta = \frac{\partial^2 f_2}{\partial x_5 \partial x_1}, \quad \frac{\partial^2 f_4}{\partial x_9 x_4} = -\rho\alpha a,$$

$$\frac{\partial^2 f_5}{\partial x_3{}^2} = \theta\beta = \frac{\partial^2 f_5}{\partial x_5 x_3}, \quad \frac{\partial^2 f_5}{\partial x_4 \partial x_3} = \theta\beta k, \quad \frac{\partial^2 f_5}{\partial x_5 x_4} = \rho\alpha a,$$

$$\frac{\partial^2 f_7}{\partial x_3 x_7} = -\alpha b, \quad \frac{\partial^2 f_7}{\partial x_5 x_7} = -\alpha b\delta, \quad \frac{\partial^2 f_8}{\partial x_3 x_7} = \alpha b, \quad \frac{\partial^2 f_8}{\partial x_5 x_2} = \alpha b\delta. \tag{54}$$

A direct calculation from the expression above yield

$$a_o = v_1 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_1}{\partial x_i \partial x_j} + v_2 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}$$
$$+ v_3 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_3}{\partial x_i \partial x_j} + v_4 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_4}{\partial x_i \partial x_j}$$

$$+v_5 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j} + v_6 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_6}{\partial x_i \partial x_j}$$

$$+v_7 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_7}{\partial x_i \partial x_j} + v_8 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_8}{\partial x_i \partial x_j}$$

$$+v_9 \sum_{i,j=1}^{9} w_i w_j \frac{\partial^2 f_9}{\partial x_i \partial x_j}. \tag{55}$$

Since $v_3 = v_7 = v_8 = v_9 = 0$ in (52), after some re-arrangement and simplification, (54) and (55) yield

$$a_o = v_1(-\alpha a w_1 w_9 - \beta(w_1 w_3 + w_1 w_5) - \beta k(w_1 w_4)$$

$$+v_2(\alpha a w_1 w_9 + \beta(w_1 w_3 + w_1 w_5) - \beta(w_1 w_3 + w_1 w_5) + \beta k(w_1 w_4) - v_4(\rho \alpha a(w_4 w_9)))$$

$$+v_5(\theta\beta(w_3{}^2 + w_1 w_5) + \theta\beta k(w_3 w_4) + \rho\alpha a(w_4 w_9)) > 0. \tag{56}$$

To compute for b_o for the transformed system of (50), the associated nonzero partial derivatives of f (evaluated at the disease-free steady state) are given by

$$\frac{\partial^2 f_2}{\partial x_4 \partial \beta k^*} = \beta, \quad \frac{\partial^2 f_5}{\partial x_3 \partial \beta k^*} = \theta\beta, \tag{57}$$

so that

$$b_o = v_2 w_4 \frac{\partial^2 f_2}{\partial x_4 \partial \beta k^*} + v_5 w_3 \frac{\partial^2 f_5}{\partial x_3 \partial \beta k^*}, \tag{58}$$

$$b_o = \beta(v_2 w_4) + \theta\beta(v_5 w_3) > 0. \tag{59}$$

Since $a_o > 0$, $b_o > 0$, then the model system (13) will undergo a subcritical bifurcation, otherwise it will exhibit a forward bifurcation. Hence the endemic equilibrium is locally asymptotically stable.

5   Numerical simulations

Table 1. Variables in Model (13) and Their Meanings

Variable	Descriptions	Values	Source
$S_h(0)$	Antenatal compliant susceptible pregnant women	50	Assumed
$S_2(0)$	Antenatal noncompliant susceptible pregnant women	30	Assumed
$I_m(0)$	Malaria infected pregnant women	10	Assumed
$I_f(0)$	Filariasis infected pregnant women	15	Assumed
$I_{mf}(0)$	Malaria - filariasis infected pregnant women	20	Assumed
$T(0)$	Treatment of malaria - filariasis infected pregnant women	10	Assumed
$S_v(0)$	Susceptible mosquitos	25	Assumed
$E_v(0)$	Exposed mosquitos	20	Assumed
$I_v(0)$	Infected mosquitos	10	Assumed

Table 2. Parameter Values and Descriptions

Parameters	Symbols	Values	Source
Recruitment rate of susceptible pregnant women	πA_h	4.21×10^{-5}	[19]
Per capita birth rate of mosquitos	λ_v	1000	Varies
Per capita natural mortality rate of pregnant women	μ_h	0.00004	Assumed
Per capita natural mortality rate of mosquitos	μ_v	0.05	[16]
Proportion of noncompliant susceptibles	$(1-\pi)A_h$	3×10^{-5}	Varies
Progression rate from compliant to noncompliant class	δ_1	0.12	Assumed
Rate of loss of immunity to both malaria and filarisis	τ	0.0006	Assumed
Rate of malaria symptoms in pregnant women	σ_h	0.8331	[16, 23]
Modification parameter	ϵ	0.7	Assumed
Modification parameter	θ	1.2	Assumed
Modification parameter	ρ	1.4	Assumed
Modification parameter	δ	0.00211	Assumed
Modification parameter	k	0.011	Assumed
Modification parameter	η	0.003	Assumed
Modification parameter	υ	0.0036	Assumed
Disease induced death rate of the codisease	γ	0.00231	[19]
Progression rate of malaria leading to filariasis symptoms	ψ	1.0	[23]
Biting rate of mosquitos	α	0.2	[19]
Transmission probability of malaria in mosquitos	b	0.09	Varies
Transmission probability of malaria in humans	a	0.8333	[16]
Rate of mosquito bite leading to malaria infection	βI_m	0.0183	[16, 20]
Rate of mosquito bite leading to filariasis infection	βI_f	3.6×10^{-5}	[23]
Rate of mosquito bite leading to codisease infection	βI_{mf}	2.7×10^5	[23]
Treatment rate of malaria	ϕ_1	0.00341	Assumed
Treatment rate of filariasis	ϕ_2	0.00061	Assumed
Treatment rate of malaria - filariasis	ϕ_3	0.00072	Assumed
Transmission rate between susceptible and infected mosquitos	K_v	0.502	[23]
Natural death rate of mosquitos	μ_v	0.1429	[16, 23]
Progression rate of exposed to infected mosquitos	σ	0.0112	Assumed

Figure 1 shows the profile of the pregnant women treated from malaria - filariasis coinfection by varying ϕ_3 between $0.00021 - 0.00072$. The decline in the graph shows that use of drugs, e.g., ACT and Mectizan, will bring about the drastic
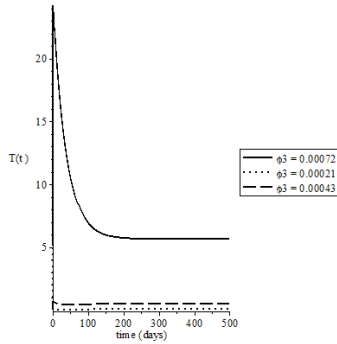
reduction of the codisease in their system.

Figure 1. Graph of Disease Profile of T against Time (t)

Figure 2 presents the disease profile of pregnant women infected with malaria - filariasis. The rise in the graph shows that, in the absence of medical interventions, there will be a full blown epidemic leading to mortality and morbidity.
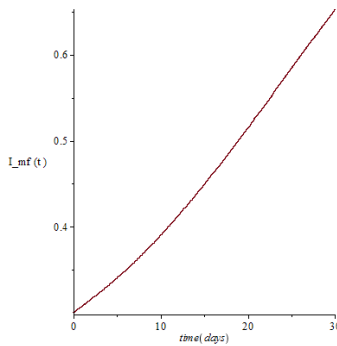

Figure 2. Graph of I_{mf} against Time (t)

Figure 3 shows the disease profile of the susceptible pregnant women non compliant to medical antenatal program. The rise in the graph depicts the group having a high tendency of contacting the malaria - filariasis coinfection.
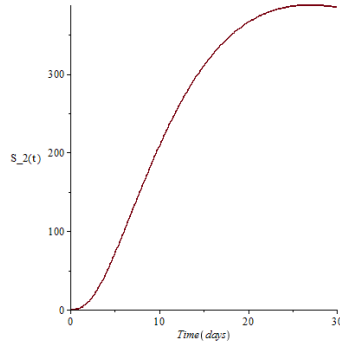
Figure 3. Graph of S_2 against Time (t)

Figure 4 presents the variation of the loss of immunity rate τ between $0.0002 - 0.0006$. The decline shows that after appropriate treatment, they lose immunity overtime, thereby making them susceptible to the coinfection.
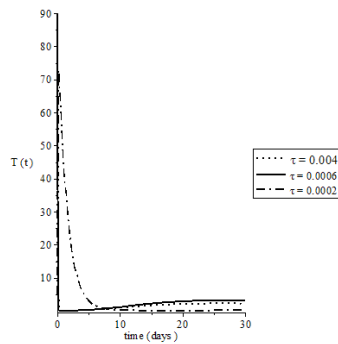

Figure 4. Graph of T against Time (t)

Figure 5 illustrates the disease profile's number of deaths of Pregnant women infected with malaria and filariasis in the absence of medical interventions, varying the disease induced death rate γ between $0.00176 - 0.00231$.
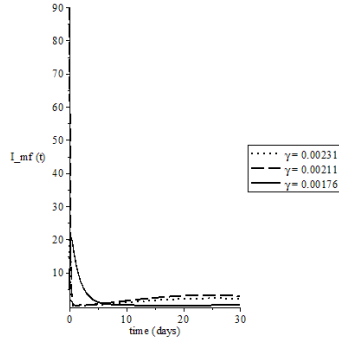
Figure 5. Graph of I_{mf} against Time (t)

Figure 6 shows the S_h group of pregnant women who are susceptible to malaria - filariasis coinfections but are still compliant to antenatal medical program. The gradual rise and decline on the graph shows that the coinfections will be minimized overtime with their compliance to antenatal program.
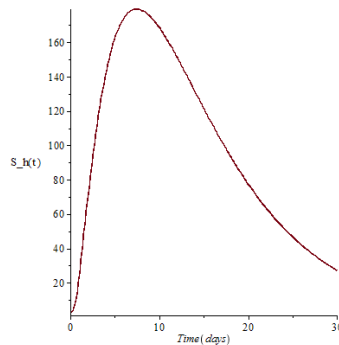

Figure 6. Graph of S_h against Time (t)

6 Conclusion and recommendations

A deterministic model describing the transmission of malaria - filariasis coinfections in susceptible host of pregnant women population is considered. A domain where the model is well posed and realistic in an epidemic sense is presented. Also, the basic reproduction number of malaria - filariasis coinfections (R_{mf}) is obtained and the local and global behavior is analyzed. It was investigated that if $R_{mf} < 1$, malaria - filariasis coinfections vanish out of the population, and if $R_{mf} > 1$, the coinfections persist and become endemic. However, variations

in the values of state variables and parameters involved in the model formulation bring about the need for medical intervention strategies like treatment, to curtail the spread of the coinfectious disease. This work can be further extended by considering the impact of climatic factors, optimal controls, cost effective strategies.

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