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A Mathematical Modelling of Lymphatic Filariasis and Malaria Co-infection

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ABSTRACT
Lymphatic Filariasis (LF) and Malaria continue to pose significant public health burden globally and are co-endemic in many sub-Saharan African regions. In this work, we developed and analyzed a mathematical model of Lymphatic filariasis and malaria co-infection model. Friedman and Lunge method was used to find the positivity of the solution, the disease-free equilibrium was obtained, the model stability was analyzed, and the basic reproductive number was also obtained. The findings suggest that with the use of a bed-net and insecticide as a control measure, the treatment of LF and malaria co-infection can be reduced to a minimum.

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INTRODUCTION

Statistics reveal that the goal of eradicating parasitic diseases like lymphatic filariasis and malaria has not been entirely realized; instead, these two old diseases, particularly lymphatic filariasis (also known as elephantiasis), have been neglected or completely ignored. Lymphatic filariasis and malaria are two vector-borne diseases that account for the largest morbidity and mortality rates globally. Lymphatic filariasis was derived from the Latin word 'filarial', it is a vector-borne disease of human-caused by Wuchcreria Banocrofti, Brugia and skin dwelling Onchocerca volvulus (Ottesen, 1984). Among these three species, Wuchcreria Banocrofti is the most common and accounts for 90% of infection globally (Anosike et al, 2005) and over 120 million people are infected with filariasis, claiming over 40 million people with Africa accounting for 40% of all the global cases (Lenhart et al, 2007).

Despite the increase in the literacy level of the pathology of lymphatic filariasis and the efforts made to treat this disease with diethylcarbamazine and albendazole, filariasis continues to pose a major public health threat in tropical and subtropical regions. This is because the disease is more prevalent in the region with a higher incidence of poverty (Tan, 2013) and making it a disease of the poor and most often serves as an indicator of underdevelopment (WHO, 1993). It is also noted that areas with a higher prevalence of malaria and filariasis have poor environmental and settlement planning and other activities that favour the breeding reservoirs for mosquito vectors (Haddix *et al.,* 2000, Ngwa, 2004). Coincidentally, both malaria and filariasis have feverish symptoms. In rural areas, it is often hard to distinguish between drug-resistant falciparum malaria and periodic fever due to filariasis infection (Ojiako *et al.,*2009). In order to uncover solutions to control diseases in the population, several studies have been undertaken using a mathematical model method. (Ayoola *et al.,* 2021, Peter *et al.,* 2018, Peter *et al.,* 2019, Peter *et al.,* 2020, Peter *et al.,* 2021a, Peter *et al.,* 2021b, Peter *et al.,* 2021c, Peter *et al.,* 2022, Abioye *et al.,* 2018, Peter *et al.,* 2021, Peter *et al.,* 2020, Ojo *et al.,* 2022)

The mathematical modelling of this disease has been a subject of research by scientists in recent times and we discuss some papers in this direction. A program to simulate the modelling of LF disease and its control was called LYMFASIM was proposed by Plaisier *et al,* 1998. The framework is flexible and can be adapted to the modelling of LF disease. Norman *et al.,*2000 presented another framework called EPIFIL which take into account the age structure of the population of LF disease and it was concluded in

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this research that this analytical tool gives an effective result for controlling LF disease.

A mathematical model for studying the transmission dynamics of LF that improves on previous studies of LF was studied. In the work, the existence and uniqueness of the model were discovered, and stability analyses were also obtained (Oguntolu (2019), Oguntolu *et al,* 2021). Gweryina and Tersoo (2014) developed a deterministic model for malaria-filariasis co-infection with chemoprevention and treatment is formulated and analyzed. Furthermore, results from numerical simulation suggest that high chemotherapy and treatment hold great promise for helping to stem the tide of new malaria and filariasis infections.

The interactions between malaria-LF co-infection were studied by (Slater *et al,* 2013) and the results also highlight the potential perverse effects of vector controls on human-human infection prevalence in co-endemic regions. (Mwamtobe *et al,* 2017) derived the basic reproduction number and its interpretation suggests that LF can be best-treated fatter using quarantine. Swaminathan *et al,* 2008 proposed a mathematical model to look deeply into the transmission and control of LF. They gave an analysis of some of the challenges being faced in order to combat this disease. Das and Subramanian (2002) provide an overview of the development of the relevant mathematical and statistical models applicable to the transmission and control of LF outbreaks.

Due to some of the gaps we observed in previous research, our goal is to present a mathematical model that incorporates relevant features such as chemoprevention, treatment, infected humans at the acute stage, chronic stage, filariasis, the use of bed net and insecticide.

The remainder of the article is divided as follows: Section 2 describes the model development, Section 3 presents the mathematical analysis, Section 4 displays the results derived from this article, and Section 5 & 6 presents the discussion of the results and conclusion from the article.

MODEL DEVELOPMENT

Model formulation and description

A mathematical model provides a framework within which we can communicate an understanding of the spread of disease in the human population, both in space and time. In this chapter, we developed and analyzed a mathematical model and optimal control of malaria and Lymphatic filariasis co-infection. The model incorporates relevant features such as chemoprevention, treatment, infected humans at the acute stage, chronic stage, filariasis, the use of bed net and insecticide.

The Human population is sub diving into the susceptible $\left(S_h\left(t\right)\right)$, the malaria-infected class $\left(I_m\left(t\right)\right)$,the infected acute stage $\left(I_{hal}\right)$, the infected chronic stage $\left(I_{hcl}\right)$, the Lymphatic filariasis acid alaria co-infected class, the treatment class and the chemoprevention class $\left(V_h\left(t\right)\right)$ d the mosquito population is subdivided into two classes the susceptible vector and the infected vector class

The filariasis-malaria co-infection transmission model has been modeled based on the epidemiology aspects of two diseases.
The mosquitoes and Human beings are recruited into their susceptible corresponding populations at rate Λ_v and Λ_h res pectively. Mosquito experiences natural death rate μ_v and death by insecticide at a rat δ_v which is proportional to the number in each mosquito class. Similarly, human beings experience a natural death μ_h, which is proportional to the number in each human class.

The mosquito ingests microfilarias or malaria parasite or both when it bites a human who is infected with filariasis or malaria or both. This filariasis could be at the acute stage or chronic stage in the human at the rate

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$$\lambda_h = \beta_h \sigma_v \frac{\left(I_{hal} + I_{hcl} + I_m + I_{lm}\right)}{N_h}$$

The meaning of and with the other parameters are given in the definition of the model parameters. For filariasis infection, upon getting infected, susceptible mosquitoes enter the infected class $I_v(t)$. The filari form juveniles escape from the mosquito's proboscis when the insect is feeding and penetrates the wound structure of human beings.

There is an interaction between the infected vector and the chemoprevention, the susceptible human and the infected vector. The susceptible vector and ria-infected tend human, susceptible vector and both the acute and chronic stage of the infected humans with filariasis, Lymphatic and malaria co-infected humans. In Figure 1, we present the graphical representation of the model formulation and interactions between sub-population.

Model equations

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \sigma_v(1-\theta)(\beta_m + \beta_l + \beta_{lm})\frac{I_v S_h}{N_v} - (\tau_1 + \mu_h)S_h + \alpha_1 T_h + \alpha_2 V_h \\
\frac{dV_h}{dt} &= \tau_1 S_h - (\mu_h + \alpha_2)V_h - \beta_m \sigma_v \frac{I_v V_h}{N_v} \\
\frac{dI_{hal}}{dt} &= \beta_l \sigma_v (1-\theta)\frac{I_v S_h}{N_v} - \beta_l \sigma_v (1-\theta)\frac{I_v I_{hal}}{N_v} - (\mu_h + \tau_1 + \rho)I_{hal} \\
\frac{dI_{hcl}}{dt} &= \beta_l \sigma_v (1-\theta)\frac{I_v I_{hal}}{N_v} + \rho I_{hal} - (\mu_h + \tau_1)I_{hcl} \\
\frac{dI_m}{dt} &= \beta_m \sigma_v (1-\theta)\frac{I_v S_h}{N_v} - (\tau_2 + \delta_m + \mu_h)I_m + \beta_m \sigma_v \frac{I_v V_h}{N_v} \\
\frac{dI_{lm}}{dt} &= \beta_{lm} \sigma_v (1-\theta)\frac{I_m S_h}{N_v} - (\tau_3 + \delta_m + \mu_h)I_{lm} \\
\frac{dT_h}{dt} &= \tau_2 I_m + \tau_1 (I_{hal} + I_{hcl}) + \tau_3 I_{lm} - (\alpha_1 + \mu_h)T_h \\
\frac{dS_v}{dt} &= \Lambda_v - \beta_h \sigma_v \frac{(I_{hal} + I_{hcl} + I_{lm} + I_m)S_v}{N_h} - (\mu_v + \delta_v)S_v \\
\frac{dI_v}{dt} &= \beta_h \sigma_v \frac{(I_{hal} + I_{hcl} + I_{lm} + I_m)}{N_h} - (\mu_v + \delta_v)I_v
\end{aligned} \tag{1}$$

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Table 1: Definition of variable/Parameter of the model

Variable	Description
$S_h(t)$	A class of susceptible Human
$V_h(t)$	A class of susceptible individuals taking drugs (chemoprevention) at time (t)
$I_{hal}(t)$	A class of infected acute stage of LF (not showing sign of Lymphatic filariasis) at time (t)
$I_{hcl}(t)$	A class of infected-chronic stage of LF at time (t)
$I_m(t)$	A class of malaria infected human at time (t)
$I_{lm}(t)$	A class of Lymphatic filariasis and malaria co-infected humans at time t
$T_h(t)$	Recovered human population at time t
$S_v(t)$	Susceptible mosquitoes at time t
$I_v(t)$	Infected mosquitoes at time t
β_h	The infectivity of an infection malaria, Lymphatic filariasis and co-infection humans defined as the probability that a bite by a susceptible mosquitoes on an infected human with transfer the infection to the mosquito
β_m	The infectivity of the mosquito, define as the probability that a bite by an infected mosquito on a susceptible human will transfer malaria infection to the Human
β_l	The infectivity of the mosquito, defined as the probability that a bite by an infected mosquito on a susceptible human will transfer Lymphatic filariasis infection to the Human
β_{lm}	The infectivity of the mosquito, defined as the probability that a bite by an infected mosquito on a susceptible human will transfer Lymphatic filariasis infection to the Human
σ_v	The main biting rate of mosquitoes, define as the average number of bites given to humans by each mosquito per unit time
Λ_h	Recruitment rate of the human population
Λ_v	Recruitment rate of the mosquito population
θ	Proportion of the susceptible population using mosquito net and insecticide
μ_h	Natural death rate for the human population
μ_v	Natural death rate for the mosquito population
δ_m	Death rate due to malaria infection
ρ	Rate of progression of human from $I_{hal}(t)$ to $I_{hcl}(t)$

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δ_v	Mosquitoes death rate due to the use of insecticide
τ_1	Treatment rate for Lymphatic filariasis infected individuals
τ_2	Treatment rate for malaria infected individuals
τ_3	Treatment rate for Lymphatic filariasis and malaria co-infected individuals
α_1	Progression rate at which malaria, Lymphatic filariasis and co-infected Lymphatic filariasis maleness full recovered human after treatment move to susceptible class
α_2	Rate at which the treatment immunity wanes off

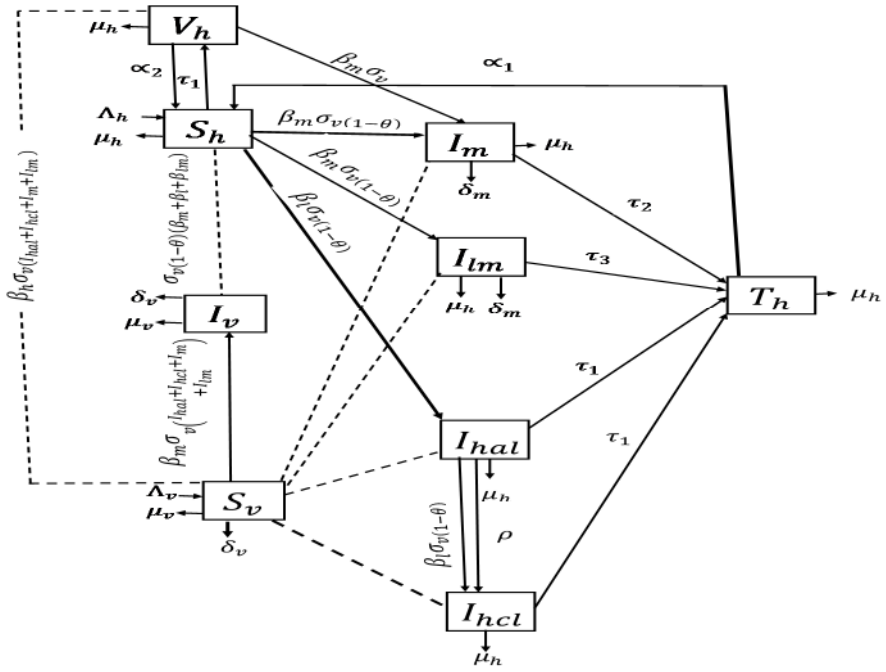

Figure 1. Schematic diagram of the model

MATHEMATICAL ANALYSIS

Positivity of the Solutions

In this subsection, since the model consist of both human and vector classes, we employ the technique of Friedman and Lungu to demonstrate that the model equation is positively invariant and well posed. We consider the system in matrix form

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$$\begin{pmatrix} \dot{S}_h \\ \dot{V}_h \\ \dot{I}_{hal} \\ \dot{I}_{hcl} \\ \dot{I}_m \\ \dot{I}_{cm} \\ \dot{T}_h \\ \dot{S}_v \\ \dot{I}_v \end{pmatrix} = \begin{pmatrix} -k_1 & \alpha_2 & 0 & 0 & 0 & 0 & \alpha_1 & 0 & 0 \\ \tau_1 S_h & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \rho & -k_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -k_5 & 0 & 0 & 0 \\ 0 & 0 & \tau_1 & \tau_1 & \tau_2 & \tau_3 & -k_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -k_7 - \omega_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_5 & -k_7 \end{pmatrix} \begin{pmatrix} \dot{S}_h \\ \dot{V}_h \\ \dot{I}_{hal} \\ \dot{I}_{hcl} \\ \dot{I}_m \\ \dot{I}_{cm} \\ \dot{T}_h \\ \dot{S}_v \\ \dot{I}_v \end{pmatrix} +$$

$$\begin{pmatrix} \Lambda_h - \omega_1 I_v S_h \\ -\frac{\beta_m \sigma_v}{N_v} I_v V_h \\ \omega_2 I_v S_h - \omega_2 I_v I_{hal} \\ \omega_2 I_v I_{hal} \\ \frac{\beta_m \sigma_v}{N_v} I_v V_h + \omega_3 I_v S_h \\ \omega_4 I_v S_h \\ 0 \\ \Lambda_v \\ 0 \end{pmatrix}$$

which in compact form can be written as :

$$\frac{dx_i}{dt} = f(x,t) = A_i x_i + H(x_i)$$

For $x = (x_1, x_2, ..., x_n)$ where $x_1 = S_h$, $x_2 = V_h$, $x_3 = I_{hal}$, $x_4 = I_{hcl}$, $x_5 = I_m$, $x_6 = I_{lm}$ $x_7 = T_h$, $x_8 = S_v$, $x_9 = I_v$ and $(.)'$ denote transpose. Then it can be written as

$$\frac{dx_i}{dt} = f(x,t)$$

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For $x=\left(x_1,x_2,...,x_n\right)'$. One can easily show that the equation satisfies the differential inequality

$$\frac{dx_i}{dt} = A_i x_i + \sum_{v=1}^{n} C_{ij} x_i + \varepsilon$$

For $i=1,2,3,...,n$ with $C_{ij} \geq 0$ and $t \succ 0$ if $x_i\left(0\right) \geq \varepsilon$

For $i=1,2,3,...,n$ then $x_i\left(t\right) \geq \varepsilon$ for all $t \geq o$

Proof

Assuming without loss of generality that $\mathcal{E} > 0$. The case $\mathcal{E}$ =0 is trivial through approximation with a sequence $\varepsilon = \varepsilon_k$, which converges to zero as k goes to infinity.

Suppose now that $x_i\left(0\right) \geq \varepsilon \succ 0$, for $1 \leq i \leq n$ does not hold, then there exist $t_0 \succ 0$ such that $x_i\left(t\right) \geq 0$ for $1 \leq i \leq n$, $0 \leq t \prec t_0$ and $x_i\left(t_0\right) = 0$ for at least one i, say $i = i_0$.

Then $x\left(i_0\right)$ is a decreasing function such that $\frac{dx_{i_0}}{dt}\left(t_0\right) \leq 0$

From the differential $\frac{dx_{i_0}}{dt} \geq A_i x_{i_0} + \sum_{j=1}^{n} C_{ij} x_{i_0} + \varepsilon$ which is a contradiction. Thus, if $x_i\left(0\right) \geq \varepsilon$

For $i=1,2,3,...,n$ then $x_i\left(t\right) \geq 0$ for all $t \geq 0$, since this hold hence we've showed that the equations are all positive.

Basic Reproductive Number (R_c)

In the model equation (1), the infectious compartments includes

I_{hal}, I_{hcl}, I_m, I_{lm}, T_h, I_v, S_v and the expected secondary infections depends on these classes. The rate of appearance of new infections in the compartment is given by the matrix.

The Jacobean matrix of F evaluated at the disease free equilibrium point is given by:

$$F = \left(\frac{\partial f_i\left(E^0\right)}{\partial x_i} \right) \quad x_j = I_{hal}, I_{hcl}, I_m, I_{lm}, I_v \text{ for } j=1,2,3,4 \text{ and } E^0 \text{ is the}$$

disease free equilibrium.

The jacobian of Infection term matrix is given as

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$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\beta_l \sigma_v (1-\theta) S_h^*}{N_v} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_m \sigma_v (1-\theta) S_h + \beta_m \sigma_v V_h}{N_v} \\ 0 & 0 & 0 & 0 & \frac{\beta_{lm} \sigma_v (1-\theta) S_h^*}{N_v} \\ \frac{\beta_h \sigma_v S_v}{N_h} & \frac{\beta_h \sigma_v S_v}{N_h} & \frac{\beta_h \sigma_v S_v}{N_h} & \frac{\beta_h \sigma_v S_v}{N_h} & 0 \end{pmatrix}$$

and the jacobian of transmission term matrix is

$$V = \begin{pmatrix} k_3 & 0 & 0 & 0 & 0 \\ -\rho & k_6 & 0 & 0 & 0 \\ 0 & 0 & k_4 & 0 & 0 \\ 0 & 0 & 0 & k_5 & 0 \\ 0 & 0 & 0 & 0 & k_7 \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} -\lambda & 0 & 0 & 0 & \frac{\beta_l \sigma_v (1-\theta) S_h}{k_7 N_v} \\ 0 & -\lambda & 0 & 0 & 0 \\ 0 & 0 & -\lambda & 0 & \frac{\beta_m \sigma_v (1-\theta) S_h}{k_7 N_v} + \frac{\beta_m \sigma_v V_h}{k_7 N_v} \\ 0 & 0 & 0 & -\lambda & \frac{\beta_{lm} \sigma_v (1-\theta) S_h}{k_7 N_v} \\ \frac{\beta_h \sigma_v S_v}{k_1 k_3 N_h} + \frac{\beta_h \sigma_v S_v \rho}{k_1 k_3} & \frac{\beta_h \sigma_v S_v}{k_7 N_h} & \frac{\beta_h \sigma_v S_v}{k_4 N_h} & \frac{\beta_h \sigma_v S_v}{k_5 N_h} & -\lambda \end{pmatrix}$$

$$\left.\begin{aligned} \lambda_1 &= 0 \\ \lambda_2 &= 0 \\ \lambda_3 &= 0 \\ \lambda_4 &= 0 \end{aligned}\right\}$$

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$$\lambda_5 = \sqrt{\frac{\beta_h\beta_{lm}\sigma_v\sigma_v k_1k_2k_3k_4(1-\theta)+\beta_h\beta_m\sigma_v\sigma_v k_1k_2k_3k_5(1-\theta)+\beta_h\beta_m\sigma_v\sigma_v\tau_1k_1k_3k_4+\beta_h\beta_l\sigma_v\sigma_v k_2k_4k_5(1-\theta)(k_1+\rho)}{k_1k_3k_4k_5k_7(k_2+\tau_1)}}$$

$$\lambda_6 = -\sqrt{\frac{\beta_h\beta_{lm}\sigma_v\sigma_v k_1k_2k_3k_4(1-\theta)+\beta_h\beta_m\sigma_v\sigma_v k_1k_2k_3k_5(1-\theta)+\beta_h\beta_m\sigma_v\sigma_v\tau_1k_1k_3k_4+\beta_h\beta_l\sigma_v\sigma_v k_2k_4k_5(1-\theta)(k_1+\rho)}{k_1k_3k_4k_5k_7(k_2+\tau_1)}}$$

Clearly, we can see that λ_5 is the dominant Eigen values.

$$R_c = \sqrt{\frac{\beta_h\beta_{lm}\sigma_v\sigma_v k_1k_2k_3k_4(1-\theta)+\beta_h\beta_m\sigma_v\sigma_v k_1k_2k_3k_5(1-\theta)+\beta_h\beta_m\sigma_v\sigma_v\tau_1k_1k_3k_4+\beta_h\beta_l\sigma_v\sigma_v k_2k_4k_5(1-\theta)(k_1+\rho)}{k_1k_3k_4k_5k_7(k_2+\tau_1)}}$$

.***Disease Free Equilibrium State (DFE)***

Disease free equilibrium states are steady when all the infectious classes in a population are zero, that is;

at Disease Free Equilibrium;

$$E^0 = \left(S_h, V_h, \mathrm{I}_{hal}, \mathrm{I}_{hcl}, I_m, \mathrm{I}_{lm}, T_h, S_v, \mathrm{I}_v\right) = \left(\frac{\Lambda_h k_2}{k_1k_2-\alpha_2\tau_1}, \frac{\tau_1\Lambda_h}{k_1k_2-\alpha_2\tau_1}, 0,0,0,0,0, \frac{\Lambda_h}{k_7}, 0\right).$$

Local stability of disease-free equilibrium state.

The Disease-free equilibrium E^0 of the model equation (1) is locally asymptotically stable (LAS) of R_c <1.

Proof: Linearization of the model (1) at any arbitrary equilibrium point (E^0) gives the Jacobian

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$$J(E^0)=\begin{pmatrix} -k_1 & \alpha_2 & 0 & 0 & 0 & 0 & \alpha_1 & 0 & -\frac{\sigma_v(1-\theta)(\beta_m+\beta_l+\beta_{lm})\Lambda_h k_2 k_7}{\Lambda_v(k_1k_2-\alpha_2\tau_1)} \\ 0 & -\frac{k_1k_2-\alpha_2\tau_1}{k_1} & 0 & 0 & 0 & 0 & \frac{\alpha_1\tau_1}{k_1} & 0 & -\frac{\Lambda_h k_7\tau_1(\beta_m k_1\sigma_v+\sigma_v(1-\theta)(\beta_m+\beta_l+\beta_{lm})k_2)}{k_1\Lambda_v(k_1k_2-\alpha_2\tau_1)} \\ 0 & 0 & -k_3 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_l\sigma_v(1-\theta)\Lambda_h k_2k_7}{\Lambda_v(k_1k_2-\alpha_2\tau_1)} \\ 0 & 0 & 0 & -k_1 & 0 & 0 & 0 & 0 & \frac{\rho\beta_l\sigma_v(1-\theta)\Lambda_h k_2k_7}{k_3\Lambda_v(k_1k_2-\alpha_2\tau_1)} \\ 0 & 0 & 0 & 0 & -k_4 & 0 & 0 & 0 & \frac{\Lambda_h\beta_m k_7(\sigma_v(1-\theta)k_2+\sigma_v\tau_1)}{\Lambda_v(k_1k_2-\alpha_2\tau_1)} \\ 0 & 0 & 0 & 0 & 0 & -k_5 & 0 & 0 & \frac{\beta_{lm}\sigma_v(1-\theta)\Lambda_h k_2k_7}{\Lambda_v(k_1k_2-\alpha_2\tau_1)} \\ 0 & 0 & \tau_1 & 0 & 0 & 0 & -k_6 & 0 & \frac{k_7\Lambda_h(\tau_3\beta_{lm}\sigma_v(1-\theta)k_1k_2k_3k_4+z_1+z_2)}{k_1k_3\Lambda_v(k_1k_2-\alpha_2\tau_1)k_4k_5} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -k_7 & -\frac{\sigma_v\beta_h\Lambda_h(\beta_{lm}\sigma_v(1-\theta)k_1k_2k_3k_4+z_3+z_4)}{\Lambda_h(\tau_1+k_2)k_1k_3k_4k_5} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\sigma_v\beta_h\Lambda_h(\beta_{lm}\sigma_v(1-\theta)k_1k_2k_4+z_3+z_4+k_1k_3k_4k_5k_7(k_2+\tau_1))}{\Lambda_h(\tau_1+k_2)k_1k_3k_4k_5} \end{pmatrix}$$

Therefore, the eigenvalues are

$$\lambda_1 = -k_1 < 0, \quad \lambda_2 = -\frac{k_1k_2-\alpha_2\tau_1}{k_1}$$

$$\lambda_2 = -\left(\frac{\mu_h^2+\mu_h\alpha_2+\mu_h\tau_1}{k_1}\right) < 0$$

$$\lambda_3 = -k_3 < 0$$

$$\lambda_4 = -k_1 < 0$$

$$\lambda_5 = -k_4 < 0$$

$$\lambda_6 = -k_5 < 0$$

$$\lambda_7 = -k_6 < 0$$

$$\lambda_8 = -k_7 < 0$$

$$\lambda_9 = \frac{\sigma_v\beta_h\Lambda_h(\beta_{lm}\sigma_v(1-\theta)k_1k_2k_4+z_3+z_4-k_1k_3k_4k_5k_7(k_2+\tau_1))}{\Lambda_h(\tau_1+k_2)k_1k_3k_4k_5k_7}$$

Therefore R_c <1
This implies that $\lambda_9 < 0$ if $R_c < 1$
Hence, the disease free equilibrium (E^0) of the Model equation is locally asymptotically stable (LAS).

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RESULTS

We varied some of the parameters (δ_v, τ_1, τ_2, τ_3) of the model using the values between 0 and 1 in order to see the effect of this variation on the model we formulate. For the numerical simulations, the time are in weeks. Other parameters are presented in Table 1. The graphical representation of various numerical simulations is presented in Figure 2.

Table 2. Values for Population Independent Parameter of the Model

Parameter	Values	Source
β_h	0.09	(Chunky Choole,2012)
β_v	0.50	Assumed
β_m	0.8333	(Chunky Choole,2012)
β_l	0.10	(Chunky Choole,2012)
β_{lm}	0.8333	(Chunky Choole,2012)
σ_v	0.125	(Lawi *et al*, 2011)
δ_m	0.00049312	(Lawi *et al*, 2011)
α_1	0.25	Assumed
β_h	0.09	(Chunky Choole,2012)
Λ_v	0.071	(Gweryina Reuben, 2014)
Λ_h	0.3	Assumed
μ_v	0.05	(Gweryina Reuben, 2014)
μ_h	0.017	(Gweryina Reuben, 2014)
β_{lm}	0.8333	(Chunky Choole,2012)
σ_v	0.125	(Lawi *et al*, 2011)
Θ	0.25	Assumed
δ_v	0.25	Assumed
P	0.00002797	(Bhunu and Mushayabasa, 2012)
τ_1	0.25	Assumed
τ_2	0.25	Assumed
τ_3	0.25	Assumed
α_1	0.25	Assumed
α_2	0.25	Assumed

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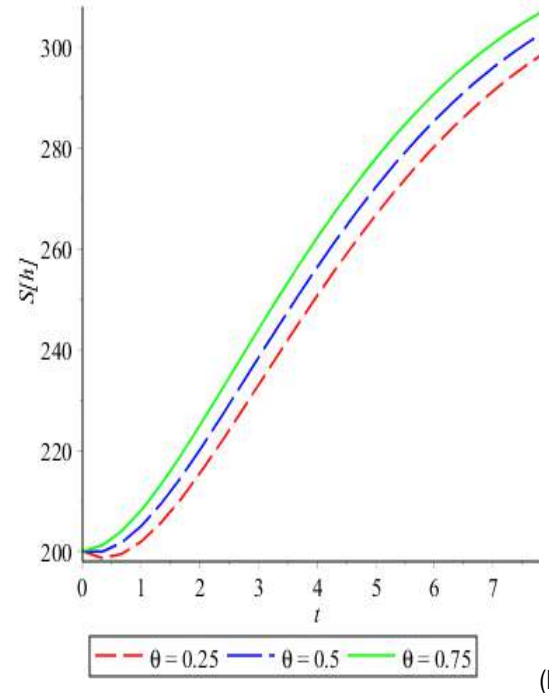
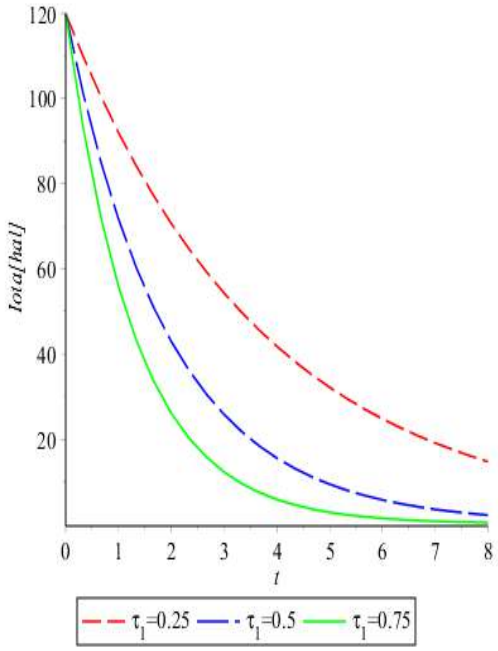
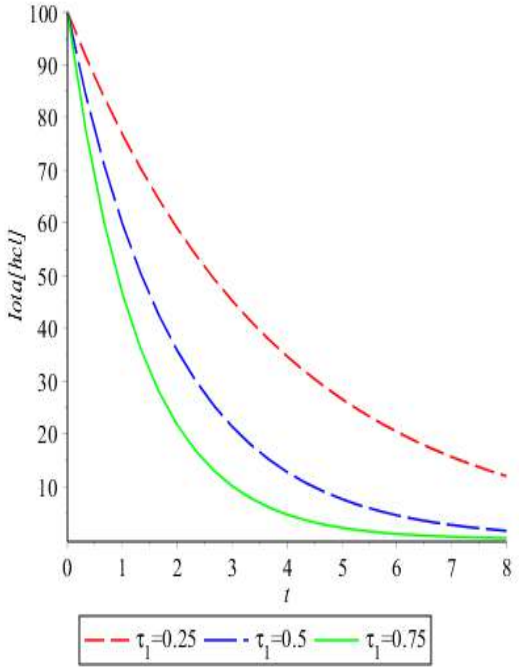


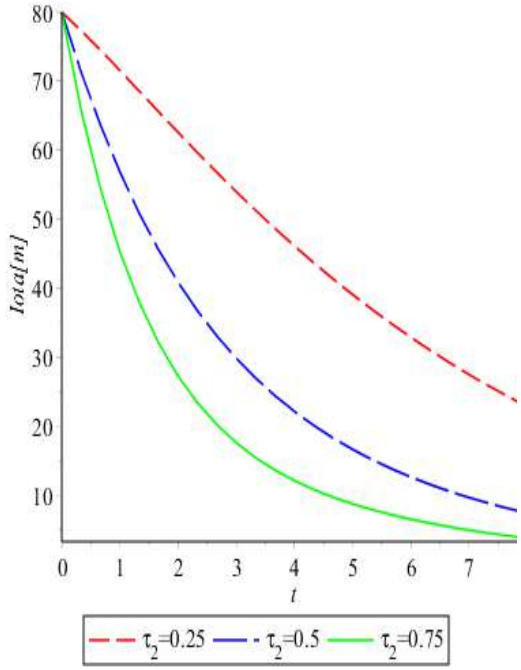
(a)



(b)



(c)



(d)

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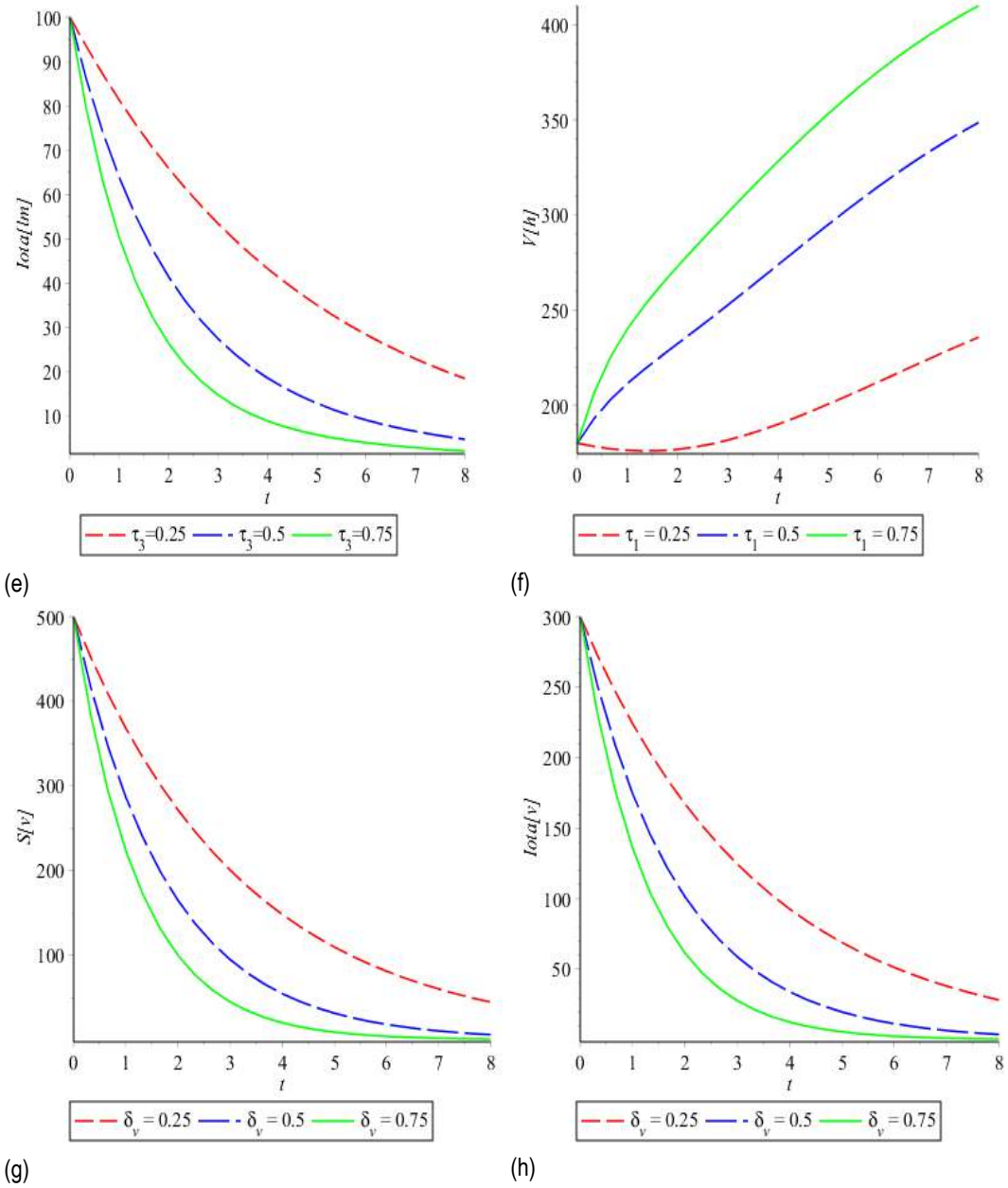

Figure 2. (a) Effect of the rate of the use of bed-net and insecticides on the Susceptible population. (b) Effect of treatment of LF on the Acute stage LF population. (c) Effect of treatment of LF on the Chronic stage LF population. (d) Effect of treatment of Malaria on those who are infected malaria. (e) The Effect of the rate of treatment of LF and malaria co-infection. (f) Effect of Treatment on the Chemoprevention class (V_h). (g) Effect of the use of insecticide on the Susceptible Vector Population. (h) Effect of the use of insecticide on the Infected Vector Population (I_v).

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DISCUSSION OF RESULTS

Figures 2a to 2h are the different graph of the solution using the populations of vectors and human against time with different parameters of the model. Maple software was used to plot the visualization of the result. Figure 2a shows that, due to the increased use of bed nets and insecticides, the susceptible population increases due to a decrease in the contact rate β with mosquitoes. Figures 2b to 2e, show a reduction in the population of the infected classes when treated for Malaria, LF, or both LF and malaria co-infection. Figure 2f depicts an increase in the chemoprevention population when drugs are administered, while Figures 2g to 2h depict the effects of using insecticides (δ_v), with the results indicating that as the use of insecticides increases, there is a reduction in both the susceptible and infected vector populations. Our findings suggest that combining all of the controls produces the best overall reduction in disease spread. As a result, we should encourage these controls in order to keep the disease from spreading.

CONCLUSION

In this study, the mathematical model of Lymphatic Filariasis and malaria co-infection was developed using a system of first order differential equations. The positivity of the solution was obtained using Lungu method. The equilibrium state of the Disease-Free Equilibrium state (DFE) was obtained and the basic reproduction number R_c of the model was also obtained. The Disease-Free Equilibrium (DFE) was analyse for local stability. The result from the analysis of the DFE showed that, the DFE is locally asymptotically stable if $R_c < 1$. The analytical solutions of the model were presented graphically in order to have a better understanding of the model.

In future work, we intend to solve the model using a semi-analytic approach and present the mathematical analysis in terms of optimal control. We hope that the findings of this study will help policymakers and health practitioners in the future to properly control diseases.

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