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Optimal Control Analysis for a Lymphatic Filariasis Model

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In this paper, a mathematical model for the transmission dynamics of lymphatic *filariasis* is presented. Human and mosquito populations are divided based on their lymphatic *filariasis* status. The human population is subdivided into six (6) compartments, while the mosquito population is subdivided into three (3) compartments. The disease-free equilibrium (DFE) and the endemic equilibrium states are proven to be the model's two equilibrium states. In terms of the model's demographic and epidemiological characteristics, an explicit formula for the effective reproduction number was found. The disease-free equilibrium state was discovered to be locally asymptotically stable using the Routh-Hurwitz criterion if the basic reproduction number is less than one. By using Castillo-Chavez, the disease-free equilibrium state was found to be globally asymptotically stable. This means that lymphatic *filariasis* could be put under control in a population when the reproduction number is less than one. Sensitivity analysis was carried out on the basic reproduction number to ascertain the parameters that have an impact on the reproduction number The results show that some parameters that appeared in the reproduction number have an impact on the reproduction number. An optimal control problem was formulated and analyzed using Pontryagin's Maximum Principle to determine the optimality system. The system was solved numerically using the forward and backward sweep method and results show that the combination of treated bed nets, antibiotics, and indoor residual spray is the most effective way to prevent the spread of *filariasis* in a community.

Keywords: Lymphatic filariasis, Microfilariae, Optimal control, Reproduction number, Stability analysis.

1. Introduction

Lymphatic filariasis, a debilitating disease, is one of the most prevalent and yet one of the most neglected tropical diseases with serious economic and social consequences [23, 29]. Lymphatic filariasis affects women, men, and children of all ages. It is a mosquito-borne disease caused by tissue dwelling nematodes of Brugia malayi, Brugia timori, and Wuchereria bancrofti species [28,29] and is estimated to affect about 120 million people worldwide [13, 14, 27]. Wuchereria bancrofti is responsible for 90% of the cases and is found throughout the tropical and subtropical areas of the world; Brugia malayi is confined to southeast and eastern Asia; Brugia timori is found only in Timor and its adjacent islands [19]. Infection leads to lymphedema, a buildup of fluid due to impaired function of the lymph vessels, in only a small proportion people, even in areas of intense transmission [6], as most people with long-term infections are clinically asymptomatic.

A number of mathematical models have looked into Malaria, a mosquito-borne infection [2, 5, 17, 18, 30,31], to mention just a few, but only a few have looked into lymphatic filariasis [4,8,19,22, 26]. However, these models have some limitations as they do not account for intervention measures such as quarantine. While EPIFIL uses a constant force of infection and accounts for the impact of age structure of the human community [19], LYMFASIM accounts for the role of the immune system in regulating parasite numbers [22].

[2] used nonlinear mathematical model to study the transmission dynamics of lymphatic filariasis with three (3) stages of infection. Result of their analysis suggested that effective lymphatic filariasis control requires strategies beyond filariasis treatment only. However, we proposed a nonlinear mathematical model with four (4) stages of infection namely: exposed human

(E_h), Acutely infected human $\left(I_h^a\right)$, Chronically infected human $\left(I_h^c\right)$ and infected mosquito $\left(I_m\right)$ with the aim of studying the transmission dynamics of the disease and optimal control strategies was incorporated to reduce disease transmission and infection in the population. The control measures proposed in this work include treated bed-nets, indoor residual spray as a prevention from getting infected with *filariasis* and treatment by the use of antibiotics.

2. Model Formulation

Human and mosquito populations are divided based on their lymphatic filariasis status. However, the human population is sub-divided into, susceptible humans $S_h(t)$, exposed human $E_h(t)$, acute infected human $I_h^a(t)$, chronically infected human $I_h^c(t)$, recovered human $R_h(t)$ and recovered humans with disability $D_h(t)$, with total human population given by $N_h(t)=S_h(t)+E_h(t)+I_h^a(t)+I_h^c(t)+R_h(t)+D_h(t)$ while, the mosquito population is sub-divided into three compartments which includes; susceptible mosquito $S_m(t)$, exposed mosquitoes $E_m(t)$ and infected mosquitoes $I_m(t)$, with total mosquito population given by $N_m(t)=S_m(t)+E_m(t)+I_m(t).$

Recruitment into human population is Λ_h while Λ_m is the recruitment rate of the mosquito population. The natural death rates of human and mosquito population are μ_h and μ_m respectively. These death rates are proportional to the number of each human or mosquito class. The mosquito ingests microfilariae when biting a human who is infected with *filariasis* at a rate $(1-u_1)\dfrac{\varepsilon_h\beta_h\left(\theta_hE_h+\phi_hI_h^a+I_h^c\right)}{N_m}$, where $(1-u_1)$ reduces the force of infection due to the use of treated bed nets as a prevention from getting *filariasis* infection, ε_h is the success rate of microfilariae transmission from human to susceptible mosquitoes , β_h is the average number of mosquito bites which cause transmission of disease from infected human to susceptible mosquito; θ_h and $\phi_h\in(0,1)$ account for reduced number of microfilariae in the blood stream of $E_h(t)$ and $I_h^a(t)$ respectively. Susceptible mosquito can experience death (artificial) due to indoor residual spray at a rate $u_3\theta_m$ which is proportional to the number of each mosquito population. The susceptible mosquito population decreased by $(1-u_1)\dfrac{\varepsilon_h\beta_h\left(\theta_hE_h+\phi_hI_h^a+I_h^c\right)}{N_m}$.

Then the exposed mosquito $E_m(t)$ population increased by $(1-u_1)\dfrac{\varepsilon_h\beta_h\left(\theta_hE_h+\phi_hI_h^a+I_h^c\right)}{N_m}$ and reduced due to progression to infected mosquito population at a rate ρ_m. At this stage, the microfilariae ingested by the mosquito developed into infective *filariform* larvae to become infectious and hence, these mosquitoes move into the infected class $I_m(t)$ at a rate ρ_m. *Filariform* larvae escape from infected mosquito's proboscis $I_m(t)$ when the insect is feeding and then penetrate a wound structure of a susceptible human at a rate $(1-u_1)\dfrac{\varepsilon_m\beta_{mI_m}}{N_h}S_h$ where ε_m is the success rate of microfilariae transmission from infected mosquito to susceptible human, β_m is the average number of mosquito bites which cause transmission of disease from infectious mosquito to susceptible human per mosquito and move to the exposed class $E_h(t)$. The population of the exposed individuals at time t, $E_h(t)$, increased by progression of newly infected individuals at a rate $(1-u_1)\dfrac{\varepsilon_m\beta_{mI_m}}{N_h}S_h$ and reduced due to progression to infected-acute humans $I_h^a(t)$ at a rate $\theta_h\rho_h$. The population of the acute infected individuals at time t increases due to progression rate $\theta_h\rho_h$ and reduces due to progression to chronically infectious at a rate σ_h and recovery from the illness at a rate $u_2\gamma_a$.

The population of $I_h^c(t)$ increased by progression from $I_h^a(t)$ at a rate σ_h and decreased by $u_2\gamma_c$. The population of $R_h(t)$ increased by progression from $I_h^a(t)$ and $I_h^c(t)$ at a rate $u_2\gamma_a$ and $u_2\gamma_c$ respectively and decreases at a rate α_h and π_h respectively. The population of recovered with disability $D_h(t)$ increases by progression from $R_h(t)$ at a rate π_h and decreases at a rate φ_h. The population of recovered with disability become susceptible to the disease at a rate φ_h.

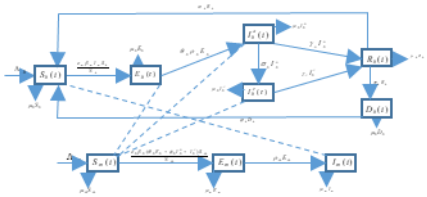


Figure. 1 Schematic Diagram of the model

The *lymphatic filariasis* model equations with constant controls are:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \left(\frac{\varepsilon_m\beta_m I_m}{N_h} + \mu_h\right)S_h + \alpha_h R_h + \varphi_h D_h \\
\frac{dE_h}{dt} &= \left(\frac{\varepsilon_m\beta_m I_m}{N_h}\right)S_h - (\theta_h\rho_h + \mu_h)E_h \\
\frac{dI_h^a}{dt} &= \theta_h\rho_h E_h - (\mu_h + \sigma_h + \gamma_a)I_h^a \\
\frac{dI_h^c}{dt} &= \sigma_h I_h^a - (\gamma_c + \mu_h)I_h^c \\
\frac{dR_h}{dt} &= \gamma_a I_h^a + \gamma_c I_h^c - (\alpha_h + \mu_h + \pi_h)R_h \\
\frac{dD_h}{dt} &= \pi_h R_h - (\varphi_h + \mu_h)D_h \\
\frac{dS_m}{dt} &= \Lambda_m - \left(\frac{\varepsilon_h\beta_h(\theta_h E_h + \phi_h I_h^a + I_h^c)}{N_m} + \mu_m\right)S_m \\
\frac{dE_m}{dt} &= \frac{\varepsilon_h\beta_h(\theta_h E_h + \phi_h I_h^a + I_h^c)}{N_m}S_m - (\rho_m + \mu_m)E_m \\
\frac{dI_m}{dt} &= \rho_m E_m - \mu_m I_m
\end{aligned} \quad (1)$$

3. Models Analysis

3.1 Sensitivity Analysis

To obtain the sensitivity results, we used the normalized index formula to obtain the results in Table 1. From the results in Table1, it follows that, a positive index sign indicates that an increase in the parameter's value will result in an increase in the value of the reproduction number and a reduction in the parameter's value will reduce the value of the reproduction number. A negative index sign indicates that an increase in the parameter's value will result in a reduction in the value of the reproduction number and a reduction in the parameter's value will result in an increase in the value of the reproduction number [see 35].

Table 1: Sensitivity indices results

Parameters	Sensitivity index
ε_m	0.5
ε_h	0.5
μ_h	–0.3570124
μ_m	–0.17
ρ_h	–0.4853767
ρ_m	0.41869
β_m	0.5
θ_h	0.005403
ϕ_h	0.0007007
σ_h	–0.00497
γ_a	–0.0327
γ_c	0.011195
β_h	0.512

3.2 Optimal Control Analysis

Based on the sensitivity results in Table 1, we updated the filariasis model to get a model given as

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \left((1-u_1)\frac{\varepsilon_m\beta_m I_m}{N_h} + \mu_h\right)S_h + \alpha_h R_h + \varphi_h D_h \\
\frac{dE_h}{dt} &= \left((1-u_1)\frac{\varepsilon_m\beta_m I_m}{N_h}\right)S_h - (\theta_h\rho_h + \mu_h)E_h \\
\frac{dI_h^a}{dt} &= \theta_h\rho_h E_h - (\sigma_h + u_2\gamma_a + \mu_h)I_h^a \\
\frac{dI_h^c}{dt} &= \sigma_h I_h^a - (u_2\gamma_c + \mu_h)I_h^c \\
\frac{dR_h}{dt} &= u_2\gamma_a I_h^a + u_2\gamma_c I_h^c - (\alpha_h + \pi_h + \mu_h)R_h \\
\frac{dD_h}{dt} &= \pi_h R_h - (\varphi_h + \mu_h)D_h \\
\frac{dS_m}{dt} &= \Lambda_m - \left((1-u_1)\frac{\varepsilon_h\beta_h\left(\theta_h E_h + \phi_h I_h^a + I_h^c\right)}{N_m} + (\mu_m + u_3\theta_m)\right)S_m \\
\frac{dE_m}{dt} &= (1-u_1)\frac{\varepsilon_h\beta_h\left(\theta_h E_h + \phi_h I_h^a + I_h^c\right)}{N_m}S_m - (\rho_m + \mu_m + u_3\theta_m)E_m \\
\frac{dI_m}{dt} &= \rho_m E_m - (\mu_m + u_3\theta_m)I_m
\end{aligned} \tag{2}$$

The factor $(1-u_1)$ is a control function representing prevention from lymphatic *filariasis* infection through the use of treated bed nets by human populations. u_2 is a control representing treatment using antibiotic by the human population. u_3 is a control representing indoor residual spray to eradicate the mosquito populations. The use of treated bed nets will reduce the spread of the *filariasis* disease from infected to susceptible individuals. Treatments will also enhance the health status of the infectious individual and confer temporary immunity against reinfection. Where t_f is the final time and the coefficients $A_1, A_2, A_3, A_4, c_1, c_2$ and c_3 are positive weights to balance the factors. The aim is to minimize the number of infected individuals and total number of mosquitoes population, while minimizing the cost of controls $u_1(t)$, $u_2(t)$, $u_3(t)$.Thus, we seek an optimal controls u_1^*, u_2^*, u_3^* such that $J(u_1^*, u_2^*, u_3^*) = \min_{u_1,u_2,u_3\in\Pi} J(u_1, u_2, u_3)$.

where $\Pi = \{u_1, u_2, u_3\}$ such that u_1, u_2, u_3 are measurable with $0 \le u_1 \le 1, 0 \le u_2 \le 1$ and $0 \le u_3 \le 1$ for $t \in [0, t_f]$ is the control set. The essential conditions critically needed for an optimal solution to satisfy emanate from Pontryagin's Maximum Principle. The Hamiltonian is expressed as

$$\begin{aligned}
H = {} & A_1E_h + A_2I_h^a + A_3I_h^c + A_4N_m + \frac{c_1u_1^2}{2} + \frac{c_2u_2^2}{2} + \frac{c_3u_3^2}{2} \\
& + \lambda_{S_h}\left[\Lambda_h - (1-u_1)\frac{\varepsilon_m\beta_m I_m}{N_h}S_h - \mu_h S_h + \alpha_h R_h + \varphi_h D_h\right] \\
& + \lambda_{E_h}\left[(1-u_1)\frac{\varepsilon_m\beta_m I_m}{N_h}S_h - (\theta_h\rho_h + \mu_h)E_h\right] \\
& + \lambda_{I_h^a}\left[\theta_h\rho_h E_h - (\sigma_h + u_2\gamma_a + \mu_h)I_h^a\right] \\
& + \lambda_{I_h^c}\left[\sigma_h I_h^a - (u_2\gamma_c + \mu_h)I_h^c\right] \\
& + \lambda_{R_h}\left[u_2\gamma_a I_h^a + u_2\gamma_c I_h^c - (\alpha_h + \pi_h + \mu_h)R_h\right] \\
& + \lambda_{D_h}\left[\pi_h R_h - (\varphi_h + \mu_h)D_h\right] \\
& + \lambda_{S_m}\left[\Lambda_m - (1-u_1)\frac{\varepsilon_h\beta_h\left(\theta_h E_h + \phi_h I_h^a + I_h^c\right)}{N_m}S_m - (\mu_m + u_3\theta_m)S_m\right] \\
& + \lambda_{E_m}\left[(1-u_1)\frac{\varepsilon_h\beta_h\left(\theta_h E_h + \phi_h I_h^a + I_h^c\right)}{N_m}S_m - (\rho_m + \mu_m + u_3\theta_m)E_m\right] \\
& + \lambda_{I_m}\left[\rho_m E_m - (\mu_m + u_3\theta_m)I_m\right]
\end{aligned} \tag{3}$$

where $\lambda_{S_h}, \lambda_{E_h}, \lambda_{I_h^a}, \lambda_{I_h^c}, \lambda_{R_h}, \lambda_{D_h}, \lambda_{S_m}, \lambda_{E_m}$, and λ_{I_m} den otes the adjoin variables.

Theorem 4. For the optimal controls u_i^*, for $i = 1, 2, 3$ and solution $S_h, E_h, I_h^a, I_h^c, R_h, D_h, S_m, E_m$ and I_m of the associated state system (23) that minimizes $J = (u_1, u_2, u_3)$ over Π

Then there exist adjoin variables $\lambda_{S_h}, \lambda_{E_h}, \lambda_{I_h^a}, \lambda_{I_h^c}, \lambda_{R_h}, \lambda_{D_h}, \lambda_{S_m}, \lambda_{E_m}$, and λ_{I_m} sati sfying

$$-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial i} \tag{4}$$

where $i = S_h, E_h, I_h^a, I_h^c, R_h, D_h, S_m, E_m, I_m$ with transversality conditions

$$\lambda_{S_h} = \lambda_{E_h} = \lambda_{I_h^a} = \lambda_{I_h^c} = \lambda_{R_h} = \lambda_{D_h} = N_{S_m} = \lambda_{E_m} = \lambda_{I_m} = 0 \tag{5}$$

and

$$u_1^* = \min\left\{1, \max\left(1, \frac{\frac{\varepsilon_m \beta_m I_m S_h}{N_h}\left(\lambda_{E_h} - \lambda_{S_h}\right) + \frac{\varepsilon_h \beta_h \left(\theta_h E_h + \phi_h I_h^a + I_h^c\right) S_m}{N_m}\left(\lambda_{E_m} - \lambda_{S_m}\right)}{c_1}\right)\right\}$$

$$u_2^* = \min\left\{1, \max\left(1, \frac{\gamma_a I_h^a\left(\lambda_{I_h^a} - \lambda_{R_h}\right) + \gamma_c I_h^c\left(\lambda_{I_h^c} - \lambda_{R_h}\right)}{c_2}\right)\right\}$$

$$u_3^* = \min\left\{1, \max\left(1, \frac{\theta_m\left(S_m \lambda_{S_m} + E_m \lambda_{E_m} + I_m \lambda_{I_m}\right)}{c_3}\right)\right\} \quad (6)$$

By determining the solution for u_i^*, for $i = 1, 2, 3$ subject to the constraints, the characterization (3) is obtained.

Table 2: Parameter Values

Parameters	Values	Reference
Λ_h	2500	[20, 25]
Λ_m	1000	[20]
ε_m	0.01	[25].
ε_h	0.01	[25].
μ_h	0.000039	[1,15]
μ_m	0.1429	[15, 20]
ρ_h	0.0238	[10]
ρ_m	0.0555	[10]
β_m	0.86	[9]
β_h	0.283	[11]
θ_h	0.25	[3]
θ_m	0.001	[7]
ϕ_h	0.15	Assumed
σ_h	10	[16]
γ_a	0.7	[16]
γ_c	0.97	Assumed
π_h	0.97	Assumed
α_h	0.125	[3]
φ_h	0.125	Assumed
A_1	1	[7]
A_2	1	Assumed
A_3	2	Assumed
A_4	4	[7]
C_1	1	[7]
C_2	10^i, $i = 0, 2, 4$	[21]
C_3	3	[7]

Table 3: State Variables Values

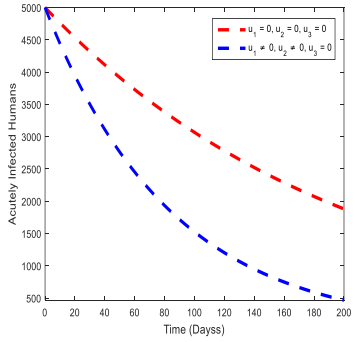
Variables	Values	Reference
$S_h(0)$	10,000	[11]
$E_h(0)$	6,000	[11]
$I_h^a(0)$	5,000	[11]
$I_h^c(0)$	1,500	Assumed
$R_h(0)$	3,000	[11]
$D_h(0)$	1,455	Assumed
$S_m(0)$	100,000	[11]
$E_m(0)$	16,000	Assumed
$I_m(0)$	30,000	Assumed

5. Numerical Simulations

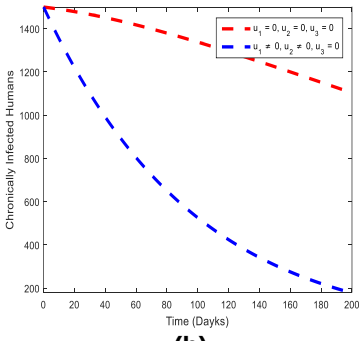
Using the data from Tables (2) and (3), we numerically simulated the optimality system in this section. We used an iterative approach to find the best option.

The results are presented in Figure 2 to Figure 5.

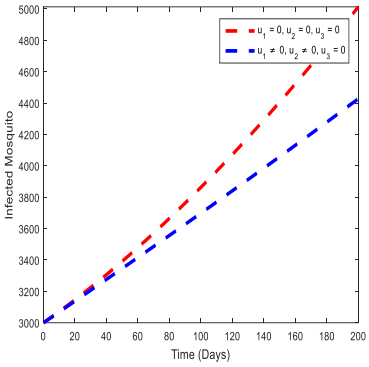
Case 1: use of treated net and antibiotics bed



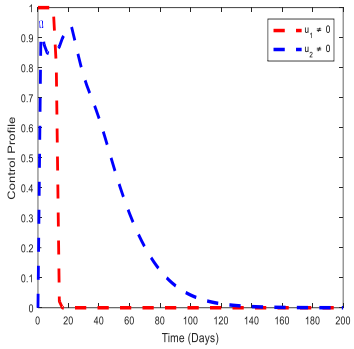
(a)



(b)

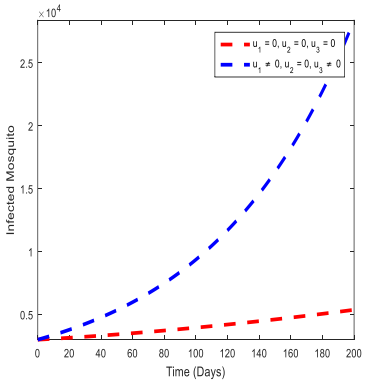


(c)

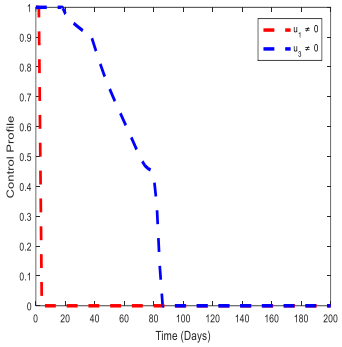


(d)

Figure 2: Optimal use of treated bed net and antibiotics as control strategy



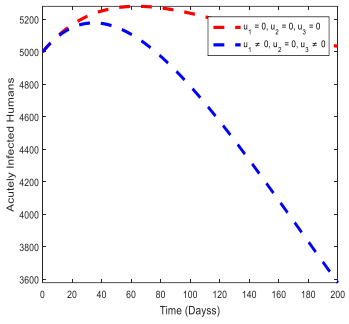
(c)



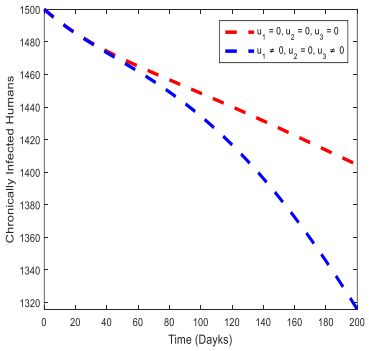
(d)

Figure 3: Optimal use of treated bed net and indoor residual spray as control strategy

Case 2: use of treated bed net and indoor residual spray

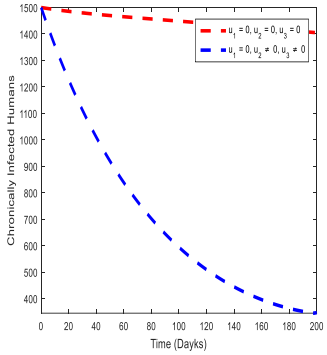


(a)



(b)

Case 3: use of antibiotics and indoor residual spray



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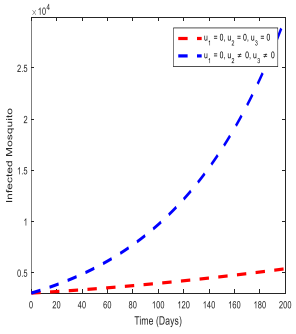
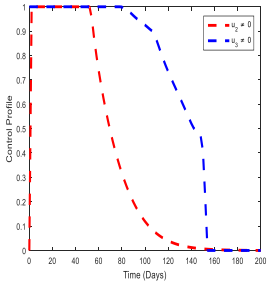


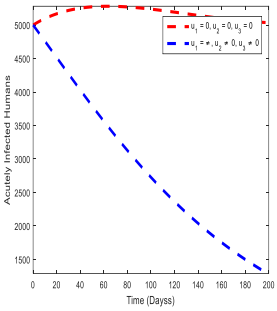
(b)



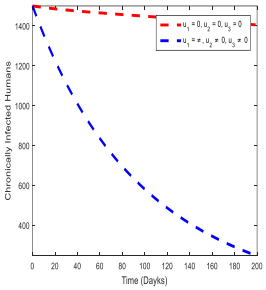
(c)

Figure 4: Optimal use of antibiotics and indoor residual spray as control strategy

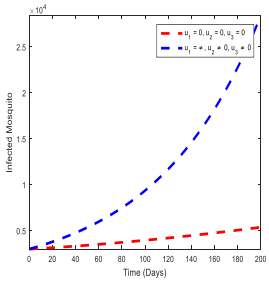
Case 4: use of treated bed net, antibiotics, and indoor residual spray



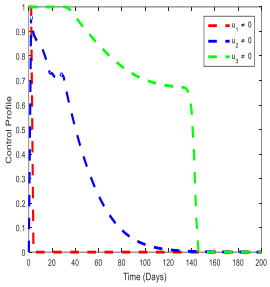
(a)



(b)



(c)



(d)

Figure 5: Optimal use of treated bed net, antibiotics, and indoor residual spray as control strategy

Case 1:

In case 1, we present the simulation of optimal control system for treated bed net and antibiotics $(u_1 = u_2 \neq 0, u_3 = 0)$ as intervention to control *filariasis* disease. Figure 2 (a-c) are the simulation results of case 1. On the figure, we observed that there is a moderate decline in the population of acutely infected human and infected mosquito, there is a significant decline in the population of chronically infected human compared to the population without optimal control in case 1 when $(u_1 = u_2 = u_3 = 0)$. Figure 2 (d) shows the control profile for case 1. We observed that the curve for treated bed net remained at the upper bound for almost 20 days before dropping to the lower bound, while the curve for the antibiotics remained at upper bound for 30 days before dropping to the lower bound

Case 2:

We simulated the model by incorporating optimized treated bed net (u_1) and indoor residual spray (u_3) as control strategy for *filariasis* disease, setting $u_1 = u_3 \neq 0$ while we set $u_2 = 0$, to minimize the objective functional. Figure 3 (a-c) are the simulation result of case 2. On the figure, we observed that there is a significant decline in the population of Acutely infected human and also there is a moderate decline in the population of chronically infected human while an increase in the population of infected mosquito. Figure 3 (d) shows the control profile for case 2. We observed that the curve for treated bed net stays at the upper bound for about 4 days before reaching the lower bound, the control curve for indoor residual spray remained at the upper bound for 20 days before reaching the lower bound. Hence, we can say that this strategy is effective in eradicating the disease from the population in a specified period of time.

Case 3:
In case 3, we present the simulation of optimal control system for antibiotic and indoor residual spray $(u_2 = u_3 \neq 0, u_1 = 0)$ as a control strategy for *filariasis* disease. figure 4 (a-c) are the simulation of case 3. On the figure, we observed that there is a significant decline in the population of acutely infected human and chronically infected human, while there is increase in the population of infected mosquito when the strategy is implemented compared to the case with no optimal control. Figure 4 (d) shows the control profile for case 3. We observed that the curve for the antibiotic remained at the upper bound for about 55 days before dropping to the lower bound while the control curve for indoor residual spray stays at the upper bound for about 90 days before reaching its lower bound.

Case 4:
Case 4 presents the simulation of optimal system for treated bed net, antibiotics, and indoor residual spray $(u_1 = u_2 = u_3 \neq 0)$ as intervention to control *filariasis* disease. Figure 5 (a-c) are the simulation results of case 4. On the figure, we observed that there is a significant decline in the population of acutely infected human and chronically infected human, we also observed that there is significant increase in the population of infected mosquito. Figure 5 (d) shows the control profile for case 4. We observed that the curve for the treated bed net remained at upper bound for about 4 days before reaching its lower bound, the curve for antibiotic stays at the upper bound for about 8 days before gradually dropping to its lower bound and the curve for indoor residual spray remained at the upper bound for about 35 days before it's started dropping gradually to the lower bound.

6. Conclusion

The filariasis model consist of nine (9) system of nonlinear differential equations. The model consists of three (3) controls namely: treated bed net, antibiotics, and indoor residual spray. We considered the control parameters as constant in our first line of analysis. The positivity of the solutions analysis show that the system of differential equations has non-negative solutions for all time t, and is bounded within the given region. The disease-free and the present equilibrium were obtained. We used Routh-Hurwitz criterion to determine the local stability while Castillo-Chavez conditions were applied to prove the global asymptotic stability. The disease-free equilibrium is proved to be locally asymptotically stable (LAS) and globally asymptotically stable (GAS) when the reproduction number $R_0 < 1$. This implies that the disease will be completely die out if the system is stable and unstable if otherwise. By resolving the optimality system, which consists of nine (9) ordinary differential equations derived from the state and adjoint equations, we were able to achieve the desired control. Using forward backward sweep method by Lenhart Suzanne, we solved the state equations for the controls over the simulated. We used different strategies such as a combination of two controls at a time and a combination of all the three controls at a time.

Conflict of interest

The authors declare no conflict of interest.

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