Global Stability and Backward Bifurcation for a Lymphatic filariasis model

Egberanmwen Barry Iyare (✉ barriyare@yahoo.com)

Samuel Adegboyega University, Nigeria <https://orcid.org/0000-0002-2196-3354>

Daniel Okuonghae

University of Benin, Nigeria

Francis Osagiede

University of Benin, Nigeria

Research Article

Keywords: Backward bifurcation, Lymphatic filariasis, Threshold conditions, Global Stability

Posted Date: February 9th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-191972/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Global Stability and Backward Bifurcation for a Lymphatic *filariasis* Model

Egberanmwun Barry Iyare¹

Daniel Okuonghae^{2a}

Francis Osagiede^{2b}

1. Dept. of Mathematical and Physical Sciences, Samuel Adegboyega University, Ogwa, Edo, Nigeria.

2a&b. Dept. of Mathematics, University of Benin, Nigeria.

Corresponding author: Egberanmwun Barry Iyare; Email: barriyare@yahoo.com

January, 2021

Abstract

An ODE model of Lymphatic *filariasis* is proposed with eight mutually disjoint compartments. The model is proven to be mathematically and epidemiologically well posed. Epidemiological interpretation of the effective reproduction number is presented. A special case is considered where the death rate due to the disease is negligible. An endemic equilibrium under this special scenario is explicitly computed and the presence of backward bifurcation under this condition is suggested. Bifurcation analysis is performed using the Castillo-Chavez and Song theorem in the special case where death due to disease is zero. When the re-infection rate is zero, backward bifurcation is shown not to be present. In such a situation, global asymptotic stability of the endemic equilibrium is established.

Keywords: Backward bifurcation, Lymphatic *filariasis*, Threshold conditions, Global Stability.

1 Introduction

Infectious diseases has always been a great concern of humans. The outbreak and spread of infectious diseases has been questioned and studied for many years, they are still the major causes of death in the developing countries. Lymphatic *filariasis*, a neglected tropical disease, is still a major public health problem in Africa, South America and Asia [1]. Despite existing knowledge of the disease pathology and global treatment campaign, nearly 1.4 billion people in 73 countries worldwide are threatened by the disease, of which over 120 million people are currently infected [2].

There are more than 500 filarial parasites that are known to infect mammals, birds, reptiles and amphibians [3]. *Wuchereria bancrofti*, *brugia malayi*, *onchocerca volvulus*, *loa loa*, *mansonella perstans*, *mansonella streptocerca*, *mansonella ozzardi* and *brugia timori* are the common parasites of man. The adult of *brugia malayi*, *brugia timori* and *wuchereria bancrofti* inhabit the lymphatic system, hence, the disease they cause is called lymphatic *filariasis* [3]. The worms have an estimated active reproductive span of 4-6 years, producing millions of small miniature microfilariae, which circulates in the peripheral blood [2]. They are transmitted from person to person by several species of mosquitoes [2]. Some of the symptoms of lymphatic *filariasis* include recurrent episodes of chills and fever during the day and at night, especially when the microfilariae circulates across the bloodstream, paroxysmal coughing at night, swellings of legs and arms known as elephantiasis etc. Treatment include use of diethylcarbamazine (DEC). This drug focuses on killing the microfilariae which are active and circulating through the bloodstream. It is also effective against the adult *wuchereria bancrofti* worms and can effectively treat lymphatic *filariasis*. In cases where the lymph nodes are severely clogged and limbs are inflame, surgical procedures maybe prescribed to reduce the swellings and relieve the obstruction.

*Corresponding author: , barriyare@yahoo.com

Several mathematical models have been formulated to understand the transmission dynamics of lymphatic *filariasis* under various circumstances. [4] developed and analysed a lymphatic *filariasis* disease transmission model to determine the impact of multi-interventions campaign via health education and sterile insect technique (SIT) on the spread of lymphatic *filariasis*. [5] formulated and analysed a nonlinear differential equation model to study the effect of chemoprophylaxis on the exposed individuals. [6] developed a mathematical model to investigate the impact that vector genus specific dependent processes may have on overall lymphatic *filariasis* transmission. [7] used lymphatic *filariasis* Simulation Model (LYMFASIM) to estimate the duration of Mass Drug Administration (MDA) required for elimination and residual levels of microfilaria (Mf) and antigenaemia (Ag) prevalence reached after that duration in different transmission settings varying from low to high. The result indicated that the duration of annual MDA increased with higher baseline endemicity and lower coverage. [8] used a model-based assessment to develop different plausible scale-up scenarios to reach global elimination and eradication of lymphatic *filariasis*. They predicted the duration of MDA to reach local elimination for different transmission archetypes and estimated the required number of treatments and the implication of rapid scale-up. [9] used mathematical models to assess the feasibility and strategic value of including vector control in the GPELF initiative to achieve the global elimination of lymphatic *filariasis*. [10] developed and analysed a mathematical model to quantify the potential effect that heterogeneous infection processes occurring in the major mosquito vector genera may have on parasite transmission and control. [11] formulated and analysed a deterministic differential equation model with two key control measures; quarantine and treatment. They assumed that no infection exists at the initial stage and that there is no vertical transmission in both human and mosquito population. In all the models considered, none has created a separate class for the symptomatic and asymptomatic individuals. In this work, we proposed eight non-linear mathematical models to study the transmission dynamics of lymphatic *filariasis* with treatment in the symptomatic class and those re-infected after treatment enters the exposed class.

2 Methods

The pathogenesis of lymphatic filariasis (LF), signs and symptoms, diagnosis and treatment as well as current epidemiological data were presented. Thereafter, an extensive review of lymphatic filariasis (LF) were carried out. Mathematical tools and techniques employed in the development and analysis of the formulated model were identified and studied. These methods and techniques include; use of Lyapunov functionals, Metzler matrix theory, bifurcation analysis, and other methods used for the study of dynamical systems. ([12],[13],[14],[15],[16],[17]). Based on the biology and natural history of lymphatic filariasis, we formulated novel deterministic mathematical model. The model was rigorously analyzed. We formulated the following system of 8 nonlinear ordinary differential equations. The model is given below.

The total human population at time t , denoted by $N_h(t)$, is subdivided into five mutually-disjoint compartments of susceptible humans ($S_h(t)$), latent individual ($E_h(t)$), Asymptomatic individual ($A_h(t)$), Symptomatic individual ($I_h(t)$), and Treated individual ($T_h(t)$). The total vector population at time t , denoted by $N_v(t)$ is subdivided into three mutually-disjoint compartments of susceptible mosquitoes ($S_v(t)$), latent mosquitoes ($E_v(t)$) and infectious mosquitoes ($I_v(t)$), so that

$$N_h(t) = S_h(t) + E_h(t) + A_h(t) + I_h(t) + T_h(t). \quad (2.1)$$

$$N_v(t) = S_v(t) + E_v(t) + I_v(t). \quad (2.2)$$

Based on the assumptions above, the model is given by the following system of nonlinear ordinary differential equations in (2.3)

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_v S_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \lambda_v S_h + \nu \lambda_v T_h - (k_1 + \mu_h) E_h, \\
\frac{dA_h}{dt} &= k_1 E_h - (k_2 + \mu_h) A_h \\
\frac{dI_h}{dt} &= k_2 A_h - (\tau_{h1} + \delta_L + \mu_h) I_h \\
\frac{dT_h}{dt} &= \tau_{h1} I_h - \nu \lambda_v T_h - \mu_h T_h \\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_h S_v - \mu_v S_v, \\
\frac{dE_v}{dt} &= \lambda_h S_v - k_v E_v - \mu_v E_v, \\
\frac{dI_v}{dt} &= k_v E_v - \mu_v I_v,
\end{aligned} \tag{2.3}$$

where

$$\lambda_v = \frac{\beta_v \sigma_v \sigma_h I_v}{\sigma_v N_v + \sigma_h N_h}$$

and

$$\lambda_h = \frac{\beta_h \sigma_v \sigma_h (\eta_1 E_h + \eta_{h1} A_h + I_h)}{\sigma_v N_v + \sigma_h N_h}$$

are the forces of infection.

Table 1: Description of state variables of the model

Variable	Description
$S_h(t)$	Population of susceptible individuals
$E_h(t)$	Population of individuals with latent LF
$A_h(t)$	Population of individuals with asymptomatic LF
$I_h(t)$	Population of individuals with symptomatic LF
$T_h(t)$	Population of individuals treated of LF
$S_v(t)$	Population of susceptible mosquitoes
$E_v(t)$	Population of exposed mosquitoes.
$I_v(t)$	Population of infectious mosquitoes.

2.1 Explanation of Terms of the Equations in (2.3)

The equations in (2.3) represent the derivatives, with respect to time, of the different human and mosquito compartments. The first equation in (2.3) represent the derivatives of the susceptible human population, S_h . The first term is

Table 2: Description of parameters of the model

Parameter	Description
μ_h, μ_v	Natural death rates
Λ_h, Λ_v	Recruitment rates
β_v	Transmission probability from mosquitoes to humans
β_h	Transmission probability from humans to mosquitoes
b_v	Number of bites on humans per unit time
b_h	Number of bites per mosquito per unit time
σ_v	Number of times a mosquito bites human per unit time
σ_h	Maximun number of mosquito bites a human can receive per unit time
$\tau_{h1}, \tau_{h2}, \tau_{h3}$	Treatment rates for LF
k_1, k_2	Progression rate for LF
ν	Reinfection rate for LF after treatment
k_v	Progression rates for mosquitoes
δ_L	Disease-induced death rate for individuals with LF

the rate of recruitment Λ_h . The second term is the interaction between the susceptible human, S_h and the infectious mosquitoes that leads to new cases of lymphatic *filariasis* in humans. This leaves the susceptible human class S_h and moves into the exposed or latent class E_h . The last term is the death rate for the susceptible human population, S_h .

The second equation in (2.3) represent the derivatives of the latent or exposed class. The first term represent the number of new latent lymphatic *filariasis* cases. The second term represent those that got reinfected after treatment. The third term represent those that developed the lymphatic *filariasis* disease, are infective but does not show symptoms of the disease. They are regarded as the asymptomatic lymphatic *filariasis* cases. This leaves the latent class E_h and move into the asymptomatic class, A_h . The last term is the death rate for the latent humans.

The third equation in (2.3) represent the derivatives of the asymptomatic humans A_h class (i.e. those with lymphatic *filariasis* disease but does not show symptoms). The first term is the number of asymptomatic individuals. The second term represent those that have developed the disease, are infective and shows symptoms of the disease. They are regarded as the symptomatic lymphatic *filariasis* cases. This leaves the asymptomatic class, A_h and moves into the symptomatic class, I_h . The last term is the natural mortality rate of the asymptomatic class.

The fourth equation in (2.3) represent the derivatives of the symptomatic class, I_h (i.e. those showing symptoms of the disease). The first term represent the number of symptomatic individuals. The second term represent those undergoing treatment, they leaves the symptomatic class and moves into the recovered or treated class. The third term represent those that died due to the disease and the last term is the death rate.

The fifth equation in (2.3) represent the derivatives of the treated or recovered class. The first term on the right hand side represent the number of treated individuals. The second term of the equation represent those that are re-infected after they have been treated of the disease. These leaves the treated class and moves into the latent class. The last term is the death rate for the treated class.

The sixth equation in (2.3) represent the derivatives of the susceptible class, S_v for mosquito population. The first term on the right hand side of the sixth equation is the recruitment term for mosquitoes, Λ_v . The second term of the sixth equation is the interaction between the susceptible mosquito, I_v , the exposed human, E_h asymptomatic, A_h and symptomatic humans, I_h , leading to new cases of latent or exposed mosquitoes. These leaves the susceptible class S_v and moves into the expose class, E_v . The last term is the death rate for susceptible mosquitoes.

The seventh equation in (2.3) represent the derivatives of the latent or exposed class for mosquitoes. The first term represent the number of new cases of latent mosquitoes. The second term represent those mosquitoes that have fully develop the filarial parasite and can transmit the disease. The last term is the death rate for latent mosquitoes.

The eight equation in (2.3) represent the derivatives of the infectious class for mosquitoes. The first term on the right hand side is the number of new infectious mosquitoes. The last term is the death rate for infectious mosquitoes.

Theorem 1: Let the initial data for the LF model (2.3) be given as $S_h(0) > 0, E_h(0) > 0, A_h(0) > 0, I_h(0) > 0, T_h(0) > 0, S_v(0) > 0, E_v(0) > 0$ and $I_v(0)$. Then the orbits $(S_h(t), E_h(t), A_h(t), I_h(t), T_h(t), S_v(t), E_v(t), I_v(t))$ of the model with positive initial conditions, will remain positive for all time $t > 0$.

Proof:

Consider the first equation of model (2.3), given below as

$$\frac{dS_h}{dt} = \Lambda_h - \lambda_v S_h - \mu_h S_h, \quad (2.4)$$

We can write (2.4) as

$$\frac{dS_h}{dt} \geq -(\lambda_v + \mu_h)S_h, \quad (2.5)$$

$$\frac{dS_h}{S_h(t)} \geq -(\lambda_v + \mu_h)dt, \quad (2.6)$$

Thus, Integrating (2.6) with respect to t in $[0, t_1]$, yields

$$S_h(t_1) \geq S_h(0) \exp - \left\{ \int_0^{t_1} \lambda_v dt + \mu_h t \right\} > 0, \forall t > 0. \quad (2.7)$$

Hence $S_h(t) > 0, \forall t > 0$.

Using similar approach, it can be shown that the other state variables of the model (2.3) remain positive for all $t > 0$.

Theorem 2: Let $(S_h(t), E_h(t), A_h(t), I_h(t), T_h(t), S_v(t), E_v(t), I_v(t))$ be trajectories of the system (2.3) with initial conditions and the biological feasible regions given by the set $\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_v$ where $\mathcal{D}_h = \{(S_h, E_h, A_h, I_h, T_h) \in \mathbb{R}_+^5 : N_h \leq \frac{\Lambda_h}{\mu_h}\}$ $\mathcal{D}_v = \{(S_v, E_v, I_v) \in \mathbb{R}_+^3 : N_v \leq \frac{\Lambda_v}{\mu_v}\}$ is positively-invariant and attracts all the positive trajectories of model (2.3).

Proof:

Adding up the first five equations of the right hand side of (2.3), yields

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h. \quad (2.8)$$

From (2.8), it follows that $\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$. Hence, $\frac{dN_h}{dt} \leq 0$ if $N_h(t) \geq \frac{\Lambda_h}{\mu_h}$. A standard comparison theorem in [16] can be used to show that

$$N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}). \quad (2.9)$$

In particular, if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ for all $t > 0$. Similarly, it can also be shown for the total vector population. And if $N_v(0) \leq \frac{\Lambda_v}{\mu_v}$, then $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$ for all $t > 0$. Hence, the set $\mathcal{D}$ is positively invariant.

Moreover, if $N_h(0) > \frac{\Lambda_h}{\mu_h}$, and $N_v(0) > \frac{\Lambda_v}{\mu_v}$, then either the orbits enters the domain $\mathcal{D}$ in finite time or $N_h(t)$ asymptotically approaches $\frac{\Lambda_h}{\mu_h}$ as $t \rightarrow \infty$. and $N_v(t)$ asymptotically approaches $\frac{\Lambda_v}{\mu_v}$ as $t \rightarrow \infty$. Thus, the domain $\mathcal{D}$ attracts all trajectories in $\mathbb{R}_+^8$. Since the domain $\mathcal{D}$ is positively-invariant, it is enough to study the dynamics of the flows generated by the system (2.3) in $\mathcal{D}$.

We conclude, therefore, that the model (2.3) is both mathematically and epidemiologically well posed. $\square$

3 Local Asymptotic Stability (LAS) of the Disease Free Equilibrium (DFE)

The DFE of the model (2.3) is given by

$$\mathcal{E}_0 = (S_h^0, E_h^0, A_h^0, I_h^0, T_h^0, S_v^0, E_v^0, I_v^0) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right). \quad (3.1)$$

Applying the method in [17], we investigate the LAS of the DFE:

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\beta_v \mu_v \Lambda_h \sigma_v \sigma_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\eta_1 \beta_h \mu_h \Lambda_v \sigma_v \sigma_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v} & \frac{\eta_{h1} \beta_h \mu_h \Lambda_v \sigma_v \sigma_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad (3.2)$$

and

$$V = \begin{bmatrix} (k_1 + \mu_h) & 0 & 0 & 0 & 0 \\ -k_1 & (k_2 + \mu_h) & 0 & 0 & 0 \\ 0 & -k_2 & (\tau_{h1} + \delta_L + \mu_h) & 0 & 0 \\ 0 & 0 & 0 & (k_v + \mu_v) & 0 \\ 0 & 0 & 0 & -k_v & \mu_v \end{bmatrix}, \quad (3.3)$$

The basic reproduction number is

$$\rho(FV^{-1}) = \mathcal{R}_L = \sqrt{R_1 R_2} \quad (3.4)$$

where $R_1 = \frac{\beta_v \sigma_v \sigma_h k_v \Lambda_h}{g_4 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)}$.

$R_2 = \frac{\beta_h \sigma_v \sigma_h \mu_h \Lambda_v}{g_4 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \left[\frac{\eta_1}{g_1} + \frac{\eta_{h1} k_1}{g_1 g_2} + \frac{k_1 k_2}{g_1 g_2 g_3} \right]$,
and $g_1 = (k_1 + \mu_h)$, $g_2 = (k_2 + \mu_h)$, $g_3 = (\tau_{h1} + \delta_L + \mu_h)$, $g_4 = (k_v + \mu_v)$,
 R_1 and R_2 are highly dependent on the biting rate $\frac{\sigma_v \sigma_h}{(\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)}$

Using Theorem 2 in [17], we state the following:

Lemma 1: The DFE of the model (4.5) is LAS in $\mathcal{D}$ if $\mathcal{R}_L < 1$, and unstable if $\mathcal{R}_L > 1$.

3.0.1 Implication Of Lemma 1

- LF can be eradicated from a population *where there is diagnosis of both asymptomatic and symptomatic LF cases*, when $\mathcal{R}_L < 1$.
- This implies that if a small number of infectious LF patients enters a population, no outbreak of the disease in the population where LF control programmes are already being implemented.

The threshold quantity R_0 is the effective reproduction number for lymphatic *filariasis*. It represent the average number of secondary cases that one infectious individual (or mosquito) would generate over the duration of the infectious period if introduced into a susceptible human (or mosquito) population.

3.1 Epidemiological Interpretation of the terms of R_0

The epidemiological interpretation of the terms in the effective reproduction number R_L is as follows. Susceptible humans S_h acquire lymphatic *filariasis* infection following effective contact with an infected mosquito I_v . The number of human infection generated by an infected mosquito is the product of the infectious rate of infected mosquito, the probability that an exposed mosquito survives the exposed stage and moves to the infectious stage, and the average life expectancy of the infected mosquito.

The average number of new human infection is given as

$$\frac{\beta_v \sigma_v \sigma_h S_h^0}{\sigma_v N_v^0 + \sigma_h N_h^0} * \frac{k_v}{g_4} * \frac{1}{\mu_v} \quad (3.5)$$

Using the fact that $N_h^0 = S_h^0 = \frac{\Lambda_h}{\mu_h}$ and $N_v^0 = S_v^0 = \frac{\Lambda_v}{\mu_v}$, we have

$$\frac{\beta_v \sigma_v \sigma_h k_v \Lambda_h}{g_4 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \quad (3.6)$$

Equation (3.6) represents the total number of secondary lymphatic *filariasis* infection in human caused by one infected mosquito.

Similarly, susceptible mosquito S_v acquire lymphatic *filariasis* parasite infection following effective contact with an exposed human E_h , asymptomatic human A_h and infectious human I_h . The number of mosquito infections generated by an exposed human is given by the product of the infection rate of exposed humans and the average duration in the exposed E_h class.

Thus,

Number of mosquito infection generated by exposed humans is given by

$$\frac{\eta \beta_h \sigma_v \sigma_h S_v^0}{\sigma_v N_v^0 + \sigma_h N_h^0} * \frac{1}{g_1} \quad (3.7)$$

Using the fact that $N_h^0 = S_h^0 = \frac{\Lambda_h}{\mu_h}$ and $N_v^0 = S_v^0 = \frac{\Lambda_v}{\mu_v}$, we have

$$\frac{\eta \beta_h \sigma_v \sigma_h \Lambda_v \mu_h}{g_1 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \quad (3.8)$$

The number of mosquito infection generated by an asymptomatic human A_h is given by the product of the infection rate of asymptomatic humans, the probability that an exposed human survives the exposed stage and moves to the asymptomatic stage and the average duration in the asymptomatic class.

Thus, the average number of mosquito infections generated by the asymptomatic humans is given by

$$\frac{\beta_h \sigma_v \sigma_h S_v^0}{\sigma_v N_v^0 + \sigma_h N_h^0} * \frac{k_1}{g_1} * \frac{1}{g_2} \quad (3.9)$$

Using the fact that $N_h^0 = S_h^0 = \frac{\Lambda_h}{\mu_h}$ and $N_v^0 = S_v^0 = \frac{\Lambda_v}{\mu_v}$,

We have

$$\frac{k_1 \beta_h \sigma_v \sigma_h \mu_h \Lambda_v}{g_1 g_2 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \quad (3.10)$$

Finally, the number of mosquito infections generated by infectious human I_h is given by the infection rate of infectious humans, the probability that an asymptomatic humans survives the asymptomatic stage and moves to the infectious class and the average duration in the infectious class.

Thus, the average number of mosquito infection generated by the infectious humans is given by

$$\frac{\beta_h \sigma_v \sigma_h S_v^0}{\sigma_v N_v^0 + \sigma_h N_h^0} * \frac{k_1 k_2}{g_1 g_2} * \frac{1}{g_3} \quad (3.11)$$

Using the fact that $N_h^0 = S_h^0 = \frac{\Lambda_h}{\mu_h}$ and $N_v^0 = S_v^0 = \frac{\Lambda_v}{\mu_v}$,

We have

$$\frac{k_1 k_2 \beta_h \sigma_v \sigma_h \mu_h \Lambda_v}{g_1 g_2 g_3 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \quad (3.12)$$

The average number of new mosquito infections generated by infected humans (exposed, asymptomatic and infectious) is given by the addition of equations (3.8), (3.10) and (3.12).

That is

$$\frac{\eta \beta_h \sigma_v \sigma_h \Lambda_v \mu_h}{g_1 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} + \frac{k_1 \beta_h \sigma_v \sigma_h \mu_h \Lambda_v}{g_1 g_2 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} + \frac{k_1 k_2 \beta_h \sigma_v \sigma_h \mu_h \Lambda_v}{g_1 g_2 g_3 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \quad (3.13)$$

Simplifying, we have

$$\frac{\beta_h \sigma_v \sigma_h \mu_h \Lambda_v (\eta g_2 g_3 + k_1 g_3 + k_1 k_2)}{g_1 g_2 g_3 (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)} \quad (3.14)$$

The expression in equation (3.14) represents the total number of lymphatic *filariasis* infected mosquitoes caused by (exposed, asymptomatic and infectious humans).

The geometric mean of equations (3.6) and (3.14) gives the effective reproduction number R_L and this is equal to R_0 .

4 Existence Of Endemic Equilibrium Point

We consider a special case of the model (2.3) where $\delta_L = 0$. We now have

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_v S_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \lambda_v S_h + \nu \lambda_v T_h - (k_1 + \mu_h) E_h, \\
\frac{dA_h}{dt} &= k_1 E_h - (k_2 + \mu_h) A_h, \\
\frac{dI_h}{dt} &= k_2 A_h - (\tau_{h1} + \mu_h) I_h, \\
\frac{dT_h}{dt} &= \tau_{h1} I_h - \nu \lambda_v T_h - \mu_h T_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_h S_v - \mu_v S_v, \\
\frac{dE_v}{dt} &= \lambda_h S_v - k_v E_v - \mu_v E_v, \\
\frac{dI_v}{dt} &= k_v E_v - \mu_v I_v,
\end{aligned} \tag{4.1}$$

where

$$\lambda_v^* = \frac{\beta_v \sigma_v \sigma_h I_v^*}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{4.2}$$

and

$$\lambda_h^* = \frac{\beta_h \sigma_v \sigma_h (\eta_1 E_h^* + \eta_{h1} A_h^* + I_h^*)}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{4.3}$$

are the forces of infection at endemic steady state.

Adding up the first five equations and the last three equations in (4.1), we have

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h. \tag{4.4}$$

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v \tag{4.5}$$

It follows that $N_h(t) \rightarrow \frac{\Lambda_h}{\mu_h}$ as $t \rightarrow \infty$ and $N_v(t) \rightarrow \frac{\Lambda_v}{\mu_v}$ as $t \rightarrow \infty$ that is $N_h^*(t) = \frac{\Lambda_h}{\mu_h}$ and $N_v^*(t) = \frac{\Lambda_v}{\mu_v}$,

Let

$$\mathcal{E}_1 = (S_h^*, E_h^*, A_h^*, I_h^*, T_h^*, S_v^*, E_v^*, I_v^*)$$

be the EEP for the mass-action model.

The effective reproduction number now denoted by $\mathcal{R}_{L1}$, is given as:

$$\mathcal{R}_{L1} = \sqrt{\frac{\sigma_h^2 \sigma_v^2 k_v \beta_h \beta_v \Lambda_h \Lambda_v \mu_h (\eta_1 g_2 g_{3a} + k_1 (\eta_{h1} g_{3a} + k_3))}{(\Lambda_h \mu_v \sigma_h + \Lambda_v \mu_h \sigma_v)^2 g_1 g_2 g_{3a} g_4}} \tag{4.6}$$

where $g_1 = (k_1 + \mu_h)$, $g_2 = (k_2 + \mu_h)$, $g_{3a} = (\tau_{h1} + \mu_h)$, $g_4 = (k_v + \mu_v)$

The EEP of the mass-action system is given as:

$$\begin{aligned}
S_h^* &= \frac{\Lambda_h}{\lambda_v^* + \mu_h}, \\
E_h^* &= \frac{\Lambda_h \lambda_v^* g_2 g_{3a} (\nu \lambda_v^* + \mu_h)}{(\lambda_v^* + \mu_h) [g_1 g_2 g_{3a} (\nu \lambda_v^* + \mu_h) - \nu \lambda_v^* \tau_{h1} k_1 k_2]}, \\
A_h^* &= \frac{k_1 \Lambda_h \lambda_v^* g_{3a} (\nu \lambda_v^* + \mu_h)}{(\lambda_v^* + \mu_h) [g_1 g_2 g_{3a} (\nu \lambda_v^* + \mu_h) - \nu \lambda_v^* \tau_{h1} k_1 k_2]}, \\
I_h^* &= \frac{k_1 k_2 \Lambda_h \lambda_v^* (\nu \lambda_v^* + \mu_h)}{(\lambda_v^* + \mu_h) [g_1 g_2 g_{3a} (\nu \lambda_v^* + \mu_h) - \nu \lambda_v^* \tau_{h1} k_1 k_2]}, \\
T_h^* &= \frac{\tau_{h1} k_1 k_2 \Lambda_h \lambda_v^*}{(\lambda_v^* + \mu_h) [g_1 g_2 g_{3a} (\nu \lambda_v^* + \mu_h) - \nu \lambda_v^* \tau_{h1} k_1 k_2]}, \\
S_v^* &= \frac{\Lambda_v}{\lambda_h^* + \mu_v}, \\
E_v^* &= \frac{\Lambda_v \lambda_h^*}{g_4 (\lambda_h^* + \mu_v)}, \\
I_v^* &= \frac{k_v \Lambda_v \lambda_h^*}{\mu_v g_4 (\lambda_h^* + \mu_v)}
\end{aligned} \tag{4.7}$$

where

$$\lambda_v^* = \frac{\beta_v \sigma_v \sigma_h I_v^*}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{4.8}$$

and

$$\lambda_h^* = \frac{\beta_h \sigma_v \sigma_h (\eta_1 E_h^* + \eta_{h1} A_h^* + I_h^*)}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{4.9}$$

are the forces of infections at the EEP, respectively

Substituting the expressions for the EEP in (4.7) into the forces of infection in (4.8), it follows (after several algebraic manipulations) that the EEP of the special case satisfies the following polynomial at the steady:

$$(\lambda_v^*)^2 A + \lambda_v^* B + C = 0. \tag{4.10}$$

where

$$\begin{aligned}
A &= \nu \beta_h \sigma_v \sigma_h \Lambda_h (\eta_1 g_2 g_{3a} + \eta_{h1} k_1 g_{3a} + k_1 k_2) + \mu_v \nu (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v) \\
&\quad [g_{3a} (k_1 + k_2 + \mu_h) + k_1 k_2], \\
B &= \beta_h \sigma_v \sigma_h \Lambda_h \mu_h (\eta_1 g_2 g_{3a} + \eta_{h1} k_1 g_{3a} + k_1 k_2) + g_1 g_2 g_{3a} (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v) \\
&\quad + \nu \mu_h (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v) [g_{3a} (k_1 + k_2 + \mu_h) + k_1 k_2], \\
&\quad - \nu g_1 g_2 g_{3a} (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v) \mathcal{R}_{L1}^2, \\
C &= \mu_h g_1 g_2 g_{3a} (\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v) (1 - \mathcal{R}_{L1}^2),
\end{aligned} \tag{4.11}$$

The components of the EEP are then obtained by solving for λ_v^* from the polynomial (4.10).

The number of positive roots of (4.10) depends on the sign of B and C , since $A > 0$

The following results can be deduced.

Theorem 3 The model (4.1) with $\delta_L = 0$ has

- i. two endemic equilibria if $B < 0$ and $\mathcal{R}_L < 1$.
- ii. unique endemic equilibrium if $B > 0$ and $\mathcal{R}_L > 1$.
- iii. no endemic equilibrium otherwise, when $\mathcal{R}_L < 1$.

Item 1 of Theorem 3 suggests the possibility of backward bifurcation (BB) in the model (4.1) with $\delta_L = 0$. This is a significant result because models of vector-borne diseases have been established in literature that disease-induced death is responsible for backward bifurcation. ([18], [19], [20])

5 Bifurcation Analysis

Here, we investigate the possibility of the co-existence of a stable DFE with a stable EEP when $R_{L1} < 1$. We claim the following

Theorem 4 The model (4.1) with $\delta_L = 0$ exhibit backward bifurcation at $\mathcal{R}_{L1} = 1$ whenever a bifurcation coefficient, denoted by α is positive.

Proof:

Consider the case with $\beta_v = \beta_v^*$, a bifurcation parameter. Solving for $\beta_v = \beta_v^*$ from $\mathcal{R}_{L1} = 1$ yields

$$\beta_v^* = \frac{(\Lambda_h \mu_v \sigma_h + \Lambda_v \mu_h \sigma_v)^2 g_1 g_2 g_{3a} g_4}{\sigma_h^2 \sigma_v^2 k_v \beta_h \Lambda_h \Lambda_v \mu_h (\eta_1 g_2 g_{3a} + k_1 (\eta_{h1} g_{3a} + k_2))}. \quad (5.1)$$

Let $S_h = x_1$, $E_h = x_2$, $A_h = x_3$, $I_h = x_4$, $T_h = x_5$, $S_v = x_6$, $E_v = x_7$, and $I_v = x_8$. It follows, that the model (4.1) with $\delta_L = 0$ can be re-written as

$$\begin{aligned} \dot{x}_1 &\equiv f_1 = \Lambda_h - \lambda_v x_1 - \mu_h x_1, \\ \dot{x}_2 &\equiv f_2 = \lambda_v x_1 + \nu \lambda_v x_5 - g_1 x_2, \\ \dot{x}_3 &\equiv f_3 = k_1 x_2 - g_2 x_3, \\ \dot{x}_4 &\equiv f_4 = k_2 x_3 - g_{3a} x_4, \\ \dot{x}_5 &\equiv f_5 = \tau_{h1} x_5 - \mu_h x_5 - \nu \lambda_v x_5, \\ \dot{x}_6 &\equiv f_6 = \Lambda_v - \lambda_h x_6 - \mu_v x_6, \\ \dot{x}_7 &\equiv f_7 = \lambda_h x_6 - g_4 x_7, \\ \dot{x}_8 &\equiv f_8 = k_v x_7 - \mu_v x_8, \end{aligned} \quad (5.2)$$

where $g_1 = k_1 + \mu_h$, $g_2 = k_2 + \mu_h$, $g_{3a} = \tau_{h1} + \mu_h$, $g_4 = k_v + \mu_v$

The Jacobian of the transformed system at the DFE with $\beta_v = \beta_v^*$, is given by:

$$J_{\beta_v^*} = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & \sigma & 0 & 0 & -G_1 \\ 0 & -g_1 & 0 & 0 & 0 & 0 & 0 & G_1 \\ 0 & k_1 & -g_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_2 & -g_{3a} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_{h1} & -\mu_h & 0 & 0 & 0 \\ 0 & -\eta_1 G_2 & -\eta_{h1} G_2 & -G_2 & 0 & -\mu_v & 0 & 0 \\ 0 & \eta_1 G_2 & \eta_{h1} G_2 & G_2 & 0 & 0 & -g_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_v & -\mu_v \end{pmatrix}. \quad (5.3)$$

where $G_1 = \frac{\beta_v \mu_v \Lambda_h \sigma_v \sigma_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v}$ and $G_2 = \frac{\beta_h \mu_h \Lambda_v \sigma_v \sigma_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v}$. The matrix $J_{\beta_v^*}$ has a **simple zero eigenvalue** and all other eigenvalues have negative real part.

Eigenvectors of $J_{\beta_v^*}$

The Jacobian $J_{\beta_v^*}$ has a right eigenvector $\mathbf{w} = (w_1, w_2, \dots, w_8)^T$, where

$$\begin{aligned} w_1 &= \frac{G_1}{\mu_h} w_8, \\ w_2 &= \frac{G_1}{g_1} w_8, \\ w_3 &= \frac{k_1 G_1}{g_1 g_2} w_8, \\ w_4 &= \frac{k_1 k_2 G_1}{g_1 g_2 g_{3a}} w_8, \\ w_5 &= \frac{k_1 k_2 \tau_{h1} G_1}{g_1 g_2 g_{3a} \mu_h} w_8 \\ w_6 &= -\frac{G_1 G_2}{g_1 \mu_v} \left[\eta_1 + \frac{\tau_{h1} k_1}{g_2} + \frac{k_1 k_2}{g_2 g_{3a}} \right] w_8, \\ w_7 &= -\frac{G_1 G_2}{g_1 g_4} \left[\eta_1 + \frac{\tau_{h1} k_1}{g_2} + \frac{k_1 k_2}{g_1 g_4} \right] w_8 \\ w_8 &= w_8 > 0 \end{aligned} \tag{5.4}$$

The Jacobian $J_{\beta_v^*}$ has a left eigenvector $\mathbf{V} = (v_1, v_2, \dots, v_8)^T$, where

$$\begin{aligned} v_1 &= 0, v_5 = 0, v_6 = 0. \\ v_2 &= \frac{k_v G_2}{g_1 g_4} \left[\frac{k_1}{g_2} \left(\frac{k_2}{g_{3a}} + \eta_{h1} \right) + \eta_1 \right] v_8, \\ v_3 &= \frac{k_v G_2}{g_2 g_4} \left[\frac{k_2}{g_{3a}} + \eta_{h1} \right] v_8, \\ v_4 &= \frac{k_v G_2}{g_{3a} g_4} v_8, \\ v_7 &= \frac{k_v}{g_4} v_8, \\ v_8 &= v_8 > 0 \end{aligned} \tag{5.5}$$

Applying the *Center Manifold Theory* in [14], we compute the associated non-zero partial derivatives of the right hand sides of the transformed system (5.2), (evaluated at the DFE with $\beta_v = \beta_v^*$) that the associated bifurcation coefficients, a and b , are given by

$$a = \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \quad \text{and} \quad b = \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0), \tag{5.6}$$

Computation of a

For the system (5.2), the associated non-zero partial derivatives are given by:

It follows from the above expressions, **(after several algebraic manipulations)**, that

$$\begin{aligned}
a &= v_2 \sum_{i,j=1}^8 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + v_7 \sum_{i,j=1}^8 w_i w_j \frac{\partial^2 f_4}{\partial x_i \partial x_j}, \\
&= \frac{2\sigma_h \sigma_v \mu_v \mu_h}{(\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)^2} \left[v_2 w_5 w_8 \beta_v \sigma_h \Lambda_h \mu_v (v-1) + v_7 w_6 \beta_h \sigma_h \Lambda_h \mu_v \right. \\
&\quad (\eta_1 w_2 + \eta_{h1} w_3 + w_4) + v_2 w_8 \beta_v \sigma_v \Lambda_v \mu_h (w_1 + \nu w_5) - (w_1 + w_2 + w_3 + w_4 + w_5) \\
&\quad [v_7 w_2 \beta_h \eta_1 \sigma_h \mu_h \Lambda_v + v_7 w_3 \beta_h \eta_{h1} \sigma_h \mu_h \Lambda_v + v_7 w_4 \beta_h \sigma_h \mu_h \Lambda_v] - (w_7 + w_8) \\
&\quad [v_7 w_2 \beta_h \eta_1 \sigma_v \mu_h \Lambda_h + v_7 w_3 \beta_h \eta_{h1} \sigma_v \mu_h \Lambda_h + v_7 w_4 \beta_h \sigma_v \mu_h \Lambda_v] - v_2 w_8 \beta_v \sigma_h \mu_v \Lambda_h (w_2 + w_3 \\
&\quad \left. + w_4) - v_2 w_8 \beta_v \sigma_v \mu_v \Lambda_h (w_6 + w_7 + w_8) \right].
\end{aligned} \tag{5.8}$$

Computation of b using $\beta_v = \beta_v^*$ as a bifurcation parameter gives

$$\frac{\partial^2 f_2}{\partial x_8 \partial \beta_v^*} = \frac{\sigma_v \sigma_h \mu_v \mu_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v} \tag{5.9}$$

It follows also from the above expressions that

$$\begin{aligned}
b &= v_2 \sum_{i=1}^8 w_i \frac{\partial^2 f_2}{\partial x_i \partial \beta_v^*}, \\
&= v_2 w_8 \frac{\sigma_v \sigma_h \mu_v \mu_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v}.
\end{aligned} \tag{5.10}$$

Obviously $b > 0$ for all biologically feasible parameter values.

Hence, backward bifurcation occurs if and only if $\mathbf{a} > 0$. □

5.1 Non-existence of backward bifurcation

Consider the special case of the model (2.3) with $\delta_L = 0$ and negligible re-infection rate (i.e., $\nu = 0$). Then the backward bifurcation coefficient, a , given by equation (5.8) reduces to:

$$\begin{aligned}
a &= - \frac{2\sigma_h \sigma_v \mu_v \mu_h}{(\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v)^2} \left[v_2 w_5 w_8 \beta_v \sigma_h \Lambda_h \mu_v + v_7 w_6 \beta_h \sigma_h \Lambda_h \mu_v \right. \\
&\quad (\eta_1 w_2 + \eta_{h1} w_3 + w_4) + v_2 w_8 \beta_v \sigma_v \Lambda_v \mu_h w_1 + (w_1 + w_2 + w_3 + w_4 + w_5) \\
&\quad [v_7 w_2 \beta_h \eta_1 \sigma_h \mu_h \Lambda_v + v_7 w_3 \beta_h \eta_{h1} \sigma_h \mu_h \Lambda_v + v_7 w_4 \beta_h \sigma_h \mu_h \Lambda_v] + (w_7 + w_8) \\
&\quad [v_7 w_2 \beta_h \eta_1 \sigma_v \mu_h \Lambda_h + v_7 w_3 \beta_h \eta_{h1} \sigma_v \mu_h \Lambda_h + v_7 w_4 \beta_h \sigma_v \mu_h \Lambda_v] \\
&\quad \left. + v_2 w_8 \beta_v \sigma_h \mu_v \Lambda_h (w_2 + w_3 + w_4) + v_2 w_8 \beta_v \sigma_v \mu_v \Lambda_h (w_6 + w_7 + w_8) \right] < 0.
\end{aligned} \tag{5.11}$$

Hence, this study has confirmed that the presence of re-infection induces backward bifurcation in the transmission dynamics of LF in a system where there are negligible disease-induced deaths. Hence, as was previously postulated in literature, the absence of disease-induced death does not necessarily rule out the possibility of a backward bifurcation as seen from this analysis.

6 Global Asymptotic Stability (GAS) OF EEP

In this subsection, we consider the global asymptotic stability (GAS) of the endemic equilibrium point (EEP) of model (2.3). We consider a special case where $\nu = \delta_L = 0$, so that equation (2.3) becomes

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h - \lambda_v S_h - \mu_h S_h, \\
 \frac{dE_h}{dt} &= \lambda_v S_h - (k_1 + \mu_h) E_h, \\
 \frac{dA_h}{dt} &= k_1 E_h - (k_2 + \mu_h) A_h, \\
 \frac{dI_h}{dt} &= k_2 A_h - (\tau_{h1} + \mu_h) I_h, \\
 \frac{dT_h}{dt} &= \tau_{h1} I_h - \mu_h T_h, \\
 \frac{dS_v}{dt} &= \Lambda_v - \lambda_h S_v - \mu_v S_v, \\
 \frac{dE_v}{dt} &= \lambda_h S_v - k_v E_v - \mu_v E_v, \\
 \frac{dI_v}{dt} &= k_v E_v - \mu_v I_v,
 \end{aligned} \tag{6.1}$$

Since T_h does not appear in the other equations in system (6.1), we only need to consider the reduced system given below

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h - \lambda_v S_h - \mu_h S_h, \\
 \frac{dE_h}{dt} &= \lambda_v S_h - (k_1 + \mu_h) E_h, \\
 \frac{dA_h}{dt} &= k_1 E_h - (k_2 + \mu_h) A_h, \\
 \frac{dI_h}{dt} &= k_2 A_h - (\tau_{h1} + \mu_h) I_h, \\
 \frac{dS_v}{dt} &= \Lambda_v - \lambda_h S_v - \mu_v S_v, \\
 \frac{dE_v}{dt} &= \lambda_h S_v - k_v E_v - \mu_v E_v, \\
 \frac{dI_v}{dt} &= k_v E_v - \mu_v I_v,
 \end{aligned} \tag{6.2}$$

where

$$\lambda_v^* = \frac{\beta_v \sigma_v \sigma_h I_v^*}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{6.3}$$

and

$$\lambda_h^* = \frac{\beta_h \sigma_v \sigma_h (\eta_1 E_h^* + \eta_{h1} A_h^* + I_h^*)}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{6.4}$$

are the forces of infection at endemic steady state.

We claim the following

Theorem 5: The unique EEP, $\mathcal{E}_2$, of model (6.2) with $\nu = \delta_L = 0$ is GAS in $\mathcal{D} \setminus \mathcal{D}_0$ whenever $\mathcal{R}_{L1} > 1$.

Proof:

In proving the GAS of the EEP of system (6.2), we consider the following non-linear Lyapunov function

$$\begin{aligned} \mathcal{F} = & S_h - S_h^* - S_h^* \ln \left(\frac{S_h}{S_h^*} \right) + E_h - E_h^* - E_h^* \ln \left(\frac{E_h}{E_h^*} \right) \\ & + A_1 \left[A_h - A_h^* - A_h^* \ln \left(\frac{A_h}{A_h^*} \right) \right] + A_2 \left[I_h - I_h^* - I_h^* \ln \left(\frac{I_h}{I_h^*} \right) \right] \\ & + S_v - S_v^* - S_v^* \ln \left(\frac{S_v}{S_v^*} \right) + E_v - E_v^* - E_v^* \ln \left(\frac{E_v}{E_v^*} \right) \\ & + A_3 \left[I_v - I_v^* - I_v^* \ln \left(\frac{I_v}{I_v^*} \right) \right], \end{aligned} \quad (6.5)$$

where

$$A_1 = \frac{\tilde{\beta}_h S_v^* \eta_{h1}}{k_2 + \mu_h}, \quad A_2 = \frac{\tilde{\beta}_h S_v^*}{\tau_{h1} + \mu_h}, \quad A_3 = \frac{\tilde{\beta}_v S_h^*}{\mu_v} \quad (6.6)$$

$\mathcal{F}$ has Lyapunov derivatives, given as:

$$\begin{aligned} \dot{\mathcal{F}} = & \left(1 - \frac{S_h^*}{S_h} \right) \dot{S}_h + \left(1 - \frac{E_h^*}{E_h} \right) \dot{E}_h + A_1 \left(1 - \frac{A_h^*}{A_h} \right) \dot{A}_h + A_2 \left(1 - \frac{I_h^*}{I_h} \right) \dot{I}_h \\ & + \left(1 - \frac{S_v^*}{S_v} \right) \dot{S}_v + \left(1 - \frac{E_v^*}{E_v} \right) \dot{E}_v + A_3 \left(1 - \frac{I_v^*}{I_v} \right) \dot{I}_v. \end{aligned} \quad (6.7)$$

Substituting the right hand side of the equations of model (6.2) into (6.7), we have:

$$\begin{aligned} \dot{\mathcal{F}} = & \left(1 - \frac{S_h^*}{S_h} \right) (\Lambda_h - \bar{\beta}_v I_v^* S_h^* - \mu_h S_h^*) + \left(1 - \frac{E_h^*}{E_h} \right) (\bar{\beta}_v I_v^* S_h^* - (k_1 + \mu_h) E_h) \\ & + A_1 \left(1 - \frac{A_h^*}{A_h} \right) (k_1 E_h - (k_2 + \mu_h) A_h) + A_2 \left(1 - \frac{I_h^*}{I_h} \right) (k_2 A_h - (\tau_{h1} + \mu_h) I_h) \\ & + \left(1 - \frac{S_v^*}{S_v} \right) (\Lambda_v - \bar{\beta}_h S_v (\eta_{11} E_h + \eta_{h1} A_h + I_h) - \mu_v S_v) \\ & + \left(1 - \frac{E_v^*}{E_v} \right) (\bar{\beta}_h S_v (\eta_{11} E_h + \eta_{h1} A_h + I_h) - (k_v + \mu_v) E_v) \\ & + A_3 \left(1 - \frac{I_v^*}{I_v} \right) (k_v E_v - \mu_v I_v). \end{aligned} \quad (6.8)$$

Using equation (6.6) and simplifying, we have

$$\begin{aligned}
\dot{\mathcal{F}} = & \mu_h S_h^* \left(2 - \frac{S_h}{S_h^*} - \frac{S_h^*}{S_h} \right) + \mu_v S_v^* \left(2 - \frac{S_v}{S_v^*} - \frac{S_v^*}{S_v} \right) \\
& + \tilde{\beta}_v S_h^* I_v^* \left(3 - \frac{S_h^*}{S_h} - \frac{E_v I_v^*}{E_v^* I_v} - \frac{I_v S_v E_v^*}{I_v^* S_h^* E_v} \right) \\
& + \tilde{\beta}_h S_v^* I_h^* \left(3 - \frac{S_v^*}{S_v} - \frac{A_h I_h^*}{A_h^* I_h} - \frac{I_h S_v A_h^*}{I_h^* S_v^* A_h} \right) \\
& + \tilde{\beta}_h S_v^* A_h^* \eta_{h1} \left(3 - \frac{S_v^*}{S_v} - \frac{E_h A_h^*}{E_h^* A_h} - \frac{S_v A_h E_h^*}{S_v^* A_h^* E_h} \right) \\
& + \tilde{\beta}_h S_v^* E_h^* \eta_{h1} \left(3 - \frac{S_v^*}{S_v} - \frac{E_v E_h^*}{E_v^* E_h} - \frac{S_v E_h E_v^*}{S_v^* E_h^* E_v} \right)
\end{aligned} \tag{6.9}$$

Finally, using the arithmetic mean geometric mean relation, the following inequalities hold:

$$\begin{aligned}
2 - \frac{S_h}{S_h^*} - \frac{S_h^*}{S_h} & \leq 0, \\
2 - \frac{S_v}{S_v^*} - \frac{S_v^*}{S_v} & \leq 0, \\
3 - \frac{S_h^*}{S_h} - \frac{E_v I_v^*}{E_v^* I_v} - \frac{I_v S_h E_v^*}{I_v^* S_h^* E_v} & \leq 0, \\
3 - \frac{S_v^*}{S_v} - \frac{A_h I_h^*}{A_h^* I_h} - \frac{I_h S_v A_h^*}{I_h^* S_v^* A_h} & \leq 0, \\
3 - \frac{S_v^*}{S_v} - \frac{E_h A_h^*}{E_h^* A_h} - \frac{S_v A_h E_h^*}{S_v^* A_h^* E_h} & \leq 0, \\
3 - \frac{S_v^*}{S_v} - \frac{E_v E_h^*}{E_v^* E_h} - \frac{S_v E_h E_v^*}{S_v^* E_h^* E_v} & \leq 0.
\end{aligned} \tag{6.10}$$

So that $\dot{\mathcal{F}} \leq 0$ for $\mathcal{R}_{L1} > 1$. Considering the system (6.2) at EEP, the necessary state variables can be substituted into the equation for T_h , hence $T_h(t)$ approaches T_h^* as $t \rightarrow \infty$. Hence, $\dot{\mathcal{F}}$ is a Lyapunov function of system (6.2) with $\nu = \delta_L = 0$ on D/D_O .

Thus, by the Lyapunov function $\mathcal{F}$ and Lasalle's Invariance Principle [21], every solution to the equations in the reduced model (6.2), with $\nu = \delta_L = 0$ approaches $\mathcal{E}_2$ as $t \rightarrow \infty$ for $\mathcal{R}_{L1} > 1$.

7 Results and Discussion

We developed a new compartmental model to describe the transmission dynamics of lymphatic *filariasis*. We found out that, the disease-free state was globally asymptotically stable when the reproduction number is less than unity; indicating that lymphatic *filariasis* cannot successfully invade the community. When the reproduction number was greater than one, the endemic state was globally stable. We established that the model exhibit backward bifurcation even when the disease-induced death was negligible. We confirmed that the presence of re-infection induces backward bifurcation in the transmission dynamics of lymphatic *filariasis* in a system where there are negligible disease-induced deaths. This is a significant result because models of vector-borne diseases (Malaria, Dengue, West Nile Virus etc.) have been established in literature that disease-induced death is responsible for backward bifurcation ([18], [19], [20]). We conclude that the absence of disease-induced death does not necessarily rule out the possibility of a backward bifurcation.

8 Conclusion

In this paper, we confirmed that the presence of re-infection induces backward bifurcation in the transmission dynamics of Lymphatic *filariasis* in a system where there are negligible disease-induced deaths. Hence, as was previously postulated in literature, the absence of disease-induced death does not necessarily rule out the possibility of a backward bifurcation. We also showed that the unique endemic equilibrium point (EEP) with negligible re-infection and disease-induced death is globally asymptotically stable (GAS) whenever $\mathcal{R}_{L1} > 1$.

9 Abbreviations

DFE: Disease-free equilibrium
EEP: Endemic equilibrium point
GAS: Global asymptotic stability
GPELE: Global program for the elimination of LF
LF: Lymphatic *filariasis*
LYMFASIM: Lymphatic *filariasis* simulation
MDA: Mass drug administration
ODE: Ordinary differential equation
WHO: World health organization

10 Declaration

10.1 Availability of Data and Material

All data used during this study are included in this article.

10.2 Competing Interest

The authors declared that they have no competing interests.

10.3 Funding

This research is partially supported by the European Mathematical Society (EMS-Simons for Africa) collaborative research visit grant (Type 2) to CRM, Barcelona, Spain. The funder partially supported my accommodation and feeding during my stay at the Centre for Research in Mathematics (CRM).

10.4 Authors' contributions

EI formulated the model equations, analysed the existence of the endemic equilibrium point, bifurcation analysis and was a major contributor in writing the manuscripts. DO and FO jointly analysed the global stability of the endemic equilibrium point. All authors read and approve the final manuscript.

10.5 Acknowledgement

Not applicable

References

- [1] Li X. X. and Zhou, X. N. (2013). Coinfection of tuberculosis and parasitic diseases in humans; a systematic review. *Parasite and vectors* 6:79
- [2] WHO, (2018) Accessed August 23, 2018,
- [3] Ukaga C.N., Nwoke B.E.B., Nwoke E. A.and Nwachukwu M.J., *Epidemiological characteristics of Bancrofti filariasis and the Nigerian environment.*, *Journal of Publi Health and Epidemiology*.vol.2(6), 2010.
- [4] Iddi, A. J., Massane, E. S. Kgosimore M., Nkwengulik, G. (2016a): Modeling the impact of Multi-interventions campaign on LF disease. *IJMTER* vol. 3. Issue 02.
- [5] Iddi, A. J., Estomih Massawe, Gamba, N. and Kgosimore, M. (2016b). The effect of Chemoprophylaxis for Exposed individuals on lymphatic filariasis model. *Applied and Computational Mathematics*. 5(1): 30–39.
- [6] Yan Cheng; Wang Xiaogun; Pan Qiuhui and He Mingfeng (2017). Modeling the Parasitic Filariasis spread by mosquito in periodic environment. *Computational and Mathematical Methods in Medicine*. Vol. 2017, Article ID 4567452. <http://doi.org/10.1155/2017/4567452>.
- [7] Jambulingam, P. Subramanian, S. De Vlas, S. J. Vinubala, C. and Stolk, W. A. (2015). Mathematical modeling of lymphatic filariasis elimination programs in India: required duration of mass drug administration and post treatment of infection indicators. *Parasites and vectors* vol. 2015.
- [8] Kastner, R. J., Stone, C. M., Steinmann, P. Tanner, M. and Techosi, F. (2015) What is needed to eradicate lymphatic filariasis? A model-based assessment on the impact of scaling up mass drug administration programs. *PLOS Neglected tropical* Vol. 9(10).
- [9] Bockarie, M. J., Pedersen, E. M., White, G. B. and Michael, E. (2009). Role of Vector control in the global program to eliminate lymphatic filariasis. *Annu. Rev. Entomol* 4: 469–487. Doc:10.1146/annu-rev.ento.54.110807.090626.
- [10] Michael, E. and Ganibnir, M. (2010): Vector transmission heterogeneity and the population dynamics and control of lymphatic filariasis. *Adv. Exp. Med. Biol.* 673:13–31.
- [11] Nwantobe, P. M. Simelane, S. M., Abelmay, S. and Tchuench, J. M. (2017): Mathematical Analysis of a Lymphatic filariasis Model with Quarantine and Treatment. *BMC Public Health*. 17. 265.
- [12] Blower, S.M. and Dowlatabadi, H. (1994). Sensitivity and Uncertainty Analysis of Complex Models of Disease Transmission: An HIV Model, as an Example. *International Statistical Review*. 62, No. 2: pp. 229–243
- [13] Carr, J. (1981). *Application of Center Manifold Theory*. Springer-Verlag, New York.
- [14] Castillo-Chavez, C. and Song B. (2004): *Dynamical Models of Tuberculosis and their Applications* *Math. Bio, and Eng.*, 1, No2, 361–404.
- [15] Fleming, W.H. and Rishel, R.W. (1975). *Deterministic and Stochastic Optimal Control*. Springer, New York.
- [16] Lakshmikantham V., Leela S., and Martnyuk A. A. (1989): *Analysis of Nonlinear Systems*. Marces Dekker, Inc., New York.
- [17] Van den Driesscle, P. and Watmough, J. (2002): Reproduction numbers and sub-threshold endemic equilibrium for compartmental models of disease transmission. *Math. Biosc.*, 180:29–48.

- [18] Garba, S. M., Gumel, A. B. and Bakar, M. R. A. (2008): Backward bifurcation in dengue transmission dynamics. *Math. Biosc.*, 215: 11–25.
- [19] Mukandavire Z., Bowa K., and Garira W. (2007). Modelling Circumcision and Condom use as HIV/AIDS Preventive Control Strategies. *Mathematical and Computer Modelling*. 46 (11):1353–1272.
- [20] Blayneh, K. W., Gumel A.B., Lenhart S., and Clayton T. (2010), *Backward Bifurcation and Optimal control in Transmission Dynamics of West Nile Virus*. *Bulletin of Mathematical Biology* vol.2, no. 23, pp. 1004–1028.
- [21] Lasalle, J. P. (1976): *The stability of Dynamical systems*, vol. 25. SIAM.

11 Appendix

Here, we show how the EEP in (4.7) was obtained.

Setting the right hand side of the first equation in (4.1) to zero and solving for S_h , we have

$$S_h^* = \frac{\Lambda_h}{\lambda_v^* + \mu_h} \quad (11.1)$$

where the * at the superscript indicates values at the steady states.

From the second equation in (4.1), setting the right hand side of this equation to zero, we have

$$E_h^* = \frac{\lambda_v^* S_h^* + \nu \lambda_v^* T_h^*}{k_1 + \mu_h} \quad (11.2)$$

From the third equation in (4.1), setting the right hand side of this equation to zero, we have

$$A_h^* = \frac{k_1 E_h^*}{K_2 + \mu_h} \quad (11.3)$$

From the fourth equation in (4.1), setting the right hand side of this equation to zero, we have

$$I_h^* = \frac{k_2 A_h^*}{\nu \lambda_v^* + \mu_h} \quad (11.4)$$

From the fifth equation in (4.1), setting the right hand side of this equation to zero, we have

$$T_h^* = \frac{\tau_{h1} I_h^*}{\nu \lambda_v^* + \mu_h} \quad (11.5)$$

Substituting the value of A_h^* in (11.4), we have

$$I_h^* = \frac{k_1 K_2 E_h^*}{(\tau_{h1} + \mu_h)(k_2 + \mu_h)} \quad (11.6)$$

Substituting the new value of I_h^* in (11.5), we have

$$T_h^* = \frac{\tau_{h1}k_1k_2E_h^*}{(\nu\lambda_v^* + \mu_h)(\tau_{h1} + \mu_h)(k_2 + \mu_h)} \quad (11.7)$$

Substituting the value of S_h^* and the new value of T_h^* in (11.2) and after some simplification, we have

$$E_h^* = \frac{\Lambda_h\lambda_v^*g_2g_{3a}(\nu\lambda_v^* + \mu_h)}{(\lambda_v^* + \mu_h)[g_1g_2g_{3a}(\nu\lambda_v^* + \mu_h) - \nu\lambda_v^*\tau_{h1}k_1k_2]} \quad (11.8)$$

where $g_1 = (k_1 + \mu_h)$, $g_2 = (k_2 + \mu_h)$, $g_3 = (\tau_{h1} + \mu_h)$, $g_4 = (k_v + \mu_v)$

Substituting the new value of E_h^* in (11.3), we have

$$A_h^* = \frac{k_1\Lambda_h\lambda_v^*g_{3a}(\nu\lambda_v^* + \mu_h)}{(\lambda_v^* + \mu_h)[g_1g_2g_{3a}(\nu\lambda_v^* + \mu_h) - \nu\lambda_v^*\tau_{h1}k_1k_2]} \quad (11.9)$$

Substituting the new value of A_h^* in (11.4), we have

$$I_h^* = \frac{k_1k_2\Lambda_h\lambda_v^*(\nu\lambda_v^* + \mu_h)}{(\lambda_v^* + \mu_h)[g_1g_2g_{3a}(\nu\lambda_v^* + \mu_h) - \nu\lambda_v^*\tau_{h1}k_1k_2]} \quad (11.10)$$

Substituting the new value of I_h^* in (11.5), we have

$$T_h^* = \frac{\tau_{h1}k_1k_2\Lambda_h\lambda_v^*}{(\lambda_v^* + \mu_h)[g_1g_2g_{3a}(\nu\lambda_v^* + \mu_h) - \nu\lambda_v^*\tau_{h1}k_1k_2]} \quad (11.11)$$

From the sixth equation in (4.1), setting the right hand side to zero, we have

$$S_v^* = \frac{\Lambda_v}{\lambda_h^* + \mu_v} \quad (11.12)$$

From the seventh equation in (4.1), setting the right hand side to zero and substituting the value of S_v^* , we have

$$E_v^* = \frac{\Lambda_v\lambda_h^*}{g_4(\lambda_h^* + \mu_v)} \quad (11.13)$$

From the eighth equation in (4.1), setting the right hand side to zero and substituting the value of E_v^* , we have

$$I_v^* = \frac{k_v\Lambda_v\lambda_h^*}{\mu_vg_4(\lambda_h^* + \mu_v)} \quad (11.14)$$

Figure 1: Model schematic diagram.

Figures

Figure 1

Model schematic diagram.