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Lymphatic Filariasis Model with Prevention and Treatment in Human Under Treatment Barriers

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ABSTRACT

In this paper, a deterministic *Lymphatic Filariasis* (LF) model with preventive measures in human and treatment barriers is developed and analysed to assess the impact of treatment barriers on the transmission dynamics of LF in endemic areas. Qualitative analysis and numerical simulation are presented in terms of the reproduction number of the model in the absence and presence of treatment barriers. It is established that the treatment intervention has shown improvement in the reduction of LF infection in the population. Furthermore, in the absence of treatment barriers the model guaranteed disease extinction behaviour, while in the presence of treatment barriers the model shows disease persistence behaviour when $R_e < 1$. This means that in the presence of treatment barriers there is coexistence of the stable disease-free state and the stable persistent state of the disease when $R_e < 1$. The persistence behaviour may be due to plentiful infected individuals who accumulate in the community due to treatment barriers while the disease has no natural recovery. The numerical simulations are performed to complement the analytical results.

Keywords : Lymphatic Filariasis, Mass Drug Administration (MDA), Treatment Barrier, Reproduction Number

I. INTRODUCTION

Lymphatic Filariasis (LF) is a public health problem causing long term disability in the world as considered by World Health Organization (WHO 2016). Currently, 790 million people are at risk of the disease and 68 million are infected, with a further 20 million suffering from chronic morbidity (Irvine et al., 2017). Chronic infection with mosquito-transmitted filarial worms leads to lymphatic dysfunction, resulting in progressive, irreversible swelling of the limbs, breasts or genitals.

A Global Programme to Eliminate *Lymphatic Filariasis* (GPELF) was initiated in 1998 aimed at the worldwide elimination of LF as a public health problem. (Simonsen *et al.,* 2013).

The control of Lymphatic Filariasis is based on annual Mass Drug Administration (MDA) of the combined drug regime of *albendazole* and *diethyl-carbamazine* or *albendazole* and *ivermectin*) to interrupt the spread of the disease over at least 4-6 years. Drug consumption, as preventive chemotherapy and treatment of infected individuals are crucial factors in the success of Lf elimination program. The goal of MDA is to reduce the density of parasites circulating in the blood of infected persons and the intensity of infection in communities to levels where transmission is no longer sustainable by the mosquito vector (Gyapong et al., 2018). LF elimination is unlikely to be achieved as planned (Morgan et al., 2017) due to delaying in elimination program implementation, compliance, and drug contraindications. This is because a proportion of the population fails to comply

with MDA, and thus a potential reservoir for the parasite is left untreated, opening the door to recrudescence of the microfilaraemia (*mf*) that reduce the probability of the program's success (Dickson et al 2018).

The major challenge with the currently available drugs and their regimens is that the interruption of transmission requires very high treatment coverage (probably >85% of the total population) to achieve elimination (Ambulingam et al.2016). However treatment coverage is much affected by poor community compliance with MDA programme as well as barrier to compliance such as fear of drug side effects, social and cultural beliefs (Jones et al.,2012).
Transmission models exist to describe transmission processes and predict the impact of interventions on transmission and chances of elimination. Three different transmission models (LYMFASIM, EPIFIL and TRANSFIL) have been developed to compare the number of rounds of mass drug administration needed to achieve a prevalence of microfilaraemia less than 1% with the triple-drug regimen and with current two-drug regimens (Arvine *et al.,* 2017).

A number of epidemic models have been developed to study the parasite transmission and control of LF (Malecela *et al.,* 2004; Bani *et al.*, 2013; Iddi *et al.,* 2016, Mwamtobe *et al.,* 2017, Arvine *et al.,* 2017) to mention the few. Simulation modelling suggests that the triple-drug regimen has potential to accelerate the elimination of Lymphatic Filariasis if high population coverage of mass drug administration can be achieved and if systematic non-adherence with mass drug administration is low. None of those mathematical models considered the aspect of treatment barriers in the transmission dynamics of LF disease. This study, aims to develop a mathematical model with preventive measures in human and incorporate saturated function to describe the impact of treatment barriers in the transmission dynamics of LF epidemic in society.

II. MODEL FORMULATION AND ANALYSIS

The Lymphatic Filariasis model with human population under study being divided into 4 compartments and vector population into 3 populations is formulated. Horizontal standard incidence with homogeneous mixing so that individuals have equal chances of acquiring infection is assumed as well as no reinfections. The recruited individuals are assumed to replenish through births or immigration. The model subdivides the human population at time t, $N_h(t)$ into the class of susceptible individuals, $S_h(t)$ (who are individuals at risk of acquiring LF infection upon effective contact). The educated individuals $A(t)$ (education on vector control preventive measures), the latent or exposed class $E_h(t)$ (individual is infected but not infectious), LF infected $I_h(t)$ (infectious) individuals and the removed individuals $R(t)$. The vector (mosquitoes) population at time t $N_v(t)$ is subdivided into, susceptible mosquitoes, $S_v(t)$, the exposed vector (infected mosquitoes but not yet infectious), $E_v(t)$ and the infected vector $I_v(t)$, with the potential to spread LF parasite through effective contact with susceptible humans. The susceptible humans S_h are recruited into the population by birth and immigration at a constant rate Λ. Susceptible individuals are subjected to Mass Drug Administration (MDA) as a preventive chemotherapy and progress to education class A at a rate ϕ (public health education includes use of Insecticides Treated Nets (ITN), Indoor Residual Spraying (IRS) and environmental cleanliness). It is assumed that those who received mass prevention are educated to comply with MDA. A discounted factor Φ is the transmission rate of the education. Clearly, health education will be effective if there would be no progression of individuals from educated class to exposed class (i.e. $\Phi = 0$) and ineffective if $\Phi = 1$. Susceptible human S_h acquire LF through contact

with infected mosquito at a rate λ_h and move to exposed class. The exposed class develops the disease and progresses to infected human individuals at a constant rate ν. Infected human individuals progress to the recovery state R at a saturated treatment function $T(I_h) = \frac{\varepsilon I_h}{1+\sigma I_h}$ where ε is positive and σ is non-negative. The saturated treatment function demonstrates the limitations of implementing the treatment program due to social barriers like ignorance, reluctance, being wary, hence delaying treatment. ε is the cure rate while σ measures the extent of the effect of infected individuals being delayed for treatment. Since the disease has temporary immunity, and assuming that there is no reinfection, the fraction of removed individuals lose their immunity at the rate of $(1-\tau)\omega$ and return to the susceptible class and , while another fraction of the removed individuals who are educated returns to educated class at the rate of $\tau\omega$. All human populations are subject to constant natural death rate μ_h. The disease is not fatal, so there is no death associated with the disease. Susceptible mosquitoes S_v are generated through births at a constant rate π. Susceptible mosquitoes acquire LF through effective contact with infected humans at a rate λ_v. Newly infected mosquitoes move to exposed class E_v and later progress to infectious class I_v at a constant rate of γ. The vector population suffers deaths naturally at a constant rate μ_v. As is the case with humans there is no disease induced death rate in the vector population. We assume standard incidence form of forces of infection $\lambda_h = \frac{\beta_{vh}\alpha I_v}{N_v}$ and $\lambda_v = \frac{\beta_{hv}\alpha I_h}{N_h}$ (Tumwiine *et al,* 2007), of the human hosts and the vector, respectively. The rate of infection of the human host depends on proportion of infected mosquito while that of the vector depends of the fraction of infected human host.

β_{vh} is the probability that a human host becomes infectious and β_{hv} is the probability that susceptible mosquitoes become infected by biting infected humans while α refers to mosquito biting rate. Therefore, the total human population is given by $N_h = S_h + A + E_h + I_h + R$ while that of the vector (mosquito) population is $N_v = S_v + E_v + I_v$. Assuming that all state variables and parameters are positive for all $t \geq 0$, the above assumptions give the following model equations which describe the progress of the disease.

The human population model is given by

$$\frac{dS_h}{dt} = \Lambda + (1-\tau)\omega R - \lambda_h S_h - (\phi + \mu_h)S_h$$

$$\frac{dA}{dt} = \phi S_h + \tau\omega R - (\Phi\lambda_h + \mu_h)A$$

$$\frac{dE_h}{dt} = \lambda_h S_h + \Phi\lambda_h A - (v + \mu_h)E_h$$

$$\frac{dI_h}{dt} = vE_h - (\in + \mu_h)I_h$$

$$\frac{dR}{dt} = \in I_h - (1-\tau)\omega R - (\tau\omega + \mu_h)R$$

where $\in (I_h) = \frac{\varepsilon}{1+\sigma I_h}$

The vector population model is given by

$$\frac{dS_v}{dt} = \pi - (\lambda_v + \mu_v)S_v$$

$$\frac{dE_v}{dt} = \lambda_v S_v - (\gamma + \mu_v)E_v \qquad (1)$$

$$\frac{dI_v}{dt} = \gamma E_v - \mu_v I_v$$

The model represented by the model (1) will be analysed in the feasible region

$$\Gamma_h = \left\{ \left(S_h, A, E_h, I_h, R\right) \in R_+^5 \mid N_h = \frac{\Lambda}{\mu_h} \right\} \quad \text{and}$$

$$\Gamma_v = \left\{ \left(S_v, E_v, I_v\right) \in R_+^3 \mid N_v = \frac{\pi}{\mu_v} \right\}$$

and all state variables and parameters are assumed to be non-negative for all $t \geq 0$ since the model is dealing with a population. The invariant region can be obtained using the differential equation inequality theorem as in [Kelatlhegile and Kgosimore 2015]

$$\lim_{t\to\infty} \sup S_h(t) \leq \frac{\Lambda}{\mu_h}$$

Adding equations of system (1), yields

$$\frac{dN_h}{dt} = \Lambda - \mu_h N_h \text{ and } \frac{dN_v}{dt} = \pi - \mu_v N_v,$$

Integrating the two equations and applying the initial conditions, when $t = 0$

, the result is

$$N_h \leq \frac{\Lambda}{\mu_h} + N_h(0) e^{-\mu_h t}$$

and $$N_v \leq \frac{\pi}{\mu_v} + N_v(0),$$

$$\text{As } t \to \infty,\ 0 \leq N_h \leq \frac{\Lambda}{\mu_h} \text{ and } 0 \leq N_v \leq \frac{\pi}{\mu_v}$$

$$\Gamma = \left\{ \begin{array}{c} \left(S_h, A, E_h, I_h, R, S_v, E_v, I_v\right) \in R_+^8 : \\ S_h, A, E_h, I_h, R, S_v, E_v, I_v \geq 0; \\ N_h \leq \frac{\Lambda}{\mu_h}, \quad N_v \leq \frac{\pi}{\mu_v} \end{array} \right\},$$

This is well posed in the invariant set of the model system (1). Thus Γ is positively invariant meaning that all solutions with initial conditions remain in Γ for $t \geq 0$. The global existence, uniqueness and continuity of the model hold in Γ, making the model to be epidemiologically and mathematically well posed. Thus, the model (1) dynamics is considered in Γ.

Setting the right side of the system (1) to zero in terms of λ_h^* and λ_v^* we obtained the following

$$E_h^* = \frac{\lambda_h^* S_h^* + \Phi \lambda_h^* A^*}{(\nu + \mu_h)} \qquad I_h^* = \frac{\nu E_h^*}{(\in + \mu_h)}$$

$$E_v^* = \frac{1}{(\gamma + \mu_v)} \lambda_v^* S_v^* \quad ,$$

$$I_v^* = \frac{\gamma}{(\gamma + \mu_v)\mu_v} \lambda_v^* S_v^*$$

where $$\in (I_h) = \frac{\varepsilon}{1 + \sigma I_h} \qquad (2)$$

Substitute I_h^* and I_v^* into forces of infection as follows to get:

$$\lambda_h^* = \frac{\alpha \beta_{vh} \gamma}{(\gamma + \mu_v)\mu_v} \left(\frac{S_v^*}{N_v} \right) \lambda_v^* \qquad (3)$$

$$\lambda_v^* = \frac{\alpha \beta_{hv} \nu}{(\nu + \mu_h)} \frac{\alpha \beta_{vh} \gamma}{(\gamma + \mu_v)} \frac{1}{(\in + \mu_h)} \left(\frac{S_h^* + \Phi A}{N_h} \right) \left(\frac{S_v^*}{N_v} \right) \lambda_h^* \qquad (4)$$

Substituting λ_v^* into λ_h^* yields:-

$$\lambda_h^* = \frac{\alpha^2 \beta_{vh} \beta_{hv} \gamma \nu}{(\nu + \mu_h)(\gamma + \mu_v)\mu_v} \frac{1}{(\in + \mu_h)} \left(\frac{S_h^* + \Phi A}{N_h} \right) \frac{S_v^*}{N_v} \lambda_h^*$$

This gives $$\lambda_h^* = 0 \qquad (5)$$

Or

$$\frac{\alpha^2 \beta_{vh} \beta_{hv} \gamma \nu}{(\nu + \mu_h)(\gamma + \mu_v)\mu_v} \frac{1}{(\in + \mu_h)} \left(\frac{S_h^* + \Phi A}{N_h} \right) \frac{S_v^*}{N_v} = 1 \qquad (6)$$

The solution $\lambda_h^* = 0$ leads to the disease-free equilibrium point given by

$$E_0 = \left(\frac{\Lambda}{(\phi + \mu_h)}, \frac{\Lambda \phi}{\mu_h (\phi + \mu_h)}, 0, 0, 0, \frac{\pi}{\mu_v}, 0, 0 \right) \qquad (7)$$

The solution of (6) leads to the endemic equilibrium point given by

$$S_h^* = \frac{\Lambda + (1-\tau)\omega R^*}{\lambda_h^* + (\phi + \mu_h)}$$

$$A^* = \frac{\phi S_h^* + \tau\omega R^*}{\Phi\lambda_h^* + \mu_h} \qquad E_h^* = \frac{(S_h^* + \Phi A^*)\lambda_h^*}{(\nu + \mu_h)}$$

$$I_h^* = \frac{\nu E_h^*}{(\in + \mu_h)}$$

$$R^* = \frac{\in I_h^*}{(\omega + \mu_h)}$$

$$S_v^* = \frac{\pi}{(\lambda_v^* + \mu_v)} \qquad E_v^* = \frac{\lambda_v^* S_v^*}{(\gamma + \mu_v)}$$

$$I_v^* = \frac{\gamma\lambda_v^* S_v^*}{\mu_v(\gamma + \mu_v)} \qquad (8)$$

The basic reproduction number or contact number R_e represents the average number of secondary infections that a single infection host can generate in a totally susceptible population of hosts and vectors. We calculate the basic reproduction number R_e by using the next generation method by (Van den Driessche and Watmough) of the system (1).

$$A = \left[\frac{\partial F_i(E_0)}{\partial x_i}\right].\left[\frac{\partial V_i(E_0)}{\partial x_i}\right]^{-1} = FV^{-1} \qquad (9)$$

where

F_i is the rate of appearance of new infections and $V_i = V_i^- - V_i^+$ is the net rate of transfer of individuals into compartment i, with V_i^- denoting transfer out of compartment i and V_i^+ the transfer of individuals into compartment i. This model has four (4) infected classes, thus $m = 4$; and are ordered as follows: $E_h, I_h, E_v,$ and I_v . The matrices F and V are obtained from model (1) as:-

The Jacobian matrix of **F** and **V** evaluated at E_0 is given by:-

$$\mathbf{F} = \begin{bmatrix} 0 & 0 & 0 & \frac{\alpha\beta_{vh}\Lambda\mu_v(\Phi\phi + \mu_h)}{\pi\mu_h(\phi + \mu_h)} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\alpha\beta_{hv}\pi\mu_h}{\Lambda\mu_v} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

and $\mathbf{V} = \begin{bmatrix} (\nu + \mu_h) & 0 & 0 & 0 \\ -\nu & (\varepsilon + \mu_h) & 0 & 0 \\ 0 & 0 & (\gamma + \mu_v) & 0 \\ 0 & 0 & -\gamma & \mu_v \end{bmatrix}$

R_e is defined as the spectral radius (dominant eigenvalues) of the matrix $\mathbf{FV}^{-1}$ which is

$$R_e = \sqrt{\frac{\alpha^2\beta_{vh}\beta_{hv}\nu\gamma(\Phi\phi + \mu_h)}{(\phi + \mu_h)(\nu + \mu_h)(\varepsilon + \mu_h)(\gamma + \mu_v)\mu_v}}$$

$$= R_{e_h} \times R_{e_v} \qquad (10)$$

As the reproduction number is generated by infections of two populations, therefore we have:-

where

$$R_{e_h} = \frac{\alpha\beta_{vh}\nu(\Phi\phi + \mu_h)}{(\phi + \mu_h)(\nu + \mu_h)(\varepsilon + \mu_h)}$$

and $R_{e_v} = \frac{\alpha\beta_{hv}\gamma}{(\gamma + \mu_v)\mu_v}$

The persistence or disappearance of the disease in the society depends on the magnitude of the basic reproduction number R_e.

The Jacobian matrix of the system (1) is computed by differentiating each equation in system (1) with respect to the state variables and evaluated at E_0 to obtain the characteristic polynomial of $\mathbf{J}_{E_0}$ as $|\mathbf{J}_{E_0} - \lambda I| = 0$ with four explicitly negative eigenvalues μ_h, $(\phi + \mu_h)$, $(\omega + \mu_h)$ and μ_v of $\mathbf{J}_{E_0}$.

The remaining eigenvalues are the roots of $\left(J_{E_0^*}\right)$ the fourth order characteristic equation which their negativity depend on R_e. Excluding these columns and the corresponding rows we have,

$$` J_{E_{01}} = \begin{bmatrix} -a_1 & 0 & 0 & a_5 \\ x & -a_2 & 0 & 0 \\ 0 & a_3 & -a_4 & 0 \\ 0 & 0 & y & -z \end{bmatrix}$$

The remaining eigenvalues are the roots of $\left(J_{E_{01}}\right)$ the fourth order characteristic equation,

$$(\lambda + a_1)(\lambda + a_2)(\lambda + a_4)(\lambda + z) - a_3 a_5 xy = 0$$

where

$$a_1 = (\nu + \mu_h),\ a_2 = (\varepsilon + \mu_h),\ a_3 = \frac{\alpha \beta_{hv} \pi \mu_h}{\Lambda \mu_h},$$

$$a_4 = (\gamma + \mu_v),\ a_5 = \frac{\alpha \beta_{vh} \Lambda \mu_v (\Phi\phi + \mu_h)}{\pi \mu_h (\phi + \mu_h)},$$

$$x = \nu,\ y = \gamma,\ z = \mu_v$$

This reduces to

$$\lambda^4 + P_3\lambda^3 + P_2\lambda^2 + P_1\lambda + P_0 = 0 \qquad (11)$$

where

$$P_3 = (a_1 + a_2 + a_4 + z),$$

$$P_2 = (a_1 + a_2 + a_4 + z + a_1 a_2 + a_4 z)$$

$$P_1 = a_1 a_4 z + a_4^2 z$$

$$P_0 = -a_3 a_5 xy + a_1 a_2 a_4 z = (1 - R_e)(1 + R_e)$$

We apply the Routh-Hurwitz criterion to check the stability of J_{E_0}. The Routh-Hurwitz conditions hold when all the eigenvalues have negative real parts making J_{E_0} to be locally asymptotically stable. For the polynomial (11) Routh –Hurwitz conditions are $P_0 > 0,\ P_1 > 0,\ P_2 > 0$ and $P_3 > 0$.

Thus, $H_1 = P_3 > 0$,

$$H_2 = \begin{vmatrix} P_3 & 1 \\ P_1 & P_2 \end{vmatrix} = P_3 P_2 - P_1 > 0,$$

$$H_3 = \begin{vmatrix} P_3 & 1 & 0 \\ P_1 & P_2 & P_3 \\ 0 & P_0 & P_1 \end{vmatrix} = P_1 (P_2 P_3 - P_1) - P_0 P_3^2 > 0,$$

$$H_4 = \begin{vmatrix} P_3 & 1 & 0 & 0 \\ P_1 & P_2 & P_3 & 1 \\ 0 & P_0 & P_1 & P_2 \\ 0 & 0 & 0 & P_0 \end{vmatrix} = P_0 H_3 > 0$$

Since $a_1 > 0,\ a_2 > 0,\ a_3 > 0,\ a_4 > 0,\ x > 0,\ y > 0$ and $z > 0$, then we check the positivity that

$$H_2 = P_3 P_2 - P_1$$
$$= (a_1 + a_4)(a_2 + z)(a_4 + z) + a_1^2 (a_2 + a_4 + z) + a_1 (a_2 + a_4 + z)^2$$

H_2 is positive quantity, and also

$$H_3 = P_1 (P_2 P_3 - P_1) - P_0 P_3^2$$

$$= (a_1 + a_2)(a_1 + a_4)(a_2 + a_4)(a_1 + z)(a_2 + z)$$
$$+ (a_1 + a_2 + a_4 + z)^2 a_3 a_5 xy$$

Clearly $H_3 > 0$ then $H_4 = P_0 H_3 > 0$.

These conditions guarantee that all roots of the polynomial (11) have negative eigenvalues of the Jacobian J_{E_0}, that is the negative real parts when $R_e < 1$, but when $R_e > 1$ implies that $P_0 < 0$. Moreover, with $P_1 > 0,\ P_2 > 0$ and $P_3 > 0$ means that not all roots of polynomial (11) can have negative real part, which means that when $R_e > 1$ the disease free is unstable. Since all the above conditions hold for J_{E_0} to be stable, then the following theorem holds.

Theorem 1: *The disease-free equilibrium point is locally asymptotically stable if and only if* $R_e < 1$, *and is unstable if* $R_e > 1$.

We computed sensitivity analysis for model (1) and the results are displayed below.

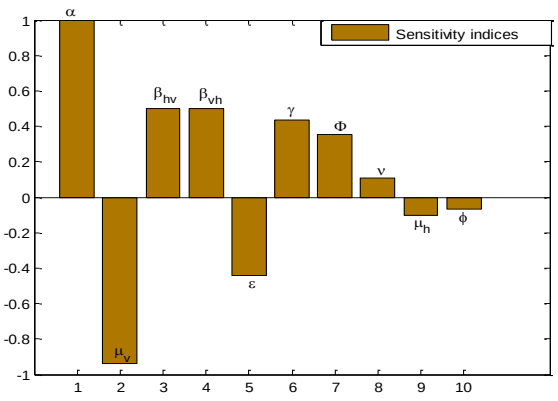


Figure 1 : Sensitivity indices of R_e

Interpretation of Sensitivity Indices shows that, the most sensitive parameters are the mosquito biting rate α, mosquitos' mortality rate μ_v, the probability that infectious mosquitoes infect a human host and the probability that mosquitoes become infected by infectious human β_{vh} and β_{hv} respectively. However, the cure rate ε, progression rate from exposed mosquito's class to infected mosquito class ν and the transmission rate of the educated Φ are important parameters. Therefore, this could be interpreted as reducing the mosquitoes biting rate α by increasing vectors death rate (through Insecticide Treated Nets (ITN), Indoor Residual Spraying (IRS) and environmental cleanliness) and increasing mass treatment would have the positive effect in controlling Lymphatic Filariasis transmission in the population.

III. MODEL APPLICATION

1) Parameter Estimation

Mathematical models in the form of ODE express the input-output properties of the dynamical populations. Moreover, systems of ODEs rarely provide quantitative solutions that are close to real field observations or experimental data because natural systems are subject to environmental and demographic noise and biologists are often indeterminate about the exact parameterization (Cao *et al.*, 2008). In this section we carry out parameter estimation using Least Squares to estimate model 1 parameters so as the system (ODE) solutions fits the actual data well.

Consider a nonlinear model as

$$y = f(x,\theta) + \varepsilon \qquad (12)$$

To compute the LSQ estimate for the parameters, direct formulas (as in regression models) are no longer available, and one has to numerically minimize the sum of squares. The likelihood function $L(\theta)$ is computed by sum of squares of Residual methods and used the Least Squares (LSQ) methods to estimate the parameters. Least Squares (LSQ) is commonly used in data fitting of which the "best fit" minimizes the sum of squared residual (SSR). A residual or error is the difference between observed/actual value and approximated value of the model. Having the Likelihood function $L(\theta)$ the fminsearch a built in Matlab optimizer is used to maximize $L(\theta)$ by minimizing the sum of squares term $\sum_{i=1}^{n}\left(y_i - f(x_i,\theta)\right)^2$ of the model predictions from the real data. That is

$$L(\theta) = \sum_{i=1}^{n}\left(y_i - f(x_i,\theta)\right)^2$$

Where, y_i is the observed value/data and $f(x_i,\theta)$ is the model function, with θ being the vector of unknown parameters and n is the number of observations.

Table 1. LF infective individuals' data from Morogoro Tanzania

YEARS/ DISTRICT	2009	2010	2011	2012	2013	2014
MOROGORO RURAL	1780	1741	293	220	749	690
MVOMERO	250	354	100	354	229	257
KILOSA	76	108	36	24	20	15
ULANGA	1178	1078	1038	986	944	914
TOTAL	3284	3281	1467	1584	1942	1876

The estimated parameters are shown in **table 2** and the corresponding ODE solutions of system (1) fit the actual data well.

Table 2 : Parameter values from literature and their Estimates

Parameter	Parameter Value	Source	Estimated
β_{vh}	0.86 day^{-1}	Mirand, 2009	0.8709
β_{hv}	0.083 day^{-1}	Niger, 2008	0.0826
α	0.29 day^{-1}	Ishikawa, 2003	0.2935
γ	0.0017 day^{-1}	Iddi,, 2016	0.0017
ϕ	0.0833 day^{-1}	Mwamtobe, 2017	0.0832
Φ	0.475 day^{-1}	Mwamtobe, 2017	0.4700
ν	0.0588 day^{-1}	Blayneh, 2009	0.0579
ε	0.125 day^{-1}	Bhunu, 2012	0.1232
μ_h	0.000039 day^{-1}	Mwamtobe, 2017	0.000037
μ_v	0.017 day^{-1}	Bhunu, 2012	0.0167
σ	0.045 day^{-1}	Assumed [0,1]	0.022
π	0.71 day^{-1}	Niger, 2008	0.0708
Λ	0.0384 day^{-1}	Iddi *et al,* 2016	0.0365
ω	0.000137 day^{-1}	Mwamtobe, 2017	0.0001
τ	0.11 day^{-1}	Assumed [0,1] as a measure of proportion	0.1112

1) Model Fitting

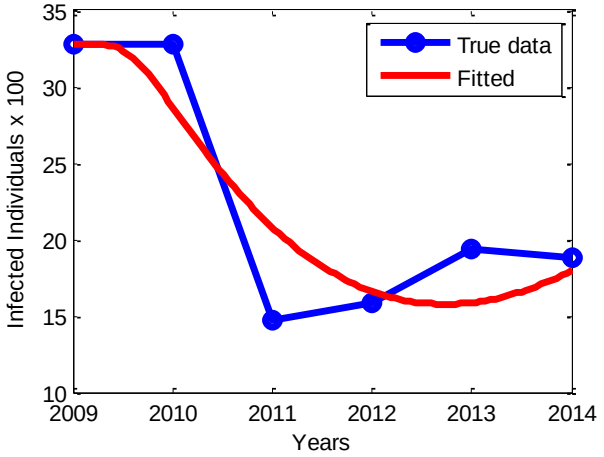


Figure 2 : Model fitting using LF infective individuals' actual data

Figure 4 shows the model system 1 on fitting to data for infective individuals of Lymphatic Filariasis obtained from Morogoro Regional Hospital in Tanzania. The fitting consist data for six years (2009-2014) due to scarcity of data. This is due to the fact that the LF elimination program in Morogoro region was implemented for only six years. The blue solid line shows the actual data and the red solid line represents the model fitted to the data. The model fits the data well for the given estimated parameter values.

2) Numerical Simulation

Numerical simulation was carried out using a set of parameter values sourced mainly from the literature and assumptions as shown in table 1. The model systems are simulated using Matlab ODE solvers and we considered the following initial conditions: $S_h = 10000,\ A = 8000,\ E_h = 4000,\ I_h = 900,\ R = 300,$ $S_v = 100,000,\ E_v = 80,000,\ I_v = 30,000.$

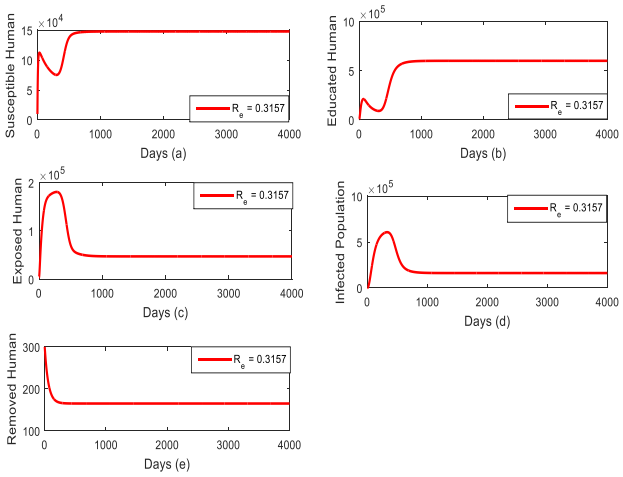


Figure 3: Dynamics of the human populations of the LF model in the presence of treatment barriers when $R_e < 1$ $(R_e = 0.3157)$

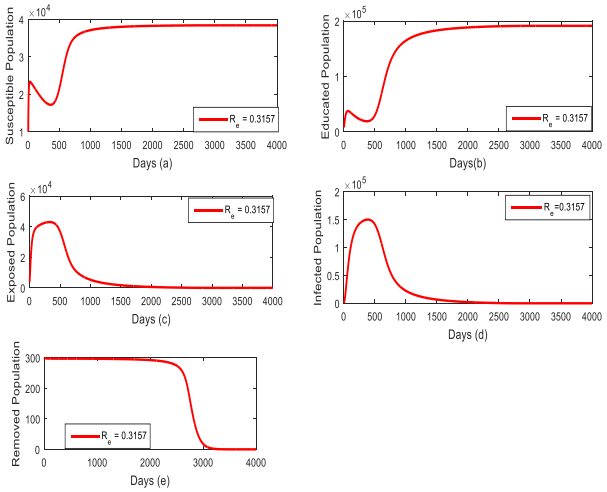


Figure 4: Dynamics of subpopulations of the LF model in the absence of treatment barriers when $R_e < 1$ $(R_e = 0.3157)$

Computation of R_e for the selected parameter values gave $R_e = 0.3157$. Clearly the disease dies out of the population as time increases. It shows that at initial stages the epidemic rises to a maximum level accompanied by a corresponding decrease in the susceptible and educated population. After a maximum level, infective classes and removed class gradually decrease until they vanish from the population. Susceptible and educated classes rise to maximum levels and settle at equilibrium level.

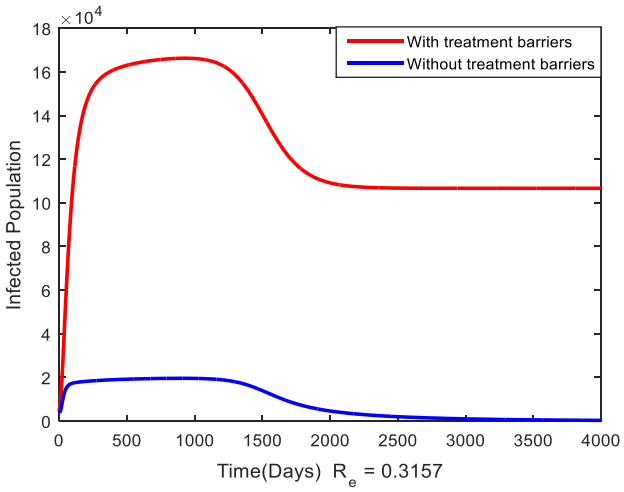


Figure 5: The effect of treatment barriers on LF transmission

It is observed from figure 5 that in the presence of treatment barriers, susceptible and educated individuals decrease while infected individuals' increase tremendously. However, in the absence of treatment barriers, the susceptible and educated individuals increase while the infected individuals' decrease.

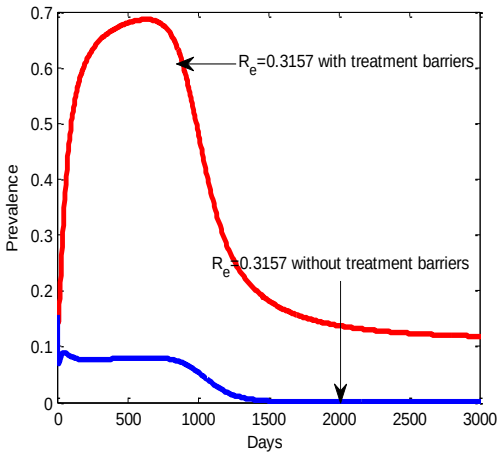
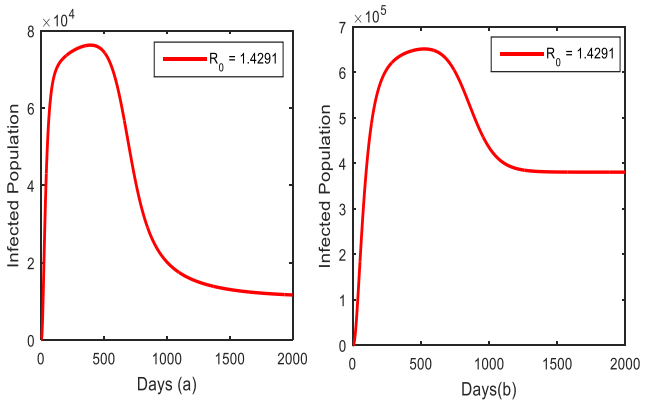


Figure 6: The Effect of Treatment Barriers on LF Prevalence

Figure 6 shows that in the presence of treatment barrier the prevalence increases tremendously than in the absence of treatment barriers, even if $R_e < 1$.



(a)Without treatment barrier (b) With treatment barrier

Figure 7: Influence of Treatment Barriers on Infected population when $R_e > 1$.

Figure 7(a) shows that in the absence of treatment barriers the infected population is reduced to equilibrium level with few infected individuals <250, but in the presence of treatment barriers the endemicity level increases to maximum level with infected individuals reaching > 3,600 as shown in 7(b).

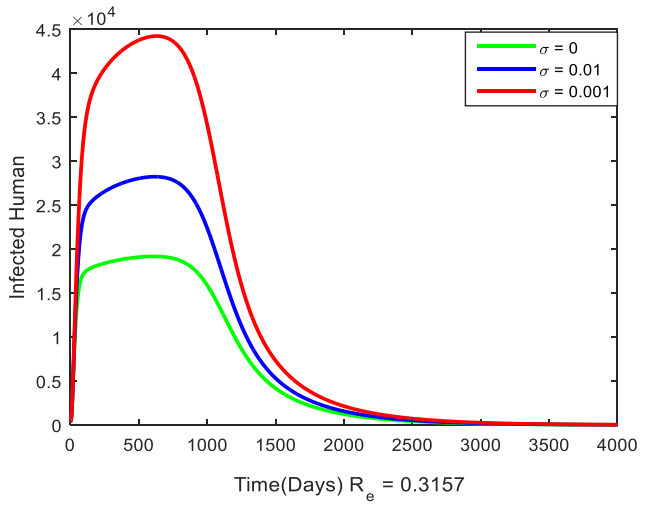


Figure 8: Variation of treatment barriers' rate σ.

From figure 8 the results show that an increase in the parameter value of σ leads to the increase of infected people in the society.

IV. DISCUSSION

The treatment function measured the effect of treatment and treatment barriers on transmission dynamics of the disease. Qualitative analysis and numerical simulation of the model were explored in terms of reproduction number. Numerical simulations indicated that, in the absence of treatment barriers, when $R_e < 1$ the model guaranteed disease extinction. However, the numbers of infective individuals showed disease persistence behaviour when $R_e > 1$. On the other hand, in the presence of treatment barriers, the numbers of infective individuals show persistent behaviour when $R_e < 1$ and when $R_e > 1$. This indicates that, there is multiple equilibria when $R_e < 1$ or there is coexistence between the stable disease free state and stable persistent state of the disease when $R_e < 1$ in presence of treatment barriers. The findings also suggested that the disease could be reduced when *Lymphatic Filariasis* elimination program increases uptake of drugs for prevention, scale up the health education campaigns on vector control and increase acceptance rate into timely treatment of the chronic infected individuals in the community. This may be attributed to the fact that many infected individuals, delay seeking for treatment. The reasons for delays in seeking treatment may be social barriers through scepticism, stigmatization or reluctance to travel long distances health posts, particularly so if they bear a disability. Therefore, more treatment barriers lead to more people being infected and consequently, a poor and disabled society.

V. CONCLUSION

In this work, the LF model with preventive measures in human and treatment coupled with treatment barriers aspect on the transmission dynamics of LF is developed. Qualitative analysis and numerical simulation are presented in terms of the reproduction number of the model in the absence and presence of treatment barriers. It is established that the treatment intervention has shown improvement in the

reduction of LF infection in the population as shown in figure 5 and figure 6. Furthermore, in the absence of treatment barriers the model guaranteed disease extinction behaviour, while in the presence of treatment barriers the model shows disease persistence behaviour when $R_e < 1$. This means that in the presence of treatment barriers there is coexistence of the stable disease-free state and the stable persistent state of the disease when $R_e < 1$. The persistence behaviour may be due to plentiful infected individuals who accumulate in the community because they denied treatment due to treatment barriers while the disease has no natural recovery. Thus, the results indicate that, driving $R_e < 1$ is not a necessary condition for eliminating the disease, more important, however, is the epidemic threshold R_e that should be taken below a determined threshold R_e^c. The study suggests that the disease can be reduced when LF elimination program targets at increasing the uptake of drugs for prevention, scale up of education on vector control and an increase of acceptance rate on timely treatment of infected individuals in the community.

VI. REFERENCES

[1]. Mwamtobe P.M., Simelane M.S, Abelman S. and Tchuenche J. M.. Mathematical Analysis of a Lymphatic Filariasis model with quarantine and treatment. BMC Public Health 17:265, 2017.

[2]. Nandha B, Krishnamoorthy K and Jambulingam P, "Towards elimination of Lymphatic Filariasis: social mobilization issues and challenges in mass drug administration with anti-filarial drugs in Tamil Nadu, South India" Vector Control Research Centre (Indian Council of Medical Research), Medical Complex, Indira Nagar Pondicherry 605 006, India.

[3]. Jones C, "Prevalence of Wuchereria Bancrofti Antigenemia and Associated Factors among Children of School Age Born after Initiation of Mass Drug Administration with Ivermectin and Albendazole in Rufiji District, Tanzania". MSc thesis Muhimbili University of Health and Allied Sciences , 2012.

[4]. World Health Organization. Global programme to eliminate Lymphatic Filariasis: progress report, 2015. Weekly Epidemiological Record. 91(39):441–60, 2016.

[5]. Bani R, Tisch D, Yunusa M. D, Mubayi A, Gidea M. "Modeling the transmission dynamics and control of Lymphatic Filariasis (Wuchereria bancrofti parasites) in Papua New Guinea"

[6]. Mwamtobe P.M., Simelane M.S, Abelman S. S and Tchuenche J. M.. Mathematical Analysis of a Lymphatic Filariasis model with quarantine and treatment. BMC Public Health 17:265, 2017.

[7]. Jambulingam P, "Towards elimination of Lymphatic Filariasis: social mobilization issues and challenges in mass drug administration with anti-filarial drugs in Tamil Nadu, South India" Vector Control Research Centre (Indian Council of Medical Research), Medical Complex, Indira Nagar Pondicherry 605 006, India.

[8]. Jones C, "Prevalence of Wuchereria Bancrofti Antigenemia and Associated Factors among Children of School Age Born after Initiation of Mass Drug Administration with Ivermectin and Albendazole in Rufiji District, Tanzania". MSc thesis Muhimbili University of Health and Allied Sciences , 2012.

[9]. World Health Organization. Global programme to eliminate Lymphatic Filariasis: progress report.Weekly Epidemiological Record. 91(39):441–60, 2016.

[10]. Bani R, Tisch D, Yunusa M. D, Mubayi A, Gidea M. "Modeling the transmission dynamics and control of Lymphatic Filariasis (Wuchereria bancrofti parasites) in Papua New Guinea"

[11]. Gyapong J. O, Owusu I. O, da-Costa Vroom F. B, Mensah E. O, Gyapong M.

[12]. Benjamin F. R. D, Graves P. M., Ni Ni Aye, Thet Wai Nwe, Tint Wai, San San Win, Mynt Shwe, Janet Douglass, Richard S. Bradbury, William J. McBride "The prevalence of Lymphatic Filariasis infection and disease following six rounds of mass drug administration in Mandalay Region, Myanmar" (2018), PLOS

[13]. Blayneh K, CaoY and Kwon H "Optimal Control of Vector borne Diseases: Treatment and Prevention," Discrete and Continuous Dynamical Systems Series B, 11(3): 1-xx. 2009

[14]. Bhunu CP and Mushayabasa S "Transmission Dynamics of Lymphatic Filariasis, Mathematical Approach" ISRN Biomathematics, 2012.

[15]. Cao J, Fussman GF and Ramsay JO. Estimating a predator-prey dynamical model with the parameter cascades method. Biometrics 64 (3):959-67, 2008.

[16]. Iddi A.J, Massawe E, Nkwengulila G, Kgosimore M, "The Effect of Chemoprophylaxis for Exposed Individuals on Lymphatic Filariasis Model." Applied and Computational Mathematics 5(1): 30-39, 2016.

[17]. Michael E, and Gambhir M .Vector Transmission Heterogeneity and the Population Dynamics and Control of Lymphatic Filariasis: Modelling Parasite Transmission and Control, Bioscience and Springer Science Business Media, 2010.

[18]. Michael E, Malecela-Lazaro M.N, Simonsen P.E, Pedersen E.M., Barker G, Kumar A and Kazura J.W.. Mathematical Modelling and the Control of Lymphatic Filariasis. Lancet Infect Dis 4: 223-234, 2004.

[19]. Miranda I, Ewungkem T, Chandra N, Podder, and Gumel B. A. Mathematical Study of the Role of Gametocytes and an Imperfect Vaccine on Malaria Transmission Dynamics. Bulletin of Mathematical Biology, 11: pp 538-009-9437-3, 2009.

[20]. Niger A.M and Gumel A.B. Mathematical Analysis of the Role of Repeated Exposure on Malaria Transmission Dynamics. Differential Equations and Dynamical Systems, 16(3) pp 251-287, 2008.

[21]. Irvine M. A., Stolk W. A, Smith M. E, Subramanian S., Singh B.K, Weil G.J. " Effectiveness of a triple-drug regimen for global elimination of Lymphatic Filariasis: a modelling study" , 2017.

[22]. "Elimination of Lymphatic Filariasis: Current perspectives on mass drug administration" Research and Reports in Tropical Medicine 9: 25–33, 2018.

[23]. Morgan E, Smith B.K, Singh & Michael E, " Assessing endgame strategies for the elimination of Lymphatic Flariasis: A model-based evaluation of the impact of DEC-medicated salt". Scientific Reports 7: 7386, 2017.

[24]. Kelatlhegile R., Kgosimore M, "Bifurcation analysis of vertical transmission model with preventive strategy". Journal of the Egyptian Mathematical Society, 1–7, 2015.

[25]. Tumwiine J, Mugisha J.Y.T. and Luboobi L.S.. On oscillatory pattern of malaria dynamics in a population with temporary immunity. Computational and Mathematical Methods in Medicine 8(3) 191–203, 2007.

[26]. Van den Driessche P., Watmough J, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission", Math. Biosci. 180:29-48 , 2002.

[27]. Mohammad A H, Ashok K S, Subhashisa S, Shridhar K and Sanghamitra P. "Mass drug administration for lymphatic filariasis elimination in a coastal state of India: a study on barriers to coverage and compliance". Infectious Diseases of Poverty, 3:31, 2014.

[28]. Simonsen PE, Derua YA, Kisinza WN, Magesa SM, Malecela MN and Pedersen EM Lymphatic Filariasis Control in Tanzania: Effect of Six Rounds of Mass Drug Administration with Ivermectin and Albendazole on Infection and Transmission. BMC Infectious Diseases, 13:335, 2013.

[29]. P. Jambulingam , S. Subramanian, S. J. de Vlas , C. Vinubala1 and W. A. Stolk. Mathematical modelling of lymphatic filariasis elimination programmes in India: required duration of mass drug administration and post-treatment level of infection indicators. Parasites & Vectors 9:501, 2016.

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