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Derivation of the Reproduction Number and Simulation of the Tuberculosis-Lymphatic *filariasis* Co-infection Model with Treatment for both Diseases

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Abstract

An Ordinary Differential Equation(ODE) co-infection model of Tuberculosis-Lymphatic *filariasis* is proposed with 17 mutually disjoint compartments. We showed that the model is Mathematically and Epidemiologically well-posed and the disease-free equilibrium(DFE) of the co-infection model is locally asymptotically stable if $R_O < 1$, it is unstable if $R_O > 1$. The numerical results show that lymphatic *filariasis* infection increases susceptibility to tuberculosis infection. This is in agreement with literature that, persons with lowered immunity such as HIV, diabetes, immune disorder etc are at a higher risk of contacting infectious diseases. We also found that increasing the rate of diagnosis and treatment of active tuberculosis and symptomatic lymphatic *filariasis* cases, the incidence of co-infection in the community can be reduced, and that if resources are limited, efforts should be targeted at treating only the co-infected cases.

Keywords: Tuberculosis, Lymphatic *filariasis*, Co-infection, Tuberculosis-Lymphatic *filariasis*.
MSC2010: 92BXX.

1 Introduction

In recent times, we have witnessed a resurgence of deadly infectious diseases which were once thought to have been eradicated appearing in new geographical areas, where they were initially not present. Malaria, tuberculosis(TB),HIV/AIDS, Dengue fever, cholera, West Nile virus, lymphatic *filariasis*(LF), Chagas, Lassa fever e.t.c. and recently, the Corona virus disease, 2019 (COVID '19) are just a few diseases which continue to persist despite all efforts committed to getting them eradicated.

LF and TB, according to World Health Organisation, are endemic in India, Indonesia and Nigeria. There is an overlap of endemic regions between LF and TB which may lead to co-infection

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[1]. Moreover, microbiological studies have suggested that LF infection could affect the pathogenesis of TB in co-infection cases. Persons with lowered immunity such as HIV, diabetes, immune disorder etc are at a higher risk of contracting TB [2]. LF, commonly known as elephantiasis, is a painful and profoundly disfiguring disease. According to WHO, the disease impairs the lymphatic system and lead to the abnormal enlargement of body parts, causing pain, severe disability and social stigma. Those who are infected, are not only physically disable, but suffer mental, social and financial losses contributing to stigma and poverty. *Filariasis* is a group of human and animal infectious diseases caused by nematode parasites commonly called "*filariae*" [3]. The thread-like adult parasites live in vessels, tissues or body cavities of the vertebrate hosts. The female worms are viviparous and produce microscopic embryos called microfilariae [3]. The microfilariae circulate in the blood or migrate through the skin from where they are ingested by vectors during blood meal [3]. The worms have an estimated active reproductive span of 4-6 years, producing millions of small immature microfilariae, which circulate in the peripheral blood [4]. They are transmitted from person to person by several species of mosquitoes [4]. The drug diethylcarbamazine (DEC) is used to kill the microfilariae in the blood stream. But in the case of elephantiasis, surgical procedure may be prescribed to reduce the swelling and relieve the obstruction. (TB) is an airborne transmitted disease caused by slowly-replicating bacterium. [4, 5]. It affects the lungs (pulmonary TB) but can also affect other parts of the body (extra pulmonary TB). The disease is released into the air as droplets when those who have active TB cough or sneeze [6].Individuals having latent TB cannot transmit TB [7]. Only 10% of the latent TB individuals actually develop active TB. Latent TB individuals can remain in the latent state for a long time, and may die without developing the disease [8]. The good news is that TB and LF can be treated.

TB and LF are widespread and serious public health problems in developing countries. While TB has continued to cause high mortality in humans, LF is a major cause of chronic morbidity in humans [4]. It is estimated that TB infects one-third of the world′s population resulting in 2–3 million deaths per year [1]. And over 120 million people are affected by lymphatic *filariasis* (LF) disease worldwide [4]. TB and LF are endemic in Nigeria, suggesting an overlap of endemic region between TB and LF that may lead to co-infection [1]. LF impedes the smooth functioning of the immune system [9] which can lower the immune defence system, making the individual to be highly susceptible to TB and other infectious diseases. Helminths co-infections with other diseases alone are thought to occur in over 800 million people and are especially prevalent among the global poor [10–12]. TB patients with helminth infections have severe pulmonary disease, which can interfere with diagnosis test for TB [13–15]. Six high burden countries (HBC) account for 60% of global TB incidences. These countries are India, Indonesia, China, Nigeria, Pakistan and South Africa. According to WHO [16] TB mortality accounts for 1.4 million deaths and 0.4 million (additional) deaths due to HIV/AIDS and TB. There are about 600,000 new (incident) TB cases in Nigeria and 250,000 TB-related deaths (WHO, 2016). LF infect 120 million people globally with about 40 million disfigured and incapacitated [4]. India, Indonesia and Nigeria are the countries with the highest LF burden in the world.

Several mathematical models have been formulated to understand the transmission dynamics of TB and LF under various circumstances. [17] developed and analysed an LF disease transmission model to determine the impact of multi-interventions campaign via health education and sterile insect technique (SIT) on the spread of LF. [18] formulated and analysed a nonlinear differential equation model to study the effect of chemoprophylaxis on the exposed individuals. [19] developed a mathematical model to investigate the impact that vector genus – specific dependent processes may have on overall LF transmission. [20] used lymphatic *filariasis* Simulation Model (LYMFASIM)to estimate the duration of Mass Drug Administration (MDA) required for elimination and residual levels of microfilaemia (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. [21] used a model-based assessment to develop different plausible scale-up scenarios to reach global elimination and eradication of LF. 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-

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up. [22] used mathematical models to assess the feasibility and strategic value of including vector control in the GPELF initiative to achieve the global elimination of LF. [23] 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. [24] 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. [25] formulated a TB model with seasonality, [26] formulated and analysed a mathematical model for the transmission dynamics of TB with vaccination and screening of individuals to identify individuals with chronic TB cases to be placed on prompt TB treatment. [27] proposed and analysed an SEIR compartmental model to examine the population dynamics of TB with BCG vaccination as a control measure. [28] presented and analysed a new mathematical model for TB dynamics to study the effects of additional heterogeneities based on the level of awareness of TB within the population and active-case finding on the dynamics of the disease. [29] developed and analysed a mathematical model based on the level of exposure of individuals to mycobacterium TB. [30] formulated a co-infection model for malaria and rotavirus. [31] proposed and analysed a deterministic mathematical model for HIV/TB co-infection to examine the impact of HIV or TB infection with treatment of TB and management of HIV using Antiretroviral Therapy (ART). [32] proposed a system of nine nonlinear mathematical model to study the co-infection of both zika and malaria in a community where they are both endemic. [33] developed and analysed a mathematical model to explore the effects of early and late HIV treatment during the TB treatnment course. [34] developed and analysed a nine-dimensional deterministic ordinary differential equations model for Dengue and Chikungunya co-infection. [35] proposed and analysed a compartmental model of Listeriosis and Antrax co-infection. [36] formulated and analysed a mathematical model for the spread of HIV and TB co-infection by considering the resistance of HIV to antiretroviral (ARV) drugs. [37] proposed and analysed an SIQRM epidemic model with vaccination and relapse to study the effect of immunity obtained from vaccination and treatment. [38] proposed a 7-dimensional system of nonlinear ordinary equations to study the transmission and spread of malaria parasite in a population.Thier model incorporate a class of non-drug complient human compartment into the population. To the best of the author's knowledge, no work has been done to study the transmission dynamics of TB-LF co-infection. In this work, we proposed seventeen non-linear mathematical models for the transmission dynamics of TB-LF co-infection to study how the endemicity of LF affect the population dynamics of TB with treatment for both diseases. The rest of the paper is organized as follows. In section 2, we proposed a TB-LF co-infection model with treatment for both diseases. In section 3, we showed that the parameters of the model remains positive for all time. In section 4, we compute the basic reproduction number of TB-LF co-infection model. and in subsection 4.1, we compute the basic reproduction number of LF-only model using the epidemiological approach in [39]. In section 5, we carry out the numerical simulation of the model. In section 6, we give the results and discussion. And finally, the conclusion is given in section 7.

2 Methods

The pathogenesis of TB and LF, signs and symptoms, diagnosis and treatment as well as current epidemiological data are presented. Thereafter, an extensive review of TB and LF literature were carried out. Based on the biology and natural history of TB and LF, we formulated seventeen novel deterministic mathematical co-infection models given below in equation 2.6.

The total human and mosquito population at time t, denoted by $N_h(t)$ and $N_v(t)$ are divided into 17 mutually exclusive compartments. This is made up of 14 mutually-exclusive compartments for human population and 3 mutually-exclusive compartments for mosquito population, see Figure 1. The human population is made up of susceptible individuals $S_h(t)$, individual having latent TB $E_t(t)$, individuals having active TB $I_T(t)$, individuals treated of TB $T_t(t)$, individuals having latent LF $E_h(t)$, individuals having asymptomatic LF $A_h(t)$, individuals having symptomatic LF

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$I_h(t)$, dually infected individuals having latent LF and latent TB $E_{ht}(t)$, dually infected individuals having latent LF and active TB $E_{hT}(t)$, dually infected individuals with asymptomatic LF and latent TB $A_{ht}(t)$, dually infected individuals with asymptomatic LF and active TB $A_{hT}(t)$, dually infected individuals with symptomatic LF and latent TB $I_{ht}(t)$, dually infected individuals with symptomatic LF and active TB $I_{hT}(t)$ and individuals with treated LF $T_h(t)$,So that

$$\begin{aligned} N_h(t) = S_h(t) + E_t(t) + I_T(t) + T_t(t) + E_h(t) + A_h(t) + I_h(t) + \\ E_{ht}(t) + E_{hT}(t) + A_{ht}(t) + A_{hT}(t) + I_{ht}(t) + I_{hT}(t) + T_h(t) \end{aligned} \tag{2.1}$$

Similarly, the mosquito population at time t is made up of susceptible mosquito population, $S_v(t)$, exposed mosquito population, $E_v(t)$ and infected female mosquito population, $I_v(t)$, So that

$$N_v(t) = S_v(t) + E_v(t) + I_v(t) \tag{2.2}$$

Susceptible individuals acquire LF infection following effective contact with mosquito infected with LF at a rate given by

$$\lambda_v = \frac{\beta_v \sigma_v \sigma_h I_v}{\sigma_v N_v + \sigma_h N_h} \tag{2.3}$$

Susceptible mosquitoes can acquire LF when in contact with individuals infected with LF (both singly and dually infected) at a rate given by

$$\lambda_h = \frac{\beta_h \sigma_v \sigma_h ((\eta_1 E_h + \eta_{h1} A_h + I_h) + \eta_v (\eta_2 E_{ht} + \eta_{h2} A_{ht} + I_{ht}) + \eta_v \omega_v (\eta_3 E_{hT} + \eta_{h3} A_{hT} + I_{hT}))}{\sigma_v N_v + \sigma_h N_h} \tag{2.4}$$

Susceptible individuals acquire TB following effective contact with an infected individual at a rate given by

$$\lambda_T = \frac{\beta_T (I_T + \theta_T (\eta_{T1} E_{hT} + \eta_{T2} A_{hT} + I_{hT}))}{N_h} \tag{2.5}$$

See Tables 1 and 2 for the definition of state variables and parameters in the model.

The model for TB-LF co-infection is given by the following deterministic system of nonlinear differential equations.

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_T S_h - \lambda_v S_h - \mu_h S_h, \\
\frac{dE_t}{dt} &= (1-p_1)\lambda_T (S_h + \phi T_t + T_h) - \epsilon_1 \lambda_T E_t - \lambda_v E_t + \tau_{h2} I_{ht} - (\gamma_1 + \mu_h) E_t, \\
\frac{dI_T}{dt} &= p_1 \lambda_T (S_h + \phi T_t + T_h) + \epsilon_1 \lambda_T E_t + \gamma_1 E_t - \lambda_v I_T + \tau_{h3} I_{hT} - (\tau_{T1} + \delta_T + \mu_h) I_T, \\
\frac{dT_t}{dt} &= \tau_{T1} I_T - \phi \lambda_T T_t - \lambda_v T_t - \mu_h T_t, \\
\frac{dE_h}{dt} &= \lambda_v (S_h + T_t) - \theta_1 \lambda_T E_h + \nu \lambda_v T_h + \tau_{T2} E_{hT} - (k_1 + \mu_h) E_h, \\
\frac{dA_h}{dt} &= k_1 E_h + \tau_{T3} A_{hT} - (k_2 + \mu_h) A_h - \theta_2 \lambda_T A_h, \\
\frac{dI_h}{dt} &= k_2 A_h + \tau_{T4} I_{hT} - (\tau_{h1} + \delta_L + \mu_h) I_h - \theta_3 \lambda_T I_h \\
\frac{dT_h}{dt} &= \tau_{h1} I_h - \lambda_T T_h - \nu \lambda_v T_h - \mu_h T_h,
\end{aligned}$$

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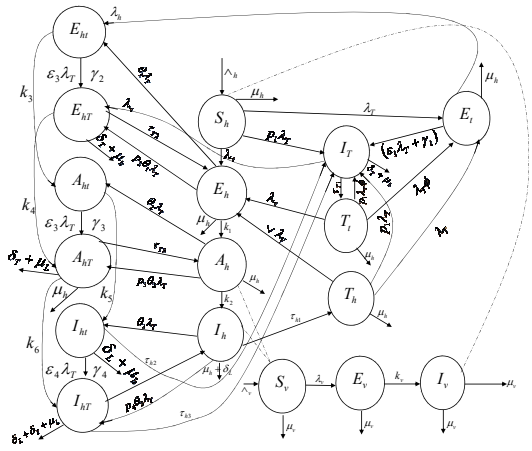

Figure 1: Model schematic diagram.

$$
\begin{aligned}
\frac{dE_{ht}}{dt} &= \lambda_v E_t + (1-p_2)\theta_1\lambda_T E_h - \epsilon_2\lambda_T E_{ht} - (\gamma_2 + k_2 + \mu_h)E_{ht},\\
\frac{dE_{hT}}{dt} &= \lambda_v I_T + p_2\theta_1\lambda_T E_h + \epsilon_2\lambda_T E_{ht} + \gamma_2 E_{ht} - (\tau_{T2} + k_4 + \delta_L + \mu_h)E_{hT},\\
\frac{dA_{ht}}{dt} &= (1-p_3)\theta_2\lambda_T A_h - \epsilon_3\lambda_T A_{ht} + k_3 E_{ht} - (\gamma_3 + k_5 + \mu_h)A_{ht}\\
\frac{dA_{hT}}{dt} &= p_3\theta_2\lambda_T A_h + \epsilon_3\lambda_T A_{ht} + \gamma_3 A_{ht} + k_4 E_{hT} - (\tau_{T3} + k_6 + \delta_T + \mu_h)A_{hT},\\
\frac{dI_{ht}}{dt} &= (1-p_4)\theta_3\lambda_T I_h - \epsilon_4\lambda_T I_{ht} + k_5 A_{ht} - (\gamma_4 + \tau_{h2} + \delta_L + \mu_h)I_{ht} \qquad (2.6)\\
\frac{dI_{hT}}{dt} &= p_4\theta_3\lambda_T I_h + \epsilon_4\lambda_T I_{ht} + k_6 A_{hT} + \gamma_4 I_{ht} - (\tau_{T4} + \tau_{h3} + \delta_L + \delta_T + \mu_h)I_{hT},\\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_h S_v - \mu_v S_v,\\
\frac{dE_v}{dt} &= \lambda_h S_v - k_v E_v - \mu_v E_v,\\
\frac{dI_v}{dt} &= k_v E_v - \mu_v I_v,
\end{aligned}
$$

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Table 1: Description of state variables of model (2.6)

Variable	Description
$S_h(t)$	Population of susceptible individuals
$E_t(t)$	Population of individuals with latent TB
$I_T(t)$	Population of individuals with active TB
$T_t(t)$	Population of individuals treated of TB
$E_h(t)$	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
$E_{ht}(t)$	Population of dually infected individuals with latent LF and latent TB
$E_{hT}(t)$	Population of dually infected individuals with latent LF and active TB
$A_{ht}(t)$	Population of dually infected individuals with asymptomatic LF and latent TB
$A_{hT}(t)$	Population of dually infected individuals with asymptomatic LF and active TB
$I_{ht}(t)$	Population of dually infected individuals with symptomatic LF and latent TB
$I_{hT}(t)$	Population of dually infected individuals with symptomatic LF and active TB
$S_v(t)$	Population of susceptible mosquitoes
$E_v(t)$	Population of exposed mosquitoes.
$I_v(t)$	Population of infectious mosquitoes.

3 Positivity of Solution of the Model

Theorem 1: The model (2.6) to be consistent with both human and mosquito population, all feasible solutions of the model will remain positive for all time $t > 0$.

Proof:

Consider the first equation of model (2.6), given below as

$$\frac{dS_h}{dt} = \Lambda_h - \lambda_T S_h - \lambda_v S_h - \mu_h S_h, \tag{3.1}$$

Without loss of generality, we can write equation (3.1) as

$$\frac{dS_h}{dt} \geq -(\lambda_T + \lambda_v + \mu_h) S_h, \tag{3.2}$$

$$\frac{dS_h}{S_h(t)} \geq -(\lambda_T + \lambda_v + \mu_h) dt \tag{3.3}$$

integrating (3.3) with respect to t in $[0, t_1]$, yields

$$S_h(t_1) \geq S_h(0) e^{-\{\int_0^{t_1} (\lambda_T + \lambda_v) dt + \mu_h t\}} > 0, \ \forall \ t > 0. \tag{3.4}$$

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Table 2: Description of parameters of model (2.6)

Parameter	Description
μ_h, μ_v	Natural death rates
Λ_h, Λ_v	Recruitment rates
β_T	Transmission rate of TB
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
$\tau_{T1}, \ldots, \tau_{T4}$	Treatment rates for TB
$\tau_{h1}, \tau_{h2}, \tau_{h3}$	Treatment rates for LF
$\gamma_1, \gamma_2, \gamma_3, \gamma_4$	TB progression rate
$\epsilon_1, \epsilon_2, \epsilon_3, \epsilon_4$	Exogeneous reinfection rate for TB
$k_1, \ldots, k_6$	Progression rates for LF
$p_1, \ldots, p_4$	Fraction of fast disease progression
ν	Reinfection rates for LF after treatment
θ_T	Increased infectiousness of dually-infected on TB transmission
η_{T1}, η_{T2}	Reduced transmissibility of dually-infected on TB in comparison with infected with TB
η_1, η_2, η_3	Reduced transmissibility of LF by asymptomatic individuals in comparison with symptomatic individuals
η_v	Mod. par. accounts for the increased infectioness of dually-infected
ω_v	Mod. par. accounts for the increased infectioness of dually-infected with active TB compared to those with latent TB
ϕ	Rate of TB reinfection after treatment
k_v	Progression rates for mosquitoes
δ_L	Disease-induced death rate for individuals with LF
δ_T	Disease-induced death rate for individuals with active TB

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

Similarly, all state variables of the model (2.6) are positive, $\forall\ t > 0$.

Theorem 2: Let $(S_h, E_t, I_T, T_t, E_h, A_h, I_h, T_h, E_{ht}, E_{hT}, A_{ht}, A_{hT}, I_{ht}, I_{hT}, S_v, E_v, I_v)$ be trajectories of the system (2.6) with initial conditions $(S_h \geq 0, E_t \geq 0, I_T \geq 0, T_t \geq 0, E_h \geq 0, A_h \geq 0, I_h \geq 0, T_h \geq 0, E_{ht} \geq 0, E_{hT} \geq 0, A_{ht} \geq 0, A_{hT} \geq 0, I_{ht} \geq 0, I_{hT} \geq 0, S_v \geq 0, E_v \geq 0, I_v \geq 0)$ and the biological feasible regions given by the set $\mathcal{D}_1 = \mathcal{D}_{h1} \times \mathcal{D}_{v1}$ where $\mathcal{D}_{h1} = \{(S_h, E_t, I_T, T_t, E_h, A_h, I_h, T_h, E_{ht}, E_{hT}, A_{ht}, A_{hT}, I_{ht}, I_{hT}) \in \mathbb{R}^{14}_+ : N_h \leq \frac{\Lambda_h}{\mu_h}\}$ $\mathcal{D}_{v1} = \{(S_v, E_v, I_v) \in \mathbb{R}^3_+ : N_v \leq \frac{\Lambda_v}{\mu_v}\}$ is positively-invariant and attracts all the positive trajectories of (2.6)

Proof:

Adding up the first 14 equations on the right hand side of (2.6), yields

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_L I_h - \delta_T I_T - \delta_T E_{hT} - \delta_T A_{hT} - \delta_L I_{ht} - \delta_L I_{hT} - \delta_T I_{hT}. \tag{3.5}$$

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From (3.5), it follows that $\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h$. Hence, $\frac{dN_h}{dt} \leq 0$ if $N_h(t) \geq \frac{\Lambda_h}{\mu_h}$. we have that

$$N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}). \tag{3.6}$$

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, 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 $\mathcal{D}_1$ 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 $\mathcal{D}_1$ in finite time or $N_h(t)$ asymptotically approaches $\frac{\Lambda_h}{\mu_h}$ as $t \to \infty$. and $N_v(t)$ asymptotically approaches $\frac{\Lambda_v}{\mu_v}$ as $t \to \infty$. Thus, the domain $\mathcal{D}_1$ attracts all trajectories in $\mathbb{R}^{17}_+$. Since the domain $\mathcal{D}_1$ is positively-invariant, it is enough to study the dynamics of the flows generated by the system (2.6) in $\mathcal{D}_1$.
We conclude, therefore, that the model (2.6) is Mathematically and Epidemiologically well posed. □

4 Computation of the Basic Reproduction Number of the Co-infection Model

The DFE of the model (2.6) is given by

$$\mathcal{E}_2 = (S_h^0, E_t, I_T, T_t, E_h^0, A_h^0, I_h^0, T_h^0, E_{ht}, E_{hT}, A_{ht}, A_{hT}, I_{ht}, I_{hT}, S_v^0, E_v^0, I_v^0) \tag{4.1}$$

$$= \Big(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0\frac{\Lambda_v}{\mu_v}, 0, 0\Big). \tag{4.2}$$

$$F = \begin{pmatrix} F_{11} & F_{12} \\ F_{21} & F_{22} \end{pmatrix}. \tag{4.3}$$

And:

$$F_{11} = \begin{pmatrix} 0 & (1-p_1)\beta_T & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.4}$$

$$F_{12} = \begin{pmatrix} (1-p_1)\beta_T\theta_T\eta_{T1} & 0 & (1-p_1)\beta_T\theta_T\eta_{T2} & 0 & (1-p_1)\beta_T\theta_T & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & G_1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.5}$$

And:

$$F_{21} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta_1 G_2 & \eta_{h1} G_2 & G_2 & \eta_v\eta_2 G_2 & \eta_v\omega_v\eta_3 G_2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.6}$$

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$$F_{22} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \eta_v G_2 & \eta_v \omega_v G_2 & \eta_v G_2 & \eta_v \omega_v G_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.7}$$

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 $G_2 = \frac{\beta_h \mu_h \Lambda_v \sigma_v \sigma_h}{\sigma_v \Lambda_v \mu_h + \sigma_h \Lambda_h \mu_v}$

$$V = \begin{pmatrix} V_{11} & V_{12} \\ V_{21} & V_{22} \end{pmatrix}. \tag{4.8}$$

$$V_{11} = \begin{pmatrix} g_5 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_1 & g_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & g_1 & 0 & 0 & 0 \\ 0 & 0 & -k_1 & g_2 & 0 & 0 \\ 0 & 0 & 0 & -k_2 & g_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & g_7 \end{pmatrix}. \tag{4.9}$$

$$V_{12} = \begin{pmatrix} 0 & 0 & 0 & -\tau_{h2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\tau_{h3} & 0 & 0 \\ -\tau_{T2} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\tau_{T3} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\tau_{T4} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.10}$$

$$V_{21} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & -\gamma_2 & g_8 \\ 0 & 0 & 0 & 0 & 0 & -k_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -k_4 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \tag{4.11}$$

$$V_{22} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ g_9 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_3 & g_{10} & 0 & 0 & 0 & 0 \\ -k_5 & 0 & g_{11} & 0 & 0 & 0 \\ 0 & -k_6 & -\gamma_4 & g_{12} & 0 & 0 \\ 0 & 0 & 0 & 0 & g_4 & 0 \\ 0 & 0 & 0 & 0 & -k_v & \mu_v \end{pmatrix}. \tag{4.12}$$

where $g_1 = k_1 + \mu_h, g_2 = k_2 + \mu_h, g_3 = \tau_{h1} + \mu_h + \delta_L, g_4 = k_v + \mu_h, g_5 = \gamma_1 + \mu_h, g_6 = \tau_{T1} + \mu_h + \delta_T, g_7 = \gamma_2 + \mu_h + k_3, g_8 = \tau_{T2} + k_4 + \mu_h + \delta_L, g_9 = \gamma_3 + \mu_h + k_5$

$\rho(FV^{-1}) = \mathcal{R}_0 = max(R_L, R_T)$

where

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$\mathcal{R}_L = \sqrt{\frac{\sigma_h^2\sigma_v^2 k_v\beta_h\beta_v\Lambda_h\Lambda_v\mu_h(\eta_1 g_2 g_3 + k_1(\eta_{h1} g_3 + k_3))}{(\Lambda_h\mu_v\sigma_h + \Lambda_v\mu_h\sigma_v)^2 g_1 g_2 g_3 g_4}}$, the basic reproduction number for LF-only model

and

$\mathcal{R}_T = \frac{p_1\beta_T}{\tau_{T1}+\delta_T+\mu_h} + \frac{\gamma_1(1-p_1)\beta_T}{(\gamma_1+\mu_h)(\tau_{T1}+\delta_T+\mu_h)}$ the basic reproduction number for TB-only model

.

4.1 Computation of Basic Reproduction of LF-only model: R_L:

Using the approach in [39], we compute the basic reproduction number R_L of LF-only model

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

$$\frac{\beta_v\sigma_v\sigma_h S_h^0}{\sigma_v N_v^0 + \sigma_h N_h^0} * \frac{k_v}{g_4} * \frac{1}{\mu_v} \tag{4.13}$$

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

$$\frac{\beta_v\sigma_v\sigma_h k_v\Lambda_h}{g_4(\sigma_v\Lambda_v\mu_h + \sigma_h\Lambda_h\mu_v)} \tag{4.14}$$

Equation (4.14) 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

$$\frac{\eta_1\beta_h\sigma_v\sigma_h S_v^0}{\sigma_v N_v^0 + \sigma_h N_h^0} * \frac{1}{g_1} \tag{4.15}$$

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

$$\frac{\eta_1\beta_h\sigma_v\sigma_h\Lambda_v\mu_h}{g_1(\sigma_v\Lambda_v\mu_h + \sigma_h\Lambda_h\mu_v)} \tag{4.16}$$

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

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$$\frac{\eta_{h_1}\beta_h\sigma_v\sigma_h S_v^0}{\sigma_v N_v^0+\sigma_h N_h^0} * \frac{k_1}{g_1} * \frac{1}{g_2} \tag{4.17}$$

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

$$\frac{k_1\eta_{h_1}\beta_h\sigma_v\sigma_h\mu_h\Lambda_v}{g_1g_2(\sigma_v\Lambda_v\mu_h+\sigma_h\Lambda_h\mu_v)} \tag{4.18}$$

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

$$\frac{\beta_h\sigma_v\sigma_h S_v^0}{\sigma_v N_v^0+\sigma_h N_h^0} * \frac{k_1k_2}{g_1g_2} * \frac{1}{g_3} \tag{4.19}$$

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

$$\frac{k_1k_2\beta_h\sigma_v\sigma_h\mu_h\Lambda_v}{g_1g_2g_3(\sigma_v\Lambda_v\mu_h+\sigma_h\Lambda_h\mu_v)} \tag{4.20}$$

The average number of new mosquito infections generated by infected humans (exposed, asymptomatic and infectious) is given by the addition of equations (4.16), (4.18) and (4.20).

That is

$$\frac{\eta_1\beta_h\sigma_v\sigma_h\Lambda_v\mu_h}{g_1(\sigma_v\Lambda_v\mu_h+\sigma_h\Lambda_h\mu_v)} + \frac{k_1\eta_{h_1}\beta_h\sigma_v\sigma_h\mu_h\Lambda_v}{g_1g_2(\sigma_v\Lambda_v\mu_h+\sigma_h\Lambda_h\mu_v)} + \frac{k_1k_2\beta_h\sigma_v\sigma_h\mu_h\Lambda_v}{g_1g_2g_3(\sigma_v\Lambda_v\mu_h+\sigma_h\Lambda_h\mu_v)} \tag{4.21}$$

Simplifying, we have

$$\frac{\beta_h\sigma_v\sigma_h\mu_h\Lambda_v(\eta_1g_2g_3+k_1\eta_{h_1}g_3+k_1k_2)}{g_1g_2g_3(\sigma_v\Lambda_v\mu_h+\sigma_h\Lambda_h\mu_v)} \tag{4.22}$$

The expression in equation (4.22) represents the total number of lymphatic *filariasis* infected mosquitoes caused by (exposed, asymptomatic and infectious humans).

The geometric mean of equations (4.14) and (4.22) gives the effective reproduction number R_L.

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Table 3: Baseline values and ranges of the parameters of the model 2.6.

Parameters	Baseline values	Ranges	References
μ_h	0.02041 year^{-1}	(0.0143, 0.03)	Assumed
μ_v	0.0714 year^{-1}	(0.0143, 0.03)	Assumed
Λ_h	3 768 410 year^{-1}	(3,000,000, 4,000,000)	[40]
Λ_v	10000 year^{-1}	(5,000, 15,000)	[40]
β_T	Variable year^{-1}	(1.55, 5)	[16]
β_v	Variable year^{-1}	(0, 1)	[16]
β_h	Variable year^{-1}	(0, 1)	[16]
σ_v	620.39 year^{-1}	(600, 800)	[41]
σ_h	1168 year^{-1}	(1000, 2000)	[41]
τ_{T1}	1.5 year^{-1}	(0, 2)	[6]
τ_{T2}	1.5 year^{-1}	(0, 2)	[6]
τ_{T3}	1.5 year^{-1}	(0, 2)	[6]
τ_{T4}	1.5 year^{-1}	(0, 2)	[6]
τ_{h1}	1.5 year^{-1}	(0, 2)	[41]
τ_{h2}	1.5 year^{-1}	(0, 2)	[41]
τ_{h3}	1.5 year^{-1}	(0, 2)	[41]
γ_1	0.5 year^{-1}	[0, 1]	[42]
γ_2	0.7 year^{-1}	[0, 1]	[42]
γ_3	0.7 year^{-1}	[0, 1]	[42]
γ_4	0.7 year^{-1}	[0, 1]	[42]
ϵ_1	1.5 year^{-1}	[1, 2.5]	[43]
ϵ_2	1.8 year^{-1}	[1, 2.5]	[43]
ϵ_3	1.8 year^{-1}	[1, 2.5]	[43]
ϵ_4	1.8 year^{-1}	[1, 2.5]	[43]
k_1	0.15 year^{-1}	(0, 0.6)	Assumed
k_2	0.15 year^{-1}	(0, 0.6)	Assumed
k_3	0.2 year^{-1}	(0, 0.6)	Assumed
k_4	0.2 year^{-1}	(0, 0.6)	Assumed
k_5	0.2 year^{-1}	(0, 0.6)	Assumed
k_6	0.2 year^{-1}	(0, 0.6)	Assumed
ν	0.0001 year^{-1}	(0, 1)	Assumed
η_{T1}	0.85 year^{-1}	[0, 1]	Assumed
η_{T2}	0.85 year^{-1}	[0, 1]	Assumed
η_1	0.3 year^{-1}	[0.1, 0.8]	[44]
η_2	0.3 year^{-1}	[[0.1, 0.8]	[44]
η_3	0.3 year^{-1}	[[0.1, 0.8]	[44]
η_v	1.3 year^{-1}	[1, 2]	[41]

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Parameters	Baseline values	Ranges	References
ω_v	1.25 year^{-1}	[1, 2.5]	[41]
η_{h1}	0.65 year^{-1}	[0.2, 0.9]	[41]
η_{h2}	0.65 year^{-1}	(0.2, 0.9)	[41]
η_{h3}	0.65 year^{-1}	(0.2, 0.9)	[41]
θ_1	2.5 year^{-1}	[0, 10]	Assumed
θ_2	2.5 year^{-1}	(0, 10)	Assumed
θ_3	2.5 year^{-1}	(0, 10)	Assumed
ϕ	1.3 year^{-1}	(1, 2.15)	[45]
p_1	0.1 year^{-1}	[0, 0.5]	[46]
p_2	0.3 year^{-1}	[(0, 0.5]	[46]
p_3	0.3 year^{-1}	[(0, 0.5]	[46]
p_4	0.3 year^{-1}	[(0, 0.5]	[46]
k_v	0.005 year^{-1}	[(0, 0.009]	[44]
δ_T	0.365 year^{-1}	[(0, 0.5]	[6]
δ_L	0.0001 year^{-1}	[(0, 0.0005]	[44]

5 Numerical simulations

Here, we carried out numerical simulation. The ranges and baseline values of the parameters are listed in Table (3). We used demographic (and epidemiological) parameters relevant to Nigeria and from literature. In 2020, Nigeria population was estimated to be 208,994,835 [40].

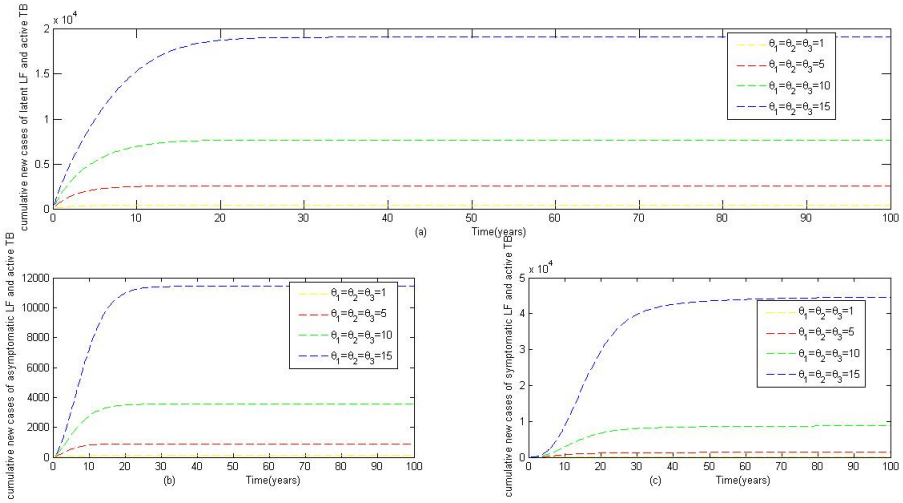

Figure 2: New cases of (a) E_{hT} (b) A_{hT} and (c) I_{hT} when θ_1, θ_2 and θ_3 are varied.

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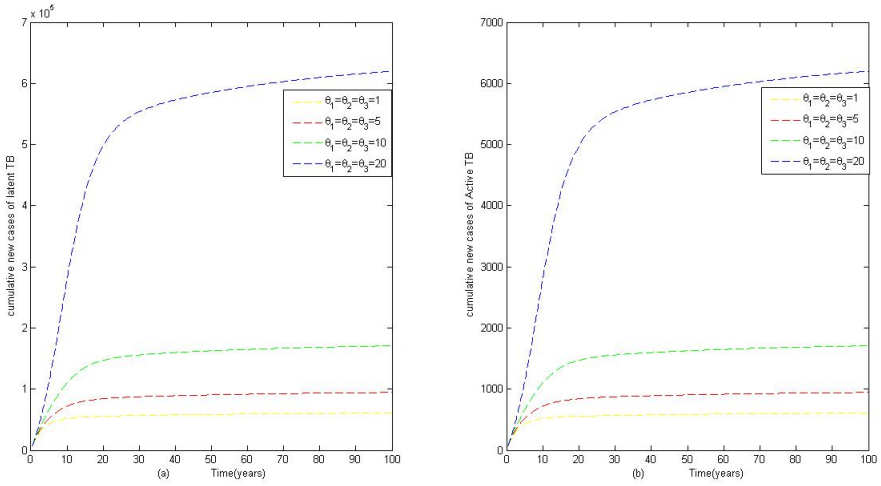

Figure 3: New cases of (a) E_t and (b) I_T when θ_1, θ_2 and θ_3 are varied.

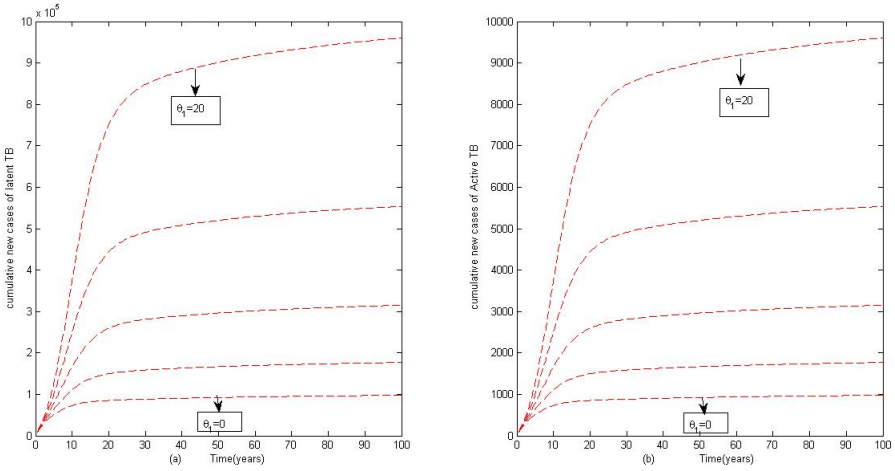

Figure 4: New cases of (a)E_t and (b) I_T when θ_1 is varied.

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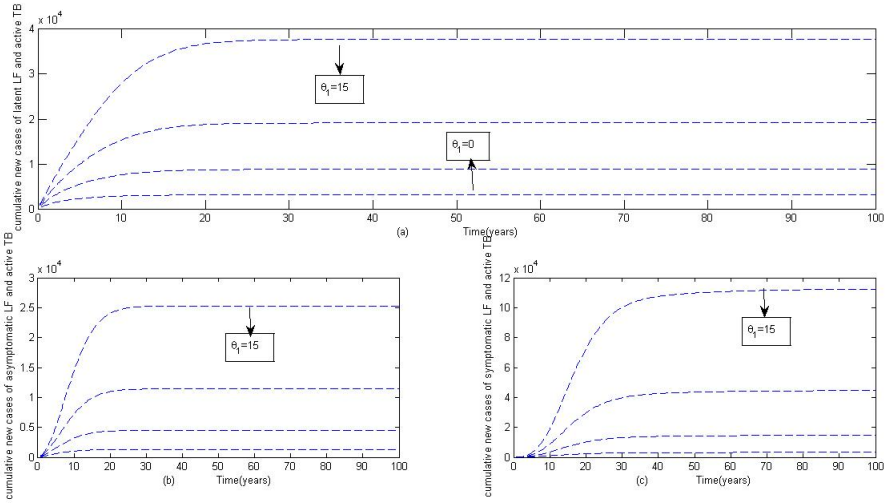

Figure 5: New cases of (a) E_{hT} (b) A_{hT} and (c) I_{hT} when θ_1 is varied.

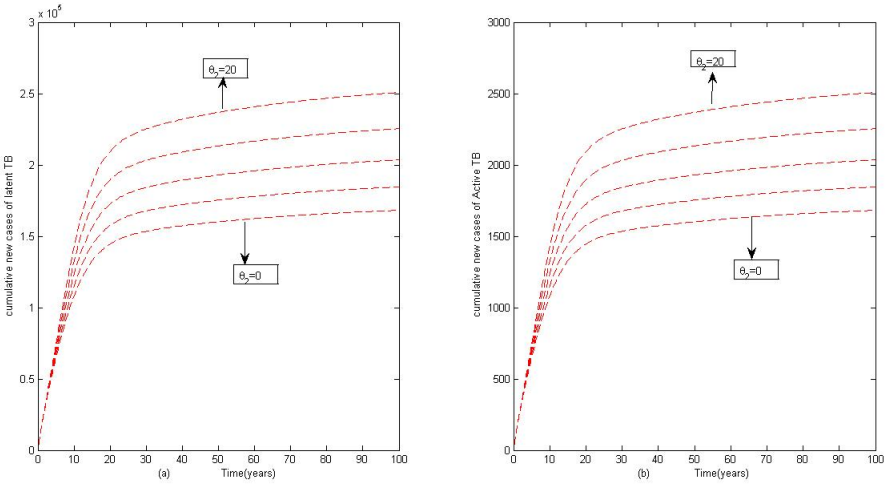

Figure 6: New cases of (a) E_t and (b) I_T when θ_2 is varied.

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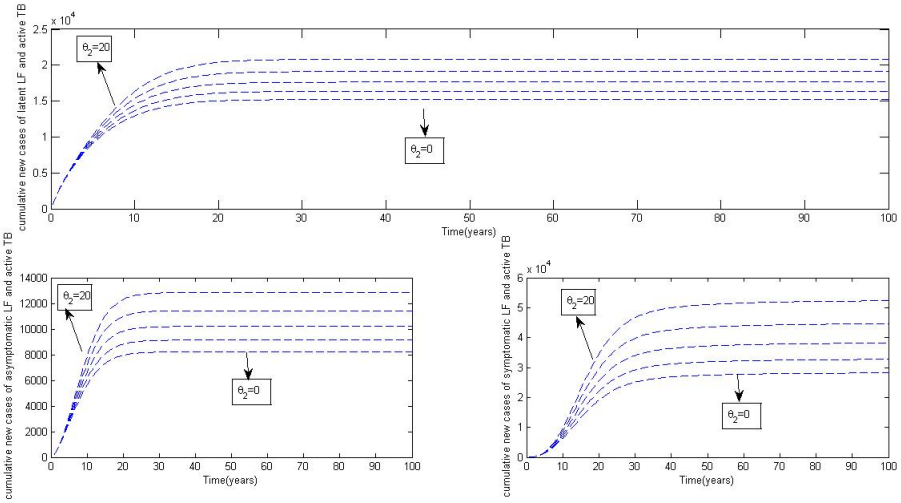

Figure 7: New cases of (a) E_{hT} (b) A_{hT} and (c) I_{hT} when θ_2 is varied.

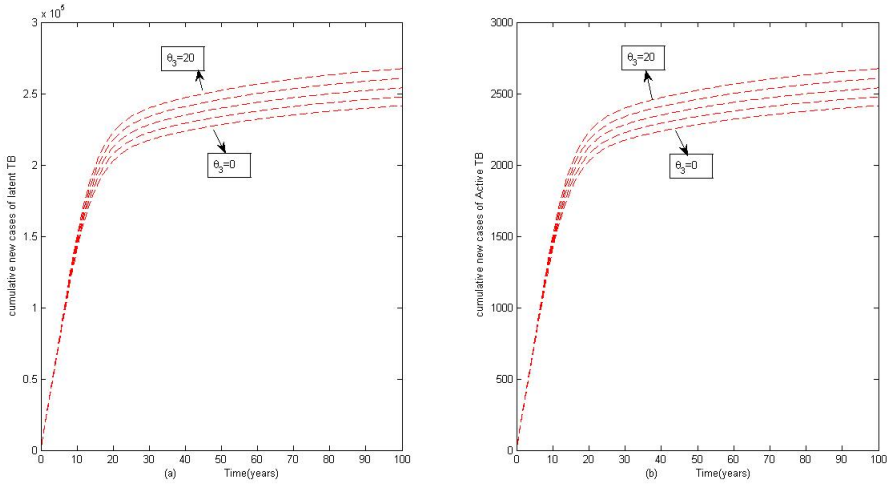

Figure 8: New cases of (a) E_t and (b) I_T when θ_3 is varied.

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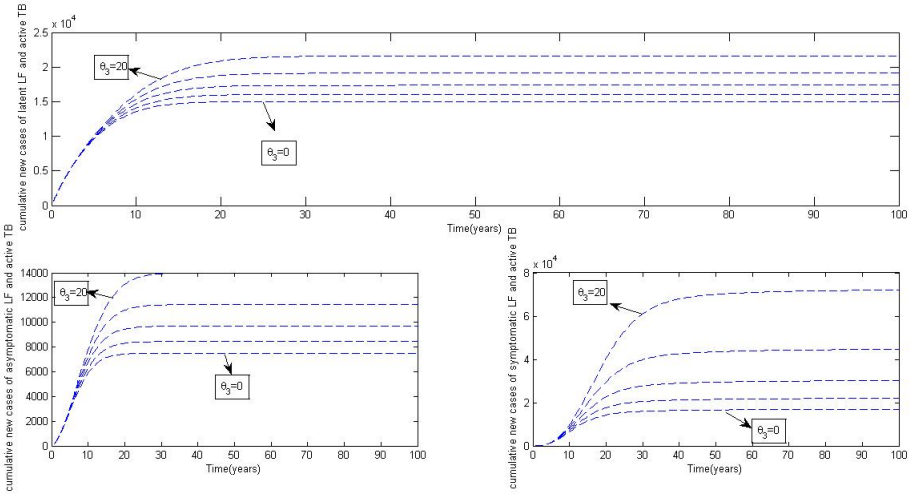

Figure 9: New cases of (a) E_{hT} (b) A_{hT} and (c) I_{hT} when θ_3 is varied.

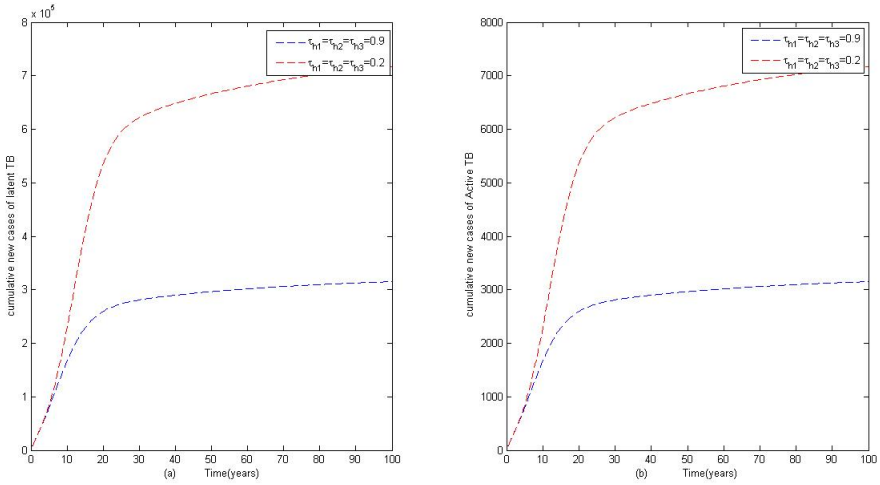

Figure 10: New cases of (a) E_t and (b) I_T with τ_{h1}, τ_{h2} and τ_{h3} varied.

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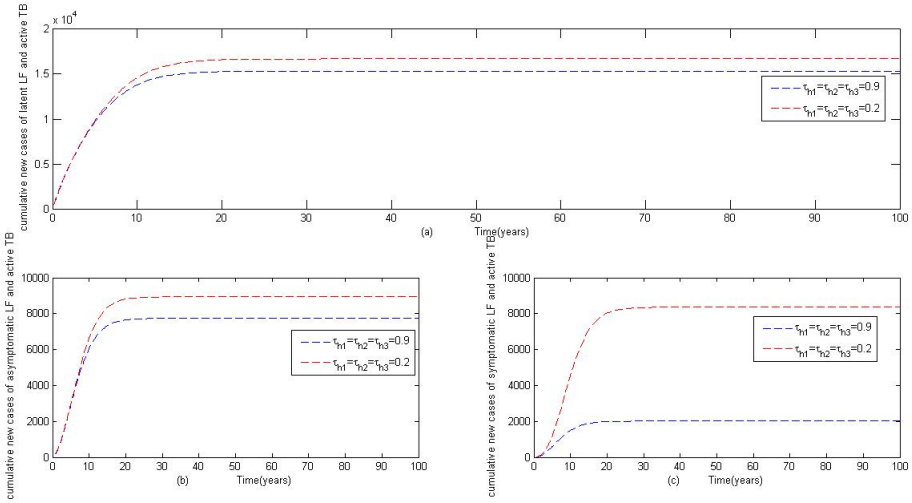

Figure 11: New cases of(a) E_{hT} (b) A_{hT} and (c) I_{hT} with τ_{h1}, τ_{h2} and τ_{h3} varied.

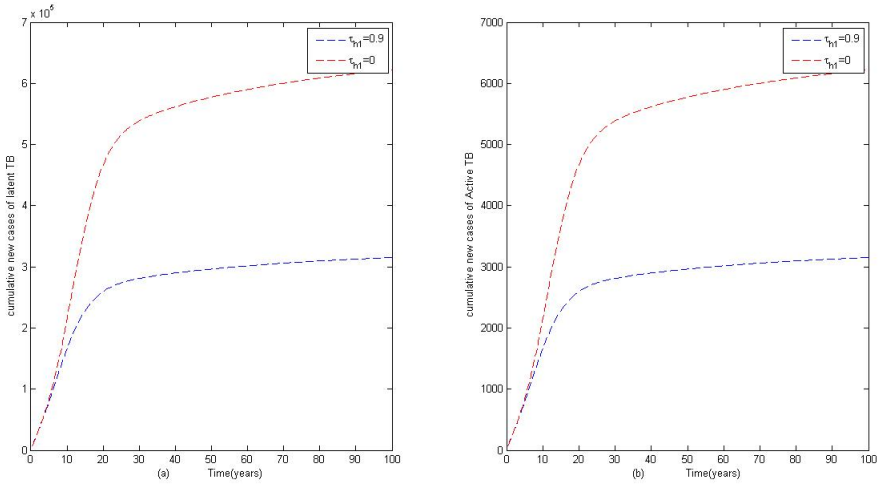

Figure 12: New cases of (a) E_t and (b) I_T with τ_{h1} varied.

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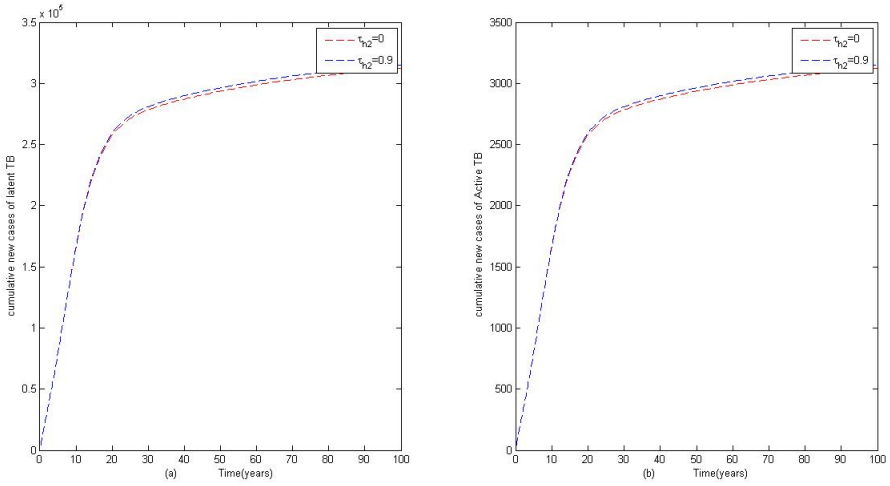

Figure 13: New cases of (a) E_t and (b) I_T with τ_{h1} varied.

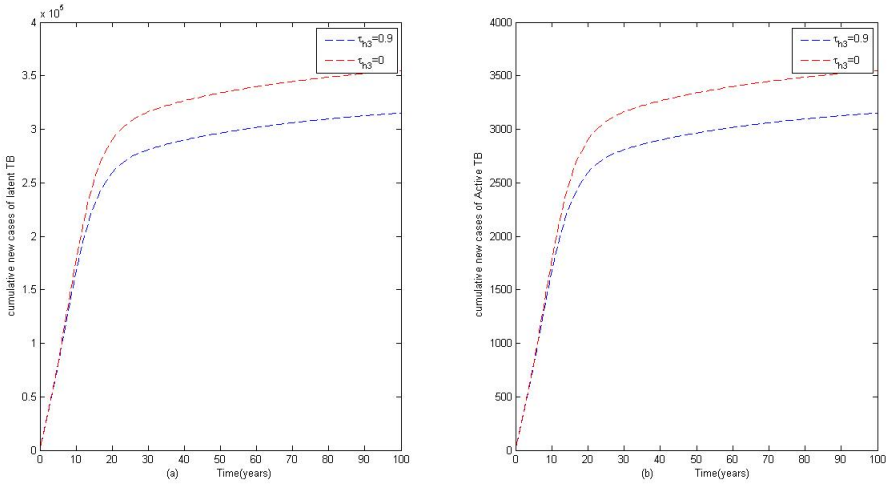

Figure 14: New cases of (a) E_t and (b) I_T with τ_{h1} varied.

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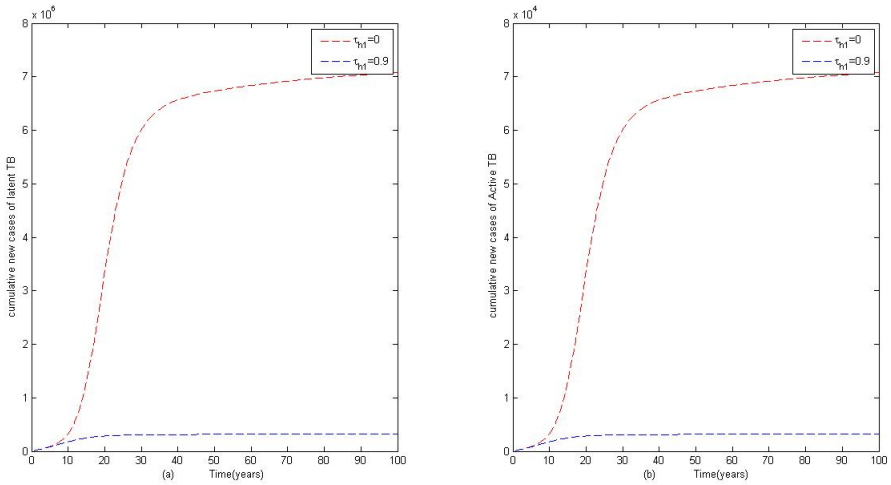

Figure 15: New cases of (a) E_t and (b) I_T with τ_{h1} varied.

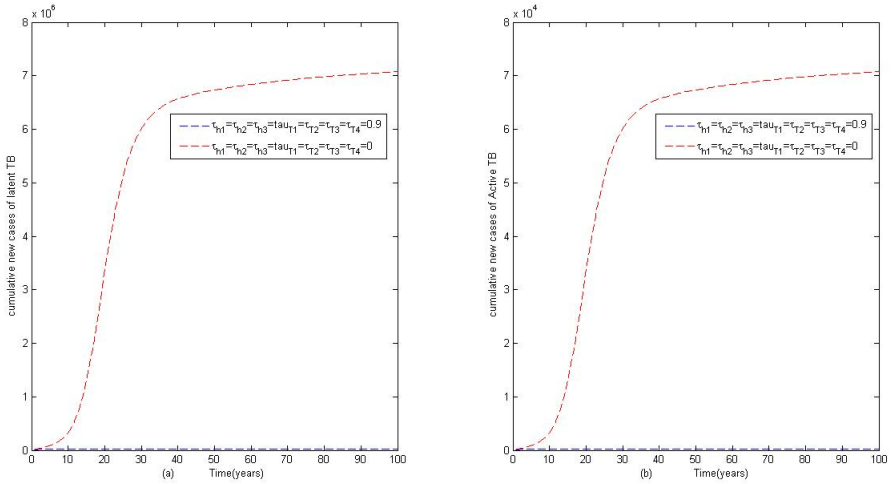

Figure 16: New cases of (a) E_t and (b) I_T with τ_{h1} varied.

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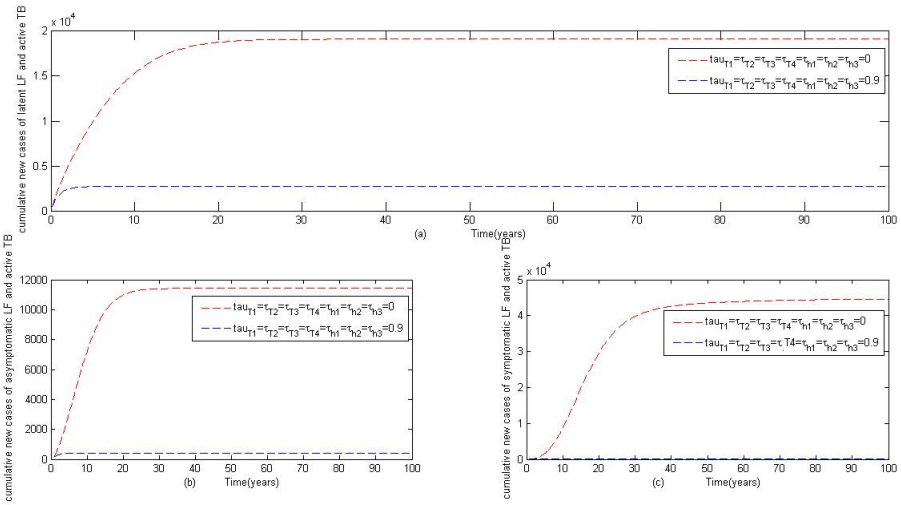

Figure 17: New cases of (a) E_t and (b) I_T with τ_{h1} varied.

6 Results and Discussion

Figures 2a, 2b and 2c show the cumulative new cases of latent LF and active TB, asymptomatic LF and active TB, and symptomatic LF and active TB when θ_1, θ_2 and θ_3 are varied from 1 to 15. θ_1, θ_2 and θ_3 are modification parameters which account for the increased susceptibility of those with LF disease to TB. According to WHO (2018), LF impede the smooth functioning of the lymphatic system. The lymphatic system houses and maintain the human immune defence system. If the functions of the lymphatic system is affected because of the presence of *filariasis*, the immunity of the organism is compromised and this can open the door for TB and other infectious diseases.
Figures 3a and 3b shows the cumulative new cases of latent and active TB when θ_1, θ_2 and θ_3 are varied from 1 to 20, indicating that those with LF disease are highly susceptible to TB and other infectious diseases. Similar trends was also observed in Figures 4a and 4b, Figures 5a, 5b and 5c when θ_1 only was varied from 0 to 20. Figures 6a and 6b shows the cumulative new cases of latent TB E_t and active TB, I_T when the modification parameters θ_2 was varies from $0\breve{}20$.
Figures 7a, 7b and 7c show the cumulative new cases of the co-infected individual with active TB cases. That is, latent LF and active TB (E_{hT}), asymptomatic if and active TB, and symptomatic LF and active TB (I_{hT}) when θ_2 was varied from $0-20$. Here, there was no significant change in the dynamic of the co-infected class. Compared to when θ_1 was varied. The same trend was also observed in Figures 8a and 8b, 9a, 9b and 9c when θ_3 was varied.
Comparing Figures (3a and 3b), (5a and 5b) and (8a and 8b), it was found that those who have latent LF disease are highly susceptible to TB in the single and co-infected cases.
Figure 10 show the cumulative new cases of latent TB (E_t) and active TB (I_T) when we applied LF-only treatment strategy. In this strategy, all the treatments for LF were adjusted from $0.2-0.9$ i.e. $\tau_{h1} = \tau_{h2} = \tau_{h3} = 0.2$ and $\tau_{h1} = \tau_{h2} = \tau_{h3} = 0.9$. It was found that the cumulative new cases of latent TB (E_t) and active TB (I_T) dropped significantly. In the co-infected cases, there was a drop in the co-infected compartment containing symptomatic LF and active TB (I_{hT}), but in the other co-infected classes, containing active TB (E_{hT}) and (A_{hT}), there was no significant drop in the number of new co-infected cases. because only those with LF symptons were treated (see Figures 11a, 11b and 11c).
In Figures 12a and 12b, when the treatment for the latent LF infected individual was varied, we found that the cumulative new case of latent TB (E_t) and active TB (I_T) dropped significantly, but in Figure 13 and 14 there was no significant changes in the cumulative new cases of latent TB (E_t) and active TB (I_T) when the treatment τ_{h2} and τ_{h3} were varied respectively.

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In Figure 15, when all those with LF disease are treated and τ_{h1} varied from $0.2 - 0.9$, there was a significant drop in the cumulative new cases of those with latent TB (E_t) and active TB (I_T). We found in Figure 16 a significant drop in the cumulative new cases of latent TB (E_t) when the treatments rate was increased from 0 to 0.9. Similar trend was also observe in Figure 17.

7 Conclusion

We proposed and analysed 17 nonlinear Mathematical model to study how the endemicity of LF affect the population dynamics of TB. The mathematical model presented in this work provide mathematical and epidemiological insight into the transmission dynamics of TB–LF co-infection. The numerical results presented in Figures 2, 3, 4, 5, 6, 7, 8 and 9 shows that LF infection increases susceptibility to TB infection. This is in agreement with [2] that, persons with lowered immunity such as HIV, diabetes, immune disorder etc are at a higher risk of contacting infectious diseases. The results presented in Figures 10 to 17 shows that increasing the rate of diagnosis and treatment of active TB and symptomatic LF cases can reduce the incidence of co-infection in the community.

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