

Mathematical Model of the Transmission Dynamics of Genital Elephantiasis (Lymphatic Filariasis)

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Abstract

This thesis presents a deterministic compartmental model, developed and analyzed to investigate the dynamics of lymphatic filariasis disease, through mosquito-borne infection. The model is in eight compartments: five for the human population and three for the mosquito population based on the microfilariae and antibody levels. The existence of the invariant region where the model is epidemiologically feasible and the positivity of the solution were established. The existence of Disease-free equilibrium (DFE) and the Endemic equilibrium (EE) were determined. Stability analysis of the disease-free equilibrium was investigated via the threshold parameter (reproduction number R_0) obtained using the next generation matrix technique. The model was found to be locally asymptotically stable when the basic reproduction number is less than unity for both special and non special case. It was also revealed that the disease is endemic when $R_0 > 1$. It was proved through Lyapunov method that the DFE and EE are globally asymptotically stable. Simulation analysis was also carried out and it was shown that even when all lymphatic filariasis cases displaying elephantiasis symptoms are put on treatment it will not be able to eradicate the disease. This result suggests that effective control of lymphatic filariasis may lie in treatment for those displaying symptoms. It was also shown that if on the long run as the biting rate of the Mosquitoes increases, the infected population increases. Then as biting rate decreases, then the chronic infected individuals are completely eradicated from the population while the highly infected humans are reduced. The simulation also showed the impact of the effectiveness of treatment on the chronic infected humans, where we see that the population reduces rigorously until we get to a period of 70 days and then begins to increase again. This shows that the treatment strategies are not effective or perfect. Hence there are chances of fail in treatment. Furthermore our analysis shows that on a long run the trend continues indefinitely.

Key words: Genital Elephantiasis, Mathematical Modeling, Lymphatic filariasis, Endemic Equilibrium (EE)

1. Introduction

Lymphatic filariasis is a vector-borne disease which is prevalent in the tropical and sub-tropical regions of the world Michael et al. (1996). The overwhelming presence of this disease in these regions can be attributed to poor hygienic environmental human activities that make the vector-mosquito to thrive. Over 120 million people have been infected with this disabling disease and about one-third of this number exhibit the clinical manifestation, Ottesen (1994).

Unlike many vector-borne diseases, lymphatic filariasis is not life threatening WHO Report, (1995). Infected mosquitoes transmit the L3 (infective stage) state larvae to the susceptible human host. These larvae travel to the lymphatic glands where they mature into adulthood and reproduce WHO Report, (1995). An average of 80,000 microfilariae a day is reproduced after mating, Manguina, et al. (2009).

The microfilariae (MF) or L1 stage larvae are released into the blood stream where a susceptible mosquito picks it during a bite, Addiss and Dreyer, (2000). People with weak immune responses to lymphatic filariasis parasite (notably wuchereria bancrofti; about 90%), they develop filarial fever, subsequently hydrocele, lymphedema and elephantiasis, if not treated early Addiss and Dreyer, (2000).

The diseases manifestation is said to be catalyzed by the co-habitation of the parasite with a bacteria called Wolbachia, McNulty, et al. (2012).

With more than 1.3 billion people at risk of the infection (Michael et al 1996) and the dismal underlying socio economic consequence of the diseases Addiss and Dreyer (2000) and WHO Report, (1995), it became imperative for the world community to work together towards the elimination of lymphatic filariasis WHO Report (1995). Elephantiasis which is caused by obstruction of the lymphatic system, which results in the accumulation of fluid called LYMPH in the affected areas.

The lymphatic system helps to protect the body against infection and diseases. It functions as part of the immune system, consisting of a network of tubular channels (lymph vessels) that drain a thin watery fluid known as lymph from different area of the body into the blood stream. Obstruction of these vessels results in the massive swelling and gross enlargement characteristic of elephantiasis.

In areas where filariasis is endemic, the most common cause of the elephantiasis is a parasitic disease known as lymphatic filariasis. It primarily affects the legs and genitals, resulting in baggy, thickened and ulcerated skin, along with fever and chills. This condition can be very painful and uncomfortable, and it reduces the sufferer's ability to live a normal life. A serious complication can be the obstruction on blood vessels, which limit blood supply and cause the skin to become infected.

Plaisier et al. (1998) considered a model based on transmission of L3 stage larvae from mosquito to the human host. Factors such as immunity of individuals, vector control and drug treatment for the patients were incorporated in their model. However, simulations were only carried out using modeling framework (LYMFASIM) to evaluate some control programs which need analytical justification.

Subramanian et al. (2004) also used LYMFASIM micro simulation model to investigate the effect of immunity, considering three variations, on the intensity of infection in Pondicherry, India after interruption of transmission through vector control. This last study was aimed at inquiring the impact of immunity on infection in controlling vectors activities. These models

predicted large reductions in the prevalence of microfilariae following 3 and 6 years after the integrated vector control management in Pondicherry, India.

Mathematical model for lymphatic filariasis transmission and control challenges and prospect was also considered by Swaminabhan, et al (2008). They highlight factors related to the efficacy of the drugs of choice, their mode of action, and the possibility that drug resistance may develop; the role of vector-parasite combinations; the magnitude of transmission thresholds; host-parasite interactions and their effects on the dynamics of infection and immunity; parasite biology, and progression to lymphatic filariasis associated disease. The two mathematical models developed offer potential decision making tools for transmission and control of lymphatic filariasis

Das and Subramanian (2002) considered modeling the epidemiology, transmission and control of lymphatic filariasis. Mathematical models have proven valuable in gaining quantitative insights into the population dynamics of the parasites, and may be used to make credible predictions of the likely outcomes of various control strategies. The article provides an overview of the development of the relevant mathematical/statistical models and of their application in studies of the epidemiology, transmission and control of lymphatic filariasis.

Chan, et al. (2008) formulated a dynamical model of infection and disease in lymphatic filariasis. An epidemiological model for the spread of lymphatic filariasis, a mosquito-borne infection, was developed and analyzed. The epidemic thresholds known as the reproduction number and equilibria for the model are determined and stabilities analyzed. And the LYMFASIM simulation program for modeling lymphatic filariasis and its controls were considered by Plaisier, et al. (1998). With LYMFASIM, a variety of hypotheses can be tested about the life history of the parasite *Wuchereria bancrofti*, its transmission from man to man through mosquitoes, the role of the immune system in regulating parasite numbers, the development of disease symptoms, and the effects of control measures (drug treatment or mosquito control).

McGill, et al (2015) also formulated a mathematical model for the transmission dynamics and control of lymphatic filariasis. In their model they considered six compartments, four for the human population and two for the mosquito population. They considered the rate at which a susceptible human get infected when there is an interaction between infected mosquitoes. They also considered the control of elephantiasis.

The pathogenesis of lymphatic filariasis has been a matter of debate for many decades. In their paper, Dreyer et al (2000) proposed a dynamical model of bancroftian filariasis, integrating clinical, parasitological, surgical, therapeutic, ultrasonographic and histopathological data. This model has profound implications for filariasis control programs and the management of the individual patient. This study describes the relationship between transmission intensity and infection and disease due to *Wuchereria bancrofti* in an endemic area of Papua New Guinea. The prevalence of microfilaremia in the entire study population was 66%. Of 1892 persons examined, 6.2% and 12.3% had lymphedema of the legs and hydroceles, respectively. The prevalence of microfilaremia and clinical morbidity were lowest

in persons less than 20 years old and increased progressively with age. Annual transmission potential and annual infective biting were monitored in five villages where *Anopheles punctulatus* and *Anopheles koliensis* are the only vectors of *W. bancrofti*. Both measures of the entomologic inoculation rate were positively associated with the village-specific microfilarial rate, mean intensity of microfilaremia, and prevalence of leg edema. These data indicate that transmission intensity is a major determinant of patent infection and morbidity rates in bancroftian filariasis. The lack of a quantitative framework that describes the dynamic relationships between infection and morbidity has constrained efforts aimed at the community-level control of lymphatic filariasis.

In their paper, Chan et al (1998) described the development and validation of EPIFIL, a dynamic model of filariasis infection intensity and chronic diseases. Infection dynamics were modeled using the well established immigration-death formulation, incorporating the acquisition of immunity to infective larvae over time. The dynamics of disease (lymphodema and hydrocele) were modeled as a catalytic function of a variety of factors, including worm load and the impact of immunopathological responses. The model was parameterized using age-stratified data collected from a Bancroftian filariasis endemic area in Pondicherry in southern India. The fitted parameters suggest that a relatively simple model including only acquired immunity to infection and irreversible progression to disease can satisfactorily explain the observed infection and disease patterns. Disease progression is assumed to be a consequence of worm induced damage and to occur at a high rate for hydrocele and a low rate for lymphodema. This suggests that immunopathology involvement may not be a necessary component of observed age-disease profiles. These findings support a central role for worm burden in the initiation and progression of chronic filarial disease.

Alison and Robert (2010) surveyed the current state of a group of parasitic and microbial diseases called the Neglected Tropical Diseases (NTDs). These diseases currently infect a billion people, primarily in socioeconomically depressed areas of the world, are a leading cause of worldwide disability, and are responsible for approximately 534,000 deaths per year. They focused on several subcategories: protozoans, helminthes and bacterial diseases. They identify the populations most at risk from these diseases, and outlined symptoms and other disease burdens. They also examined the progress being made in controlling NTDs, including the current state of drug development. They further examined mathematical modeling of NTDs. While mathematical modeling is not bound by many of the strictures of access, data collection and infrastructure funding, they nevertheless demonstrated that few NTDs have received much attention from mathematical models, and that some have received no attention at all. They concluded that simple mathematical models could contribute significantly to the understanding of these diseases and the efforts required controlling them, at very little cost. Finally they concluded that investment in prevention, treatment and awareness of NTDs is urgently warranted.

Ottesen et al (1997) reported that Lymphatic filariasis infected 120 million people in 73 countries worldwide and continues to be a worsening problem, especially in Africa and the

Indian subcontinent. Elephantiasis, lymphoedema, and genital pathology afflicted 44 million men, women and children; another 76 million have parasites in their blood and hidden internal damage to their lymphatic and renal systems. In the past, tools and strategies for the control of the condition were inadequate, but over the last 10 years dramatic research advances have led to new understanding about the severity and impact of the disease, new diagnostic and monitoring tools, and, most importantly, new treatment tools and control strategies. The new strategy aims both at transmission control through community-wide (mass) treatment programmes and at disease control through individual patient management. Annual single-dose co-administration of two drugs (ivermectin + diethylcarbamazine (DEC) or albendazole) reduces blood microfilariae by 99% for a full year; even a single dose of one drug (ivermectin or DEC) administered annually can result in 90% reductions; field studies confirm that such reduction of microfilarial loads and prevalence can interrupt transmission. New approaches to disease control, based on preventing bacterial super-infection, can now halt or even reverse the lymphoedema and elephantiasis sequelae of filarial infection. Recognizing these remarkable technical advances, the successes of recent control programmes, and the biological factors favouring elimination of this infection, the Fiftieth World Health Assembly recently called on WHO and its Member States to establish as a priority the global elimination of lymphatic filariasis as a public health problem.

Srividya et al (1991) in their study examined the relationship between the dynamics of *Wuchereria bancrofti* infection and the development of chronic lymphatic disease. Data sets from Pondicherry, South India, and Calcutta were used to estimate the age-specific proportion of the endemic population which has converted from microfilaria positive to amicrofilaraemia, and was assumed to be at risk of disease. For men, but not women, the age-prevalence profile of the estimated population 'at risk' was shown to correspond closely to the observed age-prevalence of chronic lymphatic disease in the same community. For both sexes, and independent of age, approximately 11% of the population at risk eventually developed lymphoedema. They concluded that these observations suggest that filariasis endemic populations consist of those individuals who remain amicrofilaraemic and asymptomatic, and those who progress through the sequence: uninfected, microfilaraemic, amicrofilaraemic, to develop irreversible obstructive lymphatic pathology.

Das and Subramanian (2002) in their work reported that *Wuchereria bancrofti* transmitted by *Culex quinquefasciatus* accounts for >90% of the global burden of lymphatic filariasis (LF). Recent advances in diagnostic and control tools and a better epidemiological understanding of the disease have led to hope that LF is eradicable. The World Health Organization has helped a number of member countries to launch nation-wide programmes of mass treatment with antifilarial drugs such as diethylcarbamazine, albendazole and ivermectin, for the elimination of this disease. In order to make rational decisions about control strategies, reliable predictions of the long-term impact of such treatment, and of alternative interventions, need to be made, and these can only be based on a sound, quantitative understanding of the population biology of the parasites. Mathematical models have proven valuable in gaining quantitative insights into the population dynamics of the parasites, and may be used to make credible predictions of

the likely outcomes of various control strategies. This article provides an overview of the development of the relevant mathematical/statistical models and of their application in studies of the epidemiology, transmission and control of lymphatic filariasis.

Michael et al. (2008) reported that the ultimate goal of the global programme against lymphatic filariasis is eradication through irrevocable cessation of transmission using 4 to 6 years of annual single dose mass drug administration. The costs of eradication, managerial impediments to executing national control programmes, and scientific uncertainty about transmission endpoints, are challenges to the success of this effort, especially in areas of high endemicity where financial resources are limited. We used a combined analysis of empirical community data describing the association between infection and chronic disease prevalence, mathematical modeling, and economic analyses to identify and evaluate the feasibility of setting an infection target level at which the chronic pathology attributable to lymphatic filariasis - lymphoedema of the extremities and hydroceles - becomes negligible in the face of continuing transmission as a first stage option in achieving the elimination of this parasitic disease. The results show that microfilaria prevalence below a threshold of 3.55% at a blood sampling volume of 1 ml could constitute readily achievable and sustainable targets to control lymphatic filarial disease. They also showed that as a result of the high marginal cost of curing the last few individuals to achieve elimination, maximal benefits can occur at this threshold. Indeed, a key finding from our coupled economic and epidemiological analysis is that when initial uncertainty regarding eradication occurs and prospects for resolving this uncertainty over time exist, it is economically beneficial to adopt a flexible, sequential, eradication strategy based on controlling chronic disease initially.

Bitran et al (2009) in their paper analyzed the rationale for, and costs associated with, the control and elimination of neglected tropical diseases (NTDs) in Latin America and the Caribbean. They also estimated the magnitude of potential health gains. The results suggest that lymphatic filariasis, onchocerciasis, and trachoma can be feasibly and affordably eliminated by 2020, at a total cost of US\$128 million. Control of other NTDs could produce important reductions in prevalence and incidence, along with other social and economic benefits. They finally concluded that in particular, controlling soil-transmitted helminths (roundworm and hookworm, for example) would produce total costs of \$41 million between now and 2020.

Supriatna, et al. (2009) in their paper discussed a mathematical model for the transmission of *Lymphatic Filariasis* disease in Jati Sampurna, West Java Indonesia. Their model assumes that acute infected humans are infectious and treatment is given to a certain number of acute infected humans found from screening process. The treated acute individuals are assumed to remain susceptible to the disease. The model was analyzed and it was able to found a condition for the existence and stability of the endemic equilibrium. A well known rule of thumb in epidemiological model, that is, the endemic equilibrium exists and stable if the basic reproduction number is greater than one, was shown. Moreover, it was also shown that if the level of screening n is sufficiently large, current medical treatment strategy will be able to

reduce the long-term level of incidences. However, in practice it was not realistic and cannot eliminate the disease, in terms of reducing the basic reproduction number. The reproduction number could be reduced by giving additional treatments, such as reducing the biting rate and mosquito's density. This suggests that there should be a combination of treatment to eliminate the disease.

Other useful works that were reviewed in this work were the works of Michael, et al. (2006), Michael, et al. (2004), Gersoyitz and Hammer (2003) and Chan, et al. (1998).

Here, in our this work, we develop and analyze a mathematical model that captures the transmission dynamics of lymphatic filariasis using differential equations to explore if treatment for those symptoms alone will be able to keep the infection under control. We also considered eight compartments, five for the human populations and three for the mosquito population. We also considered the rate at which susceptible humans and susceptible mosquitoes become infected. We considered only the natural death rate of humans and mosquitoes.

2. Model Formulation

2.1 Symbols and Parameters

S_H - Susceptible humans

E_H - Exposed humans

I_{Hh} - High concentration of microfilaria with antibody in humans

I_{Hc} - Chronic, very high concentration of microfilaria and antibody, symptoms showing at scrotum of humans

T_H - Treatment as a result of successful operation at scrotum in humans

S_m - Susceptible mosquitoes

E_m - Expose mosquitoes

I_m - Infected mosquitoes

Λ_H - Recruitment rate of susceptible humans through birth and migration

Λ_m - Recruitment rate of susceptible mosquitoes through breeding

b - Biting rate of the vector mosquitoes

β_H - Rate that a Human becomes infectious after interacting with infected mosquitoes

β_m - Rate that a vector mosquitoes becomes infectious after feeding from infected human

μ_H - Natural death rate of humans

μ_m - Natural death rate of mosquitoes

α_H - Rate of progression of humans from the exposed state to infectious state

α_m - Rate of progression of mosquitoes from the exposed state to infectious state

γ_H - Rate of progression of human from infectious state to chronic states.

δ_H - Rate at which chronic infected humans undergo treatment

ω - Rate at which high infected humans undergo treatment

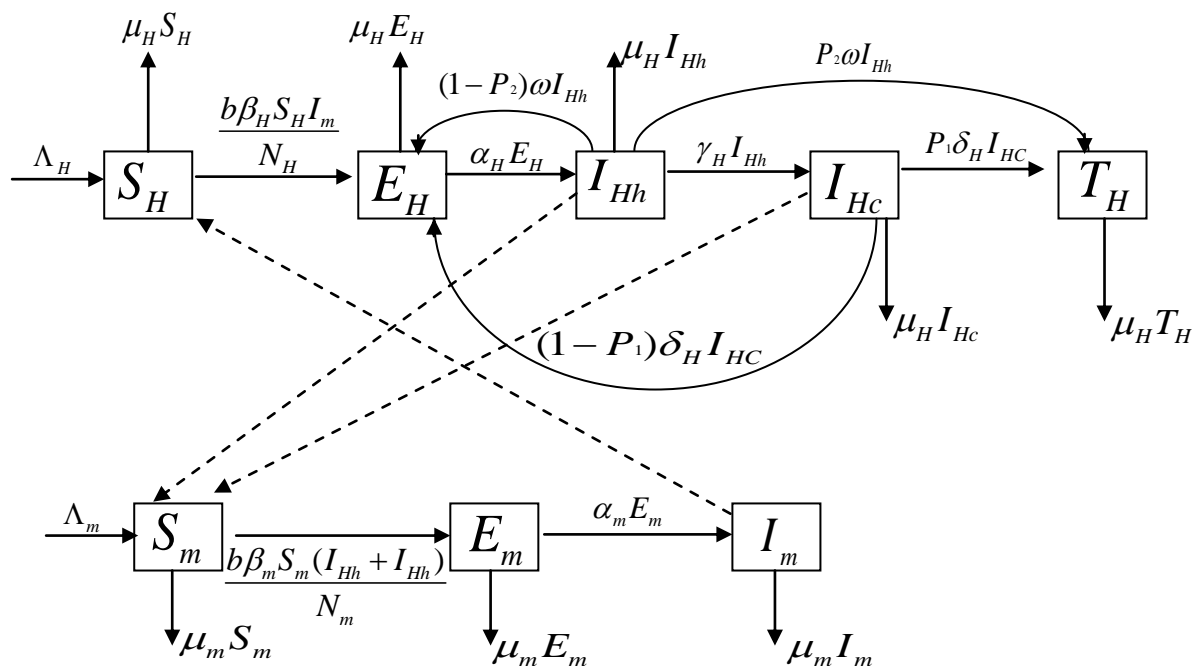
p_1 - Fraction of individuals in the chronic infected human population whose treatment were successful

p_2 - Fraction of individuals in the highly infected population whose treatment were successful

2.2 Assumptions of the Model

1. It is assumed that because infected individuals E_H have a low level of microfilaria they are un-dictated by diagnostic tests before treatment.
2. Both the highly infected and the chronic infected individuals can transmit the disease.
3. The infection cannot result to the death of the infected population.
4. Individuals can only be treated through operation at the scrotum.
5. Failure in treatment in both cases of highly and chronic infectiousness takes an individual back to the exposed.
6. Fractions of individuals whose treatment fail is the same for the highly infected and chronically infected
7. Treated individuals acquire permanent immunity against re-infection

2.3 Model Flow Chart



2.4 The Model Formulation

Using this flow diagram, the assumptions, symbols and parameters stated above, we now formulate the required model as follows:

$$\left. \begin{aligned}
 \frac{dS_H}{dt} &= \Lambda_H - \frac{b\beta_H S_H I_m}{N_H} - \mu_H S_H \\
 \frac{dE_H}{dt} &= \frac{b\beta_H S_H I_m}{N_H} + (1-p_1)\delta_H I_{Hc} + (1-p_2)\omega I_{Hh} - (\alpha_H + \mu_H)E_H \\
 \frac{dI_{Hh}}{dt} &= \alpha_H E_H - (\omega + \gamma_H + \mu_H)I_{Hh} \\
 \frac{dI_{Hc}}{dt} &= \gamma_H I_{Hh} - (\delta_H + \mu_H)I_{Hc} \\
 \frac{dT_H}{dt} &= p_1\delta_H I_{Hc} + p_2\omega I_{Hh} - \mu_H T_H \\
 \frac{dS_m}{dt} &= \Lambda_m - \frac{b\beta_m S_m (I_{Hc} + I_{Hh})}{N_m} - \mu_m S_m \\
 \frac{dE_m}{dt} &= \frac{b\beta_m S_m (I_{Hc} + I_{Hh})}{N_m} - (\alpha_m + \mu_m)S_m \\
 \frac{dI_m}{dt} &= \alpha_m E_m - \mu_m I_m
 \end{aligned} \right\} (2.1)$$

The model consists of human and mosquito interacting populations. The human population is sub-divided based on the level of microfilariae, in the human hosts that include susceptible (S_H) no parasites, low infected individual (E_H), with low level of microfilariae less than (20mf/ml), highly infected individual (I_{Hh}), with high level of MF, chronically infected individuals (I_{Hc}) which is characterized by higher MF and treated individuals (T_H). The mosquito population is divided into susceptible (S_m), Exposed (E_m) and infected mosquito, (I_m). Hence,

$$N_H(t) = S_H(t) + E_H(t) + I_{Hh} + I_{Hc} + T_H(t)$$

$$N_m(t) = S_m(t) + E_m(t) + I_m(t)$$

Recruitment into the human population is assumed to be a constant rate of Λ_H which include birth and migration. Natural mortality is the only way individuals can die at an assumed rate of μ_H which is proportional to class sizes (Hooper et al 2009) and (WHO Report, 1995). When an infectious mosquito takes a bite at a susceptible host, there exist some probability L_3 stage infective larvae, can be transmitted to the individual (Michael, 1999), where the infection rate is λ_m which results on movement of susceptible (S_H) individuals to Exposed $E_H(t)$ and the rate is defined by

$$\lambda_m = \frac{b\beta_H S_H I_m}{N_h} (1-\theta)$$

where θ represents the factor at the bed-net effect reduce the transmission rate. If $\theta=1$ means bed-nets are used by all individuals, implying $\lambda_m = 0$ and $\theta=0$ means bed-nets were not used. β_H is the rate of parasite transmission to human, b is the average bites per mosquito per day. Infected people in the Exposed class E_H can leave because of diseases progression at the rate α_H to high infectious class I_{Hh} or by natural mortality rate μ_H . Highly infected individuals can progress to chronic infectious class I_{Hc} at a constant rate γ_H . Treatment of I_{Hh} and I_{Hc} infected humans occurs at a rate of ω and δ_H that reduces the intensity level of microfilariae (MF). It's assumed that because infected individuals E_H have low level of microfilariae they are undetected by diagnostic, tests before treatment.

The rate of recruitment in mosquito population is Λ_m and the mortality at the rate μ_m . The susceptible mosquito engorges microfilariae MF (L_1 stage) when it bites infected individuals. I_{Hh} and I_{Hc} and therefore, becomes infected at a rate λ_h defined by

$$\lambda_h = \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m}$$

Where β_m the rate of parasite transmission to mosquito

Patients in chronic class whose infection status had reached an advanced stage will start showing clinical manifestation of hydrocoele, lyphoedema and elephantiasis, Hooper, et al (2009). According to Krishnamoorly et al (2004), there is a higher rate of the death of a mosquito that engorges microfilariae beyond its saturation level.

3. Model Analysis

In this paper, we analyze the elephantiasis model, we first prove that the set of solution is confined in a feasible region, and then show that all the solutions are positive. We investigate the existence and stability of the equilibrium point. Further we computed the basic reproduction number. We also proved global stability of the disease free equilibrium (DFE) and endemic equilibrium (EE) using the lyapunov function. Finally we considered the numerical solution of the model using simulation.

3.1 Basic Properties of the Model

3.1.1 Invariant Property

Theorem 3.1: The closed set

$$D = \left\{ (S_H, E_H, I_{Hh}, I_{Hc}, T_H, S_m, E_m, I_m) \in \mathfrak{R}_+^8 : S_H + E_H + I_{Hh} + I_{Hc} + T_H \geq 0; S_m + E_m + I_m \geq 0 \right\}$$

is positively- invariant and attracting with respect to the model of (2.1).

Proof:

Considering the human population we have,

$$N_H = S_H + E_H + I_{Hh} + I_{Hc} + T_H \tag{3.1}$$

Differentiating (4.1) we obtain

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_{Hh}}{dt} + \frac{dI_{HC}}{dt} + \frac{dT_H}{dt} \quad (3.2)$$

Substituting (2.1) into (3.2) gives us

$$\begin{aligned} \frac{dN_H}{dt} &= \Lambda_H - \frac{b\beta_H S_H I_m}{N_H} - \mu_H S_H + \frac{b\beta_H S_H I_m}{N_H} + (1-p_1)\delta_H I_{HC} + (1-p_2)\omega I_{Hh} - (\alpha_H + \mu_H)E_H + \alpha_H E_H \\ &\quad - (\omega + \gamma_H + \mu_H)I_{Hh} + \gamma_H I_{Hh} - (\delta_H + \mu_H)I_{HC} + p_1\delta_H I_{HC} + p_2\omega I_{Hh} - \mu_H T_H \\ &= \Lambda_H - \mu_H N_H \end{aligned}$$

$$\Rightarrow \frac{dN_H}{dt} \leq \Lambda_H - \mu_H N_H$$

$$\frac{dN_H}{dt} + \mu_H N_H \leq \Lambda_H$$

$$\frac{d}{dt} (N_H e^{\mu_H t}) \leq \Lambda_H e^{\mu_H t}$$

$$N_H e^{\mu_H t} \leq \frac{\Lambda_H}{\mu_H} e^{\mu_H t} + k$$

$$N_H \leq \frac{\Lambda_H}{\mu_H} + k e^{-\mu_H t}$$

Taking limit as $t \rightarrow \infty$

$$\Rightarrow N_H \leq \frac{\Lambda_H}{\mu_H} \quad (3.3)$$

Here we study the model for human population from the epidemiological concept in the feasible region.

$$D_H = \mathbb{R} \left\{ (S_H, E_H, I_{Hh}, I_{HC}, T_H) \in \mathbb{R}_+^5 : \leq \frac{\Lambda_H}{\mu_H} \right\}$$

Considering the mosquito population we have,

$$N_m = S_m + E_m + I_m \quad (3.4)$$

Differentiating (3.4) we have

$$\Rightarrow \frac{dN_m}{dt} = \frac{dS_m}{dt} + \frac{dE_m}{dt} + \frac{dI_m}{dt} \quad (3.5)$$

Substituting (2.1) into (3.5) we obtain

$$\Rightarrow \frac{dN_m}{dt} = \Lambda_m - \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} - \mu_m S_m + \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} - \alpha_m E_m - \mu_m E_m + \alpha_m E_m - \mu_m I_m$$

$$\frac{dN_m}{dt} = \Lambda_m - \mu_m S_m - \mu_m E_m - \mu_m I_m = \Lambda_m - \mu_m (S_m + E_m + I_m) \leq \Lambda_m - \mu_m N_m$$

$$\frac{dN_m}{dt} \leq \Lambda_m - \mu_m N_m$$

$$\frac{dN_m}{dt} + \mu_m N_m \leq \Lambda_m$$

$$\frac{d}{dt} (N_m e^{\mu_m t}) \leq \Lambda_m e^{\mu_m t}$$

$$N_m e^{\mu_m t} \leq \frac{\Lambda_m}{\mu_m} e^{\mu_m t} + k$$

$$N_m \leq \frac{\Lambda_m}{\mu_m} + k e^{-\mu_m t}$$

Taking limit as $t \rightarrow \infty$

$$N_m \leq \frac{\Lambda_m}{\mu_m} \quad (3.6)$$

Also we study the model for mosquito population from the epidemiological concept in the feasible region

$$D_m = \left\{ (S_m, E_m, I_m) \in \mathbb{R}_+^3 : N_m \leq \frac{\Lambda_m}{\mu_m} \right\}$$

Generally, $D = D_H \times D_m \subset \mathbb{R}_+^5 \times \mathbb{R}_+^3$ this means that both the human population at a given time $N_H(t)$ and the mosquito population $N_m(t)$ are confined in the feasible regions D_H and D_m respectively.

That is all the solutions for both human and mosquito are within this region.

Then the total population $N(t) = N_H(t) + N_m(t)$ is confined within the feasible region given by

$$D = \left\{ (S_H, E_H, I_{Hh}, I_{HC}, T_H, S_m, E_m, I_m) \in \mathbb{R}_+^8 : N_H \leq \frac{\Lambda_H}{\mu_H}, N_m \leq \frac{\Lambda_m}{\mu_m} \right\}$$

So we are studying our model in the feasible region $\in \mathbb{R}_+^8$. ■

3.2 POSITIVITY OF SOLUTION

Theorem 3.2: The solutions of the elephantiasis model with positive initial values in the feasible region D remains positive at all time $t > 0$.

Proof: We will adopt the ideas of Chiyaka et. al. (2008) and Lashari et al. (2012) to prove the positivity of solution of our model

From the first equation of the model it is obvious that $S_H(t) > 0$ for all $t \geq 0$; otherwise let there exist $t_* > 0$ such that $S_H(t_*) = 0$, $S'_H(t_*) \leq 0$ and $S_H, E_H, I_{Hh}, I_{HC}, I_H, S_m, E_m, I_m > 0$

For $0 < t < t_*$

$$\Rightarrow S'_H(t_*) = \Lambda_H - \frac{b\beta_H S_H(t_*) I(t_*)}{N_H} - \mu_H S_H(t_*)$$

$$S'_H(t_*) = \Lambda_H > 0$$

Which is a contradiction of the assumption that $S_H(t_*) \leq 0$

Hence $S_H(t) > 0$

In the second equation

Let there exist a $t_* = \sup\{t > 0 : S_H, E_H, I_{Hh}, I_{HC}, T_H, S_m, E_m, I_m > 0\}$

Then we have

$$\frac{d}{dt} (E_H e^{(\alpha_H + \mu_H)t}) = \left[\frac{b\beta_H S_H I_H}{N_H} + (1 - p_1) \delta_H I_{HC} + (1 - p_2) \omega I_{Hh} \right] e^{(\alpha_H + \mu_H)t} \quad (3.7)$$

Integral (4.7) from 0 to t_*

$$E_H(t_*) e^{(\alpha_H + \mu_H)t_*} - E_H(0) = \int_0^{t_*} f(\theta) e^{(\alpha_H + \mu_H)\theta} d\theta$$

$$E_H(t_*) e^{(\alpha_H + \mu_H)t_*} = E_H(0) + \int_0^{t_*} f(\theta) e^{(\alpha_H + \mu_H)\theta} d\theta$$

$$E_H(t_*) = E_H(0) e^{-(\alpha_H + \mu_H)t_*} + \left(e^{-(\alpha_H + \mu_H)t_*} \right) \int_0^{t_*} f(\theta) e^{(\alpha_H + \mu_H)\theta} d\theta \quad (3.8)$$

Hence $E_H(t) > 0$

Again for I_{Hh}

We assume that there exist a $t_* > 0$ such that $I_{Hh}(t_*) = 0$ and $I_{Hh}(t) = 0$

$$\Rightarrow \left[I_{Hh} e^{(\omega + \mu_H + \gamma_H)t} \right] = \alpha_H E_H e^{(\omega + \mu_H + \gamma_H)t} \quad (3.9)$$

Integrating (3.9) from 0 to t_*

$$I_{Hh}(t^*) e^{(\omega + \gamma_H + \mu_H)t^*} - I_{Hh}(0) = \int_0^{t^*} \alpha_H E_H(\theta) e^{(\omega + \gamma_H + \mu_H)\theta} d\theta$$

$$I_{Hh}(t^*) = I_{Hh}(0) e^{-(\omega + \gamma_H + \mu_H)t^*} + \left(e^{-(\omega + \gamma_H + \mu_H)t^*} \right) \int_0^{t^*} \alpha_H E_H(\theta) e^{(\omega + \gamma_H + \mu_H)\theta} d\theta \quad (3.10)$$

$$\Rightarrow I_{Hh} > 0$$

For I_{Hc} we have that

$$\frac{d}{dt} \left(I_{Hc} e^{(\delta_H + \mu_H)t} \right) = \gamma_H I_{Hh} e^{(\delta_H + \mu_H)t} \quad (3.11)$$

Integrating (3.11) from 0 to t_* we have

$$I_{Hc}(t) e^{(\delta_H + \mu_H)t^*} - I_{Hc}(0) = \int_0^{t^*} \gamma_H I_{Hh} e^{(\delta_H + \mu_H)\theta} d\theta$$

$$I_{Hc}(t) e^{(\delta_H + \mu_H)t^*} = I_{Hc}(0) e^{-(\delta_H + \mu_H)t^*} + \left(e^{(\delta_H + \mu_H)t^*} \right) \int_0^{t^*} \gamma_H I_{Hh} e^{(\delta_H + \mu_H)\theta} d\theta \quad (3.12)$$

Hence $I_{Hc}(t) > 0$

We also prove for T_H

$$\frac{dT_H}{dt} = p_1 \delta_H I_{Hc} + p_2 \omega I_{Hh} - \mu_H T_H \quad (3.13)$$

$$\frac{d}{dt} \left(T_H e^{\mu_H t} \right) = \left(p_1 \delta_H I_{Hc} + p_2 \omega I_{Hh} \right) e^{\mu_H t} \quad (3.14)$$

Integrating (3.14) from 0 to t_*

$$T_H(t) e^{(\mu_H)t^*} - T_H(0) = \int_0^{t^*} g(\theta) e^{(\mu_H)\theta} d\theta$$

$$T_H(t) = T_H(0) e^{-(\mu_H)t^*} + \left(e^{-(\mu_H)t^*} \right) \int_0^{t^*} g(\theta) e^{(\mu_H)\theta} d\theta \quad (3.15)$$

To prove the positivity of S_m , it follows the same approach as we used in the case of S_H

From the sixth equation of the model (3.1), it is obvious that $S_m(t) > 0$ for all $t \geq 0$; otherwise let there exist $t_* > 0$ such that $S_m(t_*) = 0$

$$S_m^c(t_*) \leq 0 \text{ and } S_H, E_H, I_{Hh}, I_{Hc}, T_H, S_m, E_m, I_m > 0$$

For $0 < t < t_*$

$$\Rightarrow S_m'(t_*) = \Lambda_m - \frac{b\beta_m S_m(t_*)(I_{Hh} + I_{Hc})}{N_m} - \mu_m S_m(t_*)$$

$$S_m'(t_*) = \Lambda_m > 0$$

Which is a contradiction of the assumption that $S_m^c(t_*) \leq 0$

Hence $S_m(t) > 0$

For the next

$$\frac{dE_m}{dt} = \frac{b\beta_m S_m(I_{Hh} + I_{Hc})}{N_m} - (\alpha_m + \mu_m) E_m \tag{3.16}$$

Let there exist a $t_* = \sup\{t > 0 : S_H, E_H, I_{Hh}, I_{Hc}, T_H, S_m, E_m, I_m > 0\}$

Then we have

$$\frac{d}{dt} \left(E_m e^{(\alpha_m + \mu_m)t} \right) = \left(\frac{b\beta_m S_m(I_{Hh} + I_{Hc})}{N_m} \right) e^{(\alpha_m + \mu_m)t} \tag{3.17}$$

Integrate (3.17) from 0 to t_*

$$E_m(t_*) e^{(\alpha_m + \mu_m)t_*} - E_m(0) = \int_0^{t_*} f(\theta) e^{(\alpha_m + \mu_m)\theta} d\theta$$

$$E_m(t_*) e^{(\alpha_m + \mu_m)t_*} = E(0) + \int_0^{t_*} f(\theta) e^{(\alpha_m + \mu_m)\theta} d\theta$$

$$E_m(t_*) = E(0) e^{-(\alpha_m + \mu_m)t_*} + e^{-(\alpha_m + \mu_m)t_*} \int_0^{t_*} f(\theta) e^{(\alpha_m + \mu_m)\theta} d\theta \tag{3.18}$$

Hence $E_m(t) > 0$

$$\frac{dI_m}{dt} = \alpha_m E_m - \mu_m I_m \tag{3.19}$$

$$\frac{d}{dt} \left(I_m e^{\mu_m t} \right) = \alpha_m E_m \tag{3.20}$$

Integrating (3.20) from 0 to t_*

$$I_m(t_*)e^{\mu_m t_*} - I_m(0) = \int_0^{t_*} g(\theta)e^{\mu_m \theta} d\theta$$

$$I_m(t_*)e^{\mu_m t_*} = I_m(0) + \int_0^{t_*} g(\theta)e^{\mu_m \theta} d\theta$$

$$I_m(t_*) = I_m(0)e^{-\mu_m t_*} + e^{-\mu_m t_*} \int_0^{t_*} g(\theta)e^{\mu_m \theta} d\theta$$

$$\therefore I_m(t) > 0 \tag{3.21}$$

This completes the proof that for all $t > 0$ the solution of the system (2.1) is non-negative. ■

3.3 Existence of Equilibrium Points for Non-Special Case

The points at which the differential equations of the system (2.1) are equal to zero are referred to as equilibrium points or steady-state solutions.

$$\frac{dS_H}{dt} = \frac{dE_H}{dt} = \frac{dI_{Hh}}{dt} = \frac{dI_{Hc}}{dt} = \frac{dT_H}{dt} = \frac{dS_m}{dt} = \frac{dE_m}{dt} = \frac{dI_m}{dt} = 0$$

This implies that

$$\left. \begin{aligned} \Lambda_H - \frac{b\beta_H S_H I_m}{N_H} - \mu_H S_H &= 0 \\ \frac{b\beta_H S_H I_m}{N_H} + (1-p_1)\delta_H I_{Hc} + (1-p_2)\omega I_{Hh} - (\alpha_H + \mu_H)E_H &= 0 \\ \alpha_H E_H - (\omega + \gamma_H + \mu_H)I_{Hh} &= 0 \\ \gamma_H I_{Hh} - (\delta_H + \mu_H)I_{Hc} &= 0 \\ p_1\delta_H I_{Hc} + p_2\omega I_{Hh} - \mu_H T_H &= 0 \\ \Lambda_m - \frac{b\beta_m S_m (I_{Hc} + I_{Hh})}{N_m} - \mu_m S_m &= 0 \\ \frac{b\beta_m S_m (I_{Hc} + I_{Hh})}{N_m} - (\alpha_m + \mu_m)E_m &= 0 \\ \alpha_m E_m - \mu_m I_m &= 0 \end{aligned} \right\} \tag{3.22}$$

Solving (3.22) we have

$$\left. \begin{aligned}
 S_H^* &= \frac{\Lambda_H N_H^*}{(b\beta_H I_m^* + N_H^* \mu_H)} \\
 E_H^* &= \frac{b\beta_H S_H^* I_m^* + N_H^* (1-p_1)\delta_H I_{Hc}^* + N_H^* (1-p_2)\omega I_{Hh}^*}{(\alpha_H + \mu_H) N_H^*} \\
 I_{Hh}^* &= \frac{\alpha_H E_H^*}{(\omega + \gamma_H + \mu_H)} \\
 I_{Hc}^* &= \frac{\gamma_H I_{Hh}^*}{(\delta_H + \mu_H)} \\
 T_H^* &= \frac{p_1 \delta_H I_{Hc}^* + p_2 \omega I_{Hh}^*}{\mu_H} \\
 S_m^* &= \frac{N_m \Lambda_m}{[b\beta_m (I_{Hc}^* + I_{Hh}^*)]} \\
 E_m^* &= \frac{b\beta_m S_m^* (I_{Hc}^* + I_{Hh}^*)}{N_m^* (\alpha_m + \mu_m)} \\
 I_m^* &= \frac{\alpha_m E_m^*}{\mu_m}
 \end{aligned} \right\} (3.23)$$

Therefore it is important to note that there is no trivial equilibrium points as long as the recruitment term Λ_H and Λ_m are not zero. This implies that $(S_H^*, E_H^*, I_{Hh}^*, I_{Hc}^*, T_H^*, S_m^*, E_m^*, I_m^*) \neq (0,0,0,0,0,0,0)$ and the population will not be extinct.

3.4 Stability of the Disease – Free Equilibrium point

At the disease free equilibrium point all the disease compartments are set to be zero, that is,

$$E_H = I_{Hh} = I_{Hc} = E_m = I_m = 0$$

Hence our model reduces to,

$$\Lambda_H - \mu_H S_H = 0 \Rightarrow S_H = \frac{\Lambda_H}{\mu_H}$$

$$T_H = 0 \text{ and } S_m = \frac{\Lambda_m}{\mu_m}$$

Therefore the disease-free equilibrium (DFE) of our model is given by

$$\mathcal{E}^0 = (S_H^0, E_H^0, I_{Hh}^0, I_{Hc}^0, T_H^0, S_m^0, E_m^0, I_m^0) = \left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0 \right)$$

3.5 Basic Reproduction number for the model (2.1)

The basic reproduction number R_0 is defined as the average number of secondary infection that can occur when one infected individual is introduced into an entirely susceptible human population.

The local stability of \mathcal{E}^0 will now be explored using the next generation matrix operator as developed by Vanden Driessche and Watmough (2002)

$$\text{Let } x = (E_H, I_{Hh}, I_{Hc}, E_m, I_m)^T$$

Then we can re-write our model for the disease components as

$$\frac{dX}{dt} = f(X) - v(X) \quad (3.24)$$

Where $f(X)$ is defined as the rate of appearance of new infections into the disease compartment and we have it as;

$$f(X) = \begin{bmatrix} \frac{b\beta_H S_H I_m}{N_H} \\ 0 \\ 0 \\ \frac{b\beta_m S_m (I_{Hc} + I_{Hh})}{N_m} \\ 0 \end{bmatrix}$$

And $v(X)$ is defined as the rate of transfer of individual in and out of the disease compartment, and we have it as;

$$\text{And } v(X) = \begin{pmatrix} (\mu_H + \alpha_H)E_H - (1-p_2)\omega I_{Hh} - (1-p_1)\delta_H I_{Hc} \\ (\mu_H + \alpha_H + \omega)I_{Hh} - \alpha_H E_H \\ (\mu_H + \delta_H)I_{Hc} - \gamma_H I_{Hh} \\ (\mu_m + \alpha_m)E_m \\ \mu_m I_m - \alpha_m E_m \end{pmatrix}$$

Then F which is the Jacobian of $f(X)$ evaluated at the DFE ε^0 becomes

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & b\beta_H \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & b\beta_m & b\beta_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

And V the Jacobian of $v(X)$ evaluated at the DFE ε^0 becomes

$$V = \begin{pmatrix} \mu_H + \alpha_H & -(1-p_2)\omega & -(1-p_1)\delta_H & 0 & 0 \\ -\alpha_H & (\mu_H + \gamma_H + \omega) & 0 & 0 & 0 \\ 0 & -\gamma_H & \mu_H + \delta_H & 0 & 0 \\ 0 & 0 & 0 & \mu_m + \alpha_m & 0 \\ 0 & 0 & 0 & \alpha_m & \mu_m \end{pmatrix}$$

Then

$$V^{-1} = \frac{1}{\psi} \begin{pmatrix} (\omega\gamma_H + \mu_H)(\delta_H + \mu_H) & -a_0 & a_1 & 0 & 0 \\ -\alpha_H(\delta_H + \mu_H) & (\alpha_H + \mu_H)(\delta_H + \mu_H) & -[(p_1 - 1)\mu_H\delta_H] & 0 & 0 \\ \alpha_m\mu_m(\alpha_H + \gamma_H\mu_m) & -\gamma_H(\alpha_H + \mu_H) & (1-p_2)\omega\alpha_H + (\alpha_H + \mu_H)(\omega + \mu_H) & 0 & 0 \\ 0 & 0 & 0 & a_2 + \frac{a_3}{\alpha_m + \mu_m} & 0 \\ 0 & 0 & 0 & \alpha_m a_2 + \frac{a_3}{\mu_m(\alpha_m + \mu_m)} & a_2 + \frac{a_3}{\mu_m} \end{pmatrix}$$

where, $\psi = (-1 + p_1)\alpha_H\gamma_H\delta_H + (\delta_H + \mu_H)[(-1 + p_2)\omega\alpha_H + (\delta_H + \mu_H)(\omega + \gamma_H + \mu_H)]$

$$a_0 = (1 - p_1)\gamma_H\delta_H + (-1 + p_2)\omega + (\delta_H + \mu_H)$$

$$a_1 = (1 - p_1)\delta_H + (\omega + \gamma_H + \mu_H)$$

$$a_2 = (1 - p_1)\alpha_H\gamma_H\delta_H$$

$$a_3 = (\delta_H + \mu_H)[(-1 + p_2)\alpha_H\omega + (\alpha_H + \mu_H)(\omega + \gamma_H + \mu_H)]$$

$R_0 = \rho(FV^{-1})$ where ρ is the spectral radius, is given by

$$R_0 = \frac{b\beta_H b\beta_m \alpha_H \alpha_m (\gamma_H + \delta_H + \mu_H)}{\sqrt{b\beta_H b\beta_m \alpha_H \alpha_m (\gamma_H + \delta_H + \mu_H) [\mu_H (\omega + \gamma_H + \mu_H) (\delta_H + \mu_H) + \alpha_H [\mu_H (p_2 \omega + \gamma_H + \mu_H) + \delta_H (p_2 \omega + p_1 \gamma_H + \mu_H)]]} \mu_m (\alpha_m + \mu_m)}$$

$$= \frac{b\beta_H \alpha_H (\gamma_H + \delta_H + \mu_H)}{\sqrt{b\beta_H \alpha_H (\gamma_H + \delta_H + \mu_H) [\mu_H (\omega + \gamma_H + \mu_H) (\delta_H + \mu_H) + \alpha_H [\mu_H (p_2 \omega + \gamma_H + \mu_H) + \delta_H (p_2 \omega + p_1 \gamma_H + \mu_H)]]}} \times \frac{b\beta_m \alpha_m}{\sqrt{b\beta_m \alpha_m \mu_m (\alpha_m + \mu_m)}}$$

$$\Rightarrow R_0 = R_H R_m$$

$$R_0 = R_0^N$$

$$R_H = R_H^N = \frac{b\beta_H \alpha_H (\gamma_H + \delta_H + \mu_H)}{\sqrt{b\beta_H \alpha_H (\gamma_H + \delta_H + \mu_H) [\mu_H (\omega + \gamma_H + \mu_H) (\delta_H + \mu_H) + \alpha_H [\mu_H (p_2 \omega + \gamma_H + \mu_H) + \delta_H (p_2 \omega + p_1 \gamma_H + \mu_H)]]}}$$

$$R_m = R_m^N = \frac{b\beta_m \alpha_m}{\sqrt{b\beta_m \alpha_m \mu_m (\alpha_m + \mu_m)}}$$

$$\Rightarrow R_0 = R_H R_m$$

where R_H^N (Reproductive number of Humans for non-special case) tells about the number of humans that one infectious mosquito infects during its period of infectiousness in an entirely susceptible human population and R_m^N (the reproductive number of mosquitoes for non-special case) gives us the information about the average number of mosquitoes that one infectious individual can infect during his/her period of infections in an entirely susceptible mosquito population.

3.6 Basic Reproduction number for the Special Case

Differential Equation for special case where we assumed there is no treatment that is $p_1 = p_2 = 1$

$$\left. \begin{aligned}
 \frac{dS_H}{dt} &= \Lambda_H - \frac{b\beta_H S_H I_m}{N_H} - \mu_H S_H \\
 \frac{dE_H}{dt} &= \frac{b\beta_H S_H I_m}{N_H} - k_1 E_H \\
 \frac{dI_{Hh}}{dt} &= \alpha_H E_H - k_2 I_{Hh} \\
 \frac{dI_{Hc}}{dt} &= \gamma_H I_{Hh} - k_3 I_{Hc} \\
 \frac{dT_H}{dt} &= \delta_{Hc} + \omega I_{Hh} - \mu_H T_H \\
 \frac{dS_m}{dt} &= \Lambda_m - \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} - \mu_m S_m \\
 \frac{dE_m}{dt} &= \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} - k_4 E_m \\
 \frac{dI_m}{dt} &= \alpha_m E_m - \mu_m I_m
 \end{aligned} \right\} \quad (3.25)$$

Where $k_1 = (\alpha_H + \mu_H)$, $k_2 = (\omega + \mu_H + \gamma_H)$, $k_3 = (\delta_H + \mu_H)$ and $k_4 = (\alpha_m + \mu_m)$

Hence, the equilibrium point for the special case is

$$\left. \begin{aligned}
 S_H^* &= \frac{\Lambda_H N_H^*}{(b\beta_H I_m^* + N_H^* \mu_H)} \\
 E_H^* &= \frac{b\beta_H S_H^* I_m^*}{(\alpha_H + \mu_H) N_H^*} \\
 I_{Hh}^* &= \frac{\alpha_H E_H^*}{(\omega + \gamma_H + \mu_H)} \\
 I_{Hc}^* &= \frac{\gamma_H I_{Hh}^*}{(\delta_H + \mu_H)} \\
 T_H^* &= \frac{\delta_H I_{Hc}^* + \omega I_{Hh}^*}{\mu_H} \\
 S_m^* &= \frac{N_m \Lambda_m}{[b\beta_m (I_{Hc}^* + I_{Hh}^*)]} \\
 E_m^* &= \frac{b\beta_m S_m^* (I_{Hc}^* + I_{Hh}^*)}{N_m^* (\alpha_m + \mu_m)} \\
 I_m^* &= \frac{\alpha_m E_m^*}{\mu_m}
 \end{aligned} \right\} \quad (3.26)$$

The threshold quantity R_0 , is called the reproductive number of the disease elephantiasis. It represent the expected average number of new infections produced directly or indirectly by a single infective when introduced into a completely susceptible population. When the basic reproductive number $R_0 < 1$, on average each infected individual infects fewer the one individual, and the disease dies out, if $R_0 > 1$, on average each infected individual, infect more than one other individual, so we would expect the disease to spread.

To obtain R_0 for our special case we now explored using the next generation matrix operator as developed by van den Driessche and Watmough (2002).

Let $X = (E_H, I_{Hh}, I_{Hc}, E_m, I_m)$, that is, the set of the entire disease compartment.

Our model can be written as

$$\frac{dX}{dt} = f(X) - v(X) \quad (3.27)$$

Where $f(X)$ is the rate of appearance of new infection into the disease compartments

It is defined by

$$f(X) = \begin{pmatrix} \frac{b\beta_H S_H I_m}{N_H} \\ 0 \\ 0 \\ \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} \\ 0 \end{pmatrix}$$

F is the Jacobian of $f(X)$ with respect to the disease compartments evaluated at the point ε^0

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & b\beta_H \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & b\beta_m & b\beta_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$v(X)$ is the rate of transfer of individual in and out of the disease compartment, it is defined by $v(X) = v_{(X)}^- - v_{(X)}^+$

Where $v_{(X)}^-$ is the rate of transfer of disease out of the disease compartments

$$v_{(X)}^- = \begin{pmatrix} (\alpha_H + \mu_H) E_H \\ (\omega_H + \gamma_H + \mu_H) I_{Hh} \\ (\delta_H + \mu_H) I_{Hc} \\ (\alpha_m + \mu_m) E_m \\ \mu_m I_m \end{pmatrix}$$

And $v_{(X)}^+$ is the rate of transfer of disease into the disease compartment by other means.

$$v_{(X)}^+ = \begin{pmatrix} 0 \\ \alpha_H E_H \\ \gamma_H I_{Hh} \\ 0 \\ a_m E_m \end{pmatrix}$$

$$v(X) = \begin{pmatrix} k_1 E_H \\ k_2 I_{Hh} - \alpha_H E_H \\ k_3 I_{Hc} - \gamma_H I_{Hh} \\ k_4 E_m \\ \mu_m I_m - \alpha_m E_m \end{pmatrix}$$

V is the jacobian of $v(X)$ with respect to the disease compartment at the point ε^0 .

$$V = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 \\ -\alpha_H & k_2 & 0 & 0 & 0 \\ 0 & -\gamma_H & k_3 & 0 & 0 \\ 0 & 0 & 0 & k_4 & 0 \\ 0 & 0 & 0 & -\alpha_m & \mu_m \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{k_1} & 0 & 0 & 0 & 0 \\ \frac{\alpha_H}{k_1 k_2 k_3} & \frac{1}{k_2} & 0 & 0 & 0 \\ \frac{\alpha_H \gamma_H}{k_1 k_2 k_3} & \frac{\gamma_H}{k_2 k_3} & \frac{1}{k_3} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{k_4} & 0 \\ 0 & 0 & 0 & \frac{\alpha_m}{\mu_m k_4} & \frac{1}{\mu_m} \end{pmatrix}$$

$$F \bullet V^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_H \alpha_m}{\mu_m k_4} & \frac{b\beta_H}{\mu_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{b\beta_m \alpha_m k_2}{k_1 k_2 k_3} & \frac{b\beta_m k_2}{k_2 k_3} & \frac{b\beta_m}{k_3} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$R_0 = \sqrt{\frac{b^2 \beta_H \alpha_H \beta_m \alpha_m (k_3 + \gamma_H)}{k_1 k_2 k_3 k_4 \mu_m}}$$

$$R_H = \frac{b\beta_H \alpha_H (k_3 + \gamma_H)}{k_1 k_2 k_3 k_4}, R_m = \frac{b\beta_m \alpha_m}{\mu_m}$$

$$R_0 = \sqrt{R_H R_m}$$

where R_0 = Re productive number for the special case

R_H = Re productive number of humans for the special case

R_m = Re productive number of mosquitoes for the special case

R_H describes the number of humans that one infectious mosquito infects over its expected infection period in a completely susceptible human population, and R_m is the number of mosquitoes infected by one infectious human during the period of infectiousness in a completely susceptible mosquitoes population.

Using the basic reproduction number (R_0) obtained for the model (4.31)

Theorem 3.3: The disease-free state, ε^0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ for both special and non special cases

Proof:

Linearizing (3.1) at $I_H^0 = R^0 = I_m^0 = 0$ we have,

$$J(E^0) = \begin{pmatrix} -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 & b\beta_H \\ 0 & -(\alpha_H + \mu_H) & (1-p_2)\omega & (1-p_1)\delta_H & 0 & 0 & 0 & b\beta_H \\ 0 & \alpha_H & -(\omega + \gamma_H + \mu_H) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_H & -(\delta_H + \mu_H) & 0 & 0 & 0 & 0 \\ 0 & 0 & p_2\omega & p_1\delta_H & -\mu_H & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\alpha_m + \mu_m) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -\mu_m \end{pmatrix}$$

$$|J(E^0) - I\lambda| = \begin{pmatrix} -\mu_H - \lambda & 0 & 0 & 0 & 0 & 0 & 0 & b\beta_H \\ 0 & -(\alpha_H + \mu_H) - \lambda & (1-p_2)\omega & (1-p_1)\delta_H & 0 & 0 & 0 & b\beta_H \\ 0 & \alpha_H & -(\omega + \gamma_H + \mu_H) - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_H & -(\delta_H + \mu_H) - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & p_2\omega & p_1\delta_H & -\mu_H - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_m - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\alpha_m + \mu_m) - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -\mu_m - \lambda \end{pmatrix}$$

$$|J - I\lambda| = 0$$

$$\Rightarrow (-\mu_H - \lambda_1)[-(\alpha_H + \mu_H) - \lambda_2][-\mu_H - \lambda_3][-\mu_m - \lambda_4][-(\alpha_m + \mu_m) - \lambda_5][-\mu_m - \lambda_6] \begin{bmatrix} -(\omega + \gamma_H + \mu_H) - \lambda & 0 \\ \gamma_H & -(\delta_H + \mu_H) - \lambda \end{bmatrix} = 0$$

$$(-\mu_H - \lambda_4)[-(\alpha_H + \mu_H) - \lambda_2][-\mu_H - \lambda_5][-\mu_m - \lambda_6][-(\alpha_m + \mu_m) - \lambda_7][-\mu_m - \lambda_8][-(\omega + \gamma_H + \mu_H) - \lambda_3][-(\delta_H + \mu_H) - \lambda_4] = 0$$

$$\Rightarrow \lambda_1 = -\mu_H, \lambda_2 = -(\alpha_H + \mu_H), \lambda_3 = -(\omega + \gamma_H + \mu_H), \lambda_4 = -(\delta_H + \mu_H), \lambda_5 = -\mu_H, \lambda_6 = -\mu_m, \lambda_7 = -(\alpha_m + \mu_m) \text{ and } \lambda_8 = -\mu_m$$

Since all the real parts of the seven eigen-values are negative, the DFE is locally asymptotically stable.

3.7 Global Asymptotic Stability for Disease-Free Equilibrium

Theorem 3.4: if $R_0 \leq 1$ then the disease-free equilibrium of the model (3.25) is globally asymptotically stable.

Proof: To establish the global stability of the disease-free equilibrium \mathcal{E}^0 , we construct the following linear Lyapunov function (L).

3.8 We consider the Linear Lyapunov Function below

$$L = C_1 E_H + C_2 I_{Hh} + C_3 I_{HC} + C_4 E_m + C_5 I_m \quad (3.28)$$

Where the constants are,

$$C_1 = \frac{b\beta_m \alpha_m \alpha_H (k_3 + \gamma_H)}{k_1 k_2 k_3 k_4 \mu_m}$$

$$C_2 = \frac{b\beta_m \alpha_m (k_3 + \gamma_H)}{k_1 k_2 k_3 k_4 \mu_m}$$

$$C_3 = \frac{b\beta_m \alpha_m}{k_3 k_4 \mu_m}$$

$$C_4 = \frac{R_0 \alpha_m}{k_4 \mu_m}$$

$$C_5 = \frac{R_0}{\mu_m}$$

Putting the values of C_1, C_2, C_3, C_4 and C_5 into (3.28) we have

$$L = \frac{b\beta_m \alpha_m \alpha_H (k_3 + \gamma_H)}{k_1 k_2 k_3 k_4 \mu_m} E_H + \frac{b\beta_m \alpha_m (k_3 + \gamma_H)}{k_1 k_2 k_3 k_4 \mu_m} I_{Hh} + \frac{b\beta_m \alpha_m}{k_3 k_4 \mu_m} I_{HC} + \frac{R_0 \alpha_m}{k_4 \mu_m} E_m + \frac{R_0}{\mu_m} I_m \quad (3.29)$$

Differentiating equation (4.35) and substituting the steady state values $\dot{E}_H, \dot{I}_{Hh}, \dot{I}_{HC}, \dot{E}_m$ and \dot{I}_m

$$\begin{aligned} \dot{\mathcal{E}} &= \frac{b\beta_m\alpha_m\alpha_H(k_3+\gamma_H)}{k_1k_2k_3k_4\mu_m} \left[\frac{b\beta_H S_H I_m}{N_H} - k_1 E_H \right] + \frac{b\beta_m\alpha_m(k_3+\gamma_H)}{k_2k_3k_4\mu_m} [\alpha_H E_H - k_2 I_{Hh}] \\ &+ \frac{b\beta_m\alpha_m}{k_3k_4\mu_m} [\gamma_H I_{Hh} - k_3 I_{HC}] + \frac{R_0\alpha_m}{k_4\mu_m} \left[\frac{b\beta_m S_m (I_{Hh} + I_{HC})}{N_H} - k_4 E_m \right] + \frac{R_0}{\mu_m} [\alpha_m E_m - \mu_m I_m] \\ \dot{\mathcal{E}} &= \frac{b^2\beta_m\beta_H\alpha_m\alpha_H(k_3+\gamma_H)S_H I_m}{k_1k_2k_3k_4\mu_m N_H} - \frac{b\beta_m\alpha_m\alpha_H(k_3+\gamma_H)E_H}{k_2k_3k_4\mu_m} + \frac{b\beta_m\alpha_m\alpha_H(k_3+\gamma_H)E_H}{k_2k_3k_4\mu_m} - \frac{b\beta_m\alpha_m(k_3+\gamma_H)I_{Hh}}{k_3k_4\mu_m} \\ &+ \frac{b\beta_m\alpha_m\gamma_H I_{Hh}}{k_3k_4\mu_m} - \frac{b\beta_m\alpha_m I_{HC}}{k_4\mu_m} - \frac{R_0\alpha_m b\beta_m S_m (I_{Hh} + I_{HC})}{k_4\mu_m N_H} - \frac{R_0\alpha_m E_m}{\mu_m} + \frac{R_0\alpha_m E_m}{\mu_m} - R_0 I_m \\ &= \frac{b^2\beta_m\beta_H\alpha_m\alpha_H(k_3+\gamma_H)S_H I_m}{k_1k_2k_3k_4\mu_m N_H} - \frac{b\beta_m\alpha_m I_{Hh}}{k_4\mu_m} + \frac{b\beta_m\alpha_m\gamma_H I_{Hh}}{k_3k_4\mu_m} - \frac{b\beta_m\alpha_m I_{HC}}{k_4\mu_m} - \frac{R_0\alpha_m b\beta_m S_m (I_{Hh} + I_{HC})}{k_4\mu_m N_H} - R_0 I_m - \frac{b\beta_m\alpha_m\gamma_H I_{Hh}}{k_3k_4\mu_m} \end{aligned}$$

Re-arranging we obtain;

$$\begin{aligned} &\frac{b^2\beta_m\beta_H\alpha_m\alpha_H(k_3+\gamma_H)S_H I_m}{k_1k_2k_3k_4\mu_m N_H} - R_0 I_m - \frac{\alpha_m b\beta_m I_{Hh}}{k_4\mu_m} - \frac{b\beta_m\alpha_m\gamma_H I_{Hh}}{k_3k_4\mu_m} + \frac{b\beta_m\alpha_m\gamma_H I_{Hh}}{k_3k_4\mu_m} - \frac{b\beta_m\alpha_m I_{HC}}{k_4\mu_m} \\ &+ \frac{R_0\alpha_m b\beta_m S_m I_{Hh}}{k_4\mu_m N_H} + \frac{R_0\alpha_m b\beta_m S_m I_{HC}}{k_4\mu_m N_H} \end{aligned}$$

Collecting like terms

$$\begin{aligned} &(R_0^2 - R_0)I_m + \frac{\alpha_m b\beta_m}{k_4\mu_m} (R_0 - 1)I_{Hh} + \frac{\alpha_m b\beta_m}{k_4\mu_m} (R_0 - 1)I_{HC} \\ \dot{\mathcal{L}} &\leq R_0 (R_0 - 1)I_m + \frac{\alpha_m b\beta_m}{k_4\mu_m} (R_0 - 1)I_{Hh} + \frac{\alpha_m b\beta_m}{k_4\mu_m} (R_0 - 1)I_{HC} \\ \dot{\mathcal{L}} &\leq R_0 (R_0 - 1)I_m + R_m (R_0 - 1)I_{Hh} + R_m (R_0 - 1)I_{HC} \end{aligned}$$

$\dot{\mathcal{E}} \leq 0$ for $R_0 \leq 1$, and for $\dot{\mathcal{E}} = 0$, if and only if $I_m = I_{Hh} = I_{HC} = 0$ by Lasalle's invariance principle, \mathcal{E}^0 is globally asymptotically stable, ■

3.9 Global Stability of Endemic Equilibrium Point.

Theorem 3.5: The endemic equilibrium of the model (3.25) is globally asymptotically stable whenever $R_0 > 1$
 $p_1 = p_2 = 1$

Proof: Let the endemic equilibrium of the model (3.25) be denoted by

$$M = (S_H^{**}, E_H^{**}, I_{Hh}^{**}, I_{HC}^{**}, T_H^{**}, S_m^{**}, E_m^{**}, I_m^{**})$$

and Let $R_0 > 1$ so that M exists.

Consider the following nonlinear Lyapunov function.

$$\begin{aligned}
 M = & C_1 \left[S_H - S_H^{**} - S_H^{**} \ln \left(\frac{S_H}{S_H^{**}} \right) + E_H - E_H^{**} - E_H^{**} \ln \left(\frac{E_H}{E_H^{**}} \right) \right] + C_2 \left[I_{Hh} - I_{Hh}^{**} - I_{Hh}^{**} \ln \left(\frac{I_{Hh}}{I_{Hh}^{**}} \right) \right] \\
 & + C_3 \left[I_{Hc} - I_{Hc}^{**} - I_{Hc}^{**} \ln \left(\frac{I_{Hc}}{I_{Hc}^{**}} \right) \right] + C_4 \left[S_m - S_m^{**} - S_m^{**} \ln \left(\frac{S_m}{S_m^{**}} \right) + E_m - E_m^{**} - E_m^{**} \ln \left(\frac{E_m}{E_m^{**}} \right) \right] + C_5 \left[I_m - I_m^{**} - I_m^{**} \ln \left(\frac{I_m}{I_m^{**}} \right) \right] \quad (3.30)
 \end{aligned}$$

Differentiating the nonlinear lyapounov function

$$\begin{aligned}
 \dot{M} &= C_1 \left[\dot{S}_H - \frac{S_H^{**}}{S_H} \dot{S}_H + \dot{E}_H - \frac{E_H^{**}}{E_H} \dot{E}_H \right] + C_2 \left[\dot{I}_{Hh} - \frac{I_{Hh}^{**}}{I_{Hh}} \dot{I}_{Hh} \right] + C_3 \left[\dot{I}_{Hc} - \frac{I_{Hc}^{**}}{I_{Hc}} \dot{I}_{Hc} \right] + C_4 \left[\dot{S}_m - \frac{S_m^{**}}{S_m} \dot{S}_m + \dot{E}_m - \frac{E_m^{**}}{E_m} \dot{E}_m \right] \\
 &+ C_5 \left[\dot{I}_m - \frac{I_m^{**}}{I_m} \dot{I}_m \right] \\
 \dot{M} &= C_1 \left[\left(1 - \frac{S_H^{**}}{S_H} \right) \dot{S}_H + \left(1 - \frac{E_H^{**}}{E_H} \right) \dot{E}_H \right] + C_2 \left[\left(1 - \frac{I_{Hh}^{**}}{I_{Hh}} \right) \dot{I}_{Hh} \right] + C_3 \left[\left(1 - \frac{I_{Hc}^{**}}{I_{Hc}} \right) \dot{I}_{Hc} \right] + C_4 \left[\left(1 - \frac{S_m^{**}}{S_m} \right) \dot{S}_m + \left(1 - \frac{E_m^{**}}{E_m} \right) \dot{E}_m \right] \\
 &+ C_5 \left[\left(1 - \frac{I_m^{**}}{I_m} \right) \dot{I}_m \right] \quad (3.31)
 \end{aligned}$$

Substituting model (3.25) into (3.31)

$$\begin{aligned}
 = & C_1 \left[\left(1 - \frac{S_H^{**}}{S_H} \right) \left(\Lambda_H - \frac{b\beta_H S_H I_m}{N_H} - \mu_H S_H \right) + \left(1 - \frac{E_H^{**}}{E_H} \right) \left(\frac{b\beta_H S_H I_m}{N_H} - k_1 E_H \right) \right] + C_2 \left[\left(1 - \frac{I_{Hh}^{**}}{I_{Hh}} \right) (\alpha_H E_H - k_2 I_{Hh}) \right] \\
 & + C_3 \left[\left(1 - \frac{I_{Hc}^{**}}{I_{Hc}} \right) (\gamma_H I_{Hh} - k_3 I_{Hc}) \right] + C_4 \left[\left(1 - \frac{S_m^{**}}{S_m} \right) \left(\Lambda_m - \frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} - \mu_m S_m \right) \right] \quad (3.32) \\
 & + \left(1 - \frac{E_m^{**}}{E_m} \right) \left(\frac{b\beta_m S_m (I_{Hh} + I_{Hc})}{N_m} - k_4 E_m \right) + C_5 \left[\left(1 - \frac{I_m^{**}}{I_m} \right) (\alpha_m E_m - \mu_m I_m) \right]
 \end{aligned}$$

At steady state.

$$\begin{aligned}
 \Lambda_H &= \frac{b\beta_H S_H^{**} I_m}{N_H} - \mu_H S_H^{**} \\
 \frac{b\beta_H S_H^{**} I_m}{N_H} &= k_1 E_H^{**} \Rightarrow k_1 = \frac{b\beta_H S_H^{**} I_m}{N_H E_H^{**}} \\
 \alpha_H E_H^{**} &= k_2 I_{Hh}^{**} \Rightarrow k_2 = \frac{\alpha_H E_H^{**}}{I_{Hh}^{**}} \\
 \gamma_H I_{Hh}^{**} &= k_3 I_{Hc}^{**} \Rightarrow k_3 = \frac{\gamma_H I_{Hh}^{**}}{I_{Hc}^{**}} \\
 \Lambda_m &= \frac{b\beta_m S_m^{**} (I_{Hh}^{**} + I_{Hc}^{**})}{N_m} + \mu_m S_m^{**}
 \end{aligned}$$

Substituting the values of steady state into (3.38)

$$\begin{aligned}
 \mathcal{M}^* = & C_1 \left[\left(1 - \frac{S_H^{**}}{S_H} \right) \left(\frac{b\beta_H S_H^{**} I_m}{N_H} + \mu_H S_H^{**} - \frac{b\beta_H S_H I_m}{N_H} + \mu_H S_H \right) + \left(1 - \frac{E_H^{**}}{E_H} \right) \left(\frac{b\beta_H S_H I_m}{N_H} - k_1 E_H \right) \right] \\
 & + C_2 \left[\left(1 - \frac{I_{Hh}^{**}}{I_{Hh}} \right) (\alpha_H E_H - k_2 I_{Hh}) \right] + C_3 \left[\left(1 - \frac{I_{HC}^{**}}{I_{HC}} \right) (\gamma_H I_{Hh} - k_3 I_{HC}) \right] \\
 & + C_4 \left[\left(1 - \frac{S_m^{**}}{S_m} \right) \left(\frac{b\beta_m S_m^{**} (I_{Hh}^{**} + I_{HC}^{**})}{N_m} + \mu_m S_m^{**} - \frac{b\beta_m S_m (I_{Hh} + I_{HC})}{N_m} - \mu_m S_m \right) \right] \\
 & + \left(1 - \frac{E_m^{**}}{E_m} \right) \left(\frac{b\beta_m S_m (I_{Hh} + I_{HC})}{N_m} - k_4 E_m \right) + C_5 \left[\left(1 - \frac{I_m^{**}}{I_m} \right) (\alpha_m E_m - \mu_m I_m) \right]
 \end{aligned} \tag{3.33}$$

$$C_1 = \frac{k_3 k_4 N_H N_m \mu_m}{b^2 \beta_H \beta_m S_H^{**} S_m^{**} \alpha_m (K_3 + \gamma_H)}$$

$$C_2 = \frac{k_1 k_3 k_4 N_H N_m \mu_m}{b^2 \beta_H \beta_m S_H^{**} S_m^{**} \alpha_H \alpha_m (k_3 + \gamma_H)}$$

$$C_3 = \frac{b^2 \beta_H \beta_m S_H^{**} S_m^{**} \alpha_H \alpha_m}{N_H N_m \mu_m k_1 k_2 k_3 k_4}$$

$$C_4 = \frac{b\beta_H S_H^{**} \alpha_H \alpha_m}{N_H \mu_m k_1 k_2 k_4}$$

$$C_5 = \frac{k_3 k_4 N_m}{b\beta_m S_m^{**} \alpha_m (K_3 + \gamma_H)}$$

Substituting the values of the lyapunov constants C's into equation (3.33)

$$\begin{aligned}
 \mathcal{M}^* = & \frac{k_3 k_4 N_H N_m \mu_m}{b^2 \beta_H \beta_m S_H^{**} S_m^{**} \alpha_m (k_3 + \gamma_H)} \left[\left(1 - \frac{S_H^{**}}{S_H} \right) \left(\frac{b\beta_H S_H^{**} I_m}{N_H} + \mu_H S_H^{**} - \frac{b\beta_H S_H I_m}{N_H} + \mu_H S_H \right) + \left(1 - \frac{E_H^{**}}{E_H} \right) \left(\frac{b\beta_H S_H I_m}{N_H} - k_1 E_H \right) \right] \\
 & + \frac{k_1 k_3 k_4 N_H N_m \mu_m}{b^2 \beta_H \beta_m S_H^{**} S_m^{**} \alpha_H \alpha_m (k_3 + \gamma_H)} \left[\left(1 - \frac{I_{Hh}^{**}}{I_{Hh}} \right) (\alpha_H E_H - k_2 I_{Hh}) \right] + \frac{b^2 \beta_H \beta_m S_H^{**} S_m^{**} \alpha_H \alpha_m}{N_H N_m \mu_m k_1 k_2 k_3 k_4} \left[\left(1 - \frac{I_{HC}^{**}}{I_{HC}} \right) (\gamma_H I_{Hh} - k_3 I_{HC}) \right] \\
 & + \frac{b\beta_H S_H^{**} \alpha_H \alpha_m}{N_H \mu_m k_1 k_2 k_4} \left[\left(1 - \frac{S_m^{**}}{S_m} \right) \left(\frac{b\beta_m S_m^{**} (I_{Hh}^{**} + I_{HC}^{**})}{N_m} + \mu_m S_m^{**} - \frac{b\beta_m S_m (I_{Hh} + I_{HC})}{N_m} - \mu_m S_m \right) \right] \\
 & + \left(1 - \frac{E_m^{**}}{E_m} \right) \left(\frac{b\beta_m S_m (I_{Hh} + I_{HC})}{N_m} - k_4 E_m \right) + \frac{k_3 k_4 N_m}{b\beta_m S_m^{**} \alpha_m (k_3 + \gamma_H)} \left[\left(1 - \frac{I_m^{**}}{I_m} \right) (\alpha_m E_m - \mu_m I_m) \right]
 \end{aligned}$$

Collecting those terms with dot stars in the effected classes and substituting the values of k's inside the bracket we have

$$\begin{aligned}
 M_{\&}^* = & \frac{k_3 k_4 N_H N_m \mu_m}{b^2 S_H^{**} S_m^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{b \beta_H S_H^{**} I_m^{**}}{N_H} - \frac{b \beta_H (S_H^{**})^2 I_m^{**}}{N_H} - \frac{b \beta_H S_H I_m E_H^{**}}{N_H} \right] + \frac{k_3 k_4 N_H N_m \mu_m}{b^2 S_H^{**} S_m^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{\mu_H S_H^{**} + \mu_H S_H^{**}}{S_H} - \frac{\mu_H (S_H^{**})^2}{S_H} - \mu_H S_H \right] \\
 & + \frac{k_1 k_3 k_4 N_H N_m \mu_m}{b^2 S_H^{**} S_m^{**} \beta_H \beta_m \alpha_H \alpha_m (k_3 + \gamma_H)} \left[\alpha_H E_H^{**} - \frac{I_{Hh}^{**} \alpha_H E_H^{**}}{I_{Hh}} \right] + \frac{b^2 S_H^{**} S_m^{**} \beta_H \beta_m \alpha_H \alpha_m}{k_1 k_2 k_3 k_4 N_H N_m \mu_m} \left[\gamma_H I_{Hh}^{**} - \frac{\gamma_H I_{Hh}^{**} I_{Hc}^{**}}{I_{Hc}} \right] \\
 & + \frac{b S_H^{**} \beta_H \alpha_H \alpha_m}{k_1 k_2 k_3 N_H \mu_m} \left[\frac{b \beta_m S_m^{**} (I_{Hh}^{**} + I_{Hc}^{**})}{N_m} - \frac{b \beta_m (S_m^{**})^2 (I_{Hh}^{**} + I_{Hc}^{**})}{N_m S_m} - \frac{b \beta_m S_m (I_{Hh}^{**} + I_{Hc}^{**}) E_m^{**}}{N_m E_m} + \frac{b \beta_m S_m^{**} (I_{Hh}^{**} + I_{Hc}^{**})}{N_m} \right] \\
 & + \frac{b S_H^{**} \beta_H \alpha_H \alpha_m}{k_1 k_2 k_4 N_H \mu_m} \left[\frac{\mu_m S_m^{**} + \mu_m S_m^{**} - \frac{\mu_m (S_m^{**})^2}{S_m}}{-\mu_m S_m} \right] + \frac{k_3 k_4 N_m}{b S_m^{**} \beta_m \alpha_m (k_3 + \gamma_H)} \left[\mu_m I_m^{**} - \frac{\alpha_m E_m I_m^{**}}{I_m} \right]
 \end{aligned}$$

Factorizing further

$$\begin{aligned}
 M_{\&}^* = & \frac{k_3 k_4 N_m \mu_m I_m^{**}}{b \beta_m S_m^{**} \alpha_m (k_3 + \gamma_H)} \left[2 - \frac{S_H^{**}}{S_H} - \frac{I_m E_H^{**} S_H}{S_H^{**} E_H I_m^{**}} \right] + \frac{k_3 k_4 N_H N_m \mu_m \mu_H}{b^2 S_m^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[2 - \frac{S_H^{**}}{S_H} - \frac{S_H}{S_H^{**}} \right] \\
 & + \frac{k_1 k_3 k_4 N_H N_m \mu_m E_H^{**}}{b^2 S_H^{**} S_m^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[1 - \frac{I_{Hh}^{**} E_H^{**}}{I_{Hh} E_H^{**}} \right] + \frac{b^2 S_H^{**} S_m^{**} \beta_H \beta_m \alpha_H \alpha_m \gamma_H I_{Hh}^{**}}{k_1 k_2 k_3 k_4 N_H N_m \mu_m} \left[1 - \frac{I_{Hh}^{**} I_{Hc}^{**}}{I_{Hc} I_{Hh}^{**}} \right] \\
 & + \frac{b^2 \beta_H S_H^{**} \alpha_H \beta_m S_m^{**} \alpha_m (I_{Hh}^{**} + I_{Hc}^{**})}{k_1 k_2 k_4 N_H \mu_m N_m} \left[2 - \frac{S_m^{**}}{S_m} - \frac{S_m E_m^{**}}{E_m S_m^{**}} \right] + \frac{b S_H^{**} \beta_H \alpha_H S_m^{**} \alpha_m}{k_1 k_2 k_4 N_H} \left[2 - \frac{S_m^{**}}{S_m} - \frac{S_m}{S_m^{**}} \right] \\
 & + \frac{k_3 k_4 N_m I_m^{**} \mu_m}{b S_m^{**} \beta_m \alpha_m (k_3 + \gamma_H)} \left[1 - \frac{\alpha_m E_m}{\mu_m I_m} \right]
 \end{aligned}$$

Substituting the values of $\mu_m = \frac{\alpha_m E_m^{**}}{I_m^{**}}$ and collecting the positive and negative terms, we have the following

$$M_{\&}^* = \left\{ \frac{2k_3 k_4 N_H N_m \mu_m I_m^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} + \frac{2k_3 k_4 N_H N_m \mu_m}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} + \frac{k_1 k_3 k_4 N_H N_m \mu_m E_H^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} + \frac{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m \gamma_H I_{Hh}^{**}}{k_1 k_2 k_3 k_4 N_H N_m \mu_m} + \frac{2b^2 S_H^{**} \beta_H \beta_m \alpha_H \alpha_m S_m^{**} (I_{Hh}^{**} + I_{Hc}^{**})}{k_1 k_2 k_4 N_H N_m \mu_m} + \frac{2b S_H^{**} \beta_H \alpha_H \alpha_m S_m^{**}}{k_1 k_2 k_4 N_H} + \frac{k_3 k_4 N_m \mu_m I_m^{**}}{b S_m^{**} \beta_m \alpha_m (k_3 + \gamma_H)} \right\}$$

$$\left[\begin{aligned} & \frac{k_3 k_4 N_H N_m \mu_m I_m^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{S_H^{**}}{S_H} + \frac{I_H E_H^{**}}{S_H^{**} E_H I_m^{**}} \right] + \frac{k_3 k_4 N_H N_m \mu_m}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{S_H^{**}}{S_H} + \frac{S_H}{S_H^{**}} \right] + \\ & \frac{k_1 k_3 k_4 N_H N_m \mu_m E_H^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{I_{Hh}^{**} E_H}{I_{Hh}} \right] + \frac{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m \gamma_H I_{Hh}^{**}}{k_1 k_2 k_3 k_4 N_H N_m \mu_m} \left[\frac{I_{Hh} I_{Hc}^{**}}{I_{Hc} I_{Hh}^{**}} \right] + \\ & \frac{b^2 S_m^{**} \beta_H \beta_m \alpha_H \alpha_m S_m^{**} (I_{Hh}^{**} + I_{Hc}^{**})}{k_1 k_2 k_4 N_H N_m \mu_m} \left[\frac{S_m^{**}}{S_m} + \frac{S_m E_m^{**}}{E_m S_m^{**}} \right] + \frac{b S_H^{**} \beta_H \alpha_H \alpha_m S_m^{**}}{k_1 k_2 k_4 N_H} \left[\frac{S_m^{**}}{S_m} + \frac{S_m}{S_m^{**}} \right] + \\ & \frac{k_3 k_4 N_m \mu_m I_M^{**}}{b S_m^{**} \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{I_m^{**} E_m}{E_m^{**} I_m} \right] \end{aligned} \right]$$

$\Rightarrow \dot{M}^* = A - B$ where

$$A = \frac{2k_3 k_4 N_H N_m \mu_m I_m^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} + \frac{2k_3 k_4 N_H N_m \mu_m}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} + \frac{k_1 k_3 k_4 N_H N_m \mu_m E_H^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} + \frac{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m \gamma_H I_{Hh}^{**}}{k_1 k_2 k_3 k_4 N_H N_m \mu_m} + \frac{2b^2 S_m^{**} \beta_H \beta_m \alpha_H \alpha_m S_m^{**} (I_{Hh}^{**} + I_{Hc}^{**})}{k_1 k_2 k_4 N_H N_m \mu_m} + \frac{2b S_H^{**} \beta_H \alpha_H \alpha_m S_m^{**}}{k_1 k_2 k_4 N_H} + \frac{k_3 k_4 N_m \mu_m I_M^{**}}{b S_m^{**} \beta_m \alpha_m (k_3 + \gamma_H)} \tag{3.34}$$

$$B = \frac{k_3 k_4 N_H N_m \mu_m I_m}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{S_H^{**}}{S_H} + \frac{S_H I_m E_H^{**}}{S_H^{**} E_H I_m^{**}} \right] + \frac{k_3 k_4 N_H N_m \mu_m}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{S_H^{**}}{S_H} + \frac{S_H}{S_H^{**}} \right] + \frac{k_1 k_3 k_4 N_H N_m \mu_m E_m^{**}}{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m (k_3 + \gamma_H)} \left[\frac{I_H^{**} E_H}{I_H E_H^{**}} \right] + \frac{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_m \gamma_H I_H^{**}}{k_1 k_2 k_3 k_4 N_H N_m \mu_m} \left[\frac{I_{Hh} I_{Hc}^{**}}{I_{Hc} I_{Hh}^{**}} \right] + \frac{b^2 S_m^{**} S_H^{**} \beta_H \beta_m \alpha_H \alpha_m (I_{Hh}^{**} + I_{Hc}^{**})}{k_1 k_2 k_4 N_H N_m \mu_m} \left[\frac{S_m^{**}}{S_m} + \frac{S_m E_m^{**}}{E_m S_m^{**}} \right] + \frac{b S_H^{**} \beta_H \alpha_H \alpha_m S_m^{**}}{k_1 k_2 k_4 N_H} \left[\frac{S_m^{**}}{S_m} + \frac{S_m}{S_m^{**}} \right] + \frac{k_3 k_4 N_m \mu_m I_H^{**}}{b S_m^{**} \beta_m \alpha_m (k_1 + \gamma_H)} \left[\frac{I_m^{**} E_m}{E_m^{**} I_m} \right] \tag{3.35}$$

From (3.34) and (3.35), we observe that $A < B$, then, $\dot{M}^* = \frac{dM}{dt} < 0$ (which means that M^* will be negative definite). $\dot{M}^* = \frac{dM}{dt} = 0$ if and only if $S_H = S_H^{**}, E_H = E_H^{**}, I_{Hh} = I_{Hh}^{**}, I_{Hc} = I_{Hc}^{**}, T_H = T_H^{**}, S_m = S_m^{**}, E_m = E_m^{**}$ and $I_m = I_m^{**}$

Thus the largest compact invariant set, $\left\{ (S_H^{**}, E_H^{**}, I_{Hh}^{**}, I_{Hc}^{**}, T_H^{**}, S_m^{**}, E_m^{**}, I_m^{**}) \in \Delta_1 : \frac{dM}{dt} = 0 \right\}$ is just the endemic equilibrium point ξ^* . Then by Lassalle invariant principle, if $A < B$, then ξ^* will be globally asymptotically stable in Δ_1 , ■

3.10 Numerical simulation

For numerical results, we consider the following values for the parameters involved in the model.

VARIABLES & PARAMETERS	VALUE	UNIT	SOURCE
S_H	30	----	Assumed
E_H	25	-----	Assumed
I_{Hh}	15	-----	Assumed
I_{Hc}	10	-----	Assumed
T_H	0	-----	Assumed
S_m	20	-----	Assumed
E_m	15	-----	Assumed
I_m	10	-----	Assumed
Λ_H	24	Per day	Ridouan etaal (2013)
Λ_m	4000	Per day	Ridouan etaal (2013)
β_H	3.576	Per day	Ridouan etaal (2013)
β_m	0.091	Per day	Labadin etaal (2009)
α_H	0.0045	Per year	Ridouan etaal (2013)
α_m	0.055	Per day	Ridouan etaal (2013)
μ_H	0.00048	Per day	Ridouan etaal (2013)
μ_m	0.067	Per day	Ridouan etaal (2013)
γ_H	0.000287	Per year	Ridouan etaal (2013)
δ_H	0.00196	Per day	Olaniyi and Obabiyi(2013)
ω	0.00196	Per day	Olaniyi and Obabiyi(2013)
p_1	0.005	Per day	Assumed
p_2	0.036	Per day	Assumed
B	0.12	Per day	Olaniyi and Obabiyi(2013)

For high accuracy of results, we used MATLAB 15 to carry out our simulations.

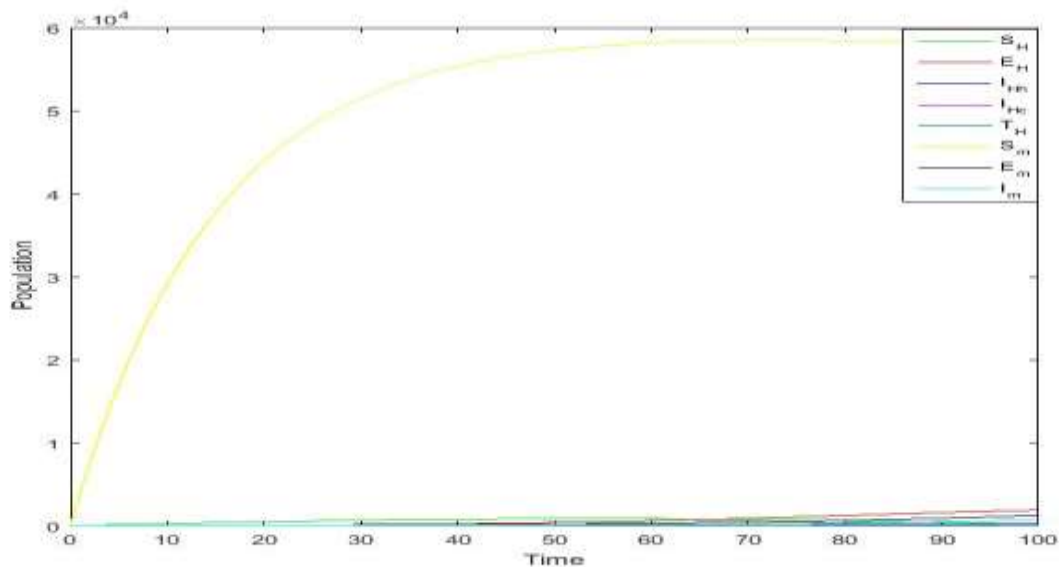


Fig.3.1: A plot of the eight compartments when $R_0 < 1$.

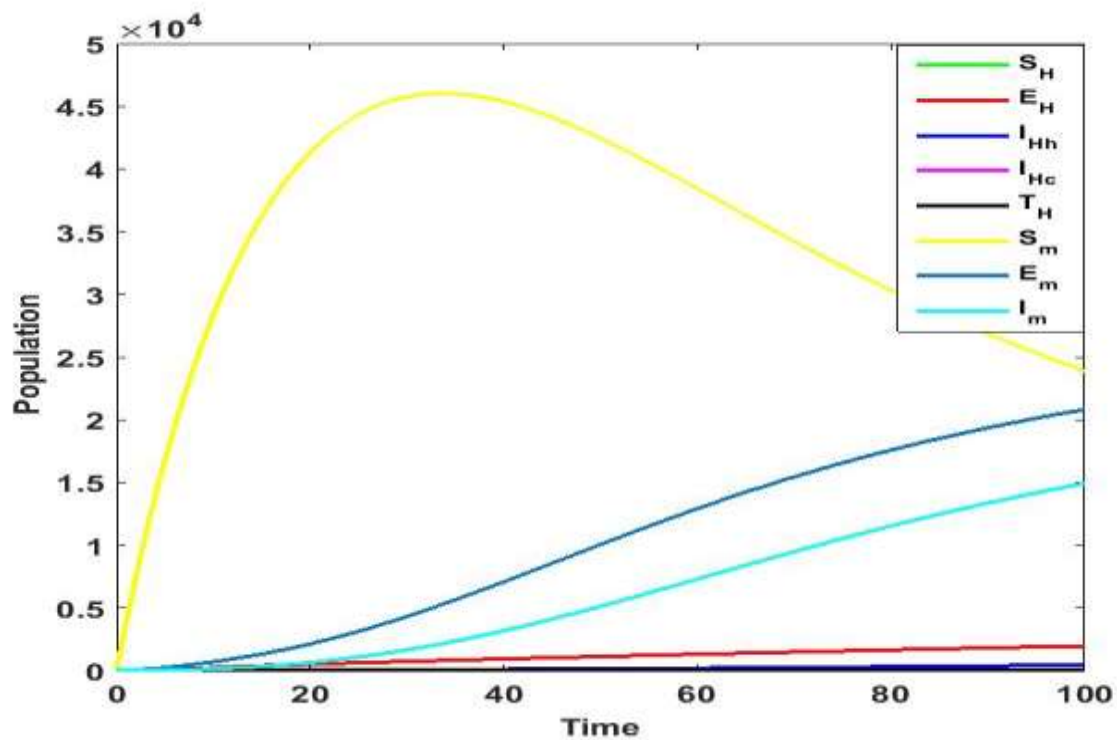


Fig.3.2: A plot of the eight compartments when $R_0 > 1$

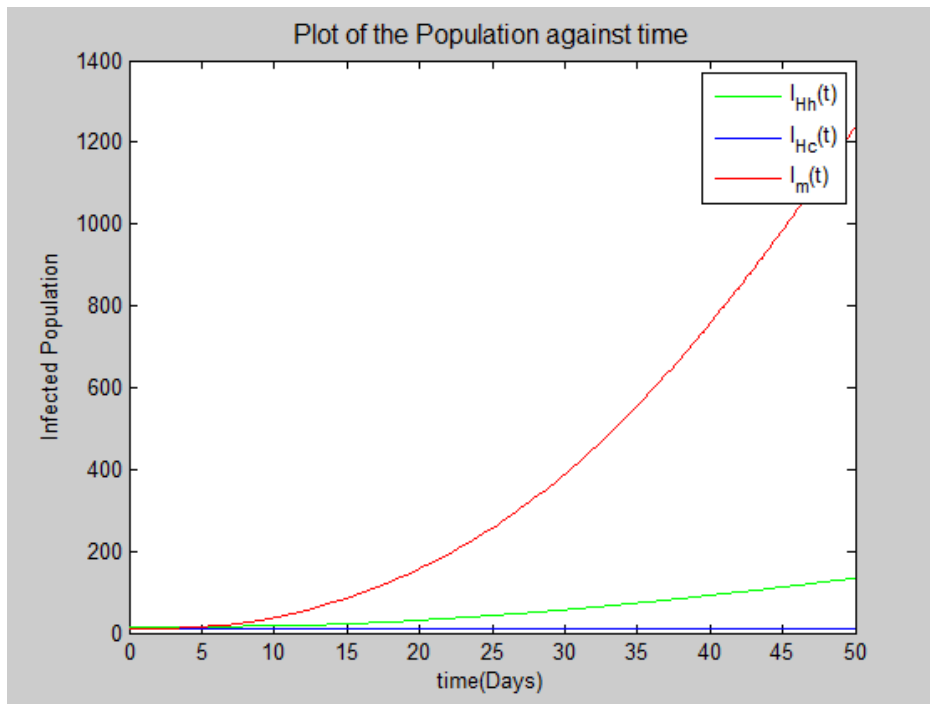


Fig. 3.3: Plot of the infected class (I_{Hh}, I_{Hc} and I_m) against time when $\delta_H = 0.002$

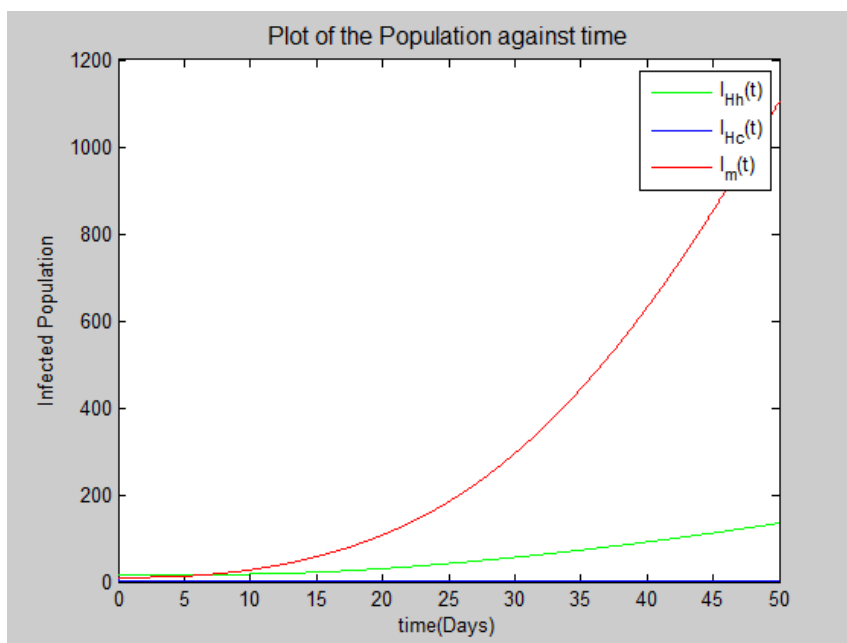


Fig. 3.4: Plot of the infected class (I_{Hh}, I_{Hc} and I_m) against time when $\delta_H = 30$

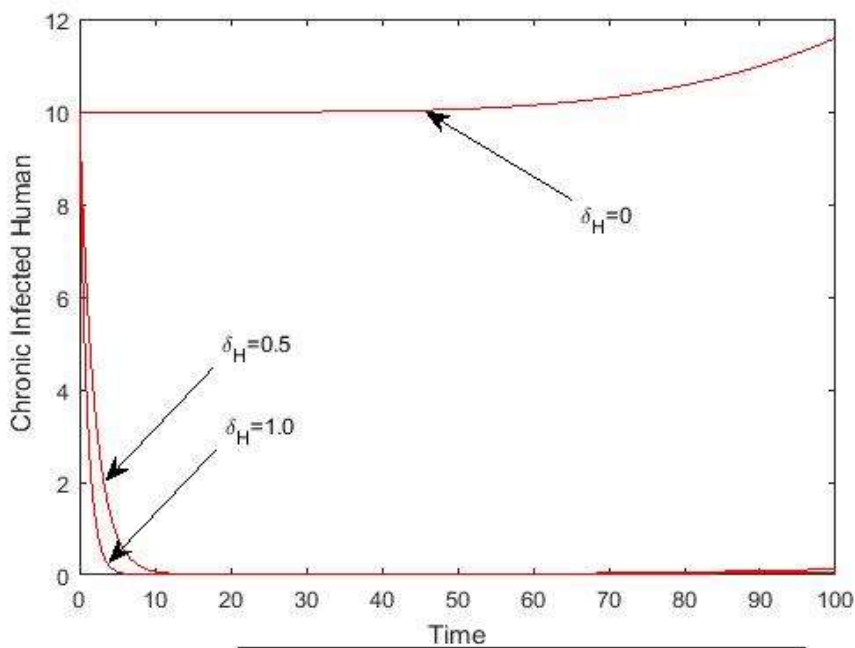


Fig.5; shows the trend of the chronic infected human when we vary δ

Fig. 3.5: Shows the trend of the chronic infected humans when we vary δ_H

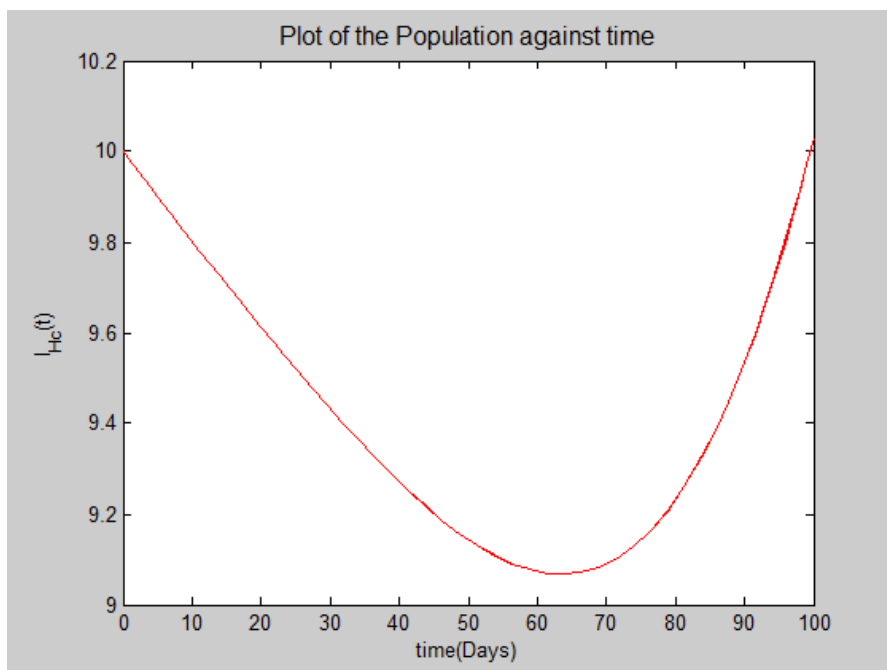


Fig.3.6: Plot of I_{Hc} against time when we vary the value of p_1 .

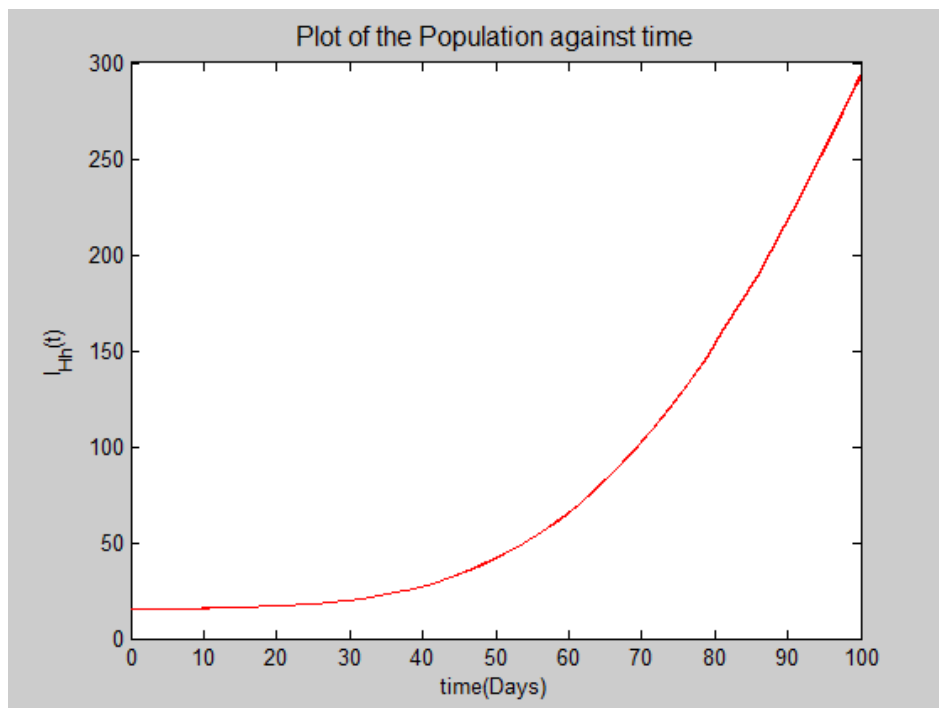


Fig. 3.7: Plot of I_{Hh} against time when we vary the value of p_2 .

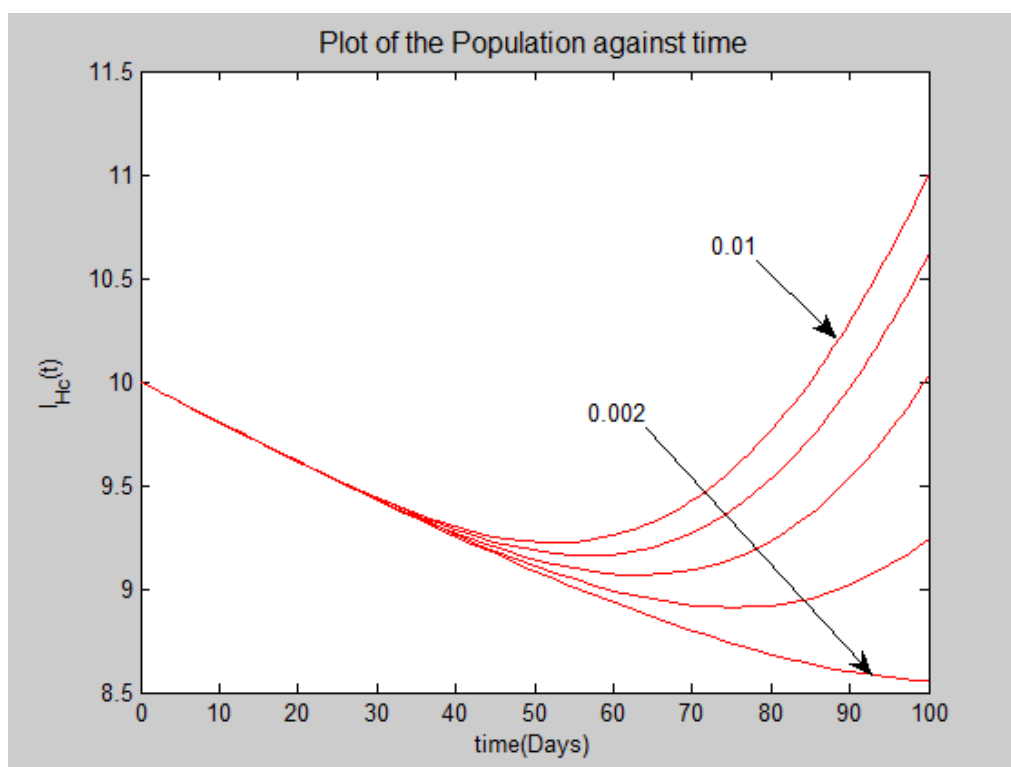


Fig. 3.8: Plot of I_{Hc} at different biting rate (b)

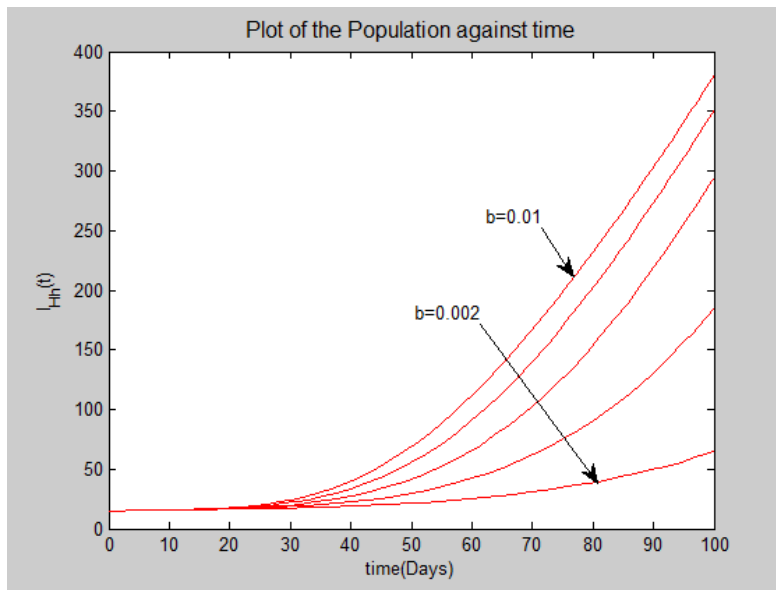


Fig. 3.9: Plot of I_{Hh} at different biting rate (b)

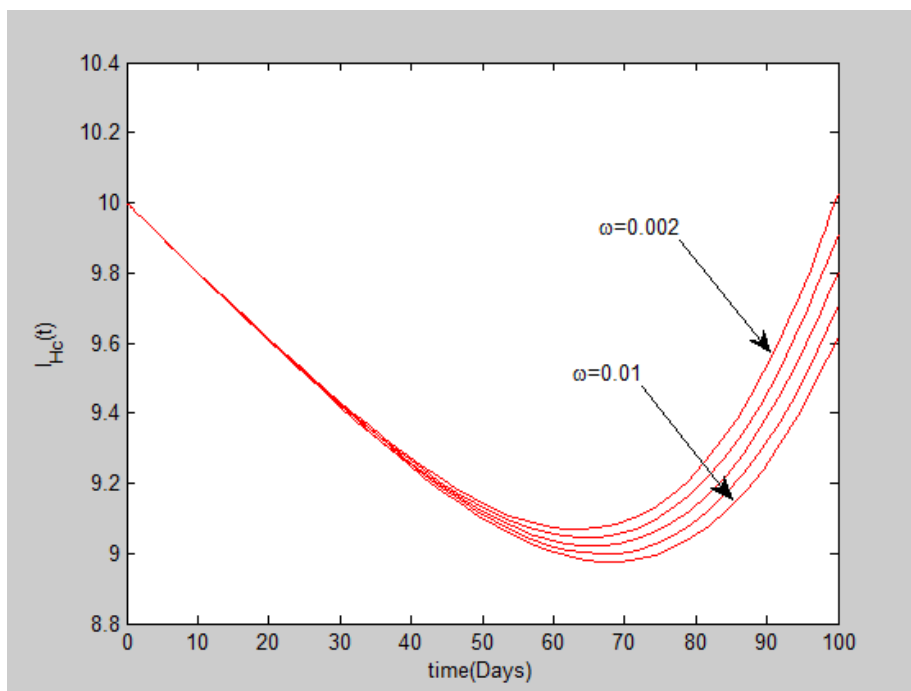


Fig. 3.10: Plot of I_{Hc} against time with different of ω

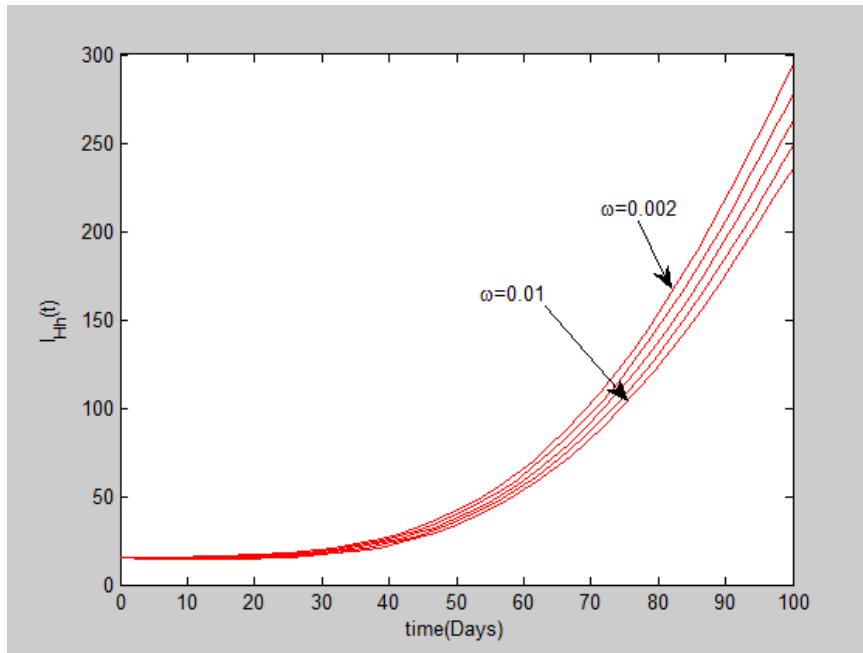


Fig. 3.11: Plot of I_{Hh} against time with different values of ω

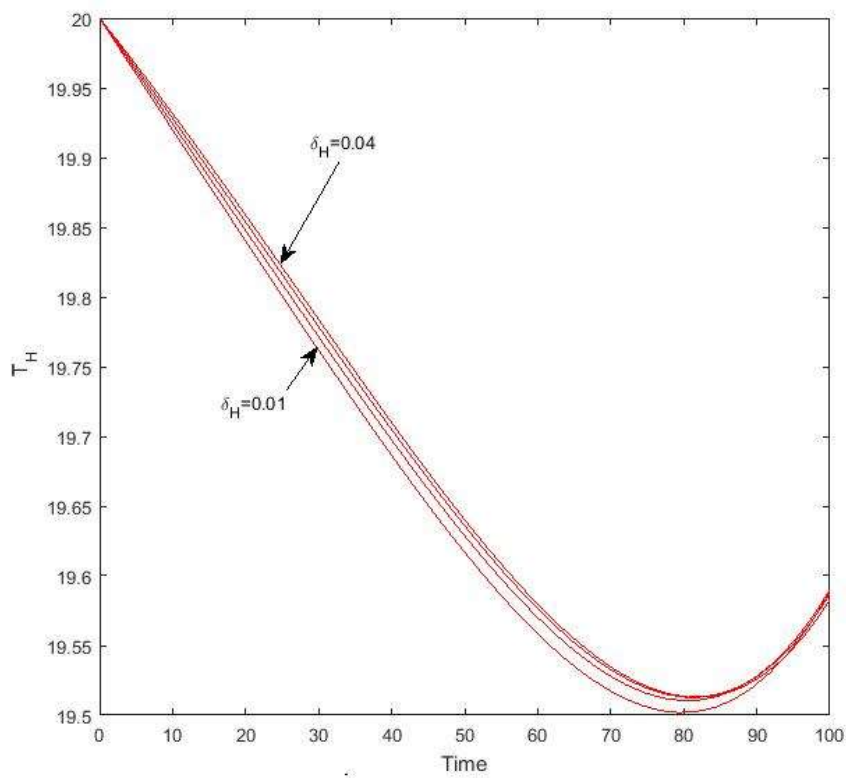


Fig. 12

Fig. 3.12: Plot of T_H against time varying rate of treatment of I_{Hc}

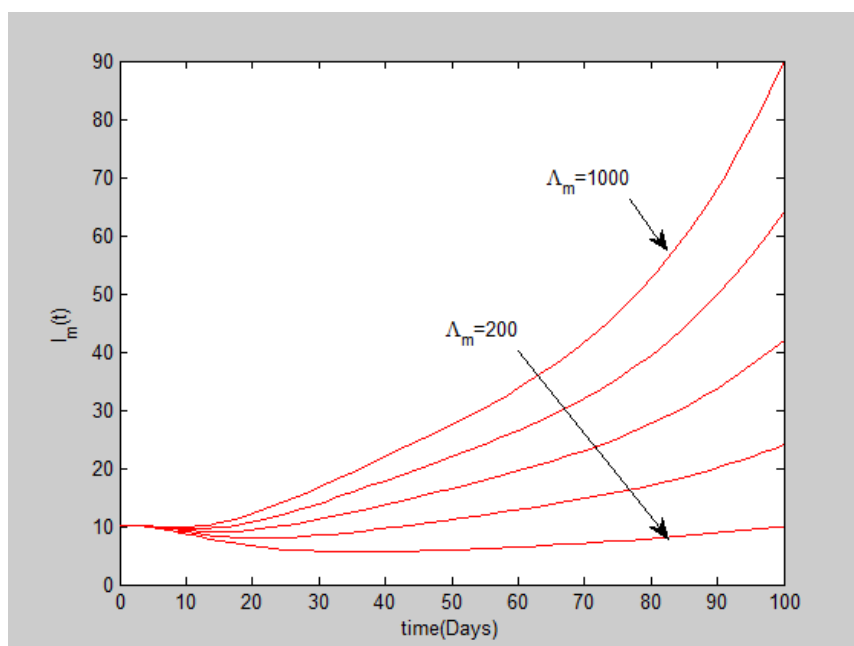


Fig.3.13: Plot of I_m against time for different values of Λ_m

3.11 Interpretation and Discussion of the Simulation Graphs

Fig 3.1 shows the simulation of all the classes against time for $R_0 = 0.3130 < 1$. We observe that the infection is completely eradicated from the population in the long run.

For Fig. 3.2, at $R_0 = 6.2655 > 1$ the graph shows that the simulation of the population in the long run and we observe that all the human population “ N_H ” is reduced. But the vector mosquito population remains in the population. This means that genital elephantiasis will not be eradicated from the population, that is, in future the menace of the infection will still be a treat to the population.

Fig. 3.3, Shows that for $\delta_H =$ or all 0.002, we have both the infective classes of humans and that of mosquito to remain in the population.

In Figure 3.4 we see that an increase in the value of the δ_H precise at $\delta_H = 30$ we have that the three classes were reduced with the I_{HC} completely out of the population in the long run.

Figure 3.5, our simulation reveals that if by any means δ_H approaches zero, the number of individual grows continuously as $t \rightarrow \infty$. Also increasing the value of δ_H have a great effect in reducing the chronic infected individual

Figure 3.6 shows the impact of the effectiveness of treatment on the I_{HC} humans, where we see that the population reduces rigorously until we get to a period of 70 days and then begins to increase again. This shows that the treatment strategies are not effective. Hence there are chances of fail in treatment. Furthermore our analysis shows that in a long run the trend stops.

In Figure 3.7 we see that I_{Hh} grows exponentially and we see that varying the parameter p_1 have little or no impact on the population. Just as we have in Figure 3.6. Our analysis show that in the long run, the trend stopped at $t = 500$.

In Figures 3.8 and 3.9 if in the long run as the biting rate increases, the infected population increases. Then as biting rate decreases, then the I_{HC} individuals are completely eradicated from the population while the I_{Hh} infected individuals are reduced.

In Figure 3.10 and Figure 3.11 we observed that both the I_{Hh} and I_{HC} infected individuals are reduced as omega (ω , the treatment rate) increases though I_{HC} reduces more. Therefore, treatment should be more focused on the I_{HC} infected individuals.

In Figure 3.12, we investigate the effect of increase in treatment rate on the treatment class, and that shows that treatment population decreases until it approaches to $t = 80$ and then increases throughout the long run. This is to say that, within the first 80 days those whose treatment where successful were very minimal irrespective of the actions of the health personnel. As the rate of treatment increases, more people recover from the infection and 80 days is the minimum time needed for full recovery.

Figure 3.13, we observe that varying the value Λ_m . As the recruitment rate of mosquitoes decreases the number of infected mosquitoes reduces. This means that if the entire infected mosquitoes can be eradicated from the environment the rate at which the infection is transmitted will be reduced.

Our research also shows that varying Λ_m reduce the susceptible mosquitoes in the population. This is to say that if effort is made to ensure that the rate at which mosquitoes breeds in the environment is control at the early stage we stand a chance to have a population free of elephantiasis.

4. Summary, Conclusion and Recommendations

4.1 Summary

In this thesis work, we have formulated and analyzed a compartmental model for the Elephantiasis (Lymphatic Filariasis) transmission in human and mosquito populations using a deterministic model. The human population was divided into five compartment: Susceptible(S_H), Exposed(E_H), Highly Infected(I_{Hh}), Chronic Infected(I_{HC}), and Treatment(T_H) class, while the mosquito population was divided into three compartment: Susceptible(S_m), Exposed(E_m), and Infected(I_m). We established a region where the model is epidemiologically feasible and mathematically well-posed, which is called the invariant property. We also showed the existence of a disease-free equilibrium (DFE) and endemic equilibrium (EE) points. We went further to establish the local and global stability of the DFE and EE using the Lyapunov method. We finally carried out a simulation study of the model.

4.2 Conclusion

The reproduction number is computed for both special cases and non-special cases. It is also analyzed for when it is less than unity and for when it is greater than unity. The analysis suggests that treatment of elephantiasis cases has some impact on reducing the spread of the lymphatic filariasis infections.

However, sensitivity analysis tend to give a better picture about the relationship between the reproduction number and the treatment factors, as it shows that the treatment factor reduces the reproduction number but not to the level necessary for the disease elimination. This result suggests that effective lymphatic filariasis control requires strategies beyond elephantiasis treatment only.

From the simulation analysis of all the classes against time for $R_0 = 0.3130 < 1$ we observe that the infection is completely eradicated from the population in the long run.

For $R_0 = 6.2655 > 1$ the graph shows that the simulation of the population in the long run and we observe that all the human population " N_H " is reduced. But the vector mosquito population remains in the population. This means that genital elephantiasis will not be eradicated from the population that is in future the menace of the infection will still be a treat to the population.

The graph also shows that for $\delta_H = 0.002$, we have both the infective classes of humans and that of mosquito to remain in the population. We also see that an increase in the value of the δ_H precise at $\delta_H = 30$ we have that the three classes where reduced with the I_{HC} completely out of the population in the long run.

Our simulation also reveals that if by any means δ_H approaches zero, the number of individual grows continuously as $t \rightarrow \infty$. Also, it shows that increasing the value of δ_H have a great effect in reducing the chronic infected individuals. It further shows that the impact of the effectiveness of treatment on the I_{HC} humans, where we see that the population reduces rigorously until we get to a period of 70 days and them begins to increase again. This shows that the treatment strategies are not effective. Hence there are chances of fail in treatment. Furthermore our analysis shows that in a long run the trend stops.

We also see that I_{Hh} grows exponentially and we see that varying the parameter p_1 have little or no impact on the population. Our analysis show that in the long run, the trend stopped at $t = 500$.

We also found out that in the long run as the biting rate increases, the infected population increases. Then as biting rate decreases, then the I_{HC} individuals are completely eradicated from the population while the I_{Hh} infected individuals are reduced.

We observed that both the I_{Hh} and I_{HC} infected individuals are reduced as omega (ω , the treatment rate) increases though I_{HC} reduces more. Therefore, treatment should be more focused on the I_{HC} infected individuals.

We investigated the effect of increase in treatment rate on the treatment class, and that shows that treatment population decreases until it approaches to $t = 80$ and then increases throughout the long run. This is to say that, within the first 80 days those whose treatment where

successful were very minimal irrespective of the actions of the health personnel. As the rate of treatment increases, more people recover from the infection and 80 days is the minimum time needed for full recovery.

4.3 Recommendations

Based on the above, humans need to boost their antibodies production to be able to subdue the invasion of parasites in the bloodstream. Eating right food and living a healthy lifestyle can help boost the level of antibodies in humans. It is also important to note that reducing human-mosquito contact rate plays a big role in inhibiting the prevalence of elephantiasis.

The use of insecticide-treated bed net, closing of doors and windows against mosquitoes. Clearing of stagnant water and drainages, are all regarded as vector control measures.

However, efforts should be intensified in developing elephantiasis vaccine as this would facilitate the stimulation of the immune system in producing antibodies against elephantiasis.

Treatment should be more focus on the Chronic Infected (I_{HC}) individual than the Highly Infected (I_{Hh}) individual as we can see in our simulation of Figure 4.10.

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