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STABILITY ANALYSIS OF THE DYNAMICS OF TUNGIASIS TRANSMISSION IN ENDEMIC AREAS

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Abstract:

Tungiasis refers to the infestation caused by the permanent penetration of the female sand flea "*Tunga penetrans*" into the skin of the human or animal hosts and causes cutaneous lesion. In this paper, we developed a deterministic model for the dynamics of tungiasis in communities of human beings, animal reservoirs and flea infested environment in order to understand the way tungiasis disease spreads in a poor resource community. We established the conditions for local and global stability of the disease-free and endemic equilibrium points. The computational results showed that the disease-free equilibrium point is locally and globally asymptotically stable when the model basic reproduction number R_0 is less than unit, that is $R_0 < 1$. Using the Lyapunov stability theory and LaSalle's Invariant Principle we found that the endemic equilibrium point (EE) is globally asymptotically stable when $R_0 > 1$. The numerical simulations have been presented to illustrate the way the model variables behave when there is no intervention and that the endemic equilibria exist and they are stable. On-host and off-host control measures which include focal spraying of insecticides to the premises, application of insecticidal dusts on animal bodies, environmental and personal hygiene, educational campaign, use of closed foot wear and application of plant based repellents should be implemented.

1 Introduction

Tungiasis is an ectoparasitic disease caused by the permanent penetration of the female sand flea "*Tunga penetrans"* into the epidermis of its host. Besides humans, various domestic and sylvatic animals are affected (Heukelbach and Feldmeier, 2002). Today, tungiasis is endemic in many countries in Latin America, the Caribbean and sub-Saharan Africa (Heukelbach *et al*. 2001). Transmission occurs when skin of the host comes into contact with soil or floor where adult female sand fleas have developed or when the host lives in close contact with infested animals (Pilger *et al.,* 2008). Models lead to a better understanding of the dynamics of the epidemic and the magnitude of the problem (Kaplan and Brandeau, 1994). Mathematical models can also help in figuring out decisions that are of significance importance on the outcomes and provides comprehensive examinations that

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enter into decisions in a way that human reasoning and debate cannot (Bawa, 2013). Generally the disease dynamics requires a variety of mathematical tools, from model creation to solving differential equations to statistical analysis (Pathak *et al.,* 2010).

The basic concept in mathematical modeling is stability analysis of the equilibria of the epidemic models. The method based on the use of stable Metzler matrices has been proposed and proven to be useful to establish the global stability of the disease free equilibrium as described by Kamgang and Sallet (2008). The importance of the Metzler matrices is well recognized in the stability of dynamical systems and positive systems, more generally in biology, engineering and economics (Gupta, *et al.,* 2014). On the other hand Lyapunov Direct Method by Korobeinikov (2002, 2004, and 2007) and McCluskey, (2006) combined with LaSalle's Invariance Principle (LaSalle, 1976) has been a powerful tool for the analysis of stability of autonomous systems of differential equations through construction of suitable Lyapunov functions. Lyapunov functions are developed to establish the global asymptotic stability of the endemic equilibrium points.

In this paper we consider the dynamics of tungiasis that involves the interaction between humans, animal reservoirs and flea contaminated environment. We determine the model equilibria and analyze their stability at local and global level. We establish the local stability of the disease-free equilibrium point using trace and determinant criterion, the global stability of the disease-free equilibrium point using stable Metzler matrix theory and we also establish the global stability of the endemic equilibrium point using Lyapunov direct Method combined with LaSalle's Invariance Principle. Finally, we perform numerical simulations of the model system in a closed population to present the results. The rest of this paper is organized as follows. In the second section, we present the model with its basic properties. In the third section we carry out a qualitative analysis of the model whereby stability conditions for the disease-free equilibrium and the endemic equilibrium are derived. The fourth section presents different computer simulations of the system. In the last section, the biological significance of our analytical and numerical findings is discussed.

2 Materials and methods

2.1 Model formulation

The total human population at any time t, denoted by N_H is subdivided into three distinct epidemiological subpopulations namely susceptible humans S_H, people who are mildly infested by jiggers I_{Hl} and the people who are severely infested by jiggers I_{Hh}. S_H is generated through birth at a rate b_H so that the human recruitment is $b_H N_H$. S_H may

acquire infestation from the infested animal reservoir I_{Ah} and the flea infested environment F_E following the force of infestation Ψ_H and move to either I_{Hl} or I_{Hh} at the rates $\rho_{AH} I_{Ah}/N_H$ and $\alpha_{EH}\beta_{EH} r_F F_E/(k+F_E)$ respectively. I_{Hl} may acquire infestation from the environment as well and progresses into I_{Hh} at a rate $\alpha_{EH}\beta_{EH} r_F F_E/(k+F_E)$. We assume that individuals in classes S_H and I_{Hl} suffer only natural death at a rate μ_H and for class I_{Hh} the individuals suffer both the natural death at a rate μ_H and disease induced death at a rate σ_H. Similarly the animal reservoir population denoted by N_A is subdivided into three subpopulations namely, the susceptible animals reservoirs S_A, animals that are mildly infested by jiggers I_{Al} and animals that are severely infested by jiggers I_{Ah}. S_A is generated through birth at a rate b_A so that its recruitment rate is $b_A N_A$. S_A may acquire infestation from infested animal reservoir I_{Ah} and the flea infested environment F_E following the force of infestation Ψ_A, and move to either I_{Al} or I_{Ah} at the rates $\rho_A I_{Ah}/N_A$ and $\alpha_{EA}\beta_{EA} r_F F_E/(k+F_E)$ respectively. I_{Al} may acquire infestation from the environment as well and progresses into I_{Ah} at a rate $\alpha_{EA}\beta_{EA} r_F F_E/(k+F_E)$. We assume that individuals in classes S_A and I_{Al} suffer only natural death at a rate μ_A and the individuals class in I_{Ah} suffers both the natural death at a rate μ_A and disease induced death at a rate σ_A. The sub-model of environmental component consists of larvae and adult fleas compartments denoted by L_E and F_E respectively. From within bodies of I_{Hh} and I_{Ah} eggs are shed by the gravid female flea into the environment at an average rate δ_e with contribution rates of $\delta_e I_{Hh}$ and $\delta_e I_{Ah}$ respectively so that they hatch and mature into adult fleas. The compartment L_E suffers a natural mortality rate μ_L and matures to adult fleas class F_E at a rate γ_L. At class F_E the fleas are recruited from larvae as they mature and from the severely infested animal reservoirs I_{Ah} as they are shed at a rate ε_A with contribution rate $\varepsilon_A I_{Ah}$ to the soil environment. The fleas are removed from the environment F_E at the rate $r_F F_E/(k+F_E)$ and μ_F for burrowing into the host and natural death respectively. The forces of infestation for human population is given by $\Psi_H = \rho_{AH} I_{Ah}/N_H + \alpha_{EH}\beta_{EH} r_F F_E/(k+F_E)$ and that of animal reservoir population is given by $\Psi_A = \rho_A I_{Ah}/N_A + \alpha_{EA}\beta_{EA} r_F F_E/(k+F_E)$. The variables and parameters of the model are summarized in Tables 1 and 2 respectively.

Table 1: The state variables of the model

Variable	Description
$S_H(t)$	Number of humans in a susceptible class at time, t
$I_{Hl}(t)$	Number of humans in a mildly infested class at time, t
$I_{Ah}(t)$	Number of humans in a severely infested class at time, t
$S_A(t)$	Number of animals in a susceptible class at time, t
$I_{Al}(t)$	Number of animals in a mildly infested class at time, t
$I_{Ah}(t)$	Number of animals in a severely infested class at time, t
$F_E(t)$	The density of jigger fleas in the environment at time, t
$L_E(t)$	The density of jigger larvae in the environment at time, t
$N_H(t)$	Total human population at time, t
$N_A(t)$	Total animal population at time, t

Table 2: The parameters of the model

Parameter	Description
k, K	Half saturation constant of the jigger fleas and Environment carrying capacity of jigger larvae
γ_L	Maturation rate from larvae to adult jigger fleas
σ_H, σ_A	disease induced mortality rates for humans and animal reservoirs respectively
μ_H, μ_A, μ_F, μ_L	Natural mortality rates for humans, animal reservoir, jigger fleas and jigger larvae respectively
r_F	The rate at which the jigger fleas leave the soil to attack the hosts
β_{EH}, β_{EA}	Effective contact rate between contaminated environment and susceptible humans, Effective contact rate between contaminated environment and susceptible animal reservoirs respectively
ρ_{AH}, ρ_A	Effective contact rate between animals with fleas and susceptible humans, Effective contact rate between animals with fleas and susceptible animals respectively
b_H, b_A	Recruitment rates for humans and animal reservoirs respectively
ε_A	The rate of jigger fleas contribution by the severely infested animal reservoirs into the environment
δ_e	The rate of deposit of jigger eggs into the environment
α_{EH}, α_{EA}	The proportions of jigger fleas that leaves the environment to infest the susceptible human and animal reservoir hosts respectively
Ψ_H, Ψ_A	The forces of infestation for humans and animal populations respectively

2.2 Model flow chart

Using the above assumptions, definition of variables and parameters, the model flow diagram that depicts the dynamics of tungiasis transmission for the human population, animal reservoir population and the jigger fleas in the environment is shown in Figure 1.

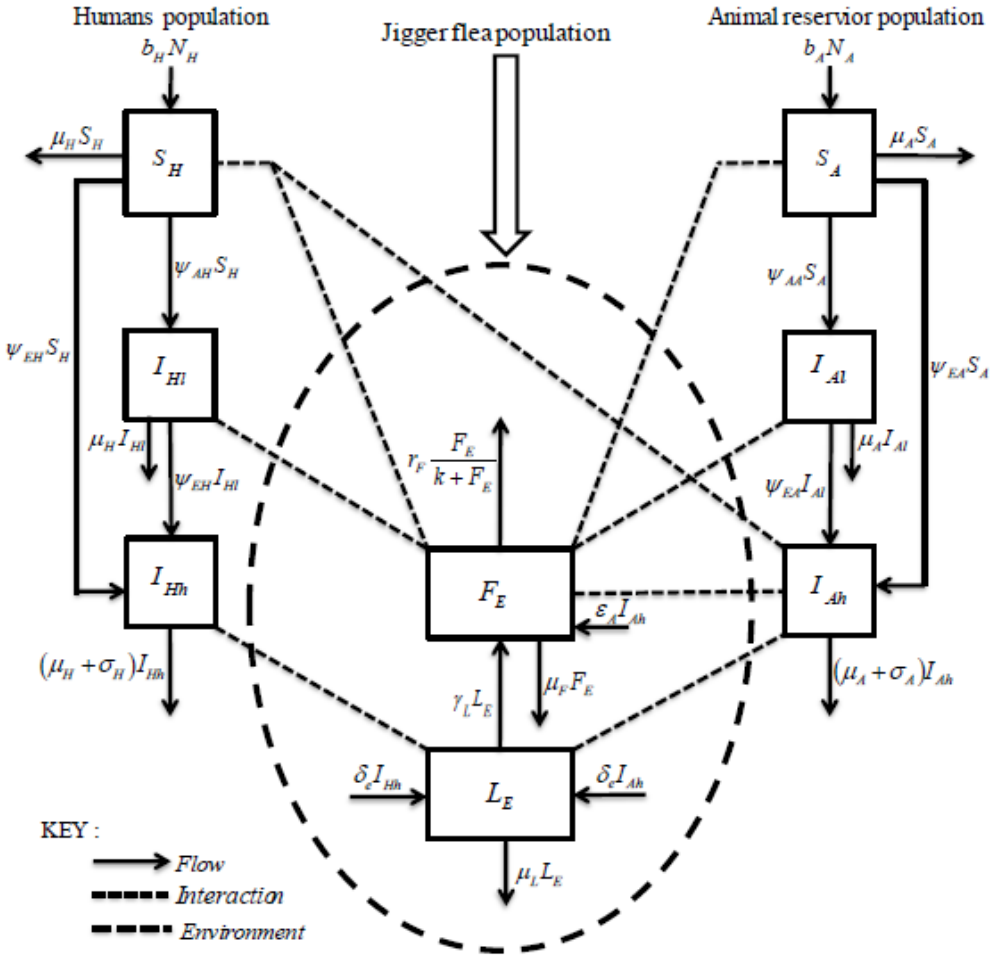

Figure 1: Tungiasis basic dynamical model

Figure 1 shows the possible interactions between humans, animal reservoirs and jigger fleas in the environment. We have the susceptible human S_H, infested humans at mildly and severe states I_{Hl} and I_{Hh}, susceptible animal S_A, infested animals at mildly and severe states I_{Al} and I_{Ah}. We have larval class L_E and the adult jigger flea class F_E.

2.3 Model differential equations

From the compartmental model Figure 1 the following dynamical systems are derived to describe the dynamics of the transmission of Tungiasis:

$$
\begin{cases}
\text{Dynamics in human population} \\
\dfrac{dS_H}{dt} = b_H N_H - \left(\rho_{AH} \dfrac{I_{Ah}}{N_H} + \alpha_{EH}\beta_{EH} r_F \dfrac{F_E}{k+F_E} \right) S_H - \mu_H S_H \\
\dfrac{dI_{Hl}}{dt} = \rho_{AH} \dfrac{I_{Ah}}{N_H} S_H - \alpha_{EH}\beta_{EH} r_F \dfrac{F_E}{k+F_E} I_{Hl} - \mu_H I_{Hl} \\
\dfrac{dI_{Hh}}{dt} = \alpha_{EH}\beta_{EH} r_F \dfrac{F_E}{k+F_E} I_{Hl} + \alpha_{EH}\beta_{EH} r_F \dfrac{F_E}{k+F_E} S_H - (\mu_H + \sigma_H) I_{Hh} \\
\text{Dynamics in animal population} \\
\dfrac{dS_A}{dt} = b_A N_A - \left(\rho_A \dfrac{I_{Ah}}{N_A} + \alpha_{EA}\beta_{EA} r_F \dfrac{F_E}{k+F_E} \right) S_A - \mu_A S_A \\
\dfrac{dI_{Al}}{dt} = \rho_A \dfrac{I_{Ah}}{N_A} S_A - \alpha_{EA}\beta_{EA} r_F \dfrac{F_E}{k+F_E} I_{Al} - \mu_A I_{Al} \\
\dfrac{dI_{Ah}}{dt} = \alpha_{EA}\beta_{EA} r_F \dfrac{F_E}{k+F_E} S_A + \alpha_{EA}\beta_{EA} r_F \dfrac{F_E}{k+F_E} I_{Al} - (\mu_A + \sigma_A) I_{Ah} \\
\text{Dynamics in Jigger flea population} \\
\dfrac{dL_E}{dt} = \delta_e \left(1 - \dfrac{L_E}{K} \right) (I_{Hh} + I_{Ah}) - (\gamma_L + \mu_L) L_E \\
\dfrac{dF_E}{dt} = \gamma_L L_E + \varepsilon_A I_{Ah} - \mu_F F_E - r_F \dfrac{F_E}{k+F_E}
\end{cases} \tag{1}
$$

where

$$N_H(t) = S_H(t) + I_{Hl}(t) + I_{Hh}(t), \quad N_A(t) = S_A(t) + I_{Al}(t) + I_{Ah}(t),$$

$$\psi_{EH} = \alpha_{EH}\beta_{EH} r_F \frac{F_E}{k+F_E}, \ \psi_{AH} = \rho_{AH} \frac{I_{Ah}}{N_H}, \psi_{EA} = \alpha_{EA}\beta_{EA} r_F \frac{F_E}{k+F_E},$$

$$\psi_{AA} = \rho_A \frac{I_{Ah}}{N_A}, \alpha_{EH} + \alpha_{EA} = 1, \ 0 < \alpha_{EH} < 1 \ \text{ and } \ 0 < \alpha_{EA} < 1$$

with initial conditions

$$S_H(0) > 0, I_{Hl}(0) \geq 0, I_{Hh}(0) \geq 0, S_A(0) > 0, I_{Al}(0) \geq 0, I_{Ah}(0) \geq 0, L_E(0) \geq 0, F_E(0) > 0$$

3 Properties of the model

3.1 Invariant region

To test whether the model is well posed epidemiologically and mathematically, we investigate the feasibility of the model solution whereby we present the model system (1) in compact form as in equation (2). We consider the approach used by (Mpeshe *et al.,* 2014).

$$\frac{dX}{dt} = A(X)X + F \tag{2}$$

where $X = (S_H, I_{Hl}, I_{Hh}, S_A, I_{Al}, I_{Ah}, L_E, F_E)^T$, (3)

$A(X)$ is the 8 by 8 matrix:

$$A(X) = \begin{bmatrix} -D_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_a & -D_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_b & A_c & -D_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -D_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_d & -D_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_e & A_f & -D_6 & 0 & 0 \\ 0 & 0 & \delta_e & 0 & 0 & \delta_e & -D_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_A & \gamma_L & -D_8 \end{bmatrix} \tag{4}$$

where

$$D_1 = \rho_{AH}\frac{I_{Ah}}{N_H} + \alpha_{EH}\beta_{EH}r_F\frac{F_E}{k+F_E} + \mu_H, D_2 = \alpha_{EH}\beta_{EH}r_F\frac{F_E}{k+F_E} + \mu_H, D_3 = (\mu_H + \sigma_H),$$

$$D_4 = \rho_A\frac{I_{Ah}}{N_A} + \alpha_{EA}\beta_{EA}r_F\frac{F_E}{k+F_E} + \mu_A, D_5 = \alpha_{EA}\beta_{EA}r_F\frac{F_E}{k+F_E} + \mu_A, A_f = \alpha_{EA}\beta_{EA}r_F\frac{F_E}{k+F_E},$$

$$D_7 = \left\{\frac{\delta_e}{K}(I_{Hh} + I_{Ah}) + (\gamma_L + \mu_L)\right\}, D_8 = \mu_F + \frac{r_F}{k+F_E}, A_a = \rho_{EH}\frac{I_{Ah}}{N_H}, A_d = \rho_A\frac{I_{Ah}}{N_A},$$

$$A_c = \alpha_{EH}\beta_{EH}r_F\frac{F_E}{k+F_E}, A_b = \alpha_{EH}\beta_{EH}r_F\frac{F_E}{k+F_E}, A_e = \alpha_{EA}\beta_{EA}r_F\frac{F_E}{k+F_E}, D_6 = (\mu_A + \sigma_A)$$

and F is the column vector such that:

$$F = (b_H N_H, 0, 0, b_A N_A, 0, 0, 0, 0)^T \geq 0 \tag{5}$$

$A(X)$ is a Metzler matrix for all $X \in \Re_+^8$ considering the fact that $F \geq 0$, model system (1) is positively invariant in $\Re_+^8$, and F is Lipschitz continuous. Thus the feasible region Φ for the model system is the set

$$\Phi = \left\{ \begin{array}{l} (S_H, I_{Hl}, I_{Hh}, S_A, I_{Al}, I_{Ah}, L_E, F_E) \in \Re_+^8 : S_H \leq N_H, I_{Hl} \leq N_H, \\ I_{Hh} \leq N_H, S_A \leq N_A, I_{Al} \leq N_H, I_{Ah} \leq N_A, S_H + I_{Hl} + I_{Hh} \leq N_H, \\ S_A + I_{Al} + I_{Ah} \leq N_A, L_E \leq K, F_E \leq \dfrac{\gamma_L}{\mu_F}K \end{array} \right\} \tag{6}$$

Therefore it can be verified that the solution remains in the feasible region Φ if it starts in this region. Hence, it is sufficient to study the dynamics of the model in Φ.

3.2 Positivity of the solution

Let the initial data be $\{(S_H(t), I_{Hl}(t), I_{Hh}(t), S_A(t), I_{Al}(t), I_{Ah}(t), L_E(t), F_E(t)) \geq 0\} \in \Phi$ then the solution set $\{S_H(t), I_{Hl}(t), I_{Hh}(t), S_A(t), I_{Al}(t), I_{Ah}(t), L_E(t), F_E(t)\}$ of the model system (1) is non-negative for all $t > 0$

From the first equation of the model system (1) we have

$$\frac{dS_H}{dt} = b_H N_H - \left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH}\beta_{EH} r_F \frac{F_E}{k + F_E} \right) S_H - \mu_H S_H$$

$$\frac{dS_H}{dt} \geq -\left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH}\beta_{EH} r_F \frac{F_E}{k + F_E} + \mu_H \right) S_H \qquad (7)$$

Integrating equation (7) by separating the variables we have;

$$\int_0^t \frac{dS_H}{S_H} \geq -\int_0^t \left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH}\beta_{EH} r_F \frac{F_E}{k + F_E} + \mu_H \right) ds$$

$$S_H(t) \geq S_H(0) e^{-\int_0^t \left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH}\beta_{EH} r_F \frac{F_E}{k+F_E} + \mu_H \right) ds} \geq 0$$

By using the similar procedure, it can be shown that the remaining variables $I_{Hl}, I_{Hh}, S_A, I_{Al}, I_{Ah}, L_E, F_E$ are also non-negative for all time $t \geq 0$. Thus the solution set $\Phi = \{S_H(t), I_{Hl}(t), I_{Hh}(t), S_A(t), I_{Al}(t), I_{Ah}(t), L_E(t), F_E(t)\}$ of the model system (1) is non-negative for all $t \geq 0$.

4 Model analysis

4.1 Existence of disease free equilibrium point

The disease free equilibrium (DFE) point of the model system (1) is given by

$$\Phi^o = (S_H^o, I_{Hl}^o, I_{Hh}^o, S_A^o, I_{Al}^o, I_{Ah}^o, L_E^o, F_E^o) = \left(\frac{b_H N_H}{\mu_H}, 0, 0, \frac{b_A N_A}{\mu_A}, 0, 0, 0, 0 \right)$$

where by N_H and N_A are constants with estimated values of 1500 humans and 1200 animal reservoirs respectively.

4.2 Local stability of the disease free equilibrium

The local stability of the disease free equilibrium point is investigated using eigenvalues or trace-determinant criteria of the Jacobian matrix which is defined as a matrix of all first-order partial derivative of a vector-valued function (Simon and Blume, 1994). An equilibrium point is locally asymptotically stable if the Jacobian matrix evaluated at that

point has a negative trace and a positive determinant or has negative eigenvalues (Mpeshe *et al.,* 2009). We prove the following Theorem

Theorem 1: *The disease free equilibrium* $\left(\Phi^{o}\right)$ *whenever it exists is locally asymptotically stable if* $R_o < 1$ *and unstable otherwise.*

Proof

At disease free equilibrium point Φ^{o} we obtain the Jacobian matrix as given in (8) as

$$J\left(\Phi^{o}\right)=\begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & -J_2 & 0 & -J_8 \\ 0 & -\mu_H & 0 & 0 & 0 & J_3 & 0 & 0 \\ 0 & 0 & -J_1 & 0 & 0 & 0 & 0 & J_9 \\ 0 & 0 & 0 & -\mu_A & 0 & -J_4 & 0 & -J_{10} \\ 0 & 0 & 0 & 0 & -\mu_A & J_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -J_6 & 0 & J_{11} \\ 0 & 0 & \delta_e & 0 & 0 & \delta_e & -J_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_A & \gamma_L & -J_{12} \end{bmatrix} \tag{8}$$

where

$$J_1 = \left(\mu_H + \sigma_H\right), J_2 = J_3 = \frac{\rho_{AH} b_H}{\mu_H}, J_4 = J_5 = \frac{\rho_A b_A}{\mu_A}, J_6 = \left(\mu_A + \sigma_A\right), J_7 = \left(\gamma_L + \mu_L\right),$$

$$J_8 = J_9 = \frac{\alpha_{EH}\beta_{EH} r_F N_H b_H}{k\mu_H}, J_{10} = J_{11} = \frac{\alpha_{EA}\beta_{EA} r_F N_A b_A}{k\mu_A}, J_{12} = \left(\mu_F + \frac{r_F}{k}\right)$$

The trace of $J\left(\Phi^{o}\right)$ is negative and its determinant is positive. Therefore all its eigenvalues have negative real parts.

$Tr\left\{J\left(\Phi^{o}\right)\right\} < 0$ which in this case implies

$$-\left\{3\mu_H + \sigma_H + 3\mu_A + \sigma_A + \gamma_L + \mu_L + \mu_F + \frac{r_F}{k}\right\} < 0 \tag{9}$$

$Det\left\{J\left(\Phi^{o}\right)\right\} > 0$ which in this case implies

$$\frac{1}{k}\left\{\mu_A\mu_H\begin{pmatrix} \mu_H\left(\mu_H + \sigma_H\right)\left(\gamma_L + \mu_L\right)\mu_A\left(\mu_A + \sigma_A\right)\left(k\mu_H + r_F\right)\left(1 - R_o\right) \\ -\delta_e r_F \gamma_L\left\{\alpha_{EH}\beta_{EH} N_H b_H \mu_A\left(\mu_A + \sigma_A\right) + \alpha_{EA}\beta_{EA} N_A b_A\left(\mu_H + \sigma_H\right)\right\} \end{pmatrix}\right\} > 0 \tag{10}$$

where $R_o = \frac{\alpha_{EA}\beta_{EA} N_A b_A \varepsilon_A}{\mu_A\left(\mu_A + \sigma_A\right)}\left(\frac{r_F}{\mu_F k + r_F}\right)$ is our basic reproduction number.

We let;

$$\Theta = \mu_H(\mu_H + \sigma_H)(\gamma_L + \mu_L)\mu_A(\mu_A + \sigma_A)(k\mu_H + r_F)(1 - R_o)$$
$$\Omega = \delta_e r_F \gamma_L \{\alpha_{EH}\beta_{EH}N_H b_H \mu_A(\mu_A + \sigma_A) + \alpha_{EA}\beta_{EA}N_A b_A(\mu_H + \sigma_H)\}$$

For the determinant of the Jacobian matrix $J(\Phi^o)$ to be positive Θ must be greater than Ω, that is $\Theta > \Omega$ and the basic reproduction number R_o must be less than one $(R_o < 1)$. It follows that the condition $Det\{J(\Phi^o)\} \geq 0$ implies that $Tr\{J(\Phi^o)\} < 0$. Therefore, the disease free equilibrium (Φ^o) is locally asymptotically stable if and only if $R_o < 1$ and thus we have proved Theorem 1.

4.3 Global stability of the disease free equilibrium

The global stability of the disease free equilibrium is determined using Metzler matrix stability method by Castillo-Chavez *et al.* (2002) whereby the system is put in the form:

$$\frac{dX_n}{dt} = B_o(X_n - X_{DFE,n}) + B_1 X_i \quad (11)$$

$$\frac{dX_i}{dt} = B_2 X_i \quad (12)$$

where X_n and X_i are vectors for non-transmitting and transmitting classes respectively, $X_{DFE,n}$ is a disease free equilibrium, and B_o, B_1 *and* B_2 are matrices. For global stability of the disease free equilibrium to hold the matrix B_o should have the eigenvalues with negative real parts and matrix B_2 need to be a Metzler matrix (i.e. the off-diagonal elements of B_2 are non-negative, denoted by $B_2(X) \geq 0, \forall i \neq j$).

From model system (1) we consider the following sub-systems

$$\begin{cases} X_n = (S_H, S_A, L_E)^T, X_i = (I_{Hl}, I_{Hh}, I_{Al}, I_{Ah}, F_E)^T, \\ X_{DFE,n} = \left(\dfrac{b_H N_H}{\mu_H}, \dfrac{b_A N_A}{\mu_A}, 0\right)^T \end{cases} \quad (13)$$

We consider the system (11) and differentiate it with respect to S_H, S_A and L_E at disease free equilibrium point Φ^o we have the matrix B_o, with which the eigenvalues are of negative real parts as indicated in equation (14)

$$B_o = \begin{bmatrix} -\mu_H & 0 & 0 \\ 0 & -\mu_A & 0 \\ 0 & 0 & -(\gamma_L + \mu_L) \end{bmatrix} \tag{14}$$

Differentiating system (11) with respect to $I_{Hl}, I_{Hh}, I_{Al}, I_{Ah}, F_E$ we obtain matrix B_1 in (15)

$$B_1 = \begin{bmatrix} 0 & 0 & 0 & -\dfrac{\rho_{AH} b_H}{\mu_H} & -\dfrac{\alpha_{EH}\beta_{EH} r_F b_H N_H}{k\mu_H} \\ 0 & 0 & 0 & -\dfrac{\rho_A b_A}{\mu_A} & -\dfrac{\alpha_{EA}\beta_{EA} r_F b_A N_A}{k\mu_A} \\ 0 & \delta_e & 0 & \delta_e & 0 \end{bmatrix} \tag{15}$$

Again from system (12) we differentiate it with respect to $I_{Hl}, I_{Hh}, I_{Al}, I_{Ah}, F_E$ we have matrix B_2 as indicated in equation (16)

$$B_2 = \begin{bmatrix} -\mu_H & 0 & 0 & \dfrac{\rho_{AH} b_H}{\mu_H} & 0 \\ 0 & -(\mu_H + \sigma_H) & 0 & 0 & \dfrac{\alpha_{EH}\beta_{EH} r_F b_H N_H}{k\mu_H} \\ 0 & 0 & -\mu_A & \dfrac{\rho_A b_A}{\mu_A} & 0 \\ 0 & 0 & 0 & -(\mu_A + \sigma_A) & \dfrac{\alpha_{EA}\beta_{EA} r_F b_A N_A}{k\mu_A} \\ 0 & 0 & 0 & \varepsilon_A & -\left(\mu_F + \dfrac{r_F}{k}\right) \end{bmatrix} \tag{16}$$

We have investigated that the eigenvalues of matrix B_o are real and negative and the matrix B_2 is a Metzler matrix therefore the model system (1) is globally asymptotically stable which is supported by the following theorem.

Theorem 2: *The disease-free equilibrium point* (Φ^o) *is globally asymptotically stable if* $R_o \leq 1$ *and unstable if* $R_o > 1$

4.4 Existence of endemic equilibrium points

Endemic equilibrium point Φ^* is a steady state solution in which the disease persists in the population i.e. $I_{Hl} \neq 0, I_{Hh} \neq 0, I_{Al} \neq 0, I_{Ah} \neq 0$ and $F_E \neq 0$. We equate all expressions for the equations in the model system (1) to zero and let $\Phi^* = (S_H^*, I_{Hl}^*, I_{Hh}^*, S_A^*, I_{Al}^*, I_{Ah}^*, L_E^*, F_E^*)$ and $P^* = r_F F_E^* / (k + F_E^*)$ considering that the animal population is constant for $b_A > \mu_A$ then from model system (1) at arbitrary equilibrium solution Φ^* we have.

$$P^* = r_F\, F_E^* / \left(k + F_E^*\right) \tag{17}$$

$$I_{Ah}^* = \frac{N_A\left(b_A - \mu_A\right)}{\sigma_A} \tag{18}$$

$$S_H^* = \frac{\sigma_A b_H \left(N_H\right)^2}{\rho_{AH} N_A\left(b_A - \mu_A\right) + \sigma_A \alpha_{EH} \beta_{EH} P^* + \mu_H} \tag{19}$$

$$I_{Hl}^* = \frac{\rho_{EH} N_A\left(b_A - \mu_A\right) b_H \left(N_H\right)^2}{N_H\left(\alpha_{EH}\beta_{EH}P^* + \mu_H\right)\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + \sigma_A \alpha_{EH}\beta_{EH}P^* + \sigma_A \mu_H\right)} \tag{20}$$

$$I_{Hh}^* = \frac{\alpha_{EH}\beta_{EH}P^* b_H \left(N_H\right)^2 \left\{\begin{matrix}\rho_{EH} N_A\left(b_A - \mu_A\right) \\ + \sigma_A N_H\left(\alpha_{EH}\beta_{EH}P^* + \mu_H\right)\end{matrix}\right\}}{\left(\mu_H + \sigma_H\right) N_H\left(\alpha_{EH}\beta_{EH}P^* + \mu_H\right)\left\{\begin{matrix}\rho_{AH} N_A\left(b_A - \mu_A\right) \\ + \sigma_A \alpha_{EH}\beta_{EH}P^* + \sigma_A \mu_H\end{matrix}\right\}} \tag{21}$$

$$S_A^* = \frac{\sigma_A b_A \left(N_A\right)^2}{\rho_A N_A\left(b_A - \mu_A\right) + \sigma_A \alpha_{EA}\beta_{EA}P^* + \sigma_A \mu_H} \tag{22}$$

$$I_{Al}^* = \frac{\sigma_A \rho_A N_A b_A \left(b_A - \mu_A\right)}{\left(\sigma_A \alpha_{EA}\beta_{EA}P^* + \sigma_A \mu_A\right)\left(\rho_A\left(b_A - \mu_A\right) + \sigma_A \alpha_{EA}\beta_{EA}P^* + \sigma_A \mu_H\right)} \tag{23}$$

$$L_E^* = \frac{DP^{*2} + UP^* + M}{QP^{*2} + RP^* + T} \tag{24}$$

where

$$D = \delta_e K N_H \sigma_A \left(\alpha_{EH}\beta_{EH}\right)^2 \left(N_A(b_A - \mu_A)(\mu_H + \sigma_H) + b_H N_H^2\right)$$

$$U = \delta_e K N_H \alpha_{EH}\beta_{EH} \left\{\begin{matrix} N_A(b_A - \mu_A)(\mu_H + \sigma_H)\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + 2\sigma_A \mu_H\right) \\ + b_H N_H\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + \mu_H\right)\end{matrix}\right\}$$

$$U = \delta_e K N_H \alpha_{EH}\beta_{EH} \left\{\begin{matrix} N_A(b_A - \mu_A)(\mu_H + \sigma_H)\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + 2\sigma_A \mu_H\right) \\ + b_H N_H\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + \mu_H\right)\end{matrix}\right\}$$

$$M = \delta_e K N_H N_A\left(b_A - \mu_A\right)\mu_H\left(\mu_H + \sigma_H\right)\left(\rho_{AH} N_A\left(b_A - \mu_A\right)\right)$$

$$Q = \left(\alpha_{EH}\beta_{EH}\right)^2 N_H \sigma_A \left(\delta_e b_H N_H^2 + (\mu_H + \sigma_H)\left(\delta_e N_A(b_A - \mu_A) + \gamma_L + \mu_L\right)\right)$$

$$R = N_H \alpha_{EH}\beta_{EH} \left\{\begin{matrix} \delta_e \rho_{AH} N_A\left(b_A - \mu_A\right)\left(N_A(b_A - \mu_A)(\mu_H + \sigma_H) + \mu_H b_H N_H\right) \\ + \left(\begin{matrix}\sigma_A\left(\mu_H + \sigma_H\right)\left(\gamma_L + \mu_L\right)\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + \sigma_A \mu_H\right) \\ + 2\delta_e \mu_H N_A\left(b_A - \mu_A\right)\end{matrix}\right)\end{matrix}\right\}$$

$$T = N_A \mu_H\left(\mu_H + \sigma_H\right)\left(\rho_{AH} N_A\left(b_A - \mu_A\right) + \sigma_A \mu_H\right)\left(\delta_e N_H(b_A - \mu_A) + \sigma_A(\gamma_L + \mu_L)\right)$$

Substituting P^* and I_{Ah}^* from equations (17) and (18) respectively into equation (25) to solve for F_E^* such that

$$0 = \gamma_L L_E^* + \varepsilon_A I_{Ah}^* - \mu_F F_E^* - r_F \frac{F_E^*}{k + F_E^*} \tag{25}$$

$$F_E^* = \frac{1}{\sigma_A \mu_F}\left\{\sigma_A \gamma_L L_E^* + \varepsilon_A N_A (b_A - \mu_A) - \sigma_A P^*\right\} \tag{26}$$

Substituting F_E^* from equation (26) into equation (17) we have

$$P^* = \frac{\sigma_A \gamma_L r_F L_E^* + r_F \varepsilon_A N_A (b_A - \mu_A) - \sigma_A r_F P^*}{\sigma_A \mu_F k + \sigma_A \gamma_L L_E^* + \varepsilon_A N_A (b_A - \mu_A) - \sigma_A P^*} \tag{27}$$

$$BP^* + \sigma_A \gamma_L L_E^* P^* + AP^* - \sigma_A P^{*2} = \sigma_A \gamma_L r_F L_E^* + r_F A - \sigma_A r_F P^*$$

$$(\sigma_A \gamma_L P^* - C) L_E^* = A + \sigma_A P^{*2} - BP^* \tag{28}$$

where $A = r_F \varepsilon_A N_A (b_A - \mu_A)$,

$B = \sigma_A \mu_F k + \varepsilon_A N_A (b_A - \mu_A) + \sigma_A r_F$,

$C = \sigma_A \gamma_L r_F$

Substituting L_E^* from equation (24) into (28) we have

$$(\sigma_A \gamma_L P^* - C)\left(\frac{DP^{*2} + UP^* + M}{QP^{*2} + RP^* + T}\right) = A + \sigma_A P^{*2} - BP^*$$

$$(\sigma_A \gamma_L P^* - C)(DP^{*2} + UP^* + M) = (A + \sigma_A P^{*2} - BP^*)(QP^{*2} + RP^* + T)$$

$$\sigma_A Q P^{*4} + (\sigma_A R - \sigma_A \gamma_L D - BQ) P^{*3} + (AQ + \sigma_A T + CD - BR - \sigma_A \gamma_L U) P^{*2}$$

$$+ (AR + CU - M\sigma_A \gamma_L - BT) P^* + CM + AT = 0$$

On simplification we have the four degree polynomial function in terms of P^* given by

$$a_0 P^{*4} + a_1 P^{*3} + a_2 P^{*2} + a_3 P^* + a_4 = 0 \tag{29}$$

where

$a_0 = \sigma_A Q$

$a_1 = (\sigma_A R - \sigma_A \gamma_L D - BQ)$

$a_2 = (AQ + \sigma_A T + CD - BR - \sigma_A \gamma_L U)$

$a_3 = (AR + CU - M\sigma_A \gamma_L - BT)$

$a_4 = CM + AT$

Substituting P^* from equation (17) into (29) we have the expression for the endemic equilibria which satisfy the following four degree polynomial in terms of F_E^* given by

$$Y_0 F_E^{*4} + Y_1 F_E^{*3} + Y_2 F_E^{*2} + Y_3 F_E^{*} + Y_4 = 0 \tag{30}$$

where

$$Y_0 = a_0 r_F^4 + a_1 r_F^3 + a_2 r_F^2 + a_3 r_F + a_4$$
$$Y_1 = \left(a_1 r_F^3 + 2a_2 r_F^2 + 3a_3 r_F + 4a_4\right)k$$
$$Y_2 = \left(a_2 r_F^2 + 3a_3 r_F k^2 + 6a_4\right)k^2$$
$$Y_3 = \left(a_3 r_F + 4a_4\right)k^3$$
$$Y_4 = a_3 k^4$$

Since the variables $S_H^*, I_{Hl}^*, I_{Hh}^*, S_A^*, I_{Al}^*, L_E^*, F_E^*$ are expressed in terms of P^* then we substitute $P^* = r_F F_E^* / \left(k + F_E^*\right)$ in equation (29) we obtain the polynomial function with degree four in (30) which represents the presence of endemic equilibrium points. This shows that there are possible four roots for F_E^* which further implies that there are at most four possible endemic equilibrium points.

4.5 Global stability of endemic equilibrium point

Global stability of the endemic equilibrium (EE) is investigated using Lyapunov method and LaSalle's invariance principle. The approach which has been found to be useful for compartmental epidemic models with any number of compartments (Korobeinikov and Maini, 2004). To achieve this we construct a suitable Lyapunov function of the form:

$$L = \sum_{i=1}^{8} A_i \left(x_i - x_i^* \ln x_i\right) \tag{31}$$

where A_i is a properly selected constant, x_i is the population of i^{th} compartment, x_i^* is the equilibrium value of x_i and $A_i > 0$. The Lyapunov function denoted by L is continuous and differentiable. We have:

$$\begin{cases} L\left(S_H, I_{Hl}, I_{Hh}, S_A, I_{Al}, I_{Ah}, L_E, F_E\right) = A_1\left(S_H - S_H^* \ln S_H\right) + A_2\left(I_{Hl} - I_{Hl}^* \ln I_{Hl}\right) \\ \qquad + A_3\left(I_{Hh} - I_{Hh}^* \ln I_{Hh}\right) + A_4\left(S_A - S_A^* \ln S_A\right) \\ \qquad + A_5\left(I_{Al} - I_{Al}^* \ln I_{Al}\right) + A_6\left(I_{Ah} - I_{Ah}^* \ln I_{Ah}\right) \\ \qquad + A_7\left(L_E - L_E^* \ln L_E\right) + A_8\left(F_E - F_E^* \ln F_E\right) \end{cases} \tag{32}$$

The global stability of endemic equilibrium (EE) holds if its time derivative $\frac{dL}{dt} \le 0$.

Proof:

The time derivative of the Lyapunov function L is given by

$$\begin{cases} \dfrac{dL}{dt} = A_1\left(1-\dfrac{S_H^*}{S_H}\right)\dfrac{dS_H}{dt} + A_2\left(1-\dfrac{I_{Hl}^*}{I_H}\right)\dfrac{dI_H}{dt} + A_3\left(1-\dfrac{I_{Hh}^*}{I_{Hl}}\right)\dfrac{dI_{Hl}}{dt} + A_4\left(1-\dfrac{S_A^*}{S_A}\right)\dfrac{dS_A}{dt} \\ \qquad + A_5\left(1-\dfrac{I_{Al}^*}{I_{Al}}\right)\dfrac{dI_{Al}}{dt} + A_6\left(1-\dfrac{I_{Ah}^*}{I_{Ah}}\right)\dfrac{dI_{Ah}}{dt} + A_7\left(1-\dfrac{L_E^*}{L_E}\right)\dfrac{dL_E}{dt} + A_8\left(1-\dfrac{F_E^*}{F_E}\right)\dfrac{dF_E}{dt} \end{cases}$$

$$\begin{aligned} \frac{dL}{dt} = & -A_1\left(1-\frac{S_H^*}{S_H}\right)^2\mu_H S_H + A_1\left(1-\frac{S_H^*}{S_H}\right)\left(\frac{I_{Ah}{}^* S_H^*}{I_{Ah}S_H}-1\right)\frac{I_{Ah}}{N_H}\rho_{AH}S_H \\ & + A_1\left(1-\frac{S_H^*}{S_H}\right)\left(\frac{F_E^* S_H^*\left(k+F_E\right)}{F_E S_H\left(k+F_E^*\right)}-1\right)\frac{F_E}{k+F_E}\alpha_{EH}\beta_{EH}r_F S_H \\ & - A_2\left(1-\frac{I_{Hl}^*}{I_{Hl}}\right)^2\mu_H I_{Hl} + A_2\left(1-\frac{I_{Hl}^*}{I_{Hl}}\right)\left(\frac{F_E^* I_{Hl}^*\left(k+F_E\right)}{F_E I_{Hl}\left(k+F_E^*\right)}-1\right)\frac{F_E I_{Hl}}{k+F_E}\alpha_{EH}\beta_{EH}r_F \\ & - A_3\left(1-\frac{I_{Hh}^*}{I_{Hh}}\right)^2\left(\mu_H+\sigma_H\right)I_{Hh} \\ & - A_4\left(1-\frac{S_A^*}{S_A}\right)^2\mu_A S_A + A_4\left(1-\frac{S_A^*}{S_A}\right)\left(\frac{I_{Ah}^* S_A^*}{I_{Ah}S_A}-1\right)\frac{I_{Ah}}{N_A}\rho_A S_A \\ & + A_4\left(1-\frac{S_A^*}{S_A}\right)\left(\frac{F_E^* S_A^*\left(k+F_E\right)}{F_E S_A\left(k+F_E^*\right)}-1\right)\frac{F_E}{k+F_E}\alpha_{EA}\beta_{EA}r_F S_A \\ & - A_5\left(1-\frac{I_{Al}^*}{I_{Al}}\right)^2\mu_A I_{Al} + A_5\left(1-\frac{I_{Al}^*}{I_{Al}}\right)\left(\frac{F_E^* I_{Al}^*\left(k+F_E\right)}{F_E I_{Al}\left(k+F_E^*\right)}-1\right)\frac{F_E I_{Al}}{k+F_E}\alpha_{EA}\beta_{EA}r_F \\ & - A_6\left(1-\frac{I_{Ah}^*}{I_{Ah}}\right)^2\left(\mu_A+\sigma_A\right)I_{Ah} \\ & - A_7\left(1-\frac{L_E^*}{L_E}\right)^2\left(\gamma_L+\mu_L\right)L_E + A_7\frac{\delta_e}{K}\left(1-\frac{L_E^*}{L_E}\right)\left(\frac{I_{Hh}^* L_E^*}{I_{Hh}L_E}-1\right)I_{Hh}L_E \\ & + A_7\frac{\delta_e}{K}\left(1-\frac{L_E^*}{L_E}\right)\left(\frac{I_{Ah}^* L_E^*}{I_{Ah}L_E}-1\right)I_{Ah}L_E \\ & - A_8\left(1-\frac{F_E^*}{F_E}\right)^2\mu_F F_E + A_8\left(1-\frac{F_E^*}{F_E}\right)\left(\frac{F_E^*\left(k+F_E\right)}{F_E\left(k+F_E^*\right)}-1\right)r_F\frac{F_E}{k+F_E} \end{aligned} \tag{33}$$

By adopting the approach by McCluskey, (2006) we have the following expressions

$$\begin{aligned}\frac{dL}{dt} &= -A_1\left(1-\frac{S_H^*}{S_H}\right)^2\mu_H S_H - A_2\left(1-\frac{I_{Hl}^*}{I_{Hl}}\right)^2\mu_H I_{Hl} - A_3\left(1-\frac{I_{Hh}^*}{I_{Hh}}\right)^2(\mu_H+\sigma_H)I_{Hh}\\ &- A_4\left(1-\frac{S_A^*}{S_A}\right)^2\mu_A S_A - A_5\left(1-\frac{I_{Al}^*}{I_{Al}}\right)^2\mu_A I_{Al} - A_6\left(1-\frac{I_{Ah}^*}{I_{Ah}}\right)^2(\mu_A+\sigma_A)I_{Ah}\\ &- A_7\left(1-\frac{L_E^*}{L_E}\right)^2(\gamma_L+\mu_L)L_E - A_8\left(1-\frac{F_E^*}{F_E}\right)^2\mu_F F_E + Z(\Phi)\end{aligned} \tag{34}$$

where

$$\begin{aligned}Z(\Phi) &= A_1\left(1-\frac{S_H^*}{S_H}\right)\left(\frac{I_{Ah}^*S_H^*}{I_{Ah}S_H}-1\right)\frac{I_{Ah}}{N_H}\rho_{AH}S_H \le 0\\ &+ A_1\left(1-\frac{S_H^*}{S_H}\right)\left(\frac{F_E^*S_H^*(k+F_E)}{F_E S_H(k+F_E^*)}-1\right)\frac{F_E}{k+F_E}\alpha_{EH}\beta_{EH}r_F S_H \le 0\\ &+ A_2\left(1-\frac{I_{Hl}^*}{I_{Hl}}\right)\left(\frac{F_E^*I_{Hl}^*(k+F_E)}{F_E I_{Hl}(k+F_E^*)}-1\right)\frac{F_E}{k+F_E}\alpha_{EH}\beta_{EH}r_F I_{Hl} \le 0\\ &+ A_4\left(1-\frac{S_A^*}{S_A}\right)\left(\frac{I_{Ah}^*S_A^*}{I_{Ah}S_A}-1\right)\frac{I_{Ah}}{N_A}\rho_A S_A \le 0\\ &+ A_4\left(1-\frac{S_A^*}{S_A}\right)\left(\frac{F_E^*S_A^*(k+F_E)}{F_E S_A(k+F_E^*)}-1\right)\frac{F_E}{k+F_E}\alpha_{EA}\beta_{EA}r_F S_A \le 0\\ &+ A_5\left(1-\frac{I_{Al}^*}{I_{Al}}\right)\left(\frac{F_E^*I_{Al}^*(k+F_E)}{F_E I_{Al}(k+F_E^*)}-1\right)\frac{F_E}{k+F_E}\alpha_{EA}\beta_{EA}r_F I_{Al} \le 0\\ &+ A_7\left(1-\frac{L_E^*}{L_E}\right)\left(\frac{I_{Hh}^*L_E^*}{I_{Hh}L_E}-1\right)\frac{\delta_e}{K}I_{Hh}L_E + A_7\left(1-\frac{L_E^*}{L_E}\right)\left(\frac{I_{Ah}^*L_E^*}{I_{Ah}L_E}-1\right)\frac{\delta_e}{K}I_{Ah}L_E \le 0\\ &+ A_8\left(1-\frac{F_E^*}{F_E}\right)\left(\frac{F_E^*(k+F_E)}{F_E(k+F_E^*)}-1\right)r_F\frac{F_E}{k+F_E} \le 0\end{aligned} \tag{35}$$

$Z(\Phi)$ is non-positive by following the approach of Korobeinikov (2002, 2004, 2007) and McCluskey (2006). Thus $Z(\Phi)\le 0$ for all $\Phi > 0$ Hence, $\frac{dL}{dt}\le 0$ in Φ and is zero when $\Phi = \Phi^*$. Therefore the largest invariant set in Φ such that $\frac{dL}{dt}=0$ is the singleton (Φ^*) which is our endemic equilibrium point. By LaSalle's invariant principle (LaSalle, 1976) we conclude that (Φ^*) is globally asymptotically stable (GAS). Thus, we establish the following Theorem

Theorem 5: *The endemic equilibrium point* Φ^* *of model system* (3) *is globally asymptotically stable in* Φ *if* $R_o > 1$ *and unstable otherwise.*

5 Simulations on the basic model state variables over time

The simulation of the model is conducted to find out the dynamics of Tungiasis disease in the population when there is no intervention. It is performed using MALAB, and we set time in weeks and days. We use parameter values whose sources are from literature and others are estimated. Furthermore through numerical simulation we illustrate the stability of the endemic equilibrium states in human, animal reservoirs and flea in the environment whenever they exist. The parameter values used for simulation are shown in Table 3.

5.1 Parameter Estimation

In this section we estimate some of the parameter values of Tungiasis model. We estimate the human mortality rate μ_H, taking into consideration that the average life expectancy of the human population in Tanzania is 60.9 years (UNICEF, 2015) which is equivalently to 4.5×10^{-5} per day. According to Tanzania population (TP, 2016) the human birth rate is 0.00011 per day. The maturation rate γ_L of larvae into adult flea is estimated to be 0.0105 per day and the shedding rate of fleas ε_A to be 0.4 per day. The death of the flea occurs around day 25 post-penetration (Eisele *et al.,* 2003), we therefore assume the life span of a flea to be 25 days, which implies that its death rate μ_F is 0.04 per day. The concentration of jigger fleas in the soil environment is not known; we therefore consider the number of fleas in one cubic meter of sand and assume the maximal larvae carrying capacity K to be $1\times10^5 \text{cell/m}^3$ and the half saturation constant for fleas k to be 1×10^4 cell/m^3. The disease induced death rate for animal reservoir population σ_A is estimated to be 0.037 per day and the disease induced death rate for human population σ_H is estimated to be 0.011 per day. The natural death rate of animal reservoir μ_A ranges from $(360-3600)$days (Radostits, 2001; Gaff *et al.,* 2007). The values of effective contact rates are $\rho_A = 0.26$ per day (Allerson *et al.,* 2013), $\rho_{AH} = 0.052$ per day (Gaff *et al.*, 2007), $\beta_{EH} = 0.19$ per day and $\beta_{EA} = 0.48$ per day are estimated.

Table 3: Parameter values used for the simulation of model variables

Parameter	Value/Range	Source/References
k	1×10^4 cell/m^3	Estimated
K	1×10^5cell/m^3	Estimated
γ_L	0.0105per day	Estimated
σ_H	0.011 per day	Estimated
σ_A	0.037 per day	Estimated
μ_H	0.000045 per day	UNICEF. (2015)
μ_A	$0.0028(360-3600)^{-1}$ per day	Gaff *et al.* (2007); Radostits. (2001)
μ_F	0.04 per day	Eisele *et al.* (2003),
r_F	0.58 per day	Estimated
μ_L	0.08 per day	Estimated
β_{EH}	0.19 per day	Estimated
β_{EA}	0.48 per day	Estimated
ρ_{AH}	0.052 per day	Gaff *et al.* (2007)
ρ_A	0.26 (0.091-0.9) per day	Allerson *et al.* (2013)
b_H	0.00011 per day	TP, (2016).
b_A	0.022 per day	Gaff *et al.* (2007)
ε_A	0.40 per day	Estimated
δ_e	0.12 per day	Estimated
α_{EH}	0.4	Estimated
α_{EA}	0.6	Estimated

5.2 Discussion of the results

Figure 2 illustrate the dynamics in humans, animal reservoirs and flea populations, showing the behavior of eight state variables $S_H, I_{Hl}, I_{Hh}, S_A, I_{Al}, I_{Ah}, L_E$ and F_E over time when there is no intervention.

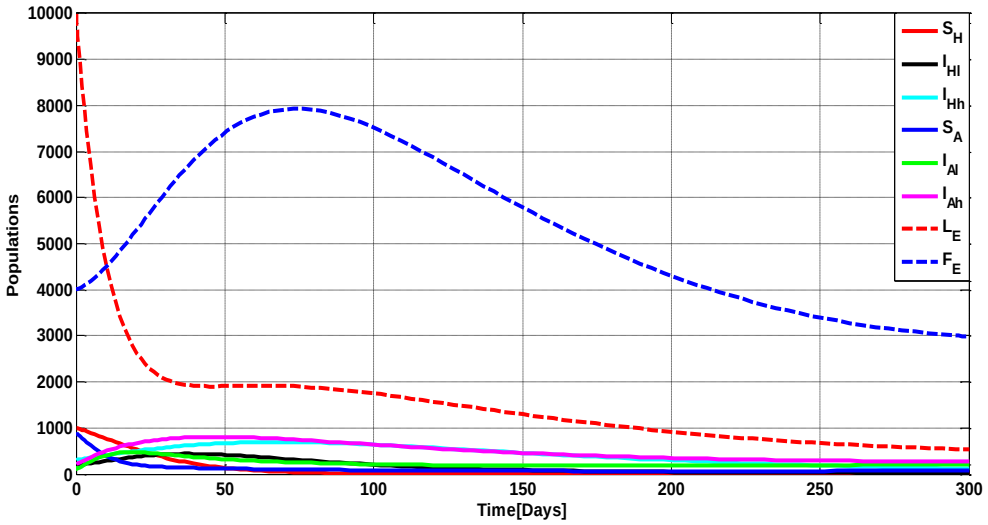

Figure 2: Dynamics in model sub-populations

From Figure 2: The red dotted line is the larvae population and the blue dotted line is the adult flea population. The larvae population decreases exponentially to attain endemic equilibrium point as they die naturally and as they mature into adult fleas. The population of adult fleas generated from larvae maturation increase exponentially to a certain point in time then decrease to attain endemic equilibrium point as they die a natural death and as they burrow into the epidermis of the hosts. The red line is the susceptible humans, the black line is mildly infested humans and the cyan line is the severely infested humans. The susceptible humans decrease exponentially due to natural death and as they acquire infestation from severely infested animal or from the flea infested environment and eventually attain the endemic equilibrium point. The decrease in susceptible human population results into the growth of the mildly infested humans that grow exponentially after sometime they start declining due to natural death and as they acquire more infestation from the environment to join the severely infested human class then eventually attains endemic equilibrium point. The severely infested humans grow exponentially to a certain point in time then start declining due to natural and disease induced death and finally attains endemic equilibrium point. The blue line is the susceptible animal reservoirs, the green line is the mildly infested animal reservoirs and the magenta line is the severely infested animal reservoirs. The susceptible animal reservoirs decrease exponentially due to natural death and as they acquire infestation from severely infested animal reservoirs or

from the flea infested environment. The decrease of susceptible animal reservoirs results into the growth of the mildly infested animal reservoirs that grow exponentially and then start declining due to natural death and as the acquire more infestation from the environment to join severely infested animal reservoirs class then eventually attain endemic equilibrium point. The severely infested animal reservoirs grow exponentially to a certain point in time then start declining due to natural and disease induced mortalities and finally they attain endemic equilibrium point.

5.3 The variation of population variables on the dynamics of Tungiasis over time

The numerical simulations as illustrated in Figures 3(a), 3(b), 3(c), 4(a), 4(b), 4(c), 5(a) and 5(b) show the variations of the model variables $S_H, I_{Hl}, I_{Hh}, S_A, I_{Al}, I_{Ah}, L_E$ and F_E over time. The trajectories of the model variables originating from different initial values converges to a common endemic equilibrium levels which implies the existence and stability of the endemic equilibrium states in human, animal reservoirs and flea populations whenever they exist.

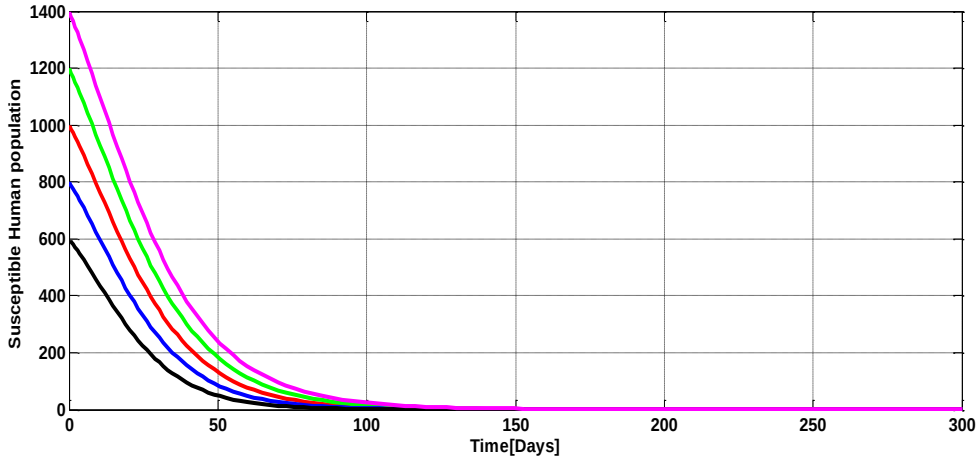

Figure 3(a): Stability of the endemic equilibrium for susceptible human population

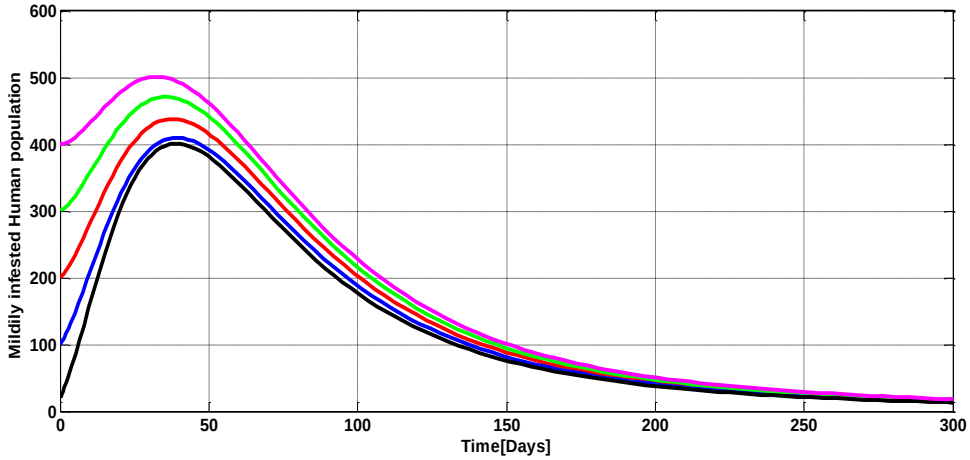

Figure 3(b): Stability of endemic equilibrium for mildly infested human population

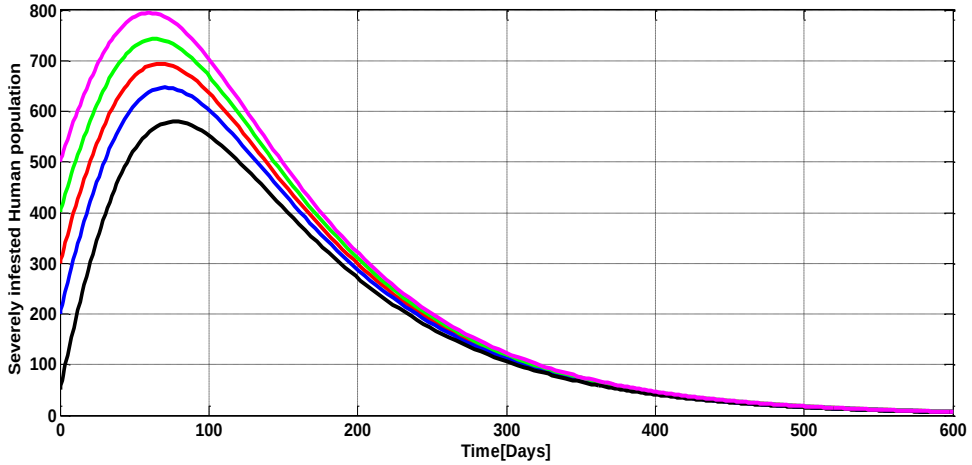

Figure 3(c): Stability of the endemic equilibrium for severely infested human population

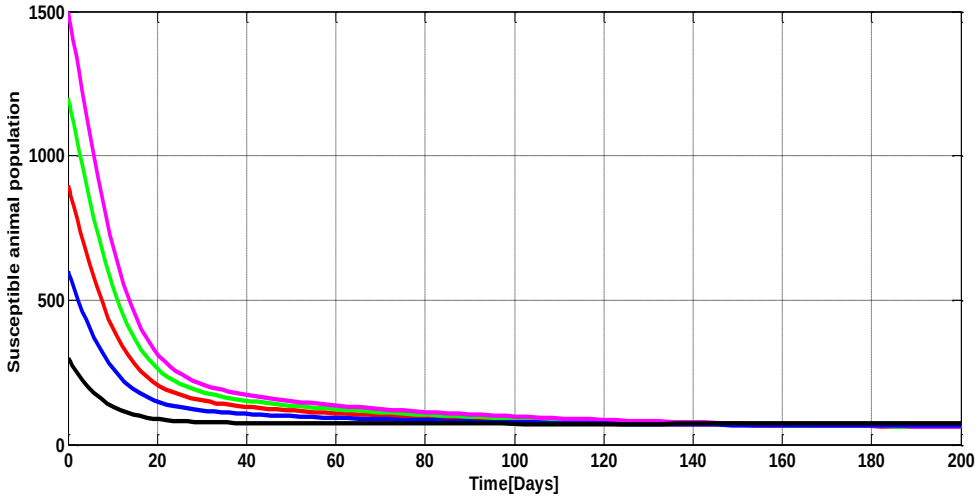

Figure 4(a): Stability of the endemic equilibrium for susceptible animal population

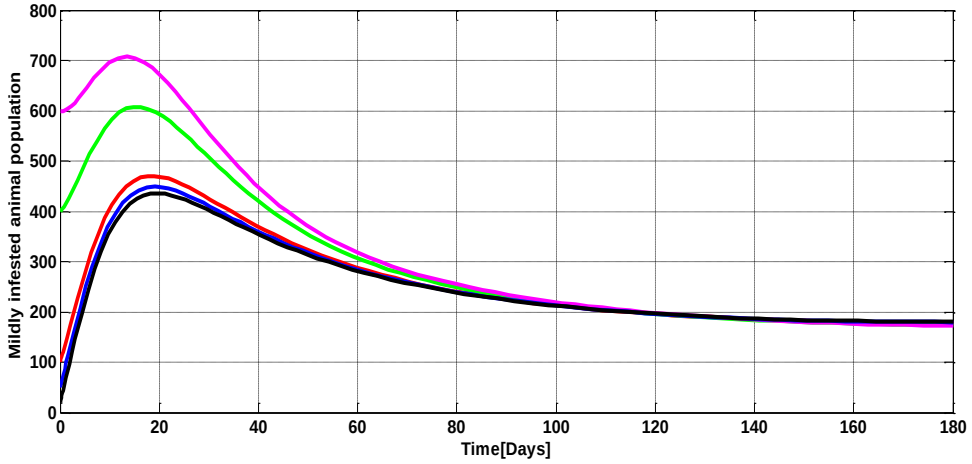

Figure 4(b): Stability of the endemic equilibrium for mildly infested animal population

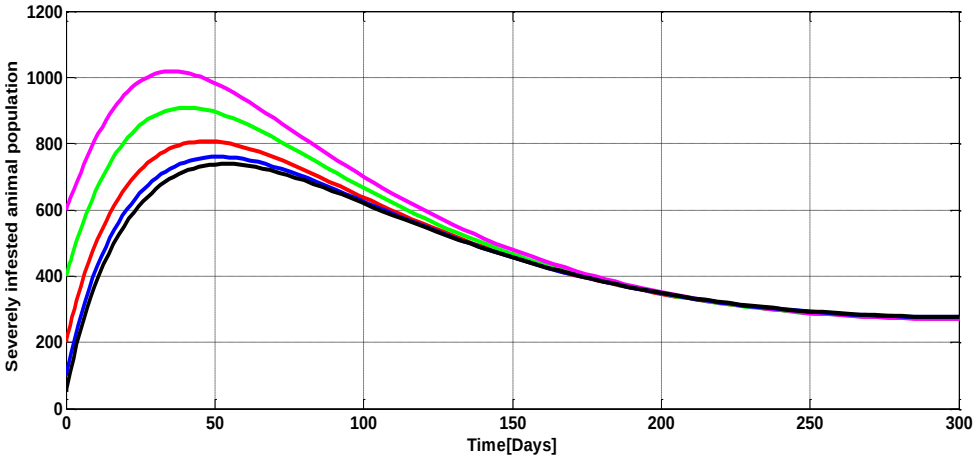

Figure 4(c): Stability of the endemic equilibrium for severely infested animal population

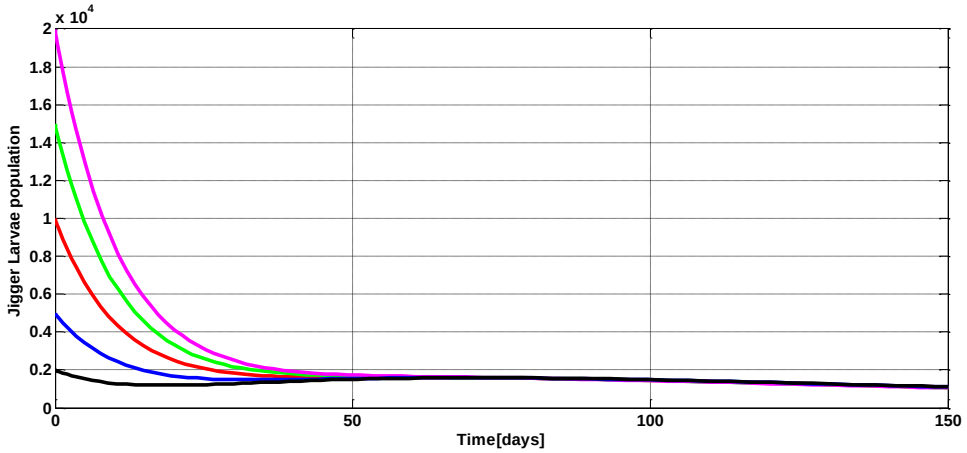

Figure 5(a): Stability of the endemic equilibrium for jigger larvae population.

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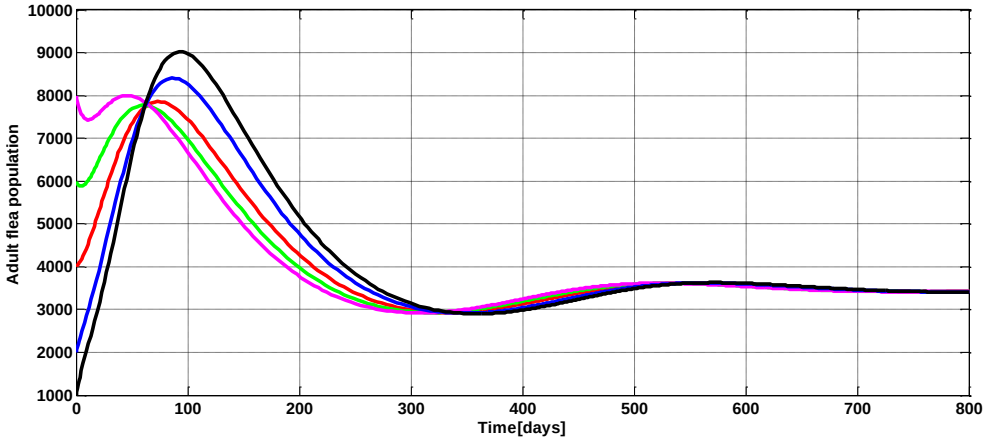

Figure 5(b): Stability of the endemic equilibrium for adult jigger flea population

6 Conclusions

The Tungiasis model with sub-models of humans, animal reservoirs and flea populations, has been developed and analyzed to study the tungiasis dynamics in endemic areas. The basic reproduction number R_0 computed was used to determine the stability of the disease-free equilibrium point (DFE). We then proved the existence and stability of endemic equilibrium point (EE) using Lyapunov direct Method combined with LaSalle's Invariance Principle. The analytical results show that the DFE is locally asymptotically stable, if $R_0 < 1$ and the EE is globally asymptotically stable when $R_0 > 1$. From numerical simulations we have observed that without intervention the populations vary for some time but ultimately approach the endemic equilibrium levels in the long run which imply the existence and stability of endemic equilibrium point.

This work provides a basic dynamical model that can be used to understand the transmission dynamics of tungiasis transmission that involves the interaction between human population, animal reservoir population and the flea infested environment. Understanding tungiasis transmission dynamics will help to design proper control strategies and evaluate their potential impact in reducing tungiasis morbidity and mortality in the endemic communities at large.

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