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MODELLING THE DYNAMICS OF TUNGIASIS TRANSMISSION IN ZOOBOTIC AREAS

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Abstract: In this paper we formulated a mathematical model for the dynamics of Tungiasis as a result of interactions between humans, animal reservoirs and sand flea populations. Tungiasis is a parasitic skin disease caused by the female flea “*Tunga penetrans*” that affect the economically disadvantaged communities in Latin America, the Caribbean and sub-Saharan Africa where prevalence is high and severe infestation occurs commonly. We obtained the basic reproduction number, R_0 which has been used to determine parameter sensitivity indices so that the key parameters for the control of the disease transmission are identified. The numerical results showed that the parameters with high impact on R_0 are; flea natural mortality rate μ_F , the contribution rate of fleas into the soil environment ε_A and the transmission rate between soil environment and susceptible animals β_{EA} . The numerical simulation showed that R_0 decreases with decreasing β_{EA} and ε_A and with increasing μ_F . Therefore to control the disease we should reduce the transmission rate between soil environment and susceptible animals by regular cleaning of the home compound, reducing the contribution rate of fleas into the environment by dusting the animals with insecticidal powder and increasing the flea mortality rate by spraying the insecticides into the premises.

Keywords: Tungiasis; basic reproduction number; sensitivity analysis; numerical simulation.

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1 Introduction

Tungiasis is an infestation caused by permanent penetration of the female sand flea "*Tunga penetrans*" into the body of the warm-blooded reservoir hosts [2], [10], this parasitic arthropod is normally found in most tropical and sub-tropical, sandy terrain of warm dry climates. It prefers deserts, beaches, stables, stock farms, and the soil dust close to farms [1]. The warm-blooded reservoir hosts include; humans, pigs, dogs, cats, rats, sheep, cattle, donkeys, monkeys, birds, and elephants [9], [22]. Originally, the ectoparasite was restricted to Latin America, South America and the Caribbean whereby between 18th and 19th century the parasite is said to have been stowed-away from Brazil to Angola in West Africa and eventually to other parts of sub-Saharan Africa [16], [26]. Currently Tungiasis is endemic in all tropical areas of the world including many countries in Latin America, the Caribbean and sub-Sahara Africa [13].

Transmission of Tungiasis occurs when susceptible hosts are in contact with sandy soil in which female fleas are present, also when the host is in contact with the infested animal reservoirs [14], [24]. The risk factors associated with Tungiasis infestation include: domestic animals in the homes perhaps because they harbour the *Tunga penetrans* fleas [20], [24]; poor housing which are dusty with cracks on the walls and with earthen floors, provides good breeding environment for *Tunga penetrans* [21]; and lack of regular use of closed foot wears are important factors for Tungiasis [20], [27]. Tungiasis results in significant morbidity, manifesting itself in a number of symptoms such as severe local inflammation, auto-amputation of digits, deformation and loss of nails, formation of fissures and ulcers, gangrene and walking difficulties [4]. Impaired physical fitness of adult household members has a negative impact on life quality, on household economics and immobility which may lead to stigmatization and social exclusion. Children with tungiasis usually show disproportionately high absenteeism at school and may be teased and ridiculed.

In economically disadvantaged communities Tungiasis prevalence may be up to 60% in the

general population and up to 80% in children [10]. The traditional treatment, i.e. removal of embedded sand fleas with sharp, non-sterile instruments may lead to transmission of blood-borne pathogens such as hepatitis B and C virus, possibly also HIV [11]. In domestic animals, Tungiasis affect weight gain, milk production and can lead to the death of offspring as a result of the mother's inability to nurse [29]. Due to multiple risk factors for Tungiasis its transmission is complex, and there is an overall lack of understanding of transmission dynamics at the population level and consequently the empirical data on its dynamics in most endemic areas is lacking. So far no mathematical models have been developed for the study of Tungiasis epidemics for the interactions between human hosts, reservoir animal hosts and the soil infested environment.

Many studies so far conducted focused on the knowledge pertaining the possible causes of Tungiasis, attitude and practices on the prevalence in different endemic communities without using mathematical analysis. Heukelbach *et al.* [15] conducted a study in order to understand the transmission of this parasitic skin disease in a typical endemic area, whereby a longitudinal study was carried out in Fortaleza north-eastern Brazil. The study shows that prevalence of Tungiasis and parasite burden vary significantly during the year with a peak in the dry season whereby, his findings showed the important consequences for the design of control measures. Pilger *et al.* [24] performed cross-sectional study to determine the role of animal reservoirs in human tungiasis, in a traditional fishing community in northeast Brazil whereby his study showed that tungiasis is highly prevalent in humans and domestic animals and concluded that animals should be included in control operation so as to reduce of disease burden in the human population. Ogbomoiko *et al.* [27] conducted a study on risk factors for Tungiasis transmission in endemic areas in Lagos Nigeria whereby individuals were examined clinically for the presence of tungiasis. His results identified the factors associated with Tungiasis including: sand or clay floor inside the house, resting usually outside the house, lack of regular use of closed footwear, and the presence of pigs on the compound. He concluded that, those are the important factors to be targeted for

intervention strategies.

In order to find an effective way to control (prevent and treat) Tungiasis, it is of great importance to establish its transmission dynamics. Mathematical dynamical models provide unique insights on the spread of diseases, relating important public-health questions to basic transmission parameters and can be used extensively in the study of ecological and epidemiological phenomena [17]. Mathematical models allows researchers to ask questions that cannot be addressed by other methods, for example, these models enable estimation of epidemiological parameters linked to key mechanisms, integration of data spanning multiple spatial scales, comparison of alternative control strategies, prediction of future trends, and explanation of observed patterns based on mechanistic hypotheses [18]. A comprehensive picture of disease dynamics requires a variety of mathematical tools, from model creation to solving differential equations and statistical analysis [23].

In this paper we provide the insights into the dynamics of Tungiasis transmission, we build a mathematical model that incorporates humans, animal reservoirs and the flea infested soil environment aiming at predicting typical infestation dynamics at the population level. Sensitivity analysis is performed to determine the sensitive parameters that could be targeted for implementation of control strategies. The rest of the paper is arranged as follows. In Section 2, we formulate a mathematical model and establish the basic properties of the model. In Section 3, we perform model analysis which includes; the disease-free equilibrium point and the basic reproduction number. In Section 4, we perform sensitivity analysis with its interpretations. In section 5, we perform numerical simulation and discuss the results and in section 6 is the conclusion.

2 Model formulation

The total human population at any time t , denoted by N_H is subdivided into three distinct

epidemiological sub-population namely susceptible humans S_H , people who are mildly infested by jiggers I_{HI} 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 $b_H N_H$. The natural death rate occurs in S_H , I_{HI} and I_{Hh} classes at a rate μ_H . Individuals in I_{Hh} compartment, suffers an additional death due to disease at a rate σ_H . S_H acquire infestation from the animal with severe infestation I_{Ah} and the environment with fleas F_E at a rate given by Ψ_H and move to either I_{HI} 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. Furthermore I_{HI} may acquire infestation from the environment as well and progresses to I_{Hh} at a rate $\alpha_{EH} \beta_{EH} r_F F_E / (k + F_E)$. The parameters ρ_{AH} and β_{EH} are the effective contact rates between susceptible humans and infested animal reservoirs and between susceptible human and the environment respectively. Ψ_H is the force of infestation for human population and is given by:

$$\Psi_H(I_{Ah}, F_E) = \left\{ \rho_{AH} I_{Ah}/N_H + \alpha_{EH} \beta_{EH} r_F F_E / (k + F_E) \right\} \quad (1)$$

Likewise the animal reservoir population denoted by N_A is also subdivided into three sub-population namely, the susceptible animals S_A , animals that are mildly infested by jiggers I_{AI} and animals that are severely infested by jiggers I_{Ah} . S_A is generated through birth at a rate b_A so that the animal recruitment is $b_A N_A$. The natural death occurs in S_A , I_{AI} and I_{Ah} classes at a rate μ_A . Individuals in I_{Ah} compartment suffers an additional death due to disease at a rate σ_A . S_A acquires infestation from the sources I_{Ah} and F_E classes at a rate given by

Ψ_A and move to either I_{AI} 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. Moreover I_{AI} may acquire infestation from the environment as well and progresses into I_{Ah} at a rate $\alpha_{EA}\beta_{EA}r_F F_E/(k + F_E)$. The parameters ρ_A and β_{EA} are the effective contact rates between susceptible and infested animal reservoirs and between the susceptible animal reservoir and the environment respectively. Ψ_A is the force of infestation for animal reservoir population and is given by:

$$\Psi_A(I_{Ah}, F_E) = \{\rho_A I_{Ah}/N_A + \alpha_{EA}\beta_{EA}r_F F_E/(k + F_E)\} \quad (2)$$

The environmental component consists of jigger larvae and adult jigger fleas' compartments denoted by L_E and F_E respectively. L_E is generated when jigger eggs are shed by the gravid female flea embedded into the human or animal reservoir hosts into the environment. Recruitment rate of larvae from eggs is given by $\delta_e(1 - L_E/K)$, where $K > 0$ is the jigger larvae environmental carrying capacity and δ_e is a constant rate of shedding eggs into the environment whereby, the contribution rates of jigger eggs into the environment are $\delta_e(1 - L_E/K)I_{Hh}$ and $\delta_e(1 - L_E/K)I_{Ah}$ for human and animal reservoir populations respectively. The eggs hatch into larvae and eventually mature into adult jigger flea. The jigger larvae are removed from class L_E as they die a natural death at a rate μ_L and as they mature at a rate γ_L to join another class F_E . F_E is generated through larvae maturation at a rate γ_L and from severely infested animal reservoirs as they shed fleas into the environment at a rate ε_A with contribution rate $\varepsilon_A I_{Ah}$. The jigger fleas are removed from class F_E at the rates $r_F F_E/(k + F_E)$ and μ_F as they burrow into the hosts and as they die naturally respectively. The burrowing rate of fleas into the humans and animal reservoirs hosts are $\alpha_{EH}r_F F_E/(k + F_E)$

and $\alpha_{EA} r_F F_E / (k + F_E)$ respectively. The model assumes that individuals mix homogeneously in the human and animal reservoir population and that transmission of the infestations occurs with a standard incidence and Holling type II functional response. Again the model assumes that for all three populations the individuals infested with Tungiasis do not recover naturally, the soil environment has a high concentration of fleas than animal reservoirs, and individuals in contact with infested environment will have severe infestation than when in contact with animal reservoirs. 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
$I_{HI}(t)$	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
$I_{AI}(t)$	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
$\mu_H, \mu_A, \mu_F, \mu_L$	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 into the environment by the severely infested animal reservoirs
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

2.1 Model flow chart

The interactions between humans, animal reservoirs and sand fleas contaminated soil environment are depicted in the compartmental diagram in Figure 1.

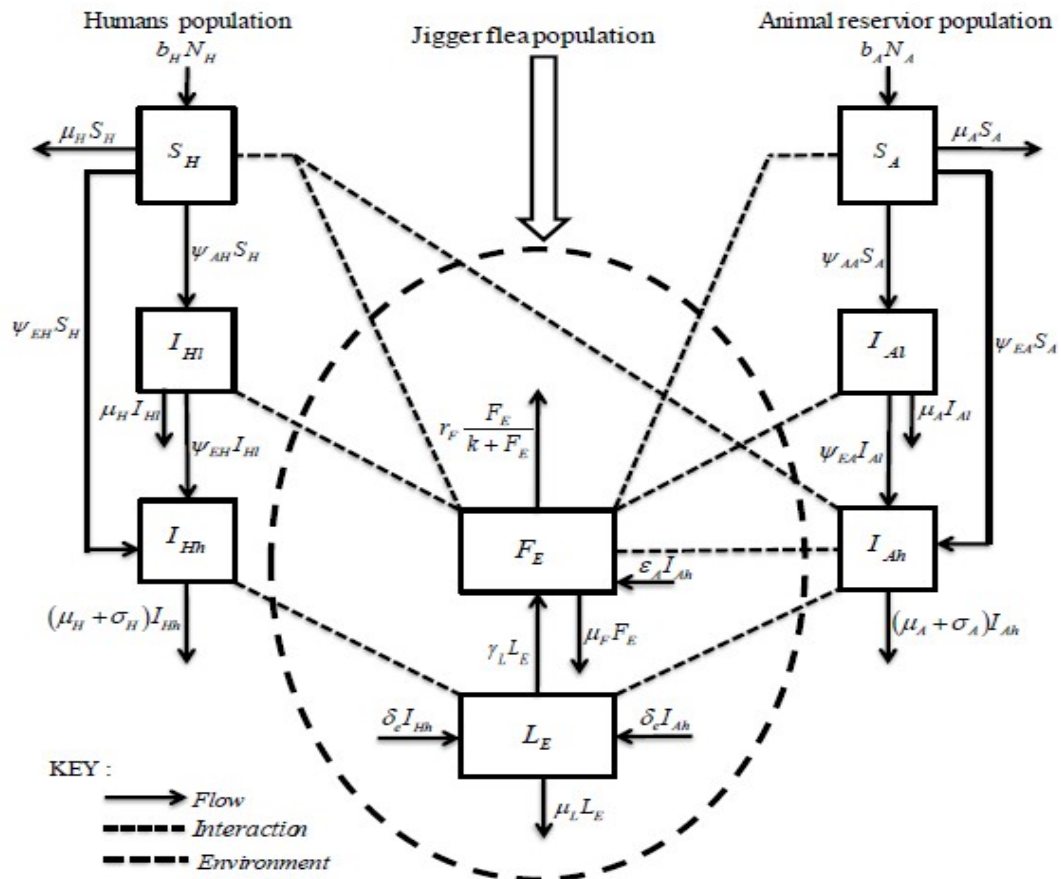


Figure 1: Tungiasis basic dynamical model

The model flow chart in Figure 1: Shows the dynamics of Tungiasis transmission that includes; susceptible human S_H , infested humans at mildly and severe states I_{Hl}, I_{Hh} , susceptible animal S_A , infested animals at mildly and severe states I_{Al}, I_{Ah} . L_E and F_E are the larvae and adult flea populations present in the soil environment. The dashed lines indicate contacts and the solid lines with arrow head indicate movements in and out of the compartments.

2.2 Model differential equations

The interactions between humans, animal reservoirs and jigger fleas contaminated environment are depicted in the compartmental diagram in Figure 1.

Dynamics in human population

$$\frac{dS_H(t)}{dt} = b_H N_H - \left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} \right) S_H - \mu_H S_H \quad (3a)$$

$$\frac{dI_{HI}(t)}{dt} = \rho_{AH} \frac{I_{Ah}}{N_H} S_H - \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} I_{HI} - \mu_H I_{HI} \quad (3b)$$

$$\frac{dI_{Hh}(t)}{dt} = \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} I_{HI} + \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} S_H - (\mu_H + \sigma_H) I_{Hh} \quad (3c)$$

Dynamics in animal population

$$\frac{dS_A(t)}{dt} = b_A N_A - \left(\rho_A \frac{I_{Ah}}{N_A} + \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E} \right) S_A - \mu_A S_A \quad (3d)$$

$$\frac{dI_{AI}(t)}{dt} = \rho_A \frac{I_{Ah}}{N_A} S_A - \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E} I_{AI} - \mu_A I_{AI} \quad (3e)$$

$$\frac{dI_{Ah}(t)}{dt} = \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E} S_A + \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E} I_{AI} - (\mu_A + \sigma_A) I_{Ah} \quad (3f)$$

Dynamics in Jigger flea population

$$\frac{dL_E(t)}{dt} = \delta_e \left(1 - \frac{L_E}{K} \right) (I_{Hh} + I_{Ah}) - (\gamma_L + \mu_L) L_E \quad (3g)$$

$$\frac{dF_E(t)}{dt} = \gamma_L L_E + \varepsilon_A I_{Ah} - \mu_F F_E - r_F \frac{F_E}{k + F_E} \quad (3h)$$

where

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

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

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

with initial conditions

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

2.3 Basic properties of the model

In this section, we determine the feasibility of the model, i.e. the invariant region and positivity of the solution. The invariant region of the model describes the region in which the solutions of the model system (3) are biological meaningful and the positivity describes non-negative solution of model system (3).

2.3.1 Invariant region

To test whether the model is well posed epidemiologically and mathematically, we need to investigate the feasibility of the model solution. Therefore we present the model system (3) in compact form as in equation (4). We adopt the approach used by Mpeshe *et al.* [19].

Lemma 1: *The model system (3) is well posed in the feasible region defined by:*

$$\Phi = \left\{ \begin{array}{l} (S_H, I_{HI}, I_{Hh}, S_A, I_{AI}, I_{Ah}, L_E, F_E) \in \mathfrak{R}_+^8 : S_H \leq N_H, I_{HI} \leq N_H, \\ I_{Hh} \leq N_H, S_A \leq N_A, I_{AI} \leq N_H, I_{Ah} \leq N_A, S_H + I_{HI} + I_{Hh} \leq N_H, \\ S_A + I_{AI} + I_{Ah} \leq N_A, L_E \leq K, F_E \leq \frac{\gamma_L}{\mu_F} K \end{array} \right\}$$

Proof:

We write the model system (3) in the form:

$$\frac{dX}{dt} = A(X)X + F \quad (4)$$

$$\text{where } X = (S_H, I_{HI}, I_{Hh}, S_A, I_{AI}, I_{Ah}, L_E, F_E)^T, \quad (5)$$

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

$$A(X) = \begin{bmatrix} -D_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_1 & -D_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ A_2 & A_3 & -D_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -D_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_4 & -D_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_5 & A_6 & -D_6 & 0 & 0 \\ 0 & 0 & \delta_e & 0 & 0 & \delta_e & -D_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon_A & \gamma_L & -D_8 \end{bmatrix} \tag{6}$$

where

$$\begin{aligned} D_1 &= \rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} + \mu_H, D_2 = \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} + \mu_H, D_3 = (\mu_H + \sigma_H), \\ D_4 &= \rho_A \frac{I_{Ah}}{N_A} + \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E} + \mu_A, D_5 = \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E} + \mu_A, D_6 = (\mu_A + \sigma_A), \\ D_7 &= \left\{ \frac{\delta_e}{K} (I_{Hh} + I_{Ah}) + (\gamma_L + \mu_L) \right\}, D_8 = \mu_F + \frac{r_F}{k + F_E}, A_1 = \rho_{EH} \frac{I_{Ah}}{N_H}, A_2 = \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E}, \\ A_3 &= \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E}, A_4 = \rho_A \frac{I_{Ah}}{N_A}, A_5 = \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E}, A_6 = \alpha_{EA} \beta_{EA} r_F \frac{F_E}{k + F_E}, \end{aligned}$$

and F is the column vector:

$$F = (b_H N_H, 0, 0, b_A N_A, 0, 0, 0, 0)^T \geq 0 \tag{7}$$

$A(X)$ is a Metzler matrix i.e. a matrix such that off diagonal terms (elements) are non-negative,

for all $X \in \mathfrak{R}_+^8$. Considering the fact that $F \geq 0$, the model system (3) is positively invariant in

\mathfrak{R}_+^8 and F is Lipschitz continuous. Thus the feasible region Φ for the model system is the set:

$$\Phi = \left\{ \begin{aligned} &(S_H, I_{HI}, I_{Hh}, S_A, I_{AI}, I_{Ah}, L_E, F_E) \in \mathfrak{R}_+^8 : S_H \leq N_H, I_{HI} \leq N_H, \\ &I_{Hh} \leq N_H, S_A \leq N_A, I_{AI} \leq N_H, I_{Ah} \leq N_A, S_H + I_{HI} + I_{Hh} \leq N_H, \\ &S_A + I_{AI} + I_{Ah} \leq N_A, L_E \leq K, F_E \leq \frac{\gamma_L}{\mu_F} K \end{aligned} \right\} \tag{8}$$

Therefore it can be verified that Φ is positively invariant with respect to model system (3), i.e. 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 .

2.3.2 Positivity of the solution

For model system (3) to be epidemiologically meaningful and well posed, we need to prove that all the state variables are non-negative for all $t > 0$

Lemma 2:

Let the initial data be $\{(S_H(0), I_{HI}(0), I_{Hh}(0), S_A(0), I_{AI}(0), I_{Ah}(0), L_E(0), F_E(0)) \geq 0\} \in \Phi$ then the solution set $\{S_H(t), I_{HI}(t), I_{Hh}(t), S_A(t), I_{AI}(t), I_{Ah}(t), L_E(t), F_E(t)\}$ of the model system (3) is non-negative for all time $t > 0$.

Proof:

From the first equation of the model system (3) we have:

$$\begin{aligned} \frac{dS_H}{dt} &= b_H N_H - \left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} \right) S_H - \mu_H S_H \\ \frac{dS_H}{dt} &\geq - \left(\rho_{AH} \frac{I_{Ah}}{N_H} + \alpha_{EH} \beta_{EH} r_F \frac{F_E}{k + F_E} + \mu_H \right) S_H \end{aligned} \quad (9)$$

Integrating equation (9) by separating the variables

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

Using the similar procedure it can also be shown that the remaining model variables

$S_H, I_{HI}, I_{Hh}, S_A, I_{AI}, I_{Ah}, L_E, F_E$ are also positive for all time $t > 0$.

Therefore the solution set $\Phi = \{S_H(t), I_{HI}(t), I_{Hh}(t), S_A(t), I_{AI}(t), I_{Ah}(t), L_E(t), F_E(t)\}$ of the model system (3) is non-negative for all $t \geq 0$. Thus the model is biologically and mathematically posed. Hence it is feasible to consider the dynamics of the three sub-models of humans, animal reservoirs and the soil environment.

3 Model analysis

In this section we conduct equilibrium analysis in order to investigate or address the existence of the disease free-equilibrium point of system (3) that will facilitate in the determination of the model basic reproduction number.

3.1 Disease free equilibrium point

The disease free equilibrium point is obtained by equating equations of the model system (3) to zero that means:

$$\frac{dS_H}{dt} = \frac{dI_{HI}}{dt} = \frac{dI_{Hh}}{dt} = \frac{dS_A}{dt} = \frac{dI_{AI}}{dt} = \frac{dI_{Ah}}{dt} = \frac{dL_E}{dt} = \frac{dF_E}{dt} = 0$$

The disease free equilibrium (DFE) point is the situation where there is no infestation in the population and environment, i.e. $I_{HI} = I_{Hh} = I_{AI} = I_{Ah} = F_E = 0$. Therefore the (DFE) point is given by:

$$\Phi^o = (S_H^o, I_{HI}^o, I_{Hh}^o, S_A^o, I_{AI}^o, I_{Ah}^o, L_E^o, F_E^o) = \left(\frac{b_H N_H}{\mu_H}, 0, 0, \frac{b_A N_A}{\mu_A}, 0, 0, 0, 0 \right) \quad (10)$$

3.2 The basic reproduction number

The basic reproduction number R_o is defined as the *expected* number of secondary infections that one infectious individual would cause over the duration of the infectious period in a fully susceptible population [8]. Its computation is based on the next generation operator technique described by Van de Driessche and Watmough [28]. Given the model system (3) we let $g_i(x)$

be the rate of appearance of new infestations of individuals into compartment i and $v_i(x)$ be the rate of transfer of individuals in and out of compartment i by all other means other than epidemic. Therefore at disease free equilibrium the Jacobian matrices for $g_i(x)$ and $v_i(x)$ are respectively given by

$$G = \frac{\partial g_i}{\partial x_j}(\Phi^o) \text{ and } V = \frac{\partial v_i}{\partial x_j}(\Phi^o) \text{ with } 1 \leq i, j \leq 5 .$$

The basic reproduction number, R_o of the model system (3) is given by the spectral radius or (largest eigenvalue) of the next generation matrix GV^{-1} . i.e. $R_o = \rho(GV^{-1})$. The Jacobian matrices G and V in this case are as given in equations (11) and (12) respectively.

$$G = \begin{bmatrix} 0 & 0 & 0 & \frac{\rho_{AH}b_H}{\mu_H} & 0 \\ 0 & 0 & 0 & 0 & \frac{\alpha_{EH}\beta_{EH}r_F b_H N_H}{k\mu_H} \\ 0 & 0 & 0 & \frac{\rho_A b_A}{\mu_A} & 0 \\ 0 & 0 & 0 & 0 & \frac{\alpha_{EA}\beta_{EA}r_F b_A N_A}{k\mu_A} \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{11}$$

$$V = \begin{bmatrix} \mu_H & 0 & 0 & 0 & 0 \\ 0 & \mu_H + \sigma_H & 0 & 0 & 0 \\ 0 & 0 & \mu_A & 0 & 0 \\ 0 & 0 & 0 & \mu_A + \sigma_A & 0 \\ 0 & 0 & 0 & -\varepsilon_A & \mu_F + \frac{r_F}{k} \end{bmatrix} \tag{12}$$

We compute the next generation matrix GV^{-1} which is the product of two Jacobian matrices G and V^{-1} where G is nonnegative and V is a nonsingular matrix. We get,

$$GV^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{\rho_{AH}b_H}{\mu_H(\mu_A + \sigma_A)} & 0 \\ 0 & 0 & 0 & \frac{\alpha_{EH}\beta_{EH}r_F N_H b_H \varepsilon_A}{\mu_H(\mu_A + \sigma_A)(\mu_F k + r_F)} & \frac{\alpha_{EH}\beta_{EH}r_F N_H b_H}{\mu_H(\mu_F k + r_F)} \\ 0 & 0 & 0 & \frac{\rho_A b_A}{\mu_A(\mu_A + \sigma_A)} & 0 \\ 0 & 0 & 0 & \frac{\alpha_{EA}\beta_{EA}r_F N_A b_A \varepsilon_A}{\mu_A(\mu_A + \sigma_A)(\mu_F k + r_F)} & \frac{\alpha_{EA}\beta_{EA}r_F N_A b_A}{\mu_A(\mu_F k + r_F)} \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad (13)$$

Therefore, according to the definition of the basic reproduction number, R_o , we have:

$$R_o = \frac{\alpha_{EA}\beta_{EA}N_A b_A \varepsilon_A}{\mu_A(\mu_A + \sigma_A)} \left(\frac{r_F}{\mu_F k + r_F} \right) \quad (14)$$

The basic reproduction number R_o for the model system (3) is independent of the human population N_H but only depends on the animal reservoir population N_A and the density of jigger fleas in the environment F_E making the human beings victims of the Tungiasis epidemic. Increasing the shedding rates ε_A , animal to environment effective contact rate β_{EA} , removal rate of jigger fleas from the environment that attack the hosts r_F , the animal reservoir population N_A , animal birth rate b_A and the proportion of sand fleas that infest animal reservoirs α_{EA} , the model's basic reproduction number R_o increases. Increasing animal natural mortality rate μ_A , the disease induced animal mortality rate σ_A , the sand flea mortality rates μ_F and the environmental jigger fleas' carrying capacity k lowers the model's basic reproduction number. Therefore the control measures against the off-host life stages of jigger fleas and On-host treatment of domestic animals should be targeted. This underlines the importance to include domestic animals in control operation aiming at the reduction of disease occurrence in the human population.

4 Sensitivity analysis and numerical simulations

In this section, we conduct sensitivity analysis of the basic reproduction number in order to investigate the parameters with high sensitivity indices and then we perform numerical simulation to determine their effect on the basic reproduction number.

4.1 Sensitivity of the basic reproduction number

Sensitivity analysis shows the impact of each parameter to the disease transmission whereby each parameter is investigated with respect to the basic reproduction number. It is commonly used to determine the robustness of model predictions to parameter values [7]. To determine how best we can reduce the mortality and morbidity due to Tungiasis infestation, it is necessary to study the relative importance of different factors responsible for its transmission and prevalence [3]. Normally we perform sensitivity analysis to determine the parameters that have a high impact on the basic reproduction number R_0 that should be targeted by intervention strategies.

4.2 Parameter Estimation

The flea dies around day 25 post-penetration [9], we therefore assume that its life span 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 half saturation constant k to be 10,000 cell/m³. The disease induced death rate for animal reservoirs σ_A is estimated to be 0.037 per day. The natural death rate of animal reservoir μ_A ranges from (360–3600)days [12], [25]. The value of effective contact rates β_{EA} is estimated to be 0.48 per day and the rate of shedding of fleas ε_A is estimated to be 0.4 per day. The parameters from literatures and others are estimated are shown in Table 3.

Table 3: Parameter values used for the simulations and sensitivity analysis

Parameter	Values /Range	Source (References)
k	1×10^4 cell/m ³	Estimated
σ_A	0.037 per day	Estimated
μ_A	$0.0028(360 - 3600)^{-1}$ per day	[12], [25]
μ_F	0.04 per day	[9]
r_F	0.58 per day	Estimated
β_{EA}	0.48 per day	Estimated
b_A	0.022 per day	[12]
ε_A	0.40 per day	Estimated
α_{EA}	0.60	Estimated
N_A	1200	Estimated

4.3 Determination of Sensitivity indices

The sensitivity indices measures the relative change in a state variable when a parameter changes. In computing the sensitivity indices, we use methods described by Chowell *et al.* [5] to derive an analytical expression for the sensitivity of R_0 to each parameter using the normalized forward sensitivity index of a variable with respect to a parameter which is the ratio of the relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives. The normalized forward sensitivity index of a variable that depends differentiably on a parameter is defined by Chitnis *et al.* [6]. These indices tell how vital each parameter is to disease transmission, epidemicity and prevalence.

Definition: The normalized forward sensitivity index of a variable with respect to a parameter is the ratio of the relative change in the variable R_0 to the relative change in the parameter p_i , is defined as

$$X_{p_i}^{R_0} = \frac{\partial R_0}{\partial p_i} \times \frac{p_i}{R_0} \quad (15)$$

We derive an analytical expression for the sensitivity of R_0 as in equation (15) to each of parameters involved in the basic reproduction number. The sensitivity indices of R_0 with respect to the parameters of the basic reproduction number are computed. Let us take an example of the parameters; b_A , β_{EA} , ε_A and N_A to compute sensitivity indices of the basic reproduction number as indicated in (16):

$$\begin{cases} X_{b_A}^{R_0} = \frac{\partial}{\partial b_A}(R_0) \times \frac{b_A}{R_0} = +1, & X_{N_A}^{R_0} = \frac{\partial}{\partial N_A}(R_0) \times \frac{N_A}{R_0} = +1 \\ X_{\varepsilon_A}^{R_0} = \frac{\partial}{\partial \varepsilon_A}(R_0) \times \frac{\varepsilon_A}{R_0} = +1, & X_{\beta_{EA}}^{R_0} = \frac{\partial}{\partial \beta_{EA}}(R_0) \times \frac{\beta_{EA}}{R_0} = +1 \end{cases} \quad (16)$$

Using the same procedure as in (16) the remaining indices $X_{\alpha_{EA}}^{R_0}$, $X_{r_F}^{R_0}$, $X_{\mu_A}^{R_0}$, $X_{\sigma_A}^{R_0}$, $X_{\mu_F}^{R_0}$ and $X_k^{R_0}$ are obtained and all the indices together are tabulated accordingly as indicated in Table 4.

Table 4: Sensitivity indices of R_0 evaluated at the baseline parameter values

Parameter symbol	Sensitivity index	Parameter symbol	Sensitivity index
b_A	+1.0000000000	k	-0.9985520995
μ_F	-0.9985520995	r_F	0.9985521005
α_{EA}	+1.0000000000	β_{EA}	+1.0000000000
N_A	+1.0000000000	ε_A	+1.0000000000
μ_A	-1.070351759	σ_A	-0.9296482412

5 Numerical simulation and discussion of the results

In this section the sensitivity indices are interpreted in order to investigate those parameters which are more positive than others and those which are more negative than others, so that the most sensitive parameters are obtained. We then plot the graphs to illustrate how the basic reproduction number R_0 varies with respect to the model sensitive parameters.

5.1 Interpretation of sensitivity indices

From Table 4, the sensitivity indices show that when the parameters $\alpha_{EA}, \beta_{EA}, b_A, N_A, \varepsilon_A$ and r_F are increased while the other parameters remain constant the value of R_0 increases implying that they increase the endemicity of the disease as they have positive indices. When the parameters μ_A, σ_A, μ_F and k are increased while keeping other parameters constant, the value of R_0 decreases implying that they decrease the chances of endemicity of the disease as they have negative indices. Analytically and by considering biological meaning of the model parameters we have observed that the basic reproduction number is more sensitive to parameters $\beta_{EA}, \varepsilon_A$ and μ_F . These parameters should be the main targets for designing control strategies. Therefore to control the disease we target to reduce the effective contact rate between animal reservoir and the environment β_{EA} , to reduce the rate of addition of sand fleas ε_A to the environment due to shedding by infested animal reservoirs I_{Ah} and to increase the mortality rate of jigger fleas μ_F in the environment. Despite the case that other parameters like α_{EA}, b_A, N_A followed by r_F are also more positive and parameters like μ_A, k followed by σ_A are more negative, they are not selected because it is difficult, impractical and meaningless to use them for designing the control strategies so they are ignored.

5.2 Numerical simulation of sensitive parameters

Figures 2, 3 and 4 illustrate the numerical simulation results of the basic reproduction number R_0 with respect to the model parameters, to show how R_0 varies with respect to the model parameters β_{EA} , μ_F and ε_A .

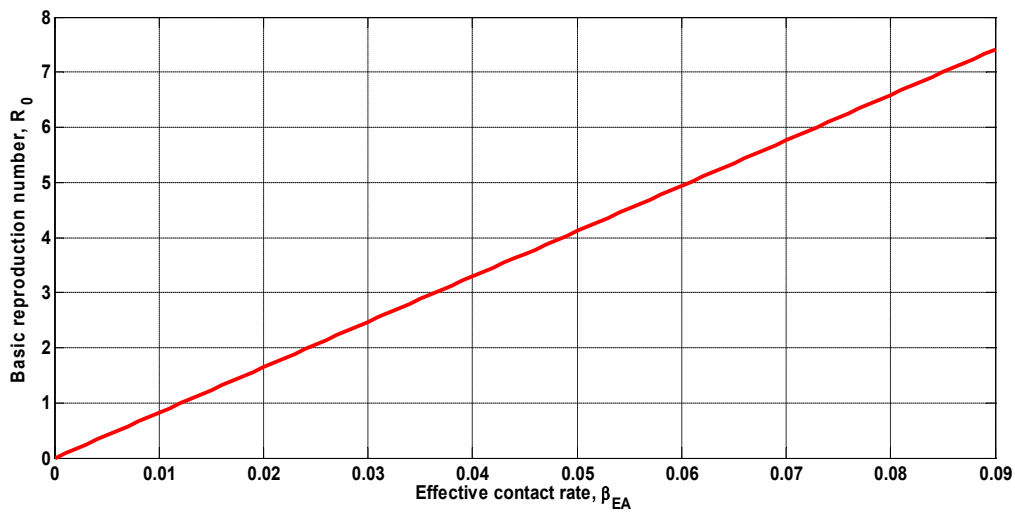


Figure 2: The effect of effective contact rate, β_{EA} on the basic reproduction number, R_0 .

In Figure 2: The positive sign of the sensitivity index of R_0 with respect to β_{EA} implies a 10% linear increase in β_{EA} leads to a 10% increase in R_0 and vice versa.

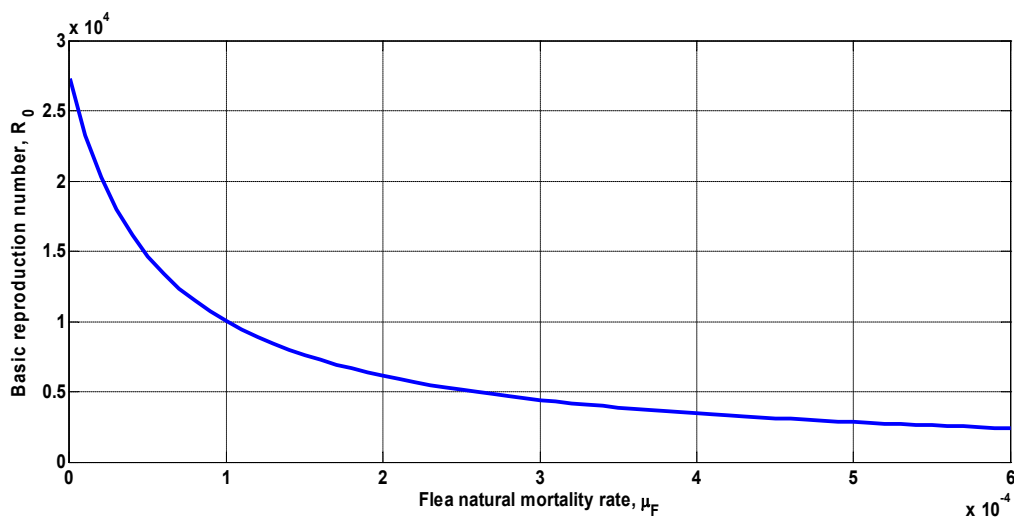


Figure 3: The effect of flea natural mortality rate, μ_F on the basic reproduction number R_0

In Figure 3: The negative sign of the sensitivity index of R_0 with respect to μ_F implies a 10% increase in μ_F leads to approximately 9.9% exponential decrease in R_0 and vice versa.

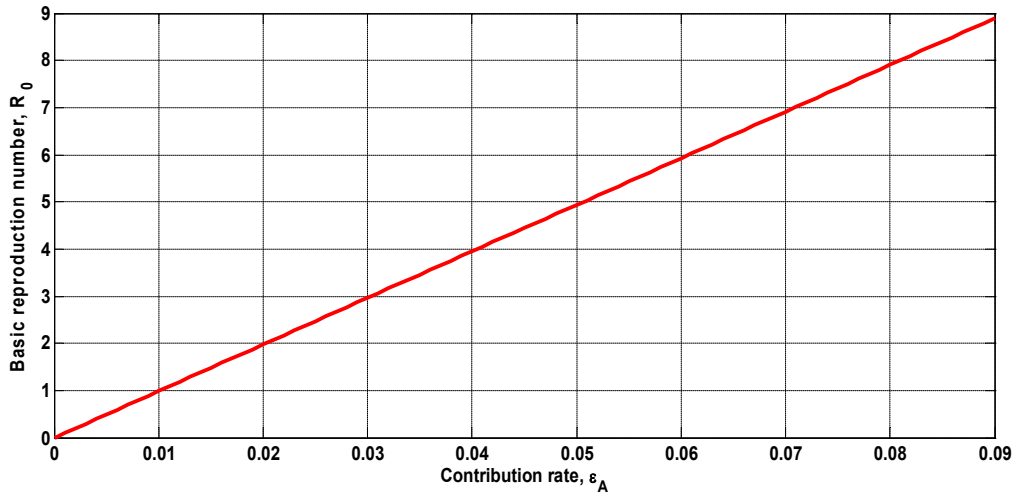


Figure 4: The effect of flea contribution rate, ε_A on the basic reproduction number R_0

In Figure 4: The positive sign of the sensitivity index of R_0 with respect to ε_A implies a 10% increase in the shedding rate ε_A leads to a 10% linear increase in R_0 and vice versa.

6 Conclusion

In this paper we have formulated and analyzed a deterministic mathematical model for the dynamics of Tungiasis. The model comprises the interactions between humans, animal reservoirs and the sand fleas in the environment. We derived an explicit formula for basic reproductive number R_0 using the next generation operator method and used it in the determination of sensitivity indices of parameters. The analytical results showed that the effective contact rate between the soil environment and the susceptible animal denoted by β_{EA} , the mortality rate of the sand flea denoted by the μ_F and the rate at which the fleas are shed by the severely infested animal reservoirs denoted by ε_A are more sensitive parameters than others. The numerical simulation show that reducing the effective contact rate β_{EA} , increasing the natural death rate of

fleas μ_F and decreasing the contribution rate of fleas ε_A into the environment lowers the model basic reproduction number R_0 which implies reduced endemicity of the disease. Therefore if policymakers wish to decrease the probability of a large transient Tungiasis epidemic, reducing on and off-host flea population and the effective contact rate between the sand flea sources and the hosts will be an effective intervention strategy. This work provides for the sensitivity analysis of the model parameters that could be used for studies to determine stability analysis and designing of control strategies.

Conflict of Interests

The authors declare that there is no conflict of interests.

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