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Mathematical Modelling of Tungiasis Disease Dynamics Incorporating Hygiene as a Control Strategy

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Authors' contributions

This work was carried out in collaboration between both authors. Author IC designed the study and wrote the first draft of the manuscript. All authors managed literature searches. Author FKM performed the Mathematical analysis of the study and the simulations. Both authors read and approved the final manuscript.

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Abstract

Tungiasis is a disease that mostly affects the children, the disabled, alcoholics and the aged in Kenya and other parts of the world. Despite the intensive research that has been done on tungiasis disease, the disease remains a threat in Muranga County. In this research, we formulated a model which is mathematical in nature and derived a system of ordinary differential equations from it, which we used to study the dynamics of tungiasis disease, incorporating proper hygiene as a control measure. The basic reproduction number, R_0, is calculated using the next generation matrix. We determined the equilibrium points of the model and also carried out their stability analysis. From stability, both disease free equilibrium and endemic equilibrium points of the model were found to be locally asymptotically stable when $R_0 < 1$ and $R_0 > 1$ respectively. Numerical simulation of the model carried out showed that effective proper hygiene leads to a faster decrease in the spread of tungiasis.

Keywords: Tungiasis; hygiene; reproduction number; stability; numerical simulation.

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1 Introduction

Tungiasis is a skin infection which is parasitic in nature. It is caused by the female sand flea tunga penetrans. Prevalence is high in the economically disadvantaged communities, especially in the Caribbean, Latin America and Sub Saharan Africa. In Kenya, tungiasis is an important but neglected health problem. The areas mostly affected by the disease include central Kenya, western Nyanza, coastal and western regions. In Muranga county by 2010, more than 1358 people from just one division were suffering from tungiasis, out of which 700 were school going children from 13 primary schools. This is according to Ahadi Kenya, [1].

Tungiasis has mostly been associated with household poverty. The Tunga penetran infestations depends on the familys ability to access clean water, sanitation, good quality housing and good nutrition. Tungiasis therefore is linked to poor hygiene, sanitation education, poverty and waste disposal methods used in the villages. It mostly affects the children, the aged, alcoholics and people with disabilities. Many affected children drop out of school due to tungiasis. This is due to the fact that the children are unable to walk to school, and they also face the challenge of discrimination and stigmatization. Towards the end of the last century, there is an observer who termed jiggers as the most fearful calamity that has ever afflicted the East African peoples after seeing affected people groaning with pain and crawling around on all fours on the slopes of Mount Kilimanjaro as recorded in the MOH Kenya Policy, [2].

Feldmeier et al. [3] did a research on Tungiasis as a neglected disease with many challenges, and concluded that Tungiasis has an important social dimension, and affects human rights, and that appropriate strategies should be formulated to address this debilitating and mutilating parasitic skin disease that has unnecessarily plagued disadvantaged communities for centuries.

Kiragu [4] did a research on the efficacy of coconut oil in the control of tungiasis. He concluded that there is a strong relationship between infestation rate and the disease morbidity. Application of coconut oil reduced both the number of embedded fleas as well as the rate of infection. This is a clear indication that if serious protection measures are put into place, the rate of tungiasis infection can reduce, hence the need to consider hygiene.

Nthiiri [5] carried out a research on mathematical modelling of jigger infection incorporating treatment as a control strategy. Her findings were that effective treatment of jigger infection prevents rapid progression of this infection. She further recommended protection measures like wearing of shoes and watering of dusty floors. This two recommendations are incorporated in hygiene as a control strategy.

Kahuru et al. [6] carried out a research on modelling the dynamics of Tungiasis transmission in zoonotic areas. The research concentrated on the interactions between sand fleas, humans and animal reservoirs. According to the findings, reducing the effective rate of contact between soil environment and the susceptible animals, increasing the natural death rate of fleas and decreasing the contribution rate of fleas lowers the basic model reproduction number. This translates to reduced disease intensity.

Kahuru et al. [7] carried out a research on optimal control techniques on a mathematical model for the dynamics of Tungiasis in a community. The findings indicate that controlling of infested soils and animal reservoirs with insecticides, environmental hygiene and cementing floors of houses may serve as a possible approach to control Tungiasis infestation. In this research we look in to hygiene (both environmental and personal) as a major component, since it carries a lot of weight in the fight against the spread of tungiasis disease.

In view of all the above research work done, it is evident that a lot of research needs to be done on protection. In this research, we carried out a study on mathematical modelling of tungiasis disease incorporating hygiene as a control strategy. We modified work done by Nthiiri [5], by researching on her recommendation of observing cleanliness.

2 Model Formulation

The model is formulated where the total population is generally in four categories. These include; proper hygiene practice group (P), the susceptible group (S), the infected group (I) and the treated group (T). This implies that the total population, N, at any time t, is given by $N(t) = P(t) + S(t) + I(t) + T(t)$. The proper hygiene practice group is recruited at birth at a rate $\delta\psi$ while the susceptible group is recruited at a rate $(1-\delta)\psi$, where ψ is the rate of recruitment at birth, and δ is the probability of getting recruited into the class of proper hygiene practice. The proper hygiene practice group (P) become susceptible(S) at a rate π. Then upon infection, the susceptible group (S) move to the infected group (I) at a rate α. After receiving treatment, the infected people (I) move to the treated group (T) at a rate β. All individuals in each compartment experience natural death at a rate ϵ. This rate is proportional to the number of individuals in each compartment. The rate of infection, α, is defined as $\alpha = \frac{\kappa c I}{N}$. Where κ is the probability of being infected following prolonged contacts with individuals who are infected, and c is the contact rate with individuals who are infected.

Assumptions of the Model are:

i. Human birth and natural death takes place at different rates.
ii. Only susceptible individuals get infected.
iii. There is permanent immunity on recovery.
iv. Infected individuals die from natural or disease induced death.

Based on the above description and assumptions we obtain the flow chart below;

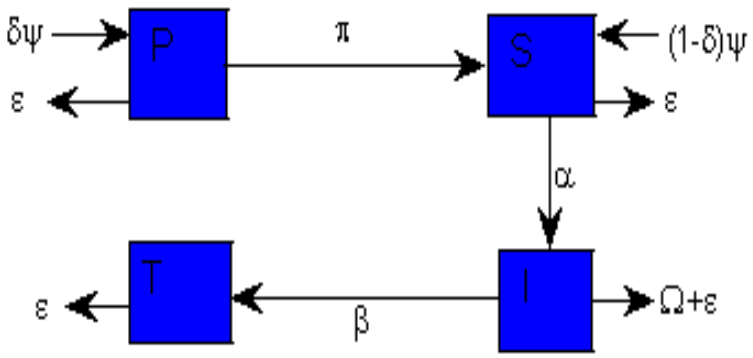

Fig. 1. Flow chart

From Fig. 1, we derive the following differential equations of the model with non-negative initial conditions.

$$\begin{aligned}
\frac{dP}{dt} &= \delta\psi - (\pi+\epsilon)P \\
\frac{dS}{dt} &= (1-\delta)\psi + \pi P - (\alpha+\epsilon)S \\
\frac{dI}{dt} &= \alpha S - (\Omega+\beta+\epsilon)I \\
\frac{dT}{dt} &= \beta I - \epsilon T
\end{aligned} \tag{2.1}$$

3 Model Analysis

Since the system (2.1) describes human population, all the solutions of state variable with non-negative initial conditions are non-negative $\forall\ t > 0$ and they are bounded in the feasible region $\Phi = \{(P,S,I,T) \in \mathbb{R}^4_+; S > 0; P,I,T, \geq 0; N \leq \frac{\psi}{\epsilon}\}$

3.1 Existence of equilibrium eoints

In this section we calculate the equilibrium points of system (2.1); disease free equilibrium (DEE) and endemic equilibrium (EE).

To obtain the equilibrium points for the model, we set the right hand side to zero, that is

$$\begin{aligned}
\delta\psi - (\pi+\epsilon)P &= 0 \\
(1-\delta)\psi + \pi P - (\alpha+\epsilon)S &= 0 \\
\alpha S - (\Omega+\beta+\epsilon)I &= 0 \\
\beta I - \epsilon T &= 0
\end{aligned} \tag{3.1}$$

Where $\alpha = \frac{\kappa c I}{N}$

To determine the DFE (E^0), we substitute $P = P^0 = 0$, $S = S^0 = N^0 = N$, $I = I^0 = 0$ and $T = T^0 = 0$ in system (2.1) to obtain

$(1-\delta)\psi - (\alpha+\epsilon)S^0 = 0$

Implying that $S^0 = \frac{(1-\delta)\psi}{(\alpha+\epsilon)}$

Thus the DFE is $E^0(0, \frac{(1-\delta)\psi}{(\alpha+\epsilon)}, 0, 0)$

Next, to determine the EE (E^*), first, we substitute $\alpha = \frac{\kappa c I}{N}$ in system (3.1) followed by $E^*(P^*, S^*, I^*, T^*)$ and $N = N^*$, to get

$$\begin{aligned}
\delta\psi - (\pi+\epsilon)P^* &= 0 \\
(1-\delta)\psi + \pi P^* - (\frac{\kappa c I^*}{N^*} + \epsilon)S^* &= 0 \\
\frac{\kappa c I^* S^*}{N^*} - (\Omega+\beta+\epsilon)I^* &= 0 \\
\beta I^* - \epsilon T^* &= 0
\end{aligned} \tag{3.2}$$

Solving for P^*, S^*, I^* and T^*, we obtain

$$E^*\begin{pmatrix} P^* \\ S^* \\ I^* \\ T^* \end{pmatrix} = E^*\begin{pmatrix} \frac{\delta\psi}{\epsilon+\pi} \\ \frac{N^*(\epsilon+\Omega+\beta)}{\kappa c} \\ \frac{1}{(\epsilon+\Omega+\beta)}\left[\frac{(\epsilon+\pi-\delta\epsilon)\psi}{(\epsilon+\pi)} - \frac{\epsilon N^*(\epsilon+\Omega+\beta)}{\kappa c}\right] \\ \frac{\beta}{\epsilon(\epsilon+\Omega+\beta)}\left[\frac{(\epsilon+\pi-\delta\epsilon)\psi}{(\epsilon+\pi)} - \frac{\epsilon N^*(\epsilon+\Omega+\beta)}{\kappa c}\right] \end{pmatrix}$$

where $N^* = P^* + S^* + I^* + T^*$

3.2 The basic reproduction number

The basic reproduction number, R_0 is the average number of secondary infections caused by a single infectious individual during his/her entire lifetime as an infective, in a purely susceptible population. Using the next generation matrix method by [8], R_0 is the spectral radius of the matrix FV^{-1}. Where F is the Jacobian of f_j, where f_j is the rate of appearance of new infections in compartment j, and V is the Jacobian of v_j, where v_j is the rate of transfer out of compartment j. R_0 is important in that it is directly related to the effort required to eliminate infection. The larger the R_0 number, the harder it is to eliminate infection and vice versa. The infected class is given by the third equation of system (2.1)

$$\frac{dI}{dt} = \alpha S - (\Omega + \beta + \epsilon)I \tag{3.3}$$

From equation (3.3), we have

$f_j = \alpha S = \frac{\kappa c I S}{N}$ and $v_j = (\Omega + \beta + \epsilon)I$

which can be expressed as

$F = \kappa c$ and $V = \Omega + \beta + \epsilon$

Hence

$FV^{-1} = \frac{\kappa c}{\Omega+\beta+\epsilon}$

or

$$R_0 = \frac{\kappa c}{\Omega + \beta + \epsilon} \tag{3.4}$$

3.3 Local stability of the Disease-free Equilibrium (DFE)

In this section we investigate the stability of its disease free equilibrium of system (2.1).

Theorem 1. *The disease free equilibrium E^0 of the model is locally asymptotically stable whenever $R_0 < 1$.*

Proof. The Jacobian matrix of system (2.1) is given by

$$J = \begin{bmatrix} -(\pi+\epsilon) & 0 & 0 & 0 \\ \pi & -\left(\epsilon + \frac{\kappa c I}{N}\right) & \frac{-\kappa c S}{N} & 0 \\ 0 & \frac{\kappa c I}{N} & \frac{\kappa c S}{N} - (\epsilon + \Omega + \beta) & 0 \\ 0 & 0 & \beta & -\epsilon \end{bmatrix}$$

At DFE, the Jacobian matrix becomes

$$J(E^0) = \begin{bmatrix} -(\pi+\epsilon) & 0 & 0 & 0 \\ \pi & -\epsilon & -\kappa c & 0 \\ 0 & 0 & \kappa c - (\epsilon+\Omega+\beta) & 0 \\ 0 & 0 & \beta & -\epsilon \end{bmatrix}$$

Upon calculation of the trace and determinant of $J(E^0)$, we obtain

$Tr(J(E^0)) = -(\pi+\epsilon) - 2\epsilon + \kappa c - (\epsilon+\Omega+\beta)$

$Det(J(E^0) = -\epsilon^2(\pi+\epsilon)(\kappa c - (\epsilon+\Omega+\beta))$

clearly $Tr(J(E^0)) < 0$ and $Det(J(E^0)) > 0$ for $R_0 < 1$ i.e $\kappa c < (\epsilon+\Omega+\beta)$

Hence by Routh-Hurwitz criteria, the DFE is locally asymptotically stable. □

3.4 Local stability of the endemic equilibrium (EE) of the model

If a disease persists in a population, it is said to be endemic

Theorem 2. *The endemic equilibrium of the model is locally asymptotically stable whenever* $R_0 > 1$

Proof. We use the Routh-Hurwitz criterion to prove this theorem. The Jacobian matrix at E^* is given by

$$J(E^*) = \begin{bmatrix} -(\pi+\epsilon) & 0 & 0 & 0 \\ \pi & -\left(\epsilon + \frac{\kappa c X}{N^*}\right) & -(\epsilon+\Omega+\beta) & 0 \\ 0 & \frac{\kappa c X}{N^*} & 0 & 0 \\ 0 & 0 & \beta & -\epsilon \end{bmatrix}$$

Where

$X = \frac{1}{(\epsilon+\Omega+\beta)}\left[\frac{(\epsilon+\pi-\delta\epsilon)\psi}{(\epsilon+\pi)} - \frac{\epsilon N^*(\epsilon+\Omega+\beta)}{\kappa c}\right]$

The characteristic equation is given by

$(\pi+\epsilon+\lambda)(\epsilon+\lambda)[\lambda^2 + \left(\epsilon + \frac{\kappa c X}{N^*}\right)\lambda + \frac{\kappa c(\epsilon+\Omega+\beta)X}{N^*}] = 0$

Clearly the first two eigenvalues are $\lambda_1 = -(\pi+\epsilon)$ and $\lambda_2 = -\epsilon$. The rest are given by

$$\lambda^2 + \left(\epsilon + \frac{\kappa c X}{N^*}\right)\lambda + \frac{\kappa c(\epsilon+\Omega+\beta)X}{N^*} = 0 \tag{3.5}$$

Upon substitution for X in equation (3.5), we obtain

$$\lambda^2 + a\lambda + b = 0 \tag{3.6}$$

$a = \frac{R_0(\epsilon+\pi-\delta\epsilon)\psi}{N^*(\epsilon+\pi)}$

$b = \frac{\kappa c(\epsilon+\pi-\delta\epsilon)\psi}{N^*(\epsilon+\pi)} - \epsilon(\epsilon+\Omega+\beta)$

clearly $a, b > 0$ for $R_0 = \frac{\kappa c}{\Omega+\beta+\epsilon} > 1$

Using Routh-Hurwitz criterion for a polynomial of degree two, the eigenvalues given by equation (3.6) are negative. Thus the EE of the model is locally asymptotically stable whenever $R_0 > 1$. □

4 Numerical Simulation

Numerical simulations are carried out to investigate the effect of proper hygiene practice on the dynamics of tungiasis infection. This was done with the help of MATLAB by using the parameter values in Table 1 below.

Table 1. Parameter values of the model

Parameter description	Symbol	Value	Source
Recruitment rate	ψ	0.0044	[9]
Natural mortality rate	ϵ	0.016	[9]
Disease induced mortality rate	Ω	0.005	Estimated
Loss of protection rate	π	0.001	Estimated
Transmission probability rate of tungiasis	κ	0.0011	Estimated
Contact rate of infection	c	0.0002	Estimated
Adjustment parameter	δ	$0 < \delta < 1$	Assumed
Rate of treatment	β	0.9	Estimated

Fig. 2. shows the effect of proper hygiene practice on infectious individuals at different rates of recruitment to proper hygiene practice class. From the figure, it can be seen that all trajectories of the solutions of infectious individuals converge to zero. Also, it can be seen that the trajectories converge to zero at different times. For instance, when $\delta = 0$ (No proper hygiene practice) the trajectory takes more than 20 days to converge zero while for $\delta = 0.9$ it takes around 10 days. This implies that as the rate of recruitment to proper hygiene practice class increases infectious individuals take shorter time to converge to zero (disease free equilibrium point).

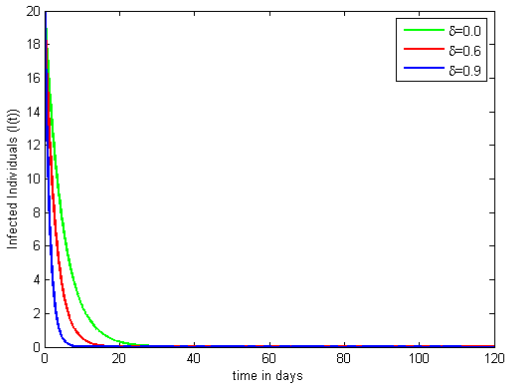

Fig. 2. The effect of proper hygiene practice on infected individuals

5 Conclusion

In this research we formulated a mathematical model of tungiasis dynamics with incorporation of proper hygiene practice. We carried out stability analysis and it showed that the disease free equilibrium is locally asymptotically stable provided that $R_0 < 1$ while endemic equilibrium is locally asymptotically stable provided that $R_0 > 1$. Numerical simulation results demonstrate that effective proper hygiene practice helps in reducing tungiasis infection.

6 Recommendations

From the results of this study, it is clear that tungiasis infection can be controlled through the incorporation of effective proper hygiene practice. Hence we recommend health policy makers of the government of Kenya and the general public to ensure that hygiene measures like wearing of shoes and living in houses with cemented floors are observed.

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Competing Interests

Authors have declared that no competing interests exist.

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