MATHEMATICAL MODELLING OF TUNGIASIS DISEASE DYNAMICS INCORPORATING HYGIENE AS A CONTROL STRATEGY.

BY

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DECLARATION

This project is my original work and has not been presented for a degree in any other university/Institution for consideration of any certificate.

Signature…… Date…….

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This project has been submitted for review with my approval as the university supervisor.

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DEDICATION

I dedicate my project work to my family and many friends.

Special gratitude to my husband Mbuthia for his great support and encouragement, and for being there for me throughout the entire Masters program.

I also dedicate this work and give special thanks to my children, Cuki, Mash and Shiko, whose words of encouragement ring in my ears.

I also dedicate this project to my brothers and sisters, and to many friends, who have supported me throughout the project.

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ACRONYMS AND ABBREVIATIONS

1. CBO – Community Based Organization
2. MOH – Ministry Of Health
3. MALDF – Ministry of Agriculture Livestock Development and Fisheries
4. MOEST – Ministry of Education Science and Technology
5. MEWNR – Ministry of Environment Water and Natural Resources
6. MICNG – Ministry of Interior and Coordination of National Government
7. NGO – Non-Governmental Organization
8. NTD – Neglected Tropical Diseases.
9. WHO – World Health Organization

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DEFINITION OF TERMS

Tungiasis - disease caused by female jigger flea

Chronic disease - one that has persisted for a long time or constantly recurring

Super infection - reinfection of an individual who is already infected

Fissures - a groove, natural divisions, deep furrow, elongated cleft or tear in various parts of the body

Ulcer - an open sore or wound that develops on the skin

Sepsis - life threatening complication of an infection (accumulation of pus)

Gangrene - dead tissue caused by an infection or lack of blood flow

ABSTRACT

Despite the intensive research that has been done on tungiasis, a lot remains to be done, especially on hygiene as a control strategy. Protection measures include, but not limited to; vaccination against tetanus in areas that are tungiasis-endemic, regular application of natural repellants like coconut oil and neem and regularly spraying domestic animals such as cats and dogs that act as reservoirs to tunga penetrants. Families in prone areas can also be provided with shoes, house floors that are sealed, clean water and soap. In this research, we particularly concentrate on hygiene as a control strategy.

We have formulated a model which is mathematical in nature. The model is based on a system of ordinary differential equations, which are used to study the dynamics of tungiasis disease, incorporating hygiene as protection against infection as a control measure. We have determined the steady state of the model and also carried out stability analysis. This has helped in determining the conditions that favor the infection of tungiasis in a community. We are then able to determine the effect of hygiene of a population against infection of tungiasis. We have established the endemic equilibrium point and also the disease free equilibrium point, which are found to be asymptotically stable. The basic reproduction number, R_O, is calculated using the next generation matrix. Numerical simulation of the model carried out showed that protection against infection using proper hygiene measures leads to a decrease in the spread of tungiasis.

1.0 INTRODUCTION

1.1. BACKGROUND INFORMATION

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, severe infestations of tungiasis has affected many communities, mostly in central Kenya, western, coast and Nyanza. The disease is debilitating to those affected. It affects all ages, but more so the children and the aged, due to their inability of taking care of themselves and also because of sitting in the same position for too long. The alcoholics and the disabled are also at risk of getting infected. Tungiasis is commonly known as nigua, jigger, ndutu, pique, pulga de areia or chigoe. It is a zoonosis because it is transmitted from animals (fleas) to humans. It also affects animals according to Heukelbach J et al, (2001). According to WHO, (2018), evidence show that tunga penetrans was imported to Africa towards the end of the 19th century, and has since spread to many Sub-Saharan countries. Many diseases that affect the resource poor countries in Africa have gone without much attention. This is because they have had less attention for medical authorities and researchers. Tungiasis is among such diseases according to Arene, (1984). It belongs to the family of neglected tropical diseases (NTDs), as recorded by Hotez P J, (2008).

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. It is in low levels in other parts of the country. 4 million Kenyans translating to 4% of the total population suffer from tungiasis according to MOH Kenya Policy, (2014). Ranking by regions, central Kenya had the highest infection rate followed by coast region as recorded by Ahadi Kenya, (2010).

In Murang'a 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, (2010).

A male fertilizes a tunga penetran only after it is burrowed and feeding on blood. The hind parts of the tunga penetran remains in contact with the air. This makes it possible for the flea to expel eggs, breathe and defecate. When eggs are expelled, they fall to the ground, after which the female dies, still embedded on the skin. In a suitable environment, like sandy dusty soil, the eggs then develop into adult sand fleas. Different mammalian animals are reservoirs for human infection. This include dogs, cats, rats and pigs according to WHO, (2018).

MOH Kenya policy, (2014), states that when the skin comes into contact with floor or soil which is infested with adult sand fleas, transmission occurs. All body parts are possible sites of infestation by tunga penetran, but the most affected parts are the feet, especially the toes, underneath the nails and the webs of the toes. This is because fleas cannot jump high. In case of heavy infestation, knees and elbows are also affected. The lesion due to infection can range from asymptomatic to pruritic and eventually becomes very painful. Multiple/severe infestations may result in a cluster of nodules with a honey comb appearance. As the lesion develops, bacterial superinfection almost inevitably occurs. During penetration, the flea breaks up the stratum corneum, allowing bacterial micro colonies on the skin surface to spread. According to WHO, (2018), deformation and loss of nails, lymphangitis, tissue necrosis, fissures, lymphedema and ulcers are chronic complications. These result in mutilation of the feet, pain and disfigurement. The characteristic changes in the way people with severe tungiasis walk is caused by mutilation and disfigurement. Life threatening complications can be caused by bacterial superinfection such as gangrene, tetanus and post-streptococcal glomerulo nephritis as stated by Heukelbach J.*et al*, (2004). Tetanus is a common

complication in children as recorded by Heukelbach J.*et al*, (2001).According to MOH policy of 2014, it is a common secondary infection that has been associated with deaths of jigger victims and the spread of HIV/AIDS which is passed from one person to person due to sharing of pins. The traditional method of treatment, involves the removal of embedded sand fleas with sharp non-sterile instruments like needles and safety pins. This may lead to transmission of blood-borne pathogens such as hepatitis B and C, possibly also HIV as recorded by FeldMeier H *et al*. (2013a).

Tungiasis has mostly been associated with household poverty. The Tunga penetran infestations depends on the family's ability to access clean water, sanitation, good quality housing and good nutrition. Tungiasis therefore is linked to poor hygiene, sanitation education, and 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 Policy,(2014).

The table below shows the symptoms and signs of acute and chronic Tungiasis.

Table 1: Symptoms and signs of acute and chronic tungiasis

Acute Tungiasis	Chronic Tungiasis
Difficulty in walking	Loss of nails
Fissures	Deformation of toes
Severe itching	Deformation of the nails
Pain upon pressure	Auto amputation of toes
Lesion in clusters	Tetanus
Ulcer	Sepsis
Skin inflammation and swelling	Gangrene

Tungiasis is neglected by the scientific community and the medical profession, but it is a disease that is debilitating to those affected.

NGOs and civil organizations have supported the MOH in the control of tungiasis. However, a lot has not been done on the protection of populations in the endemic areas. Protection measures may include, but not limited to; vaccination against tetanus in areas that are tungiasis-endemic, regular application of natural repellants like coconut oil and neem and regularly spraying domestic animals such as cats and dogs that act as reservoirs to tunga penetrants. Families in prone areas can also be provided with shoes, house floors that are sealed, clean water and soap.

Since most of the above measures are unaffordable to most affected people, various ministries, NGOs and CBOs should come together and work towards the common goal of eradicating Tungiasis. This ministries include; MOH, MALDF, MOEST, MEWNR and MICNG.

The occurrence of Tungiasis increases with the onset of dry seasons, and decreases when the rainy season starts. This means that protection measures such as spraying and fumigation must be done at the beginning of the dry seasons.

1.2. Statement of the Problem

Tungiasis is a disease that mostly affect the children, the disabled, alcoholics and the aged in Kenya and other parts of the world .There is evidence that Tungiasis results in serious acute and chronic conditions, and therefore there is need to address this issue.

In Muran'ga, people continue to suffer silently from tungiasis, and especially children.

Jigger infection can crumble communities' progress due to ineffectiveness of the infected individuals towards engaging in development projects.

Although much has been done on treatment as a control measure by the MOH, NGOs and civil organizations, a lot remains to be done on proper hygiene measures as a control.

In this research, we intend to fill this gap by doing a study on mathematical modelling of tungiasis disease, incorporating hygiene as a control strategy.

1.3. Justification of the Study

According to MOH Kenya, (2014), tungiasis (jiggers) afflicts 4% of the population in Kenya. Another 25%, mainly children, are at risk of infestation. The disease contributes to significant morbidity and sometimes mortality in endemic counties. According to the same survey, 10 million Kenyans who live in impoverished households are at risk of tungiasis.

Many people in tungiasis- endemic areas continue to silently suffer, especially children, according to Ahadi Kenya, (2010). They are unable to participate in learning activities write properly or even walk to school as stated by Ahadi Kenya, (2011)

According to Karuga, (2011), a newspaper reported that due to jigger infestations, 50,000 children dropped out of school in a period of 20 months, and 265 people died due to tungiasis related causes in the same period.

In Murang'a County, over 5,000 school going children have been reported to have dropped out of school because they were not able to walk due to tungiasis infection, as recoded by Kimani *et al.*,(2012).

Widespread use of harmful approaches to treatment of tungiasis, lack of standardized approaches to control, treatment and protection has led to the serious infection of tungiasis in endemic areas.

Notably, this infection has been neglected and go unchecked yet it impacts negatively to the development of a nation.

There is therefore need to incorporate proper hygiene as a control strategy against the infection of the disease.

1.4. Objectives of the Study

1.4.1. Main objective

To develop a mathematical model of tungiasis infection dynamics incorporating hygiene as a control strategy.

1.4.2. Specific objective

i) To develop a mathematical model of tungiasis infection incorporating hygiene.

ii) To perform stability analysis of the disease free equilibrium and endemic equilibrium.

iii) To carry out simulation to investigate the role of hygiene in tungiasis control.

1.5. Model Assumptions

The main assumption of the model is that once an individual is treated, there is no re-infection.

1.6 Significance of the Study.

The study will be significant to the policy makers on sensitizing the public on the importance of protection in Tungiasis control in a community that will lead to a reduction in dependency ratio, which can hinder development of a nation.

The study will also help health workers understand the dynamics of tungiasis hence enhance treatment.

2.0 LITERATURE REVIEW

2.1. Introduction

This chapter reviews literature on the mathematical modelling of tungiasis incorporating hygiene as a control strategy against infection.

2.2. Literature Review

The first documentation of the ectoparasite tunga penetrans was made by Fernandez in 1525, when he noted that Spanish conquerors in the native indigenous populations from Haiti quite often suffered from jigger infection according to Heukelbach J *et al,* (2001).

Gonzalo Ximenes de Quesada, (1535) reported that an entire village had been abandoned by its inhabitants because of tungiasis infection. He was a Spanish conqueror on a military expedition in Colombia. The soldiers could hardly walk since they were severely infested according to Sachse et al., (2007).

Jorg Heukelbach, (2005) did a research on diagnosis, treatment, control and prevention of tungiasis. His research findings were that education should focus on secondary prevention. He recommends use of appropriate study designs to increase knowledge on the control of tunga penetrans.

H. Feldmeier *et al*., (2014) 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.

MOH Kenya policy, (2014) came up with national policy guidelines on prevention and control of jigger infestations. One of the many recommendations stated that measures aiming to interrupt the off-host development should focus on physically changing the environment in which eggs, pupae and larva develop, hence the need to research on hygiene as a protection measure.

Kiragu G, (2015) 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, (2016) 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 J *et al*., (2016) 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.

Bashahun G. *et al*., (2017) did a research on prevalence rates of tungiasis. The research showed that the prevalence rate in Kenya is very high, and as such, new appropriate prevention and control

approaches should be designed to mitigate the persistence of the disease, particularly in vulnerable and poor communities.

Kahuru J. *et a*l., (2017) 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.

Nyangacha R.m.*et al*, (2019) carried out a research on tungiasis infection, and recommended that there is need to design control strategies for tungiasis that are cost effective and easily accessible

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 will carry out a study on mathematical modelling of tungiasis disease incorporating hygiene as a control strategy. We will modify work done by Nthiiri, (2016), by researching on her recommendation of observing cleanliness.

3.0 MATHEMATICAL FORMULATION OF THE MODEL.

3.1.Model Formulation

We have formulated a mathematical model based on ordinary differential equations, which are used to study the dynamics of tungiasis disease.

The model is formulated where the total population is in four categories. This include; the proper hygiene 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 hygienically protected group is recruited at birth at a rate $\delta\psi$, by ensuring that the people in the prone areas are provided with clean adequate water and sanitation, 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 the protected individuals. The protected group (P) become susceptible(S) at a rate λ. 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

$$\alpha = \frac{\ell c I}{N}$$

Where ℓ 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.

Table 2: Table of variables

Symbol	**Variable description**
N	Total population
P	Protected individuals
S	Susceptible individuals
I	Infected individuals
T	Treated individuals

Table 3: Description of parameters

Parameter description	**Symbol**
Recruitment rate	Ψ
Probability of getting recruited	δ
Loss of protection rate	λ
Natural mortality rate	ε
Disease induced mortality rate	Ω
Contact rate of infection	c
Probability rate of infection	ℓ
Rate of infection	α
Rate of treatment	β

The model flow diagram is as follows;

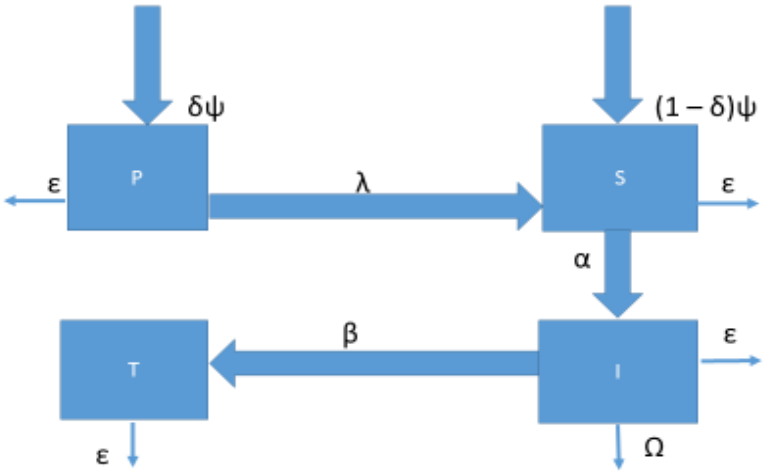

Figure 1: Model flow diagram

The model equations is given by;

$$\frac{dP}{dt} = \delta\psi - (\lambda + \varepsilon)P \quad \textbf{(3.1.1)}$$

$$\frac{dS}{dt} = (1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S \quad \textbf{(3.1.2)}$$

$$\frac{dI}{dt} = \alpha S - (\Omega + \beta + \varepsilon)I \quad \textbf{(3.1.3)}$$

$$\frac{dT}{dt} = \beta I - \varepsilon T \quad \textbf{(3.1.4)}$$

3.2.Positivity of Solutions

The model will be analyzed in the feasible region Γ, where (P (t), S (t), I (t), T (t)) $\in \Gamma \subset \mathbb{R}^4_+$.

We show that the state variables of the model remain positive ∀t > 0.

Theorem 3.3. *The state variables (P (t), S (t), I (t), T (t))* ≥ 0 $\forall t > 0$ in Γ.

Proof.

Consider equation (**3.1.1)**

$$\frac{dP}{dt} = \delta\psi - (\lambda + \varepsilon)P$$

This implies that

$$\frac{dP}{dt} \geq -(\lambda + \varepsilon)P$$

On integration,

$$\int \frac{dP}{dt} \geq \int -(\lambda + \varepsilon)P$$

$$\int \frac{dP}{P} \geq \int -(\lambda + \varepsilon)dt$$

$$\ln P(t) \geq -(\lambda + \varepsilon)t + \ln C$$

At t = 0, $\ln P(0) = \ln C$.

On substitution,

$$\ln P(t) \geq -(\lambda + \varepsilon)t + \ln P(0)$$

Therefore $\ln \frac{P(t)}{P(0)} \geq -(\lambda + \varepsilon)t$

Thus $\frac{P(t)}{P(0)} \geq e^{-(\lambda + \varepsilon)t}$

Hence $P(t) \geq P(0)\, e^{-(\lambda + \varepsilon)t}$

$$P(t) \geq 0 \ \forall \ t > 0 \qquad \textbf{(3.2.1)}$$

Consider equation (**3.1.2)**

$$\frac{dS}{dt} = (1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S$$

$$\int \frac{dS}{dt} \geq \int -(\alpha + \varepsilon)S$$

$$\int \frac{dS}{S} \geq \int -(\alpha + \varepsilon)dt$$

$$\ln S(t) \geq -(\alpha + \varepsilon)t + \ln C$$

At t = 0, $\ln s(0) = \ln C$

Substituting, $\ln S(t) \geq -(\alpha + \varepsilon)t + \ln S(0)$

$$\ln \frac{S(t)}{S(0)}) \geq -(\alpha + \varepsilon)t$$

$$\frac{S(t)}{S(0)} \geq e^{-(\alpha+\varepsilon)t}$$

Therefore $S(t) \geq S(0)\, e^{-(\alpha+\varepsilon)t}$

Hence $S(t) \geq 0 \ \forall t > 0$ **(3.2.2)**

We now consider equation (**3.1.3)**

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

$$\frac{dI}{dt} \geq -(\Omega + \beta + \varepsilon)I$$

On integration,

$$\int \frac{dI}{dt} \geq \int -(\Omega + \beta + \varepsilon)I$$

$$\int \frac{dI}{I} \geq \int -(\Omega + \beta + \varepsilon)dt$$

$$\ln I\,(t) \geq -(\Omega + \beta + \varepsilon)t + \ln C$$

At t = 0, $\ln I\,(0) = \ln C$

Substituting we get,

$$\ln I\,(t) \geq -(\Omega + \beta + \varepsilon)t + \ln I\,(0)$$

$$\ln \frac{I\,(t)}{I\,(0)} \geq -(\Omega + \beta + \varepsilon)t$$

$$I\,(t) \geq I\,(0)\, e^{-(\Omega+\beta+\varepsilon)t}$$

This implies that $I\,(t) \geq 0 \quad \forall\, t > 0$ **(3.2.3)**

We then consider equation (**3.1.4)**

$$\frac{dT}{dt} = \beta I - \varepsilon T$$

$$\frac{dT}{dt} \geq -\varepsilon T$$

On integration,

$$\int \frac{dT}{dt} \geq \int -\varepsilon T$$

$$\int \frac{dT}{T} \geq \int -\varepsilon dt$$

$$\ln T\,(t) \geq -\varepsilon t + \ln C$$

At t = 0, $\ln T\,(0) = \ln C$

On substitution,

$$\ln T(t) \geq -\varepsilon t + \ln T(0)$$

$$\ln\frac{T(t)}{T(0)} \geq -\varepsilon t$$

$$T(t) = T(0)\, e^{-\varepsilon t}$$

Hence $T(t) \geq 0 \quad \forall\, t > 0$ **(3.2.4)**

This implies that in view of equations (**3.2.1), (3.2.2), (3.2.3)** and (**3.2.4),** all state variables are non-negative $\forall\, t > 0$

3.3.Boundedness of Solutions

We now show that our solutions are bounded, implying that the model is epidemiologically posed in Γ, and thus can be analyzed.

Theorem 3.3. *All solutions are bounded in* Γ $\forall\, t > 0$, *such that* $0 \leq N \leq \frac{\psi}{\varepsilon}$, *and (S (t), T (t), I (t), P (t)) Є* $\Gamma \subset \mathbb{R}^4_+$.

Proof

Since $$\frac{dN}{dt} = \frac{dP}{dt} + \frac{dS}{dt} + \frac{dI}{dt} + \frac{dT}{dt}, \quad \textbf{(3.3.1)}$$

And $$\frac{dP}{dt} = \delta\psi - (\lambda + \varepsilon)P$$

$$\frac{dS}{dt} = (1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S$$

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

$$\frac{dT}{dt} = \beta I - \varepsilon T \quad ,$$

Then $$\frac{dN}{dt} = \psi - \varepsilon(P + S + I + T) - \Omega I \qquad \textbf{(3.3.2)}$$

But $P + S + I + T = N$

Therefore $$\frac{dN}{dt} = \psi - \varepsilon N - \Omega I$$

Assuming there's no infection, $$\frac{dN}{dt} \leq \psi - \varepsilon N$$

$$dN \leq (\psi - \varepsilon N)\, dt$$

$$1dN + \leq (\varepsilon N - \psi)\, dt \leq 0$$

Let $G = 1$, $H = \varepsilon N - \psi$

Then $\frac{\partial G}{\partial t} = 0$, $\frac{\partial H}{\partial N} = \varepsilon$

Since $\frac{\partial G}{\partial t} \neq \frac{\partial H}{\partial N}$,

Then we use an integrating factor such that $\frac{\partial G}{\partial t} = \frac{\partial H}{\partial N}$,

The integrating factor is given by $e^{\int\left[\frac{1}{P}\left(\frac{\partial H}{\partial N} - \frac{\partial G}{\partial t}\right)\right]dt} = e^{\varepsilon t}$

Since $dN \leq (\psi - \varepsilon N)\, dt$,

Then $e^{\varepsilon t}dN + (e^{\varepsilon t}\varepsilon N - e^{\varepsilon t}\psi)dt \leq 0$

On integration, i.e. $\int\left(\frac{\partial\Phi(N,t)}{\partial t}\right)dt = \int(e^{\varepsilon t}\varepsilon N - e^{\varepsilon t}\psi)\mathrm{dt} \leq 0$

$$(\mathrm{N}, \mathrm{t}) = e^{\varepsilon t}N - e^{\varepsilon t}\frac{\psi}{\varepsilon} \leq \mathrm{C}$$

Therefore, $e^{\varepsilon t}N - e^{\varepsilon t}\frac{\psi}{\varepsilon} \leq \mathrm{C}$

At t = 0, $N - \frac{\psi}{\varepsilon} = \mathrm{C}$

Substituting C, we get, $e^{\varepsilon t}N - e^{\varepsilon t}\frac{\psi}{\varepsilon} \leq N - \frac{\psi}{\varepsilon}$

$$e^{\varepsilon t}N \leq e^{\varepsilon t}\frac{\psi}{\varepsilon} + N - \frac{\psi}{\varepsilon}$$

Dividing throughout by $e^{\varepsilon t}$, we get $N \leq \frac{\psi}{\varepsilon} + (N - \frac{\psi}{\varepsilon})e^{-\varepsilon t}$

As $t \to \infty$, $N \leq \frac{\psi}{\varepsilon}$ **(3.3.3)**

This shows that all solutions are bounded in Γ, $\forall\, t > 0$, such that $0 \leq N \leq \frac{\psi}{\varepsilon}$, hence the model can be analyzed.

3.4.Existence of Equilibrium Points

In this section we quantitatively analyze the model to investigate the stability of its equilibria, both at endemic equilibrium (DFE) and at disease free equilibrium (EE).

Consider the model equations

$$\frac{dP}{dt} = \delta\psi - (\lambda + \varepsilon)P$$

$$\frac{dS}{dt} = (1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S$$

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

$$\frac{dT}{dt} = \beta I - \varepsilon T$$

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

$$\delta\psi - (\lambda + \varepsilon)P = 0 \quad \textbf{(3.4.1)}$$

$$(1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S = 0 \quad \textbf{(3.4.2)}$$

$$\alpha S - (\Omega + \beta + \varepsilon)I = 0 \quad \textbf{(3.4.3)}$$

$$\beta I - \varepsilon T = 0 \quad \textbf{(3.4.4)}$$

Where $\alpha = \frac{\ell cI}{N}$

ℓ is the probability rate of acquiring tungiasis disease and c is the contact rate of infection.

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To calculate the disease free equilibrium, we set P, I, T to be equal to zero.

From equation **(3.4.2),** $(1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S = 0$

We get $S = \frac{(1-\delta)\psi}{(\varepsilon + \alpha)}$ since P = 0

$$S = \frac{(1-\delta)\psi}{\varepsilon + \frac{\ell cI}{N}}$$

Hence $S = \frac{(1-\delta)\psi}{\varepsilon}$, since I =0

The DFE point E^0 is therefore given by

$$E^0 = \left(0, \frac{(1-\delta)\psi}{\varepsilon}, 0, 0\right) \quad \textbf{(3.4.5)}$$

To calculate the EE, we set P, S, I, T not equal to zero.

Consider equation **(3.4.1)** $\delta\psi - (\lambda + \varepsilon)P = 0$

$$P^* = \frac{\delta\psi}{\varepsilon+\lambda} \qquad \textbf{(3.4.6)}$$

Consider equation 3.5.3 $\alpha S - (\Omega + \beta + \varepsilon)I = 0$

$$S^* = \frac{N(\varepsilon+\Omega+\beta)}{\ell c} \quad , \quad \text{since} \quad \alpha = \frac{\ell c I}{N} \qquad \textbf{(3.4.7)}$$

We now consider equation **(3.4.2)** $(1 - \delta)\psi + \lambda P - (\alpha + \varepsilon)S = 0$

$$(\alpha + \varepsilon)S = (1 - \delta)\psi + \lambda P$$

Substituting the value of P*we get $(\alpha + \varepsilon)S = (1 - \delta)\psi + \frac{\lambda\delta\psi}{\lambda+\varepsilon}$

$$(\alpha + \varepsilon)S = \frac{(1-\delta)(\lambda+\varepsilon)\psi + \lambda\delta\psi}{\lambda+\varepsilon}$$

On expanding we get, $(\alpha + \varepsilon)S = \frac{\psi(\lambda+\varepsilon-\delta\varepsilon)}{\lambda+\varepsilon}$

Substituting the value of S*, we get $(\alpha + \varepsilon) = \frac{\psi(\lambda+\varepsilon-\delta\varepsilon)\ell c}{(\lambda+\varepsilon)(\varepsilon+\Omega+\beta)N}$

Therefore $\alpha = -\varepsilon + \frac{\psi(\lambda+\varepsilon-\delta\varepsilon)\ell c}{(\lambda+\varepsilon)(\varepsilon+\Omega+\beta)N}$

Then substituting α and factorizing gives

$$I^* = \frac{1}{(\varepsilon+\Omega+\beta)}\left(\frac{(\varepsilon+\lambda-\delta\varepsilon)\psi}{(\varepsilon+\lambda)} - \frac{\varepsilon N(\varepsilon+\Omega+\beta)}{\ell c}\right) \qquad \textbf{(3.4.8)}$$

Lastly we consider equation **(3.3.4)** $\beta I - \varepsilon T = 0$

$$T^* = \frac{\beta I}{\varepsilon}$$

Substituting I* we get $T^* = \frac{\beta}{(\varepsilon+\Omega+\beta)}\left(\frac{(\varepsilon+\lambda-\delta\varepsilon)\psi}{(\varepsilon+\lambda)} - \frac{\varepsilon N(\varepsilon+\Omega+\beta)}{\ell c}\right)$ **(3.4.9)**

Considering equations (**3.4.6**), (**3.4.7), (3.4.8)** and **(3.4.9),** the endemic equilibrium is therefore given by

$E^*= (P^*, S^*, I^*, T^*)$

$$= \left(\frac{\delta\psi}{\varepsilon+\lambda}, \frac{N(\varepsilon+\Omega+\beta)}{\ell c}, \frac{1}{(\varepsilon+\Omega+\beta)}\left(\frac{(\varepsilon+\lambda-\delta\varepsilon)\psi}{(\varepsilon+\lambda)} - \frac{\varepsilon N(\varepsilon+\Omega+\beta)}{\ell c}\right), \frac{\beta}{(\varepsilon+\Omega+\beta)}\left(\frac{(\varepsilon+\lambda-\delta\varepsilon)\psi}{(\varepsilon+\lambda)} - \frac{\varepsilon N(\varepsilon+\Omega+\beta)}{\ell c}\right)\right)$$

3.5.The Basic Reproduction Number

The basic reproduction number, R_O 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. It can be computed as a ratio of known rates over time, and is calculated using the next generation matrix.

It 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_O is important in that it is directly related to the effort required to eliminate infection. When R_O is greater than 1, it is not easy to eliminate infection and vice versa.

The epidemic model is given by equation **(3.1.3)**

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

From the model, the associated matrices are

$$f = \alpha S$$

$$f = \frac{\ell cIS}{N} \qquad \text{since } \alpha = \frac{\ell cI}{N}$$

Taking derivative with respect to the disease compartment I, we have

$$F = \frac{\ell cS}{N}$$

At disease free equilibrium, S = N. Then,

$$F = \ell c \qquad \textbf{(3.5.1)}$$

The v matrix is given by $v = (\Omega + \beta + \varepsilon)I$

Taking derivative with respect to I we get

$$V = (\Omega + \beta + \varepsilon) \qquad \textbf{(3.5.2)}$$

Thus $$V^{-1} = \frac{1}{(\Omega+\beta+\varepsilon)} \qquad \textbf{(3.5.3)}$$

Hence considering equations **(3.5.1)** and **(3.5.3)**

$$FV^{-1} = \frac{\ell c}{(\Omega+\beta+\varepsilon)}$$

The basic reproduction number, R_O, is the spectral radius of the matrix FV^{-1} given by

$$\rho\,(FV^{-1}) = \frac{\ell c}{(\Omega+\beta+\varepsilon)}$$

Therefore $$R_O = \frac{\ell c}{(\Omega+\beta+\varepsilon)} \qquad \textbf{(3.5.4)}$$

3.6.Local stability of the Disease-free Equilibrium (DFE)

We now analyze the model to investigate the stability of its disease free equilibrium. The DFE points of the model are its steady state solutions in the absence of infection or disease.

Theorem 3.6.*The disease free equilibrium E^0 of the model is locally asymptotically stable whenever $R_o < 1$.*

Proof:

The Jacobian matrix of the linearized system of the model system of equations (**3.1.1), (3.1.2), (3.1.3)** and (**3.1.4)** is given by

$$J = \begin{bmatrix} -(\lambda + \varepsilon) & 0 & 0 & 0 \\ \lambda & -\left(\varepsilon + \frac{\ell cI}{N}\right) & \frac{-\ell cS}{N} & 0 \\ 0 & \frac{\ell cI}{N} & \frac{\ell cS}{N} - (\varepsilon + \Omega + \beta) & 0 \\ 0 & 0 & \beta & -\varepsilon \end{bmatrix}$$

We compute the Jacobian matrix at disease free equilibrium and investigate its stability effect due to the reproduction number, R_O.

At DFE, I = 0 and N = S.

Hence at DFE, the Jacobian becomes

$$J_{E^0} = \begin{bmatrix} -(\lambda + \varepsilon) & 0 & 0 & 0 \\ \lambda & -\varepsilon & -\ell c & 0 \\ 0 & 0 & \ell c - (\varepsilon + \Omega + \beta) & 0 \\ 0 & 0 & \beta & -\varepsilon \end{bmatrix}$$

The stability of J_{E^0} is completely determined by Routh-Hurwitz criteria which states that for a system to be stable

a) The trace of J_E^0 must be negative

b) The determinant of J_E^0 must be positive.

Applying the criteria, we compute the trace and the determinant and set the conditions.

The trace (τ) at DFE, E^0, is given by

$$\tau (J_E^0) = -(\lambda + \varepsilon) - \varepsilon + \ell c - (\varepsilon + \Omega + \beta) - \varepsilon$$

$$= -(\lambda + \varepsilon) - 2\varepsilon + \ell c - (\varepsilon + \Omega + \beta)$$

$$= -(\lambda + \varepsilon) - 2\varepsilon + \left(\frac{\ell c}{(\Omega+\beta+\varepsilon)} - 1\right)(\varepsilon + \Omega + \beta)$$

Since $R_O = \frac{\ell c}{(\varepsilon + \Omega + \beta)}$ from **(3.5.4)**

Then $\tau (J_E^0) = -(\lambda + \varepsilon) - 2\varepsilon + (R_O - 1)(\varepsilon + \Omega + \beta)$

Which is negative provided that $R_O < 1$.

The determinant of DFE is given by;

$$\det (J_E^0) = -(\lambda + \varepsilon)(-\varepsilon)(\ell c - (\varepsilon + \Omega + \beta))(-\varepsilon)$$

$$= -(\lambda + \varepsilon)(\varepsilon^2)\left(\frac{\ell c}{(\varepsilon + \Omega + \beta)} - 1\right)(\varepsilon + \Omega + \beta)$$

Since $R_O = \frac{\ell c}{(\varepsilon + \Omega + \beta)}$ from **(3.5.4)**

Then $\det (J_E^0) = -(\lambda + \varepsilon)(\varepsilon^2)(R_O - 1)(\varepsilon + \Omega + \beta)$

Which is positive provided that $R_O < 1$.

This shows that both the trace and the determinant of J_E^0 satisfy the Routh-Hurwitz criterion. Thus the disease-free equilibrium is locally asymptotically stable when Ro < 1.

The model has a disease free equilibrium when $R_o < 1$.

3.7.Local stability of the endemic equilibrium (EE) of the model

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

Theorem 3.7. *The endemic equilibrium of the model is locally asymptotically stable whenever* Ro > 1.

Proof

We investigate the stability of the EE using the Routh-Hurwitz criterion, that is, the trace and the determinant of the matrix J_E^*. The Jacobian matrix at E^* is given by

$$J_E^* = \begin{bmatrix} -(\lambda + \varepsilon) & 0 & 0 & 0 \\ \lambda & -\left(\varepsilon + \frac{\ell c \kappa_1}{N}\right) & \frac{-\ell c \kappa_2}{N} & 0 \\ 0 & \frac{\ell c \kappa_1}{N} & \frac{\ell c \kappa_2}{N} - (\varepsilon + \Omega + \beta) & 0 \\ 0 & 0 & \beta & -\varepsilon \end{bmatrix}$$

Where $\kappa_1 = \frac{1}{(\varepsilon + \Omega + \beta)} \left(\frac{(\varepsilon + \lambda - \delta\varepsilon)\psi}{(\varepsilon + \lambda)} - \frac{\varepsilon N(\varepsilon + \Omega + \beta)}{\ell c}\right)$ $\kappa_2 = \frac{N(\varepsilon + \Omega + \beta)}{\ell c}$

We now compute the trace (τ) of J_E^*, which is given by

$$\tau(J_E^*) = -(\lambda + \varepsilon) - \left(\varepsilon + \frac{\ell c \kappa_1}{N}\right) + \frac{\ell c \kappa_2}{N} - (\varepsilon + \Omega + \beta) - \varepsilon$$

Substituting κ_2, we get

$$\tau(J_E^*) = -(\lambda + \varepsilon) - 2\varepsilon - \frac{\ell c\, \kappa_1}{N} + \frac{\ell c N(\varepsilon+\Omega+\beta)}{\ell c} - (\varepsilon + \Omega + \beta)$$

$$= -(\lambda + \varepsilon) - 2\varepsilon - \frac{\ell c d3(\varepsilon+\Omega+\beta)}{N(\varepsilon+\Omega+\beta)} + (\varepsilon + \Omega + \beta) - (\varepsilon + \Omega + \beta)$$

$$= -(\lambda + \varepsilon) - 2\varepsilon - \frac{Ro(\varepsilon+\Omega+\beta)\kappa_1}{N} + 0 \qquad \text{from } \mathbf{(3.5.4)}$$

$$= -(\lambda + \varepsilon) - 2\varepsilon - \frac{Ro(\varepsilon+\Omega+\beta)\kappa_1}{N}$$

Which is negative provided that $R_O > 1$

We now compute the determinant of J_E^*, which should be positive according to Routh-Hurwitz criterion. This is given by

$$\det(J_E^*) = -(\lambda + \varepsilon)\left(\left(\varepsilon + \frac{\ell c\, \kappa_1}{N}\right)\right)\left(\frac{\ell c d2}{N} - (\varepsilon + \Omega + \beta)\right)(-\varepsilon)$$

$$= \varepsilon(\lambda + \varepsilon)\left(-\varepsilon - \frac{\ell c\, \kappa_1}{N}\right)\left(\frac{\ell c\, \kappa_2}{N} - (\varepsilon + \Omega + \beta)\right)$$

$$= \varepsilon(\lambda + \varepsilon)\left(-\varepsilon\left(\frac{\ell c\, \kappa_2}{N}\right) + \varepsilon(\varepsilon + \Omega + \beta) - \left(\left(\frac{\ell c\, \kappa_2}{N}\right)\left(\frac{\ell c \kappa_1}{N}\right)\right) + \frac{\ell c \kappa_1}{N}(\varepsilon + \Omega + \beta)\right.$$

Substituting the first κ_2 , we get

$$\det(J_E^*) = \varepsilon(\lambda + \varepsilon)\left(\left(-\varepsilon\left(\frac{\ell c}{N}\right)\left(\frac{N(\varepsilon+\Omega+\beta)}{\ell c}\right) + \varepsilon(\varepsilon + \Omega + \beta) - \left(\left(\frac{\ell c \kappa_2}{N}\right)\left(\frac{\ell c \kappa_1}{N}\right)\right) + \frac{\ell c \kappa_1}{N}(\varepsilon + \Omega + \beta)\right.\right.$$

$$= \varepsilon(\lambda + \varepsilon)\left(-\varepsilon(\varepsilon + \Omega + \beta) + \varepsilon(\varepsilon + \Omega + \beta) - \left(\left(\frac{\ell c \kappa_2}{N}\right)\left(\frac{\ell c \kappa_1}{N}\right)\right) + \frac{\ell c}{N}(\varepsilon + \Omega + \beta)\right)$$

$$= \varepsilon(\lambda + \varepsilon)\left(-\left(\left(\frac{\ell c \kappa_2}{N}\right)\left(\frac{\ell c \kappa_1}{N}\right)\right)\right) + \frac{\ell c \kappa_1}{N}(\varepsilon + \Omega + \beta)$$

$$= \varepsilon(\lambda + \varepsilon)\left(-\frac{\ell c \kappa_2}{N(\varepsilon + \Omega + \beta)}\left(\frac{\ell c \kappa_1}{N}\left(\frac{\ell c \kappa_1}{N}\right)(\varepsilon + \Omega + \beta) + \left(-\frac{\ell c \kappa_2}{N(\varepsilon + \Omega + \beta)}\right)(\varepsilon + \Omega + \beta)\right.\right.\right.$$

$= \varepsilon(\lambda + \varepsilon)(-R_O(\frac{\ell c \kappa_2}{N}\frac{\kappa_1}{N})(\varepsilon + \Omega + \beta) + \frac{RO}{N}(\varepsilon + \Omega + \beta)^2\kappa_1$

$= \varepsilon(\lambda + \varepsilon)(R_O(\frac{(\varepsilon+\Omega+\beta)\kappa_1}{N})((\varepsilon + \Omega + \beta) - (\frac{\ell c \kappa_2}{N})))$ from **(3.5.4)**

Which is positive provided that R_O is positive (R_O >1)

Both the trace and the determinant of the EE satisfy the Routh-Hurwitz criterion, implying that the EE is stable when (R_O >1).

Therefore the model has an asymptotically stable endemic equilibrium provided that R_O > 0

4.0 NUMERICAL SIMULATIONS

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 the table below.

Table 4: Parameter values of the model

Parameter description	**Symbol**	**Value**	**Source**
Recruitment rate	Ψ	0.0044	Kenya demographic profile
Natural mortality rate	ε	0.016	Kenya demographic profile
Disease induced mortality rate	Ω	0.005	Estimated
Loss of protection rate	λ	0.001	Estimated
Transmission probability rate of tungiasis	ℓ	0.0011	Estimated
Contact rate of infection	c	0.0002	Estimated
Adjustment parameter	δ	0.8	Estimated
Rate of treatment	β	0.9	Estimated

Figure (2) below 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 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 to 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).

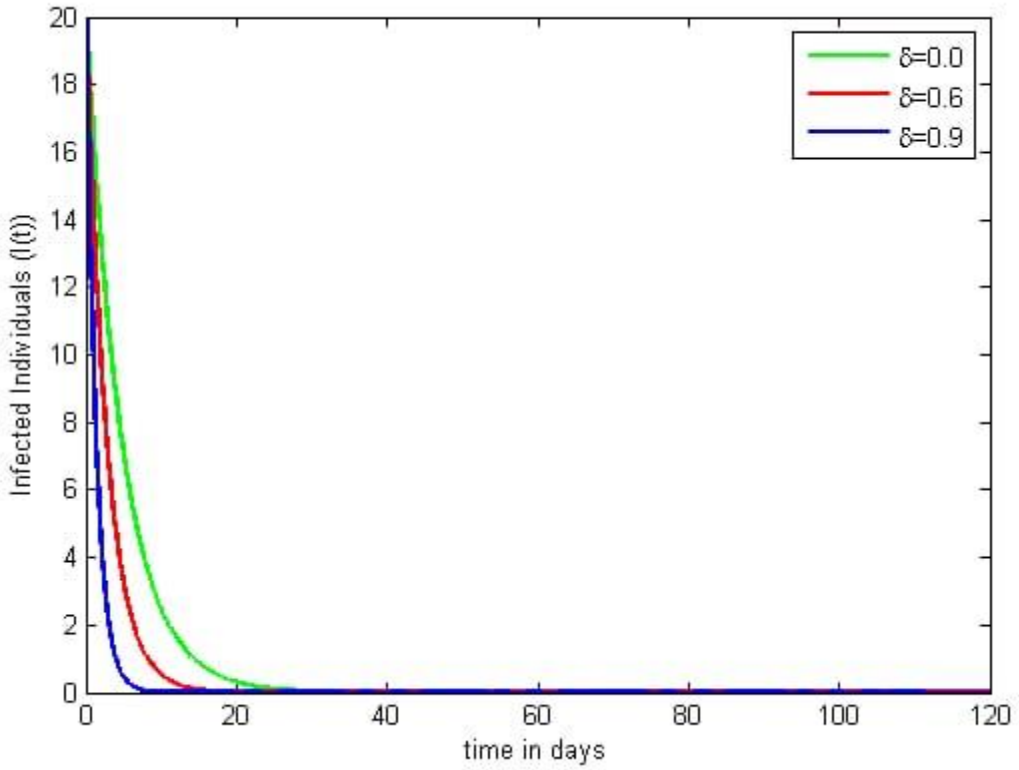

Figure 2: Effect of proper hygiene practice on infected individuals

5.0 CONCLUSIONS AND RECOMMENDATIONS

5.1. 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 $R_O > 1$. Numerical simulation results demonstrate that effective proper hygiene practice helps in reducing tungiasis infection

5.2. Recommendations

We recommend practice of proper hygiene to be enhanced as a control strategy of tungiasis infection. Proper hygiene can be practiced by, but not limited to; wearing shoes, watering dusty floors, regular application of natural repellants like coconut oil and neem and regularly spraying domestic animals such as cats and dogs that act as reservoirs to tunga penetrans

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