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# Effects of Social Media in Controlling Tungiasis: Mathematical Model

**Original Research  
Article**

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## Abstract

In this paper, a deterministic model incorporating social media in controlling tungiasis disease is considered. The model is shown to be positively invariant as well as bounded. We showed that the model has two equilibria points: disease free and endemic equilibria points. In both cases, the steady states are locally asymptotically stable provided the basic reproduction number is less than unity.

*Keywords:* Basic Reproduction Number, Local Stability, Mathematical Modeling

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## 1 Introduction

Tropical parasitic diseases are experienced by millions of the poorest population of the world. However, many of them are not listed by the World Health Organisation WHO as Neglected Tropical Diseases, as a consequence, they are ignored by governments and health care workers alike. These category of diseases therefore do not receive scientific research interest they merit. Tungiasis is one of them [11]. According to Kahuru et al [23], tungiasis is a skin disease caused by sand flea called *Tunga Penetrans*. The disease is endemic in poor resource communities where various domestic and

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sylvatic animals act as reservoirs for this zoonosis. The flea infestation is associated with poverty and occurs in many poor resource communities in the Caribbean, South America and Sub-Saharan Africa, [16].

Although by its nature a self-limiting diseases, tungiasis causes considerable morbidity, [14]. Fissures, ulcers, gangrene, lymphedema, deformation and loss of nails and auto-amputation of digits are known sequels. In a non-immunized individuals tungiasis is a risk factor for tetanus. Treatment of tungiasis involves identification of the parasite especially through mechanical removal using a sterile, sharp pointed objects such as needles or pins followed by an antiseptic dressing. It may also be effectively treated using surgical extraction of the embedded sand flea under sterile condition in medical facilities, [13]. The fleas may also be deterred by washing the affected areas with disinfectants like potassium permanganate and even coconut oil. Personal hygiene and wearing of shoes are key in the control of jigger infection. Children and the elderly need to be well taken care of through provision of primary and affordable amenities and health care facilities, [27]

Nyanginja, [27] asserts that transmission of the infectious disease has been of great interest to both medical practitioners and scholars. It is therefore critical to study epidemic transmission and take effective strategies to prevent and contain it. Individual response to disease threat depends on risk perception that is gained largely through information reported by the government to the public. Public health programs such as public vaccination or immunization, isolation through media and even education through social media can affect disease transmission during an epidemic contagion.

Mathematical modeling is an important tool used in analysing the dynamics of infectious diseases. Several models have been formulated and analysed to explain the dynamics of tungiasis disease transmission. Pigler, [34] proposed that control of Tungiasis in a resource - poor population with interventions target at the human and animal population.. He also mentioned that high prevalence of tungiasis in endemic area and the important morbidity associated with this parasitic skin disease call for the implementation of control measure. He determined the impact of repeated rounds of surgical extraction of embedded sand fleas in humans and found out that interventions were effective in controlling tungiasis for short term but failed in the long run. He recommended prevention of infestation, rather than surgical extraction of the already embedded sand fleas. Creating awareness of the disease through social media in this study forms the basis of prevention of transmission rather than treatment as effective control measure of the disease.

Kahuru et al, [22] constructed a model that investigated the dynamics of tungiasis. He employed a system of ordinary equations that incorporated interactions between humans, animal reservoirs and flea infested soil. He aimed at determining the effect of environment in controlling tungiasis disease and found that reducing on and off-host flea population and effective contact rate is an effective intervention, however, Kahuru did not consider the impact of social media in controlling the disease. Nyanginja et al, [27] formulated and analysed a susceptible  $S$ , Infected  $I$ , and Educated  $E$  (SIE) epidemic model incorporating public health education in control of transmission of tungiasis. His findings indicated that establishing public health education is an effective measure of controlling jigger menace as it reduced the spreading threshold. They further recommended proper control measure to be put in places especially in resources-poor communities where the diseases is usually endemic, however, they did not consider social media as an intervention strategy.

Social media has been known to greatly influence an individuals behaviour as well as government policies on prevention and control of infectious diseases. In this paper, we seek to understand the effects of social media in transmission of tungiasis disease

## 2 Model Formulation and Description

The human population is subdivided into classes of susceptible  $S_N$ , individuals exposed to social media  $S_E$ , the infected population  $I$ , individuals who are chronically infected  $C$  and the recovered population  $R$ .

We make the following assumptions:

1. Human birthrate and natural death rates takes place at different rates
2. Both susceptible and exposed individuals get infested but at different level.
3. The recovered group can also become susceptible
4. Chronically infested individual die from natural death or from disease induced death.

The recruitment into susceptible population  $S_N$  takes place at the rate of  $(1 - \pi)\Lambda$ , while recruitment into the exposed class occurs at  $\pi\Lambda$  where  $\pi$  is the proportion of susceptible exposed to social media. Natural death rate occurs in  $S_N, S_E, I, C$  and  $R$  classes at the rate  $\mu$ . Individuals in compartment  $C$  suffer an additional death due to the disease at the rate  $\sigma$ . Individuals leave  $S_N$  to  $S_E$  when they become exposed to health information via social media at the rate  $\xi$ . We assume that the mass action incidence transmission is defined by  $\beta S_N I$  where  $\beta$  is the effective contact rate for the disease transmission. Dissemination of information via social media may not be very effective due to either political or economic reasons and so the exposed individuals will get mildly affected at the rate  $(1 - \delta)\beta S_E I$  where  $\delta$  is the success rate of control and preventive efforts through social media and  $(1 - \delta)$  is the failure rate of the control efforts. Classes  $I$  and  $C$  recover at the rate  $\kappa$  and  $\lambda$  respectively due to treatment or other interventions. Recovered individuals revert back to the class  $S_N$  at the rate  $\omega$ . Individuals in class  $I$  move to class  $C$  at the rate  $\alpha$  due to highly infested environment. The figure below gives the schematic diagram of the model.

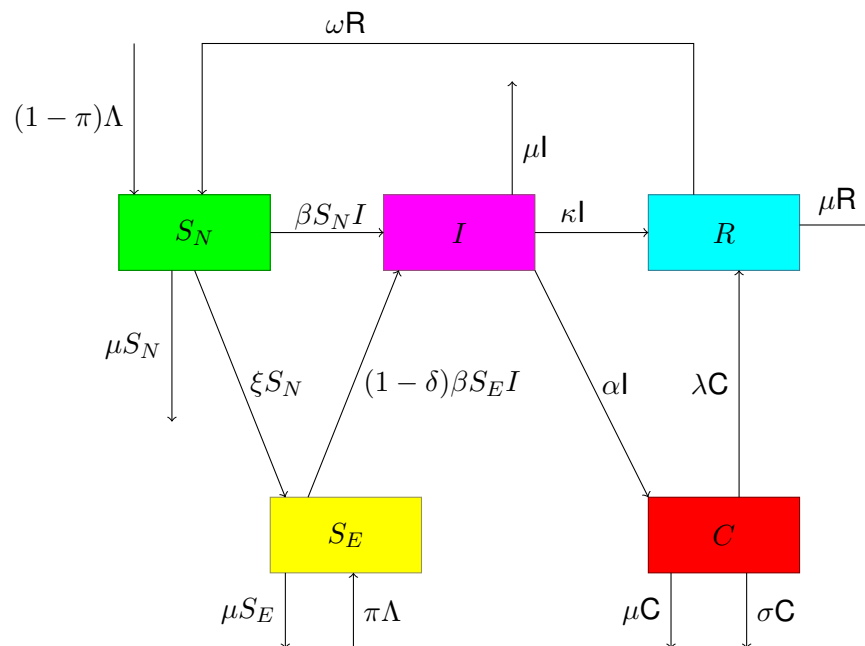


Figure 1: The flow chart showing dynamics of Tungiasis transmission

From the flow chart in figure 1 we obtain the followingsystem of ordinary differential equations

$$\begin{aligned}\frac{dS_N}{dt} &= (1 - \pi)\Lambda + \omega R - \beta S_N I - \mu S_N - \xi S_N \\ \frac{dS_E}{dt} &= \pi\Lambda + \xi S_N - (\mu + (1 - \delta)\beta I)S_E \\ \frac{dI}{dt} &= \beta S_N I + (1 - \delta)\beta S_E I - (\mu + \kappa + \alpha)I \\ \frac{dC}{dt} &= \alpha I - (\lambda + \mu + \sigma)C \\ \frac{dR}{dt} &= \kappa I + \lambda C - (\omega + \mu)R\end{aligned}\tag{2.1}$$

with initial conditions

$$S_N(0) \geq 0, S_E(0) \geq 0, I(0) \geq 0, C(0) \geq 0, R(0) \geq 0\tag{2.2}$$

### 3 Basic Qualitative Analysis of the Model

#### 3.1 Positivity of Solutions

The nonnegativity property of the model is explored in the following theorem

**Theorem 3.1.** *Suppose that condition (2.2) holds, then the solutions of the system (2.1) remain non-negative for all  $t \geq 0$ .*

*Proof.* Consider the first equation of the model system (2.1) at time  $t$

$$\frac{dS_N}{dt} + \beta S_N I - \mu S_N - \xi S_N \geq (1 - \pi)\Lambda$$

This is like a first order differential equation in  $S_N$  which has the solution

$$S_N(t) \geq S_N(0)e^{-\int_0^t (\beta I(s) + \mu + \xi) ds} + e^{-\int_0^t (\beta I(s) + \mu + \xi) ds} \times \int_0^t (1 - \pi)\Lambda e^{\int_0^u (\beta I(w) + \mu + \xi) dw} du \geq 0$$

for all  $t \geq 0$  since  $e^t > 0$  for all  $t$ .

Similarly, it can easily be shown that

$$S_E(t) \geq 0, I(t) \geq 0, C(t) \geq 0, R(t) \geq 0\tag{3.1}$$

Therefore the solutions remain non-negative for all  $t \geq 0$ .  $\square$

#### 3.2 Boundedness of Solutions

**Theorem 3.2.** *(Invariant Region) Let  $N(t) = S_N(t) + S_E(t) + I(t) + C(t) + R(t)$ . Then the feasible region of the model*

$$\Omega = \left\{ (S_N, S_E, I, C, R) \in \mathbb{R}_+^5 : N(t) \leq \frac{\pi}{\mu} \right\}$$

*is positively invariant.*

*Proof.* By adding the equations in the system (2.1) we obtain

$$\frac{dN}{dt} = \Lambda - \mu N - \sigma C$$

from which we have

$$\frac{dN}{dt} + \mu N \leq \Lambda \quad (3.2)$$

This is a first order differential equation in  $N$  whose solution is given by

$$N(t) \leq \frac{\Lambda}{\mu} + \left( N_0 - \frac{\Lambda}{\mu} \right) e^{-\mu t} \quad (3.3)$$

from which it follows that  $\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}$ . Hence  $N$  is bounded and all feasible solution sets of the model system approach or stay in  $\Omega$ . The region  $\Omega$  is therefore positively invariant and the model system is epidemiologically meaningful and Mathematically well posed in the domain  $\Omega$ . Hence it is sufficient to consider the dynamics of the flow it generates in a proper subset  $\Omega = \{(S_N, S_E, I, C, R) \in \mathbb{R}_+^5\}$   $\square$

## 4 Existence of Equilibria Points of The Model

To obtain equilibria points of the model system in equation (2.1), we equate the right hand side of (2.1) to zero and solve for the variables. That is,

$$\begin{aligned} (1 - \pi)\Lambda + \omega R^* - \beta S_N^* I^* - \mu S_N^* - \xi S_N^* &= 0 \\ \pi\Lambda + \xi S_N^* - (\mu + (1 - \delta)\beta I^*) S_E^* &= 0 \\ \beta S_N^* I^* + (1 - \delta)\beta S_E^* I^* - (\mu + \kappa + \alpha) I^* &= 0 \\ \alpha I^* - (\lambda + \mu + \sigma) C^* &= 0 \\ \kappa I^* + \lambda C^* - (\omega + \mu) R^* &= 0 \end{aligned} \quad (4.1)$$

From which we have;

$$S_N^* = \frac{(1 - \pi)\Lambda + \omega R^*}{\beta I^* + \mu + \xi} \quad (4.2)$$

$$S_E^* = \frac{\pi\Lambda + \xi S_N^*}{\mu + (1 - \delta)\beta I^*} \quad (4.3)$$

$$S_N^* = \frac{(\mu + \kappa + \alpha) - (1 - \delta)\beta S_E^*}{\beta} \quad (4.4)$$

$$C^* = \frac{\alpha I^*}{\lambda + \mu + \sigma} \quad (4.5)$$

$$R^* = \frac{\kappa(\lambda + \mu + \alpha) + \alpha\lambda}{(\mu + \omega)(\lambda + \mu + \alpha)} I^*$$

### 4.1 Disease Free Equilibrium of the Model

The model system has a steady state at a given period where there is no infestation in the population under consideration that is  $I^* = 0$ . Hence, the DFE point is given by

$$\begin{aligned} E^0 &= (S_N^0, S_E^0, I^0, C^0, R^0) \\ &= \left( \frac{(1 - \pi)\Lambda}{\mu + \xi}, \frac{(\mu + \xi)\pi\Lambda + \xi(1 - \pi)\Lambda}{\mu(\mu + \xi)}, 0, 0, 0 \right) \end{aligned}$$

## 4.2 Basic Reproduction Number

The basic reproduction number is an important epidemiological parameter. It is denoted by  $R_0$  and is the expected number of secondary infection produced by a single infective individual in a completely susceptible population, [38].

We specifically define the basic reproduction number  $R_{SE}$  of the model system equation (2.1) as the number of secondary tungiasis infections caused by a single jigger infected individual in the presence of social media awareness creation intervention. When no such programmes are employed, the basic reproduction number is defined by  $R_0$ . It measures the power of the disease to invade a population under conditions that facilitate a maximal growth. The basic reproduction number is important in that it is directly related to the effort required to eliminate infection. In general, the basic reproduction number depends on the demographic, disease and morbidity parameters.

The basic reproduction number can be obtained using the method in [38], that is, the effective reproduction number  $R_{SE}$  is given by the formula  $R_{SE} = \rho(FV^{-1})$  where  $\rho(M)$  represents the spectral radius of the matrix  $M$ ,  $F$  is the rate of occurrence of new infections while  $V$  is the rate of transferring the individuals outside the original group.

From the model system in equation (2.1), we see that

$$\mathcal{F} = \begin{pmatrix} \beta S_N I + (1 - \delta)\beta S_E I \\ 0 \end{pmatrix}, \mathcal{V} = \begin{pmatrix} (\mu + \kappa + \alpha)I \\ (\lambda + \mu + \sigma)C - \alpha I \end{pmatrix} \text{ and}$$

$$F = \begin{pmatrix} \frac{(1-\pi)\beta\Lambda}{\xi+\mu} + (1-\delta)\frac{(\mu+\xi)\pi\Lambda\beta + (1-\pi)\beta\Lambda\xi}{\mu(\mu+\xi)} & 0 \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (\mu + \kappa + \alpha) & 0 \\ -\alpha & (\lambda + \mu + \sigma) \end{pmatrix}$$

with

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu + \kappa + \alpha} & 0 \\ \frac{-\alpha}{(\mu + \kappa + \alpha)(\lambda + \mu + \sigma)} & \frac{1}{(\lambda + \mu + \sigma)} \end{pmatrix}$$

Thus,

$$FV^{-1} = \begin{pmatrix} \frac{1}{\mu + \kappa + \alpha} \cdot \left( \frac{(1-\pi)\beta\Lambda}{\xi+\mu} + (1-\delta)\frac{(\mu+\xi)\pi\Lambda\beta + (1-\pi)\beta\Lambda\xi}{\mu(\mu+\xi)} \right) & 0 \\ 0 & 0 \end{pmatrix}$$

The eigenvalues of  $FV^{-1}$  are

$$X_1 = 0$$

$$X_2 = \frac{\beta\Lambda}{\mu(\mu + \kappa + \alpha)} \left[ \frac{(1-\pi)\mu + (1-\pi)(1-\delta)\xi + (1-\delta)(\mu + \xi)\pi}{(\xi + \mu)} \right]$$

Therefore, the effective reproduction number is given by

$$R_{SE} = \frac{\beta\Lambda}{(\mu + \kappa + \alpha)} \left[ \frac{(1-\pi)}{\mu + \xi} + \frac{(1-\delta)\pi}{\mu} + \frac{(1-\delta)(1-\pi)\xi}{\mu(\mu + \xi)} \right] \quad (4.6)$$

In the absence of social media awareness,  $\xi = 0$  and  $\pi = 0$  and the effective reproduction

number reduces to

$$R_0 = \frac{\beta\Lambda}{\mu(\mu + \kappa + \alpha)}$$

This implies that

$$R_{SE} = R_0 \left[ \frac{(1-\pi)\mu + (1-\pi)(1-\delta)\xi + (1-\delta)(\mu + \xi)\pi}{(\xi + \mu)} \right] \quad (4.7)$$

Since  $0 < (1-\delta) < 1$ , it follows that

$$\left( \frac{(1-\pi)\mu + (1-\pi)(1-\delta)\xi + (1-\delta)(\mu + \xi)\pi}{(\xi + \mu)} \right) < 1$$

which implies that  $R_{SE} < R_0$ . When  $\pi = \xi = 0$ ,  $R_{SE} = R_0$ . Thus from equation (4.7), it is clear that social media creation awareness on transmission of tungiasis has a positive impact on the reduction of new infections.

### 4.3 Existence of A Unique Positive Endemic Equilibrium

In the following Lemma, we explore the existence of a unique positive endemic equilibrium for the model system (2.1).

**Lemma 4.1.** *A unique endemic equilibrium  $E^* = (S_N^*, S_E^*, I^*, C^*, R^*)$  exists provided that  $R_{SE} > 1$*

*Proof.* At disease endemic equilibrium (DEE),  $I \neq 0$  and solving for  $S_N^*, S_E^*, C^*, R^*$  in (4.1), we obtain

$$S_N^* = \frac{b_1(1-\pi)\Lambda + b_2I^*}{b_1(\beta I^* + b_4)} \quad (4.8)$$

$$S_E^* = \frac{\pi\Lambda(b_1\beta I^* + b_1b_4) + \xi b_1(1-\pi)\Lambda + b_2I^*\xi}{(b_1\beta I^* + b_1b_4)(\mu + (1-\delta)\beta I^*)} \quad (4.9)$$

$$C^* = \frac{\alpha}{b_3}I^* \quad (4.10)$$

$$R^* = \frac{b_5}{b_1}I^* \quad (4.11)$$

Using equation (4.4), we can also express  $S_N^*$  as

$$S_N^* = \frac{b_6(\mu + (1-\delta)\beta I^*) - (1-\delta)\beta\pi\Lambda}{[\mu + (1-\delta)\beta I^* + (1-\delta)\xi]\beta} \quad (4.12)$$

where

$$\begin{aligned} b_1 &= (\mu + \omega)(\lambda + \mu + \sigma), & b_2 &= \omega\alpha(\lambda + \mu + \sigma) + \omega\alpha\lambda, & b_3 &= \lambda + \mu + \sigma, \\ b_4 &= \mu + \xi, & b_5 &= \kappa(\lambda + \mu + \sigma) + \alpha\lambda & \text{and} & & b_6 &= \mu + \kappa + \alpha \end{aligned}$$

Equating equations (4.8) and (4.12), we obtain after a lengthy computation

$$A_2I^{*2} + A_1I^* + A_0 = 0 \quad (4.13)$$

Where

$$\begin{aligned} A_2 &= (1-\delta)\beta^2\{b_1b_6 - b_2\} \\ &= (1-\delta)\beta^2\{\mu b_3(b_6 + \omega) + \alpha\omega(\mu + \sigma)\} > 0 \Rightarrow b_1b_6 > b_2, \text{i.e.} \\ &(\mu + \omega)(\lambda + \mu + \sigma)(\mu + \kappa + \alpha) > \omega\kappa(\lambda + \mu + \sigma) + \omega\alpha\lambda, \end{aligned}$$

$$A_1 = (1 - \delta)\beta b_1(b_4 b_6 - \Lambda\beta) + \beta(\mu b_1 b_6 - b_2[\mu + (1 - \delta)\xi])$$

By definition  $b_4 b_6 > \Lambda\beta$  and since  $A_2 > 0$ , we see that  $\mu b_1 b_6 > b_4 b_2$ . Thus  $A_1 > 0$  and

$$A_0 = -\mu b_1 b_4 b_6 [R_{SE} - 1] \quad (4.14)$$

Where  $R_{SE}$  is as given in (4.6).

By the Descartes' Rule of Signs, existence of a positive root now depends on the sign of  $A_0$ .  $A_0$  has to be negative for a positive root,  $I^* > 0$  to exist. But from (4.14) it follows that  $A_0 < 0$  if  $R_{SE} > 1$   $\square$

With this information, one can approximate  $I^*$  using the quadratic equation in (4.13) as follows

$$\begin{aligned} I^* &\approx \frac{2A_2 \mu b_1 b_4 b_6 [R_{SE} - 1]}{A_1} \\ &= M(R_{SE} - 1) \end{aligned}$$

where  $M = \frac{2A_2 \mu b_1 b_4 b_6}{A_1} > 0$ . The endemic equilibrium point  $E^*$  can then be easily obtained using equations (4.8), (4.9), (4.10), (4.11).

## 5 Stability Analysis of Equilibria

### 5.1 Local Stability and Disease Free Equilibrium

In this section, we investigate local stability of the DFE,  $E^0$

**Theorem 5.1.** *The disease-free equilibrium of the model system (2.1) is locally asymptotically stable whenever  $R_{SE} < 1$  and unstable whenever  $R_{SE} > 1$*

*Proof.* It suffices to show that the eigenvalues of The Jacobian matrix of the mode system (2.1) have real negative parts. The Jacobian matrix at DFE,  $E^0$ , is given by

$$J(E^0) = \begin{pmatrix} -(\mu + \xi) & 0 & \frac{-\beta(1-\pi)\Lambda}{\mu + \xi} & 0 & \omega \\ \xi & -\mu & -(1-\delta)\frac{\beta(\mu + \xi)\pi\Lambda + \beta\xi(1-\pi)\Lambda}{\mu(\mu + \xi)} & 0 & 0 \\ 0 & 0 & \frac{\beta(1-\pi)\Lambda}{\mu + \xi} + (1-\delta)\frac{\beta(\mu + \xi)\pi\Lambda + \beta\xi(1-\pi)\Lambda}{\mu(\mu + \xi)} - (\mu + \kappa + \alpha) & 0 & 0 \\ 0 & 0 & \alpha & -(\lambda + \mu + \sigma) & 0 \\ 0 & 0 & \kappa & \lambda & -(\omega + \mu) \end{pmatrix}$$

The characteristic equation is given by  $|(J(E^0) - XI)| = 0$ , which yields

$$(\mu + \xi + X)(\mu + X) \left[ (\mu + \kappa + \alpha)(R_{SE} - 1) - X \right] (\lambda + \mu + \sigma + X)(\mu + \omega + X) = 0$$

Thus,



$$\begin{aligned} X_1 &= -(\mu + \xi) \\ X_2 &= -\mu \\ X_3 &= (R_{SE} - 1)(\mu + \kappa + \alpha) \\ X_4 &= -(\lambda + \mu + \sigma) \\ X_5 &= -(\omega + \mu) \end{aligned}$$

Where  $R_{SE}$  is as given in (4.6). All eigenvalues are negative provided  $R_{SE} < 1$ . If  $R_{SE} > 1$ , then  $X_3 > 0$  and therefore DFE is unstable.  $\square$

## 5.2 Global Stability of The Disease Free Equilibrium

In this section, we investigate the global stability of the DFE,  $E^0$ .

**Theorem 5.2.** *The disease free equilibrium*

$$E^0 = \left( \frac{(1-\pi)\Lambda}{\mu + \xi}, \frac{(\mu + \xi)\pi\Lambda + \xi(1-\pi)\Lambda}{\mu(\mu + \xi)}, 0, 0, 0 \right)$$

of the model system (2.1) is globally asymptotically stable (GAS) if  $R_{SE} \leq 1$ .

*Proof.* Let

$$S_N^0 = \frac{(1-\pi)\Lambda}{\mu + \xi} \quad \text{and} \quad S_E^0 = \frac{(\mu + \xi)\pi\Lambda + \xi(1-\pi)\Lambda}{\mu(\mu + \xi)}$$

and consider the following combination of linear functions and Volterra-type Lyapunov function:

$$L = L(S_N, S_E, I, C, R) = S_N - S_N^0 \ln S_N + S_E - S_E^0 \ln S_E + I + a_1 C + a_2 R$$

where  $a_1$  and  $a_2$  are constants to be determined.  $L$  is defined and continuous. It suffices to show that  $\frac{dL}{dt} \leq 0$ . Therefore, the derivative of  $L$  in the direction of the vector field given by the right-hand side of the equation (2.1) is

$$\begin{aligned} \frac{dL}{dt} &= \frac{dS_N}{dt} \left( 1 - \frac{S_N^0}{S_N} \right) + \frac{dS_E}{dt} \left( 1 - \frac{S_E^0}{S_E} \right) + \frac{dI}{dt} + a_1 \frac{dC}{dt} + a_2 \frac{dR}{dt} \\ &= \frac{-(\mu + \xi)}{S_N} (S_N - S_N^0)^2 - \frac{\mu}{S_E} (S_E - S_E^0)^2 \\ &\quad + [\beta S_N^0 + (1 - \delta)\beta S_E^0 - (\mu + \kappa + \alpha) + a_1 \alpha + a_2 \kappa] I \\ &\quad + [a_2 \lambda - a_1 (\lambda + \mu + \sigma)] C + \left[ \omega \left( \frac{S_N - S_N^0}{S_N} \right) - a_2 (\omega + \mu) \right] R \\ &\quad + \xi S_N \left( 1 - \frac{S_N^0}{S_N} \right) \left( 1 - \frac{S_E^0}{S_E} \right) \end{aligned}$$

Choose  $a_1$  and  $a_2$  such that

$$\begin{aligned} a_2 \lambda - a_1 (\lambda + \mu + \sigma) &= 0 \\ \omega \left( \frac{S_N - S_N^0}{S_N} \right) - a_2 (\omega + \mu) &= 0 \end{aligned}$$

Thus

$$\begin{aligned} a_2 &= \frac{\omega}{\mu + \omega} \left( 1 - \frac{S_N^0}{S_N} \right) \\ a_1 &= \frac{\omega \lambda}{(\mu + \omega)(\lambda + \mu + \sigma)} \left( 1 - \frac{S_N^0}{S_N} \right). \end{aligned}$$

With this in mind,  $\frac{dL}{dt}$  becomes

$$\begin{aligned} \frac{dL}{dt} = & \frac{-(\mu + \xi)}{S_N} (S_N - S_N^0)^2 - \frac{\mu}{S_E} (S_E - S_E^0)^2 + (\mu + \kappa + \alpha)(R_{SE} - 1)I \\ & + \left( \frac{\omega \lambda \alpha I}{(\mu + \omega)(\lambda + \mu + \sigma)} + \frac{\omega \kappa I}{\mu + \omega} \right) \left( 1 - \frac{S_N^0}{S_N} \right) + \xi S_N \left( 1 - \frac{S_N^0}{S_N} \right) \left( 1 - \frac{S_E^0}{S_E} \right) \end{aligned}$$

Since the arithmetic mean exceeds the geometric mean, the following inequalities hold

$$\begin{aligned} \left( \frac{\omega \lambda \alpha I}{(\mu + \omega)(\lambda + \mu + \sigma)} + \frac{\omega \kappa I}{\mu + \omega} \right) \left( 1 - \frac{S_N^0}{S_N} \right) & \leq 0 \\ \xi S_N \left( 1 - \frac{S_N^0}{S_N} \right) \left( 1 - \frac{S_E^0}{S_E} \right) & \leq 0 \end{aligned}$$

Thus  $\frac{dL}{dt} \leq 0$  if  $R_{SE} \leq 1$  with equality if  $S_N = S_N^0, S_E = S_E^0$ , and  $R_{SE} = 1$ . Since it is easy to show that the largest invariant subset contained in the set

$$L : \{ (S_N, S_E, I, C, R) \in \Omega : \frac{dL}{dt} = 0 \}$$

is the DFE,  $E^0$ , it follows from Lasalles Invariance Principal [21], that every solution of the equations in the model system (2.1) with initial conditions in  $\Omega$ , approaches  $E^0$  as  $t \rightarrow \infty$  for  $R_{SE} \leq 1$   $\square$

### 5.3 Local Stability of The Endemic Equilibrium

**Theorem 5.3.** *If*

$$(2 - \delta)\beta M(R_{SE} - 1) + \mu + \kappa + \alpha > (1 - \delta)\beta S_E^* \quad (5.1)$$

where  $S_E^*$  is as given in (4.9), then the disease-endemic equilibrium of the model system (2.1) is locally asymptotically stable

*Proof.* Since  $N(t) \rightarrow \frac{\Lambda}{\mu}$  as  $t \rightarrow \infty$  we can express  $S_N$  as

$$S_N = \frac{\Lambda}{\mu} - S_E - I - C - R$$

Thus, it is enough to study the stability of the subsystem

$$\begin{aligned} \frac{dS_E}{dt} &= \pi \Lambda + \xi \left( \frac{\Lambda}{\mu} - S_E^* - I^* - C^* - R^* \right) - (\mu + (1 - \delta)\beta I)S_E \\ \frac{dI}{dt} &= \beta \left( \frac{\Lambda}{\mu} - S_E - I - C - R \right) I + (1 - \delta)\beta S_E I - (\mu + \kappa + \alpha)I \\ \frac{dC}{dt} &= \alpha I - (\lambda + \mu + \sigma)C \\ \frac{dR}{dt} &= \kappa I + \lambda C - (\mu + \omega)C \end{aligned}$$

The Jacobian matrix at the equilibrium point  $S_E^*, I^*, C^*, R^*$  is

$$J(E^*) = \begin{pmatrix} -\xi - \mu - (1 - \delta)\beta I^* & -\xi - (1 - \delta)\beta S_E^* & -\xi & -\xi \\ -\delta\beta I^* & -2\beta I^* + (1 - \delta)\beta S_E^* - (\mu + \kappa + \alpha) & -\beta I^* & -\beta I^* \\ 0 & \alpha & -(\lambda + \mu + \sigma) & 0 \\ 0 & \kappa & \lambda & -(\mu + \omega) \end{pmatrix}$$

Let

$$\begin{aligned} c_1 &= \mu + \xi + (1 - \delta)\beta I^*, & c_2 &= -2\beta I^* + (1 - \delta)\beta S_E^* - (\mu + \kappa + \alpha) \\ c_3 &= \alpha\lambda + \kappa b_3, & c_4 &= \xi + (1 - \delta)\beta S_E^* \\ b_3 &= \lambda + \mu + \sigma, & b_4 &= \mu + \xi, & b_6 &= \mu + \kappa + \alpha, & b_7 &= \mu + \omega \end{aligned}$$

$$(J - XI) = \begin{vmatrix} -c_1 - X & -c_4 & -\xi & -\xi \\ -\delta\beta I^* & c_2 - X & -\beta I^* & -\beta I^* \\ 0 & \alpha & -b_2 - X & 0 \\ 0 & \kappa & \lambda & -b_7 - X \end{vmatrix}$$

The characteristic polynomial is then

$$\begin{aligned} &X^4 + (c_1 - c_2 + b_3 + b_7)X^3 + c_1(b_3 + b_7 - c_2) + [b_3b_7 - c_2b_3 - c_2b_7 + \alpha\beta I^* + \beta\kappa I^* \\ &- \delta\beta c_4 I^*]X^2 + [b_3b_7 - c_2b_3 - c_2b_7 + c_1\alpha\beta I^* + c_1\beta\kappa I^* - c_2b_3b_7 + \alpha\beta I^* + c_3\beta I^* \\ &- \delta\beta I^* c_4(b_3 + b_7) - \delta\beta I^* c_4\alpha\xi - \delta\beta I^* c_4\kappa\xi]X + c_1\alpha\beta b_7 I^* - c_1c_2b_3b_7 + c_1c_3\beta I^* \\ &- \delta\beta I^* c_4b_3b_7 - \delta\beta I^* \alpha\xi b_7 - \delta\beta I^* \xi c_3 \end{aligned}$$

For negative eigenvalues, it is enough to show that the independent term  $a_4$  in

$$X^4 + a_1X^3 + a_2X^2 + a_3X + a_4$$

where

$$a_4 = c_1(c_3\beta I^* + \alpha\beta b_7 I^* - c_2b_3b_7) - \delta\beta I^*(c_4b_3b_7 + \alpha\xi b_7 + \xi c_3)$$

is positive, see [2] for more details. Rewriting  $a_4$  we have

$$\begin{aligned} a_4 &= c_1(c_3\beta I^* + \alpha\beta b_7 I^* - c_2b_3b_7) - \delta\beta I^*(c_4b_3b_7 + \alpha\xi b_7 + \xi c_3) \\ &= (\lambda + \mu + \sigma)(\mu + \omega)\{\delta\beta\mu I^* + [\mu + \xi + (1 - \delta)\beta S_E^*][\mu + \kappa + \alpha + (2 - \delta)\beta I^* \\ &- (1 - \delta)\beta S_E^*]\} + \{\xi\kappa(\lambda + \mu + \sigma)\beta I^*(1 - \delta) + \alpha\xi(\mu + \omega)(1 - \delta)\beta I^* + \alpha\lambda\xi + \\ &[\mu + (1 - \delta)\beta S_E^*][\alpha\lambda + \kappa(\lambda + \mu + \sigma)\beta I^* + \alpha(\mu + \omega)\beta I^*] - \alpha\lambda\xi\delta\beta I^*\} \end{aligned}$$

The sum of in the second curly bracket is clearly positive. The sum in the first curly bracket is positive if

$$(2 - \delta)\beta I^* + (\mu + \kappa + \alpha) > (1 - \delta)\beta S_E^* \quad (5.2)$$

By the results in [2], it follows that the endemic equilibrium point is locally asymptotically stable provided condition (5.2) is satisfied.  $\square$

## 6 Numerical Simulation

Numerical simulations were carried using MATLAB R2021a to graphically illustrate the long term effect of social media intervention on the dynamics Tungiasis infestation. The simulation of the model above is done using parameter values shown in table below

Table 1: Detailed description of the state variables and relevant parameters of the proposed  $[S_N, S_E, I, C, R]$  Tungiasis Model

Detailed Description	Symbol	Value	Source
Susceptible Population	$S_N(t)$	$5.478 \times 10^7$	[36]
Mildly Infected Population	$I(t)$	$6.75 \times 10^4$	[1]
Chronically Infected Population	$C(t)$	$1.0 \times 10^1$	[1]
Fraction Exposed to Social Media	$\pi$	$3.995 \times 10^2$	Assumed
Mildly Infected Recovery Rate	$\kappa$	$6.975 \times 10^{-1}$	Assumed
Chronic Infection Rate	$\alpha$	$1.5 \times 10^{-8}$	Assumed
Susceptible Recruitment Rate	$\Lambda$	$1.245 \times 10^{-6}$	[3]
Natural Death Rate	$\mu$	$5.2 \times 10^{-8}$	[36]
Infestation Death Rate	$\sigma$	$2.607 \times 10^{-9}$	[22]
Recovered Reverting Rate to Susceptible	$\kappa$	$6.75 \times 10^{-1}$	Assumed
Effective Contact Transmission Rate	$\beta$	$3.2398 \times 10^{-9}$	[22]
Success Rate of Social Media Intervention	$\delta$	[0,1]	Assumed
Dissemination Rate of Control Strategies	$\xi$	$5.99 \times 10^{-3}$	Assumed
Chronically Infested Recovery Rate	$\lambda$	$1.375 \times 10^{-1}$	[22]
Recovered Reverting to Susceptible Population	$\omega$	$3.75 \times 10^{-4}$	Assumed

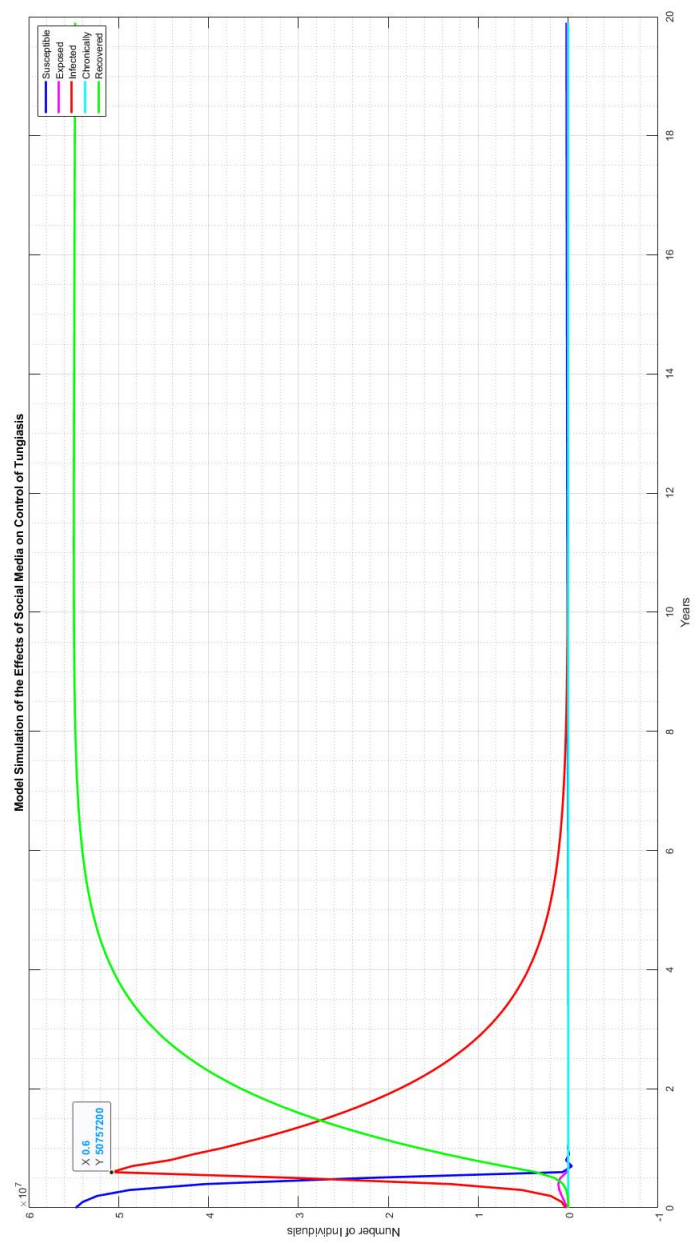


Figure 2: Effects of Social Media on Control of Tungiasis  $\delta = 0.18$

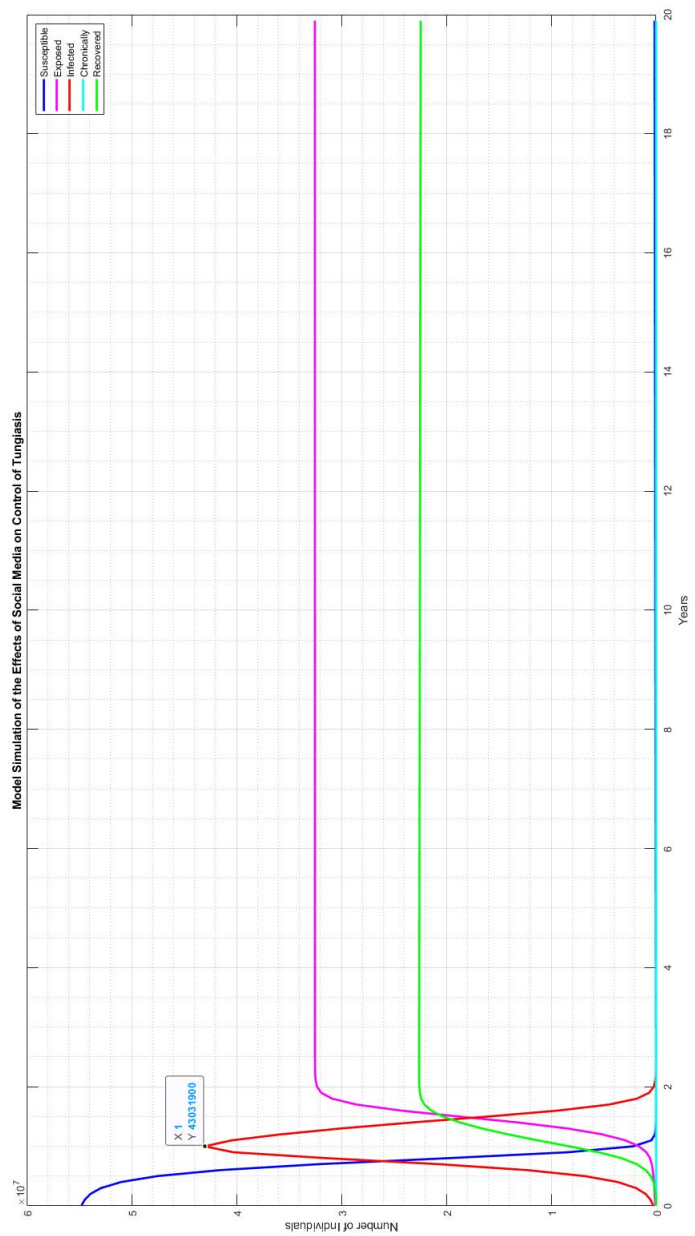


Figure 3: Effects of Social Media on Control of Tungiasis  $\delta = 0.23$

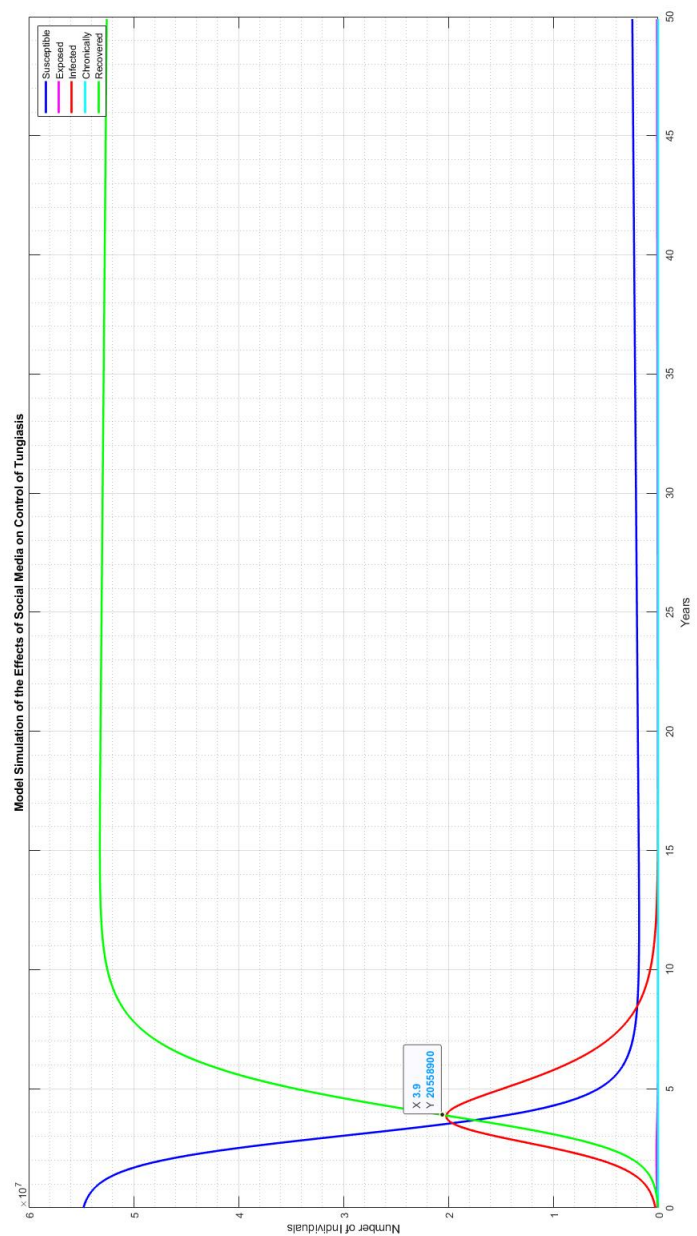


Figure 4: Effects of Social Media on Control of Tungiasis  $\delta = 0.32$

## 7 Discussion

From figure 4 we see that the population of the susceptible individuals immediately begins to drop because of the highest degree of Tungiasis infestation, consequently the removed individuals starts to rise. The number of tungiasis infested individuals increase then come to a decrease. At the same time, the population of those who are removed rises swiftly then reaches the peak showing the biological reality that tungiasis infestation can be fatal. The model is realistic to show the situation. therefore, the medical practitioners, health departments and stakeholders should focus on this moment. Moreover the population of the susceptibles also decreases at this time as more people get infected showing that the spread of the disease is high and should be controlled.

From 4 3 and 2 with  $\delta = 0.32$ ,  $\delta = 0.23$  and  $\delta = 0.18$  respectively shows that the number of infested individuals reduces from  $5.0 \times 10^7$  to  $2.0 \times 10^7$ .

$\delta$	Infested Population
0.18	$5.075 \times 10^7$
0.23	$4.303 \times 10^7$
0.32	$2.05 \times 10^7$

From the graphs, we see the relation between social media intervention and the population infested with tungiasis. It is clear that when the social media intervention success rate is increased, the number of those infected with tungiasis decreased.



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