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Modelling prevention and control of jigger infestation in Mayuge district: a mathematical approach

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Abstract

Tungiasis is a disease caused by the smallest jigger flea and found in many poor communities. Although its control involves wearing closed shoes or extraction of the flea using a very cheap sterile safety pin, the disease continues to cause death or rotting away of infested people. Numerous super infestation and deaths have been reported in Uganda, in particular, Mayuge district. This has posed a major threat to the already wanting education standards in the region. In this study, a mathematical model is presented aimed at assessing the effects of persistent jigger infestation in Mayuge district, and how it could be controlled. We use to controls, one to prevent the adult female flea from burrowing into the human host, and the other by extraction of the embedded flea from the infected human and killing the eggs to prevent further infection. Results show that without control, the disease-free equilibrium point is stable for those values of $0.3 < \mathcal{R}_0 < 1$, otherwise it is unstable. With maximum control, the disease-free is stable for values of $0.0103 < \mathcal{R}_0 < 1$. Numerical results show that if the infection rate β is less than 0.3, with other parameters kept constant, $\mathcal{R}_0$ can be controlled to less than unity and jigger infestation stopped. Numerical results further indicate that to avoid super infestation, all efforts to extract the jigger once embedded must be enforced. With the current presence of super infestation in Mayuge district, a single individual can cause more than 7 new infestations. To control this, efforts must be made to reduce the current prevalence of jigger infestation from 0.25 to 0.004. We conclude from the study that jigger infestation has an important social dimension and affects human rights. It is a zoonosis and considering its links with poverty, requires multi-disciplinary research in which public health, social sciences, health education, and animal husbandry need to interact.

Keywords: Tunga penetrans, mathematical modelling.

2020 MSC: 49J75, 92B05, 92D30, 93A30.

1. Introduction

Jiggers, also called *nvunza, chica, chigoe, pico, suthi, ivunja, liyanzi, funza, pique, nigua, tunga penetrans* and tū are burrowing fleas found in most tropical and subtropical climates [3]. The jigger is the smallest known flea and lives in soil and sand, feeding on warm-blooded hosts [27], apart from rats [21]. The female flea attacks by burrowing head-first into the host's skin where it remains and within two weeks

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swells due to eggs inside its abdomen [22]. The eggs are released to the ground when ready to hatch [6, 21] and the flea dies. The eggs hatch in the next three to four days and mature into adult fleas within three to four weeks. The entire process from burrowing into host to mature flea takes four to six weeks [6]. More details are shown in Figure 1.

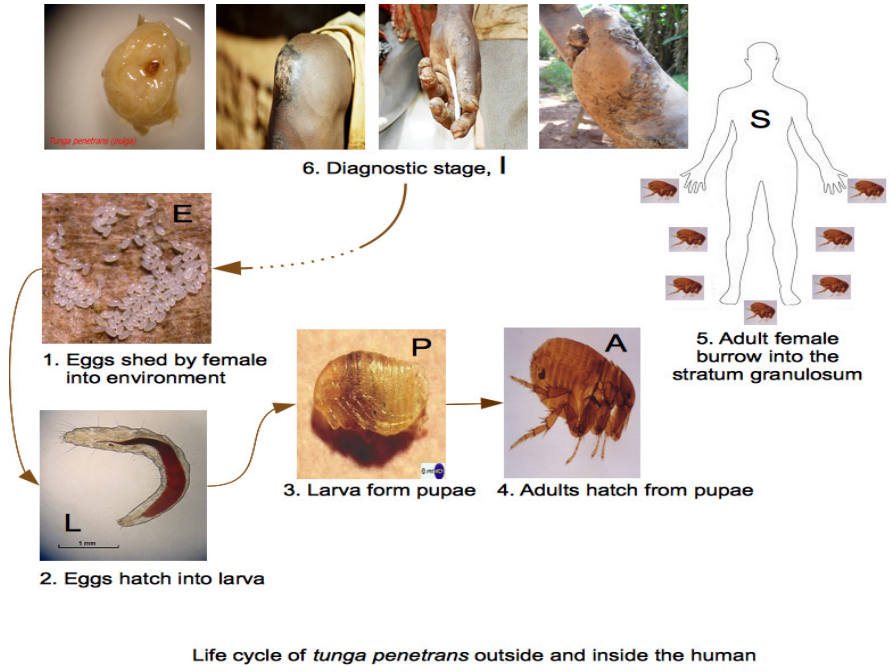

Figure 1: The cycle of *tunga penetrans*. Images extracted from [36].

The disease caused by the jigger is called tungiasis due to the intense irritation that result from breeding female chigoes inside the infected individual [21]. When left inside the host, it may result in loss of nails, toe deformation and secondary infections that lead to severe morbidity [6]. The flea burrows mostly in the host's feet because they spend most of their time on the ground [22]. In severe cases, the affected part of the body rots away [10, 36]. The disease is reported mainly in poor rural communities of Brazil [6, 21, 32], Tanzania [20], Nigeria [28] and most notably, Uganda [10, 25], where it has been reported to be at epidemic levels.

Mayuge district is found in the Eastern part of Uganda, bordered by Iganga district to the north, Bugiri district to the northeast, Namayingo district to the east, the Republic of Tanzania to the south, and Jinja district to the west [35]. Jigger infestation in Mayuge district started in homes of people and later spread to schools [16]. This posed a major threat to the already wanting education standards in the region. Researchers have established that the hemorrhagic jigger infestation in Busoga region is a result of poor hygiene. Infestation with jiggers has serious health consequences and is associated with considerable morbidity and difficulties in walking or using the hands, preventing the victims from leading productive life. Persistent infestation produces even more extremely serious outcomes such as disability and mortality due to secondary infections. Prevention of jigger infestation involves using closed shoes in contaminated soil, disinfection, and immediate extraction of embedded fleas [18, 19]. Within households, smearing floors with cow dung stops jigger infestation [31]. In generalized infestation, oral *thiabendazole* and *ivermectin* are successful treatments [9]. Although the progression and symptoms of tungiasis can be treated [9, 18, 19], it continues in poor communities [6, 10, 21, 28, 32, 34] causing deaths or rotting away of infested people due to ignorance, and perhaps neglect by respective health officials.

This study aimed at assessing the effects of persistent jigger infestation in Mayuge district, using a mathematical model. As of 2013, more than 300 families were reportedly infested with jiggers in Mayuge district, two years after government launched a campaign to curb the spread of the parasites in Busoga

region [31]. The prevalence of jigger infestation in Mayuge has been found to be 25.0%, where 58.3% had persistent jigger infestation [23]. Research has found that the factors that increased the likelihood of persistent jigger infestation in Mayuge district included low monthly income, littered compound, earthen floor, and cracked walls [23]. However, individuals knowledgeable about jigger prevention had reduced chances of being persistently infested with jiggers. Mathematical models have for long been used to study disease dynamics in humans and animals. Studies such as those in [5, 10–12, 15, 17, 24, 26, 32] predict the potential grave threat to public health due to disease outbreaks and re-emergency [2, 7, 8]. The practices that could eradicate jigger infestation in Mayuge district for example, are well known and have been applied, without evident success. It appears as though that either the practices are not effectively carried out, or that the infested individuals are not conversant with the epidemiology of the disease. It beats everyone's understanding that an entire family could die of jiggers, when it can be extracted using a very cheap sterile safety pin [18, 19]. The study in this manuscript is aimed at an in-depth analysis and understanding of the dynamics of tungiasis in humans. Two possible control methods in humans are considered. The first is stopping the adult female flea from burrowing into the human host by wearing closed shoes. The second control is by extraction of the embedded flea from the infected human and killing the eggs in the swollen flea. Although domestic animals are known to be reservoirs [27], we show dynamics of the disease in humans only. The steady states of the model are obtained and analyzed for stability and numerical simulation carried out to predict the disease outcome.

2. Model description and analysis

The model designed in this paper is Susceptible-Infected-Recovered SIR, in the human population and Egg-Larva-Pupa-Adult ELPA, in the flea. A susceptible human S, becomes infected I, when an adult female flea A burrows into the *stratum granulosum* of the individual and remains there. The force of infection is therefore set to βSA where β is the rate at which the adult jiggers attack susceptible humans. This rate incorporates both the number of contacts with contaminated environment and the probability of successful embedment of the adult fleas A into the stratum granulosum. Once infested, the human can either die due to the infection at a rate δ, or may remove the jigger before full development and recover at a per capita rate γ to join the recovered group R. If the human does not remove the flea within a period of $1/\tau_i$ days, it remains under the victim's skin and swells due to n eggs E inside its abdomen [22]. The eggs are released to the ground when ready to hatch [6, 21] and the flea dies. In the next $1/\tau_e$ days, the eggs hatch into larvae, L. The larvae then forms the pupae P after $1/\tau_l$ days, which then hatches into the adult flea A within $1/\tau_p$ days. The adult flea burrows into the non suspecting human and the cycle continues (see Figure 2).

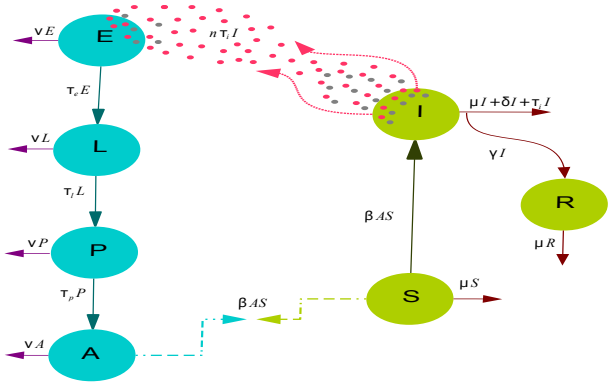

Figure 2: The flux diagram derived from the cycle of tungiasis.

It is assumed that the adult flea can only penetrate a non infected human, thereby ignoring super infestation. These assumptions and definitions give rise to the following system of differential equations:

$$\begin{aligned} S' &= b - (1-C_1)\beta AS - \mu S, \\ I' &= (1-C_1)\beta AS - (\mu+\delta+\gamma+\tau_i)I, \\ R' &= \gamma I - \mu R, \\ E' &= n(1-C_2)\tau_i I - (\nu+\tau_e)E, \\ L' &= \tau_e E - (\nu+\tau_l)L, \\ P' &= \tau_l L - (\nu+\tau_p)P, \\ A' &= \tau_p P - \nu A - (1-C_1)\beta AS, \end{aligned} \tag{2.1}$$

where b and μ are the respective per capita growth and death rates in the human host in Mayuge district, and ν is the natural death rate in the flea population. Control level C_1, where $0 \leqslant C_1 \leqslant 1$ is for preventing the adult female flea from burrowing into the human host by wearing closed shoes, while C_2, with $0 \leqslant C_2 \leqslant 1$ is the control by extraction of the embedded flea from the infected human and killing the eggs to prevent further infection. When C_1, C_2 are zero, there is no control and the disease prevails; when C_1, C_2 equal to one, the disease is controlled and dies out. This model is analyzed for existence and stability of steady states.

2.1. Equilibrium points and their stability

Equilibrium points are obtained by setting the right hand side of equation (2.1) to zero and solve the resulting system. For the disease-free point E_0, I, R, E, L, P, and A are all set to zero giving

$$E_0 = (S_0, I_0, R_0, E_0, L_0, P_0, A_0) = \left(\frac{b}{\mu}, 0, 0, 0, 0, 0, 0\right).$$

The endemic equilibrium point E_e is obtained when $I, R, E, L, P, A \neq 0$. This is given by

$$E_e = \begin{cases} S_* = \frac{\nu}{\beta(\frac{\mathcal{R}_0}{\beta}-1)}, \\ I^* = \frac{b}{(\mu+\delta+\gamma+\tau_i)}\left[\frac{\frac{\beta}{\mathcal{R}_0}+\frac{\mu\nu}{b\mathcal{R}_0}-1}{\frac{\beta}{\mathcal{R}_0}-1}\right], \\ R^* = \frac{\gamma b}{\mu(\mu+\delta+\gamma+\tau_i)}\left[\frac{\frac{\beta}{\mathcal{R}_0}+\frac{\mu\nu}{b\mathcal{R}_0}-1}{\frac{\beta}{\mathcal{R}_0}-1}\right], \\ E_* = \frac{bn(1-C_2)\tau_i}{(\nu+\tau_e)(\mu+\delta+\gamma+\tau_i)}\left[\frac{\frac{\beta}{\mathcal{R}_0}+\frac{\mu\nu}{b\mathcal{R}_0}-1}{\frac{\beta}{\mathcal{R}_0}-1}\right], \\ L_* = \frac{bn(1-C_2)\tau_i\tau_e}{(\nu+\tau_e)(\nu+\tau_l)(\mu+\delta+\gamma+\tau_i)}\left[\frac{\frac{\beta}{\mathcal{R}_0}+\frac{\mu\nu}{b\mathcal{R}_0}-1}{\frac{\beta}{\mathcal{R}_0}-1}\right], \\ P_* = \frac{b\mathcal{R}_0}{\beta\tau_p}\left[\frac{\frac{\beta}{\mathcal{R}_0}+\frac{\mu\nu}{b\mathcal{R}_0}-1}{\frac{\beta}{\mathcal{R}_0}-1}\right], \\ A_* = \frac{b}{\nu\beta}\left[1-\frac{\beta}{\mathcal{R}_0}-\frac{\mu\nu}{b\mathcal{R}_0}\right], \end{cases}$$

where

$$\mathcal{R}_0 = \frac{(1-C_1)\beta n(1-C_2)\tau_i\tau_e\tau_l\tau_p}{(\nu+\tau_e)(\nu+\tau_l)(\nu+\tau_p)(\mu+\delta+\gamma+\tau_i)}. \tag{2.2}$$

$\mathcal{R}_0$ is the basic reproduction ratio defined as the number of secondary infestions that result from a single infective infested with a single jigger, when introduced into a jigger-free population. When $\mathcal{R}_0 < 1$, the disease does not prevail, and when $\mathcal{R}_0 > 1$, the disease prevails leading to endemicity. Therefore, $\mathcal{R}_0 < 1$ leads to stability of disease-free state, while $\mathcal{R}_0 > 1$ leads to stability of the endemic state [29].

The disease-free steady state E_0 and the endemic point E_e are studied to determine the nature of their stability. The disease-free equilibrium is studied from the Jacobian of the system at the point E_0. The Jacobian of the model in equation (2.1) at steady state $E_* = (S_*, I_*, R_*, E_*, L_*, P_*, A_*)$ is given by

$$D_{E_*} = [J_{11}, J_{12}], \tag{2.3}$$

where

$$J_{11} = \begin{bmatrix} -(1-C_1)\beta A_* - \mu & 0 & 0 & 0 \\ (1-C_1)\beta A^* & -(\mu+\delta+\gamma+\tau_i) & 0 & 0 \\ 0 & \gamma & -\mu & 0 \\ 0 & n(1-C_2)\tau_i & 0 & -(\nu+\tau_e) \\ 0 & 0 & 0 & \tau_e \\ 0 & 0 & 0 & 0 \\ -(1-C_1)\beta A_* & 0 & 0 & 0 \end{bmatrix},$$

$$J_{12} = \begin{bmatrix} 0 & 0 & -(1-C_1)\beta S_* \\ 0 & 0 & (1-C_1)\beta S_* \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ -(\nu+\tau_l) & 0 & 0 \\ \tau_l & -(\nu+\tau_p) & 0 \\ 0 & \tau_p & -(\nu+(1-C_1)\beta S_*) \end{bmatrix}.$$

At the disease-free equilibrium point E_0, two of the eigenvalues of the Jacobian (2.3) are observed to be negative and equal to $-\mu, -\mu$. The dynamics of the system can then be studied using the remaining 5×5 sub matrix given by

$$\begin{bmatrix} -(\mu+\delta+\gamma+\tau_i) & 0 & 0 & 0 & (1-C_1)\beta b/\mu \\ n(1-C_2)\tau_i & -(\nu+\tau_e) & 0 & 0 & 0 \\ 0 & \tau_e & -(\nu+\tau_l) & 0 & 0 \\ 0 & 0 & \tau_l & -(\nu+\tau_p) & 0 \\ 0 & 0 & 0 & \tau_p & -(\nu+(1-C_1)\beta b/\mu) \end{bmatrix}. \tag{2.4}$$

For stability, the trace of the Jacobian (2.4) must be less than zero, while the determinant greater than zero. It is observed from Jacobian (2.4) that the trace is negative since

$$-(\mu+\delta+\gamma+\tau_i+4\nu+\tau_e+\tau_l+\tau_p+(1-C_1)\beta b/\mu) < 0.$$

To prove stability of the disease-free state, it suffices to show that the determinant is greater than zero when $\mathcal{R}_0 < 1$. Computing the determinant of Jacobian (2.4) gives

$$\begin{aligned} \frac{(1-C_1)\beta b}{\mu} n(1-C_2)\tau_i\tau_e\tau_l\tau_p - (\mu+\delta+\gamma+\tau_i)(\nu+\tau_e)(\nu+\tau_l)(\nu+\tau_p)(\nu+(1-C_1)\beta b/\mu) > 0, \\ \Rightarrow \frac{(1-C_1)\beta b n(1-C_2)\tau_i\tau_e\tau_l\tau_p}{(\mu+\delta+\gamma+\tau_i)(\nu+\tau_e)(\nu+\tau_l)(\nu+\tau_p)(\mu\nu+(1-C_1)\beta b)} - 1 > 0, \\ \equiv \frac{b}{\mu\nu+(1-C_1)\beta b}\mathcal{R}_0 - 1 > 0, \end{aligned}$$

where $\mathcal{R}_0$ is as defined in equation (2.2). Using the parameters in Table 1, it can be shown that $\frac{b}{\mu\nu+(1-C_1)\beta b} = 3.1972$ when $C_1 = 0$. The determinant is greater than zero for all values of $\mathcal{R}_0 > 0.3$, but the disease-free equilibrium point E_0 is stable for those values of $0.3 < \mathcal{R}_0 < 1$, otherwise it is unstable. When $C_1 = 1$, the determinant is positive for all values of $\mathcal{R}_0 > 0.0103$, but the disease-free is stable for values of $0.0103 < \mathcal{R}_0 < 1$.

Table 1: Parameter values used in the model.

Parameter	Description	Value and dimensions	
b	Per capita growth rate	$3.6/(100 \times 365)$ per day	[35]
β	Per capita rate of infection	$0.25/(296 \times 365)$	[23]
μ	Per capita human mortality rate	$1/(50 \times 365)$ per day	[33]
δ	Disease induced death rate	[0,1]	Estimated
γ	Per capita recovery rate	[0,1]	Estimated
τ_i	Per capita development rate for jiggers	1/25 per day	[6, 21, 22]
n	Number of eggs released per dead jigger	100	[6, 21, 22]
ν	Per capita death rate of fleas	0.023 per day	[6, 21, 22]
τ_e	Per capita development rate for jiggers	1/4 per day	[6, 21, 22]
τ_l	Per capita development rate for jiggers	1/7 per day	[6, 21, 22]
τ_p	Per capita development rate for jiggers	1/12 per day	[6, 21, 22]

Next we determine the stability of the endemic equilibrium point. This is done using a suitable Lyapunov function $\mathcal{L}(S, I, R, E, L, P, A)$ as applied in prior studies such as [13, 14, 30]. Let the Lyapunov function be given by

$$\begin{aligned}\mathcal{L}(S, I, R, E, L, P, A) = S_* \left(\frac{S}{S_*} - \ln\frac{S}{S_*}\right) + I_* \left(\frac{I}{I_*} - \ln\frac{I}{I_*}\right) + R_* \left(\frac{R}{R_*} - \ln\frac{R}{R_*}\right) \\ + \frac{E_*}{n(1-C_2)\tau_i}\left(\frac{E}{E_*} - \ln\frac{E}{E_*}\right) + \frac{L_*}{\tau_e}\left(\frac{L}{L_*} - \ln\frac{L}{L_*}\right) \\ + \frac{P_*}{\tau_l}\left(\frac{P}{P_*} - \ln\frac{P}{P_*}\right) + A_* \left(\frac{A}{A_*} - \ln\frac{A}{A_*}\right).\end{aligned} \tag{2.5}$$

The endemic equilibrium state $E_* = (S_*, I_*, R_*, E_*, L_*, P_*, A_*)$ is the only extremum and the global minimum of the function $\mathcal{L}(S, I, R, E, L, P, A) \in \mathbb{R}^7_+$. It follows from equation (2.1) at steady state that

$$\begin{aligned}\mu S_* + (1-C_1)\beta A_* S_* &= b, \\ \frac{(1-C_1)\beta A_* S_*}{I_*} &= \mu + \delta + \gamma + \tau_i, \\ \frac{\mu R_*}{I_*} &= \gamma, \\ \frac{n(1-C_2)\tau_i I_*}{E_*} &= \nu + \tau_e, \\ \frac{\tau_e E_*}{L_*} &= \nu + \tau_l, \\ \frac{\tau_l L_*}{P_*} &= \nu + \tau_p, \\ \frac{(1-C_1)\beta A_* S_*}{P_*} + \frac{\nu A_*}{P_*} &= \tau_p.\end{aligned} \tag{2.6}$$

In the case of system (2.1) the derivative of the Lyapunov function in equation (2.5) satisfies

$$\begin{aligned}\mathcal{L}' = \left(1 - \frac{S_*}{S}\right) S' + \left(1 - \frac{I_*}{I}\right) I' + \left(1 - \frac{R_*}{R}\right) R' \\ + \left(1 - \frac{E_*}{E}\right) \frac{E'}{n(1-C_2)\tau_i} + \left(1 - \frac{L_*}{L}\right) \frac{L}{\tau_e}' + \left(1 - \frac{P_*}{P}\right) \frac{P'}{\tau_l} + \left(1 - \frac{A_*}{A}\right) A'.\end{aligned}$$

Substituting the expressions for the derivatives in $\mathcal{L}'$ from equation (2.1) gives

$$\begin{aligned}
\mathcal{L}' = {} & b-(1-C_1)\beta AS-\mu S-\frac{S_*}{S}\left(b-(1-C_1)\beta AS-\mu S\right)\\
& +(1-C_1)\beta AS-(\mu+\delta+\gamma+\tau_i)I-\frac{I_*}{I}\left((1-C_1)\beta AS-(\mu+\delta+\gamma+\tau_i)I\right)\\
& +\gamma I-\mu R-\frac{R_*}{R}\left(\gamma I-\mu R\right)+\frac{1}{n(1-C_2)\tau_i}\left(n(1-C_2)\tau_i I-(\nu+\tau_e)E\right)\\
& -\frac{1}{n(1-C_2)\tau_i}\frac{E_*}{E}\left(n(1-C_2)\tau_i I-(\nu+\tau_e)E\right)+\frac{1}{\tau_e}\left(\tau_e E-(\nu+\tau_l)L\right)\\
& -\frac{1}{\tau_e}\frac{L_*}{L}\left(\tau_e E-(\nu+\tau_l)L\right)+\frac{1}{\tau_l}\left(\tau_l L-(\nu+\tau_p)P\right)\\
& -\frac{1}{\tau_l}\frac{P_*}{P}\left(\tau_l L-(\nu+\tau_p)P\right)+\tau_p P-\nu A-(1-C_1)\beta AS\\
& -\frac{A_*}{A}\left(\tau_p P-\nu A-(1-C_1)\beta AS\right).
\end{aligned}$$

Using equation (2.6) this can be shown to be equivalent to

$$\begin{aligned}
\mathcal{L}' = {} & \mu S_*+(1-C_1)\beta A_*S_*-(1-C_1)\beta AS-\mu S-\frac{S_*}{S}\left(\mu S_*+(1-C_1)\beta A_*S_*-(1-C_1)\beta AS-\mu S\right)\\
& +(1-C_1)\beta AS-\frac{(1-C_1)\beta A_*S_*}{I_*}I-\frac{I_*}{I}\left((1-C_1)\beta AS-\frac{(1-C_1)\beta A_*S_*}{I_*}I\right)\\
& +\frac{\mu R_*}{I_*}I-\mu R-\frac{R_*}{R}\left(\frac{\mu R_*}{I_*}I-\mu R\right)+I-\frac{I_*}{E_*}E\\
& -\frac{E_*}{E}\left(I-\frac{I_*}{E_*}E\right)+E-\frac{E_*}{L_*}L\\
& -\frac{L_*}{L}\left(E-\frac{E_*}{L_*}L\right)+L-\frac{L_*}{P_*}P\\
& -\frac{P_*}{P}\left(L-\frac{L_*}{P_*}P\right)+\frac{(1-C_1)\beta A_*S_*}{P_*}P+\frac{\nu A_*}{P_*}P-\nu A-(1-C_1)\beta AS\\
& -\frac{A_*}{A}\left(\frac{(1-C_1)\beta A_*S_*}{P_*}P+\frac{\nu A_*}{P_*}P-\nu A-(1-C_1)\beta AS\right),
\end{aligned}$$

such that

$$\begin{aligned}
\mathcal{L}' = {} & \mu S_*\left(2-\frac{S}{S_*}-\frac{S_*}{S}\right)+(1-C_1)\beta A_*S_*\left(2-\frac{S}{S_*}\right)+(1-C_1)\beta AS_*\left(1-\frac{I}{I_*}\frac{A_*}{A}-\frac{S}{S_*}\frac{I_*}{I}\right)\\
& +\frac{\mu R_*}{I_*}I\left(1-\frac{R_*}{R}\right)+\mu R_*\left(1-\frac{R}{R_*}\right)+\left(1-\frac{E_*}{E}\right)+I_*\left(1-\frac{E}{E_*}\right)\\
& +E\left(1-\frac{L_*}{L}\right)+E_*\left(1-\frac{L}{L_*}\right)+L\left(1-\frac{P_*}{P}\right)+L_*\left(1-\frac{P}{P_*}\right)+\frac{(1-C_1)\beta A_*S_*}{P_*}P\left(1-\frac{A_*}{A}\right)\\
& +\frac{\nu A_*}{P_*}P\left(1-\frac{A_*}{A}\right)+\nu A_*\left(1-\frac{A}{A_*}\right)+(1-C_1)\beta A_*S\left(1-\frac{A}{A_*}\right).
\end{aligned}$$

Note that $\mathcal{L}'=0$ when $S=S_*, I=I_*, R=R_*, E=E_*, L=L_*, P=P_*$, and $A=A_*$. With a choice of appropriate parameters, $\mathcal{L}'$ can be shown to be non-positive, which proves stability.

Next, bifurcation analysis is performed at the disease-free equilibrium E_0 using the Centre Manifold Theory as in Carr [1], and described in Theorem 4.1 of Castillo-Chavez [3]. In order to apply the theorem, variables of the model in equation (2.1) are re-defined as $S=x_1, I=x_2, R=x_3, E=x_4, L=x_5, P=x_6$ and

$A = x_7$. Thus, $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$. The model is now in the form $\frac{dX}{dt} = F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$, where

$$\begin{aligned}
x_1' &= f_1 = b - (1 - C_1)\beta x_7 x_1 - \mu x_1,\\
x_2' &= f_2 = (1 - C_1)\beta x_7 x_1 - (\mu + \delta + \gamma + \tau_i)x_2,\\
x_3' &= f_3 = \gamma x_2 - \mu x_3,\\
x_4' &= f_4 = n(1 - C_2)\tau_i x_2 - (\nu + \tau_e)x_4,\\
x_5' &= f_5 = \tau_e x_4 - (\nu + \tau_l)x_5,\\
x_6' &= f_6 = \tau_l x_5 - (\nu + \tau_p)x_6,\\
x_7' &= f_7 = \tau_p x_6 - \nu A - (1 - C_1)\beta x_7 x_1.
\end{aligned} \tag{2.7}$$

The disease-free equilibrium is $[x_1^* = b/\mu, x_2^* = 0, x_3^* = 0, x_4^* = 0, x_5^* = 0, x_6^* = 0, x_7^* = 0]$. Let β be the bifurcation parameter. The linearized matrix of system (2.7) around the disease-free-equilibrium when $\beta = \beta^*$ is

$$D_x f = \begin{bmatrix}
-\mu & 0 & 0 & 0 & 0 & 0 & -\frac{\beta^* b}{\mu}\\
0 & -(\mu + \delta + \gamma + \tau_i) & 0 & 0 & 0 & 0 & \frac{\beta^* b}{\mu}\\
0 & \gamma & -\mu & 0 & 0 & 0 & 0\\
0 & n(1 - C_2)\tau_i & 0 & -(\nu + \tau_e) & 0 & 0 & 0\\
0 & 0 & 0 & \tau_e & -(\nu + \tau_l) & 0 & 0\\
0 & 0 & 0 & 0 & \tau_l & -(\nu + \tau_p) & 0\\
0 & 0 & 0 & 0 & 0 & \tau_p & -(\nu + \frac{\beta^* b}{\mu})
\end{bmatrix}, \tag{2.8}$$

from which the reproduction number is shown to be as shown in equation (2.2). Consider the case when $\mathcal{R}_0 = 1$, giving

$$\beta = \beta^* = \frac{(\nu + \tau_e)(\nu + \tau_l)(\nu + \tau_p)(\mu + \delta + \gamma + \tau_i)}{(1 - C_1)n(1 - C_2)\tau_i \tau_e \tau_l \tau_p}.$$

The linearized system of the transformed system in equation (2.7) with the bifurcation parameter β^* has a simple zero eigenvalue, with all other eigenvalues having negative real part. We can now apply the center manifold approach [1], to analyze the dynamics of system (2.7) near $\beta = \beta^*$. The Jacobian (2.8) has a right eigenvector associated with the zero eigenvalue given by $w = [w_1, w_2, w_3, w_4, w_5, w_6, w_7]^T$, where

$$\begin{aligned}
w_1 &= -\frac{\beta^* b}{\mu^2} w_7, \quad w_2 = \frac{\beta^* b}{\mu(\mu + \delta + \gamma + \tau_i)} w_7, \quad w_3 = \frac{\gamma \beta^* b}{\mu^2(\mu + \delta + \gamma + \tau_i)} w_7\\
w_4 &= \frac{\beta^* b n(1 - C_2)\tau_i}{\mu(\mu + \delta + \gamma + \tau_i)(\nu + \tau_e)} w_7, \quad w_5 = \frac{\beta^* b n(1 - C_2)\tau_i \tau_e}{\mu(\mu + \delta + \gamma + \tau_i)(\nu + \tau_e)(\nu + \tau_l)} w_7,\\
w_6 &= \frac{\beta^* b n(1 - C_2)\tau_i \tau_e \tau_l}{\mu(\mu + \delta + \gamma + \tau_i)(\nu + \tau_e)(\nu + \tau_l)(\nu + \tau_p)} w_7, \quad w_7 = w_7 > 0.
\end{aligned}$$

The left eigenvector of Jacobian (2.8) associated with the zero eigenvalue at $\beta = \beta^*$ is given by $v = [v_1, v_2, v_3, v_4, v_5, v_6, v_7]$, where

$$\begin{aligned}
&v_1 = 0, v_2 = \frac{n(1 - C_2)\tau_i \tau_p \tau_l \tau_e}{(\mu + \delta + \gamma + \tau_i)(\nu + \tau_e)(\nu + \tau_l)(\nu + \tau_p)} v_7, \quad v_3 = 0,\\
&v_4 = \frac{\tau_p \tau_l \tau_e}{(\nu + \tau_e)(\nu + \tau_l)(\nu + \tau_p)} v_7, \quad v_5 = \frac{\tau_p \tau_l}{(\nu + \tau_l)(\nu + \tau_p)} v_7, \quad v_6 = \frac{\tau_p}{\nu + \tau_p} v_7,\\
&v_7 = v_7 > 0.
\end{aligned}$$

The non vanishing partial derivatives of F at the disease-free equilibrium associated with p and q are given by

$$p = \sum_{k,i,j=1}^{7} v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j} = 2v_2 w_1 w_7 \frac{\partial^2 f_2}{\partial x_1 \partial x_7} - 2v_7 w_1 w_7 \frac{\partial^2 f_7}{\partial x_1 \partial x_7} = \frac{-2\beta^* b}{\mu^2} (\mathcal{R}_0 - 1) v_7 w_7^2,$$

$$q = \sum_{k,i=1}^{7} v_k w_i \frac{\partial^2 f_k}{\partial x_i \beta^*} = v_2 w_7 \frac{\partial^2 f_2(0,0)}{\partial \beta^* \partial x_7} + v_7 w_7 \frac{\partial^2 f_3(0,0)}{\partial \beta^* \partial x_7} = v_2 w_7 \frac{b}{\mu} - v_7 w_7 \frac{b}{\mu} = \frac{b}{\mu} \left(\frac{\mathcal{R}_0}{\beta^*} - 1 \right) v_7 w_7 > 0.$$

Therefore, $p < 0$ for all $\mathcal{R}_0 > 1$, but $q > 0$ for $\mathcal{R}_0 > 0.3$. Therefore, when β^* changes from negative to positive, the disease-free equilibrium point E_0 changes its stability from stable to unstable. In turn, the negative endemic unstable equilibrium becomes positive and locally asymptotically stable. When $\mathcal{R}_0 < 0.3$, $q < 0$. This implies that when $\beta^* < 0$, with $|\beta^*| \ll 1$, the disease-free is unstable, confirming our earlier results that the disease-free state is not stable for $\mathcal{R}_0 < 0.3$. When $0 < \beta^* \ll 1$, the disease-free is locally asymptotically stable, and there exists a positive unstable equilibrium.

3. Numerical simulation

In this section, the model is simulated to study the numerical behavior using parameter values in Table 1. First the model is run under the assumption that only one flea burrows into the stratum granulosum of a susceptible human releasing 100 eggs upon completion of the cycle. The results are shown in Figure 3. It is observed from the figure that as n increases from 0 to 100, there are few infested individuals who eventually reduce to zero in about four months. As n is increased to 100, more eggs are released and we observe a progressional increase from the number of eggs to adult fleas. The figure shows that starting with one egg released in a population of 200 humans results in more than 1000 adult fleas. It is important to note that for this simulation, $\mathcal{R}_0 < 1$ and as such, the disease does not prevail in the human.

Next we simulate the model with different levels of control when $\mathcal{R}_0$ is less than unity (see Figure 4). It can be observed that as the controls decrease, $\mathcal{R}_0$ increases. Figure 4 (a) and (b) are examples of how controls C_1 and C_2 applied at equal rates affect changes in $\mathcal{R}_0$. Note that without both controls, $\mathcal{R}_0$ reaches 0.7 but does not exceed unity. Therefore, if the infection rate $\beta < 0.3$ with other parameters kept constant, $\mathcal{R}_0$ can be controlled and jigger infestation stopped.

The model is further simulated for when an individual has more than one jigger embedded into the stratum granulosum that is, $100 \leqslant n \leqslant 1000$, (Figure 5). It is seen from the figure that there are many adult fleas in the population leading to a small number of susceptibles. The number of infested individuals also increased rapidly as compared to Figure 3, but die out when the susceptible pool is depleted. It is important to note that unlike the previous case ($n \in [0, 100]$), for this case, the number of roaming young fleas increases to almost 17 times more. Therefore, super infestation should be prevented and all efforts to extract the jigger once embedded enforced. This will intercept full development of the eggs thereby preventing more egg shedding.

The corresponding plot for $\mathcal{R}_0$ and the controls in Figure 6 show that $\mathcal{R}_0$ increases to 7 when an individual is infected with more than one jigger. If we consider an extreme case of infestation with 10 jiggers (results in 1000 eggs released by the individual), then the value of β that makes $\mathcal{R}_0 = 1$ can be shown to be 0.0392. This translates into a prevalence of 0.0042352 in Mayuge district if the jigger infestation is to be controlled. From this result, we conclude that efforts must be made to reduce the current prevalence from 0.25 to 0.004. In this section, the model was simulated for different values of n, the number of eggs released in the population by one jigger. Results show that jigger infestation does not prevail when few eggs hatch to develop into adult fleas. Controls applied to reduce jigger infestation in the human and inhibit development of eggs greatly reduce $\mathcal{R}_0$. This implies that an infested individual would not spread the jiggers further. The major conclusion from the simulation is that tungiasis can be stopped if development of the flea is intercepted.

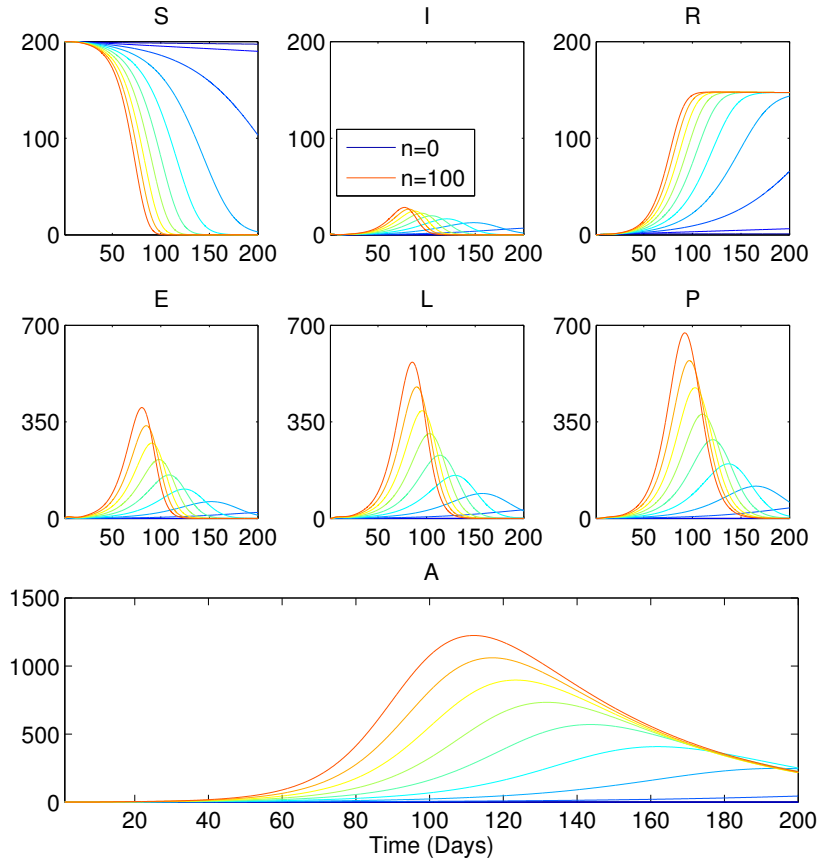

Figure 3: Simulation of the model for $0 \leqslant n \leqslant 100$. Initial conditions used are $S(0) = 200, I(0) = 0, R(0) = 0, E(0) = 1, L(0) = 0, P(0) = 0, A(0) = 0$, $\beta = 0.0003$, $\gamma = 1/7$, and $\delta = 0.01$. Other parameter vales are shown in Table 1.

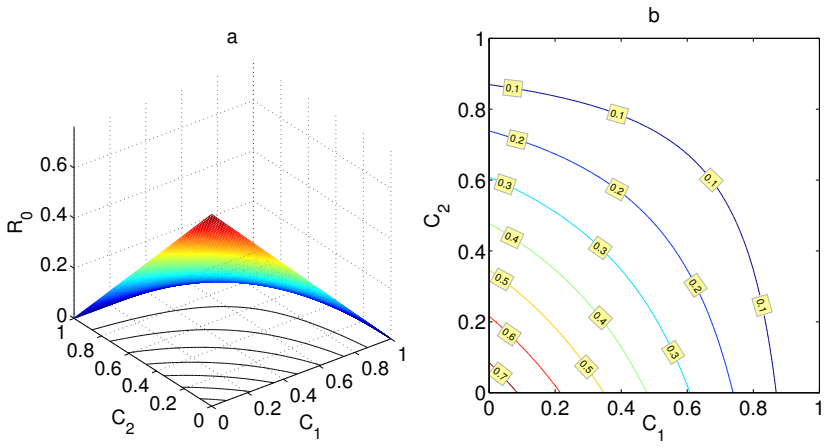

Figure 4: The basic reproduction number simulated with the controls C_1, to stop infection in the human, and C_2 that prevents eggs from developing. In this figure, $\mathcal{R}_0 < 1, \beta = 0.3$, $\gamma = 1/7$, and $\delta = 0.01$. Other parameter values used are given in Table 1. In (a), the basic reproduction number $\mathcal{R}_0$ is plotted with respect to controls C_1 and C_2, and (b) shows the level lines for $\mathcal{R}_0$. The colors of lines in (b) correspond to the shades in (a).

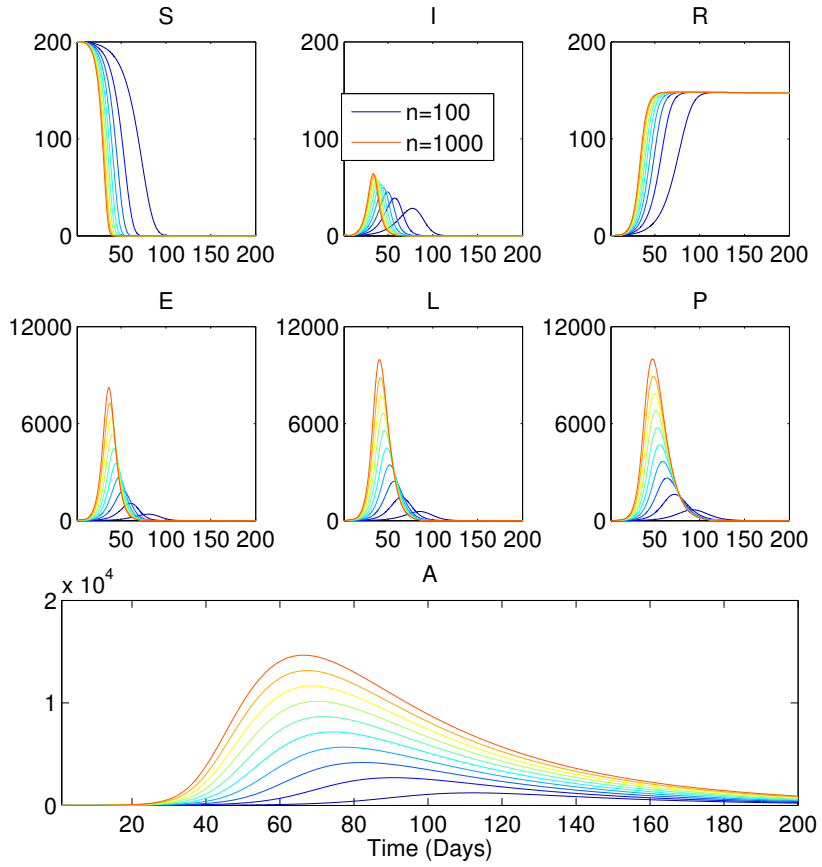

Figure 5: Simulation of the model for $100 \leqslant n \leqslant 10 \times 100$; (i.e. 10 jiggers burrow into the stratum *granulosum* of the susceptible human host). Initial conditions used are $S(0) = 200, I(0) = 0, R(0) = 0, E(0) = 1, L(0) = 0, P(0) = 0, A(0) = 0$, $(1 - C_1)\beta = 0.0003$, $\gamma = 1/7$, and $\delta = 0.01$. Other parameter vales are shown in Table 1.

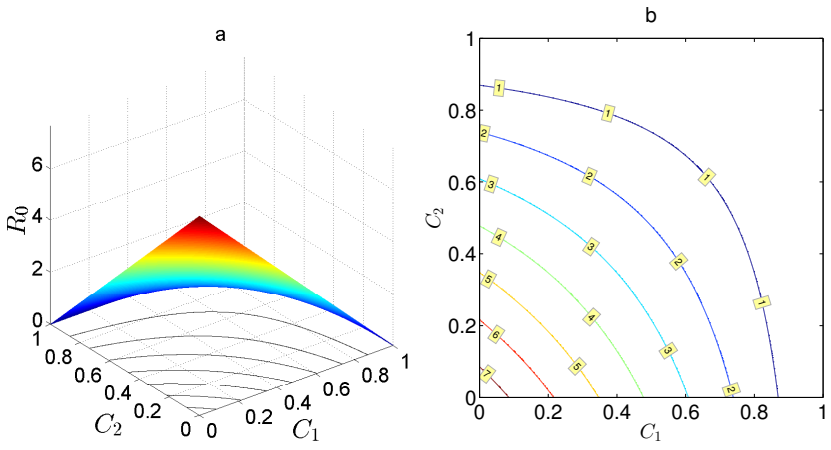

Figure 6: The basic reproduction number simulated with the controls C_1, to stop infection in the human, and C_2 that prevents eggs from developing. In this figure, $\mathcal{R}_0 > 1, (1 - C_1)\beta = 0.3$, $\gamma = 1/7$, and $\delta = 0.01$. Other parameter values used are given in Table 1. In (a), the basic reproduction number $\mathcal{R}_0$ is plotted with respect to controls C_1 and C_2, and (b) shows the level lines for $\mathcal{R}_0$. The colors of lines in (b) correspond to the shades in (a).

4. Discussion and conclusion

Jigger infestation continues to kill individuals in poor communities [6, 10, 21, 28, 32, 34] and causing rotting away of infested people due to ignorance and neglect. This paper developed and analyzed a simple epidemic model that can be used to study other diseases with similar dynamics. Two methods of analysis were used; the first using known theorems and methods (Lyapunov and the center manifold theorem), the model was analyzed for nature of stability. The second, a numerical simulation to study the behavior of solutions in a range of parameter values. Using both methods, the results showed that the disease can be eliminated if $\mathcal{R}_0 < 0.3$, and persists otherwise. Numerical results showed that when a single jigger embeds in an individual, it results in a few infested individuals who eventually reduce to zero in about four months (see Figure 3). As more eggs are shedded into the environment, there is a progressional in the number of eggs to adult fleas that eventually develop. Without both controls, $\mathcal{R}_0$ was observed to reach 0.7 but not exceed unity. Therefore, if the infection rate $\beta < 0.3$ with other parameters kept constant, secondary infestations could be controlled and jigger infestation stopped.

The model was further simulated for the case when an individual had more than one jigger embedded into the stratum granulosum that is, $100 \leqslant n \leqslant 1000$, (Figure 5). Results showed that more jigger infestation gave rise to more fleas in the environment, resulting into a small number of susceptibles in the population. The number of infested individuals increased tremendously, and the disease died out when the susceptible pool was depleted. It was also observed in this case that there were many young jiggers to almost 17 times more than the case of a single embedment. From this result we conclude that superinfection should be prevented and all efforts to extract the jigger once embedded enforced. This will intercept full development of the eggs thereby preventing more egg shedding.

When a plot of $\mathcal{R}_0$ with the controls was done, it was observed that when more than one fleas embed in an individual, there is a possibility of 7 new infestations within the population. We considered an extreme case of infestation with 10 jiggers (Mayuge district [23, 31]) to ascertain the value of the force of infection β that would prevent a single infested person to transmit to 7 new ones. Results showed that to keep $\mathcal{R}_0 \leqslant 1$, β must equal 0.0392. Therefore, with super infestation, the prevalence of tungiasis in Mayuge district should be kept to below 0.0042352 if the jiggers are to be controlled and eventually eliminated. From this result, we conclude that efforts must be made to reduce the current prevalence from 0.25 to 0.004.

Jigger infestation has an important social dimension and affects human rights. It is a zoonosis and considering its links with poverty, requires multi-disciplinary research in which public health, social sciences, health education, and animal husbandry need to interact. The disease has a potential to trigger political anti-poverty strategies by simultaneously addressing public health infrastructures in both humans and animals at the community level. All countries where jigger infestation is reported are low-income and lower middle-income economies where it afflicts the same marginalized population segments and communities affected by many neglected tropical diseases. The recent evidence of effective intervention measures against jigger infestation in Busoga region by the Uganda government [23, 31] should not be ignored by national health organizations. The Ministry of Health, through its district health offices should work to raise awareness among centers for disease control and to formulate appropriate strategies to address this debilitating and mutilating parasitic skin disease that has unnecessarily plagued disadvantaged communities for in Mayuge district.

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