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MATHEMATICAL MODELLING OF JIGGER INFECTION INCORPORATING TREATMENT AS A CONTROL STRATEGY

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Abstract: In this paper, a mathematical model based on a system of ordinary differential equations is formulated to study the dynamics of jigger infection incorporating treatment as a control strategy against infection. The basic reproduction number is computed using the next generation matrix approach. The existence of the steady states of the model are determined and the stability analysis of the model carried out. The disease free and the endemic equilibrium points are found to be locally asymptotically stable. Numerical simulation of the model carried out showed that a high probability of success of treatment leads to a low jigger prevalence in a population.

AMS Subject Classification: 92B05
Key Words: reproduction number, treatment, jigger infection

1. Introduction

A Jigger is a flea that mostly lives in dry sand and soil found in Sub Saharan climates. Jiggers are parasitic burrowers and are also known as Chigoe fleas or Tunga penetrans. A jigger is roughly 0.9mm in size. It is the smallest in the flea family. It thrives by burrowing itself into the flesh of warm blooded animals head first and hind legs out, feeding on blood within the cutaneous and subcutaneous layers of skin, laying eggs outside in the soil to continue its cycle.

Newly emerged adults are agile, jumpy and crawl on the ground until they locate a suitable host, usually man. Pigs, dogs, cats, cattle, sheep among others are important reservoirs. The dogs and cats act as important reservoirs for the

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transmission of sand fleas [4]. Both sexes feed on blood but whereas the male soon leaves the host after taking a blood meal, the fertilized female burrows by aid of its sharp and well developed mouthparts into the soft areas of the skin, such as the toe webs or under toenails. The sole, elbows and knees of heavily infested people may also be infected [8] and [5].

Effects of jiggers include; severe itching, pain, skin inflammation and swelling, desquamation of the skin, lesions and ulcerations, with black dots in the middle. If left untreated, secondary infections, such as tetanus, Lumphangitis, sepsis, gangrene and bacteremia can occur. It can also lead to Tungiasis(an ectoparasitic skin disease, caused by the penetration of the female sand flea into epidermis of the host)[6]. It is endemic in developing countries in the tropics, particularly where poverty and poor standards of basic hygiene exist, like in the resource poor communities of South America, the Caribbean and sub-Saharan Africa, where it is a serious but neglected health problem [2].

The first evidence of infection by this sand flea is a tiny black dot (lesion) on the skin at the point of penetration. The area around the embedded flea becomes very itchy and inflamed leading to ulcerations, lymphangitis and accumulation of pus (sepsis)[5].

The possible treatment may be mechanical removal of the flea with a sterile pin, followed by an antiseptic dressing. Kerosene application kills the flea but results into ulceration of the skin until the dead flea is expelled [1]. Treatment can also be via Surgical extraction of embedded sand fleas under sterile conditions in medical facilities[3]. Also Benzyl Benzoate Emulsion (BBE) and potassium permanganate may be effective in killing of the embedded fleas if applied in right concentration. The fleas may also be deterred by a repellant applied to the skin, although walking barefoot in dirt quickly removes it. If it is possible to locate the area of soil where jiggers originate, it could be burnt off or sprayed with a suitable insecticide in an effort to kill the fleas[5]. Wearing of shoes and observing cleanliness is key in controlling the infection. According to Ahadi Trust Foundation in Kenya, jiggers have continued to create havoc in rural areas and many school going children have dropped out of school because they are unable to walk [5].

2. Description and Formulation of the Model

The human population is subdivided into the class of susceptibles S(t), those who are infected I(t) and those removed, R(t) upon treatment. The total human population is given by

$$N(t) = S(t) + I(t) + R(t). \tag{1}$$

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Humans are recruited into the susceptible class at a rate Λ. The rate of infection λ is defined as

$$\lambda = \frac{\beta c I}{N}, \tag{2}$$

where β is the probability of being infected with the bug and c is the contact rate with infected environs. Upon infection, individuals in S(t) move into the class I(t). Upon treatment/physical removal of the bug, infected individuals in I(t) move to the class R(t) at a rate ρ. μ is the natural death rate, and δ is death due to jigger infestation. The resulting differential equations are:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - (\lambda + \mu)S, \\ \frac{dI}{dt} &= \lambda S - (\mu + \delta + \rho)I, \\ \frac{dR}{dt} &= \rho I - \mu R. \end{aligned} \tag{3}$$

3. Analysis of the Model

Since the model deals with human population, all the state variables are positive at all time t. We show that our solutions are bounded in the set Γ where

$$S(t), I(t), R(t) \in \Gamma \ \subset \mathbb{R}^3_+.$$

Since

$$\frac{dN}{dt} = \frac{dI}{dt} + \frac{dR}{dt} + \frac{dN}{dt}, \tag{4}$$

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I, \tag{5}$$

it follows that

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{6}$$

Then

$$dN \leq (\Lambda - \mu N)dt, \tag{7}$$

upon integration,

$$\Theta(N, t) = e^{\mu t} N - e^{\mu t} \frac{\Lambda}{\mu} \leq c \tag{8}$$

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Therefore

$$e^{\mu t}N - e^{\mu t}\frac{\Lambda}{\mu} \leq c \tag{9}$$

At t=0

$$N - \frac{\Lambda}{\mu} = c \tag{10}$$

substituting c we get

$$e^{\mu t}N - e^{\mu t}\frac{\Lambda}{\mu} \leq N - \frac{\Lambda}{\mu} \tag{11}$$

This implies

$$e^{\mu t}N \leq e^{\mu t}\frac{\Lambda}{\mu} + N - \frac{\Lambda}{\mu} \tag{12}$$

dividing by

$$e^{\mu t}$$

on both sides we have

$$N \leq \frac{\Lambda}{\mu} + \left(N - \frac{\Lambda}{\mu}\right)e^{-\mu t} \tag{13}$$

as

$$t \longrightarrow \infty$$

$$N \leq \frac{\Lambda}{\mu} \tag{14}$$

3.1. The Basic Reproduction Number

The basic reproduction number is the average number of secondary infections due to a single infectious individual in a fully susceptible population. It is the spectral radius of a matrix

$$FV^{-1} \tag{15}$$

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 of individuals into and out of compartment j. FV^{-1} is calculated by the method of next generation matrix [9] and is found to be

$$R_0 = \frac{\beta c(1-\alpha)}{\mu + \rho + \delta} \tag{16}$$

The basic reproduction number has important implications on the epidemiological trend. If $R_o < 1$ and in the absence of bifurcation, the epidemic will die out and if $R_o > 1$ the epidemic will develop in the population.

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3.2. Existence of Equilibrium Points

In this section, we analyse the model to investigate stability of its equilibria both at Disease-free equilibrium(DFE) and at endemic equilibrium(EE). The disease free equilibrium points of the model are its steady state solutions in the absence of infection or disease. Consider equation (3).

To obtain the equilibrium points for the model we set the right hand side to zero.

$$\begin{aligned} \Lambda - (\lambda + \mu)S &= 0 \\ \lambda S - (\mu + \delta + \rho)I &= 0 \\ \rho I - \mu R &= 0 \end{aligned} \tag{17}$$

Let α be the probability of success of treatment for jigger infection, thus the effective rate of infection λ becomes

$$\lambda = \frac{\beta c(1-\alpha)I}{N}$$

To calculate the DFE,we set (I, R) to be equal to zero. Thus

$$S = \frac{\Lambda}{\mu}$$

The disease-free equilibrium point E^0 is given by

$$E^0 = \left(\frac{\Lambda}{\mu}, 0, 0\right)$$

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

$$\Lambda - (\lambda + \mu)S = 0$$

$$\Rightarrow S^* = \frac{N\Lambda}{\beta c(1-\alpha)I + \mu N}$$

$$\Rightarrow S^* = \frac{N(\mu + \rho + \delta)}{\beta c(1-\alpha)}$$

$$\lambda S - (\mu + \delta + \rho)I = 0$$

$$\Rightarrow \left(\frac{\beta c(1-\alpha)I}{N}\right)\left(\frac{N\Lambda}{\beta c(1-\alpha)I + \mu N}\right) - (\mu + \delta + \rho)I = 0$$

$$\Rightarrow I^* = \frac{\beta c(1-\alpha)\Lambda - \mu N(\mu + \delta + \rho)}{\beta c(1-\alpha)(\mu + \delta + \rho)}$$

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$$\rho I - \mu R = 0$$

$$\Rightarrow \rho\left(\frac{\beta c(1-\alpha)\Lambda - \mu N(\mu+\delta+\rho)}{\beta c(1-\alpha)(\mu+\delta+\rho)}\right) - \mu R = 0$$

$$\Rightarrow R^* = \frac{\rho\beta c(1-\alpha)\Lambda - \mu N(\mu+\delta+\rho)}{\mu\beta c(1-\alpha)(\mu+\delta+\rho)}$$

Therefore the endemic equilibrium $E^* = (S^*, I^*, R^*)$ is given by

$$E^* = \left(\frac{N(\mu+\rho+\delta)}{\beta c(1-\alpha)}, \frac{\beta c(1-\alpha)\Lambda - \mu N(\mu+\delta+\rho)}{\beta c(1-\alpha)(\mu+\delta+\rho)}, \frac{\rho\beta c(1-\alpha)\Lambda - \mu N(\mu+\delta+\rho)}{\mu\beta c(1-\alpha)(\mu+\delta+\rho)}\right)$$

3.3. Local Stability of the Disease-Free Equilibrium

The disease free equilibrium points of the model are its steady state solutions in the absence of infection or disease.

The Jacobian matrix of Equation (3) is given by

$$J = \begin{bmatrix} -(\frac{\beta c(1-\alpha)I}{N} + \mu) & -(\frac{\beta cS(1-\alpha)}{N}) & 0 \\ \frac{\beta c(1-\alpha)I}{N} & \frac{\beta cS(1-\alpha)}{N} - (\mu+\delta+\rho) & 0 \\ 0 & \rho & -\mu \end{bmatrix}.$$

Since at DFE $S = N$:

$$\Rightarrow J = \begin{bmatrix} -(\frac{\beta c(1-\alpha)I}{N} + \mu) & -\beta c(1-\alpha) & 0 \\ \frac{\beta c(1-\alpha)I}{N} & \beta c(1-\alpha) - (\mu+\delta+\rho) & 0 \\ 0 & \rho & -\mu \end{bmatrix},$$

at DFE the Jacobian matrix becomes

$$J_{E^0} = \begin{bmatrix} -\mu & -\beta c(1-\alpha) & 0 \\ 0 & \beta c(1-\alpha) - (\mu+\delta+\rho) & 0 \\ 0 & \rho & -\mu \end{bmatrix}.$$

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To compute the eigenvalues, we have

$$|J_{E^0} - I\lambda| = 0$$

such that

$$\begin{vmatrix} -\mu-\lambda & -\beta c(1-\alpha) & 0 \\ 0 & \beta c(1-\alpha)-(\mu+\delta+\rho)-\lambda & 0 \\ 0 & \rho & -\mu-\lambda \end{vmatrix}.$$

Clearly $-(\mu+\lambda)$ is an eigenvalue and

$$\lambda_3 = \beta c(1-\alpha) - (\mu+\delta+\rho) < 0$$

it implies

$$\lambda_3 = \beta c(1-\alpha) < (\mu+\delta+\rho)$$
$$\lambda_3 = \frac{\beta c(1-\alpha)}{\mu+\delta+\rho} < 1$$
$$\lambda_3 = R_0 < 1$$

This shows that the eigenvalues have negative real parts, and thus the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$.

3.4. Local Stability of the Endemic Equilibrium of the Model

A disease is endemic in a population if it persists in the population. The stability of the endemic equilibrium is investigated using the trace and the determinant. The Jacobian matrix at E^* is given by

$$J = \begin{bmatrix} -(\frac{\beta c(1-\alpha)I}{N}+\mu) & -(\frac{\beta cS(1-\alpha)}{N}) & 0 \\ \frac{\beta c(1-\alpha)I}{N} & \frac{\beta cS(1-\alpha)}{N}-(\mu+\delta+\rho) & 0 \\ 0 & \rho & -\mu \end{bmatrix}$$

$$J_{E^*} = \begin{bmatrix} -\left(\frac{\beta c(1-\alpha)(\beta c(1-\alpha)\Lambda-\mu N(\mu+\delta+\rho))}{N\beta c(1-\alpha)(\mu+\delta+\rho)}+\mu\right) & -(\mu+\delta+\rho) & 0 \\ \frac{\beta c(1-\alpha)(\beta c(1-\alpha)\Lambda-\mu N(\mu+\delta+\rho))}{N\beta c(1-\alpha)(\mu+\delta+\rho)} & 0 & 0 \\ 0 & \rho & -\mu \end{bmatrix}$$

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The Trace(τ) at E^* is given by

$$\tau(J_{E^*} = -\left(\frac{\beta c(1-\alpha)(\beta c(1-\alpha)\Lambda - \mu N(\mu+\delta+\rho))}{N\beta c(1-\alpha)(\mu+\delta+\rho)} + \mu\right) - \mu$$

$$\tau(J_{E^*} = -2\mu - \left(\frac{R_0(\mu+\delta+\rho)(R_0(\mu+\delta+\rho)\Lambda - \mu N(\mu+\delta+\rho))}{NR_0(\mu+\delta+\rho)(\mu+\delta+\rho)}\right)$$

$$\tau(J_{E^*} = -2\mu - \left(\frac{R_0^2(\mu+\delta+\rho)\Lambda - \mu N)}{NR_0(\mu+\delta+\rho)}\right)$$

which is negative provided that $R_0 > 1$ and

$$Det(J_{E^*}) = (\mu+\delta+\rho)\left(\frac{\beta c(1-\alpha)(\beta c(1-\alpha)\Lambda - \mu N(\mu+\delta+\rho)}{N\beta c(1-\alpha)(\mu+\delta+\rho)}\right)(-\mu)$$

$$Det(J_{E^*}) = \mu\left(\frac{\mu N(\mu+\delta+\rho) - \beta c(1-\alpha)(\beta c(1-\alpha)\Lambda}{N\beta c(1-\alpha)}\right)$$

$$Det(J_{E^*}) = \mu\left(\frac{\mu N(\mu+\delta+\rho) - R_0^2(\mu+\delta+\rho)^2\Lambda}{NR_0(\mu+\delta+\rho)}\right)$$

$$Det(J_{E^*}) = \mu\left(\frac{\mu N - R_0^2(\mu+\delta+\rho)\Lambda}{NR_0}\right)$$

Clearly the determinant of the matrix is positive provided that $R_0^2(\mu+\delta+\rho)\Lambda < \mu N$. Therefore the model has an asymptotically stable endemic equilibrium provided that $R_0 > 1$.

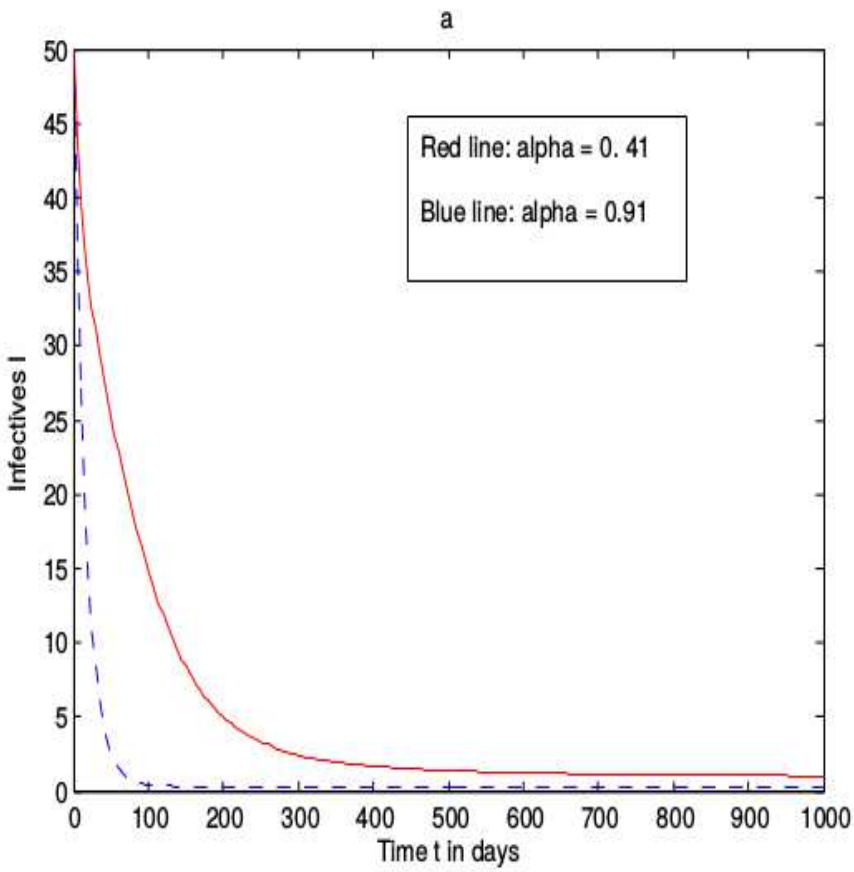
3.5. Numerical Simulations

Numerical simulations are carried out to graphically illustrate the long term effect of treatment on the dynamics of jigger infection.

Table 1: Parameter values of the model

Parameter description	Symbol	Value	Source
Recruitment rate	Λ	0.0044	[7]
Natural mortality rate	μ	0.016	[7]
Disease induced mortality rate	δ	0.005	Estimated
Rate of treatment	ρ	0.09	Estimated
Contact rate of infection	c	0.08	[7]
Probability of infection with jiggers	β	0.25, 0.85	Estimated
Probability of success of treatment	α	0.91, 0.41	Estimated

3.5.1 Figure 1:The graph of jigger infectives against time



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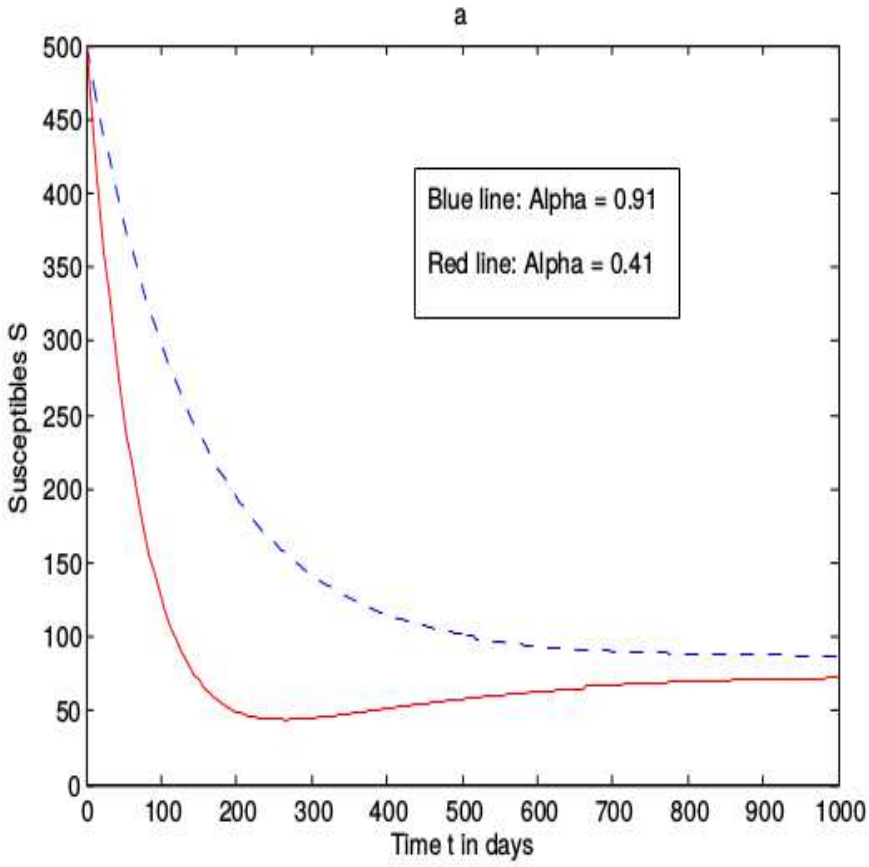
3.5.2 Figure 2: The graph of Susceptibles against time



4. Discussion

In this work, we formulated a model for the dynamics of jigger infection incorporating treatment. The existence of the disease free and endemic equilibrium was established and the stability of the same was analysed and were found to be locally asymptotically stable. From the numerical simulations, we observe that effective jigger treatment has the effect of reducing the disease prevalence. From figure 1, with an increase in the probability of the success of jigger treatment the number of infectives in the population decreases as time tends to infinity. Figure 2 shows that with a low probability of success of treatment,the number of susceptibles depletes sharply with time due to increased infections and vice versa.

In conclusion, effective treatment of jigger infection prevents rapid progression of

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this infection. Moreover improving life standards eg. observing sanitation, wearing of shoes and watering dusty floors are equally effective in jigger control. This reduces the chances of being re-infected.

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