# Modeling Jiggers' Infestation With Incomplete Recoveries Incorporating The Flea Population In Muranga County, Kenya

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#### Abstract

Sand flea insect thrives in hot and humid regions full of dust particles. It attacks humans leading to jiggers infection (tungiasis). Spread of jiggers has been recorded in Caribbean, South American and African countries. In Kenya, Murangá, Homabay and Siaya Counties are among the top regions affected by tungiasis infections. Poverty, lack of sufficient awareness, improper sanitation, and poor control methods are the major reasons for the unending spread of jiggers in Kenya. Prevention and treatment measures have been put in place by the government and NGO's to combat the unending new infections, yet the recoveries are still incomplete. A number of mathematical frameworks have been put in place to unravel the cyclic behavior of this infectious disease. However, intensive study of the dynamical behavior of the disease in both human and flea population has not been conducted. In this research, we designed a model of jiggeers infestation which incorporates the human and sand flea population in Muranga County, Kenya. We derived an ODE system from SEIR-FLA mathematical model to investigate the dynamics of jiggers infestation which incorporates both the human and flea population in Muranga County, Kenya. We used the next generation matrix approach by employing Mathematica software tools to determine the effective basic reproduction number. We incorporated the MATLAB software to generate numerical simulations and the solutions of equations. Results confirmed local stability of JFE when  $R_0 = 4.9827e - 13$  as  $t \to \infty$  for all the Susceptible, Exposed, Infectious, Recovered human compartments and the Egg, Larval, Adult sand flea compartments. All state variables are positive at all times t, and numerical analysis of the invariant region reveals that the model is well-posed. These findings confirm that treatment aid in reducing incomplete recoveries of jiggers infestation.

Keywords: Tungiasis, incomplete recoveries, invariant region, well-posed solution, Muranga county

Date of Submission: 04-05-2023

Date of Acceptance: 14-05-2023

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#### I. INTRODUCTION

Female sand flea insect, scientifically referred to as *Tunga penetrans*, permeates through human skin leading to tungiasis infection. Popular names for jiggers are *chigger*, *chigoe*, *tungiasis*, *funza*, *ndutu*, *dudu*, *pico*, *suthi*, *chica*, *cique*, *nigua*, *bicho de pe'*, *tu* – a great indication that this is an existing infestation [1]. Sand flea is a small pin-head-sized insect mainly camouflages in the wall gaps and cracks on the floors and furniture [2]. The insect survives under seasonal conditions and is more pronounced during precipitation periods in the sandy and dusty zones.

Severe inflammation coupled with pain, itching, and edema is some of the basic symptoms of tungiasis infection. Sand flea insect attacks the knees, feet, hands, and other body parts. The fecundated female sand flea burrows its head onto the host's skin, beneath the toenails or fingernails, and stays for two weeks feeding on the host's blood. It increases in size forming a sack full of eggs which it releases into the ground, and hatch within three to four days, the dies [3]. The insect completes its life cycle in the sand in three to four weeks and burrows onto the skin when it comes into contact with the host's feet and other body parts. The pregnant flea entrenches itself underneath the nails of the toe and fingernails where it burrows resulting in sores that fill with a round sack full of eggs which later form pus, leading to serious inability to walk and perform daily activities effectively [4].

Jigger infestation is associated with poverty, poor living conditions, and lack of proper sanitation in dwelling places as they provide breeding grounds for this insect. In rural schools, this pandemic has contributed to a higher proportion of school dropouts and subpar academic performance, inability to walk easily as a result of discomfort, failure to carry out daily routines, reduced self-esteem and trauma-a subsequent of stigmatization, exposure to other infectious diseases such as HIV/Aids and tetanus resulting from sharing of unsterilized needles. Related complications may lead to disabilities and even death if not properly managed.

[2]has identified some of the control and management measures such as fumigation of the wall crevices kills flea eggs, ensuring adequate sanitation, capacity building, eradication of poverty, putting on shoes all the time, as well as treatments, are effective efforts aimed at curbing the spread of this disease.

In Kenya, jiggers infestation has been ongoing in most of the communities. If jiggers outbreak is not adequately controlled, it has the potential to devastate communities' progress by causing poor living conditions and even loss of individuals who are involved in the development sectors. In as much as researchers have developed dynamical systems of this sand flea, they failed to investigate the unsuccessful recoveries in these endemic regions. As a result, there is a need to address the tungiasis outbreak. This study, therefore, focuses on critical analysis of jiggers infestations with incomplete recoveries; with the incorporation of human and flea population. The study primarily focuses on creating a dynamical model of tungiasis infestation with incomplete recoveries that includes both the flea and human populations, in Muranga County, Kenya.

Most authors have investigated the social perspective and public awareness of this disease whereas little research has been conducted on its scientific modeling. Extensive study on the dynamics of jiggers infestation which couples human and flea population has not been done.[5] formulated systems of Ordinary Differential Equations (ODEs) in his dynamical study of jigger infection whereas [6]articulated a deterministic mathematical model relating human-animal-sand flea interactions to establish an effective rate between the soil environment and susceptible human towards tungiasis. In the former, his model depicted an association between the animals, the flea, larvae, and the human. A research of dynamical systems on the impacts of public health awareness on tungiasis, which is a neglected illness that presents numerous obstacles in endemic populations, was conducted by [7].

The researchers discovered a model for tungiasis coupled with public health education aimed at determination of equilibrium analysis of the stable disease-free and the prevalence states. The authors incorporated theory of Lyapunov stability as well as the invariant principle of LaSalle for classification of global asymptotic stability of the disease-endemic critical points as unstable when  $R_E \leq 1$  and stable if  $R_E > 1$ 1. [4]modelled jiggers infestation and intervention in the human in Murang'a region. The research incorporated the study of [6] with an inclusion of the role of media campaigns towards creating awareness to the general public about the disease. This was discovered to be an effective means of minimizing the spread of tungiasis and other related complications such as HIV/Aids and Hepatitis B. National awareness campaigns through media, community health workers, community health extension workers have been established in Muranga, Bungoma, Migori, Homabay, and Siaya counties with the intent to boost appropriate sanitation among occupations thereby reducing infestation. Results displayed a positive trend in the decline of the further dissemination of this disease. [8]incorporated sanitation as a control tool in the dynamical study of tungiasis in Muranga County. Analysis of their model indicated stability points of both endemic and disease-free equilibrium points to be locally stable asymptotically for Basic Reproductive Number( $R_0$ ) above 1 and  $R_0$ below 1, respectively.[4]concluded that the jiggers infestation model displays backward bifurcation in which Jiggers Free Equilibrium (JFE) exists even if the  $R_0 < 1$ . This is insufficient information to reduce  $R_0$  below 1 for containment of this disease. Consequently, direct Lyapunov methods have been used to demonstrate the global stability of jiggers' free equilibrium infestation without backward bifurcation.

For a better understanding of the dynamical behavior of tungiasis infection, [9] employed ODEs and paired them with protection as a means of preventing the infection. Results confirmed the asymptotic stability of both the endemic and disease-free critical points. A mathematical model of thermography was developed by [10] and applied to the expansion of tungiasis on the skin. To analyze the related skin inflammation brought on by tungiasis infections, they employed a one-dimensional bioheat transfer equation known as the Pennes equation. The body may suffer harm from this Negleted Tropical Diseases (NTD), according to numerical findings, which also suggest that additional skin irritation may be connected. A mathematical model developed by [11]examined the impact of intervention methods on tungiasis transmission dynamics. Both disease-free as well as endemic stationary points were demonstrated to possess consistent asymptotic characteristics in these calculations. By applying Meltzer's matrix stability theorem & Lyapunov's approach, they discovered that the Disease Free Equilibrium (DFE) is globally stable asymptotically. When employed in combination rather than individually, control techniques have a greater influence on tungiasis disease transmission, according to the results of a numerical simulation. [12] developed a deterministic framework to assess the role of social networking sites in tungiasis disease control. Their model found solutions that are positively invariant and bounded, indicating that equilibrium conditions are asymptotically stable at the local level if the fundamental reproduction number is less than 1.0.

Having an aim of studying the most ideal trajectory of tungiasis in accordance with Pontryagin's maximum principle, [13]discovered the SIR-ELPA model. Improved human protection and treatment, as well as increased effectiveness against adult fleas, were found to have a significant impact on the ability to prevent and control the spread of the disease. [14]argued with regard to optimal-time solutions for controlling tungiasis complications with constrained resources. They applied the Pontryagin's minimal principle to the control

parametrization scheme and the interior point method to discover that only bang-bang controls are allowed for all the control policies, and then they developed a numerical strategy to implement this result. The outcomes demonstrated a trade-off within the most efficient use of resources and the quickest possible elimination. When the number of infected humans approaches a threshold  $\epsilon$ , where  $0 < \epsilon < 1$  is a predetermined positive constant, we say that T is the eradication time of the controlled human-jigger system. In as much as researchers have developed dynamical systems of this sand flea, they failed to investigate the unsuccessful recoveries in these endemic regions coupled with both human and sand flea population. This study, therefore, aims at critical analysis of jiggers infestations with incomplete recoveries between the humans and fleas.

# II. THE MODEL

In this model, we analyze both human and sand flea populations in relation to one other. The human population constitutes four compartments; the Susceptible(S), the Exposed(E), the Infested(I), and the Recovered(R) populations. The susceptible individuals are at risk of becoming infected by this parasite whereas the infested class are those that have acquired the disease. The exposed population is humans who are just beginning to be infected. The recovered group, on the other hand, are individuals who have been treated for jiggers infestation. At any time, t > 0, the total number of human is denoted by, N(t) = S(t) + E(t) + I(t) + I(t)R(t). Population of human is being drawn into the susceptible segment at the rate  $\Lambda$ . The Adult sand flea attacks and interact with the susceptible individuals at the rate  $\beta$ , leading to a force of infestation,  $\beta SA$ . Recovery at a rate,  $\gamma$  is possible through treatment. Recovered individuals are still susceptible due to lack of permanent immunity, for re-infection at a rate  $\omega$ . Individuals who have been exposed, infected, or recovered can die inevitably at the rate of  $\mu$ . The exposed group gets infected at the rate,  $\alpha$  whereas the already-infected individuals still become exposed at the rate,  $\delta$ . The flea population has three compartments; Egg stage (F), the Pupal-Larval stage (L), and the Adult stage (A). Adult sand fleas in the infected humans lay eggs onto the ground or crevices, at the rate,  $\tau$ . Some of the hatched eggs die at the rate, v while the female fleas hatch and develop into pupal and larval stage (L) at a rate,  $\sigma$ ; which later grows into adult flea at  $\frac{\epsilon \rho L}{(1+L)}$ . Some of these sand fleas( larval and adult stages) die naturally at the rate, v.

#### **Model Assumptions**

- 1. The ratio of births to deaths in any given population of human is subject to cyclical shifts.
  - 2. Infested population sheds Egg-laying fleas to the environment.
  - 3. There is permanent immunity on recovery.

# Model flow chart and Equations

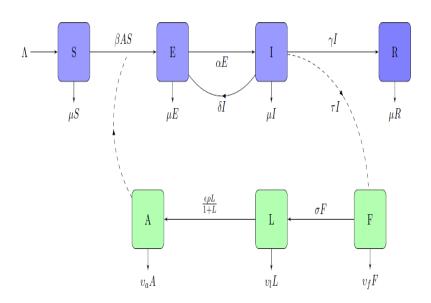


Figure 1: The Model flow chart

We have a system of ODEs arising from the chart

$$\frac{dS}{dt} = \Lambda - (\beta A + \mu)S$$

$$\frac{dE}{dt} = \beta AS - (\alpha + \mu)E + \delta I$$

$$\frac{dI}{dt} = \alpha E - (\gamma + \mu + \delta)I$$

$$\frac{dF}{dt} = \tau I - (\sigma + v_f)F$$

$$\frac{dL}{dt} = \sigma F - \frac{\epsilon\rho L}{(1+L)} - v_l L$$

$$\frac{dA}{dt} = \frac{\epsilon\rho L}{(1+L)} - v_a A$$

$$\frac{dR}{dt} = \gamma I - \mu R \quad (1)$$

where S(0) > 0,  $E(0) \ge 0$ ,  $I(0) \ge 0$ ,  $F(0) \ge 0$ ,  $L(0) \ge 0$ ,  $A(0) \ge 0$  and  $R(0) \ge 0$  are positive initial conditions.

#### Model Analysis Positivity of Solutions

For a well posed model, all the state variables must be > 0 for all  $t \ge 0$ . In this section we need to show that all the state variables are positive t > 0. In order to show this, we first express all the equations of system (1) as;

$$\frac{dS}{dt} \ge -\mu S$$

$$\frac{dE}{dt} \ge -(\alpha + \mu)E + \delta I$$

$$\frac{dI}{dt} \ge -(\gamma + \mu + \delta)I$$

$$\frac{dF}{dt} \ge -(\sigma + v_f)F$$

$$\frac{dL}{dt} \ge -(\epsilon\rho + v_l)L$$

$$\frac{dA}{dt} \ge -v_aA$$

$$\frac{dR}{dt} \ge -\mu R$$
(2)

By use of separation of variable method, we solve the first equation of system (2)as follows

$$\frac{dS}{S} \ge -\mu dt$$
  

$$\Rightarrow \int \frac{dS}{S} \ge -\int \mu dt$$
  

$$\Rightarrow \ln S \ge -(\mu t + c)$$
  

$$\Rightarrow S(t) \ge Ce^{-\mu t} \text{ where } C = e^{t}$$

At t = 0, C = S(0)Thus  $S(t) \ge S(0)e^{-\mu t} > 0$  for t > 0

Upon solving the other equations of system (2) as the first equation, we get  $E(t) \ge E(0)e^{-(\alpha+\mu)t} \ge 0$ ,

$$I(t) \ge I(0)e^{-(\gamma+\mu+\delta)t} \ge 0,$$
  

$$F(t) \ge F(0)e^{-(\sigma+\nu_f)t} \ge 0,$$
  

$$L(t) \ge L(0)e^{-(\epsilon\rho+\nu_l)t} \ge 0,$$

 $A(t) \ge A(0)e^{-v_a t} \ge 0$  and  $R(t) \ge R(0)e^{-\mu t} \ge 0$  for t > 0Clearly, all the state variables are positive t > 0

# **Invariant Region**

Here, we obtain the bounded region of solution of (1). We add the first three equations and the last equation of system (1) and get ordinary Differential Equation (ODE) for the total human population size as

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \qquad (3)$$

By using system (1), we substitute the derivatives in equation (3) and simplify the resulting equation to get  $\frac{dN}{dt} \le \Lambda - \mu N \qquad (4)$ 

Equation (4) can be written as;

$$(\mu N - \Lambda)dt + dN \le 0.$$
 (5)  
Let  $P = \mu N - \Lambda, Q = 1.$ 

(6)

Thus  $\frac{\partial P}{\partial N} = \mu$  and  $\frac{\partial Q}{\partial t} = 0$  Equation (4) is not an exact ODE. Using integrating factor given by  $e^{\int \left[\frac{1}{Q}\left(\frac{\partial P}{\partial N} - \frac{\partial Q}{\partial t}\right)\right]dt} = e^{\mu t}.$ 

Applying integrating factor on (4) we obtain

 $-10.0cm(e^{\mu t}\mu N_H - e^{\mu t}\Lambda)dt + e^{\mu t}dN \leq 0.$ Assume  $\Psi(N, t) \le k_1$  is a solution to equation (6). Then,

$$\frac{\partial}{\partial t}\Psi(N,t) = (e^{\mu t}\mu N - e^{\mu t}\Lambda)$$
$$\frac{\partial}{\partial N}\Psi(N,t) = e^{\mu t}$$

Taking integration of the first equation w.r.t t, we get

$$\int \left(\frac{\partial \Psi(N,t)}{\partial t}\right) dt = \int (e^{\mu t} \mu N - e^{\mu t} \Lambda) dt + k_2$$
  

$$\Rightarrow \Psi(N,t) = e^{\mu t} N - e^{\mu t} \frac{\Lambda}{\mu} + k_2 \le k_1$$
  

$$-4.3cm \Rightarrow e^{\mu t} N - e^{\mu t} \frac{\Lambda}{\mu} \le k \quad (for \quad k = k_1 - k_2) \qquad (7)$$

At t=0,  $k = N(0) - \frac{\Lambda}{\mu}$ .

Upon substitution of k in equation (7) and making N the subject, we get

$$N \leq \left( N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t} + \frac{\Lambda}{\mu}.$$

Thus, as  $t \to \infty$ , we obtain

$$N(t) \le \frac{\Lambda}{\mu} \tag{8}$$

Therefore  $N(t) \leq \max\left\{N(0), \frac{\Lambda}{\mu}\right\} \quad \forall t > 0$ 

In the flea cycle, the Egg stage (F), the Pupal-Larval stage (L), and the Adult stage (A) are considered as populations separately.

For the the Egg stage (F), we express the 4th equation of (1) as

$$\frac{dF}{dt} \le \frac{\tau\Lambda}{\mu} - (\sigma + v_f)F \tag{9}$$

Solving (9) by use of integrating factor, we obtain

$$F \leq \left(F(0) - \frac{\tau\Lambda}{\mu(\sigma + v_f)}\right) e^{-(\sigma + v_f)t} + \frac{\tau\Lambda}{\mu(\sigma + v_f)}.$$

Thus, as  $t \to \infty$ , we get

$$F(t) \le \frac{\tau \Lambda}{\mu(\sigma + v_f)} \quad (10)$$

Therefore,  $F(t) \le \max\left\{F(0), \frac{\tau \Lambda}{\mu(\sigma + v_f)}\right\} \quad \forall t > 0$ For the Pupal-Larval stage (L) we rewrite the fifth equation of system (1) as  $\frac{dL}{dL} < \frac{\tau \sigma \Lambda}{\sigma \sigma \Lambda} = \left(\int_{0}^{\infty} \frac{F(\sigma)}{\sigma \sigma \sigma} + \frac{\sigma \sigma \Lambda}{\sigma \sigma}\right) = \int_{0}^{\infty} \frac{F(\sigma)}{\sigma \sigma} + \frac{\sigma \sigma \Lambda}{\sigma \sigma}$ 

$$\frac{dL}{dt} \le \frac{\tau\sigma\Lambda}{\mu(\sigma+v_f)} - \left(\frac{\epsilon\rho}{(1+L)} + v_l\right)L \qquad (11)$$

Since  $\epsilon \rho < (1 + L)$ , equation (11) can be expressed as  $\frac{dL}{dt} \le \frac{\tau \sigma \Lambda}{\mu(\sigma + v_f)} - (\epsilon \rho + v_l)L (12)$ 

Solving (12) by use of integrating factor, we obtain

$$L \leq \left(L(0) - \frac{\tau \sigma \Lambda}{\mu(\sigma + v_f)(\epsilon \rho + v_l)}\right) e^{-(\epsilon \rho + v_l)t} + \frac{\tau \sigma \Lambda}{\mu(\sigma + v_f)(\epsilon \rho + v_l)}.$$

As  $t \to \infty$ , we have

$$L(t) \le \frac{\tau \sigma \Lambda}{\mu(\sigma + v_f)(\epsilon \rho + v_l)}$$
(13)

Clearly,  $L(t) \le \max \left\{ L(0), \frac{\tau \sigma \Lambda}{\mu(\sigma + v_f)(\epsilon \rho + v_l)} \right\} \quad \forall t > 0$ Lastly, for the Adult stage (A) we have

$$\frac{dA}{dt} \le \frac{\epsilon \rho \tau \sigma \Lambda}{\mu (\sigma + v_f)(\epsilon \rho + v_l) + \tau \sigma \Lambda} - v_a A \qquad (14)$$

Solving (14) by use of integrating factor, we obtain

$$A \leq \left(A(0) - \frac{\epsilon \rho \tau \sigma \Lambda}{(\mu(\sigma + v_f)(\epsilon \rho + v_l) + \tau \sigma \Lambda)v_a}\right) e^{-v_a t} + \frac{\epsilon \rho \tau \sigma \Lambda}{(\mu(\sigma + v_f)(\epsilon \rho + v_l) + \tau \sigma \Lambda)v_a},$$

Hence, as  $t \to \infty$ , we have;

$$A(t) \leq \frac{\epsilon \rho \tau \sigma \Lambda}{(\mu(\sigma + v_f)(\epsilon \rho + v_l) + \tau \sigma \Lambda)v_a}$$
(15)  
Thus,  $A(t) \leq \max \left\{ A(0), \frac{\epsilon \rho \tau \sigma \Lambda}{(\mu(\sigma + v_f)(\epsilon \rho + v_l) + \tau \sigma \Lambda)v_a} \right\} \quad \forall t > 0$ 

From the above derivations, the region in which the solution of (1) is bounded is given by =(S, E, I,R, F, L, A)  $R_+^7$ ; N(t) max N(0),, F(t) max F(0),(+\_f), L(t) max L(0),(+\_f)(+\_l), A(t) max A(0), ((+\_f)(+\_l)+)\_a Furthermore, a solution of the model (1) that starts at time  $t \ge 0$  will always stay in the region. As result, this model is well posed.

**Remark 1** The seventh equation of system (1) is redundant and since R(t) = N(t) - (S(t) + E(t) + I(t)), it is enough to consider the first six equations of system (1). As a result, the remainder of this project will focus on system (16) in the region =(S, E, I, F, L, A)  $R_+^{+6}$ ; N(t) max N(0),, F(t) max F(0),(+\_f), L(t) max L(0),(+\_f)(+\_1), A(t) max A(0), ((+\_f)(+\_1)+)\_a , our new system becomes

$$\frac{dS}{dt} = \Lambda - (\beta A + \mu)S$$

$$\frac{dE}{dt} = \beta AS - (\alpha + \mu)E + \delta I$$

$$\frac{dI}{dt} = \alpha E - (\gamma + \mu + \delta)I$$

$$\frac{dF}{dt} = \tau I - (\sigma + v_f)F$$

$$\frac{dL}{dt} = \sigma F - \frac{\epsilon \rho L}{(1+L)} - v_l L$$

$$\frac{dA}{dt} = \frac{\epsilon \rho L}{(1+L)} - v_a A (16)$$

#### **Jiggers Free Equilibrium**

Without jiggers infestation, system (16), has a steady state solution referred to as Jiggers Free Equilibrium (JFE). In order to calculate the JFE point of this model, we equate the RHS of the equations of (16)to zero then let  $S = S^0$ ,  $E = E^0$ ,  $I = I^0 = 0$ ,  $F = F^0 = 0$ ,  $L = L^0 = 0$  and  $A = A^0 = 0$  and hence  $S^0 = \frac{\Lambda}{\mu}$ . The JFE will therefore be given by  $\mathcal{E}^0 = (S^0, E^0, I^0, F^0, L^0, A^0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0)$ 

#### **Basic Reproduction Number**

Studies by [15], outlined the  $R_0$  as the estimated number of new infectious cases that a normally infected person will cause in a completely susceptible community throughout the period of his or her infectious lifetime. We calculate the  $R_0$  using next generation matrix approach [16]. Applying this approach, we consider the following equations of infectious compartments

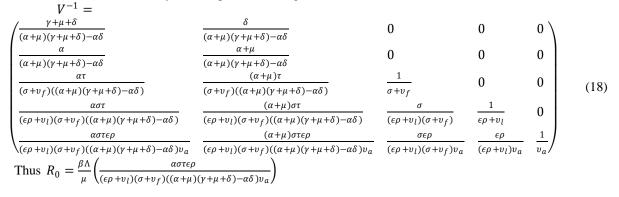
$$\frac{dE}{dt} = \beta AS - (\alpha + \mu)E + \delta I$$
$$\frac{dI}{dt} = \alpha E - (\gamma + \mu + \delta)I$$
$$\frac{dF}{dt} = \tau I - (\sigma + v_f)F$$
$$\frac{dL}{dt} = \sigma F - \frac{\epsilon \rho L}{(1+L)} - v_l L$$
$$\frac{dA}{dt} = \frac{\epsilon \rho L}{(1+L)} - v_a A (17)$$

From system (17) we have

Calculating the Jacobian matrices of  $f_i$  and  $v_i$  at  $\mathcal{E}^0$  we get

$$V = \begin{pmatrix} \alpha + \mu & -\delta & 0 & 0 & 0 \\ -\alpha & \gamma + \mu + \delta & 0 & 0 & 0 \\ 0 & -\tau & \sigma + v_f & 0 & 0 \\ 0 & 0 & -\sigma & \epsilon \rho + v_l & 0 \\ 0 & 0 & 0 & -\epsilon \rho & v_a \end{pmatrix}$$

respectively.  $R_0$  is given by  $\rho(FV^{-1})$  i.e. the largest absolute eigenvalue of the matrix  $FV^{-1}$ . Using Mathematica software to carry out computations, we get



#### Local stability of Jiggers-Free Equilibrium

Here, we will discuss the local stability of JFE.

**Theorem 1** The Jiggers-free equilibrium  $\mathcal{E}^0$  is locally asymptotically stable for  $R_0 < 1$  and unstable  $R_0 > 1$ .

*Proof.* We begin the proof by evaluating the Jacobian matrix of system (16) at  $\mathcal{E}^0$ 

$$J(\mathcal{E}^{0}) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & -\frac{\beta\Lambda}{\mu} \\ 0 & -(\alpha+\mu) & \delta & 0 & 0 & \frac{\beta\Lambda}{\mu} \\ 0 & \alpha & -(\gamma+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \tau & -(\sigma+v_{f}) & 0 & 0 \\ 0 & 0 & 0 & \sigma & -(\epsilon\rho+v_{l}) & 0 \\ 0 & 0 & 0 & 0 & \epsilon\rho & -v_{a} \end{pmatrix}$$
(19)

From matrix (19), the determinant is given by

 $Det = (\alpha + \mu)(\gamma + \mu + \delta)(\sigma + v_f)(\epsilon \rho + v_l)v_a\mu - (\sigma + v_f)(\epsilon \rho + v_l)v_a\alpha\delta\mu - \beta\Lambda\epsilon\rho\alpha\sigma\tau$ and the trace is obtained as

 $tr = -(\mu + (\alpha + \mu) + (\gamma + \mu + \delta) + (\sigma + v_f) + (\epsilon \rho + v_l) + v_a)$ 

Applying the Routh Hurwitz criterion [17], matrix (19) has negative eigenvalues because the trace (tr) < 0 and the determinant (Det) > 0 when  $R_0 < 1$ . Hence, the JFE point  $E^0$  is locally asymptotically stable whenever  $R_0 < 1$ 

#### **Endemic Equilibrium point**

This is a steady state when jiggers spread in the population. We denote Endemic Equilibrium by  $\mathcal{E}^* = (S^*, E^*, I^*, F^*, L^*, A^*)$ .

**Theorem 2** A unique endemic equilibrium point exists if  $R_0 > 1$ 

*Proof.* We begin the proof by replacing of (S, E, I, F, L, A) with  $\mathcal{E}^*$  in system (16) and equating the RHS to zero to obtain

 $0 = \Lambda - (\beta A^* + \mu)S^*$   $0 = \beta A^*S^* - (\alpha + \mu)E^* + \delta I^*$   $0 = \alpha E^* - (\gamma + \mu + \delta)I^*$   $0 = \sigma I^* - (\sigma + v_f)F^*$   $0 = \sigma F^* - \frac{\epsilon \rho L^*}{(1+L^*)} - v_l L^*$  $0 = \frac{\epsilon \rho L^*}{(1+L^*)} - v_a A^*(20)$ 

Inferring to the  $5^{th}$  and  $6^{th}$  equations of system (20), we deduce

$$F^* = \frac{v_l L^{2^*} + (v_l + \epsilon \rho) L^*}{\sigma(1 + L^*)}$$
(21)

 $A^* = \frac{\epsilon \rho L^*}{(1+L^*)v_a}.$  (22)

Using the  $4^{th}$  equation of system (20) and equation (21), we get

$$I^* = \frac{(\sigma + v_f)(v_l L^{2*} + (v_l + \epsilon \rho)L^*)}{\tau \sigma (1 + L^*)}.$$
(23)

Substituting equation (23) in the third equation of system (20) and rearranging, we deduce

$$E^* = \frac{(\gamma + \mu + \delta)(\sigma + v_f)(v_l L^{2*} + (v_l + \epsilon \rho)L^*)}{\alpha \tau \sigma (1 + L^*)}.$$
 (24)

Using the first of system (20) and equation (22), we get

$$S^* = \frac{(1+L^*)v_a\Lambda}{\beta\epsilon\rho\,L^* + (1+L^*)v_a\mu} \tag{25}$$

Now we substitute equations (22), (23), (24) and (25) in the the  $2^{nd}$  equation of system (20) an simplify the resulting equation to obtain

$$A_2 L^{2*} + A_1 L^* + A_0 = 0 (26)$$

where

$$A_0 = -\beta \epsilon \Lambda \alpha \tau \sigma + (\epsilon \rho + v_l) (\sigma + v_f) ((\alpha + \mu)(\gamma + \mu + \delta) - \alpha \delta) v_a \mu$$

$$A_{1} = -\beta\epsilon\Lambda\alpha\tau\sigma + (\epsilon\rho + v_{l})(\sigma + v_{f})((\alpha + \mu)(\gamma + \mu + \delta) - \alpha\delta)v_{a}\mu + (\alpha(\gamma + \mu) + \mu(\gamma + \mu + \delta))(\sigma + v_{f})(v_{a}\mu v_{l} + \beta\epsilon\rho(v_{l} + \epsilon\rho))$$

$$A_{2} = \alpha(\gamma + )(\sigma + v_{f})\beta\epsilon\rho v_{l} + \mu(\gamma + \mu + \delta)(\sigma + v_{f})\beta\epsilon\rho v_{l} + v_{a}\mu\alpha(\gamma + \mu)(\sigma + v_{f})v_{l}$$

$$+ v_{a}\mu^{2}(\gamma + \mu + \delta)(\sigma + v_{f})v_{l}$$

Hence, the number of possible positive real zeros of equation (26) is dependent on the signs of  $A_2$ ,  $A_2$  and  $A_0$ . Using the Descartes Rule of signs by [18], we analyze equation (26).

Clearly, 
$$A_2 > 0$$
,  $A_0 \begin{cases} > 1ifR_0 < 1 \\ < 1ifR_0 > 1 \end{cases}$  and  
$$A_1 \begin{cases} > 1ifR_0 > 1 \end{cases}$$
 and  
 $A_1 \begin{cases} > 1ifR_0 > 1 \\ < 1ifR_0 > 1and \begin{pmatrix} \alpha(\gamma + \mu) + \mu(\gamma + \mu + \delta))(\sigma + v_f)(v_a\mu v_l + \beta\epsilon\rho(v_l + \epsilon\rho)) < \\ ((\epsilon\rho + v_l)(\sigma + v_f)((\alpha + \mu)(\gamma + \mu + \delta) - \alpha\delta)v_a\mu - \beta\epsilon\rho\Lambda\alpha\tau\sigma) \end{pmatrix}$   
 $> 1ifR_0 > 1and \begin{pmatrix} \alpha(\gamma + \mu) + \mu(\gamma + \mu + \delta))(\sigma + v_f)(v_a\mu v_l + \beta\epsilon\rho(v_l + \epsilon\rho)) > \\ ((\epsilon\rho + v_l)(\sigma + v_f)((\alpha + \mu)(\gamma + \mu + \delta) - \alpha\delta)v_a\mu - \beta\epsilon\rho\Lambda\alpha\tau\sigma) \end{pmatrix}$ 

According to [18], the number of positive real zeros of equation (6.1) is equal to the number of changes in the signs of the coefficients of equation (6.1) or less than this by an even number. Thus we summarize the possibilities of positive zeros (6.1) in Table 1

Cases	A <sub>2</sub>	A <sub>1</sub>	$A_0$	$\hat{R}_0$	Number of	Number of Positive real zeros
i.	+	+	+	$R_0 < 1$	0	0
ii.	+	-	-	$R_0 > 1$	1	1
iii.	+	+	-	$R_0 > 1$	1	1

Table1: Zeros of Equation (26)

From cases ii and iii in Table 1 it is clear that a unique endemic equilibrium exists whenever  $R_0 > 1$ 

#### Local stability of endemic equilibrium point

Here, we investigate local stability of  $\mathcal{E}^*$ 

**Theorem 3**  $\mathcal{E}^*$  is locally asymptotically stable if  $R_0 > 1$ 

*Proof.* We use Center manifold theory [19]to prove this theorem by investigating the existence of forward bifurcation at  $R_0 = 1$ . Applying Theorem 4.1 of [19], we consider the transmission rate  $\beta$  as bifurcation parameter such that  $R_0 = 1$  if and only if

$$\beta = \beta^* = \frac{(\epsilon \rho + v_l)(\sigma + v_f)((\alpha + \mu)(\gamma + \mu + \delta) - \alpha \delta)v_a \mu}{\Lambda \alpha \sigma \tau \epsilon \rho}$$

DOI: 10.9790/5933-1403024659

Then we let  $S = x_1$ ,  $E = x_2$ ,  $I = x_3$ ,  $F = x_4$ ,  $L = x_5$ ,  $A = x_6$  and  $\beta = \beta^*$ Thus system (16) becomes

$$\frac{dx_1}{dt} = \Lambda - (\beta^* x_6 + \mu) x_1$$

$$\frac{dx_2}{dt} = \beta^* x_6 x_7 - (\alpha + \mu) x_2 + \delta x_3$$

$$\frac{dx_3}{dt} = \alpha x_2 - (\gamma + \mu + \delta) x_3$$

$$\frac{dx_4}{dt} = \tau x_3 - (\sigma + v_f) x_4$$

$$\frac{dx_5}{dt} = \sigma x_4 - \frac{\epsilon \rho x_5}{(1 + x_5)} - v_l x_5$$

$$\frac{dx_6}{dt} = \frac{\epsilon \rho x_5}{(1 + x_5)} - v_a x_6$$
(27)

**Remark 2** System (26) can be rewritten as  $\frac{dX}{dt} = H(x)$  where  $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$  and  $H = (h_1, x_2, x_3, x_4, x_5, x_6)^T$  $h_2$ ,  $h_3$ ,  $h_4$ ,  $h_5$ ,  $h_6$ )<sup>T</sup> Computing the Jacobian matrix of system (26), at  $\mathcal{E}^0$  we obtain

$$J(\mathcal{E}^{0}) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & -\frac{\beta^{*}\Lambda}{\mu} \\ 0 & -(\alpha+\mu) & \delta & 0 & 0 & \frac{\beta^{*}\Lambda}{\mu} \\ 0 & \alpha & -(\gamma+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \tau & -(\sigma+v_{f}) & 0 & 0 \\ 0 & 0 & 0 & \sigma & -(\epsilon\rho+v_{l}) & 0 \\ 0 & 0 & 0 & 0 & \epsilon\rho & -v_{a} \end{pmatrix}$$
(28)

Let  $\mathbf{u} = (u_1, u_2, u_3, u_4, u_5, u_6)^T$  be the right eigenvector of matrix (27) when  $R_0 = 1$  then  $\beta^* \Lambda$ 

$$\begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & -\frac{\mu}{\mu} \\ 0 & -(\alpha + \mu) & \delta & 0 & 0 & \frac{\beta^* \Lambda}{\mu} \\ 0 & \alpha & -(\gamma + \mu + \delta) & 0 & 0 & 0 \\ 0 & 0 & \tau & -(\sigma + v_f) & 0 & 0 \\ 0 & 0 & 0 & \sigma & -(\epsilon\rho + v_l) & 0 \\ 0 & 0 & 0 & 0 & \epsilon\rho & -v_a \end{pmatrix} \begin{pmatrix} u_1 \\ u_2 \\ u_3 \\ u_4 \\ u_5 \\ u_6 \end{pmatrix} = \mathbf{0}$$

$$\Rightarrow \begin{cases} -\mu u_1 - \frac{\beta^* \Lambda}{\mu} u_6 = 0 \\ -(\alpha + \mu) u_2 + \delta u_3 + \frac{\beta^* \Lambda}{\mu} u_6 = 0 \\ -(\alpha + \mu) u_2 + \delta u_3 + \frac{\beta^* \Lambda}{\mu} u_6 = 0 \\ \pi u_2 - (\gamma + \mu + \delta) u_3 = 0 \\ \tau u_3 - (\sigma + v_f) u_4 = 0 \\ \sigma u_4 - (\epsilon\rho + v_l) u_5 = 0 \\ \epsilon\rho u_5 - v_a u_6 = 0 \end{cases}$$

$$\Rightarrow \begin{cases} u_1 = -\frac{\beta^* \Lambda \alpha \tau \sigma \epsilon \rho}{\mu^2 (\epsilon\rho + v_l) (\sigma + v_f) (\gamma + \mu + \delta) v_a} u_2 \\ u_2 = u_2 > 0 \\ u_3 = \frac{\alpha \tau}{(\gamma + \mu + \delta)} u_2 \\ u_4 = \frac{\alpha \tau \sigma}{(\epsilon\rho + v_l) (\sigma + v_f) (\gamma + \mu + \delta)} u_2 \\ u_5 = \frac{\alpha \tau \sigma \epsilon \rho}{(\epsilon\rho + v_l) (\sigma + v_f) (\gamma + \mu + \delta) v_a} u_2 \end{cases}$$

Also, let  $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6)^T$  be the left eigenvector of matrix (28) corresponding with zero eigenvalue

then

$$\begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\alpha + \mu) & \alpha & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\sigma + v_f) & \sigma & 0 \\ 0 & 0 & 0 & 0 & -(\epsilon\rho + v_l) & \epsilon\rho \\ -\frac{\beta^*\Lambda}{\mu} & \frac{\beta^*\Lambda}{\mu} & 0 & 0 & 0 & -v_a \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \end{pmatrix} = \mathbf{0}$$

$$\Rightarrow \begin{cases} -\mu v_1 = 0 \\ -(\alpha + \mu)v_2 + \alpha v_3 = 0 \\ \delta v_2 - (\gamma + \mu + \delta)v_3 + \tau v_4 = 0 \\ -(\sigma + v_f)u_4 + \sigma v_5 = 0 \\ -(\epsilon\rho + v_l)v_5 + \epsilon\rho v_6 = 0 \\ -\frac{\beta^*\Lambda}{\mu}v_1 + \frac{\beta^*\Lambda}{\mu}v_2 - v_a v_6 = 0 \\ v_3 = \frac{\beta^*\Lambda\epsilon\rho\sigma + \delta(\epsilon\rho + v_l)(\sigma + v_f)v_a\mu}{(\gamma + \mu + \delta)(\epsilon\rho + v_l)(\sigma + v_f)v_a\mu}v_2 \\ v_4 = \frac{\beta^*\Lambda\epsilon\rho\sigma}{(\epsilon\rho + v_l)(\sigma + v_f)v_a\mu}v_2 \\ v_5 = \frac{\beta^*\Lambda\epsilon\rho}{(\epsilon\rho + v_l)v_a\mu}v_2 \\ v_6 = \frac{\beta^*\Lambda}{v_a\mu}v_2 \end{cases}$$

Now, as explained in Theorem 4.1 of [19], we derive the related bifurcation parameters, a and b;  $n = \frac{1}{2}$ 

$$a = \sum_{k,i,j=1}^{n} v_k u_i u_j \frac{\partial^2 h_k}{\partial x_i \partial x_j} (0,0)$$
$$b = \sum_{k,i=1}^{n} v_k u_i \frac{\partial^2 h_k}{\partial x_i \partial \beta^*} (0,0)$$

We first get the non-zero partial derivatives of model system (27) evaluated at  $\mathcal{E}^0$  to get the corresponding bifurcation coefficient a. As a result, it follows that

$$\frac{\frac{\partial^2 h_1}{\partial x_1 \partial x_6} = -\beta}{\frac{\partial^2 h_2}{\partial x_1 \partial x_6} = \beta}$$
$$\frac{\frac{\partial^2 h_2}{\partial x_5} = 2\epsilon\rho}{\frac{\partial^2 h_6}{\partial x_5^2} = -2\epsilon\rho$$
(29)

so that

$$a = v_{1}u_{1}u_{6}\frac{\partial^{2}h_{1}}{\partial x_{1}\partial x_{6}} + v_{2}u_{1}u_{6}\frac{\partial^{2}h_{2}}{\partial x_{1}\partial x_{6}} + v_{5}u_{5}^{2}\frac{\partial^{2}h_{5}}{\partial x_{5}^{2}} + v_{6}u_{5}^{2}\frac{\partial^{2}h_{6}}{\partial x_{5}^{2}}$$

$$= v_{2}u_{2}^{2}\left[-\frac{\beta^{2}\Lambda\alpha^{2}\tau^{2}\sigma^{2}\epsilon^{2}\rho^{2}}{(\epsilon\rho+v_{l})^{2}(\sigma+v_{f})^{2}(\gamma+\mu+\delta)^{2}v_{a}^{2}\mu^{2}} + \frac{2\beta\Lambda\epsilon^{2}\rho^{2}\alpha^{2}\tau^{2}\sigma^{2}}{(\epsilon\rho+v_{l})^{3}(\sigma+v_{f})^{2}(\gamma+\mu+\delta)^{2}v_{a}\mu} - \frac{2\beta\Lambda\epsilon\rho\alpha^{2}\tau^{2}\sigma^{2}}{(\epsilon\rho+v_{l})^{2}(\sigma+v_{f})^{2}(\gamma+\mu+\delta)^{2}v_{a}\mu}\right]$$

$$= -v_{2}u_{2}^{2}\left[\frac{\beta^{2}\Lambda\alpha^{2}\tau^{2}\sigma^{2}\epsilon^{2}\rho^{2}}{(\epsilon\rho+v_{l})^{2}(\sigma+v_{f})^{2}(\gamma+\mu+\delta)^{2}v_{a}^{2}\mu^{2}} + \frac{2\beta\Lambda\epsilon\rho\alpha^{2}\tau^{2}\sigma^{2}}{(\epsilon\rho+v_{l})^{3}(\sigma+v_{f})^{2}(\gamma+\mu+\delta)^{2}v_{a}\mu}\left(1 - \frac{\epsilon\rho}{(\epsilon\rho+v_{l})}\right)\right]$$
(30)

(30)

Furthermore, the non-zero partial derivatives associated with b are

$$\frac{\partial^2 h_1}{\partial x_6 \partial \beta^*} = -\frac{\Lambda}{\mu}$$
$$\frac{\partial^2 h_2}{\partial x_6 \partial \beta^*} = \frac{\Lambda}{\mu}$$
(31)

DOI: 10.9790/5933-1403024659

so that

$$b = v_1 u_6 \frac{\partial^2 h_1}{\partial x_6 \partial \beta^*} (0,0) + v_2 u_6 \frac{\partial^2 h_2}{\partial x_6 \partial \beta^*} (0,0)$$
  
$$= v_2 \frac{\alpha \tau \sigma \epsilon \rho}{(\epsilon \rho + v_l)(\sigma + v_f)(\gamma + \mu + \delta) v_a} u_2 \frac{h}{\mu}$$
  
$$= v_2 u_2 \left( \frac{\Lambda \alpha \tau \sigma \epsilon \rho}{(\epsilon \rho + v_l)(\sigma + v_f)(\gamma + \mu + \delta) v_a \mu} \right)$$
  
$$> 0$$
  
(32)

Forward bifurcation happens at  $\beta = \beta^*(R_0 = 1)$  according to Theorem 4.1 in[19]. If  $\beta > \beta^*$ , the theorem states that there exists a positive equilibrium point that is locally asymptotically stable. It's worth noting that  $\beta > \beta^*$  implies  $R_0 > 1$ . As a result, if  $R_0 > 1$ , the sole endemic equilibrium point that occurs if  $R_0 > 1$  is locally asymptotically stable.

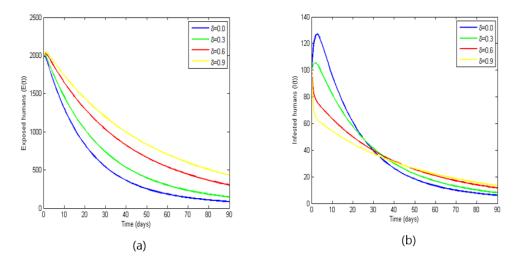
### **III. NUMERICAL SIMULATION**

Utilizing the values in Table 2, numerical simulations are run to visually depict the dynamics of a Jiggers infestation.

Parameter	Description	Value	Source
Λ	The recruitment rate of the human population into susceptible	5.36	[20]
β	Exposure rate of the susceptible humans	$8.5e^{-6}$	Estimated
α	The infection rate of the exposed humans	$5.03e^{-2}$	Estimated
γ	The recovery rate of the infected humans	0.731	Estimated
μ	The natural death rate of humans	$5.4e^{-5}$	[20]
δ	The rate at which the infested humans become exposed	0.2	Estimated
$v_f$	The rate at which sand flea eggs die naturally	0.09	[21]
$v_l$	Sand flea larval and pupal natural mortality rates	0.049525	[21]
$v_a$	Adult sand flea mortality rates	0.005	[21]
τ	The rate at which eggs are laid by sand flea from infected humans	0.001	Estimated
σ	The rate at which flea eggs develop into larval and pupal stages	0.0126665	[21]
ρ	The rate at which larvae develop into adult sand fleas	0.016665	[21]
$\epsilon$	The proportion of larvae that develop into adult fleas	0.2	[21]

Table 2: Parameter valu	ec

# IV. RESULTS AND DISCUSSIONS



#### Figure 2: The dynamics of exposed and infested groups at different rates of incomplete recoveries

Figure 2 displays the curves of the solutions of exposed and infested classes at different rates of incomplete recovery in a and b respectively.

Figure 2: (a) The dynamics of exposed population at different rates of incomplete recoveries. The

incomplete recovery rate ( $\delta$ ) ranges from 0 to 0.9, where  $\delta = 0$  implies that there is no incomplete recovery. As the incomplete recovery rate increases, the curves of the exposed class take much time to converge to zero

(b) The dynamics of infested population at different rates of incomplete recoveries. The incomplete recovery rate ( $\delta$ ) ranges from 0 to 0.9, where  $\delta = 0$  implies that there is no incomplete recovery. As incomplete recovery rate increases, the curves of the infested class take much time to converge to zero.

This indicates that when there is high incomplete rate, jiggers infestation will somehow persist in he population before eradication.

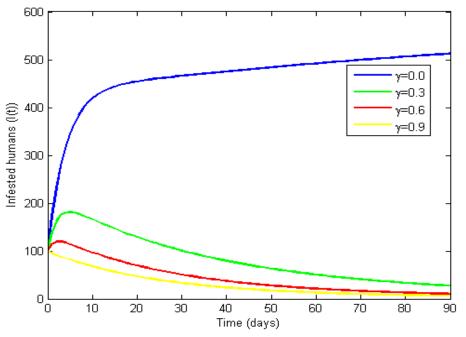
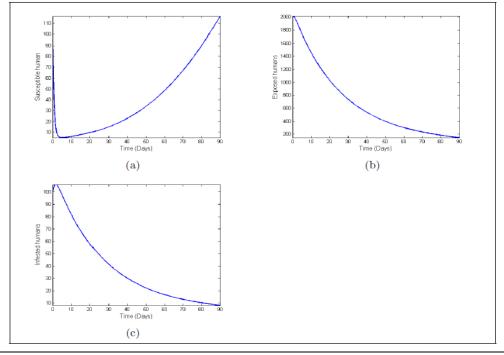


Figure 3: The dynamics of infested population at different rates of treatments.

Figure 3 shows the curves of the solutions of infested class at different rates of treatment. The treatment rate of  $\gamma = 0$  represents no treatment. When  $\gamma = 0$  the curve demonstrate that Jiggers infestation persists in the population. Also as the rate of treatment increases, Jiggers infestation decreases in the population.



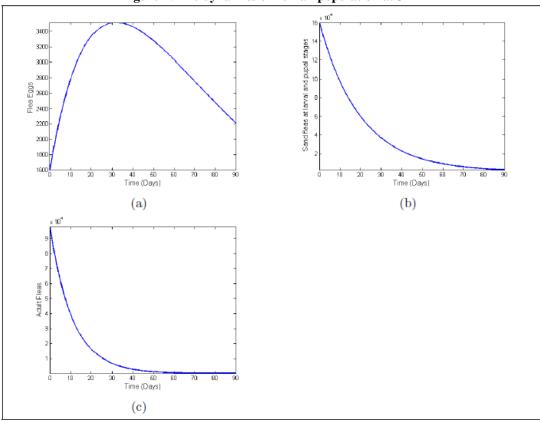


Figure 4: The dynamics of human population at JFE

Figure 5: The dynamics of sand flea population at JFE

Figure 4 and 5 shows the local stability of jiggers-free equilibrium when  $R_0 = 4.9827e^{-13} < 1$  as  $t \to \infty$  for susceptible population (a), exposed population (b), infested population (c) in humans whereas; flea eggs (a), sand fleas at larval and pupal stages (b) and adult fleas (c) in the flea segments. From the Figure 4 (a)-(c) and Figure 5 (a)-(c), it can be seen that the solution curves of various compartments tend to the JFE. That is, the solution curve of susceptible population tends to  $\frac{\Lambda}{\mu}$  while other solution curves tend to zero. Thus, the theoretical results in Theorem 1 are similar to numerical simulation results.

# V. CONCLUSION

A mathematical model of jiggers' infestation with incomplete recoveries incorporating the flea population was developed in this research project. We conducted an analysis of the model taking into account the invariant region, and came to the conclusion that the model is well-posed and that the solution sets enter and remain in the region for all time to come. After examining the model's positivity, it was determined that all state variables are positive for all time t.

We computed the  $R_0$  using next generation matrix approach[16]. We also analyzed the local stability of Jiggers free equilibrium point and endemic equilibrium point by applying the Routh Hurwitz criterion [17] and Center manifold theory [19] respectively. This analysis depicted that, the Jiggers free equilibrium point is locally asymptotically stable when  $R_0 < 1$  and the endemic equilibrium point is locally asymptotically stable when  $R_0 > 1$ .

Numerical Simulation results showed that the given system approaches the Jiggers-free equilibrium (JFE) which is consistent with Theorem 1. It also showed that Jiggers infestation decreases with increase of treatment, in Muranga County, Kenya.

# VI. ACKNOWLEDGEMENTS

For their help throughout this research, I am grateful to the faculty and employees of Kenyatta University's Department of Mathematics. Special acknowledgments to Dr Nancy Imbusi Matendechere, Technical University of Kenya, for her assistance towards research topic.

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