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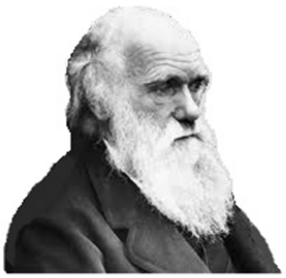

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Mathematical Modeling of Control Strategies for *Visceral leishmaniasis*

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ABSTRACT

In this paper, a deterministic mathematical model for the transmission dynamics of *Visceral leishmaniasis* (VL) was formulated and analyzed. The basic reproductive number R_0 was obtained using the next generation matrix method. The model which was parameterized using the 2011 cases of Visceral Leishmaniasis in South Sudan, was used to assess two control measures. Numerical simulation reveals that the exposed population is reduced by high detection rate, and low biting rate of sandflies. Further, our model simulation gave the values of the contact rate of susceptible human, α_1 and the detection rate of new cases, σ at which the basic reproductive number is less than one, ($R_0 < 1$), equal to one ($R_0 = 1$) and greater than one ($R_0 > 1$).

***Keywords*:** Stability, equilibrium, parameterization, endemic, basic reproductive number, simulation, visceral Leishmaniasis, *Visceral leishmaniasis*, kala-azar, *Cutaneous leishmanisis*

1. INTRODUCTION

The Leishmaniases are a conglomerate of ailments caused by protozoan parasites from more than 20 *Leishmania* species. These parasites are transmitted to human following the bite of an infected female Phlebotomine sandfly [1]. There exist three major configuration Leishmaniasis: *Cutaneous leishmanisis* (CL), *Visceral leishmaniasis* (VL), also known as kala – azar, and *Mucocutaneous leishmaniasis* (MCL). CL is the most prevalent type, VL is the most critical type and MCL is the most crippling type of the ailment.

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Some cases of visceral leishmaniasis is associated with symptoms which involve the lungs, pleura, oral mucosa, larynx, esophagus, stomach, small intestine, skin and bone marrow [2]. Post-kala-azar dermal leishmaniasis (PKDL) is a type of VL that usually results in skin eruption, either extemporaneously or as the aftermath of treatment [3]. Different regions and sub-regions of the world are associated with varying *Leishmania* species, for instance *Leishmania donovani* is common in East Africa, while *Leishmania infantum* is the leading species in Central Asia and North Africa.

The use of mathematical modeling to seek understanding of disease dynamics and in the assessment of control strategies dates back to the 19th century [4]. Bacaër and Guernaoui [5] developed a model that considered the progression from latent to symptomatic stage in human as well as seasonality and fitted their model to real data. Stauch *et al.* [6] supported the elimination of VL program in the Indian sub-region by providing information on the basic reproductive number and other intervention parameters. Mohsim *et al.* [7] presented and analyzed a mathematical model that describes the dynamics of visceral leishmaniasis in a population with immigration of infective humans under mass vaccination strategy. Ribas *et al.* [8] used mathematical model to compare the performance of five control strategies in the control of zoonotic *Visceral leishmaniasis.*

Subramanian *et al*. [9] developed a compartment-based mathematical model of zoonotic *Visceral leishmaniasis* transmission among three different populations—human, animal and sandfly; dividing the human class into asymptomatic, symptomatic, post-kala-azar dermal leishmaniasis and transiently infected. Zamir *et al.* [10] proposed a mathematical model of visceral leishmaniasis epidemic with saturated infection incidence. Lutte *et al.* [11] fitted different visceral leishmaniasis transmission models that accounts for structural differences to data from six districts in Bihar India.

Biswas [12] studied and analyzed the dynamics of *Visceral leishmaniasis* incidence data from South Sudan. Agyingi & Wiandt [13] incorporated time delays into a SIS model that describes the transmission dynamics of *Cutaneous leishmaniasis*. Pantha & Elmojtaba [14] developed a deterministic model of *Visceral leishmaniasis* in human, vector and reservoir, carried out sensitivity of the basic reproductive number and applied optimal control theory using three variables. Gandhi *et al.* [15] characterized *Visceral leishmaniasis* time delay dynamical model and established the condition for the system's stability to switch and produces a periodic solution.

In this paper we coupled an SEIR model for human to an SI model for sandfly. The aim is to obtain data driven characteristics of leishmanisis in endemic population and evaluate two main leishmaniasis control strategies. The rest of the papers is organized as follows. In section 2 the model equation is described following sandfly life cycle and some basic assumptions. Section 3 captured the equilibrium and stability analysis via the threshold, basic reproductive number. Section 4 is dedicated for the model evaluation where data from South Sudan was used to parameterize the model followed by numerical simulation.

2. MATERIALS AND METHODS

2. 1. Model formulation

Here we used compartmental modeling strategy, where the human and sandfly population are enclosed in appropriate epidemiological groups. Specifically, we coupled an SEIR model

for the human population to an SI model for the sandfly population. We assumed that the susceptible human population are recruited by either immigration or by birth at the rate β_1 and that a certain proportion of it become infected at the rate $\alpha_1, = ab_1$, where a is the per capital biting rate of sandfly on human and, b_1, is the transmission probability of VL per bite per human. This newly infected proportion is transferred to the exposed compartment where they remain until the expiration of the incubation period. A certain proportion in the exposed compartment, are detected and moved to the infectious compartment at the rate σ.

Following a successful treatment, a certain proportion of the infectious compartment are transferred to the recovered compartment at the rate γ. Members in all human compartments die naturally at the rate μ_1 and in addition human in the infectious compartment experiences death due to VL at the rate ρ. Thus, the human population at time t, $N_H(t)$, comprises of the $S(t)$, for the susceptible human population at a time t, $E(t)$ for the exposed human population at a time t, $I(t)$ for the infected human population at time t and $R(t)$ for the recovered human population at time t, such that $N_H(t) = S_1(t) + E(t) + I_1(t) + R(t)$.

The sandfly population is represented by two compartments, namely the susceptible and the infectious compartments. The susceptible compartment is recruited by birth at the rate β_2, and became infectious following a bite for blood meal from an infected human at the rate $\alpha_2 = ab_2$, where a is as above and b_2 is the transmission probability for sandfly infection with VL from human.

Since sandfly due not become ill of leishmanisis, sandfly population experiences only natural death at the rate μ_2. Similarly the sandfly population at time t, $N_S(t)$ comprises of $S_2(t)$ for the susceptible sandfly population at time t, and $I_2(t)$ for the infected sandfly population at time t, such that $N_S(t) = S_2(t) + I_2(t)$.

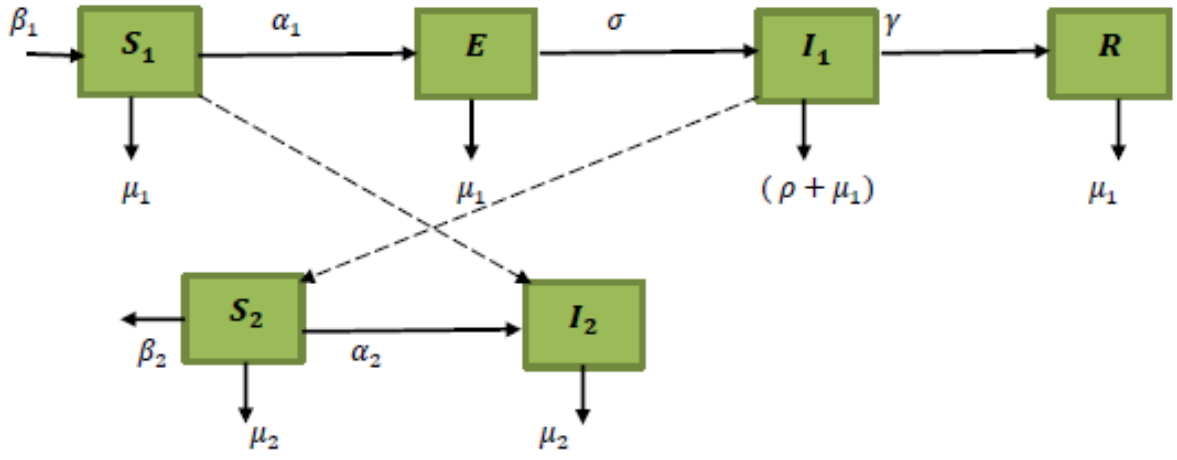

Figure 1. The schematic diagram for VL transmission.

Following the model formulation above and the schematic diagram Figure 1, we present our model as follows:

$$\frac{dS_1}{dt} = \beta_1 - \frac{\alpha_1 S_1 I_2}{N_H} - \mu_1 S_1, \quad (1)$$

$$\frac{dE}{dt} = \frac{\alpha_1 S_1 I_2}{N_H} - (\sigma + \mu_1)E, \quad (2)$$

$$\frac{dI_1}{dt} = \sigma E - (\gamma + \rho + \mu_1)I_1, \quad (3)$$

$$\frac{dR}{dt} = \gamma I - \mu_1 R, \quad (4)$$

$$\frac{dS_2}{dt} = \beta_2 - \frac{\alpha_2 S_2 I_1}{N_H} - \mu_2 S_2, \quad (5)$$

$$\frac{dI_2}{dt} = \frac{\alpha_2 S_2 I_1}{N_H} - \mu_2 S_2. \quad (6)$$

Table 1. Model parameters, their description and values.

Parameters	Description	Value	Reference
β_1	Recruitment rate of susceptible human,	200 day^{-1}	[19]
β_2	Recruitment rat of sand flies,	500 day^{-1}	[19]
α_1	Transmission rate of human,	0.00003	Estimated
μ_1	The natural death rate of human	$2.7\times10^{-5}\,days^{-1}$	[20]
a	Biting rate of sandflies on human	0.25	[6]
b_1	Transmission probability from infected sandfly to human	0.00012	Estimated
b_2	Transmission probability from infected human to sandfly	0.045	Estimated
σ	Detection rate of infected human	0.045	Estimated
γ_1	Recovery rate of infected human	0.21	[19]
α_2	Transmission rate of sand flies	0.025	Estimated
μ_2	Natural death rate of sand flies	$0.073\,days^{-1}$	[19]
ρ	Death rate of infected human due to visceral leishmaniasis	0.0112	[21]

2. 2. Positivity of solutions

Consider the region: $D=\left\{\left(S_1,E,I_1,R,S_2,I_2\right)\in\Re_+^6 : N_H\le\frac{\beta_1}{\mu_1},N_S\le\frac{\beta_2}{\mu_2}\right\}$. It can be shown that the set D is positively invariant and an attractor of all positive solutions of the system (1) to (6)

Lemma 1: The region D is invariant for the system (1) to (6)

Proof:

The rate of change of the total human population is given by

$$\frac{dN_H}{dt}=\frac{dS_1}{dt}+\frac{dE}{dt}+\frac{dI_1}{dt}=\beta_1-(\rho I_1+\mu_1 N_1). \tag{7}$$

While the rate of change of the total sandfly population is given by

$$\frac{dN_S}{dt}=\frac{dS_2}{dt}+\frac{dI_R}{dt}=\beta_2-\mu_2 N_S. \tag{8}$$

By standard comparison theorem Martynyuk *et al.* [16],

$$\frac{dN_H}{dt}\le\beta_1-\mu_1 N_H \quad \text{and} \quad \frac{dN_S}{dt}\le\beta_2-\mu_2 N_S.$$

Using the integrating factor method we have, $N_H(t)\le\frac{\beta_1}{\mu_1}+\left(N_H(0)-\frac{\beta_1}{\mu_1}\right)e^{-\mu_1 t}$, and $N_S(t)\le\frac{\beta_1}{\mu_2}+\left(N_S(0)-\frac{\beta_2}{\mu_2}\right)e^{-\mu_2 t}$.

If $N_H(0)\le\frac{\beta_1}{\mu_1}$, then $N_H(t)\le\frac{\beta_1}{\mu_1}$ and if $N_S(0)\le\frac{\beta_2}{\mu_2}$, then $N_S(t)\le\frac{\beta_2}{\mu_2}$.

Then all solutions enter D in finite time and remain non-negative for non-negative initial conditions. In this region, the system (1) to (6) is said to be well posed mathematically and epidemiologically.

2. 3. Local Stability of Disease-free Equilibrium

The disease-free equilibrium of the model is obtained by setting the right hand side of the model (1) to (6) to zero and solving the resulting equation to obtain;

$$E^* = \begin{pmatrix} S_1^* \\ E^* \\ I_1^* \\ R \\ S_2^* \\ I_2^* \end{pmatrix} = \begin{pmatrix} \frac{\beta_1}{\mu_1} \\ 0 \\ 0 \\ 0 \\ \frac{\beta_2}{\mu_2} \\ 0 \end{pmatrix} \tag{10}$$

2. 4. The basic reproductive number

The basic reproductive number is a principal threshold in disease control. It is the number of persons and infected persons will infect during their infectious period. It is one of the principal tools threshold that can be used to determine whether a steady state is an attractor or fiddler. To establish the basic reproductive number of our model, we use the technique in Driessche & Watmough [17] Our model has three infective compartments namely the exposed human E , infected human, I_1 infected sandfly I_2. Given that F is the matrix of new infections and V is the matrix of transfer of infective proportion, we have;

$$F = \begin{pmatrix} 0 & 0 & \frac{\alpha_1 S_1}{N} \\ 0 & 0 & 0 \\ \frac{\alpha_1 S_1}{N} & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} -(\sigma+\mu_1) & 0 & 0 \\ \sigma & -(\gamma+\rho+\mu_1) & 0 \\ 0 & 0 & -\mu_1 \end{pmatrix} \tag{11}$$

where:

$$Adj(V) = \begin{bmatrix} \mu_2(\gamma+\rho+\mu_1) & 0 & 0 \\ \delta\mu_2 & \mu_2(\sigma+\mu_1) & 0 \\ 0 & 0 & (\sigma+\mu_1)(\gamma+\rho+\mu_1) \end{bmatrix}$$

and

$$\det(V) = -\mu_2(\sigma+\mu_1)(\gamma+\rho+\mu_1) \tag{12}$$

$$V^{-1} = \begin{bmatrix} -\frac{1}{\sigma+\mu_1} & 0 & 0 \\ -\frac{\delta}{(\sigma+\mu_1)(\gamma+\rho+\mu_1)} & -\frac{1}{(\gamma+\rho+\mu_1)} & 0 \\ 0 & 0 & -\frac{1}{\mu_2} \end{bmatrix} \tag{13}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\alpha_1 S_1}{N} \\ 0 & 0 & 0 \\ \frac{\alpha_2 S_2}{N} & 0 & 0 \end{bmatrix} \cdot \begin{bmatrix} -\frac{1}{\delta+\mu_1} & 0 & 0 \\ \frac{-\delta}{(\sigma+\mu_1)(\gamma+\rho+\mu_1)} & \frac{-1}{(\gamma+\rho+\mu_1)} & 0 \\ 0 & 0 & \frac{-1}{\mu_2} \end{bmatrix},$$

$$= \begin{bmatrix} 0 & 0 & \frac{-\alpha_1 S_1}{N\mu_2} \\ 0 & 0 & 0 \\ \frac{-\alpha_2 S_2}{N(\sigma+\mu_1)} & 0 & 0 \end{bmatrix}. \quad (14)$$

The characteristic equation $(A-\lambda I)=0$ of equation (14) is given as

$$\begin{bmatrix} -\lambda & 0 & \frac{-\alpha_1 S_1}{N_H \mu_2} \\ 0 & -\lambda & 0 \\ \frac{-\alpha_2 S_2}{N_H(\sigma+\mu_1)} & 0 & -\lambda \end{bmatrix} = 0 \quad (15)$$

The spectral radius (dominant eigenvalue) of (15) and hence the basic reproductive number is

$$R_0 = \sqrt{\frac{\alpha_1 \alpha_2 S_1^* S_2^*}{\mu_2(\sigma+\mu_1)(N_H^*)^2}} \quad (16)$$

2. 4. 1. Interpretation of the basic reproductive number

From the next-generation matrix approach Driessche & Watmough [17], and in reference to (16), $\frac{\alpha_1 S_1}{N_H \mu_2}$ is the number of individuals infected by one infected sandfly in a population where everyone is naïve, $\frac{\alpha_2 S_2}{N_H(\sigma+\mu_2)}$ is the number of sandfly an infected human will infect as long as he/she remains infectious. This reproduction number derived by the next-generation approach serves as a threshold condition for the stability of the disease-free equilibrium, but it is difficult to interpret epidemiologically.

Theorem 1. The disease free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.

Proof:

The local stability of the disease free equilibrium is a direct consequence of Driessche & Watmough [17]. This can be supported by showing that the Jacobian (17) of the system (1) to (6) at the disease free equilibrium (10) has negative eigenvalues.

At the disease free equilibrium, the Jacobian of the model is given by:

$$J_{E^*} = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & -\frac{\alpha_1 S_1^*}{N} \\ 0 & -(\sigma+\mu_1) & 0 & 0 & 0 & \frac{\alpha_1 S_1^*}{N} \\ 0 & \sigma & -(\mu_1+\rho+\gamma) & 0 & 0 & 0 \\ 0 & 0 & \gamma & -\mu_1 & 0 & 0 \\ 0 & 0 & -\frac{\alpha_2 S_2^*}{N} & 0 & -\mu_2 & 0 \\ 0 & 0 & \frac{\alpha_2 S_2^*}{N} & 0 & 0 & -\mu_2 \end{pmatrix} \tag{17}$$

Such that the characteristic equation $(J_{E^*} - \lambda I) = 0$ becomes

$$\begin{pmatrix} \mu_1-\lambda_1 & 0 & 0 & 0 & 0 & \frac{\alpha_1 S_1^*}{N} \\ 0 & (\sigma+\mu_1)-\lambda_2 & 0 & 0 & 0 & -\frac{\alpha_1 S_1^*}{N} \\ 0 & \sigma & (\mu_1+\rho+\gamma)-\lambda_3 & 0 & 0 & 0 \\ 0 & 0 & -\gamma & \mu_1-\lambda_4 & 0 & 0 \\ 0 & 0 & \frac{\alpha_2 S_2^*}{N} & 0 & \mu_2-\lambda_5 & 0 \\ 0 & 0 & -\frac{\alpha_2 S_2^*}{N} & 0 & 0 & \mu_2-\lambda_6 \end{pmatrix} = 0 \tag{18}$$

simplifying (18) we have,

$$(\mu_1-\lambda_1)(\mu_1-\lambda_4)(\mu_1-\lambda_5)\begin{pmatrix} (\sigma+\mu_1)-\lambda_2 & 0 & -\frac{\alpha_1 S_1^*}{N} \\ \sigma & (\mu_1+\rho+\gamma)-\lambda_3 & 0 \\ 0 & -\frac{\alpha_2 S_2^*}{N} & \mu_1-\lambda_6 \end{pmatrix} = 0 \tag{19}$$

$$(\mu_1-\lambda_1)(\mu_1-\lambda_4)(\mu_1-\lambda_5)((\sigma+\mu_1)-\lambda_2)((\mu_1+\rho+\gamma)-\lambda_3)(\mu_1-\lambda_6) = 0 \tag{20}$$

such that

$$\lambda_1 = \lambda_4 = \lambda_5 = \lambda_6 = -\mu_1,$$
$$\lambda_2 = -(\sigma + \mu_1),$$
$$\lambda_3 = -(\mu_1 + \rho + \gamma).$$

Since all the eigenvalues are negative we have confirmed that the DFE locally, asymptotically stable.

2. 5. Global stability of disease free equilibrium

To ascertain the global stability of the disease free equilibrium (DFE) we employ the technique in Hove-Musekwa *et al*. [18]. Consider the infected compartments (2), (3) and (5) of the model equations. From (16) let F_i be the rate of appearance of new infections in compartment i and $V_i = V_i^- - V_i^+$ be the difference between the rate of transfer of individual out of and into compartment i by all other means, we can write equation (2), (3) and (5) in terms of $F_i - V_i$ (rate of transfer in compartment i), taking the Jacobian of F_i and V_i as F and V respectively, we have:

$$\begin{pmatrix} E'(t) \\ I_1'(t) \\ I_2'(t) \end{pmatrix} = (F - V)\begin{pmatrix} E \\ I_1 \\ I_2 \end{pmatrix} - \begin{pmatrix} \alpha_1\left(\frac{\beta_1\mu_2}{\beta_1\mu_2 + \beta_2\mu_1} - \frac{S_1}{N}\right) \\ 0 \\ \alpha_2\left(\frac{\beta_2\mu_1}{\beta_1\mu_2 + \beta_2\mu_1} - \frac{S_2}{N}\right) \end{pmatrix} \tag{21}$$

given that matrices F and V are defined by (11). Even so, because $S_1 \le \frac{\beta_1}{\mu_1}$, we have $\frac{S_1}{N} \le \frac{\beta_1}{\mu_1}$ for all $t \le 0$ *in* D. Similarly, we also have $\frac{S_2}{N} \le \frac{\beta_2}{\mu_2}$. Thus

$$\begin{pmatrix} E'(t) \\ I_1'(t) \\ I_2'(t) \end{pmatrix} < (F - V)\begin{pmatrix} E \\ I_1 \\ I_2 \end{pmatrix}. \tag{22}$$

Since the matrix F – V is a Hurwitz matrix, it follows that (22) is stable for $R_0 < 1$, following Lemma 1 of Driessche & Watmough [17]. Hence $(E, I_1, I_2) \to (0,0,0)$ as $t \to \infty$. By comparison theorem Martynyuk *et al.*, [16] we have that $(E, I_1, I_2) \to (0,0,0)$. Thus $S_1 \to \frac{\beta_1}{\mu_1}$ and $S_2 \le \frac{\beta_2}{\mu_2}$ at DFE whenever $E = I_1 = I_2 = 0$. Thus, $(E, I_1, I_2) \to E^*$ as $t \to \infty$, for $R_0 < 1$, so E^* is globally asymptotically stable.

2. 6. Endemic Equilibrium of the model

This is an equilibrium state where at least one of the infected compartments of the model is non-zero. If we represent the endemic equilibrium (EE) by $E^{**} = (S_1^{**}, E^{**}, I_1^{**}, R^{**}, S_2^{**}, I_2^{**})$, solving equations (1) to (6) simultaneously, we have that

$$S_1^{**} = \frac{\beta_1 N_H}{\alpha_1 I_2^{**} + \mu_1 N_H} \tag{23}$$

$$E^{**} = \frac{\alpha_1 \beta_1 I_2^{**}}{(\alpha_1 I_2^{**} + \mu_1 N)(\sigma + \mu_1)} \tag{24}$$

$$I_1^{**} = \frac{\sigma \alpha_1 \beta_1 I_2^{**}}{(\mu_1 + \rho + \gamma)(\alpha_1 I_2^{**} + \mu_1 N)(\sigma + \mu_1)} \tag{25}$$

$$R^{**} = \frac{\sigma \alpha_1 \beta_1 I_2^{**}}{(\mu_1 + \rho + \gamma)(\alpha_1 I_2^{**} + \mu_1 N)(\sigma + \mu_1)} \tag{26}$$

$$S_2^{**} = \frac{\beta_2 N(\mu_1 + \rho + \gamma)(\alpha_1 I_2^{**} + \mu_1 N)(\sigma + \mu_1)}{\alpha_2 \sigma \alpha_1 \beta_1 I_2^{**} + \mu_2 N(\mu_1 + \rho + \gamma)(\alpha_1 I_2^{**} + \mu_1 N)(\sigma + \mu_1)} \tag{27}$$

$$I_2^{**} = \frac{\alpha_2 \beta_2 \beta_1 \sigma \alpha_1 - N\mu_2^2(\mu_1+\rho+\gamma)(\mu_1 N\sigma+\mu_1^2 N)}{\mu_2[\alpha_1 \alpha_2 \beta_1 \sigma + N\mu_2(\mu_1+\rho+\gamma)(\sigma\alpha_1+\alpha_1\mu_1)]} \tag{28}$$

Therefore the endemic equilibrium (EE) of the model in terms of the infected sandfly I_2 can be obtained explicit by substituting (28) into, (23), (24), (25), (26), and (28).

On the other hand, the matrix containing all partial derivative of the model (1) to (6) evaluated at the endemic equilibrium is given by

$$J_{E^{**}} = \begin{pmatrix} -\left(\mu + \frac{\alpha_1 I_2^{**}}{N}\right) & 0 & 0 & 0 & 0 & -\frac{\alpha_1 S_1^{**}}{N} \\ \frac{\alpha_1 I_2^{**}}{N} & -(\sigma + \mu_1) & 0 & 0 & 0 & \frac{\alpha_1 S_1^{**}}{N} \\ 0 & \sigma & -(\mu_1 + \rho + \gamma) & 0 & 0 & 0 \\ 0 & 0 & \gamma & -\mu_1 & 0 & 0 \\ 0 & 0 & -\frac{\alpha_2 S_2^{**}}{N} & 0 & -\left(\frac{\alpha_2 I_1^{**}}{N} + \mu_2\right) & 0 \\ 0 & 0 & \frac{\alpha_2 S_2^{**}}{N} & 0 & \frac{\alpha_2 I_1^{**}}{N} & -\mu_2 \end{pmatrix} \tag{29}$$

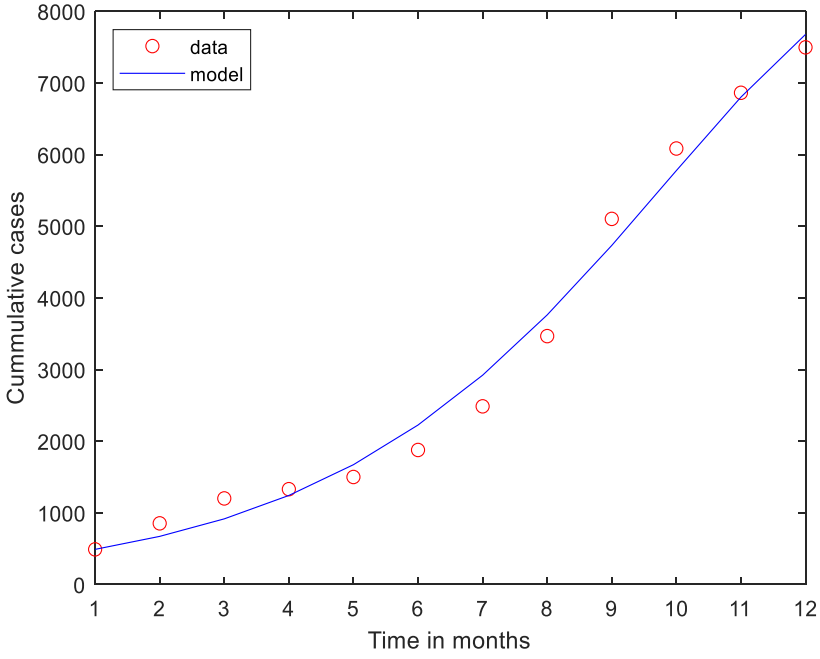

Figure 2. Fitting of the monthly cases of *Visceral leishmaniasis* in South Sudan from Jan to December 2011. Data is from [22].

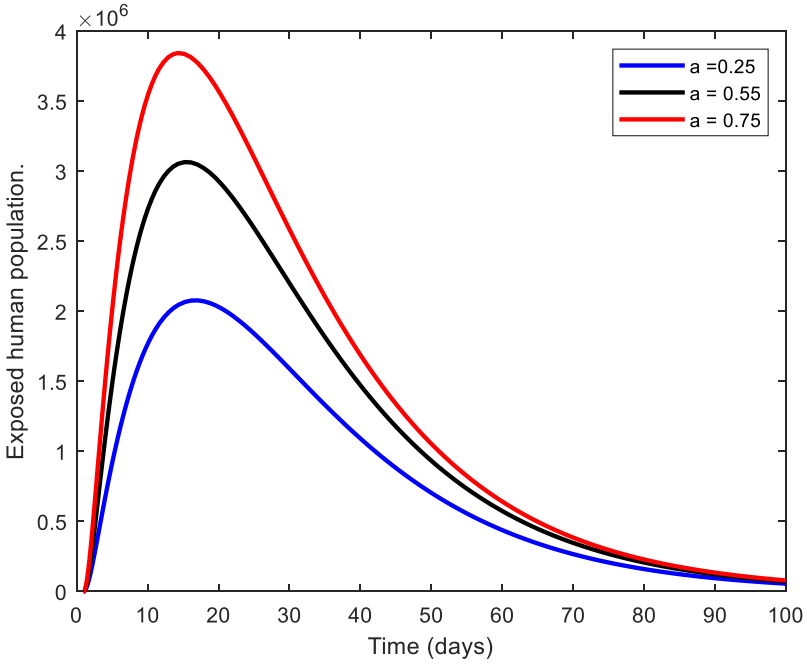

Figure 3. Simulation of exposed population with various values of biting rate of sandflies (a). The figure shows the effect of low biting rate of sandflies $(a = 0.25)$, average biting rate $(a = 0.55)$ and high biting rate $(a = 0.75)$. Other parameters are as listed in Table 1

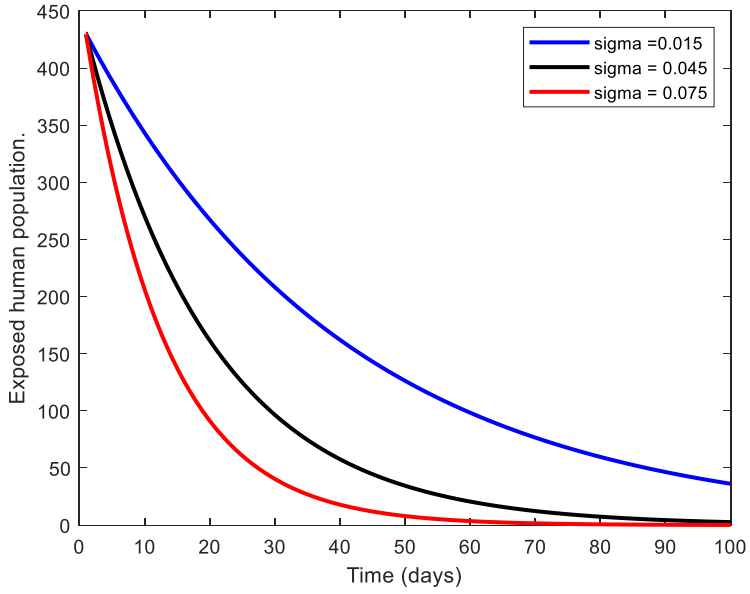

Figure 4. Simulation of the exposed population with various values of sigma. The figure displays the effect of low (sigma = 0.015), average (sigma = 0.045) and high (sigma = 0.075) detection rate of new cases on the exposed population.

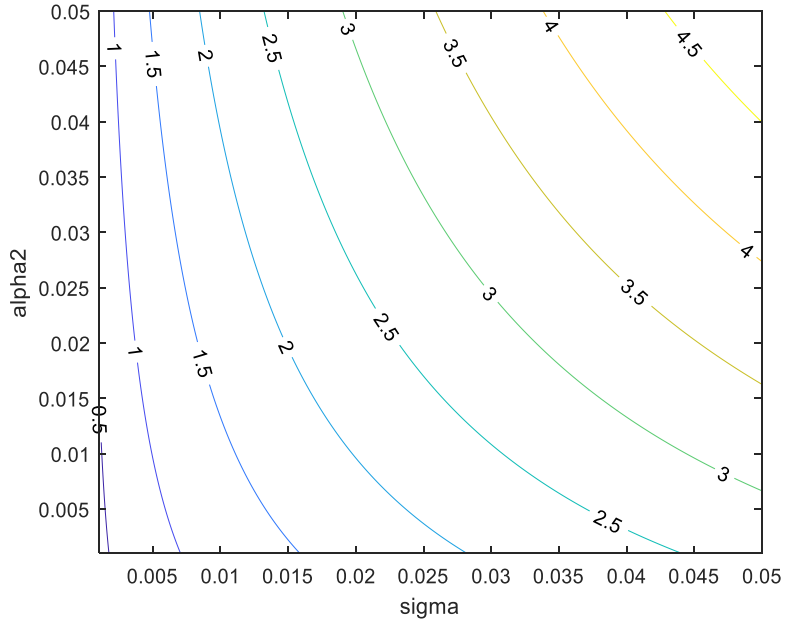

Figure 5. 2D contour plot of the basic reproductive number R_0 as a function of sandfly to human infection rate α_1, and recovery rate σ, parameter values are as listed in Table 1. The figure shows the values of α_1 and σ at which $R_0 < 1$, $R_0 = 1$ and $R_0 > 1$. The parameter values are as listed in Table 1.

2. 7. Model parameterization

Models that cannot relate well to live data are assumed to be a mere academic exercise that may not be much of help in solving real life problem that is modelled. Linking our models to data is necessary, for it helps us not only to gain more confidence in the model that we have created, but also to obtain realistic estimates of the parameters. The parameter *values* must be determined before we can think about the model simulation, this process of determining the values of the parameters of a model is called model *parameterization*. Parameter values can sometimes be found by performing specific experiments or by literature searches. However, as is most often, our model parameter values were found by *calibration*, where parameters are estimated by fitting the model to data. The data used here is the monthly cases of visceral leishmaniasis in South Sudan from January to December 2014 [22].

We opted for the least–square approach as against interpolation since the data may contain errors and capturing every little change in them may be impractical. In the least-squares approach, we assume that the time coordinates of the data are exact, but their *y*-coordinates may be distorted. We fit the solution curve through the data (see Figure 3) so that the sum of the squares of the vertical distances from the data points to the point on the curve is as small as possible.

In particular we fitted the reported cases $E(t)$ to the monthly data $(t_i, Y_i),...(t_n, Y_n)$ with sum of squares error $SSE = \sum_{i=1}^{n} (Y_i - E(t_i))^2$ which is a function of the parameters of the model is then minimized. This minimization is implemented using the matlab function *fminsearch,* the best fit parameter values from this iterative procedure is then used for model simulation.

2. 8. Model simulation

We use the values of the parameters that best fit our model to data alongside other parameter values Figure 2 to determine the effectiveness of various control measure of *Visceral leishmaniasis.*

3. CONCLUSIONS

We represented the transmission dynamics of Visceral Leishmaniasis in human and sandfly populations using a set of six ordinary differential equations. Our model was parameterized using the monthly cases of LV in South Sudan from January to December 2011 [22]. We explored the qualitative features of the model in other to assess some control strategies. The basic reproductive number of the model was obtained using the next generation approach. The local stability of the disease free equilibrium of the model was shown to be stable using the eigenvalues of the Jacobian matrix evaluated at the same equilibrium, whereas the global stability was shown to be stable using the standard comparison theory. Our result reveals that the number of exposed populations will be reduced if the biting rate of sandfly is kept at the barest minimum Figure 3. Also the value of the detection rate that will further reduce the burden of new cases is depicted in Figure 4. Finally, the values of α_1 and σ at which the basic

reproductive number will be less, equal, or greater than unity is depicted in Figure 5. This model can be improved by incorporating the reservoir population and other control parameters.

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