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Modelling and Analysis of Trypanosomiasis Transmission Mechanism

Domitila Wayua Muia[1]
Department of Mathematics and Actuarial Science,
The Catholic University of Eastern Africa,
Box 62157-00200, Nairobi-Kenya.

Shaibu Osman
Department of Mathematics and Actuarial Science,
The Catholic University of Eastern Africa,
Box 62157-00200, Nairobi-Kenya.

Mary Wainaina
Department of Mathematics and Actuarial Science,
The Catholic University of Eastern Africa,
Box 62157-00200, Nairobi-Kenya.

Abstract

Trypanosomiasis commonly known as sleeping sickness is a parasitic vector-borne disease that is mostly found in Sub-Saharan Africa. The infection can be categorised into two forms, namely; Human African Trypanosomiasis (HAT) and African Animal Trypanosomiasis (AAT). In this paper, an epidemic model is developed to give an account of the transmission mechanism of the disease in animal population. The basic reproductive rate of the disease was determined and analysed. The disease free-equilibrium of the Trypanosomiasis model was examined for local stability and its associated reproductive rate. The disease free equilibrium of the trypanosomiasis model was found to be locally asymptotically stable whenever the reproductive number was less than unity. The contribution of each parameter to the basic reproductive rate were determined. It was found that, an increase both the animal and vector recruitment rates would increase the basic reproduction number. We performed numerical simulations of the system of equations of the model. We found

[1]Corresponding author e-mail: tilawmuia@gmail.com

that there have been an increase in the populations of both the infectious animals and vectors.

AMS subject classification:
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1. Introduction

Trypanosomiasis commonly known as sleeping sickness is a parasitic vector-borne disease found in sub-Saharan Africa. There are two types of the disease, Human African Trypanosomiasis (HAT) and African Animal Trypanosomiasis (AAT). HAT is transmitted to human beings after a bite of a tsetse fly infected with the protozoa Trypanosomiasis brucei (T.b) or Rhodesience in East and Southern Africa and T.b gambiense in West and Central Africa. AAT is spread by tsetse flies infected with a variety of trypanosomes which include T.vivax, T. congolense, T.b brucei and T. simiae. Most of the tsetse transmission is cyclic and begins when the fly feeds on the blood from an animal infected with the trypanosome. The trypanosome then loses its surface coat, and multiplies in the fly and become infective after it reacquires a surface coat [3].

In Kenya, tsetse fly occupies 138 000 square kilometers. This is approximately 23 percent of the country's landmark area. That is 38 out of 47 counties with a total human population of about 11m are at risk of infection. Further according to KENTTEC, tsetse fly population is expected to grow once the rains start thus the spread of the disease (Daily Nation Mon, 30th Jan 2017).

A study conducted by authors in [3] on the parasite that causes human African Trypanosomiasis or sleeping sickness revealed that keeping approximately eighty two percent of cattle on insecticide treated environment can help reduced the problem of Trypanosomiasis among cattle's. Their study area was Tororo, a district in South Eastern Uganda where animal and human populations are approximately 40,000 and 500000 respectively. Their study focused on Trypanosomiasis in cattle and humans only. This study does not give a true reflection of the disease. Trypanosomiasis is referred to as zoonotic disease. It affects both animals and humans in general. Therefore, limiting the study of Trypanosomiasis on only cattle would not be realistic. This study in tends to improve on existing work on the fight against the disease in the entire East African belt.

According to [7], accurate foci distribution is based on the reported cases, but many infected patients die before diagnosis thus such cases are not captured. Report of exported cases is also a challenge. WHO calls for active surveillance systems that would make available accurate information in the active Atlas of HAT. Further according to WHO, it is necessary to stimulate interaction between the health workers and vet nary scientists. Mahamat et al., (2017) modeled HAT in southern Chad, his study revealed that adding tsetse control to the 'screen and treat' strategy had a mark able impact on the transmission of sleeping sickness. This was in agreement with similar findings from Guinea where

tsetse control also resulted in a significant decrease of the incidence of HAT. Vector-borne disease is one big challenge that confronts the present and future human well being. They affect about ten million people especially those living in poor conditions. They are responsible for over 10 percent of human deaths and cause impoverishment. Most developed countries are not spared due to climatic changes that have modified the spatial distribution of vectors and pathogens.

[12] evaluated the long-term effectiveness of sleeping sickness control measure in Guinea. The control measure evaluated included vector control combined with screen and treat strategy in order to assess the possibility of WHO attaining elimination of Trypanosomiasis by 2020 as one of its objectives. The findings were that interruption of the control measure would result in the disease prevalence beyond 2020. They further suggested that intervention measures must extend even after 2020 to avoid flare-ups of the disease.

Authors in [2] carried out a study on African Human Trypanosomiasis infections. Their study revealed humans play a vital role in the spread and transmission of the disease by serving as a reservoir. Moreover, it was revealed that the Democratic republic of Congo is the worse affected area and approximately seventy five percent of cases are declared.

Rock et al., (2015) quantitatively evaluated the strategy to eliminate HAT in the Democratic Republic of Congo. Their findings showed that Active screening had reduced new human infections by 52 to 53 percent over the 15 year period (1998–2012). Further projections indicated that the WHO elimination goal in Congo may be met not earlier than 2059-2092 under the then current intervention. They suggested active detection, a rise in the screening level and widespread vector control.

2. Model description and formulation

The total population of animals is divided into three groups. This is made up of susceptible animals, (S_p). These are animals who are likely to get the disease or get the infection. Infectious animals, (I_p); these are those who are currently infected with trypanosomiasis disease. Recovered animals,(R_p). These are animals that had the disease and have now recovered from trypanosomiasis. The vector population is sub-divided into two groups. Susceptible vector, (S_v) and Infectious vector, (I_v). Susceptible animals are recruited at a rate of (Λ_p), they may die as a result of natural causes at a rate of (μ_p). Whereβ is the rate at which trypanosomiasis is transmitted to animals. Infected animals either die naturally at a rate of (μ_p) or from the infection at the rate of (σ_p). Recovered animals may lose immunity and join the susceptible class at a rate of γ they also may die naturally at a rate of (μ_p). It is assumed that the recovery of the infected animals is not immediate. Susceptible flies are recruited at a rate of (Λ_v), they may naturally die at a rate of (μ_v) or from contact with Insecticide treated animals at a rate of (σ_p). They become infected after a blood meal from infective individuals. Infectious tsetse flies may die naturally, at a rate of (μ_v) or from contact with insecticide treated animals at a rate of (σ_v). Below is the Model Representation.

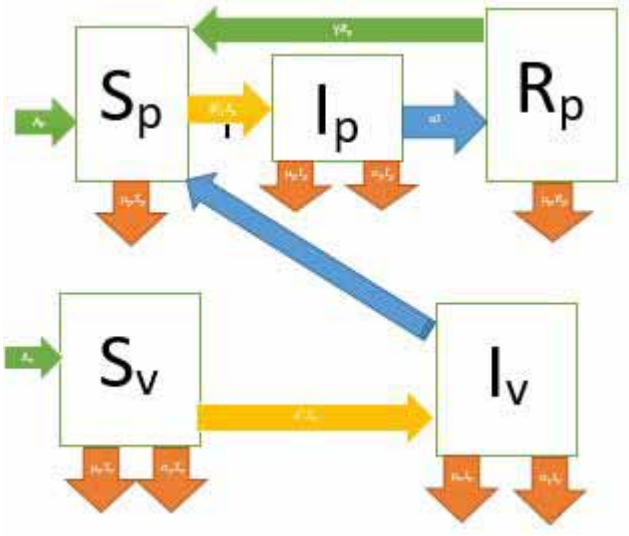

Figure 1: Model flow chart showing the compartments.

The following system of equations are obtained from the Trypanosomiasis model in figure 1;

$$\left.\begin{aligned}
\frac{dS_p}{dt} &= \Lambda_p - \beta S_p I_p + \gamma R_p - \mu_p S_p \\
\frac{dI_p}{dt} &= \beta S_p I_p - \left(\mu_p + \sigma_p + \alpha\right) I_p \\
\frac{dR_p}{dt} &= \alpha I_p - \left(\mu_p + \gamma\right) R_p \\
\frac{dS_v}{dt} &= \Lambda_v - \lambda S_v I_v - \mu_v S_v \\
\frac{dI_v}{dt} &= \lambda S_v I_v - \left(\mu_v + \sigma_v\right) I_v
\end{aligned}\right\} \tag{2.1}$$

3. The Positivity and solution boundedness

The trypanosomiasis model is uniformly bounded in the subset $\psi \subset R_+^5$. By considering the total human population of the model in figure 1;

$$N_p(t) = S_p(t) + I_p(t) + R_p(t) \tag{3.1}$$

The total population given by the sum of all the susceptible, infectious and recovered humans;

$$\frac{dN_p}{dt} = \frac{dS_p}{dt} + \frac{dI_p}{dt} + \frac{dR_p}{dt} \tag{3.2}$$

$$\frac{dN_p}{dt} = \Lambda_p - \beta S_p I_p + \gamma R - \mu_p S_p + \beta S_p I_p - (\mu_p + \sigma_p + \alpha) I_p + \alpha I_p - (\mu_p + \gamma) R_p$$

By simplifications;

$$\frac{dN_p}{dt} = \Lambda_p - \mu_p S_p - \mu_p I_p - \sigma_p I_p - \mu_p R_p$$

In the absence of mortality rate due to trypanosomiasis infections;

$$\frac{dN_p}{dt} = \Lambda_p - \mu_p S_p \tag{3.3}$$

$$\frac{dN_p}{dt} = \Lambda_p - \mu_p N_p$$

$$\frac{dN_p}{\Lambda_p - \mu_p N_p} = dt$$

$$\frac{dN_p}{\Lambda_p - \mu_p N_p} \leq dt$$

$$\int \frac{dN_p}{\Lambda_p - \mu_p N_p} \leq \int dt$$

$$\frac{\ln(\Lambda_p - \mu_p N_p)}{-\mu_p} \leq t + A$$

where A is a constant.

$$\ln(\Lambda_p - \mu_p N_p) \leq -\mu_p t - \mu_p t$$

$$\Lambda_p - \mu_p N_p \geq e^{-\mu_a t}.e^{-\mu_a A},$$

let $C = e^{-\mu_a A}$

$$\Lambda_p - \mu_p N_p \geq C e^{-\mu_a t}$$

Applying the initial conditions,

$$N_p(0) = N_p(0)$$

$$\Lambda_p - \mu_p N_p(0) = C$$

$$\Lambda_p - \mu_p N_p \geq (\Lambda_p - \mu_p N_p(0)) e^{-\mu_a t}$$

$$N_p \leq \frac{\Lambda_p}{\mu_p} - \left(\frac{\Lambda_p - \mu_p N_p(0)}{\mu_p}\right) e^{-\mu_a t}$$

As $t \to \infty$,the population$N_p \to \dfrac{\Lambda_p}{\mu_p}$

$$0 = N_p \leq \frac{\Lambda_p}{\mu_p}$$

and

$$N_p(t) \leq \frac{\Lambda_p}{\mu_p}$$

If $N_p(0) \leq \frac{\Lambda_p}{\mu_p}$ then $N_p \leq \frac{\Lambda_p}{\mu_p}$ Hence;

$$\psi_p = \left\{ \left(S_p, I_p, R_p\right) \in R_+^3 : S_p + I_p + R_p \leq \frac{\Lambda_a}{\mu_a} \right\} \tag{3.4}$$

Also, considering the vector population of the trypanosomiasis model in figure 1;

$$\left.\begin{aligned} \frac{dS_v}{dt} &= \Lambda_v - \lambda S_v I_v - \mu_v S_v \\ \frac{dI_v}{dt} &= \lambda S_v I_v - (\mu_v + \sigma_v) I_v \end{aligned}\right\} \tag{3.5}$$

$$N_v(t) = S_v(t) + I_v(t) \tag{3.6}$$

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dI_v}{dt}$$

$$\frac{dN_v}{dt} = \Lambda_v - \lambda S_v I_v - \mu_v S_v + \lambda S_v I_v - (\mu_v + \sigma_v) I_v$$

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v S_v - (\mu_v + \sigma_v) I_v$$

In the absence of infections; there are no recovery;

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v S_v \tag{3.7}$$

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v$$

$$\frac{dN_v}{\Lambda_v - \mu_v N_v} = dt$$

$$\int \frac{dN_v}{\Lambda_v - \mu_v N_v} \leq \int dt$$

$$\frac{In(\Lambda_v - \mu_v N_v)}{-\mu_v} \leq t + A$$

where A is a constant.

$$In(\Lambda_v - \mu_v N_v) \geq -\mu_v t - \mu_v A$$

$$\Lambda_v - \mu_v N_v \geq e^{-\mu_v t}.e^{-\mu_v A}$$

where $e^{-\mu_v A} = C$

$$\Lambda_v - \mu_v N_v \geq Ce^{-\mu_v t}$$

$$N_v(0) = N_v(0)$$

$$\Lambda_v - \mu_v N_v = C$$

$$\Lambda_v - \mu_v N_v \geq \Lambda_v - \mu_v N_v(0)e^{-\mu_v t}$$

$$N_v \leq \frac{\Lambda_v}{\mu_v} - \left(\frac{\Lambda_v - \mu_v N_v(0)}{\mu_v}\right) e^{-\mu_v t}$$

As $t \to \infty$,the population size $N_v \to \dfrac{\Lambda_v}{\mu_v}$

$$0 \leq N_v \leq \frac{\Lambda_v}{\mu_v}$$

and

$$N_v \leq \frac{\Lambda_v}{\mu_v}$$

Hence;

$$\psi_v = \left\{(S_v, I_v) \in R_+^2 : S_v + I_v \leq \frac{\Lambda_v}{\mu_v}\right\} \tag{3.8}$$

The trypanosomiasis model in figure 1 is bounded in;

$$\Delta = \psi_v + \psi_p. \tag{3.9}$$

Therefore, the trypanosomiasis model in the region is well-posed.

The solution of the system remains positive at any given point in time, if the initial values of all the model variables are positive. The solution of the system in the model continue to remain positive at any point in time if the initial values of all the variables are positive [11, 8].

Theorem 3.1. Considering

$$\Omega = \left\{\left(S_p(t), I_p(t), R_p(t)\right) \in R_+^3 : S_p(0) > 0, I_p(0) > 0, R_p(0) > 0\right\},$$

then the solution of $\left\{S_p(t), I_p(t), R_p(t)\right\}$ are positive for $t \geq 0$.

4. Disease free equilibrium

The disease free equilibrium is obtained by setting the system of differential equations to zero. At disease free equilibrium, there are no infections and recovery.
$I_p = 0$ and $R_p = 0$.

$$\frac{dS_p}{dt} = \Lambda_p - \beta S_p I_p + \gamma R_p - \mu_p S_p = 0 \tag{4.1}$$

$$\Lambda_p - \beta S_p I_p + \gamma R_p - \mu_p S_p = 0$$

but $I_p = 0$ and $R_p = 0$.

$$\Lambda_p - \mu_p S_p = 0$$

$$S_p^* = \frac{\Lambda_p}{\mu_p} \tag{4.2}$$

Also, considering the vector population in the flow diagram in figure 1;

$$\frac{dS_v}{dt} = \Lambda_v - \lambda S_v I_v - \mu_v S_v = 0 \tag{4.3}$$

$$\Lambda_v - \lambda S_v I_v - \mu_v S_v = 0$$

but $I_v = 0$

$$\Lambda_v - \mu_v S_v = 0$$

$$S_v^* = \frac{\Lambda_v}{\mu_v} \tag{4.4}$$

The disease free equilibrium of the Trypanosomiasis model in figure 1 is given by;

$$\left(\frac{\Lambda_a}{\mu_a}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0\right) \tag{4.5}$$

4.1. Stability of the disease free equilibrium of the Trypanosomiasis model

The disease free equilibrium of the Trypanosomiasis model in figure 1 was obtained as;

$$\left(\frac{\Lambda_p}{\mu_p}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0\right)$$

Computing the Jacobian matrix of the system of equations of the model;

$$J = \begin{pmatrix} (-\beta I_p - \mu_p) & -\beta S_p & \gamma & 0 & 0 \\ \beta S_p & \beta I_p - (\mu_p + \sigma_p + \alpha) & 0 & 0 & 0 \\ 0 & \alpha & -(\mu_p + \gamma) & 0 & 0 \\ 0 & 0 & 0 & -\lambda S_v - \mu_v & -\lambda S_v \\ 0 & 0 & 0 & -\lambda I_v & -\lambda S_v - (\mu_v + \sigma_v) \end{pmatrix}$$

The Jacobian of the system at disease free equilibrium;

$$J\left(\frac{\Lambda_a}{\mu_a},0,0,\frac{\Lambda_v}{\mu_v},0\right)=\begin{pmatrix} -\mu_p & -\frac{\beta\Lambda_p}{\mu_p} & \gamma & 0 & 0 \\ 0 & -(\mu_p+\sigma_p+\alpha) & 0 & 0 & 0 \\ 0 & \alpha & -(\mu_p+\gamma) & 0 & 0 \\ 0 & 0 & 0 & -\mu_v & -\frac{\lambda\Lambda_v}{\mu_v} \\ 0 & 0 & 0 & 0 & -\lambda\Lambda_v-(\mu_v+\sigma_v) \end{pmatrix}$$

Eigenvalues of the Jacobian matrix at disease free equilibrium;

$$\begin{vmatrix} -\mu_p-A & -\frac{\beta\Lambda_p}{\mu_p} & \gamma & 0 & 0 \\ 0 & -(\mu_p+\sigma_p+\alpha)-A & 0 & 0 & 0 \\ 0 & \alpha & -(\mu_p+\gamma)-A & 0 & 0 \\ 0 & 0 & 0 & -\mu_v-A & -\frac{\lambda\Lambda_v}{\mu_v} \\ 0 & 0 & 0 & 0 & \frac{\lambda\Lambda_v}{\mu_v}-(\mu_v+\sigma_v)-A \end{vmatrix}=0$$

The eigenvalues are as follows;

$$A_1=-\mu_p, A_2=-\left((\mu_v+\sigma_v)-\frac{\lambda\Lambda_v}{\mu_v}\right),$$

$$A_3=-\mu_p, A_4=-(\mu_p+\sigma_p+\alpha, A_5=-(\mu_p+\gamma)$$

Since all eigenvalues are negative, then the disease free equilibrium is locally asymptotically stable.

5. Basic reproductive number

This is the number of secondary infections that an infected person can cause in a completely susceptible population. This is a threshold value that governs the spread of a disease population. Using the approach in [10], the basic reproduction number of the trypanosomiasis model is computed. By considering the infective compartments of the system of equations from the model in figure 1;

$$\left.\begin{aligned} \frac{dI_p}{dt} &= \beta S_p I_p-\left(\mu_p+\sigma_p+\alpha\right)I_p \\ \frac{dI_v}{dt} &= \lambda S_v I_v-(\mu_v+\sigma_v)I_p \end{aligned}\right\} \tag{5.1}$$

Applying the concept of the next generation matrix;

Let $f=\begin{pmatrix}\beta S_p I_p\\ \lambda S_v I_v\end{pmatrix}$ and $v=\begin{pmatrix}\left(\mu_p+\sigma_p+\alpha\right)I_p\\ (\mu_v+\sigma_v)I_p\end{pmatrix}$

By obtaining the Jacobian Matrix of f and v with respect to I_p and $I_v$at disease free equilibrium,

$$F = \begin{pmatrix} \beta S_p & 0 \\ 0 & \lambda S_v \end{pmatrix} \text{ and } V = \begin{pmatrix} (\mu_p + \sigma_p + \alpha) & 0 \\ 0 & (\mu_v + \sigma_v) \end{pmatrix}$$

The Jacobian matrix of F and V at disease free equilibrium is given by;

$$F = \begin{pmatrix} \frac{\beta \Lambda_p}{\mu_p} & 0 \\ 0 & \frac{\lambda \Lambda_v}{\mu_v} \end{pmatrix}, V = \begin{pmatrix} (\mu_p + \sigma_p + \alpha) & 0 \\ 0 & (\mu_v + \sigma_v) \end{pmatrix}$$

$$V^{-1} = \frac{1}{(\mu_p + \sigma_p + \alpha)(\mu_v + \sigma_v)} \begin{pmatrix} (\mu_v + \sigma_v) & 0 \\ 0 & (\mu_p + \sigma_p + \alpha) \end{pmatrix} \tag{5.2}$$

Now finding the product of F and V^{-1}

$$FV^{-1} = \begin{pmatrix} \frac{\beta \Lambda_p}{\mu_p} & 0 \\ 0 & \frac{\lambda \Lambda_v}{\mu_v} \end{pmatrix} \begin{pmatrix} \frac{(\mu_v + \sigma_v)}{(\mu_p + \sigma_p + \alpha)(\mu_v + \sigma_v)} & 0 \\ 0 & \frac{(\mu_p + \sigma_p + \alpha)}{(\mu_p + \sigma_p + \alpha)(\mu_v + \sigma_v)} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{\beta \Lambda_p}{\mu_p (\mu_p + \sigma_p + \alpha)} & 0 \\ 0 & \frac{\lambda \Lambda_v}{\mu_v (\mu_v + \sigma_v)} \end{pmatrix}$$

Now, by obtaining the eigenvalues of FV^{-1}

$$\begin{vmatrix} \frac{\beta \Lambda_p}{\mu_p (\mu_p + \sigma_p + \alpha)} - A & 0 \\ 0 & \frac{\lambda \Lambda_v}{\mu_v (\mu_v + \sigma_v)} - A \end{vmatrix} = 0 \tag{5.3}$$

Where A is the eigenvalue of FV^{-1}

$$\left(\frac{\beta \Lambda_p}{\mu_p (\mu_p + \sigma_p + \alpha)} - A \right) \left(\frac{\lambda \Lambda_v}{\mu_v (\mu_v + \sigma_v)} - A \right) = 0$$

$$\left(\frac{\beta \Lambda_p}{\mu_p (\mu_p + \sigma_p + \alpha)} - A \right) = 0$$

or

$$\left(\frac{\lambda\Lambda_v}{\mu_v\left(\mu_v+\sigma_v\right)}-A\right)=0$$

$$A_1=\frac{\beta\Lambda_p}{\mu_p\left(\mu_p+\sigma_p+\alpha\right)}$$

and

$$A_2=\frac{\lambda\Lambda_v}{\mu_v\left(\mu_v+\sigma_v\right)}$$

The basic reproduction number is the maximum of the spectral radial of $\left(FV^{-1}\right)$.

Therefore, the basic reproduction number is given by;

$$R_0=\left(\frac{\beta\Lambda_p}{\mu_p\left(\mu_p+\sigma_p+\alpha\right)}\right)+\left(\frac{\lambda\Lambda_v}{\mu_v\left(\mu_v+\sigma_v\right)}\right) \tag{5.4}$$

6. Endemic equilibrium

The endemic equilibrium point is the state at which the infection is present in the dynamical system. By equating the system if the differential equations to zero,

$$\frac{dI_p}{dt}=\beta S_pI_p-(\mu_p+\sigma_p+\alpha)I_p=0$$

$$\beta S_pI_p=(\mu_p+\sigma_p+\alpha)I_p$$

$$S_p^*=\frac{(\mu_p+\sigma_p+\alpha)}{\beta}$$

$$\frac{dR_p}{dt}=\alpha I_p-(\mu_p+\gamma)R_p=0$$

$$\alpha I_p=(\mu_p+\gamma)R_p$$

$$R_p^*=\frac{\alpha I_p^*}{(\mu_p+\gamma)}$$

$$\frac{dS_p}{dt}=\Lambda_p-\beta S_pI_p+\gamma R_p-\mu_pS_p=0$$

$$\beta S_pI_p=\Lambda_{p+}\gamma R_p-\mu_pS_p$$

$$I_p^*=\frac{\Lambda_{p+}\gamma R_p^*-\mu_pS_p^*}{\beta S_p^*}$$

Also the vector set of equations,

$$\frac{dS_v}{dt}=\Lambda_v-\lambda S_vI_v-\mu_v\sigma_v=0$$

$$\lambda S_v I_v = \Lambda_v - \mu_v \sigma_v$$
$$I_v^* = \frac{\Lambda_v - \mu_v \sigma_v}{\lambda S_v^*}$$
$$\frac{dI_v}{dt} = \lambda S_v I_v - (\mu_v + \sigma_v) I_v = 0$$
$$\lambda S_v I_v = (\mu_v + \sigma_v) I_v$$
$$S_v^* = \frac{(\mu_v + \sigma_v)}{\lambda}$$

Hence, the endemic equilibrium is given by;

$$\left(\frac{(\mu_p + \sigma_p + \alpha)}{\beta}, \frac{\Lambda_{p+} \gamma R_p^* - \mu_p S_p^*}{\beta S_p^*}, \frac{\alpha I_p^*}{(\mu_p + \gamma)}, \frac{(\mu_v + \sigma_v)}{\lambda}, \frac{\Lambda_v - \mu_v \sigma_v}{\lambda S_v^*} \right) \tag{6.1}$$

6.1. Stability of the endemic equilibrium

Theorem 6.1. If the basic reproduction number is greater than one, $(R_0 > 1)$, then the endemic equilibrium is globally asymptotically stable and unstable otherwise.

Proof. By using the concept of Lyapunov function defined by;

$$\left.\begin{aligned} L\left(S_p^*, I_p^*, R_p^*, S_v^*, I_v^*\right) = \left(S_p - S_p^* - S_p^* \ln \frac{S_p^*}{S_p}\right) + \left(I_p - I_p^* - I_p^* \ln \frac{I_p^*}{I_p}\right) + \\ \left(R_p - R_p^* - R_p^* \ln \frac{R_p^*}{R_p}\right) + \left(S_v - S_v^* - S_v^* \ln \frac{S_v^*}{S_v}\right) + \left(I_v - I_v^* - I_v^* \ln \frac{I_v^*}{I_v}\right) \end{aligned}\right\} \tag{6.2}$$

By computing the derivative of the L along the solutions of the system of equations directly;

$$\frac{dL}{dt} = \left(\frac{S_p - S_p^*}{S_p}\right) \frac{dS_p}{dt} + \left(\frac{I_p - I_p^*}{I_p}\right) \frac{dI_p}{dt} + \left(\frac{R_p - R_p^*}{R_p}\right) \frac{dR_p}{dt} + \left(\frac{S_v - S_v^*}{S_v}\right) \frac{dS_v}{dt} + \left(\frac{I_v - I_v^*}{I_v}\right) \frac{dI_v}{dt}$$

$$\left.\begin{aligned} \frac{dL}{dt} &= \left(\frac{S_p - S_p^*}{S_p}\right) \left[\Lambda_p - \beta S_p I_p + \gamma R - \mu_p S_p\right] \\ &+ \left(\frac{I_p - I_p^*}{I_p}\right) \left[\beta S_p I_p - (\mu_p + \sigma_p + \alpha) I_p\right] \\ &+ \left(\frac{R_p - R_p^*}{R_p}\right) \left[\alpha I_p - (\mu_p + \gamma) R_p\right] \\ &+ \left(\frac{S_v - S_v^*}{S_v}\right) \left[\Lambda_v - \lambda S_v I_v - \mu_v S_v\right] + \left(\frac{I_v - I_v^*}{I_v}\right) \left[\lambda S_v I_v - (\mu_v + \sigma_v) I_v\right] \end{aligned}\right\}$$

By expansion;

$$\left.\begin{aligned}\frac{dL}{dt} &= \Lambda_p - \beta S_p I_p + \gamma R_p - \mu_p S_p - \frac{\Lambda_p S_p^*}{S_p} + \beta S_p^* I_p - \frac{\gamma S_p^* R_p}{S_p} \\ &+\mu_p S_p^* + \beta S_p I_p - (\mu_p + \sigma_p + \alpha) I_p \\ &-\beta S_p I_p^* + (\mu_p + \sigma_p + \alpha) I_p^* + \alpha I_p - (\mu_p + \gamma) R_p \\ &-\frac{\alpha I_p R_P^*}{R_p} + (\mu_p + \gamma) R_p^* \\ &+\Lambda_v - \lambda S_v I_v - \mu_v S_v - \frac{\Lambda_v S_v^*}{S_v} + \lambda S_v^* I_v + \mu_v S_v^* \\ &+\lambda S_v I_v - (\mu_v + \sigma_v) I_v - \lambda S_v I_v^* + (\mu_v + \sigma_v) I_v^*\end{aligned}\right\}$$

From;

$$\frac{dL}{dt} = U - V, \tag{6.3}$$

where U are the positive terms and V are the negative terms;

$$\begin{aligned}U &= \Lambda_p + \gamma R_p - \mu_p S_p + \beta S_p^* I_p + \mu_p S_p^* + (\mu_p + \sigma_p + \alpha) I_p^* + \alpha I_p + (\mu_p + \gamma) R_p^* \\ &\quad + \Lambda_v + \lambda S_v^* I_v + \mu_v S_v^* + \lambda S_v I_v + (\mu_v + \sigma_v) I_v^* \\ V &= \mu_p S_p + \frac{\Lambda_p S_p^*}{S_p} + \frac{\gamma S_p^* R_p}{S_p} + (\mu_p + \sigma_p + \alpha) I_p + \beta S_p I_p^* + (\mu_p + \gamma) R_p + \frac{\alpha I_p R_P^*}{R_p} \\ &\quad + \lambda S_v I_v + \mu_v S_v + \frac{\Lambda_v S_v^*}{S_v} + (\mu_v + \sigma_v) I_v + \lambda S_v I_v^*\end{aligned}$$

If $U < V$, then $\frac{dL}{dt} \leq 0$. $\frac{dL}{dt} = 0$, if and only if $S_p = S_p^*$, $I_p = I_p^*$, $R_p = R_p^*$, $S_v = S_v^*$, and $I_v = I_v^*$.

The largest compact invariant set in

$$\left\{\left(S_p^*, I_p^*, R_p^*, S_v^*, I_v^*\right) \in \Omega : \frac{dL}{dt} = 0\right\} \tag{6.4}$$

is a singleton E^*, where E^* is the endemic equilibrium.

Therefore,the endemic equilibrium is globally asymptotically stable in the invariant Ωif $M < N$ as in [9, 10].

7. Sensitivity analysis

The contribution of parameter to the reproduction number, reveals the effectiveness of each of the parameter value to persistence of the disease in the environment with time. The objective of this project is to help reduce the spread of the Trypanosomiasis infection

or give an informed decision with respect to the spread or die out of the disease. The technique of parameter contribution usually referred to sensitivity analysis is performed to indicate the level of contribution of each of the parameters to the reproductive number. The level of contribution of each parameter of the model is usually evaluated by the relationship between each parameter and the reproductive number [8, 4, 5]. Contribution of a model variable C to the reproductive number, R_0, is given by;

$$S_C^{R_0} = \frac{\partial R_0}{\partial C} * \frac{C}{R_0} \tag{7.1}$$

For β

$$S_\beta^{R_0} = \frac{dR_0}{d\beta} * \frac{\beta}{R_0}$$

$$S_\beta^{R_0} = -\frac{\beta\Lambda_p\mu_v(\mu_v+\sigma_v)}{\mu_p(\mu_p+\sigma_p+\alpha)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For α

$$S_\alpha^{R_0} = \frac{dR_0}{d\alpha} * \frac{\alpha}{R_0}$$

$$S_\alpha^{R_0} = \frac{\beta\alpha\Lambda_p\mu_v(\mu_v+\sigma_v)}{\mu_p(\mu_p+\sigma_p+\alpha)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For α

$$S_\lambda^{R_0} = \frac{dR_0}{d\lambda} * \frac{\lambda}{R_0}$$

$$S_\lambda^{R_0} = -\frac{\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)}{\mu_v(\mu_v+\sigma_v)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For Λ_p

$$S_{\Lambda p}^{R_0} = \frac{dR_0}{d\Lambda_p} * \frac{\Lambda_p}{R_0};$$

$$S_{\Lambda p}^{R_0} = -\frac{\beta\Lambda_p\mu_v(\mu_v+\sigma_v)}{\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For σ_p

$$S_{\sigma_p}^{R_0} = \frac{dR_0}{d\sigma_p} * \frac{\sigma_p}{R_0};$$

$$S_{\sigma_p}^{R_0} = \frac{\beta\Lambda_p\mu_v\sigma_p(\mu_v+\sigma_v)}{\mu_p(\mu_p+\sigma_p+\alpha)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For Λ_v

$$S_{\Lambda_v}^{R_0} = \frac{dR_0}{d\Lambda_v} * \frac{\Lambda_v}{R_0};$$

$$S_{\Lambda_v}^{R_0} = -\frac{\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)}{\mu_v(\mu_v+\sigma_v)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For μ_v

$$S_{\mu_v}^{R_0} = \frac{dR_0}{d\mu_v} * \frac{\mu_v}{R_0};$$

$$S_{\mu_v}^{R_0} = \frac{\lambda\Lambda_v\mu_p(2\mu_v+\sigma_v)(\mu_p+\sigma_p+\alpha)}{(\mu_v+\sigma_v)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

For σ_v

$$S_{\sigma_v}^{R_0} = \frac{dR_0}{d\sigma_v} * \frac{\sigma_v}{R_0};$$

$$S_{\sigma_v}^{R_0} = \frac{\lambda\Lambda_v\sigma_v\mu_p(\mu_p+\sigma_p+\alpha)}{(\mu_v+\sigma_v)\left(\beta\Lambda_p\mu_v(\mu_v+\sigma_v)+\lambda\Lambda_v\mu_p(\mu_p+\sigma_p+\alpha)\right)}$$

Table 1: Sensitivity indices of the basic reproduction number.

Parameter	Sensitivity index $(-/+)$
β	$-$
Λ_p	$-$
μ_p	$+$
σ_p	$+$
α	$+$
μ_v	$+$
Λ_v	$-$
λ	$-$
σ_v	$+$

Table 1 shows the contribution of parameters to the basic reproduction number. A decrease in both the animal and vector recruitment rates would decrease the basic reproduction number. Since the reproduction number should always be less than unity, an increase in the value of α would increase the basic reproduction number.

8. Numerical simulations

Numerical simulations was performed on the Trypanosomiasis model parameters to see the dynamics of the population of susceptible, infectious and recovered animals and vector in the system. This is done to see the how the population of the susceptible, infectious and the recovered change with time. The numerical simulations was done using Runge-Kutta fourth order scheme [1, 6]. The following parameter values were taken from existing published data and others assumed for the numerical simulations; $\beta = 0.005$, $\Lambda_v = 1200$, $\Lambda_p = 25$, $\lambda = 0.48$, $\mu_p = 0.0045$, $\mu_v = 0.05$, $\sigma_p = 0.008$, $\sigma_v = 0.002$, $\gamma = 0.01$, $\alpha = 0.0003$.

8.1. Susceptible animal and vector populations

Figure 2, 3 and 4 shows the population change in the susceptible compartments with time. The population of the susceptible animals and vectors decreases with time. The recruitment rates of both the susceptible animals and vectors increases at a constant rate but the population of both the susceptible animals and vectors decreases. This could be attributed to the increase in the population of the infectious animals and vectors. An increase in the population of the infectious animals and vectors can cause a decrease in the population of both the susceptible animals and vectors. This can only be possible as the population of the recovered animals decrease with time in the population as indicated in figure 7.

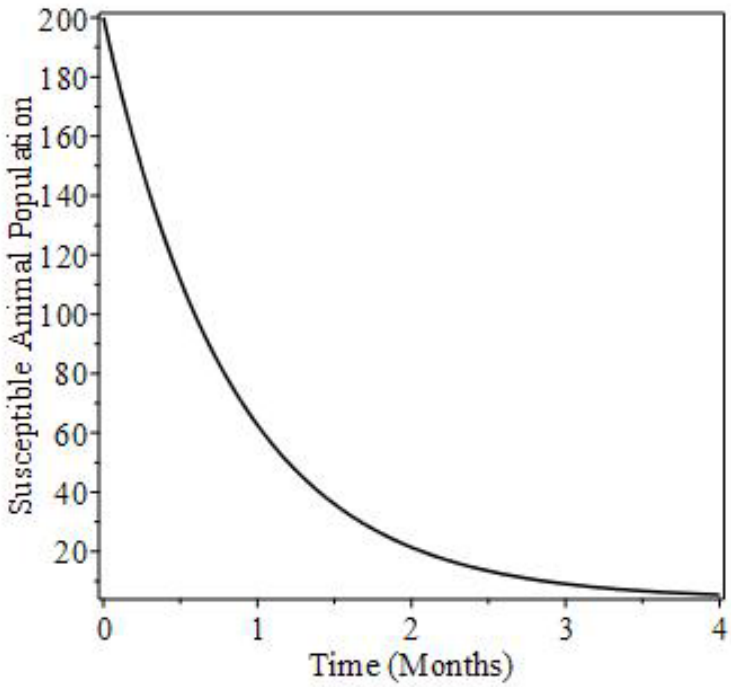

Figure 2: Susceptible animal populations.

Figure 4, indicates a comparison between the susceptible animal and vector populations in epidemics. The population of the susceptible vectors and susceptible animals varies. This is an indication that animals are more susceptible to the Trypanosomiasis infections to animals.

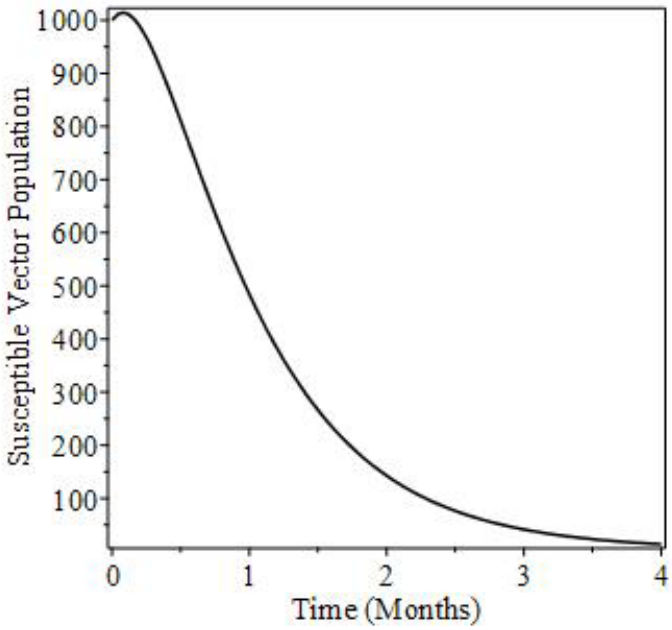

Figure 3: Susceptible vector population.

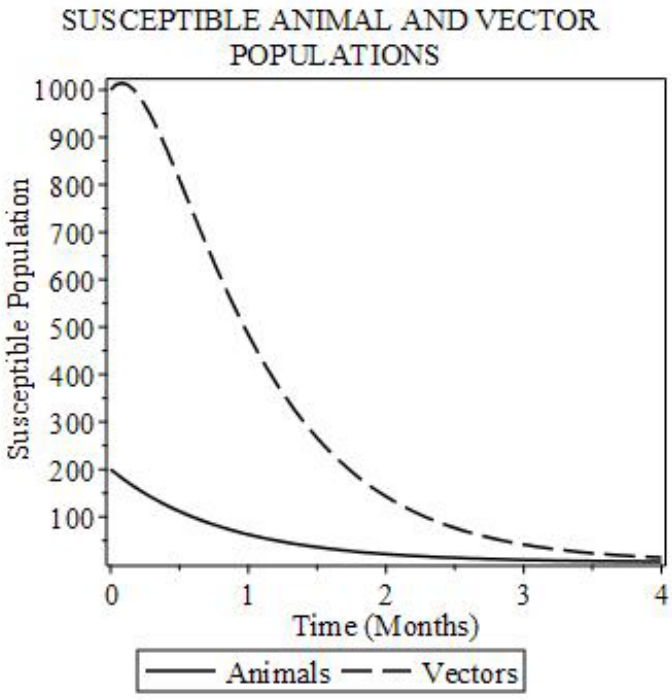

Figure 4: Comparison of susceptible animal and vector populations.

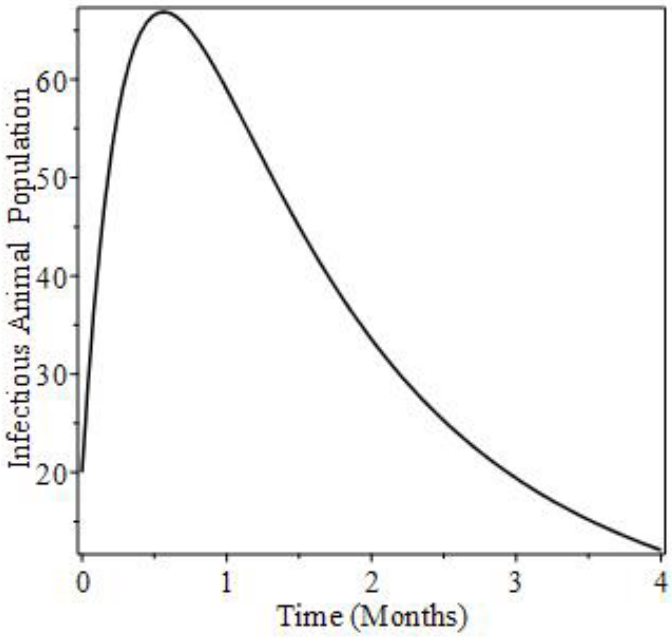

Figure 5: Infectious animal population.

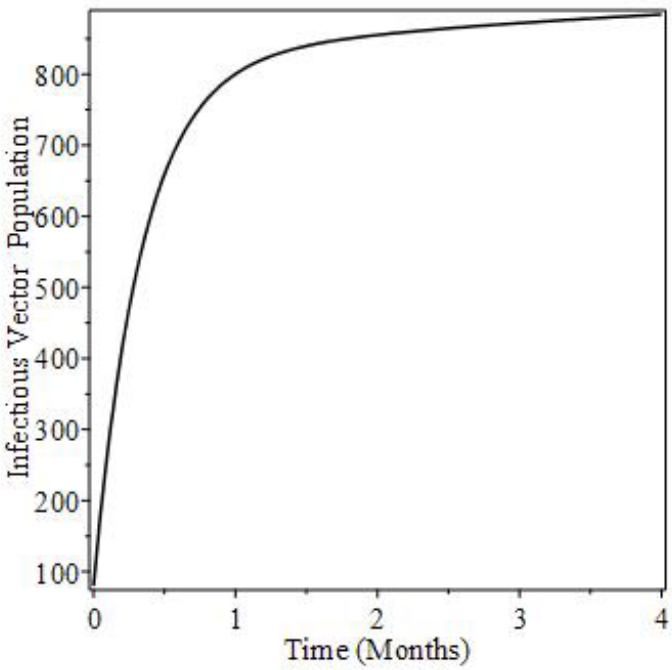

Figure 6: Infectious vector population.

8.2. Infectious animal and vector populations

Figure 5 and 6 indicates the population change in the population of the infectious animals and vectors. There have been an increase in the populations of both the infectious animals and vectors. This can be explain by the decrease in the number of both the susceptible vectors and animals as shown in figure 2 and 3. This means that more animals and vectors that are susceptible usually move to the infectious compartment. Therefore, there are increase in the population of the infectious vectors and animals.

8.3. Recovered animal population

The population change in the class of susceptible animals and that of the infectious animals are inversely proportional. The relationship could explain the continuous decrease in the population of the recovered populations of animals as shown in figure 2 and 5. This means that the rate of recovery is proportional to the rate of the rate at which the susceptible move to the infectious compartment as indicated in figure and.

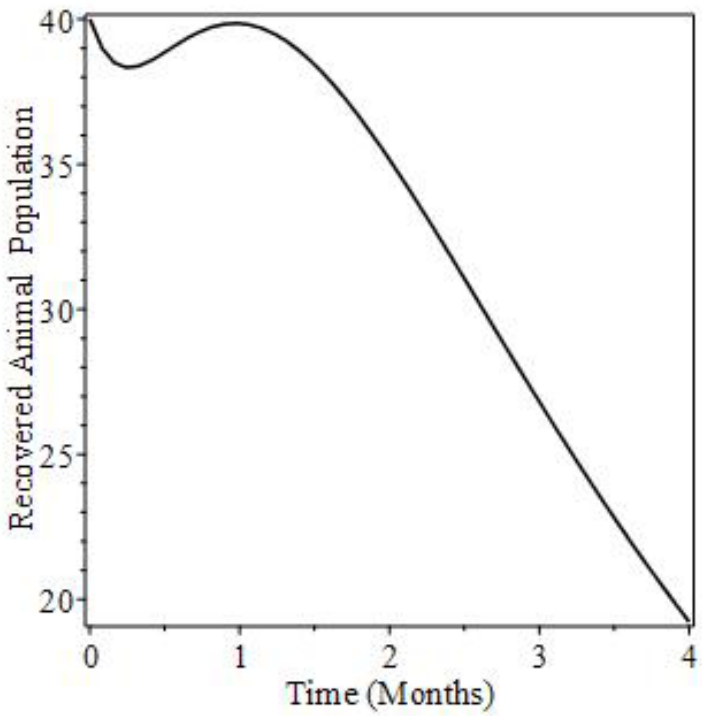

Figure 7: Recovered animal population.

9. Conclusion

We formulated a deterministic model for trypanosomiasis infections. The threshold value for the determination of the disease was computed using the next generation matrix approach. The disease free-equilibrium of the Trypanosomiasis model was determined for local stability and its associated reproductive rate. The disease free equilibrium of the trypanosomiasis model was found to be locally asymptotically stable whenever the reproductive number was less than unity. Sensitivity analysis was carried out to determine the contribution of each parameter to the basic reproductive number. It was found that, a decrease in both the animal and vector recruitment rates would decrease the basic reproduction number. We performed numerical simulations of the system of equations of the model. We found that there have been an increase in the populations of both the infectious animals and vectors.

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