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Parameter Driven Dynamics of Trypanosomiasis in a Cattle Population

J. Otieno

Maseno University, Box 333, Maseno, Kenya

J. Y. T. Mugisha and B. K. Nannyonga

Makerere University, Box 7062, Kampala, Uganda

P. Oleche

Maseno University, Box 333, Maseno, Kenya

Abstract

In this paper we study the disease dynamics of trypanosomiasis in a cattle population. The compartmental model presented includes the wild animal population which provides an alternative feeding source for the tsetse fly. An epidemiological parameter, the basic reproduction number is calculated. Based on this parameter, conditions for the global stability of the disease-free and endemic equilibrium points of the model are established. To aid decision making on which parameters to monitor in order to control the disease, a sensitivity analysis of the parameters which define the basic reproduction number is carried out. Results obtained from the sensitivity analysis indicate that the parameters with the highest influence on the spread of the disease are the vector biting rate, the vector survival rate and the vector death rate. These results indicate that an effective control of the disease would require a reduction of the contact rate between the cattle and the vector population.

Keywords: Parameter-driven, vector biting rate, vector survival rate

1 Introduction

Trypanosomiasis in cattle is a vector-borne disease transmitted by the tsetse fly and caused by a range of protozoan parasites of the *genus Typanosoma*. Trypanosomes are multi-host parasites equally capable of infecting a wide range of domestic and wildlife species, which constitute a reservoir for cattle infections. In most parts of Africa, cattle are the main species affected, due to the tsetse fly feeding preferences and the fact that they can shield other domesticated animals such as goats and pigs from the effects of trypanosomiasis [3].
The disease is a major impediment to livestock particularly cattle farming in sub-Saharan Africa. It limits the full potential of agricultural development in the 36 countries where it is endemic, and leads to loss of productivity in animals. Without treatment, it is frequently fatal [18]. It has a direct impact on the average number of livestock kept by farmers, and even more important in effect are the indirect impacts the disease has on settlement patterns, land use, draught power use, animal husbandry and farming [4].
Though much is known about the biology and ecology of the vector, the transmission of the disease and a variety of control measures developed and demonstrated, trypanosomiasis is still a significant constraint on animal production, human health and agricultural livelihoods in many parts of Africa [9]. To establish a correct perspective view of the complex dynamics of disease in given populations, models of disease transmission with correct parameter values, provide estimates of transmission thresholds that are the key to disease eradication.
A number of mathematical models both simulation [22, 23] and analytical [19, 5] have been proposed to describe African trypanosomiasis. The analytical models are derived from formulations similar to the MacDonald-type models [8]. They describe the new cases of a vector-borne disease which will arise at some time in the future from one case in the present time, the basic reproduction number [6]. Milligan and Baker [19] in their study described a compartmental model for trypanosomiasis transmission to determine a criteria for successful disease control by treatment and by vector control. The study indicated that the long life span of the vectors lead to high infection rates in the vector and high values of R_0. A sensitivity analysis of the model carried out using Monte Carlos methods emphasized the need to carry out studies of the wild animal reservoir alongside entomological surveys.
Their study incorporated the heterogeneity in transmission due to different tsetse fly species and feeding preferences so that the rate of disease transmission from the vector to host and vice versa vary from species to species. The age difference in the vector population and seasonality in cattle parasitaemia which present differences in susceptibility to infection were also incorporated into the model. The current study however is interested in a more strategic

model which simplifies the system to its bare essentials and is useful for studying general disease dynamics and control, [13].
Although the disease is complex, involves several trypanosomes with varying transmission effects and is transmitted by a wide range of tsetse fly species, a further consequence of the unusual life history of tsetse is their tendency to have low genetic variability within a given population [21]. This study therefore assumes a single trypanosome is transmitted by a particular species of the tsetse fly in this cattle population. The vector categories considered are those that directly affect the dynamics of the disease regardless of their age, the susceptible and infectious vectors. The susceptible vectors affect disease transmission depending on the rate of infection while the already infected ones transmit the disease depending on the proportion of bites on an exposed host. The vector populations exhibit a range of seasonal behavior, from populations with large fluctuations with a peak in the late rainy season, to those which appear to be constant or to show only slight seasonality [19], however the disease dynamics in this study are restricted to the vector population at peak seasons. This paper hence describes an analytical model for trypanosomiasis in a cattle population that incorporates the wild animal population. Its objective is to establish parameters that are key to eradicating the disease.

2 Model formulation

The model is formulated assuming that the cattle, vector and wild animal populations are each divided into two compartments each, the susceptible S and the infective I populations. The subscripts c, v and w denote the cattle, vector and wild animal populations respectively. The susceptible populations are replenished by birth or recruitment at constant rates Λ_c, Λ_v and Λ_w and decreased by both natural death rates μ_c, μ_v and μ_w and rates of infection λ_c, λ_v and λ_w. A further assumption is that the vector population is a single species transmitting a single pathogen.
The rates of infection are functions of $\alpha_i, i = 1, 2$, the per-capita biting rates, $\tau_i, i = 1, 2, 3, 4$, the transmission probabilities, ε, the vector survival rate and $\frac{I_i}{N_i}$, the ratio of the infected transmission population to the total susceptible populations. Hence the rates of infection are $\lambda_c = \alpha_1\tau_1\varepsilon\frac{I_v}{N_c}$, $\lambda_w = \alpha_2\tau_4\varepsilon\frac{I_v}{N_w}$ and $\lambda_v = \alpha_1\tau_2\frac{I_c}{N_c} + \alpha_2\tau_3\frac{I_w}{N_w}$ in the cattle, wild animal and vector populations respectively. The vectors get infected when they either come in contact with infected cattle or infected wild animals.
The infected compartments in the cattle, vector and wild animal populations are generated by infection of the corresponding susceptible compartments and decreased by natural death rates μ_c, μ_v and μ_w coupled with a disease induced death rate κ in the cattle population.

From the above assumptions and description, the following system of differential equations is formulated:

$$\begin{aligned}
\frac{dS_c}{dt} &= \Lambda_c - \left\{\mu_c + \alpha_1\tau_1\varepsilon\frac{I_v}{N_c}\right\}S_c,\\
\frac{dI_c}{dt} &= \alpha_1\tau_1\varepsilon\frac{I_v}{N_c}S_c - (\mu_c+\kappa)I_c,\\
\frac{dS_v}{dt} &= \Lambda_v - \left\{\alpha_1\tau_2\frac{I_c}{N_c} + \alpha_2\tau_3\frac{I_w}{N_w} + \mu_v\right\}S_v,\\
\frac{dI_v}{dt} &= \left\{\alpha_1\tau_2\frac{I_c}{N_c} + \alpha_2\tau_3\frac{I_w}{N_w}\right\}S_v - \mu_v I_v,\\
\frac{dS_w}{dt} &= \Lambda_w - \left\{\alpha_2\tau_4\varepsilon\frac{I_v}{N_w} + \mu_w\right\}S_w,\\
\frac{dI_w}{dt} &= \alpha_2\tau_4\varepsilon\frac{I_v}{N_w}S_w - \mu_w I_w.
\end{aligned} \tag{1}$$

with

$$\begin{aligned}
\frac{dN_c}{dt} &= \Lambda_c - \mu_c N_c - \kappa I_c,\\
\frac{dN_v}{dt} &= \Lambda_v - \mu_v N_v,\\
\frac{dN_w}{dt} &= \Lambda_w - \mu_w N_w.
\end{aligned} \tag{2}$$

Since (1) describes the dynamics of trypanosomiasis in cattle, tsetse fly and wild animal populations, it makes sense to expect the state variables defined in the biologically feasible region

$$\Omega = (S_c, I_c, S_v, I_v, S_w, I_w) \in \mathbb{R}^6_+,$$

to be non-negative for all values of $t \geq 0$ and that the solution to the system remain positive for all $t \geq 0$. Further density-dependent factors ensure that the respective populations have bounded recruitment functions.

Theorem 2.1. *Let the initial values be* $\{S_c(0), I_c(0), S_v(0), I_v(0), S_w(0), I_w(0)\} \geq 0 \in \Omega$*; then the solution set* $\{S_c(t), I_c(t), S_v(t), I_v(t), S_w(t), I_w(t)\}$ *of the system* (1) *is positive* $\forall t \geq 0$.

Proof. From the first equation of (1),

$$\begin{aligned}
\frac{dS_c}{dt} &= \Lambda_c - \left\{\mu_c + \alpha_1\tau_1\varepsilon\frac{I_v}{N_c}\right\}S_c,\\
\frac{dS_c}{S_c} &\geq -(\mu_c + \alpha_1\tau_1\varepsilon)dt,
\end{aligned}$$

integrating both sides gives

$$S_c(t) \geq S_c(0)e^{-(\mu_c+\alpha_1\tau_1\varepsilon)t} \geq 0.$$

Clearly, $S_c(t)$ is positive for all $t \geq 0$. Similarly, for the rest of the equations in (1) it is clear that the solution for the system remains positive for all $t \geq 0$. This result shows that solutions with initial values in Ω, remain non-negative for all $t \geq 0$. □

Theorem 2.2. *Ω is positively invariant in* (1) *(i.e. all solutions in Ω remain in Ω for all time).*

Proof. From the first equation of (2)

$$\begin{aligned} \frac{dN_c}{dt} &\leq \Lambda_c - \mu_c N_c, \\ N_c &\leq \frac{\Lambda_c}{\mu_c} - \left\{ \frac{\Lambda_c - \mu_c N_0}{\mu_c} \right\} e^{-\mu_c t}. \end{aligned} \tag{3}$$

As $t \to \infty$ in (3), $N_c \to \dfrac{\Lambda_c}{\mu_c}$. Hence $N_c \leq \dfrac{\Lambda_c}{\mu_c}$. It is clear from the second and third equations of (2) that, $N_v \to \dfrac{\Lambda_v}{\mu_v}$; $N_w \to \dfrac{\Lambda_w}{\mu_w}$. Consequently Ω is positively invariant under (1). □

From the above discussion, a set Ω is defined as

$$\Omega = \left\{ 0 \leq N_c \leq \frac{\Lambda_c}{\mu_c}, 0 \leq N_v \leq \frac{\Lambda_v}{\mu_v}, 0 \leq N_w \leq \frac{\Lambda_w}{\mu_w} \right\}. \tag{4}$$

In the remaining part of this paper the state variables are restricted to the set (4).

3 Analysis of the model

In this section, the model (1) is analyzed to establish some of the results. The stability of the equilibrium points in epidemic modelling is important. When a disease-free equilibrium is stable (especially globally), the outbreak of the disease is not expected, for life, but when it is unstable, another outbreak would be anticipated when particular conditions for the disease reappear.
The steady states of the model (1), are obtained from setting the right hand side of Equation (1) to zero. The disease-free equilibrium, ε^o, is obtained when $I_c = I_v = I_w = 0$ and is given by

$$\varepsilon^o = \{S_c, I_c, S_v, I_v, S_w, I_w\} = \left\{ \frac{\Lambda_c}{\mu_c}, 0, \frac{\Lambda_v}{\mu_v}, 0, \frac{\Lambda_w}{\mu_w}, 0 \right\}. \tag{5}$$

The disease-free equilibrium is the point at which the population remains in the absence of disease.

3.1 Local stability analysis of ε^o

The local stability of ε^o is established using the next generation operator approach [15] on the system (1). In this approach, a threshold quantity, the basic reproduction number R_0, is estimated which is critical to the asymptotic stability of the disease-free equilibrium.
Using the notation in [15], the infection matrix F and the transition matrix V, are given by (noting that $S_c^* = N_c^*$, $S_v^* = N_v^*$, and$S_w^* = N_w^*$ at the disease-free equilibrium, ε^o)

$$F = \begin{pmatrix} 0 & \alpha_1\tau_1\varepsilon & 0 \\ \alpha_1\tau_2 h & 0 & \alpha_2\tau_3\rho \\ 0 & \alpha_2\tau_4\varepsilon & 0 \end{pmatrix},$$

where the constants $h = \dfrac{N_v}{N_c}$ and $\rho = \dfrac{N_v}{N_w}$ represent the vector-cattle and vector-wild animal ratios respectively. These ratios are assumed to be constant because it is known that a vector takes a fixed number of blood-meals per unit time independent of the population density in the host [12].
The (i, j) entry of F is the rate at which the infected individuals in compartment j produce new infections in the compartment i. The infected vectors produce new infections in the cattle population at the rate $\alpha_1\tau_1\varepsilon$ and in the wild animal population at the rate $\alpha_2\tau_4\varepsilon$, while the infected cattle and wild animal populations produce new infections in the vector population at the rate $\alpha_1\tau_2 h$ and $\alpha_2\tau_3\rho$ respectively. The transition matrix

$$V = \begin{pmatrix} (\mu_c + \kappa) & 0 & 0 \\ 0 & \mu_v & 0 \\ 0 & 0 & \mu_w \end{pmatrix}.$$

The (i, j) entry of V is the rate individuals in stage j progress to stage i and its inverse given by

$$V^{-1} = \begin{pmatrix} \dfrac{1}{(\mu_c + \kappa)} & 0 & 0 \\ 0 & \dfrac{1}{\mu_v} & 0 \\ 0 & 0 & \dfrac{1}{\mu_w} \end{pmatrix},$$

is the expected time spent in compartment i by an individual initially in compartment j over the course of its infection. The average length of time an infected cow, vector and wild animal spends in the infected compartment during its lifetime assuming that the population remains near the DFE and barring infection is $\dfrac{1}{(\mu_c + \kappa)}$, $\dfrac{1}{\mu_v}$ and $\dfrac{1}{\mu_w}$ respectively. The next generation matrix or

operator is hence given by

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\alpha_1\tau_1\varepsilon}{\mu_v} & 0 \\ \frac{\alpha_1\tau_2 h}{(\mu_c+\kappa)} & 0 & \frac{\alpha_2\tau_3\rho}{\mu_w} \\ 0 & \frac{\alpha_2\tau_4\varepsilon}{\mu_v} & 0 \end{pmatrix}. \tag{6}$$

The (i, j) entry of (6) is the expected number of secondary infections produced in compartment i by an index case initially in the compartment j. The expected number of new infections in the infected vectors compartment, produced by the infected vectors originally introduced into the infected cattle and wild animal population is $\frac{\alpha_1\tau_1\varepsilon}{\mu_v}$ and $\frac{\alpha_2\tau_4\varepsilon}{\mu_v}$ respectively. The infected cattle and wild animal population produce infected vectors and vice versa causing the offdiagonal structure of FV^{-1}.

Thus, the basic reproduction number R_0, the spectral radius of the matrix FV^{-1}, is given by

$$R_0 = \sqrt{\frac{\alpha_2^2\tau_4\tau_3\varepsilon\rho}{\mu_v\mu_w} + \frac{\alpha_1^2\tau_2\tau_1\varepsilon h}{\mu_v(\mu_c+\kappa)}}. \tag{7}$$

The basic reproduction number given by (7) is biologically meaningful because as expected, it is jointly proportional to the probability of infection per contact between a susceptible and an infectious individual, the average rate of contact between a susceptible and an infectious individual and the duration of infectiousness [11]. Near the disease-free equilibrium, each infected wild animal produces $\frac{\alpha_2\tau_3\rho}{\mu_w}$ infected vectors over its expected infectious period, and each infected vector produces $\frac{\alpha_2\tau_4\varepsilon}{\mu_v}$ new infected wild animals over its expected infectious period. Similarly each infected cow produces $\frac{\alpha_1\tau_2 h}{(\mu_c+\kappa)}$ new infected vectors over its expected infectious period and each infected vector produces $\frac{\alpha_1\tau_1\varepsilon}{\mu_v}$ new infected cows over its infectious period. As indicated by Chitnis *et. al.*, [14], R_0 measures the initial disease transmission.

The number of new infections in cattle that one cow causes through their infectious period is R_0^2 and not R_0. This is because the definition of R_0 in (7) based on the next generation approach [16], counts the number of infections from one generation to the next [14]. In this case, the number of new infections in the tsetse flies count as one generation, the waves of secondary infections that flow from each previous infection. The first generation of an epidemic is all the secondary infections that result from infectious contact with the index case, who is of generation zero. R_0 refers to the number of infections generated by the index case, i.e., generation zero. The square root in Equation (7)

arises from the two "generations" required for an infected vector or host to "reproduce" itself, that is, one to transmit and one to get infected [11].
Since (2) satisfies axioms (A1)-(A5) of the Theorem 2 in van den Driessche and Watmough [17], and the R_0 calculated above is biologically meaningful, then:

Lemma 3.1. *The disease free equilibrium is locally stable for $R_0 < 1$ and unstable when $R_0 > 1$.*

3.2 Global stability analysis of ε^o

To establish the global stability conditions for the disease-free equilibrium when $R_0 < 1$, (2) is written in the form as in Castillo-Chavez *et. al.*, [1]

$$\begin{aligned} \frac{dZ}{dt} &= G(X, Z), \quad G(X, 0) = 0, \\ \frac{dX}{dt} &= F(X, Z). \end{aligned}$$

where $X \in \mathbb{R}^m$ denotes the number of uninfected individuals and $Z \in \mathbb{R}^n$ denotes the number of infected individuals. Then the two conditions:

1. For $\dfrac{dX}{dt} = F(X, 0), \quad X^*$ is globally asymptotically stable,

2. $G(X, Z) = AZ - \widehat{G}(X, Z), \quad \widehat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$

where $A = D_Z G(X^*, 0)$ is an M-matrix (the off diagonal elements of A are non-negative) and Ω is the region where the model makes biological sense, if met, also guarantee the global asymptotic stability of the disease free state [1]. If (1) satisfies the above two conditions then the following theorem holds:

Theorem 3.2. *If $R_0 < 1$, then the disease-free equilibrium point is globally asymptotically stable in Ω.*

Proof. Defining new variables and breaking the system given by (1) into two: the susceptible and the infected sub-systems, coupled with $Z = (I_c, I_v, I_w)$ and $X = (S_c, S_v, S_w)$, then (1) can be written as:

$$\begin{aligned} \frac{dZ}{dt} &= G(X, Z), \quad G(X, 0) = 0, \\ \frac{dX}{dt} &= F(X, Z). \end{aligned} \tag{8}$$

where the two functions are given by:

$$G(X,Z) = \Bigg[\alpha_1\tau_1\varepsilon\frac{I_v}{N_c}S_c - (\mu_c+\kappa)I_c; \quad \left\{\alpha_1\tau_2\frac{I_c}{N_c} + \alpha_2\tau_3\frac{I_w}{N_w}\right\}S_v - \mu_v I_v; \\ \alpha_2\tau_4\varepsilon\frac{I_v}{N_w}S_w - \mu_w I_w\Bigg]^T, \tag{9}$$

$$F(X,Z) = \Bigg[\Lambda_c - \left\{\mu_c + \alpha_1\tau_1\varepsilon\frac{I_v}{N_c}\right\}S_c; \Lambda_v - \left\{\alpha_1\tau_2\frac{I_c}{N_c} + \alpha_2\tau_3\frac{I_w}{N_w} + \mu_v\right\}S_v; \\ \Lambda_w - \left\{\alpha_2\tau_4\varepsilon\frac{I_v}{N_w} + \mu_w\right\}S_w\Bigg]^{\mathrm{T}}.$$

Consider the reduced system: $\dfrac{dX}{dt} = F(X,0)$

$$\begin{aligned} \frac{dS_c}{dt} &= \Lambda_c - \mu_c S_c, \\ \frac{dS_v}{dt} &= \Lambda_v - \mu_v S_v, \\ \frac{dS_w}{dt} &= \Lambda_w - \mu_w S_w. \end{aligned} \tag{10}$$

$X^* = \{S_c^*, S_v^*, S_w^*\} = \left\{\dfrac{\Lambda_c}{\mu_c}, \dfrac{\Lambda_v}{\mu_v}, \dfrac{\Lambda_w}{\mu_w}\right\}$ is a globally asymptotically stable equilibrium point for the reduced system $\dfrac{dX}{dt} = F(X,0)$. This is clear when the first equation in (10) is solved to obtain

$$S_c = \frac{\Lambda_c}{\mu_c} - \left\{\frac{\Lambda_c}{\mu_c} - S_c(0)\right\}e^{-\mu_c t} \to \frac{\Lambda_c}{\mu_c} \quad as \quad t\to\infty.$$

Similarly solving the second and third equations give

$$S_v = \frac{\Lambda_v}{\mu_v} - \left\{\frac{\Lambda_v}{\mu_v} - S_v(0)\right\}e^{-\mu_v t} \to \frac{\Lambda_v}{\mu_v} \quad as \quad t\to\infty,$$

and

$$S_w = \frac{\Lambda_w}{\mu_w} - \left\{\frac{\Lambda_w}{\mu_w} - S_w(0)\right\}e^{-\mu_w t} \to \frac{\Lambda_w}{\mu_w} \quad as \quad t\to\infty$$

respectively. These asymptotic dynamics are independent of the initial conditions in Ω. This implies that the convergence of the solutions of (10) is global in Ω. Since $X^* = \{S_c^*, S_v^*, S_w^*\} = \left\{\dfrac{\Lambda_c}{\mu_c}, \dfrac{\Lambda_v}{\mu_v}, \dfrac{\Lambda_w}{\mu_w}\right\}$ so that

$$G(X^*,Z) = \begin{pmatrix} \alpha_1\tau_1\varepsilon I_v - (\mu_c+\kappa)I_c \\ \alpha_1\tau_2\dfrac{I_c}{N_c}\dfrac{\Lambda_v}{\mu_v} + \alpha_2\tau_3\dfrac{I_w}{N_w}\dfrac{\Lambda_v}{\mu_v} - \mu_v I_v \\ \alpha_2\tau_4\varepsilon I_v - \mu_w I_w \end{pmatrix},$$

$G(X,Z) = AZ - \widehat{G}(X,Z)$ where $A = D_Z G(X^*, 0)$

$$A = \begin{pmatrix} -(\mu_c + \kappa) & \alpha_1\tau_1\varepsilon & 0 \\ \alpha_1\tau_2\dfrac{\Lambda_v}{\mu_v N_c} & -\mu_v & \alpha_2\tau_3\dfrac{\Lambda_v}{\mu_v N_w} \\ 0 & \alpha_2\tau_4\varepsilon & -\mu_w \end{pmatrix}$$

and

$$\widehat{G}(X,Z) = \begin{pmatrix} \widehat{G_1}(X,Z) \\ \widehat{G_2}(X,Z) \\ \widehat{G_3}(X,Z) \end{pmatrix}$$
$$= \begin{pmatrix} \alpha_1\tau_1\varepsilon I_v\left\{1 - \dfrac{S_c}{N_c}\right\} \\ \alpha_1\tau_2\dfrac{I_c}{N_c}\left\{\dfrac{\Lambda_v}{\mu_v} - S_v\right\} + \alpha_2\tau_3\dfrac{I_w}{N_w}\left\{\dfrac{\Lambda_v}{\mu_w} - S_v\right\} \\ \alpha_2\tau_4\varepsilon I_v\left\{1 - \dfrac{S_w}{N_w}\right\} \end{pmatrix}.$$

$\widehat{G_1}(X,Z)$ and $\widehat{G_3}(X,Z)$ are both greater than 0 since $\dfrac{S_w}{N_w}$ and $\dfrac{S_c}{N_c}$ are proportions. Equally since the vector and wild animal populations equilibrate at $\dfrac{\Lambda_v}{\mu_v}$ and $\dfrac{\Lambda_w}{\mu_w}$ respectively, the expression $\alpha_1\tau_2\dfrac{I_c}{N_c}\left\{\dfrac{\Lambda_v}{\mu_v} - S_v\right\} + \alpha_2\tau_3\dfrac{I_w}{N_w}\left\{\dfrac{\Lambda_v}{\mu_w} - S_v\right\}$ in $\widehat{G_2}(X,Z)$ is non-negative. Therefore since the disease-free equilibrium point is locally asymptotically stable for $R_0 < 1$, the globally stability equilibrium follows from the theorem. □

3.3 The Endemic Equilibrium, ε^*

To analyse the stability of the equilibrium point ε^*, the Centre Manifold theorem as described in Theorem 4 of [1] is used. It states that if f is C^r (r times continuously differentiable) then at every equilibrium point there is a unique C^r stable manifold, a unique C^r unstable manifold and a (not necessarily unique) C^{r-1} centre manifold, [10]. As the stability of the equilibrium correlates with the stability of its manifolds, the existence of the centre manifold brings up the question of the dynamics of the centre manifold. However, before stating our main result, we give the following theorem which will be useful in the subsequent section.

Theorem 3.3. *With reference to Castillo and Song [2], consider the general system of ordinary differential equations with a parameter ϕ, that is*

$$\frac{dx}{dt} = f(x, \phi), f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n \quad \textit{and} \quad f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}). \tag{11}$$

Without loss of generality, it is assumed that 0 *is an equilibrium for* (11) *for all values of the parameter ϕ.*
Assume

A1 $A = D_x f(0,0) = \left\{ \dfrac{\partial f_i}{\partial x_i}(0,0) \right\}$, *is the linearization of* (11) *around the equilibrium* 0 *with ϕ evaluated at* 0. *Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;*

A2 *Matrix A has a nonnegative right eigenvector $\boldsymbol{w}$ and a left eigenvector $\boldsymbol{v}$ corresponding to the zero eigenvalue.*

Let f_k be the k^{th} component of f and

$$\begin{aligned} a &= \sum v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \\ b &= \sum v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \end{aligned} \tag{12}$$

The local dynamics of (11) *around* 0 *are totally determined by a and b.*

i *$a > 0$, $b > 0$. When $\phi < 0$ with $\phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium;*

ii *$a < 0$, $b < 0$. When $\phi < 0$ with $\phi \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium;*

iii *$a > 0$, $b < 0$. When $\phi < 0$ with $\phi \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable and a positive unstable equilibrium appears;*

iv *$a < 0$, $b > 0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.*

3.4 Local stability analysis of ε^*

To establish the local asymptotic stability of the endemic equilibrium ε^*, using the Centre Manifold theorem the following definitions are made: $S_c = x_1$, $I_c = x_2$, $S_v = x_3$, $I_v = x_4$, $S_w = x_5$ and $I_w = x_6$. Using the vector notation $X = (x_1, x_2, x_3, x_4, x_5)^T$, the system (2) under these condition can be written in the form $\dfrac{dX}{dt} = F = (f_1, f_2, f_3, f_4, f_5)^T$, such that

$$\begin{aligned}
\frac{dx_1}{dt} = f_1 &= \Lambda_c - (\mu_c + \alpha_1\tau_1\varepsilon\frac{x_4}{x_1+x_2})x_1,\\
\frac{dx_2}{dt} = f_2 &= \alpha_1\tau_1\varepsilon\frac{x_4}{x_1+x_2}x_1 - (\mu_c+\kappa)x_2,\\
\frac{dx_3}{dt} = f_3 &= \Lambda_v - (\alpha_1\tau_2\frac{x_2}{x_1+x_2} + \alpha_2\tau_3\frac{x_6}{x_5+x_6} + \mu_v)x_3,\\
\frac{dx_4}{dt} = f_4 &= (\alpha_1\tau_2\frac{x_2}{x_1+x_2} + \alpha_2\tau_3\frac{x_6}{x_5+x_6})x_3 - \mu_v x_4,\\
\frac{dx_5}{dt} = f_5 &= \Lambda_w - (\alpha_2\tau_4\varepsilon\frac{x_4}{x_5+x_6} + \mu_w)x_5,\\
\frac{dx_6}{dt} = f_6 &= \alpha_2\tau_4\varepsilon\frac{x_4}{x_5+x_6}x_5 - \mu_w x_6.
\end{aligned} \tag{13}$$

The method involves evaluating the Jacobian of (13) at the disease-free equilibrium (ε^o) denoted by $J(\varepsilon^o)$. The reproduction number of the (13) is given as in (7).

The tsetse fly bites both the wild animal and cattle populations yet the rate at which they bite the wild animal population is higher [19], so that $\alpha_1 < \alpha_2$. This means that $\alpha_2 = \theta\alpha_1$, where $\theta > 1$ is the modification parameter which captures the increased transmissibility of animal trypanosomiasis when the vectors feed on the wild animal population. If we let $\alpha = \alpha_1$ and choose α to be the bifurcation parameter, solving for $\alpha = \alpha^*$ when $R_0 = 1$ gives,

$$\alpha^* = \sqrt{\frac{\mu_v\mu_w(\mu_c+\kappa)}{\theta^2\tau_4\tau_3\varepsilon\rho(\mu_c+\kappa) + \tau_2\tau_1\varepsilon h\mu_w}}. \tag{14}$$

The linearized system of the transformed (13) with $\alpha = \alpha^*$ chosen as a bifurcation parameter has a simple zero eigenvalue. Hence, the Jacobian of (13) at $\alpha = \alpha^*$ has a right eigenvector (corresponding to the zero eigenvalue) given by $\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6)^T$, with

$$\begin{aligned}
&w_1 = \frac{\alpha^*\tau_1\varepsilon}{\mu_c}\eta w_4; \quad w_2 = \frac{\alpha^*\tau_1\varepsilon}{(\mu_c+\kappa)}w_4; \quad w_3 = \eta w_4;\\
&w_4 > 0; \quad w_5 = -\frac{\theta\alpha^*\tau_4\varepsilon}{\mu_w}w_4; \quad w_6 = \frac{\theta\alpha^*\tau_4\varepsilon}{\mu_w}w_4,
\end{aligned}$$

where $\eta = \dfrac{\alpha^{*2}\tau_2\tau_1\varepsilon h}{\mu_v(\mu_c+\kappa)} + \dfrac{\theta\alpha^{*2}\tau_4\tau_3\varepsilon\rho}{\mu_v\mu_w}$, and it has a left eigenvector (corresponding to the zero eigenvalue) given by $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6)^T$, with

$$v_1 = 0; \quad v_2 = \frac{\alpha^*\tau_2 h}{(\mu_c+\kappa)}v_4; \quad v_3 = 0; \quad v_4 > 0; \quad v_5 = 0; \quad v_6 = \frac{\theta\alpha^*\tau_3\rho}{\mu_w}v_4.$$

For (13), the associated non-zero partial derivatives of F at ε^o are given by

$$\begin{aligned}
&\frac{\partial^2 f_2}{\partial x_2\partial x_4} = -\frac{\alpha^*\tau_1\varepsilon\mu_c}{\Lambda_c}; \quad \frac{\partial^2 f_4}{\partial x_1\partial x_2} = -\frac{\alpha^*\tau_2 h\mu_c}{\Lambda_c}; \quad \frac{\partial^2 f_4}{\partial x_2^2} = -\frac{2\alpha^*\tau_2 h\mu_c}{\Lambda_c};\\
&\frac{\partial^2 f_4}{\partial x_2\partial x_3} = \frac{\alpha^*\tau_2\mu_c}{\Lambda_c}; \quad \frac{\partial^2 f_4}{\partial x_3\partial x_6} = \frac{\theta\alpha^*\tau_3\mu_w}{\Lambda_w}; \quad \frac{\partial^2 f_4}{\partial x_5\partial x_6} = -\frac{\theta\alpha^*\tau_3\rho\mu_w}{\Lambda_w};\\
&\frac{\partial^2 f_4}{\partial x_6^2} = -\frac{2\theta\alpha^*\tau_3\rho\mu_w}{\Lambda_w}.
\end{aligned} \tag{15}$$

From (15), the parameter a as defined in (12) is given by

$$a = -2\alpha^{*2}\left[M_1M_4 + \eta\left\{M_5 + \frac{M_1}{\Lambda_c} + M_1M_3\right\}\right]v_4w_4^2 \; < \; 0 \tag{16}$$

where $M_1 = \dfrac{\tau_2\tau_1\varepsilon h}{\mu_c+\kappa}$, $M_2 = \dfrac{\theta^2\tau_4\tau_3\varepsilon}{\Lambda_w}$, $M_3 = \dfrac{2\theta\alpha^*\tau_4\varepsilon\rho}{\mu_w}$, $M_4 = \dfrac{3\alpha^*\tau_1\varepsilon\mu_c}{\Lambda_c}$ and $M_5 = \dfrac{\alpha^*\tau_1}{\mu_c+\kappa}$.
To calculate b, the associated non-vanishing partial derivatives are:

$$\frac{\partial^2 f_4}{\partial x_2\partial\alpha} = \tau_2 h; \quad \frac{\partial^2 f_4}{\partial x_6\partial\alpha} = \theta\tau_3\rho$$

so that

$$b = \left\{\frac{\alpha\tau_2\tau_1\varepsilon h}{\mu_c+\kappa} + \frac{\theta^2\alpha\tau_4\tau_3\varepsilon\rho}{\mu_w}\right\}v_4w_4 \; > \; 0. \tag{17}$$

From (16) and (17), $a < 0$, $b > 0$ Lemma (3.4) follows.

Lemma 3.4. *The endemic equilibrium for the system* (2) *exists and is locally asymptotically stable whenever* $R_0 > 1$ *as stated in [2] Theorem 4.1 (iv).*

The system (1) exhibits a supercritical bifurcation. This means that the exchange of stability between the disease-free and endemic steady states guarantees that the endemic steady state is locally asymptotically stable whenever $R_0 > 1$.

3.5 Global stability analysis of ε^*

In this sub-section, the global stability of the endemic equilibrium of (2) is established using the Lyapunov direct method (also called the second method of Lyapunov). This method makes it possible to determine the stability of a system without explicitly integrating the differential equations in the system.

Theorem 3.5. *Let $L(x,t)$ be a non-negative function with derivative $\dot{L}$ along the trajectories of the system. If $L(x,t)$ is a positive definite, then the origin of the system is globally asymptotically stable.*

Theorem (3.5) gives sufficient conditions for the global stability of a system. Though the search for a Lyapunov function establishing stability of an equilibrium point could be arduous, the Lyapunov function of the form $\widehat{L}(x_1,x_2,...,x_n) = \sum_{i=1}^{n} c_i \left\{ x_i - x_i^* - x_i^* log\frac{x_i}{x_i^*} \right\}$ can be especially useful for host-vector models with any number of compartments, [7].
From the general theory a unique endemic equilibrium exists which satisfies the following relations:

$$\begin{aligned}
\Lambda_c &= \mu_c S_c^* + \alpha_1\tau_1\varepsilon\frac{I_v^*}{N_c^*}S_c^*,\\
(\mu_c+\kappa)I_c^* &= \alpha_1\tau_1\varepsilon\frac{I_v^*}{N_c^*}S_v^*,\\
\Lambda_v &= \alpha_1\tau_2\frac{I_c^*}{N_c^*}S_v^* + \alpha_2\tau_3\frac{I_w^*}{N_w^*}S_v^* + \mu_v S_v^*,\\
\mu_v I_v^* &= \alpha_1\tau_2\frac{I_c^*}{N_c^*}S_v^* + \alpha_2\tau_3\frac{I_w^*}{N_w^*}S_v^*,\\
\Lambda_w &= \alpha_2\tau_4\varepsilon\frac{I_v^*}{N_w^*}S_w^* + \mu_w S_w^*,\\
\mu_w I_w^* &= \alpha_2\tau_4\varepsilon\frac{I_v^*}{N_w^*}S_w^*.
\end{aligned} \tag{18}$$

Theorem 3.6. *If $R_0 > 1$ then ε^* is globally asymptotically stable in Ω.*

Proof. We define the possible Lyapunov function
$L : (S_c, I_c, S_v, I_v, S_w, I_w) \in \Omega : S_c, I_c, S_v, I_v, S_w, I_w > 0 \to \mathbb{R}$ by

$$\begin{aligned}
L(S_c,I_c,S_v,I_v,S_w,I_w) =& c_1\left\{S_c - S_c^* - S_c^*\log\frac{S_c}{S_c^*}\right\} + c_2\left\{I_c - I_c^* - I_c^*\log\frac{I_c}{I_c^*}\right\} +\\
& c_3\left\{S_v - S_v^* - S_v^*\log\frac{S_v}{S_v^*}\right\} + c_4\left\{I_v - I_v^* - I_v^*\log\frac{I_v}{I_v^*}\right\} +\\
& c_5\left\{S_w - S_w^* - S_w^*\log\frac{S_w}{S_w^*}\right\} + c_6\left\{I_w - I_w^* - I_w^*\log\frac{I_w}{I_w^*}\right\}
\end{aligned}$$

where

$$\begin{aligned} c_1 = c_2 &= \alpha_1\tau_1\varepsilon\frac{I_v^*}{N_c^*}S_c^*, \\ c_3 = c_4 &= \left\{\alpha_1\tau_2\frac{I_c^*}{N_c^*} + \alpha_2\tau_3\frac{I_w^*}{N_w^*}\right\}S_v^*, \\ c_5 = c_6 &= \alpha_2\tau_4\varepsilon\frac{I_v^*}{N_w^*}S_w^*. \end{aligned}$$

This means L is C^1 (one time continuously differentiable) on the interior of Ω, ε^* is the global minimum of L on Ω and $L(S_c^*, I_c^*, S_v^*, I_v^*, S_w^*, I_w^*) = 0$.
The derivative of L computed along the solutions of (1) is given by

$$\begin{aligned} \frac{dL}{dt} =& m^*S_c^*\left\{1-\frac{S_c^*}{S_c}\right\}\frac{dS_c}{dt} + m^*S_c^*\left\{1-\frac{I_c^*}{I_c}\right\}\frac{dI_c}{dt} + n^*S_v^*\left\{1-\frac{S_v^*}{S_v}\right\}\frac{dS_v}{dt} \\ &+ n^*S_v^*\left\{1-\frac{I_v^*}{I_v}\right\}\frac{dI_v}{dt} + p^*S_w^*\left\{1-\frac{S_w^*}{S_w}\right\}\frac{dS_w}{dt} + p^*S_w^*\left\{1-\frac{I_w^*}{I_w}\right\}\frac{dI_w}{dt} \end{aligned} \tag{19}$$

where $m^* = \alpha_1\tau_1\varepsilon\frac{I_v^*}{N_c^*}$, $m = \alpha_1\tau_1\varepsilon\frac{I_v}{N_c}$, $n^* = \alpha_1\tau_2\frac{I_c^*}{N_c^*} + \alpha_2\tau_3\frac{I_w^*}{N_w^*}$, $n = \alpha_1\tau_2\frac{I_c}{N_c} + \alpha_2\tau_3\frac{I_w}{N_w}$, $p^* = \alpha_2\tau_4\varepsilon\frac{I_v^*}{N_w^*}$ and $p = \alpha_2\tau_4\varepsilon\frac{I_v}{N_w}$.
Substituting for the expressions of $\frac{dS_c}{dt}$, $\frac{dI_c}{dt}$, $\frac{dS_v}{dt}$, $\frac{dI_v}{dt}$, $\frac{dS_w}{dt}$ and $\frac{dI_w}{dt}$ in (19) and simplifying we get

$$\begin{aligned} \frac{dL}{dt} =& -\mu_c m^*\frac{S_c^*}{S_c}\{S_c^* - S_c\}^2 - mm^*S_c^{*2}\left\{\frac{S_c}{S_c^*}\frac{I_c^*}{I_c} - 1\right\} - m^{*2}S_c^{*2}\left\{\frac{S_c^*}{S_c}\right. \\ &\left. + \frac{I_c}{I_c^*} - 2\right\} - n^{*2}\frac{S_v^*}{I_v^*}\frac{S_v^*}{S_v}\{S_v^* - S_v\}^2 - nn^*S_v^{*2}\left\{\frac{S_v}{S_v^*}\frac{I_v^*}{I_v} - 1\right\} - \\ &n^{*2}S_v^{*2}\left\{\frac{S_v^*}{S_v} - 1\right\} - p^{*2}\frac{S_w^*}{S_w}\frac{S_w^*}{I_w^*}\{S_w^* - S_w\}^2 - p^{*2}S_w^{*2}\left\{\frac{S_w^*}{S_w}\right. \\ &\left. + \frac{I_w}{I_w^*} - 2\right\} - pp^*S_w^{*2}\left\{\frac{I_w^*}{I_w}\frac{S_w}{S_w^*} - 1\right\} \end{aligned} \tag{20}$$

Since the arithmetic mean is greater than or equal to the geometric mean we have: $\frac{S_c}{S_c^*}\frac{I_c^*}{I_c} \geq 1$, $\frac{S_c^*}{S_c} + \frac{I_c}{I_c^*} \geq 2$, $\frac{S_v}{S_v^*}\frac{I_v^*}{I_v} \geq 1$, $\frac{S_v^*}{S_v} \geq 1$, $\frac{S_w^*}{S_w} + \frac{I_w}{I_w^*} \geq 2$, $\frac{I_w^*}{I_w}\frac{S_w}{S_w^*} \geq 1$, $\forall S_c, I_c, S_v, I_v, S_w, I_w \geq 0$.

Clearly from (20), $\frac{dL}{dt} \leq 0$ always holds except at the steady state ε^*, the endemic equilibrium of the system (2). Furthermore $\frac{dL}{dt} = 0$ if and only if

$S_c = S_c^*$, $I_c = I_c^*$, $S_v = S_v^*$, $I_v = I_v^*$, $S_w = S_w^*$ and $I_w = I_w^*$. By LaSalle's invariant principle, ε^* is globally asymptotically stable in Ω. This completes the proof of Theorem (3.6). □

From the analysis of the model (1), it is clear that the dynamics of trypanosomiasis in a cattle population is determined by R_0. However, since R_0 is a function of a number of disease and population parameters, a sensitivity analysis of these parameter value will aid decision making especially on which parameters to monitor in order to control the disease.

4 Sensitivity analysis of R_0

Whereas R_0 reports a single summary outcome, the actual number of infected(s) will depend on the level of confidence or uncertainty in the various parameters that define R_0. Sensitivity analysis of R_0 allows the evaluation of its estimate to the uncertainty in estimating the values of each of its input parameters. A sensitivity index gives the ratio of the change of the output to change in input while other parameters remain constant [24]. When an explicit algebraic equation describes the relationship between the independent variable and the dependent variable, the sensitivity index $\Upsilon_i^{R_0}$ for a particular independent variable can be calculated from the partial derivative of the dependent variable, i.e.

$$\Upsilon_{\alpha_1}^{R_0} = \frac{\partial R_0}{\partial \alpha_1}\frac{\alpha_1}{R_0},$$

where the quotient, $\dfrac{\alpha_1}{R_0}$, is introduced to normalize the index by removing the effects of units [**?**].
The normalized sensitivity indices for the thirteen model parameters are given by $\Upsilon_{\alpha_2}^{R_0} = \frac{\eta_1}{R_0^2\gamma_2}$, $\Upsilon_{\alpha_1}^{R_0} = \frac{\eta_2}{R_0^2\gamma_1}$, $\Upsilon_{\tau_4}^{R_0} = \frac{1}{2}\frac{\eta_1}{R_0^2\gamma_2}$, $\Upsilon_{\tau_3}^{R_0} = \frac{1}{2}\frac{\eta_1}{R_0^2\gamma_2}$, $\Upsilon_{\tau_2}^{R_0} = \frac{1}{2}\frac{\eta_2}{R_0^2\gamma_1}$, $\Upsilon_{\tau_1}^{R_0} = \frac{1}{2}\frac{\eta_2}{R_0^2\gamma_1}$, $\Upsilon_{\varepsilon}^{R_0} = \frac{1}{2}$, $\Upsilon_{h}^{R_0} = \frac{1}{2}\frac{\eta_2}{R_0^2\gamma_1}$, $\Upsilon_{\rho}^{R_0} = \frac{1}{2}\frac{\eta_1}{R_0^2\gamma_2}$, $\Upsilon_{\mu_v}^{R_0} = -\frac{1}{2}$, $\Upsilon_{\mu_w}^{R_0} = -\frac{1}{2}\frac{\eta_1}{R_0^2\gamma_2}$, $\Upsilon_{\kappa}^{R_0} = -\frac{1}{2}\frac{\eta_2\kappa}{R_0^2 m}$ and $\Upsilon_{\mu_c}^{R_0} = -\frac{1}{2}\frac{\eta_2\mu_c}{R_0^2 m}$. where R_0 is (7) and

$$\eta_1 = \alpha_2^2\tau_4\tau_3\varepsilon\rho, \quad \eta_2 = \alpha_1^2\tau_2\tau_1\varepsilon h, \quad \gamma_1 = \mu_v(\mu_c+\kappa), \quad \gamma_2 = \mu_v\mu_w,$$
$$m = \mu_v(\mu_c+\kappa)^2.$$

All the sensitivity indices are positive except $\Upsilon_{\mu_v}^{R_0}$, $\Upsilon_{\mu_w}^{R_0}$, $\Upsilon_{\mu_c}^{R_0}$ and $\Upsilon_{\kappa}^{R_0}$. The natural death rate of the vectors, the natural death rate of the wild animals and the natural and disease-induced death rate in cattle that have the effect of reducing R_0. Further, all are functions of the parameter values except $\Upsilon_{\varepsilon}^{R_0}$ and $\Upsilon_{\mu_v}^{R_0}$ and will change as the parameter values change. The parameter values from previous studies are used to determine how sensitive parameter values are.

Parameter	**Description**	**Value**	**Ref**
α_1	Rate at which tsetse fly bite cattle	0.032	[19]
α_2	Rate at which tsetse fly bite wild animals	0.97	[19]
μ_c	Natural mortality rate of cattle (inc. slaughter)$days^{-1}$	0.00055	[25]
μ_v	Natural mortality rate of vector $days^{-1}$	0.97	[5]
μ_w	Natural mortality rate of wild animals $days^{-1}$	0.0006	[19]
κ	Disease-induced death rate in cattle $days^{-1}$	0.006	[20]
τ_1	Transmission probability from vector to cattle	0.62	[5]
τ_2	Transmission probability from cattle to vector	0.7	[25]
τ_3	Transmission probability from wild animal to vector	0.05	[19]
τ_4	Transmission probability from vector to wild animal	0.2	[19]
ε	Survival rate of vector	0.5	*Estimated*
ρ	Ratio of susceptible vectors to cattle population	76	[19]
h	Ratio of susceptible vectors to wild animal population	76	[19]
Λ_c	Cattle recruitment rate	22.0	[25]
Λ_v	Tsetse recruitment rate	24.0	*Estimated*
Λ_w	Wild animal recruitment rate	27.5	*Estimated*

Table 1: Parameter values from previous studies

Using the parameter values in Table 1, the sensitivity indices are provided in Table 2. The magnitude of the sensitivity indices indicates the more sensitive parameter in R_0, while the sign of the sensitivity index of R_0 indicates whether R_0 increases (+) or decreases (−) when a parameter increases, the rest of the parameters being constant [14].
The most sensitive parameter, the one with the highest influence on R_0, is the rate at which the vectors bite the wild animal population, α_2. Decreasing (or increasing) α_2 by 10% decreases (or increases) R_0 by 9.997%. Other important parameters include the vector survival rate ε and the vector death rate μ_v .

5 Discussion

From the analysis, the disease can invade into the susceptible cattle population and a unique endemic state exists if $R_0 > 1$, whereas the disease dies out when $R_0 < 1$. However, R_0 is a function of various disease and population parameters. Controlling the disease suggests reducing R_0 to less than 1. Targeting the parameters which have a high influence on R_0, the vector biting rate on the wild animal population, the survival rate of the vectors and vector death rate would provide suggestions on control.
The vector death rate has the highest effect of reducing R_0 as it increases. Obviously, with no vectors to transmit the disease, the disease will be eradicated from the population. The wild animal population are tolerant to and provide

Parameter	Sensitivity Index
α_2	+0.99569
ε	+0.50000
μ_v	- 0.50000
τ_4	+0.49785
τ_3	+0.49785
ρ	+0.49785
μ_w	- 0.49785
α_1	+0.00431
τ_2	+0.00215
τ_1	+0.00215
h	+0.00215
κ	- 0.00197
μ_c	- 0.00018

Table 2: Sensitivity indices of parameter values in R_0

a reservoir to the trypanosome so that vectors that contact the them are more likely to be infected. They accelerate the disease in a cattle population. Just as in [19], a close monitoring of the disease dynamics in the wild animal population would suggest methods of disease control in the cattle population.
The vector survival rate if increased, increases endemicity of the disease. The cyclic transmission of the disease implies that once the tsetse fly become infected, it remains infective for a long period [18]. The longer the vector survives, the more cattle one would expect it to infect. Figures 1 and 2 show the number of infected cattle for varying values of α_2 and ε. The parameters that drive the disease in a cattle population if targeted would greatly enhance disease control. in this particular case, control strategies that minimize contact between the vector and the cattle populations would be a way of eradicating the disease.
The particular values of the sensitivity indices of the reproduction number, R_0, to the different parameters depend on the parameter values provided in Table 2 and on the assumptions on which the model (1) is made. To effectively guide policy, the model and parameter values would need to be tested against recent data from trypanosomiasis-endemic regions. The current analysis however remains an important step in simplifying the study of the general trypanosomiasis disease dynamics in a given cattle population.

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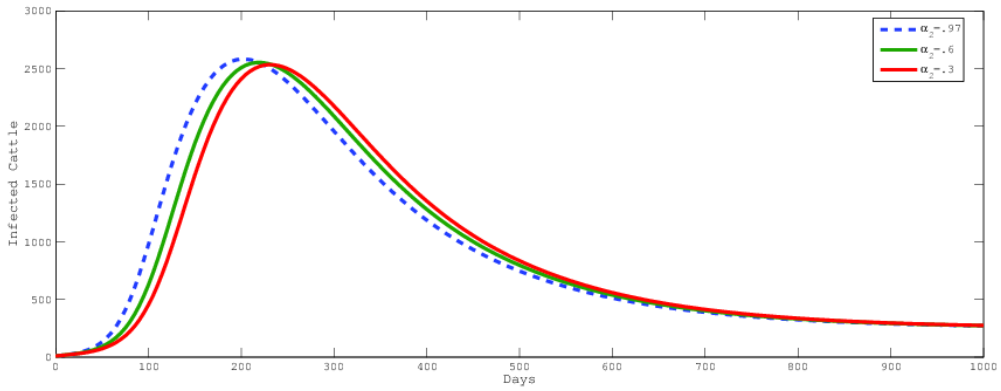

Figure 1: Infected cattle population at various vector wild animal biting rate

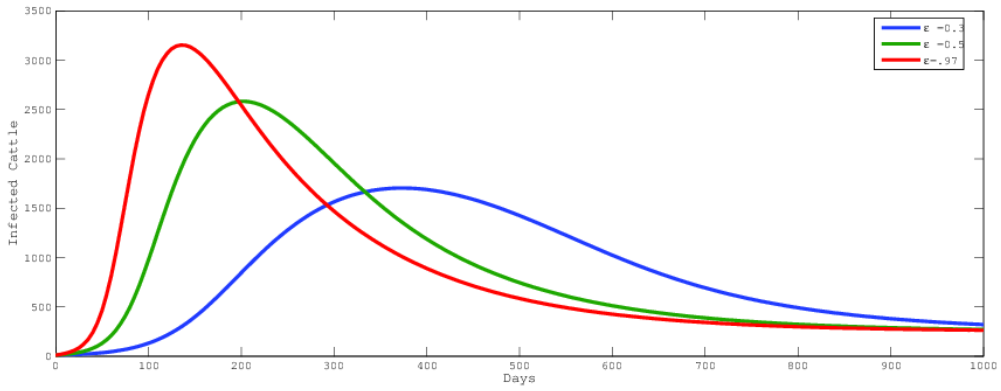

Figure 2: Infected cattle population at various vector survival rate