Mathematical Modelling of African Animal Trypanosomiasis Incorporating Spraying of Vector Population and Treating the Host Population

Abstract

In this paper, we have formulated a mathematical model for the transmission dynamics of African Animal Trypanosomiasis (AAT) by incorporating spraying of the tsetse fly population(vector) and treating the cattle population (host) as control strategies. It has been shown that the disease free equilibrium point is globally asymptotically stable and the endemic equilibrium point is locally asymptotically stable. We have also shown that treatment of the host population reduces significantly infection by AAT as compared to spraying of the vector population.

1 Introduction

African Animal Trypanosomiasis (AAT) is a well known disease caused by tsetse fly (genus: Glossina), which carry and transmit different species of trypanosomes such as Trypanosoma Vivax, T.Congolence, T. brucei, T. theileri and T. evansi which infect domestic as well as wild animal, see [8, 15]. It is known as sleeping sickness among humans and nagana among livestock population [3]. The disease has adverse effects both on human and livestock populations. AAT is a menace in countries of the Sub - Saharan Africa, limiting agricultural production in these regions causing serious food shortages. It is estimated that more than 50 million cattle are at the risk of getting the disease. In Kenya for example, AAT is prevalent in Western Kenya, Rift Valley and Coastal regions causing huge economic losses in the affected regions. This has led to decrease in livestock population especially in the rural areas [15, 17].

AAT spreads in a cattle population when an infected tsetse fly feeds on the blood of susceptible cattle and when a susceptible tsetse fly feeds on the blood of an infected cattle. During trypanosome interchange, the fly picks the trypanosome from the infected cattle and transfers it to another cattle and the cycle continues enhancing the disease transmission cycle. The cattle-tsetse fly-cattle cycle increases when cattle are introduced in grazing grounds, water points and other places occupied by Glossina pallidipes (the species that transmit AAT),see [1, 4].

Tsetse fly eradication schemes are pegged on climatic patterns of the region. During hot seasons, tsetse fly mortality rate is high and as a result, transmission rate is low. However, during cold seasons, the birth rate is high and the population hit maximum thus increasing the transmission of AAT, see [1, 3, 10]. This information is used by stakeholders to decide the period of the month to launch tsetse fly eradication campaigns. Tsetse fly eradication schemes include vector control through Aerial spraying, ground spraying, use of treated cloths (traps), sterile insect technique and clearing the surrounding environment [12, 15] and injecting a good number of susceptible cattle with anti-parasitic drugs and use

of trypanocidal drugs on infected cattle (such as Diminazene aceturate and isometamidium chloride).

Due to the destructive nature of AAT, a multi-dimesional approach is applied involving stakeholders. This has led to the establishment of Pan Africa Tsetse and Trypanosomiasis Eradication Campaign (PATTEC) [18] and other initiatives by governments.

Many studies have been conducted on the control of AAT. Ng'wena *et al* [12] assessed the potential re-emergence of AAT in both cattle and human population in Lambwe valley amid robust use of vector control measures. They employed clinical and veterinary approaches by testing blood samples of both animals and humans to check for traces of trypanosomes in their blood. They found that cases of African Trypanosomiasis are still registered in both cattle and human populations despite the presence of various control strategies in the Lambwe region.

Muriuki *et al* [11] applied time-series aerial photograph interpretation, social survey methods, and a review of human population to study tsetse fly control and the effects of land use in Lambwe valley. The result of their study showed a tremendous increase in land use leading to destruction of tsetse fly habitat. The study found that application of aerial insecticide, bush clearing using herbicides and ground spraying to destroy tsetse habitat, use of insecticide-treated cloths (targets), have been used as control measures. Kajunguri [8] developed a multi-host model to study the control of tsetse fly and Trypanosoma brucei rhodesiense (TBR) in Southeastern Uganda by incorporating insecticide treated cattle as a control strategy of Human African Trypanosomiasis(HAT) on humans. The study showed that the effective application of insecticides is cost effective and environmentally friendly. Milligan and Baker [9] modelled the effects of chemoprophylaxis on cattle and vector control immigration. They observed that in order to achieve disease reduction through chemoprophylaxis, mass treatment should be prioritised and the efficacy of the drug should also be taken into consideration.

Joyce [16] studied optimal control of trypanosomiasis in cattle population by using treatment of infected cattle as a control strategy. It was observed that treating $75\% - 90\%$ of infected cattle is sufficient in controlling AAT. This study did not consider the combined effect of treatment of host and spraying of the vector in the control of AAT. Inertia [6] modelled a Multi- Drug Resistance(MDR) during chemotherapy animal african trypasomiasis in Kwale, Kenya to predict the most effective use of trypanocides in controlling AAT. The study revealed that treatment of cattle with a combination of two trypanocides was the most optimal treatment strategy to restrict development of MDR to AAT.

Most studies on control of AAT have used clinical and veterinary approaches relying heavily on the outcome of the experiments and focussing on the treatment of cattle. In this study, we have formulated a mathematical model of AAT transmission, incorporating treatment of cattle as well as spraying of the vector population using insecticides in order to determine the combined effect of spraying the vector and treatment of cattle on the control of AAT.

2 Model Formulation

In this study, we formulate and analyze a host and vector mathematical model describing the transmission of Animal African Trypanosomiasis in cattle. The total cattle population at any time t denoted by $N_c(t)$ is divided into susceptible cattle, $S_c(t)$, Treated cattle, $E_c(t)$, Infectious cattle, $I_c(t)$ and Recovered cattle, $R_c(t)$.
The tsetse fly population at any time t denoted by $N_v(t)$ is divided into susceptible tsetse fly, $S_v(t)$, sprayed tsetse fly, $E_v(t)$ and infectious tsetse fly, $I_v(t)$.
The cattle population is determined by cattle natural birth rate λ. The cattle population is then partitioned into those kept under treatment with Trypanocides and those under no treatment. The proportion of cattle population under treatment is α. Thus, $1-\alpha$ is the proportion of cattle population under no treatment. The recruitment into the susceptible class is at the rate of $(1-\alpha)\lambda$ while recruitment into the treated class is at the rate of $\alpha\lambda$. The susceptible cattle population can transit to the treated class at the rate of τ. The treatment can either be successful, in which case the cattle develops immunity against Trypanosomes or the treatment can fail, in which case the cattle can be infected. The failure rate of treatment is given by σ. The treated cattle transit to the infected compartment at the rate of $\sigma\beta$, where β is the force of infection in cattle given by $\beta = \eta p$ in which η is the average biting rate of tsetse fly and p is the transmission rate of infection from tsetse fly to cattle. The susceptible cattle transit to the infected compartment at the rate β. The infected cattle can recover from the disease after treatment at the rate of ω. The recovered cattle loses immunity and become susceptible at the rate of γ.

The vector population is replenished at the rate of π. The tsetse fly population is then partitioned into those being sprayed with insecticide and those that are not sprayed. The proportion of the tsetse fly population being sprayed is κ. Thus, recruitment into the susceptible tsetse fly population is $(1-\kappa)\pi$ and recruitment into the sprayed tsetse fly population is $\kappa\pi$. The susceptible tsetse fly population are sprayed with insecticide at the rate of ϑ. The spraying can either be successful in which case the tsetse fly die due to spraying at the rate of ξ or the spraying can fail in which case the tsetse fly can become infectious. The force of infection in tsetse fly is given by $\varphi = \eta\rho$ where ρ is the transmission rate of infection from cattle to tsetse fly. The rate of spraying tsetse fly population fails at the rate of ϵ. The sprayed tsetse fly population transit to the infectious compartment at the rate of $\epsilon\varphi$. The cattle and the vector are reduced by natural death rates of μ and ν respectively. The infected cattle also suffer an additional death rate of δ due to the disease. The table below gives a summary of the variables and parameters of the model.

Table 1: *A table showing a list of variables and parameters defined and their corresponding meanings*

Variable/Parameter	Description
S_c	Susceptible cattle.
E_c	Population of cattle kept on drugs .
I_c	Infectious cattle population
R_c	Removed cattle population due to treatment.
S_v	Susceptible vector population .
E_v	Population of Vector kept under control through spraying
I_v	Infectious tsetse flies.
N_c	Cattle total population.
N_v	Tsetse total population.
μ	Natural death rate of cattle.
ν	Natural death rate of tsetse fly
λ	Rate at which cattle population are replenished.
π	Rate at which vector population are replenished.
ξ	Tsetse fly death rate due to spraying.
ϵ	Failure rate of spraying
β	Force of infection in Cattle.
δ	Disease-induced death rate in cattle.
φ	Force of infection in tsetse fly
γ	Rate at which recovered cattle transit to susceptible
α	Proportion of cattle kept under treatment with Trypanocides.
ϑ	rate of spraying susceptible tsetse fly.
p	Transmission rate of infection from cattle to tsetse fly.
ρ	Transmission rate of infection from tsetse fly to cattle
η	Average biting rate of tsetse fly.
τ	Rate of treating susceptible cattle with anti-parasitic drugs.
ω	Recovery rate of infected cattle.
κ	Proportion of tsetse fly kept under spraying.
σ	Failure rate of treatment

The data used in this study are estimated and some taken from existing literature. The following assumptions are made:

1. State variables and parameters of the model are positive real values
2. There is no AAT related mortalities in tsetse fly, however, tsetse fly can die as a result of a natural cause and spraying
3. The recruitment rates are limited to only births in both cattle and tsetse fly populations, making all newborns susceptible to the disease.
4. No transmission of the disease by other biting flies except tsetse fly, hence the transfer of Trypanosomes is done by only a single species of the tsetse fly and assumed to be the one causing infection
5. No mortalities arise from other infectious diseases among the susceptible cattle population

6. There is a constant population for both the host and the vector
7. Tsetse fly cannot recover from the disease and infected tsetse fly remain infectious throughout the rest of its life.

The following figure gives the schematic diagram of the model.

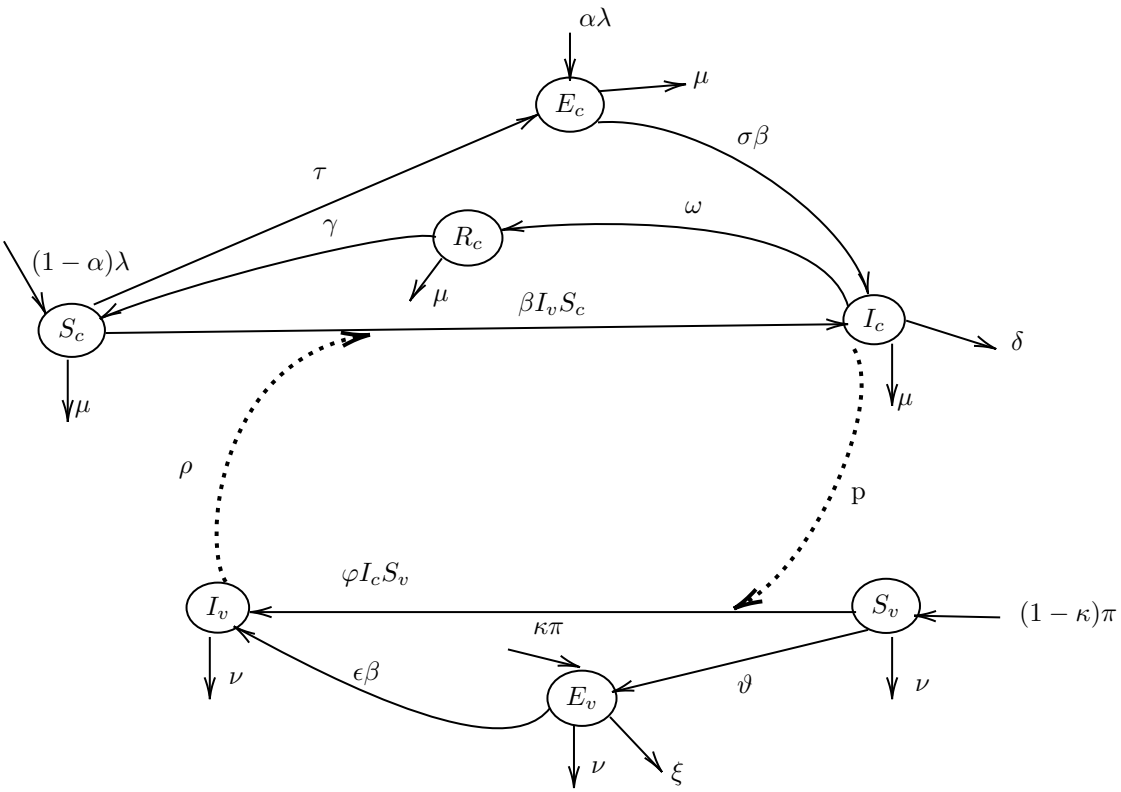

Figure 1: *Compartmental diagram showing cattle and vector populations together with AAT dynamics and the transition between compartments. Solid lines indicate the movement of cattle and vector from one compartment to another while the dashed lines indicate infection pathways*

The governing equation from the flow chart in figure 1 is given by;

$$\begin{aligned}
\frac{dS_c}{dt} &= (1-\alpha)\lambda - (\beta I_v + b_1)S_c + \gamma R_c, \\
\frac{dE_c}{dt} &= \alpha\lambda + \tau S_c - b_2 E_c, \\
\frac{dI_c}{dt} &= \beta I_v S_c + \sigma\beta E_c - b_3 I_c, \\
\frac{dR_c}{dt} &= \omega I_c - b_4 R_c, \\
\frac{dS_v}{dt} &= (1-\kappa)\pi - (\varphi I_c + b_5)S_v, \\
\frac{dE_v}{dt} &= \kappa\pi + \vartheta S_v - b_6 E_v, \\
\frac{dI_v}{dt} &= \varphi I_c S_v + \epsilon\varphi E_v - \nu I_v
\end{aligned} \tag{2.1}$$

Where $b_1 = \mu+\tau, b_2 = \sigma\beta+\mu, b_3 = \mu+\delta+\omega, b_4 = \gamma+\mu, b_5 = \vartheta+\nu, b_6 = \epsilon\varphi+\xi+\nu$
System (2.1) is appended with the initial conditions

$$S_c(0) \geq 0,\ E_c(0) \geq 0,\ I_c(0) \geq 0,\ R_c(0) \geq 0,\ S_v(0) \geq 0,\ E_v(0) \geq 0,\ I_v(0) \geq 0 \tag{2.2}$$

Adding all equations in (2.1), we obtain;

$$\frac{dN_c}{dt} = \lambda - \delta I_c - \mu N_c \quad \text{and} \quad \frac{dN_v}{dt} = \pi - \nu N_v - \xi E_v \tag{2.3}$$

Where, $N_c = S_c + E_c + I_c + R_c$ and $N_v = S_v + E_v + I_v$

2.1 Positivity and Boundedness of the solutions

In this section, we shall show that the solutions of system (2.1) are positive and bounded.

Theorem 2.1. *Suppose that conditions in (2.2) hold, then the solution of system (2.1) will remain non-negative at all time t.*

Proof. From the first equation in system (2.1), we have

$$\frac{dS_c}{dt} \geq -(\beta I_v + b_1)S_c$$

Separating the variables and integrating, we obtain

$$S_c(t) = S_c(0)e^{-\int_0^t (\beta I_v(s)+b_1)ds} \geq 0\ \forall t > 0 \tag{2.4}$$

Similarly, it can easily be shown that $E_c(t) \geq 0$, $I_c(t) \geq 0$, $R_c(t) \geq 0$, $S_v(t) \geq 0$, $E_v(t) \geq 0$, $I_v(t) \geq 0$. Thus, the solutions remain non-negative for all $t \geq 0$. □

Theorem 2.2. *The set* $\Omega = \{(S_c, E_c, I_c, R_c, S_v, E_v, I_v) \in \Re_+^7 : N_c \leq \frac{\lambda}{\mu}, N_v \leq \frac{\pi}{\nu}\}$ *is positively invariant.*

Proof. Let $S_c(t), E_c(t), I_c(t), R_c(t), S_v(t), E_v(t), I_v(t)$ be solutions of system (2.1) satisfying conditions in (2.2). Then, from (2.3), we have that

$$\frac{dN_c(t)}{dt} \leq \lambda - \mu N_c \quad \text{and} \quad \frac{dN_v(t)}{dt} \leq \pi - \nu N_v$$

from which we obtain

$$\begin{aligned} N_c(t) &\leq \frac{\lambda}{\mu} + \left(N_c(0) - \frac{\lambda}{\mu}\right) e^{-\mu t} \quad \text{and} \\ N_v(t) &\leq \frac{\pi}{\nu} + \left(N_v(0) - \frac{\pi}{\nu}\right) e^{-\nu t} \end{aligned} \tag{2.5}$$

It follows that

$$\limsup_{t\to\infty} N_c \leq \frac{\lambda}{\mu} \quad \text{and} \quad \limsup_{t\to\infty} N_v \leq \frac{\pi}{\nu}$$

Hence, N_c and N_v are bounded and all feasible solution sets of the system approach or stay in Ω. The region Ω is therefore positively invariant. □

2.2 Model equilibria and stability Analysis

The computation of the model equilibria, the basic reproduction number R_{eff} and the stability analysis of the equilibria points are done in this section.

2.2.1 Disease-Free Equilibrium(DFE) Point

Equating the right hand side of (2.1) to zero with $I_c = I_v = 0$ we obtain the DFE point E^0 as

$$\begin{aligned} E^0 &= (S_c^0,\ E_c^0,\ I_c^0,\ R_c^0,\ S_v^0,\ E_v^0,\ I_v^0) \\ &= \left(\frac{(1-\alpha)\lambda}{b_1}, \frac{\alpha\lambda + \tau S_c^0}{b_2}, 0, 0, \frac{(1-\kappa)\pi}{b_5}, \frac{\kappa\pi + \vartheta S_v^0}{b_6}, 0\right) \end{aligned} \tag{2.6}$$

2.2.2 Basic Reproduction Number

The basic reproduction number, R_0 is an essential epidemiological parameter defined as the number of secondary infections caused by one infected host or vector in a population where everyone is susceptible [19]. Using the method in [19], the effective reproduction number, R_{eff}, is given by.

$$R_{eff} = \sqrt{\left(\frac{\beta S_c^0}{b_3}\right)\left(\frac{\varphi S_v^0}{\nu}\right)}, \tag{2.7}$$

where S_c^0 and S_v^0 are as given in equation (2.6).

2.2.3 Stability of the Disease Free Equilibrium (DFE) Point E^0

In this section, we analyse the local and global stability of the DFE point E^0 for our model.

Theorem 2.3. *The DFE point E^0 for the system in (2.1) is locally asymptotically stable if $R_{eff} < 1$ and if $\beta\varphi b_4 S_c^0 S_v^0 > \gamma\nu\omega$*

Proof. We begin by finding the Jacobian matrix, $J(E^0)$ of the system at the DFE point E^0

$$J(E^0) = \begin{pmatrix} -b_1 & 0 & 0 & \gamma & 0 & 0 & -\beta S_c^0 \\ \tau & -b_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma\beta & -b_3 & 0 & 0 & 0 & \beta S_c^0 \\ 0 & 0 & \omega & -b_4 & 0 & 0 & 0 \\ 0 & 0 & -\varphi S_v^0 & 0 & -b_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \vartheta & -b_6 & 0 \\ 0 & 0 & \varphi S_v^0 & 0 & 0 & \epsilon\varphi & -\nu \end{pmatrix}$$

The eigenvalues of $J(E^0)$ are obtained using the characteristic equation

$$det(J(E^0) - zI) = 0$$

where z is a spectral parameter.
The Characteristic equation yields;

$$z^7 + a_6 z^6 + a_5 z^5 + a_4 z^4 + a_3 z^3 + a_2 z^2 + a_1 z + a_0 = 0 \tag{2.8}$$

Where

$$\begin{aligned}
a_6 &= b_1 + b_2 + b_3 + b_4 + b_5 + b_6 + \nu, \\
a_5 &= b_1(b_2 + b_3 + b_4 + b_5 + b_6 + \nu) + b_2(b_3 + b_4 + b_5 + b_6 + \nu) + \\
&\quad b_3(b_4 + b_5 + b_6) + b_4(b_5 + b_6 + \nu) + b_5(b_6 + \nu) + b_6\nu + b_3\nu(1 - R_{eff}^2) \\
a_4 &= b_3\nu(b_1 + b_2 + b_4 + b_5 + b_6)(1 - R_{eff}^2) + b_3(b_1 + b_2)(b_4 + b_5 + b_6) + \\
&\quad b_4(b_1 + b_2)(b_5 + b_6 + \nu) + b_5(b_1 + b_2 + b_4)(b_6 + \nu) + \nu b_6(b_1 + b_2 + b_4 + b_5) \\
&\quad + b_1 b_2(b_3 + b_4 + b_5 + b_6 + \nu) + b_3 b_4(b_5 + b_6) + b_3 b_5 b_6 \\
a_3 &= \nu b_3[b_1(b_2 + b_4 + b_5 + b_6) + b_2(b_4 + b_5 + b_6) + b_4(b_5 + b_6) + b_5 b_6][1 - R_{eff}^2] \\
&\quad + \sigma\beta\tau(\beta\varphi S_c^0 S_v^0 - \gamma\omega) + \beta\varphi^2\epsilon\vartheta S_c^0 S_v^0 + b_5(b_6 + \nu)(b_1 b_2 + b_1 b_4 + b_2 b_4) + \\
&\quad b_3 b_4(b_5 + b_6)(b_1 + b_2) + b_3 b_5 b_6(b_1 + b_2) + \nu b_6(b_1 + b_2)(b_4 + b_5) + \\
&\quad b_1 b_2 b_3(b_4 + b_5 + b_6) + b_1 b_2 b_4(b_5 + b_6 + \nu) + \nu b_1 b_2 b_6 + b_4 b_5 b_6(b_3 + \nu) \\
a_2 &= \nu b_3[b_1 b_2(b_4 + b_5 + b_6) + b_4(b_1 + b_2)(b_5 + b_6) + b_5 b_6(b_1 + b_2 + b_4)][1 - R_{eff}^2] \\
&\quad + b_1 b_2 b_4(b_3 + \nu)(b_5 + b_6) + b_1 b_2 b_5 b_6(b_3 + b_4) + b_3 b_4 b_5 b_6(b_1 + b_2) + \\
&\quad \nu b_1 b_5 b_6(b_2 + b_4) + \nu b_2 b_4 b_5 b_6 + \beta\varphi^2\epsilon\vartheta S_c^0 S_v^0(b_1 + b_2 + b_4) + \\
&\quad \tau\sigma\beta[(b_5 + b_6)(\beta\varphi S_c^0 S_v^0 - \gamma\omega) + (\beta\varphi b_4 S_c^0 S_v^0 - \gamma\nu\omega)] \\
a_1 &= \nu b_3[b_1 b_2 b_4(b_5 + b_6) + b_1 b_5 b_6(b_2 + b_4) + b_2 b_4 b_5 b_6][1 - R_{eff}^2] + \\
&\quad b_1 b_2 b_4 b_5 b_6(b_3 + \nu) + \beta\varphi^2\epsilon\vartheta S_c^0 S_v^0 b_4(b_1 + b_2) + \beta\varphi^2\epsilon\vartheta\mu S_c^0 S_v^0(b_2 + \tau) \\
&\quad + \tau\sigma\beta(\beta\varphi b_4 S_c^0 S_v^0 - \gamma\nu\omega)(b_5 + b_6) + \tau\sigma\beta b_5 b_6(\beta\varphi S_c^0 S_v^0 - \gamma\omega) \\
a_0 &= \nu b_1 b_2 b_3 b_4 b_5 b_6[1 - R_{eff}^2] + \beta\varphi^2\epsilon\vartheta\mu S_c^0 S_v^0 b_4(b_2 + \tau) + \tau\sigma\beta b_5 b_6(\beta\varphi b_4 S_c^0 S_v^0 - \gamma\nu\omega)
\end{aligned}$$

Clearly, the coefficients $a_0, a_1, \ldots, a_6$ are all positive provided $R_{eff} < 1$ and $\beta\varphi b_4 S_c^0 S_v^0 > \gamma\nu\omega$. By the Descartes' Rule of signs, all the roots of equation (2.8) have negative real parts. Therefore, the DFE point E^0 is stable provided $R_{eff} < 1$ and $\beta\varphi b_4 S_c^0 S_v^0 > \gamma\nu\omega$. □

Next, we study the global stability of the DFE point using the Lyapunov function approach for host-vector models as used in [13]

Theorem 2.4. *The DFE point E^0 is globally asymptotically stable (g.a.s) in Ω if $R_{eff} \leq 1$ and $\beta = \varphi = 0$*

Proof. To establish the global stability of the DFE point E^0, we construct the following Lyapunov function

$$\begin{aligned} V(t) &= A_1(S_c - S_c^0 \ln S_c) + A_2(E_c - E_c^0 \ln E_c) + A_3 I_c + A_4 R_c + A_5(S_v - S_v^0 \ln S_v) \\ &\quad + A_6(E_v - E_v^0 \ln E_v) + A_7 I_v \end{aligned} \tag{2.9}$$

where $A_i, i = 1, 2, \ldots 7$ are some positive constants to be chosen later.

Calculating the time derivative along the solutions of (2.1), we obtain

$$\begin{aligned} V'(t) &= A_1\left(\frac{S_c - S_c^0}{S_c}\right) S_c' + A_2\left(\frac{E_c - E_c^0}{E_c}\right) E_c' + A_3 I_c' + A_4 R_c' + A_5\left(\frac{S_v - S_v^0}{S_v}\right) S_v' \\ &\quad + A_6\left(\frac{E_v - E_v^0}{E_v}\right) E_v' + A_7 I_v' \end{aligned} \tag{2.10}$$

Using

$$S_c^0 = \frac{(1-\alpha)\lambda}{b_1}, E_c^0 = \frac{\alpha\lambda + \tau S_c^0}{b_2}, S_v^0 = \frac{(1-\kappa)\pi}{b_5} \text{ and } E_v^0 = \frac{\kappa\pi + \vartheta S_v^0}{b_6}$$

in (2.10), we get

$$\begin{aligned} V'(t) &= -A_1 b_1\left(\frac{(S_c - S_c^0)^2}{S_c}\right) - A_2 b_2\left(\frac{(E_c - E_c^0)^2}{E_c}\right) - A_5 b_5\left(\frac{(S_v - S_v^0)^2}{S_v}\right) \\ &\quad - A_6 b_6\left(\frac{(E_v - E_v^0)^2}{E_v}\right) - A_4 b_4 R_c - \nu A_t I_v - \varphi A_5(S_v - S_v^0) I_c + \\ &\quad \tau A_2 S_c\left(1 - \frac{S_c^0}{S_c}\right)\left(1 - \frac{E_c^0}{E_c}\right) + \vartheta A_6 S_v\left(1 - \frac{S_v^0}{S_v}\right)\left(1 - \frac{E_v^0}{E_v}\right) + \\ &\quad \varphi A_7(I_c S_v + \epsilon E_v) + \beta A_3(I_v S_c + \sigma E_c) + (\omega A_4 - A_3 b_3) I_c \end{aligned} \tag{2.11}$$

Let $A_1 = A_2 = A_5 = A_6 = A_7 = 1, A_3 = \omega\nu, A_4 = \beta\varphi S_c^0 S_v^0$. Equation (2.11) reduces to

$$\begin{aligned} V'(t) &= -A_1 b_1\left(\frac{(S_c - S_c^0)^2}{S_c}\right) - A_2 b_2\left(\frac{(E_c - E_c^0)^2}{E_c}\right) - A_5 b_5\left(\frac{(S_v - S_v^0)^2}{S_v}\right) \\ &\quad - A_6 b_6\left(\frac{(E_v - E_v^0)^2}{E_v}\right) - A_4 b_4 R_c - \nu A_t I_v - \varphi A_5(S_v - S_v^0) I_c + \\ &\quad \tau A_2 S_c\left(1 - \frac{S_c^0}{S_c}\right)\left(1 - \frac{E_c^0}{E_c}\right) + \vartheta A_6 S_v\left(1 - \frac{S_v^0}{S_v}\right)\left(1 - \frac{E_v^0}{E_v}\right) + \\ &\quad \varphi A_7(I_c S_v + \epsilon E_v) + \beta A_3(I_v S_c + \sigma E_c) + \omega\nu b_3(R_{eff}^2 - 1) I_c \end{aligned} \tag{2.12}$$

Using the Arithmetic Mean-Geometric Mean (AM-GM) inequality, we have that $\tau A_2 S_c\left(1 - \frac{S_c^0}{S_c}\right)\left(1 - \frac{E_c^0}{E_c}\right) \leq 0$ and $A_6 S_v\left(1 - \frac{S_v^0}{S_v}\right)\left(1 - \frac{E_v^0}{E_v}\right) \leq 0$

Thus, $V'(t) \leq 0$ provided $R_{eff}^2 \leq 1$ and $\beta = \varphi = 0$. Note that $V'(t) = 0$ holds only for $S_c = S_c^0, S_v = S_v^0, E_c = E_c^0, E_v = E_v^0, I_c = R_c = I_v = 0$ and $\beta = \varphi = 0$. Therefore, the largest compact invariant set in $\{(S_c, E_c, I_c, R_c, S_v, E_v, I_v) \in \Omega | V'(t) = 0\}$ is the singleton set $\{E^0\}$. Hence, Lasalle's Invariant Principle [7] then implies that E^0 is globally asymptotically stable in Ω. □

2.2.4 Endemic Equilibrium(EE)

The endemic equilibrium describes a situation where the disease persists in the cattle population. This is the case where there exists a positive endemic equilibrium point E^*. The following theorem gives conditions for the existence of a unique positive endemic equilibrium point for the system in equation (2.1).

Theorem 2.5. *There exists a unique positive endemic equilibrium point E^* if $R_{eff} \geq 1$, $\beta\gamma\omega c_2 > 2b_3c_4$ and $\beta\varphi(1-\alpha)\lambda > b_3b_5$.*

Proof. Let $E^* = (S_c^*, E_c^*, I_c^*, R_c^*, S_v^*, E_v^*, I_v^*)$ be the endemic equilibrium point of system (2.1). This point is obtained by equating the right hand side of system (2.1) to zero and solving for $S_c^*, E_c^*, I_c^*, R_c^*, S_v^*, E_v^*,$ *and* I_v^*. We obtain

$$\begin{aligned}
S_c^* &= \frac{\nu\gamma\omega\varphi b_6 I_c^{*2} + \nu[b_4(1-\alpha)\lambda\varphi + \gamma\omega b_5]b_6 I_c^* + \nu(1-\alpha)\lambda b_4 b_5 b_6}{c_3 I_c^* + c_4} \\
E_c^* &= \frac{1}{b_2(c_3 I_c^* + c_4)}\big\{\nu\varphi\tau\gamma\omega I_c^{*2} + \\
&\quad [\alpha\lambda\beta b_4 c_1 + \alpha\lambda\varphi b_1 b_4 b_6 + (1-\alpha)\lambda\tau\varphi^2\nu b_4 b_6 + \nu\tau\gamma\omega b_5 b_6]I_c^* \\
&\quad + \alpha\lambda\beta b_4 c_1 + \nu b_4 b_5 b_6[\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda]\big\} \\
R_c^* &= \frac{\omega}{b_4} I_c^*, \quad S_v^* = \frac{(1-\kappa)\pi}{\varphi I_c^* + b_5}, \quad E_v^* = \frac{\kappa\pi b_5 + \vartheta(1-\kappa)\pi + \kappa\pi\varphi I_c^*}{b_6(\varphi I_c^* + b_5)} \\
I_v^* &= \frac{c_1 I_c^* + c_2}{\nu b_6(\varphi I_c^* + b_5)}
\end{aligned}$$

where

$$\begin{aligned}
c_1 &= \varphi(1-\kappa)\pi b_6 + \kappa\pi\epsilon\varphi^2, \qquad c_2 = \kappa\pi\epsilon\varphi b_5 + \vartheta(1-\kappa)\pi\epsilon\varphi \\
c_3 &= \beta b_4 c_1 + \nu\varphi b_1 b_4 b_6, \qquad c_4 = \beta b_4 c_2 + \nu b_1 b_4 b_5 b_6
\end{aligned}$$

Using these expressions in

$$\beta I_v^* S_c^* + \sigma\beta E_c^* - b_3 I_c^* = 0,$$

we obtain after a lengthy computation

$$d_4 I_c^{*4} + d_3 I_c^{*3} + d_2 I_c^{*2} + d_1 I_c^* + d_0 = 0 \tag{2.13}$$

where

$$
\begin{aligned}
d_4 &= -\nu\varphi b_6 c_3\{\beta b_2 c_1[\mu b_3 + (\mu+\delta)\gamma] + \\
&\quad \nu\varphi b_6[b_1 b_2(\mu b_3 + (\mu+\delta)\gamma) + \mu\gamma\omega(b_1+\sigma\beta)]\} \\
d_3 &= \beta\nu\gamma\varphi b_2 b_6 + b_2 c_3[\beta(1-\alpha)\lambda\varphi b_4 c_1 + b_5(\beta\gamma\omega c_1 - b_3 c_3)] + \\
&\quad \sigma\beta\nu b_6[\varphi^2\nu\tau\gamma\omega b_6 c_4 + \nu\varphi\tau\gamma\omega b_5 b_6 c_3] + \nu\varphi b_2 b_6 c_4[\beta\gamma\omega c_1 - 2b_3 c_3] + \\
&\quad \sigma\beta\nu\varphi b_6 c_3[\alpha\lambda\beta b_4 c_1 + \nu\varphi b_4 b_6(\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda) + \nu\tau\gamma\omega b_5 b_6] \\
d_2 &= \nu(1-\alpha)\lambda\beta b_2 b_4 b_5 b_6 c_1 c_3 + \beta\varphi b_4 c_2[\nu\gamma\omega\beta b_2 b_6 c_2 + \nu\gamma\omega b_1 b_2 b_5 b_6^2 - b_3] + \\
&\quad \nu^2\varphi\tau\gamma\omega\sigma b_5 b_6^2 c_4 + \nu\sigma\beta\varphi b_6 c_3[\alpha\lambda\beta b_4 c_2 + \nu b_4 b_5 b_6(\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda)] + \\
&\quad \nu\sigma\beta b_6[\alpha\lambda b_4 c_1 + \nu\varphi b_5 b_6(\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda) + \nu\tau\gamma\omega b_5 b_6][\varphi c_4 + b_5 c_3] + \\
&\quad \beta[(1-\alpha)\lambda\varphi^3\kappa\pi\epsilon b_4 + \gamma\omega b_5 c_1] + \nu^2\varphi b_1 b_2 b_3 b_4 b_5 b_6^2 c_4[R_{eff}^2 - 1] + \\
&\quad \nu\beta(1-\alpha)\lambda\varphi b_2 b_4 b_6 c_2 c_3 + \nu b_2 b_5 b_6 c_3(\beta\gamma\omega c_2 - 2b_3 c_4) \\
d_1 &= \sigma\beta b_5 c_4\{\alpha\lambda\beta b_4 c_1 + \nu\varphi b_4 b_6[\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda] + \nu\tau\gamma\omega b_5 b_6\} + \\
&\quad \sigma\beta[\varphi c_4 + b_5 c_3][\alpha\lambda\beta b_4 c_2 + \nu b_4 b_5 b_6(\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda)] + \\
&\quad \nu\beta\varphi(1-\alpha)\lambda b_2 b_4 b_6[\varphi\kappa\pi\epsilon b_5 + c_2 c_4] + \nu\beta\gamma\omega b_2 b_5 b_6 c_2 c_4 + \\
&\quad \nu b_2 b_4 b_6 c_2 c_4[\beta\varphi(1-\alpha)\lambda - b_3 b_5] + \nu^2 b_1 b_2 b_3 b_4 b_5^2 b_6^2 c_4[R_{eff}^2 - 1] \\
d_0 &= \gamma(1-\alpha)\lambda b_4 b_5 b_6 c_2 c_4 + b_5 c_4[\alpha\lambda\beta b_4 c_2 + \nu b_4 b_5 b_6(\alpha\lambda b_1 + \tau\varphi(1-\alpha)\lambda)]
\end{aligned}
$$

Clearly $d_0 > 0$ and $d_4 < 0$. $d_1 > 0$ provided $R_{eff} \geq 1$ and $\beta\varphi(1-\alpha)\lambda > b_3 b_5$ and $d_2 > 0$ provided $R_{eff} \geq 1$ and $\beta\gamma\omega c_2 > 2b_3 c_4$. Therefore, by the Descarte's Rule of signs, a unique positive endemic equilibrium point E^* exists irrespective of the sign of d_3 provided $R_{eff} \geq 1, \beta\varphi(1-\alpha)\lambda > b_3 b_5$ and $\beta\gamma\omega c_2 > 2b_3 c_4$. □

Next, we analyse the stability of the endemic equilibrium point.

Theorem 2.6. *The endemic equilibrium point E^* is locally asymptotically stable provided $\nu b_3 \geq \beta\varphi S_c^* S_v^*$.*

Proof. The Jacobian matrix of model system (2.1) at the point E^* is given by

$$
J(E^*) = \begin{pmatrix}
-\beta I_v^* - b_1 & 0 & 0 & \gamma & 0 & 0 & -\beta S_c^* \\
\tau & -b_2 & 0 & 0 & 0 & 0 & 0 \\
\beta I_v^* & \sigma\beta & -b_3 & 0 & 0 & 0 & \beta S_c^* \\
0 & 0 & \omega & -b_4 & 0 & 0 & 0 \\
0 & 0 & -\varphi S_v^* & 0 & -\varphi I_c^* - b_5 & 0 & 0 \\
0 & 0 & 0 & 0 & \vartheta & -b_6 & 0 \\
0 & 0 & \varphi S_v^* & 0 & \varphi I_c^* & \epsilon\varphi & -\nu
\end{pmatrix}
$$

The characteristic polynomial of matrix $J(E^*)$ is given by

$$
\begin{aligned}
P_{J(E^*)}(w) &= det(J(E^*) - wI_7) \\
&= w^7 + a_6 w^6 + a_5 w^5 + a_4 w^4 + a_3 w^3 + a_2 w^2 + a_1 w + a_0
\end{aligned}
$$

If all the coefficients of the $P_{J(E^*)}(w)$ are positive, then by the Descartes Rule of signs, all the roots of $P_{J(E^*)}(w)$ will have negative real parts.

By a result in [2], all the coefficients of $P_{J(E^*)}(w)$ are positive provided the independent term a_0 is positive. It remains to show that the independent term a_0 is positive.

The independent term a_0 can be obtained using the formula $a_0 = (-1)^n det(J(E^*))$. Thus, we have

$$\begin{aligned} a_0 &= \nu\varphi b_6 I_c^*\{\sigma\beta\tau[\mu b_3 + (\mu+\delta)\gamma] + \mu(\sigma\beta + b_1)b_3 b_4\} + \mu\varphi\sigma\beta^2\tau b_5 b_6 S_c^* S_v^* + \\ &\quad \nu\beta b_2 b_6 I_v^*[\varphi I_c^* + b_5][\mu b_3 + (\mu+\delta)\gamma] + \mu\beta\varphi^2\epsilon\vartheta(\sigma\beta + b_1)b_4 S_c^* S_v^* + \\ &\quad (\mu+\delta)\nu\sigma\beta\tau\gamma b_5 b_6 + \mu b_5 b_6[\tau b_2 + b_2 b_4 + \tau\gamma][\nu b_3 - \beta\varphi S_c^* S_v^*] \end{aligned}$$

The independent term a_0 is positive provided $\nu b_3 \geq \beta\varphi S_c^* S_v^*$. Therefore, all the eigenvalues of the matrix $J(E^*)$ have negative real parts provided $\nu b_3 \geq \beta\varphi S_c^* S_v^*$. Thus, the endemic equilibrium point E^* is locally asymptotically stable provided $\nu b_3 \geq \beta\varphi S_c^* S_v^*$. □

3 Numerical Results

Table 2: *Numerical values for the parameters of the AAT model incorporating treatment of cattle and spraying of tsetse fly*

Symbol	Description	Value	Unit	Source
μ	Natural mortality rate of cattle	$\frac{1}{30\times365}$	days^{-1}	*Estimated*
ν	Natural mortality rate of vector	$\frac{1}{30}$	days^{-1}	[8]
δ	Disease-induced death rate in cattle	0.006	days^{-1}	[8]
ρ	Transmission rate of infection from tsetse fly to cattle	0.62	days^{-1}	[8]
p	Transmission rate of infection from cattle to tsetse fly	0.065	days^{-1}	[8]
η	Average biting rate of tsetse fly	0.0714	days^{-1}	*estimated*
$\beta = \eta \cdot \rho$	Infection force in Cattle	0.0443	days^{-1}	*estimated*
$\varphi = \eta \cdot p$	Infection force in vector	0.0046	days^{-1}	*estimated*
π	Rate at which vector population are replenished	0.07	days^{-1}	[5]
λ	Rate at which cattle population are replenished	$\frac{1}{15\times365}$	days^{-1}	[14]
γ	Rate at which recovered cattle reintegrate with the susceptible cattle population	$\frac{1}{50}$	days^{-1}	[14]
α	Proportion of cattle kept under treatment with Trypanocides	0.5	days^{-1}	*Estimated*
τ	Rate of treating susceptible cattle using anti-parasitic drugs	0.6	days^{-1}	*Varying*
ϑ	Rate of spraying susceptible vector	0.75	days^{-1}	*Varying*
κ	Proportion of tsetse fly kept under spraying	0.75	days^{-1}	*Estimated*
σ	Failure rate of treatment	0.02	days^{-1}	*Estimated*
ω	Recovery rate of infected cattle	$\frac{1}{10}$	day^{-1}	*estimated*
ϵ	Failure rate of spraying	0.07	days^{-1}	*estimated*
ξ	Tsetse death rate due to spraying	0.78	days^{-1}	[8]

To simulate the model, some parameter values were obtained from [5, 8, 14] and others were estimated. The parameter values are as given in Table 2 above. The model in equation (2.1) is simulated using deSolve in R-programming language for different treatment of cattle and spraying of tsetse fly levels with the initial conditions $S_c = 100, E_c = 10, I_c = 5, R_c = 0, S_v = 1000, E_v = 15, I_v = 10$.

The simulation is carried out to investigate combined effect of treatment of cattle and spraying of the tsetse fly on the control of AAT spread in Cattle. Varying rate of treatment of cattle (τ) from $50\% - 90\%$ while holding rate of spraying of tsetse fly at 75% , the simulation results are as shown in figure 2

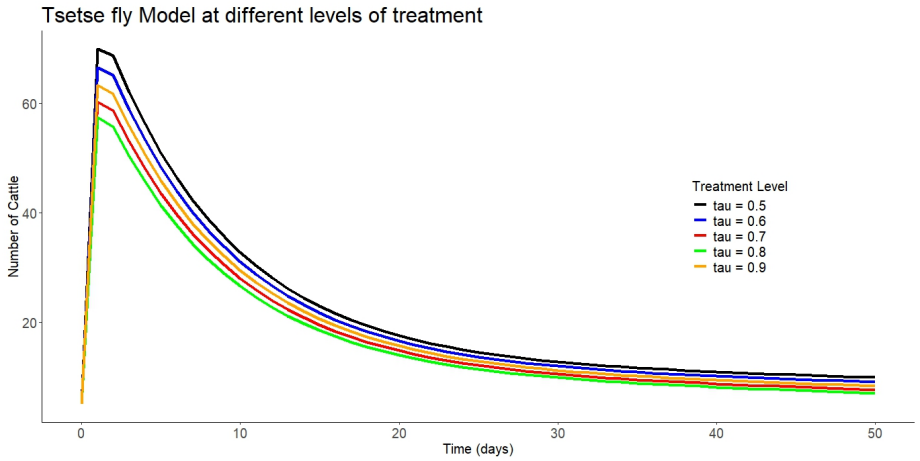

Figure 2: *Treatment at different levels with rate of spraying at* 75%

Varying rate of spraying of tsetse fly (ϑ) from $75\% - 95\%$ while holding rate of treatment of cattle at 70% , the simulation results are as shown in figure 3. The results shown in Figure 2 show that treating cattle at different levels

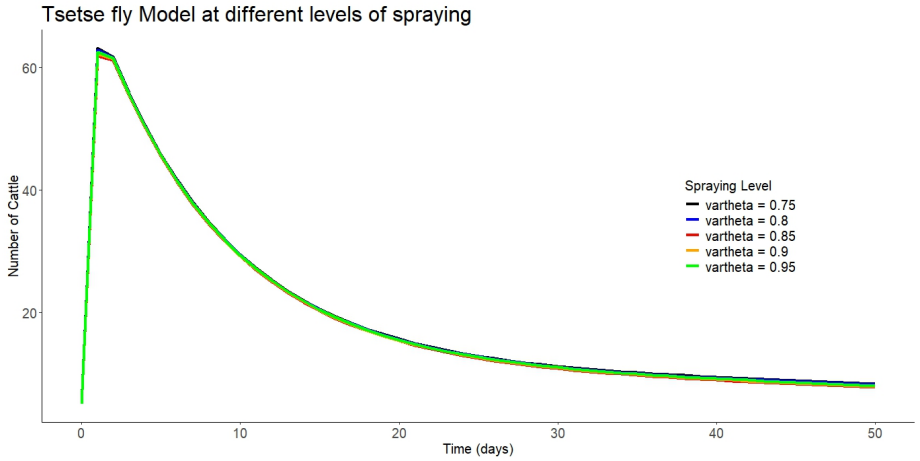

Figure 3: *Spraying at different levels with rate of treatment at* 70%

while keeping spraying of tsetse fly constant help reduce the number of infected cattle significantly. On the other hand, spraying tsetse fly at different levels while holding treatment rate of cattle constant has insignificant reduction on

the number of infected cattle as shown in Figure 3. From Fig.2 and Fig.3, it can be seen that the best strategy for controlling AAT is treating cattle at different rates while holding rate of spraying tsetse fly constant at 75%.

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