Research Article

African Trypanosomiasis Dynamics: Modelling the Effects of Treatment, Education, and Vector Trapping

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1. Introduction

African trypanosomiasis is a disease caused by microscopic parasites of the species Trypanosoma brucei, and it is transmitted through bites of infected tsetse flies of the genus Glossina which are most common in woodland and savannah areas of sub-Saharan Africa. Trypanosoma brucei infects both humans and animals, and if not treated early, it can lead to death [1]. Human African trypanosomiasis (HAT) is caused by Trypanosoma brucei gambiense and Trypanosoma brucei rhodesiense while African animal trypanosomiasis (AAT) is mainly caused by Trypanosoma brucei vivax, Trypanosoma brucei congolense, and Trypanosoma brucei rhodesiense [2].

Even though both humans and animals are infected by Trypanosoma brucei, cattle are mostly infected because of tsetse flies’ feeding preferences. Both male and female tsetse flies can transmit Trypanosoma and depend only on hosts’ blood to survive or for all their nutritional needs, unlike other vector-borne diseases such as malaria, where only a female mosquito can feed on blood and can transmit the disease [3].

The disease has affected at least 37 countries in sub-Saharan Africa, threatening the lives of millions of people in rural areas. Around 10 million square kilometers in sub-Saharan Africa have been affected by Trypanosoma brucei species [3]. On average, 70,000 cases of HAT are reported each year in sub-Saharan Africa, and more than 1 million cattle die every year due to trypanosomiasis and cause an economic loss of between $2 and $4.5 billion annually [4, 5]. It is claimed that the high mortality rate of livestock can lead to low production of meat and milk up to 50% in sub-Saharan Africa every year [6].

So far, much work has been done to investigate the dynamics of African trypanosomiasis. For example, Moore et al. [4] developed a model on the effect of climate change on African trypanosomiasis dynamics. The result from their study predicted that, by the year 2090, about 46–77 million people would be exposed to trypanosomiasis disease. Otieno et al. [7] studied the dynamics of trypanosomiasis in a cattle population by including the wild animals as an alternative feeding source for tsetse flies. The results obtained from the
study show that wild animals accelerate the disease in the cattle population. The sensitivity analysis also revealed that the vector biting rate and the vector survival rate are parameters with the greatest influence on the disease spread. These results indicate that the control strategies that target to decrease the contact between the vector and cattle populations would be the best way to eliminate the disease in the population. Kajunguri et al. [8] developed a model to control tsetse flies using insecticide-treated cattle in a multihost population. The results showed that the treatment of both infected humans and cattle combined with insecticide-treated cattle effectively decreases trypanosomes’ prevalence. Meisner et al. [9] researched the role of trypanocide treatment on cattle. The result showed that as the coverage of treated cattle with trypanocide increases, the disease prevalence decreases in both humans and cattle. Ndondo et al. [10] analyzed gambiense sleeping sickness on both human and cattle by including tsetse fly growth from its larval stage to the adult stage. The study’s findings show that human African trypanosomiasis cannot persist in the human population in the absence of cattle.

Despite the numerous studies conducted to control African trypanosomiasis, the disease remains a major health threat to both human livelihood and livestock production and affects economic development in Africa. African trypanosomiasis has caused around 500 million farmers in rural Africa villages to live under food shortage and poverty [11]. The African trypanosomiasis model developed by Ndondo et al. [10] considered treatment as the only control strategy while leaving out public health education and tsetse fly traps. Therefore, this paper aimed to extend Ndondo et al.’s [10] work by incorporating public health education and tsetse fly traps to address the question “How does tsetse-fly traps, public health education, and treatment of both human and cattle affect the dynamics of African trypanosomiasis?”. The rest of this paper is structured as follows: in Section 2, the model is formulated, and in Section 3, the model analysis is carried out. In Section 4, sensitivity analysis and its interpretations are made, and in Section 5, we have numerical simulation while concluding remarks are covered in Section 6.

2. The Model

The model considered here consists of the submodels of human, cattle, and tsetse fly populations. For the control of trypanosomiasis disease infection, we consider three interventions, namely, public health education, treatment, and trapping of tsetse flies. Human population at time $t$ is subdivided into five subpopulations of uneducated susceptible $S_h(t)$, educated susceptible $S_e(t)$, exposed $E_h(t)$, infected $I_h(t)$, and recovered individuals $R_h(t)$. The total human population $N_h(t)$ is thus given by $N_h(t) = S_h(t) + S_e(t) + E_h(t) + I_h(t) + R_h(t)$. Human individuals are recruited into the population at a constant rate of $\Lambda_h$. We assume that education strategy is implemented at the rate $\theta$ only to susceptible people to make them aware of how to protect themselves from tsetse fly biting (public health education is given on the importance of clearing the environment, wearing long-sleeved clothes, and using repellents). Uneducated susceptible individuals can acquire infection and move to exposed class through the bite of infectious tsetse flies at a rate of $\lambda_h$. Due to the education campaign, it is assumed that only a small fraction of educated individuals move to the exposed class at a rate of $(1 - \epsilon)\lambda_h$, where $\epsilon$ is the efficacy of the education campaign. Both educated susceptible and uneducated susceptible may also leave their respective classes through natural death at a rate of $\mu_h$. Exposed humans become infectious at a rate $\alpha_h$ and the infectious humans leave the infected class through natural death, disease-induced death, or recovery at the rates $\mu_h$, $\sigma_h$, and $\beta_h$, respectively. Individuals in the recovered class may leave the compartment by either natural death at a rate of $\psi_h$ or through losing temporary immunity and move to the susceptible class at a rate of $\psi_h$. We further assume that the infected human recovers through treatment, implying that no human individual recovers naturally, and also, this study assumes that all humans are born susceptible. The model for the human population takes the following form:

$$\begin{align*}
\frac{dS_h}{dt} &= \Lambda_h + \psi_h R_h - \lambda_h S_h - (\mu_h + \theta) S_h, \\
\frac{dS_e}{dt} &= \theta S_h - (1 - \epsilon) \lambda_h S_e - \mu_h S_e, \\
\frac{dE_h}{dt} &= \lambda_h S_e + (1 - \epsilon) \lambda_h S_e - (\mu_h + \alpha_h) E_h, \\
\frac{dI_h}{dt} &= \alpha_h E_h - (\mu_h + \sigma_h + \beta_h) I_h, \\
\frac{dR_h}{dt} &= \beta_h I_h - (\mu_h + \psi_h) R_h,
\end{align*}$$

where $\lambda_h$ is the force of infection given by $\lambda_h = (1 - \rho)kbkI_c/N_h$, with $k$ as the probability that the infectious vector infects a susceptible individual, $b$ is the tsetse fly blood-feeding rate per day, and $\rho$ is the proportion of tsetse fly feeding on cattle, and the complimentary (1 - $\rho$) is the proportion of tsetse fly feeding on a human per day. It is assumed that a tsetse fly can only become infected at its first blood meal and remains so throughout its lifespan [12].

The cattle population at time $t$ is subdivided into four compartments of susceptible $S_c(t)$, exposed $E_c(t)$, infected $I_c(t)$, and recovered $R_c(t)$. The total cattle population denoted by $N_c(t)$ is given as $N_c(t) = S_c(t) + E_c(t) + I_c(t) + R_c(t)$. At any moment in time, it is assumed that cattle are recruited into the population at a constant rate of $\Lambda_c$. Cattle leave the susceptible class through natural death at a rate of $\mu_c$ or by getting infected and joining the exposed class at $\lambda_c$. The exposed cattle become infectious and move to the infected class at a rate of $\alpha_c$. The infectious cattle leave the infected class through natural death, disease-induced death, or recovery at the rates $\mu_c$, $\sigma_c$, and $\beta_c$, respectively. We also assume that cattle acquire temporary immunity, and recovered cattle may leave the recovered class either by natural
death at the rate \( \mu_c \) or through the waning of temporary immunity and move to susceptible class at a rate \( \psi_c \). Furthermore, it is assumed in this study that all cattle are born susceptible, and there are no cattle that can experience natural recovery from the disease.

The model for cattle population takes the following form:

\[
\begin{align*}
\frac{dS_v}{dt} &= \Lambda_v - \lambda_v S_v - (\mu_v + \omega)S_v, \\
\frac{dE_v}{dt} &= \lambda_v S_v - (\mu_v + \omega + \alpha_v)E_v, \\
\frac{dI_v}{dt} &= \alpha_v E_v - (\mu_v + \omega)I_v, \\
\frac{dR_v}{dt} &= \beta_v I_v - (\mu_v + \psi_v)R_v,
\end{align*}
\]

where \( \lambda_v \) is the force of infection given by \( \lambda_v = b dp t / N_c \) and \( d \) is the probability that an infectious tsetse fly infects cattle.

The tsetse fly vector population at time \( t \) is divided into three compartments of susceptible \( S_v(t) \), exposed \( E_v(t) \), and infectious \( I_v(t) \). Therefore, the total vector population is \( N_v(t) = S_v(t) + E_v(t) + I_v(t) \). It is assumed that tsetse flies are recruited through birth at a rate of \( \Lambda_v \). We assume that the tsetse fly population, regardless of its status, dies naturally at a rate of \( \mu_v \) or by being trapped at a rate of \( \omega \). The tsetse flies move to exposed class after acquiring the infection at a rate of \( \lambda_v \), and also, the infected flies progress to being infectious at a rate of \( \alpha_v \). In this study, all tsetse flies are assumed to be born susceptible, and once a tsetse fly becomes infected, it is assumed to remain in that condition throughout its lifespan.

The model for vector population takes the following form:

\[
\begin{align*}
\frac{dS_v}{dt} &= \Lambda_v - \lambda_v S_v - (\mu_v + \omega)S_v, \\
\frac{dE_v}{dt} &= \lambda_v S_v - (\mu_v + \omega + \alpha_v)E_v, \\
\frac{dI_v}{dt} &= \alpha_v E_v - (\mu_v + \omega)I_v, \\
\frac{dR_v}{dt} &= \beta_v I_v - (\mu_v + \psi_v)R_v,
\end{align*}
\]

where \( \lambda_v = (1 - \rho) bg l / N_h + \rho bg l / N_c \) is the force of infection in the vector population.

The tsetse fly becomes infected either by biting infected humans or by biting infected cattle. Therefore, \( g \) is a probability that a susceptible tsetse fly becomes infected after biting an infected human host and \( z \) is the probability that a susceptible tsetse fly becomes infected after biting infected cattle. Tables 1 and 2 summarize the definitions of all state variables and the associated parameters, respectively.

**Table 1: Descriptions of variables of model (4).**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Descriptions</th>
</tr>
</thead>
<tbody>
<tr>
<td>( S_h )</td>
<td>Susceptible uneducated human</td>
</tr>
<tr>
<td>( S_e )</td>
<td>Susceptible educated human</td>
</tr>
<tr>
<td>( E_h )</td>
<td>Exposed human</td>
</tr>
<tr>
<td>( I_h )</td>
<td>Infected human</td>
</tr>
<tr>
<td>( R_h )</td>
<td>Recovered human</td>
</tr>
<tr>
<td>( S_v )</td>
<td>Susceptible cattle</td>
</tr>
<tr>
<td>( I_v )</td>
<td>Infected cattle</td>
</tr>
<tr>
<td>( R_v )</td>
<td>Recovered cattle</td>
</tr>
<tr>
<td>( S_c )</td>
<td>Susceptible vector</td>
</tr>
<tr>
<td>( I_c )</td>
<td>Infected vector</td>
</tr>
<tr>
<td>( R_c )</td>
<td>Recovered vector</td>
</tr>
</tbody>
</table>

From the model diagram in Figure 1 and the relevant assumptions, we get a full model that describes the dynamics of African trypanosomiasis disease:

\[
\begin{align*}
\frac{dS_h}{dt} &= \lambda_h S_h - (\mu_h + \sigma_h + \beta_h)E_h, \\
\frac{dE_h}{dt} &= \alpha_h E_h - (\mu_h + \sigma_h + \beta_h)I_h, \\
\frac{dI_h}{dt} &= \beta_h I_h - (\mu_h + \psi_h)R_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_v S_v - (\mu_v + \omega)S_v, \\
\frac{dE_v}{dt} &= \alpha_v E_v - (\mu_v + \omega + \alpha_v)I_v, \\
\frac{dI_v}{dt} &= \beta_v I_v - (\mu_v + \psi_v)R_v,
\end{align*}
\]

The corresponding initial conditions are \( S_h(0) \geq 0, S_v(0) \geq 0, S_c(0) \geq 0, I_h(0) \geq 0, I_v(0) \geq 0, R_h(0) \geq 0, R_c(0) \geq 0, E_h(0) \geq 0, I_c(0) \geq 0, \).
$E_0(0) \geq 0, I_0(0) \geq 0$, and $R_0(0) \geq 0, S_0(0) \geq 0, E_0(0) \geq 0$, $I_0(0) \geq 0$.

### 3. Model Analysis

Since model (4) monitors population of humans, cattle, and tsetse flies, all its associated variables are assumed to be nonnegative $\forall t \geq 0$. We need to show that model (4) is mathematically and epidemiologically well defined by proving that all the state variables are always positive $\forall t \geq 0$.

**Theorem 1.** Let the feasible region for the three populations be $\Omega = \Omega_h \cup \Omega_c \cup \Omega_v \in \mathbb{R}_+^5 \times \mathbb{R}_+^4 \times \mathbb{R}_+^3$, where $\Omega_h = [S_h, S_h + E_h + I_h + R_h = N_h \leq \Lambda_h/\mu_h]$, $\Omega_c = [S_c, S_c + E_c + I_c + R_c = N_c \leq \Lambda_c/\mu_c]$, and $\Omega_v = [S_v, S_v + E_v + I_v = N_v \leq \Lambda_v/\mu_v]$. It is sufficient to consider
the solutions in $\Omega$ since it is positively invariant and attracting with respect to model (4).

**Proof.** To prove the feasible region, we use model (4) and compute the total population of humans, cattle, and tsetse flies.

For human population,
\[
\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \sigma_h I_h + .
\] (5)

For cattle population,
\[
\frac{dN_c}{dt} = \Lambda_c - \mu_c N_c - \sigma_c I_c \leq \Lambda_c - \mu_c N_c.
\] (6)

For tsetse fly population,
\[
\frac{dN_v}{dt} = \Lambda_v - (\mu_v + \omega) N_v \leq \Lambda_v - \mu_v N_v.
\] (7)

Then, solving for $N_h$, $N_c$, and $N_v$ from equations (5)–(7), we obtain the following inequalities as in [13] (see "theorem"):

\[
N_h(t) \leq \frac{\Lambda_h}{\mu_h} \left( \frac{N_h(0)}{\mu_h} \right) e^{-\mu_h t} \rightarrow N_h(t) \leq \frac{\Lambda_h}{\mu_h} \text{ as } t \rightarrow \infty,
\] (8)

\[
N_c(t) \leq \frac{\Lambda_c}{\mu_c} \left( \frac{N_c(0)}{\mu_c} \right) e^{-\mu_c t} \rightarrow N_c(t) \leq \frac{\Lambda_c}{\mu_c} \text{ as } t \rightarrow \infty,
\] (9)

\[
N_v(t) \leq \frac{\Lambda_v}{\mu_v} \left( \frac{N_v(0)}{\mu_v} \right) e^{-\mu_v t} \rightarrow N_v(t) \leq \frac{\Lambda_v}{\mu_v} \text{ as } t \rightarrow \infty.
\] (10)

Therefore, from equations (8)–(10), we see that the solutions for human, cattle, and tsetse fly populations enter the following invariant regions:

\[
\Omega_h = \left\{ S_u, S_c, E_h, I_h, R_h, S_u + S_c + E_h + I_h + R_h = N_h \leq \frac{\Lambda_h}{\mu_h} \right\},
\]

\[
\Omega_c = \left\{ S_c, E_c, I_c, R_c, S_c + E_c + I_c + R_c = N_c \leq \frac{\Lambda_c}{\mu_c} \right\}, \text{ and}
\]

\[
\Omega_v = \left\{ S_v, E_v, I_v, S_v + E_v + I_v = N_v \leq \frac{\Lambda_v}{\mu_v} \right\}.
\] (11)

The results imply that the region is bounded, well posed, and biologically meaningful as it attracts all solutions in $\Omega$. \hfill \Box

### 3.1. Local Stability of Disease-Free Equilibrium.

Model (4) has a unique disease-free equilibrium (DFE) which was obtained by setting the right-hand sides of the equations to zero. The DFE is given by

\[
\begin{align*}
E_0 &= \left( S_u^*, S_c^*, E_h^*, I_h^*, R_h^*, S_c^*, E_c^*, I_c^*, R_c^*, S_v^*, E_v^*, I_v^* \right) \\
&= \left[ \frac{\Lambda_h}{\theta + \mu_h}, \frac{\Lambda_c}{\mu_c}, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v + \omega}, 0, 0 \right].
\end{align*}
\] (12)

Using the next generation method as in Van den Driessche and Watmough [14], the associated matrices $F$ for new infection terms and $V$ for the remaining transition terms are evaluated at $E_0$ and given by
where \( A_1 = \mu_h + \theta, \quad A_2 = \mu_h + \alpha, \quad A_3 = \mu_h + \sigma_h + \beta_h, \)
\( A_4 = \mu_h + \psi_h, \quad A_5 = \mu + \alpha, \quad A_6 = \mu + \sigma + \beta, \quad A_7 = \mu + \psi, \)
\( A_8 = \mu + \omega, \quad A_9 = \mu + \omega + \alpha, \quad B_1 = 1 - \epsilon, \) and \( B_2 = 1 - \rho. \)

It follows that the effective or control reproduction number denoted by \( R_c \) is the spectral radius of the next generation matrix \( \rho(FV^{-1}) \) given as

\[
R_c = \sqrt{R_c c + R_c h},
\]

where \( R_c c \) represents the effective reproduction number for cattle and \( R_c h \) is the effective reproduction number for humans. The reproduction number is used to determine whether the disease will die out or persist. That is, the disease persists in the community if \( R_c \) is greater than 1 and it dies out if \( R_c \) is less than 1.

The following theorem summarizes the result.

**Theorem 2.** The DFE of the model system (4) is locally asymptotically stable if \( R_c < 1 \) and unstable if \( R_c > 1 \).

Biologically, it implies that African trypanosomiasis can be eliminated from the community provided that the initial size of the subpopulations of the model (4) is in the basin of attraction of disease-free equilibrium point \( E_0 \) when \( R_c < 1 \). In other words, the introduction of infectious individuals into a population of susceptible individuals does not induce an epidemic outbreak. On the contrary, if \( R_c > 1 \), it implies that African trypanosomiasis disease will persist in the population.

### 3.2. Global Stability of the Disease-Free Equilibrium Point

Here, we apply Castillo-Chavez et al. [15] approach to analyze the global stability of the disease-free equilibrium point of the model (4).
Theorem 3. The disease-free equilibrium point is globally asymptotically stable if \( R_e < 1 \) and unstable if \( R_e > 1 \).

Proof. The model (4) can be written in the following format:

\[
\begin{align*}
\frac{dY_n}{dt} &= A_1(Y_n - Y_{E_0}) + A_2 Y_i, \\
\frac{dY_i}{dt} &= A_3 Y_i,
\end{align*}
\]

where \( Y_n \) is a vector of nontransmitting variables, \( Y_{E_0} \) is \( Y_n \) at disease-free equilibrium point \( E_0 \), and \( Y_i \) is a vector consisting of infectious variables.

From model (4), we have

\[
Y_n = (S_u, S_e, R_h, S_c, R_c, S_v)^T
\]

and

\[
Y_i = (E_h, I_h, E_c, I_c, E_v, I_v)^T
\]

from which we get

\[
\begin{bmatrix}
S_u \\
S_e \\
R_h \\
S_c \\
R_c \\
S_v
\end{bmatrix} - \begin{bmatrix}
\frac{\Lambda_h}{\theta + \mu_h} \\
\frac{\theta}{\mu_h} \left( \frac{\Lambda_h}{\theta + \mu_h} \right) \\
0 \\
\frac{\Lambda_c}{\mu_c} \\
0 \\
\frac{\Lambda_v}{\mu_v + \omega}
\end{bmatrix} = \begin{bmatrix}
S_u - \frac{\Lambda_h}{\theta + \mu_h} \\
S_c - \frac{\Lambda_c}{\mu_c} \\
R_h \\
S_c - \frac{\Lambda_c}{\mu_c} \\
R_c \\
S_v - \frac{\Lambda_v}{\mu_v + \omega}
\end{bmatrix}.
\]

The disease-free equilibrium is globally asymptotically stable if the matrix \( A_1 \) has real negative eigenvalues and \( A_3 \) is a Metzler matrix (i.e., the off diagonal elements of \( A_3 \) are nonnegative, which means \( A_3(i, j) \geq 0 \) for all indices of \( i \neq j \)). That is,

\[
A_1 = \frac{\partial Y_n}{\partial (S_u, S_e, R_h, S_c, R_c, S_v)} = \begin{bmatrix}
-(\theta + \mu_h) & 0 & \psi_h & 0 & 0 & 0 \\
\theta & -\mu_h & 0 & 0 & 0 & 0 \\
0 & 0 & -(\mu_h + \psi_h) & 0 & 0 & 0 \\
0 & 0 & 0 & -\mu_c & \psi_c & 0 \\
0 & 0 & 0 & 0 & -(\mu_c + \psi_c) & 0 \\
0 & 0 & 0 & 0 & 0 & -(\mu_v + \omega)
\end{bmatrix}.
\]
with eigenvalues $\lambda_1 = -\mu_h$, $\lambda_2 = -(\theta + \mu_h)$, $\lambda_3 = -(\mu_h + \psi_h)$, $\lambda_4 = -\mu_e$, $\lambda_5 = -(\mu_e + \psi_e)$, and $\lambda_6 = -(\mu_e + \omega)$. Also,

$$A_2 = \frac{\partial Y_u}{\partial (E_h, I_h, E_c, I_c, E_v, I_v)} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{-(1 - \rho)bkS_u}{N_h} \\ 0 & 0 & 0 & 0 & 0 & \frac{-(1 - e)bkS_c}{N_h} \\ 0 & \beta_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -b \rho S_c & \frac{-b \rho S_c}{N_c} \\ 0 & 0 & 0 & \beta_c & 0 & 0 \\ 0 & 0 & 0 & 0 & -\rho b z S_v & 0 & -0 \end{pmatrix},$$

and

$$A_3 = \frac{\partial Y_i}{\partial (E_h, I_h, E_c, I_c, E_v, I_v)} = \begin{pmatrix} -P_1 & 0 & 0 & 0 & 0 & \frac{(1 - \rho)bk(S_u + (1 - e)S_c)}{N_h} \\ \alpha_h & -P_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_3 & 0 & 0 & \frac{b \rho \rho S_c}{N_c} \\ 0 & 0 & \alpha_c & -P_4 & 0 & 0 \\ 0 & 0 & 0 & \alpha_v & -P_5 & 0 \\ 0 & 0 & 0 & 0 & \alpha_v & \frac{1 - \rho)bk}{N_h} \end{pmatrix},$$

where $P_1 = (\mu_h + \alpha_h)$, $P_2 = (\mu_h + \sigma_h + \beta_h)$, $P_3 = (\mu_c + \alpha_c)$, $P_4 = (\mu_c + \sigma_c + \beta_c)$, $P_5 = (\mu_c + \omega + \alpha_v)$, and $P_6 = (\mu_e + \omega)$. It can be seen that matrix $A_3$ has all eigenvalues which are real and negative and matrix $A_3$ is the Metzler matrix as its off-diagonal elements are positive. Thus, the system

$$\frac{dY}{dt} = A_1(Y - Y_{E_0}) + A_2 Y_u + A_3 Y_i$$

is globally asymptotically stable at disease-free equilibrium point.

4. Sensitivity Analysis

The sensitivity analysis is carried out to determine the parameters that have a higher impact on the effective reproduction number. To reduce disease transmission, the parameters that have a higher impact on the effective reproduction number should be targeted for control purposes.

Analytically, the sensitivity index of $R_e$ is calculated by using the normalized forward sensitivity index defined as $Y^R_p = \partial R_e/\partial \rho \times p/R_e$, where $p$ stands for any parameter in effective reproduction number $R_e$ [16]. For example, the sensitivity index of $R_e$ corresponding to the parameter $\alpha_v$ is given as $Y^R_{\alpha_v} = \partial R_e/\partial \alpha_v \times \alpha_v/R_e = +0.3182$. Other indices are calculated using similar approach, and the results are summarized in Table 3 and in Figure 2.

4.1. Interpretation of the Sensitivity Indices. From Table 3, the parameters $\alpha_h$, $\alpha_v$, $\Lambda_h$, $b$, $k$, $g$, $z$, $d$, $\rho$, and $\alpha_v$ have positive indices, indicating that increasing one of these parameters while keeping others constant increases the effective reproduction number, hence increasing the possibility of the disease outbreak. On the contrary, the parameters $\mu_h$, $\mu_e$, $\Lambda_h$, $\theta$, $e$, $\beta_h$, $\sigma_h$, $\Lambda_c$, $\sigma_c$, $\beta_c$, $\mu_v$, and $\omega$ have negative indices, implying that increasing one of these parameters and
keeping others constant decreases the effective reproduction number, hence reducing the disease burden among human, cattle, and vector population. From these results, the most sensitive parameters are the tsetse fly biting rate $b$, the proportion of tsetse fly biting on cattle $\rho$, the rate at which the tsetse flies die due to trapping $\omega$, and tsetse fly natural mortality rate $\mu_v$, followed by the tsetse fly recruitment rate $\Lambda_v$.

Therefore, increasing tsetse fly death rate and reducing tsetse fly biting rate through public health education campaigns on the importance of wearing long-sleeved clothes, clearing the bushes, and the use of repellent solutions to avoid vector-host contact rate would have a higher positive impact in controlling trypanosomiasis transmission in a community.

5. Numerical Simulation

In this section, we simulate model (4) using parameter values shown in Table 4. The Matlab ODE45 solver is used to simulate the model system (4). The initial conditions of the state variables are given as follows: $S_h = 200$, $S_c = 180$, $E_h = 150$, $I_h = 100$, $R_h = 80$, $S_c = 200$, $E_c = 170$, $I_c = 140$, $R_c = 100$, $S_v = 3000$, $E_v = 800$, and $I_v = 500$. The initial conditions of the state variables are arbitrarily chosen to illustrate specific behaviour of the model (4).

5.1. Effects of Interventions on Infected Human and Cattle Population. Figure 3 illustrates the impact of different combinations of interventions (human treatment, public health education, and tsetse fly trapping) on the dynamics of African trypanosomiasis in the human population. Combining all three interventions tends to diminish disease transmission in a community faster than treating infected humans only. It is also observed from Figure 3 that treating the infected humans while increasing people’s awareness about the disease has a greater impact than using treatment alone. This implies that apart from using other control measures like public health education and human treatment, there must be an effort to eliminate the tsetse fly vector to eradicate the disease from a community.

Table 3: Sensitivity indices of $R_e$ using parameter values in Table 4.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Index value</th>
<th>Parameter</th>
<th>Index value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Lambda_h$</td>
<td>$-0.0036$</td>
<td>$\beta_c$</td>
<td>$-0.2775$</td>
</tr>
<tr>
<td>$\theta$</td>
<td>$-0.0638$</td>
<td>$\sigma_v$</td>
<td>$+0.3182$</td>
</tr>
<tr>
<td>$\mu_h$</td>
<td>$-0.0042$</td>
<td>$\mu_v$</td>
<td>$-0.5649$</td>
</tr>
<tr>
<td>$\epsilon$</td>
<td>$-0.034$</td>
<td>$\omega$</td>
<td>$-0.7532$</td>
</tr>
<tr>
<td>$\alpha_h$</td>
<td>$+0.000192$</td>
<td>$\Lambda_c$</td>
<td>$+0.5$</td>
</tr>
<tr>
<td>$\beta_h$</td>
<td>$-0.025$</td>
<td>$k$</td>
<td>$+0.0036$</td>
</tr>
<tr>
<td>$\sigma_h$</td>
<td>$-0.0011$</td>
<td>$\beta_c$</td>
<td>$+1$</td>
</tr>
<tr>
<td>$\Lambda_c$</td>
<td>$-0.4964$</td>
<td>$g$</td>
<td>$+0.0036$</td>
</tr>
<tr>
<td>$\mu_c$</td>
<td>$-0.4748$</td>
<td>$z$</td>
<td>$+0.4964$</td>
</tr>
<tr>
<td>$\sigma_c$</td>
<td>$-0.2006$</td>
<td>$d$</td>
<td>$+0.4964$</td>
</tr>
<tr>
<td>$\alpha_c$</td>
<td>$+0.0033$</td>
<td>$\rho$</td>
<td>$+0.9761$</td>
</tr>
</tbody>
</table>

Figure 2: Sensitivity analysis of $R_e$ using results in Table 3.
Figure 4 shows that treatment alone cannot control the disease in the cattle populations. Therefore, to control the disease, the tsetse fly traps must be used as a complementary intervention to cattle treatment.

### 5.2. Effect of Varying Some Parameter Values

Figure 5 illustrates the effects of varying human and cattle treatment rates while fixing rates of tsetse fly trapping and public health education constant. As the rate of treating the infected

Table 4: Epidemiological data for model (4).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Values (days)</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Lambda_h )</td>
<td>27.5</td>
<td>Kajunguri et al. [8]</td>
</tr>
<tr>
<td>( \theta )</td>
<td>0.0002</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \mu_h )</td>
<td>0.000046</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \epsilon )</td>
<td>0.6</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \psi_h )</td>
<td>0.02</td>
<td>Gervas et al. [18]</td>
</tr>
<tr>
<td>( a_h )</td>
<td>0.083</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( \beta_h )</td>
<td>0.009</td>
<td>Moore et al. [4]</td>
</tr>
<tr>
<td>( \sigma_h )</td>
<td>0.004</td>
<td>Gervas et al. [18]</td>
</tr>
<tr>
<td>( k )</td>
<td>0.62</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( \Lambda_c )</td>
<td>22</td>
<td>Otieno et al. [7]</td>
</tr>
<tr>
<td>( \mu_c )</td>
<td>0.00055</td>
<td>Kajunguri et al. [8]</td>
</tr>
<tr>
<td>( \alpha_c )</td>
<td>0.06</td>
<td>Otieno et al. [7]</td>
</tr>
<tr>
<td>( \sigma_c )</td>
<td>0.083</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( \beta_c )</td>
<td>0.0083</td>
<td>Moore et al. [4]</td>
</tr>
<tr>
<td>( \psi_c )</td>
<td>0.013</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( d )</td>
<td>0.62</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( \rho )</td>
<td>0.7</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>0.03</td>
<td>Kajunguri et al. [8]</td>
</tr>
<tr>
<td>( \omega )</td>
<td>0.04</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \Lambda_v )</td>
<td>120</td>
<td>Assumed</td>
</tr>
<tr>
<td>( b )</td>
<td>0.33</td>
<td>Meisner et al. [9]</td>
</tr>
<tr>
<td>( g )</td>
<td>0.01</td>
<td>Ndondo et al. [10]</td>
</tr>
<tr>
<td>( z )</td>
<td>0.1</td>
<td>Assumed</td>
</tr>
<tr>
<td>( \alpha_v )</td>
<td>0.04</td>
<td>Meisner et al. [9]</td>
</tr>
</tbody>
</table>
human and cattle increases, the number of infected humans and cattle reduced with time, as shown in Figures 5(a) and 5(b). It indicates that treating the infected humans and cattle has a significant impact on controlling the African trypanosomiasis disease as it reduces the number of sick individuals in the population. Since tsetse flies depend only on blood for their survival, reducing the number of infected humans and cattle by treating them reduces the probability of tsetse flies biting the infected human/cattle, hence reducing the disease’s spread.

Figure 6 shows the effect of varying tsetse fly trapping rates on infected human and cattle populations while
keeping human/cattle treatment and public health education rates constant. It is clear from Figures 6(a) and 6(b) that increasing the trapping tsetse flies’ rates has a great contribution toward African trypanosomiasis elimination. It reduces the number of infected humans and cattle in the population. The reason behind this is that tsetse fly trapping reduces the density of tsetse flies in the population. As the number of vectors is reduced in the population, the contact rate of vector to human/cattle is also reduced.

Figure 7 shows the effect of varying public health education rates while constantly fixing trapping and human/cattle treatment rates. As more individuals become aware of the disease and use protective measures such as insect repellent solution, wearing long-sleeved clothes, and clearing their environment, the number of infected humans is diminished in the population with time. This scenario shows that if public health education campaigns are properly conducted, particularly in endemic areas, there is a plausibility of reducing disease transmission, as it reduces the vector human-contact rate.

From Figure 8(a), we observe that as the rates of public health education increase, the number of susceptible uneducated individuals decreases as well. This implies that more people have become aware of the disease and join the susceptible educated population. On the contrary, as public health education rates increase, the number of susceptible educated individuals increases, as shown in Figure 8(b). This indicates that more people have become aware of the disease and avoid contact with the tsetse fly by wearing long-sleeved clothes, using insect repellent, and clearing their surroundings.

Figure 9 indicates the effect of varying the efficacy of public health education on the susceptible educated human population. It is clear that as the efficacy of public health education increases, the number of susceptible educated human population increases with time. It means that increasing the public health education efficacy rates reduces the number of people contacting tsetse flies and hence reducing the number of people joining the exposed class.

5.3. Effects of Interventions on Effective Reproduction Number. When only one intervention is used to control African trypanosomiasis in a population, it is observed that the disease will not be cleared out since the effective reproduction number is greater than the unit, as shown in Figure 10. The tsetse fly trapping seems to be the best control as its effective reproduction number is lower than treatment and public health education. This observation indicates that, for the African trypanosomiasis to be eliminated in the population, a combination of interventions should be considered rather than the application of a single control method.

From Figure 11, we see that the effective reproduction number increases as the vector biting rates increases. When only two interventions are used, treating the infectious human/cattle and using tsetse fly trapping seem to be effective at reducing the threshold, $R_c$ compared to a combination of public health education and tsetse fly trapping, as well as treatment and public health education. The effective reproduction number seems to be very high when only a combination of treatment and public health education is in place. This is because treatment is only applied to sick human/cattle, preventing new cases from occurring. Public health education helps prevent new cases from occurring in
Figure 7: Variation in public health education on infected human population.

Figure 8: Variation in public health education rate on susceptible human population.
a human population, but it does not prevent new cases from occurring in the cattle populations. It can also be observed from Figure 11 that a combination of all three interventions (treatment, public health education, and tsetse fly trapping) is the best strategy for eliminating African trypanosomiasis as it adequately reduces the effective reproduction number than when only two interventions are used.

6. Conclusion

In this study, we formulated and analyzed the African trypanosomiasis model with interventions. The model consists of three interventions, namely, public health education for humans, trapping for tsetse flies, and treatment for humans and cattle. Human, cattle, and vector populations were subdivided into different classes concerning their disease status. The invariant set was derived, and the model’s solution was found to be biologically and mathematically meaningful by using the theory of differential equations. We computed the threshold $R_e$ and used it to discuss the local and global stability of the equilibria points. The disease-free equilibrium point was established, and by using the effective reproduction number, its stability was also investigated. The disease-free equilibrium point was locally asymptotically stable when the reproduction number is less than one and unstable when the reproduction number is greater than one. By applying the Metzler stability theory, the disease-free equilibrium point was globally asymptotically stable when the reproduction number is less than one. The sensitivity analysis shows that the control measures based on public health education, tsetse fly trapping, and human and cattle treatment have negative values because increasing them reduces disease transmission in a community. The numerical simulations showed that the combination of all three interventions (treatment, public health education, and
trapping) considerably cleared out the population’s disease burden faster than using only two interventions. Furthermore, the numerical results showed that, with an increase in public health education rates against African trypanosomiasis disease, the number of susceptible educated individuals increases gradually. We recommend that, to keep the disease under control, public health education campaigns through seminars and media like television, radio, magazines, and mobile networks should be spread, especially in the rural areas, to make people aware of the disease. We also recommend that the community, especially those living in endemic areas, should be encouraged to use tsetse traps as they are cheap. They have shown a great impact on tsetse control and trypanosomiasis elimination. The model presented in this study is not exhaustive. Therefore, the assumptions made during model formulation can be relaxed to incorporate the aspect of infected immigrants, age structure, climatic change, and the cost-effectiveness of the control strategies.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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References