Hindawi
International Journal of Mathematics and Mathematical Sciences
Volume 2020, Article ID 3690472, 15 pages
https://doi.org/10.1155/2020/3690472

Research Article

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

Yustina A. Liana,[1] **Nyimvua Shaban**,[1] **Goodluck Mlay**,[1] **and Anitha Phibert**[2]

[1]*Department of Mathematics, University of Dar es Salaam, Dar es Salaam, Tanzania*
[2]*Department of Zoology and Wildlife Conservation, University of Dar es Salaam Postal address, P.O.Box 35062, Dar es Salaam, Tanzania*

Correspondence should be addressed to Yustina A. Liana; lianayustina@yahoo.com

Received 21 May 2020; Revised 2 November 2020; Accepted 12 November 2020; Published 26 November 2020

Academic Editor: Raúl E. Curto

Copyright © 2020 Yustina A. Liana et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

African trypanosomiasis is a vector-borne disease that is mainly transmitted by infected tsetse flies. A deterministic model of tsetse fly vector, human, and cattle hosts is formulated and analyzed to gain insights into the disease dynamics. The roles of public health education, treatment, and tsetse fly traps are studied. The effective reproduction number, a threshold used to determine whether the disease persists or dies out in the population, is determined. The sensitivity analysis of the model parameters is performed to determine their relationship with the effective reproduction number. The results show that the tsetse fly biting rate is the most sensitive parameter to the effective reproduction number. Furthermore, the model's numerical simulation shows that a combination of all three interventions has the most significant impact on the control of African trypanosomiasis. Thus, we recommend that these control measures be put concurrently in endemic areas for effective control of the disease transmission.

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 $S_u(t)$, 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 $N_h(t) = S_u(t) + S_e(t) + E_h(t) + I_h(t) + R_h(t)$. Human individuals are recruited into the population at a constant rate of Λ_h. We assume that education strategy is implemented at the rate θ 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 λ_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 ϵ 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 μ_h. Exposed humans become infectious at a rate α_h, and the infectious humans leave the infected class through natural death, disease-induced death, or recovery at the rates μ_h, σ_h, and β_h, respectively. Individuals in the recovered class may leave the compartment by either natural death at a rate of μ_h or through losing temporary immunity and move to the susceptible class at a rate of ψ_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:

$$\begin{cases} \dfrac{dS_u}{dt} = \Lambda_h + \psi_h R_h - \lambda_h S_u - (\mu_h + \theta) S_u, \\[2ex] \dfrac{dS_e}{dt} = \theta S_u - (1-\epsilon)\lambda_h S_e - \mu_h S_e, \\[2ex] \dfrac{dE_h}{dt} = \lambda_h S_u + (1-\epsilon)\lambda_h S_e - (\mu_h + \alpha_h) E_h, \\[2ex] \dfrac{dI_h}{dt} = \alpha_h E_h - (\mu_h + \sigma_h + \beta_h) I_h, \\[2ex] \dfrac{dR_h}{dt} = \beta_h I_h - (\mu_h + \psi_h) R_h, \end{cases} \tag{1}$$

where λ_h is the force of infection given by $\lambda_h = (1-\rho) bkI_v/N_h$, with k as the probability that the infectious vector infects a susceptible individual, b is the tsetse fly blood-feeding rate per day, and ρ 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 Λ_c. Cattle leave the susceptible class through natural death at a rate of μ_c or by getting infected and joining the exposed class at λ_c. The exposed cattle become infectious and move to the infected class at a rate of α_c. The infectious cattle leave the infected class through natural death, disease-induced death, or recovery at the rates μ_c, σ_c, and β_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 μ_c or through the waning of temporary immunity and move to susceptible class at a rate ψ_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:

$$\begin{cases} \frac{dS_c}{dt} = \Lambda_c + \psi_c R_c - \lambda_c S_c - \mu_c S_c, \\ \frac{dE_c}{dt} = \lambda_c S_c - (\mu_c + \alpha_c) E_c, \\ \frac{dI_c}{dt} = \alpha_c E_c - (\mu_c + \sigma_c + \beta_c) I_c, \\ \frac{dR_c}{dt} = \beta_c I_c - (\mu_c + \psi_c) R_c, \end{cases} \quad (2)$$

where λ_c is the force of infection given by $\lambda_c = b\,d\rho I_v / N_c$ 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 Λ_v. We assume that the tsetse fly population, regardless of its status, dies naturally at a rate of μ_v or by being trapped at a rate of ω. The tsetse flies move to exposed class after acquiring the infection at a rate of λ_v, and also, the infected flies progress to being infectious at a rate of α_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:

$$\begin{cases} \frac{dS_v}{dt} = \wedge_v - \lambda_v S_v - (\mu_v + \omega) S_v, \\ \frac{dE_v}{dt} = \lambda_v S_v - (\mu_v + \omega + \alpha_v) E_v, \\ \frac{dI_v}{dt} = \alpha_v E_v - (\mu_v + \omega) I_v, \end{cases} \quad (3)$$

where $\lambda_v = (1-\rho) bgI_h/N_h + \rho bzI_c/N_c$ 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).

Variable	Descriptions
S_u	Susceptible uneducated human
S_e	Susceptible educated human
E_h	Exposed human
I_h	Infected human
R_h	Recovered human
S_c	Susceptible cattle
λ_h	Force of infection for human
λ_c	Force of infection for cattle
E_c	Exposed cattle
I_c	Infected cattle
R_c	Recovered cattle
S_v	Susceptible vector
E_v	Exposed vector
I_v	Infected vector
λ_v	Force of infection for vector

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

$$\begin{cases} \frac{dE_h}{dt} = \lambda_h S_u + (1-\epsilon)\lambda_h S_e - (\mu_h + \alpha_h) E_h, \\ \frac{dI_h}{dt} = \alpha_h E_h - (\mu_h + \sigma_h + \beta_h) I_h, \\ \frac{dR_h}{dt} = \beta_h I_h - (\mu_h + \psi_h) R_h, \\ \frac{dS_c}{dt} = \Lambda_c + \psi_c R_c - \lambda_c S_c - \mu_c S_c, \\ \frac{dE_c}{dt} = \lambda_c S_c - (\mu_c + \alpha_c) E_c, \\ \frac{dI_c}{dt} = \alpha_c E_c - (\mu_c + \sigma_c + \beta_c) I_c, \\ \frac{dR_c}{dt} = \beta_c I_c - (\mu_c + \psi_c) R_c, \\ \frac{dS_v}{dt} = \Lambda_v - \lambda_v S_v - (\mu_v + \omega) S_v, \\ \frac{dE_v}{dt} = \lambda_v S_v - (\mu_v + \omega + \alpha_v) E_v, \\ \frac{dI_v}{dt} = \alpha_v E_v - (\mu_v + \omega) I_v. \end{cases} \quad (4)$$

The corresponding initial conditions are $S_u(0) \geq 0$, $S_e(0) \geq 0, R_h(0) \geq 0, S_c(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0,$

Table 2: Description of parameters of model (4).

Parameter	Descriptions
Λ_h	Human birth rate
μ_h	Human natural death rate
ϵ	Education efficacy
ψ_h	Immunity waning rate for the recovered human
ψ_c	Immunity waning rate for the recovered cattle
α_h	Human incubation rate
α_c	Cattle incubation rate
α_v	Vector incubation rate
β_h	Human recovery rate/treatment rate
β_c	Cattle recovery rate/treatment rate
θ	Rate at which humans become aware of the disease
z	Probability that a vector becomes infected after biting infected cattle
σ_h	Human death rate due to disease
σ_c	Cattle death rate due to disease
Λ_c	Cattle birth rate
μ_c	Cattle natural death rate
μ_v	Vector natural death rate
Λ_v	Vector recruitment rate
ω	Vector death rate due to trapping
d	Probability that infectious vector infects cattle
b	Vector biting rate per day
k	Probability that infectious vector infects human
g	Probability that a vector becomes infected by biting infected human
ρ	Proportion of vector feeding on cattle

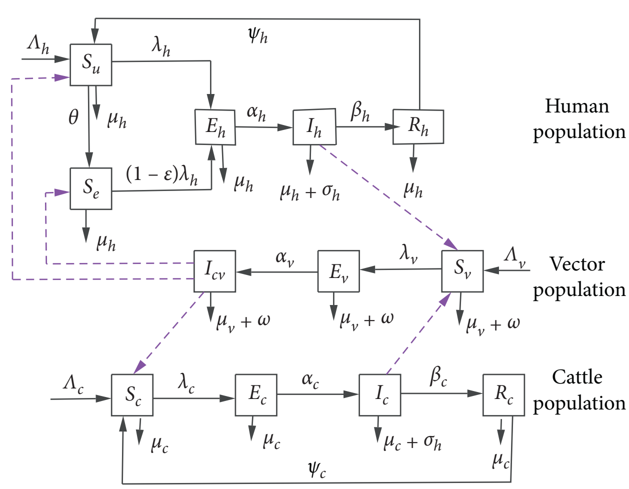

Figure 1: Flow diagram for the dynamics of African trypanosomiasis.

$E_c(0) \geq 0, I_c(0) \geq 0,$ and $R_c(0) \geq 0, S_v(0) \geq 0, E_v(0) \geq 0,$ $I_v(0) \geq 0.$

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* $\Omega_h = \{S_u, S_e, E_h, I_h, R_h \in \mathbb{R}_+^5, S_u + S_e + E_h + I_h + R_h = N_h \leq \Lambda_h/\mu_h\}$, $\Omega_c = \{S_c, E_c, I_c, R_c, S_c + E_c + I_c + R_c = N_c \leq \Lambda_c/\mu_c\}$, *and* $\Omega_v = \{S_v, E_v, I_v, S_v + E_v + I_v = N_v \leq \Lambda_v/\mu_v\}$. *It is sufficient to consider* *the solutions in Ω 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,

$$\frac{\mathrm{d}N_h}{\mathrm{d}t} = \Lambda_h - \mu_h N_h - \sigma_h I_h + . \quad (5)$$

For cattle population,

$$\frac{\mathrm{d}N_c}{\mathrm{d}t} = \Lambda_c - \mu_c N_c - \sigma_c I_c \leq \Lambda_c - \mu_c N_c. \quad (6)$$

For tsetse fly population,

$$\frac{\mathrm{d}N_v}{\mathrm{d}t} = \Lambda_v - (\mu_v + \omega) N_v \leq \Lambda_v - \mu_v N_v. \quad (7)$$

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

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} - \left(\frac{\Lambda_h - \mu_h N_h(0)}{\mu_h}\right) e^{-\mu_h t} \longrightarrow N_h(t) \leq \frac{\Lambda_h}{\mu_h} \text{ as } t \longrightarrow \infty, \quad (8)$$

$$N_c(t) \leq \frac{\Lambda_c}{\mu_c} - \left(\frac{\Lambda_c - \mu_c N_c(0)}{\mu_c}\right) e^{-\mu_c t} \longrightarrow N_c(t) \leq \frac{\Lambda_c}{\mu_c} \text{ as } t \longrightarrow \infty, \quad (9)$$

$$N_v(t) \leq \frac{\Lambda_v}{\mu_v} - \left(\frac{\Lambda_v - \mu_v N_v(0)}{\mu_v}\right) e^{-\mu_v t} \longrightarrow N_v(t) \leq \frac{\Lambda_v}{\mu_v} \text{ as } t \longrightarrow \infty. \quad (10)$$

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

$$\begin{aligned} \Omega_h &= \left\{ S_u, S_e, E_h, I_h, R_h \in \mathbb{R}_+^5, S_u + S_e + E_h + I_h + R_h = N_h \leq \frac{\Lambda_h}{\mu_h} \right\}, \\ \Omega_c &= \left\{ S_c, E_c, I_c, R_c, S_c + E_c + I_c + R_c = N_c \leq \frac{\Lambda_c}{\mu_c} \right\}, \text{ and} \\ \Omega_v &= \left\{ S_v, E_v, I_v, S_v + E_v + I_v = N_v \leq \frac{\Lambda_v}{\mu_v} \right\}. \end{aligned} \quad (11)$$

The results imply that the region is bounded, well posed, and biologically meaningful as it attracts all solutions in Ω. □

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

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

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

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{bk\theta B_1 B_2 \Lambda_h}{A_1 \left(\Lambda_h/A_1 + t\theta\Lambda_h/A_1\mu_h\right)\mu_h} + \frac{bkB_2\Lambda_h}{A_1 \left(\Lambda_h/A_1 + t\theta\Lambda_h/A_1\mu_h\right)} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & b\,d\rho \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{bgB_2\Lambda_V}{A_8 \left(\Lambda_h/A_1 + t\theta\Lambda_h/A_1\mu_h\right)} & 0 & \frac{bz\rho\Lambda_V\mu_c}{A_8\Lambda_c} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} A_2 & 0 & 0 & 0 & 0 & 0 \\ -\alpha_h & A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & A_5 & 0 & 0 & 0 \\ 0 & 0 & -\alpha_c & A_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & A_9 & 0 \\ 0 & 0 & 0 & 0 & -\alpha_v & A_8 \end{pmatrix}, \tag{13}$$

where $A_1 = \mu_h + \theta$, $A_2 = \mu_h + \alpha_h$, $A_3 = \mu_h + \sigma_h + \beta_h$, $A_4 = \mu_h + \psi_h$, $A_5 = \mu_c + \alpha_c$, $A_6 = \mu_c + \sigma_c + \beta_c$, $A_7 = \mu_c + \psi_c$, $A_8 = \mu_v + \omega$, $A_9 = \mu_v + \omega + \alpha_v$, $B_1 = 1 - \epsilon$, and $B_2 = 1 - \rho$.

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

$$\begin{aligned} R_e &= \sqrt{\frac{b^2\alpha_v\Lambda_v\left(A_1A_5A_6B_2^2gk\Lambda_c\alpha_h\mu_h\left(B_1\theta + \mu_h\right) + A_2A_3d\rho^2 z\alpha_c\mu_c\Lambda_h\left(\mu_h + \theta\right)^2\right)}{A_2A_3A_5A_6A_8^2A_9\Lambda_c\Lambda_h\left(\mu_h + \theta\right)^2}}, \\ &= \sqrt{\frac{b^2\alpha_v\alpha_c d\rho^2\Lambda_v\mu_c z}{\left(\mu_v + \omega\right)^2\Lambda_c\left(\mu_c + \alpha_c\right)A_6A_9} + \frac{b^2\Lambda_v\alpha_v\alpha_h\left(1-\rho\right)^2 gk\mu_h\left(\mu_h + (1-\epsilon)\theta\right)}{\left(\mu_v + \omega\right)^2\Lambda_h\left(\mu_h + \alpha_h\right)\left(\mu_h + \theta\right)A_3A_9}}, \\ &= \sqrt{R_{ec} + R_{eh}}, \end{aligned} \tag{14}$$

where R_{ec} represents the effective reproduction number for cattle and R_{eh} 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 $R_e > 1$ and it dies out if $R_e < 1$. The following theorem summarizes the result.

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

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 $R_e < 1$. In other words, the introduction of infectious individuals into a population of susceptible individuals does not induce an epidemic outbreak. On the contrary, if $R_e > 1$, 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:

$$\begin{cases} \dfrac{dY_n}{dt} = A_1\left(Y_n - Y_{Eo}\right) + A_2 Y_i, \\ \\ \dfrac{dY_i}{dt} = A_3 Y_i, \end{cases} \quad (15)$$

where Y_n is a vector of nontransmitting variables, Y_{Eo} 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

$$Y_n - Y_{Eo} = \begin{bmatrix} S_u \\ S_e \\ R_h \\ S_c \\ R_c \\ S_v \end{bmatrix} - \begin{bmatrix} \dfrac{\Lambda_h}{\theta + \mu_h} \\ \dfrac{\theta}{\mu_h}\left(\dfrac{\Lambda_h}{\theta + \mu_h}\right) \\ 0 \\ \dfrac{\Lambda_c}{\mu_c} \\ 0 \\ \dfrac{\Lambda_v}{\mu_v + \omega} \end{bmatrix} = \begin{bmatrix} S_u - \dfrac{\Lambda_h}{\theta + \mu_h} \\ S_e - \dfrac{\theta}{\mu_h}\left(\dfrac{\Lambda_h}{\theta + \mu_h}\right) \\ R_h \\ S_c - \dfrac{\Lambda_c}{\mu_c} \\ R_c \\ S_v - \dfrac{\Lambda_v}{\mu_v + \omega} \end{bmatrix}. \quad (16)$$

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 $A_1(i, j) \geq 0$ for all indices of $i \neq j$). That is,

$$A_1 = \frac{\partial Y_n}{\partial\left(S_u, S_e, R_h, S_c, R_c, S_v\right)} = \begin{pmatrix} -(\theta + \mu_h) & 0 & \psi_h & 0 & 0 & 0 \\ \theta & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu_h + \psi_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mu_c & \psi_c & 0 \\ 0 & 0 & 0 & 0 & -(\mu_c + \psi_c) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\mu_v + \omega) \end{pmatrix}, \quad (17)$$

with eigenvalues $\lambda_1 = -\mu_h$, $\lambda_2 = -(\theta + \mu_h)$, $\lambda_3 = -(\mu_h + \psi_h)$, $\lambda_4 = -\mu_c$, $\lambda_5 = -(\mu_c + \psi_c)$, and $\lambda_6 = -(\mu_v + \omega)$. Also,

$$A_2 = \frac{\partial Y_n}{\partial(E_h, I_h, E_c, I_c, E_v, I_v)} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \frac{-(1-\rho)bkS_u}{N_h} \\ 0 & 0 & 0 & 0 & 0 & \frac{-(1-\epsilon)bkS_e}{N_h} \\ 0 & \beta_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{-b\,d\rho\,S_c}{N_c} \\ 0 & 0 & 0 & \beta_c & 0 & 0 \\ 0 & \frac{-(1-\rho)bgS_v}{N_h} & 0 & \frac{-\rho bzS_v}{N_c} & 0 & -0 \end{pmatrix},$$

$$A_3 = \frac{\partial Y_i}{\partial(E_h, I_h, E_c, I_c, E_v, I_v)} = \begin{pmatrix} -P_1 & 0 & 0 & 0 & 0 & \frac{(1-\rho)bk(S_u + (1-\epsilon)S_e)}{N_h} \\ \alpha_h & -P_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_3 & 0 & 0 & \frac{b\,d\rho\,S_c}{N_c} \\ 0 & 0 & \alpha_c & -P_4 & 0 & 0 \\ 0 & \frac{(1-\rho)bgS_v}{N_h} & 0 & \frac{\rho bzS_v}{N_c} & -P_5 & 0 \\ 0 & 0 & 0 & 0 & \alpha_v & -P_6 \end{pmatrix}, \quad (18)$$

where $P_1 = (\mu_h + \alpha_h)$, $P_2 = (\mu_h + \sigma_h + \beta_h)$, $P_3 = (\mu_c + \alpha_c)$, $P_4 = (\mu_c + \sigma_c + \beta_c)$, $P_5 = (\mu_v + \omega + \alpha_v)$, and $P_6 = (\mu_v + \omega)$. It can be seen that matrix A_1 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

$$\frac{dY_n}{dt} = A_1(Y_n - Y_{Eo}) + A_2 Y_i \quad (19)$$

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 $\Upsilon_p^{R_e} = \partial R_e/\partial p \times p/R_e$, 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 α_v is given as $\Upsilon_{\alpha_v}^{R_e} = \partial R_e/\partial\alpha_v \times \alpha_v/R_e = +0.3182$. 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 $\alpha_h, \alpha_c, \Lambda_v, b, k, g, z, d, \rho$, and α_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 $\mu_h, \mu_c, \Lambda_h, \theta, \epsilon, \beta_h, \sigma_h, \Lambda_c, \sigma_c, \beta_c, \mu_v$, and ω have negative indices, implying that increasing one of these parameters and

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

Parameter	Index value	Parameter	Index value
Λ_h	−0.0036	β_c	−0.2775
θ	−0.0638	α_v	+0.3182
μ_h	−0.0042	μ_v	−0.5649
ϵ	−0.034	ω	−0.7532
α_h	+0.000192	Λ_v	+0.5
β_h	−0.025	b	+1
σ_h	−0.0011	k	+0.0036
Λ_c	−0.4964	g	+0.0036
μ_c	−0.4748	z	+0.4964
σ_c	−0.2006	d	+0.4964
α_c	+0.0033	ρ	+0.9761

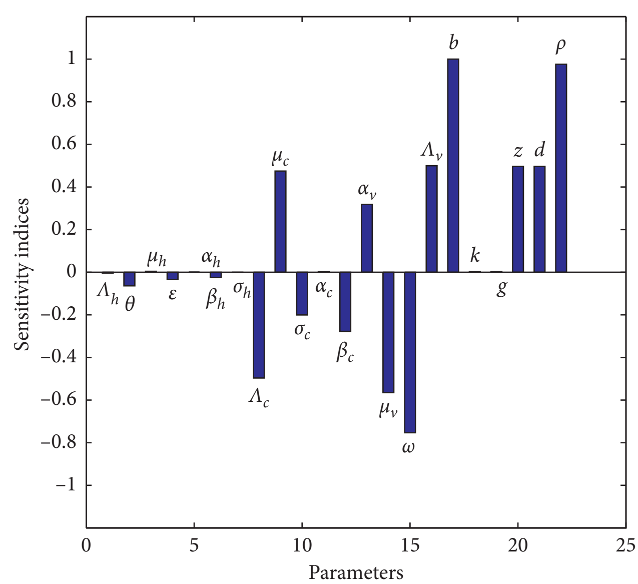

Figure 2: Sensitivity analysis of R_e using results in Table 3.

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 ρ, the rate at which the tsetse flies die due to trapping ω, and tsetse fly natural mortality rate μ_v, followed by the tsetse fly recruitment rate Λ_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: $S_u = 200$, $S_e = 180$, $E_h = 150$, $I_h = 100$, $R_h = 80$, $S_c = 200$, $E_c = 170$, $I_c = 140$, $R_c = 100$, $S_v = 3000$, $E_v = 800$, 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 4: Epidemiological data for model (4).

Parameter	Values (days)	Source
Λ_h	27.5	Kajunguri et al. [8]
θ	0.0002	Misra et al. [17]
μ_h	0.000046	Assumed
ϵ	0.6	Assumed
ψ_h	0.02	Gervas et al. [18]
α_h	0.083	Ndondo et al. [10]
β_h	0.009	Moore et al. [4]
σ_h	0.004	Gervas et al. [18]
k	0.62	Ndondo et al. [10]
Λ_c	22	Otieno et al. [7]
μ_c	0.00055	Kajunguri et al. [8]
σ_c	0.006	Otieno et al. [7]
α_c	0.083	Ndondo et al. [10]
β_c	0.0083	Moore et al. [4]
ψ_c	0.013	Ndondo et al. [10]
d	0.62	Ndondo et al. [10]
ρ	0.7	Ndondo et al. [10]
μ_v	0.03	Kajunguri et al. [8]
ω	0.04	Assumed
Λ_v	120	Assumed
b	0.33	Meisner et al. [9]
g	0.01	Ndondo et al. [10]
z	0.1	Assumed
α_v	0.04	Meisner et al. [9]

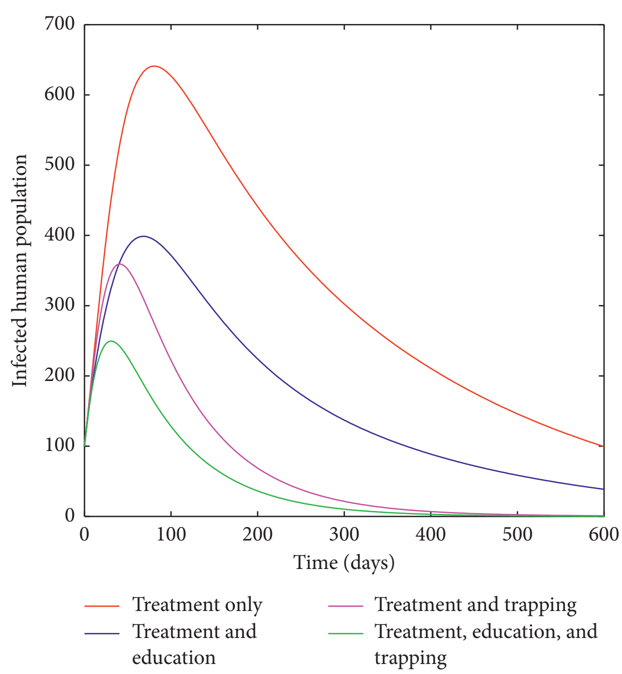

Figure 3: Impact of various interventions on infected human population when $\theta = 0.5$.

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

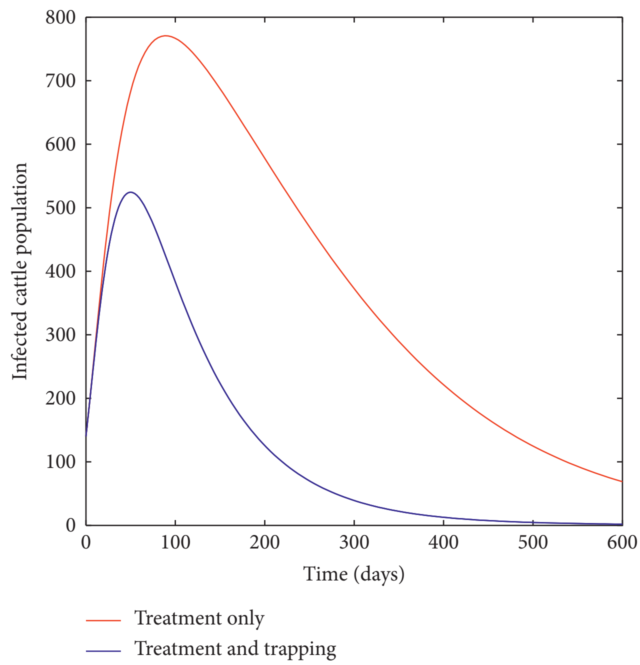

Figure 4: Impact of interventions on infected cattle population.

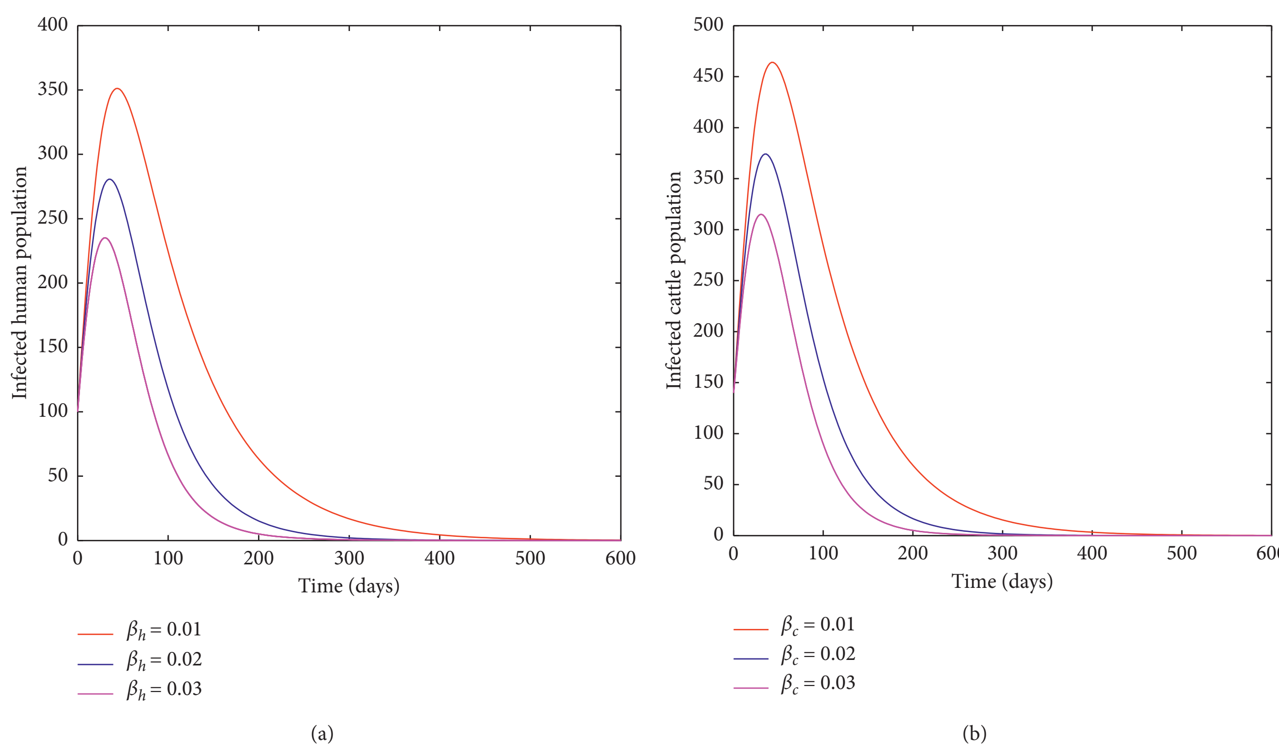

Figure 5: Variation in treatment rate on infected human and cattle population.

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

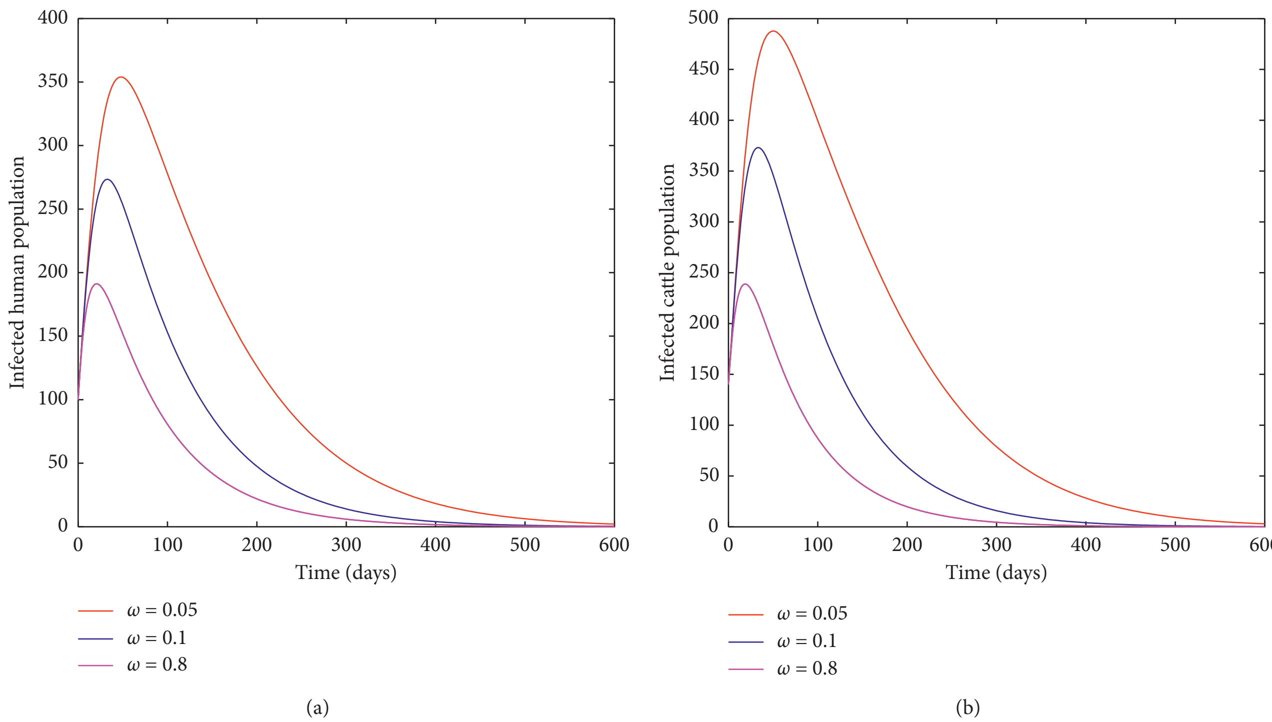

FIGURE 6: Variation in trapping rate on infected human and cattle population.

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_e 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

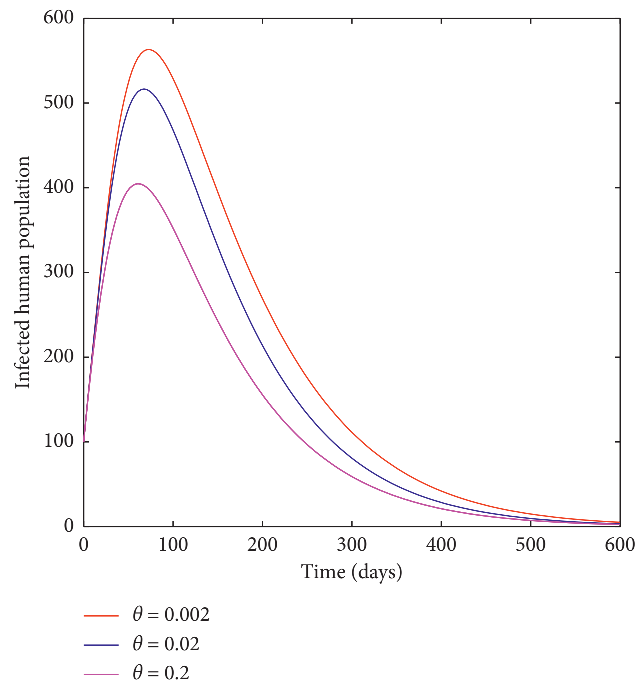

Figure 7: Variation in public health education on infected human population.

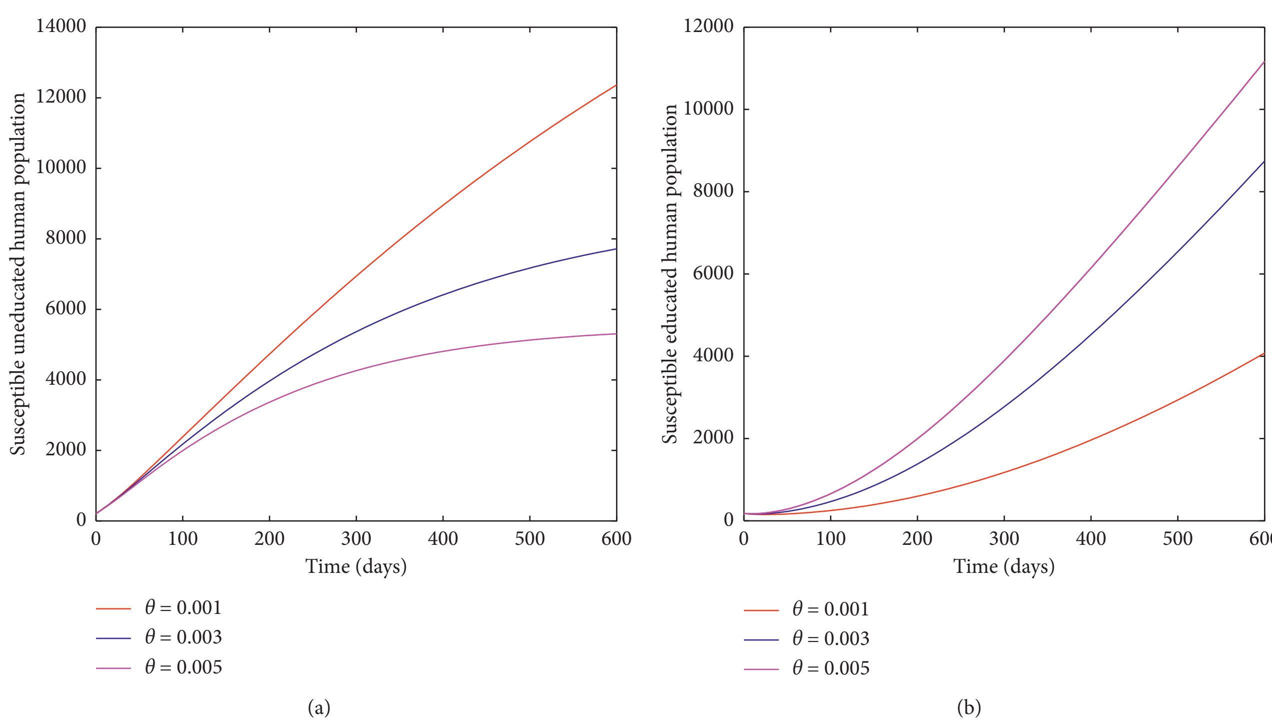

Figure 8: Variation in public health education rate on susceptible human population.

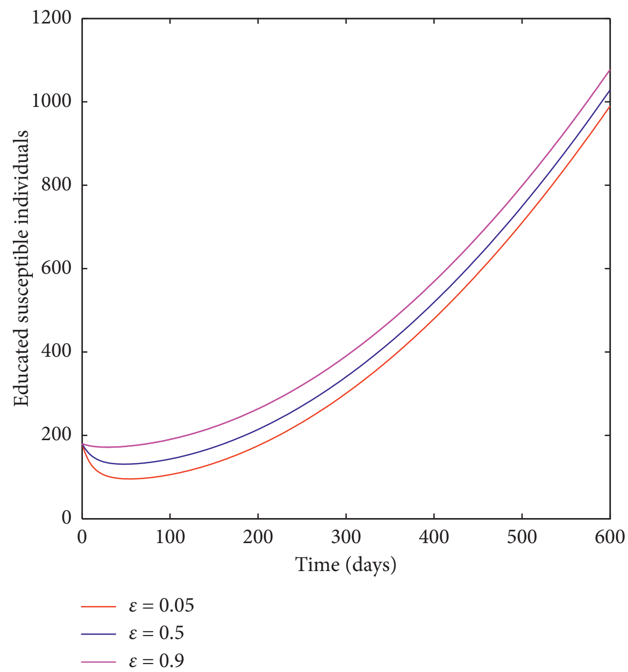

Figure 9: Variation in efficacy of public health education on susceptible human population.

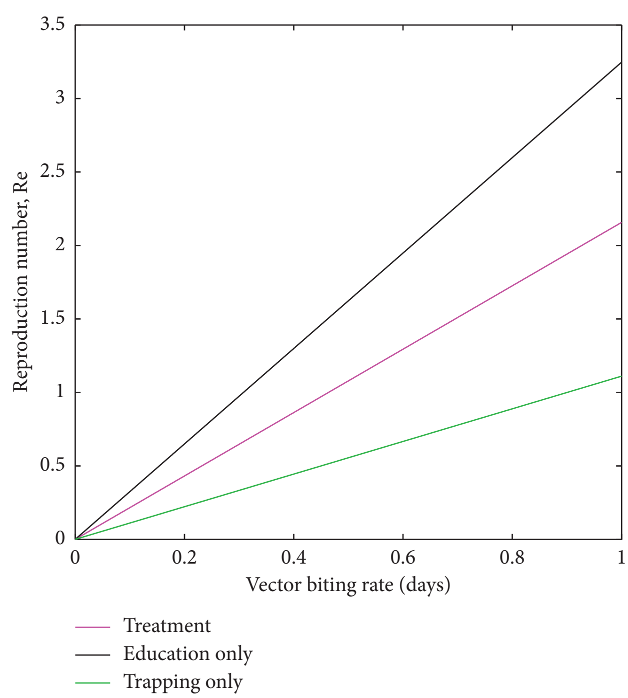

Figure 10: Effects of single intervention on effective reproduction number with respect to vector biting rate.

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)

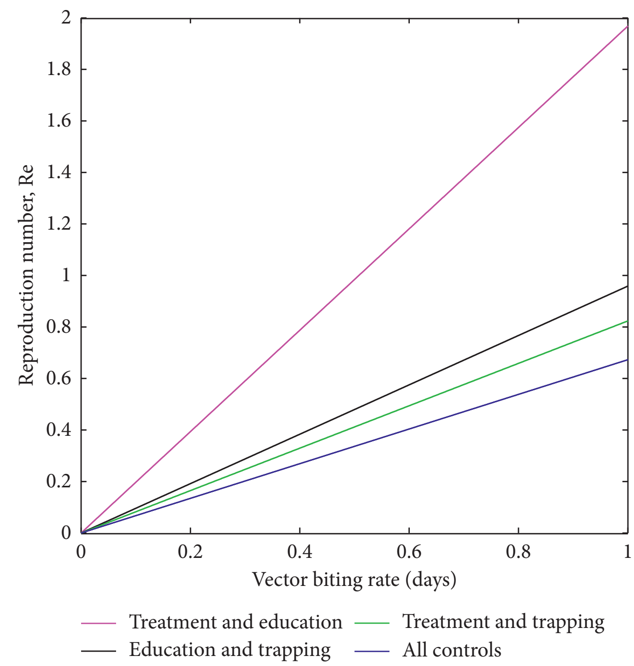

Figure 11: Effects of combining interventions on effective reproduction number with respect to vector biting rate.

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.

Acknowledgments

The authors are grateful to the University of Dar Salaam (UDSM) administration for providing facilities during the study.

References

[1] N. E. Anderson, J. Mubanga, E. M. Fevre et al., "Characterisation of the wildlife reservoir community for human and animal trypanosomiasis in the luangwa valley, Zambia," *PLoS Neglected Tropical Diseases*, vol. 5, no. 6, p. e1211, 2011.

[2] J. Pepin and H. Meda, "The epidemiology and control of human African trypanosomiasis," *Advances in Parasitology*, vol. 49, pp. 72–132, 2001.

[3] K. S. Rock, C. M. Stone, I. M. Hastings, M. J. Keelin, S. J. Torr, and N. Chitnis, "Mathematical models of human African trypanosomiasis epidemiology," *Advances in Parasitology*, vol. 87, pp. 53–133, 2015.

[4] S. Moore, S. Shrestha, K. W. Tomlinson, and H. Vuong, "Predicting the effect of climate change on African trypanosomiasis: integrating epidemiology with parasite and vector biology," *Journal of the Royal Society Interface*, vol. 9, no. 70, pp. 817–830, 2011.

[5] A. P. M. Shaw, S. J. Torr, C. Waiswa et al., "Estimating the costs of tsetse control options: an example for Uganda," *Preventive Veterinary Medicine*, vol. 110, no. 3-4, pp. 290–303, 2013.

[6] E. Prowse, "Trypanosomosis, the disease and its control: an analysis of a new tsetse-repellent technology," Doctoral Dissertation, Sveriges Lantbruksuniv, Garpenberg, Sweden, 2005.

[7] J. Otieno, J. Y. T. Mugisha, B. K. Nannyonga, and P. Oleche, "Parameter driven dynamics of trypanosomiasis in a cattle population," *Applied Mathematical Sciences*, vol. 8, no. 54, pp. 2665–2685, 2014.

[8] D. Kajunguri, J. W. Hargrove, R. Ouifki, J. Y. T. Mugisha, P. G. Coleman, and S. C. Welburn, "Modelling the use of insecticide-treated cattle to control tsetse and Trypanosoma brucei rhodesiense in a multi-host population," *Bulletin of Mathematical Biology*, vol. 76, no. 3, pp. 673–696, 2014.

[9] J. Meisner, R. V. Barnabas, and P. M. Rabinowitz, "A mathematical model for evaluating the role of trypanocide treatment of cattle in the epidemiology and control of Trypanosoma brucei rhodesiense and T. b. gambiense sleeping sickness in Uganda," *Parasite Epidemiology and Control*, vol. 5, Article ID e00106, 2019.

[10] A. M. Ndondo, J. M. W. Munganga, J. N. Mwambakana, C. M. Saad-Roy, P. Van den Driessche, and R. O. Walo, "Analysis of a model of gambiense sleeping sickness in humans and cattle," *Journal of Biological Dynamics*, vol. 10, no. 1, pp. 347–365, 2016.

[11] J. C. Deveze, *Challenges in African Agriculture*, The International Bank for Reconstruction and Development, Washington, DC, USA, 2010.

[12] D. J. Rogers, "A general model for the African trypanosomiases," *Parasitology*, vol. 97, no. 1, pp. 193–212, 1988.

[13] G. Birkhoff and G. Rota, *Ordinary Differential Equations*, Wiley and Sons, Hoboken, NY, USA, 1989.

[14] P. Van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, no. 1-2, pp. 29–48, 2002.

[15] C. Castillo-Chavez, Z. Feng, and W. Huang, C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, and A. A. Yakubu, On the computational of R0 and its role of global stability," *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods and Theory*, Springer, Berlin, Germany, 2002.

[16] N. Chitins, J. M. Hyman, and J. M. Cushing, "Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model," *Bulletin of Mathematical Biology*, vol. 70, pp. 1272–1296, 2008.

[17] A. K. Misra, A. Sharma, and J. B. Shukla, "Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases," *Mathematical and Computer Modelling*, vol. 53, no. 5-6, pp. 1221–1228, 2011.

[18] H. E. Gervas, N. K. D. O. Opoku, and S. Ibrahim, "Mathematical modelling of human african trypanosomiasis using control measures," *Computational and Mathematical Methods in Medicine*, vol. 2018, Article ID 5293568, 13 pages, 2018.