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MATHEMATICAL MODELING AND OPTIMAL CONTROL ANALYSIS OF HUMAN AFRICAN TRYPANOSOMIASIS TRANSMISSION WITH RELAPSE RESPONSE

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ABSTRACT

Human African Trypanosomiasis (HAT), a neglected tropical disease transmitted by tsetse flies, remains a health issue in sub-Saharan Africa. This study develops a seven-compartment mathematical model that includes human and vector dynamics, along with relapse mechanisms. The model's consistency is confirmed through boundedness and positivity analysis. Equilibrium points are calculated, and stability is discussed. The transmission potential via the basic reproduction number, R_0 was derived. An optimal control framework integrates three strategies: public awareness, regular screening and treatment, and vector control with insecticide traps. Using Pontryagin's Principle, cost-effective approaches are identified to reduce infections. Simulations show that combined interventions effectively lower HAT prevalence, with relapse management preventing resurgence. The findings highlight the importance of coordinated, timely strategies and relapse-aware healthcare, providing valuable insights for policymakers to implement resource-efficient measures for HAT elimination

1. INTRODUCTION

Trypanosomiasis, also known as sleeping sickness in humans and Nagana in animals, is an infectious disease caused by protozoan parasites of the genus Trypanosoma, transmitted primarily by the tsetse fly which appears mostly in sub-Saharan Africa. Human African Trypanosomiasis (HAT) is one of the neglected tropical diseases (NTD). It is a vector-borne parasitic disease. The pathogenic parasites are transmitted to humans and animals through the bite of infected tsetse flies, which have acquired their infection from humans and animals that harbor the pathogenic parasite. Trypanosomiasis has a long history as a disease that has claimed many lives, particularly during the era of slave trade and over centuries. However, Sir David Brace, a Scottish pathologist, made a significant breakthrough in 1894 by identifying and discovering the causative agent of the disease in Uganda as Trypanosoma Brucei during an investigation into a disease outbreak.

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Mathematical modeling is a crucial tool in epidemiology, enabling researchers to understand and predict the spread of diseases, evaluate intervention strategies, and inform public health policy decisions. The basic framework for studying the spread of infectious diseases using the SIR (Susceptible-Infected-Recovered) model was first proposed and used by [1]. Subsequently, other researchers introduced, developed, and analyzed additional compartments to fit specific diseases. For example, the SEIR (Susceptible-Exposed-Infected-Recovered) model was developed for diseases with a latency period, such as HIV Hethcote [2], and dynamical studies have been conducted for COVID-19 epidemic models Ayoade and Ibrahim (2022).

[3] and [4], evaluated the effectiveness of containment measures and many others, including (Peter, O. J., Ibrahim, M. O., Edogbanya, H. O., Oguntolu, F. A., Oshinubi & Ibrahim, A. A., .Lawal, 2021), [6], [7] and [8].

Many researchers have extensively investigated the causes, effects, and possible control strategies for Human African Trypanosomiasis (HAT). The World Health Organization (WHO) has set ambitious targets to eliminate both gambiense (gHAT) and rhodesiense (rHAT) forms of the disease by 2030 through elimination of transmission (EoT). Despite significant progress, more efforts are needed to model Trypanosomiasis with optimal control strategies in Africa. Be- low are several notable contributions from various authors in this field: [9] constructed and assessed a comprehensive compart- mental framework integrating tsetse flies, Human, cattle and wildlife, along- side diverse disease control strategies. Each population has several compartments with the variables, $S_j(t)$, $E_j(t)$, $I_j(t)$, and $R_j(t)$, where $j = c, h, w$ represents the number of susceptible, exposed, infected and recovered host at time t, such that the total population of each host is equivalent to $N_j(t) = S_j(t) + E_j(t) + I_j(t) + R_j(t)$. The vector population is divided into three classes such as at time t, there are Susceptible $S_v(t)$, Exposed $E_v(t)$, and Infected $I_v(t)$.

Thus, the total vector population is $N_v(t) = S_v(t) + E_v(t) + I_v(t)$. Their study aimed to evaluate the efficacy of various HAT control approaches in mitigating disease burdens within communities characterized by human-cattle-wildlife interactions. Employing optimal control theory, they identified a synergistic blend of control strategies capable of minimizing both human and cattle infections over time while optimizing implementation costs.

[10] and [11] both analyzed the transmission dynamics of (HAT) and determine optimal control strategies that are both effective in combating the disease and cost-effective. The research evaluated the impact of education campaigns, treatment, and insecticide use on reducing the transmission of HAT in African communities. They employed mathematical modeling techniques and cost-effectiveness analysis; the study seeks to provide insights into the most efficient strategies for eliminating HAT while considering the associated costs and resource allocation.

Numerous studies have proposed improved control strategies for HAT, yet the disease continues to pose a significant health threat to humans and livestock, negatively impacting economic development in Africa.

[12], formulate and analyze mathematical models for the spread of Human African Trypanosomiasis. The study utilized an ordinary differential equation framework to obtain the basic reproductive number (R_0). He also used optimal control model to evaluate the effectiveness of various intervention strategies such as treatment, vector control and public health education in reducing the disease's prevalence and transmission using Pontryagin's principle. He used numerical simulation to highlight interaction between these strategies.

Building on the work of [12], our study introduces a relapse response into the mathematical model, enhancing its realism. Furthermore, we conduct numerical simulations within an optimal control framework. The findings show that combining multiple intervention strategies substantially lowers R_0, while targeted relapse treatments help prevent disease resurgence.

The paper is structured as follows: Section 2 presents a general description of the model formulation, positivity and boundedness of the solution, disease- free and endemic equilibrium points and basic reproduction number. Section 3 focuses on the stability analysis of local disease-free equilibrium state. In Section 4 we considered the optimal control model and provides numerical simulations of the model using a set of data. Finally, Section 5 discusses the findings and presents the conclusions.

2.0 Material and Methods

2.1 Model formulation

A mathematical model that describes the effect of vector-borne diseases on hosts and vectors was developed by dividing the total human population into four compartments: Susceptible human class $S_h(t)$, Exposed human class $E_h(t)$, Infected human class $I_h(t)$, and treated human class $T_h(t)$. The total vector population is divided into three compartments: Susceptible vector class $S_V(t)$, Exposed vector class $E_V(t)$, and Infected vector class $I_V(t)$. Let $N_h(t)$ represent the total populations of host-humans at time *t*, and $N_V(t)$represent the total populations of vectors-tsetse flies at time *t*. Table 1 shows the details of the other parameters used in the schematic diagram of the model (figure 1).

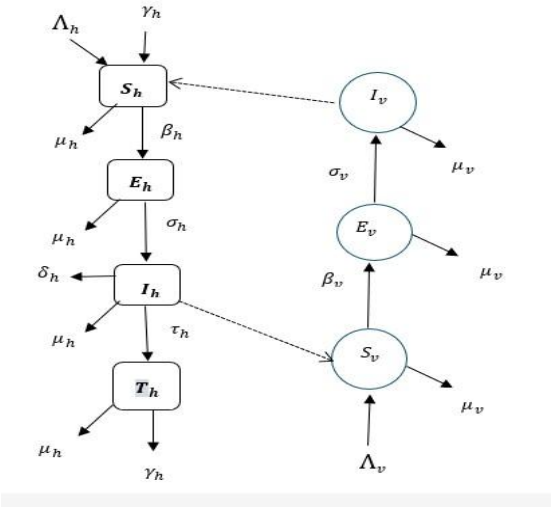

Figure 1: Schematic diagram of the model transmission dynamics

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \gamma_h T_h - \beta_h S_h I_v - \mu_h S_h \\
\frac{dE_h}{dt} &= \beta_h S_h I_v - (\sigma_h + \mu_h) E_h \\
\frac{dI_h}{dt} &= \sigma_h E_h - (\rho_h + \delta_h + \mu_h) I_h \\
\frac{dT_h}{dt} &= \rho_h I_h - (\gamma_h + \mu_h) T_h \\
\frac{dS_v}{dt} &= \Lambda_v - \beta_v S_v I_h - \mu_v S_v \\
\frac{dE_v}{dt} &= \beta_v S_v I_h - (\sigma_v + \mu_v) E_v \\
\frac{dI_v}{dt} &= \sigma_v E_v - \mu_v I_v
\end{aligned}\right\} \quad (1)$$

Table 1: Notation and description of variables and parameters

Notation	Description of variables and parameters
$S_h(t)$	Number of susceptible humans at time *t*
$E_h(t)$	Number of exposed humans at time *t*
$I_h(t)$	Number of infectious humans at time *t*
$T_h(t)$	Number of treated humans at time *t*
$S_v(t)$	Number of susceptible tsetse flies at time *t*
$E_v(t)$	Number of exposed tsetse flies at time *t*
$I_v(t)$	Number of infectious tsetse flies at time *t*
$u_1(t)$	Effect of public awareness and educating the population at time *t*
$u_2(t)$	Regular screening of the population at risk and prompt treatment of infected individuals at time *t*
$u_3(t)$	The impact of vector control using insecticide-treated traps at time *t*
Λ_h	Recruitment rate of human population
Λ_v	Recruitment rate of tsetse fly population
δ_h	Disease-induced death
μ_h	Natural death rate of human population
μ_v	Natural death rate of tsetse fly population
β_h	Rate at which susceptible humans get bitten by an infected tsetse fly
β_v	Rate at which susceptible tsetse flies get a blood meal from an infected person
σ_h	Rate at which the exposed human gets infected
σ_v	Rate at which the exposed tsetse fly gets infected
ρ_h	Rate at which the infected get medical attention as outpatients or hospitalized
γ_h	Rate at which treated human loss immunity and returns to the susceptible class

$$N_h = S_h + E_h + I_h + T_h \quad (2)$$

$$N_v = S_v + E_v + I_v \quad (3)$$

Invariant Region of the Model

By adding the system of equations (1), we have:

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} + \frac{dT_h}{dt} = \Lambda_h - (\delta_h + \mu_h)N_h \quad (4)$$

and

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt} = \Lambda_v - \mu_v N_v \quad (5)$$

Theorem 1

The basic dynamic features of these model equations (1) has solutions which are contain in the feasible region

$\Omega = \Omega_h \times \Omega_v$ for all $t > 0$

Proof

Let

$$\Omega = (S_h, E_h, I_h, T_h, S_v, E_v, I_v) \epsilon \Re_+^7 \quad (6)$$

with non-negative initial conditions using the differential inequality theorem, [13] on equation (4). In the absence of the disease induced death in the human population, equations (4) becomes,

$$\frac{dN_v}{dt} \leq \Lambda_h - \mu_h N_h \quad (7)$$

It follows that

$$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}, \quad (8)$$

hence

$$\Lambda_h - \mu_h N_h \geq K e^{-\mu_h t} \quad (9)$$

Where K is the constant

Similarly, for the vector population equation (5) becomes,

$$\Lambda_v - \mu_v N_v \geq K e^{-\mu_v t} \quad (10)$$

Where K is the constant

Therefore, all feasible solutions of the human and vector of the system model are in the regions:

$$\Omega_h = \{(S_h, E_h, I_h, T_h) \epsilon \Re_+^4 : S_h, E_h, I_h, T_h \geq 0, N_h \leq \Lambda_h - \mu_h N_h\} \quad (11)$$

$$\Omega_v = \{(S_v, E_v, I_v) \epsilon \Re_+^3 : S_v, E_v, I_v \geq 0, N_v \leq \Lambda_v - \mu_v N_v\} \quad (12)$$

Thus, the feasible set of the model is given by

$$\Omega = \{(S_h, E_h, I_h, T_h, S_v, E_v, I_v) \epsilon \Re_+^7 : S_h, E_h, I_h, T_h, S_v, E_v, I_v \geq 0; N_h \leq \frac{\Lambda_h}{\mu_h}, N_v \leq \frac{\Lambda_v}{\mu_v}\} \quad (13)$$

Which is a positively invariant (i.e. solutions remain positive for all time, t) and the model is well pose and biologically meaningful.

2.2 Positivity and Boundedness of the solutions

In this subsection, we show the positivity and boundedness of system equation (1) above.

Lemma 1

Let the initial data be

$\{(S_h(0), E_h(0), I_h(0), T_h(0), S_v(0), E_v(0), I_v(0)) \geq 0\} \epsilon \Omega$

Then, the solution set

$\{S_h(t) + E_h(t) + I_h(t) + T_h(t) + S_v(t) + E_v(0) + I_v(t)\}$ of the system (1) is positive for all $t > 0$.

proof

From the system of equation (1) above, we solve the following

$$\frac{dS_h}{dt} = \Lambda_h + \gamma_h R_h - \beta_h S_h I_v - \mu_h S_h \geq \Lambda_h - \mu_h S_h \quad (14)$$

$$\frac{dS_h}{dt} \geq \Lambda_h - \mu_h S_h \quad (15)$$

$$\frac{dS_h}{dt} + \mu_h S_h \geq \Lambda_h \quad (16)$$

using integrating factor, we have

$$\frac{d}{dt}(S_h e^{\mu_h t}) \geq \Lambda_h e^{\mu_h t} \quad (17)$$

$$S_h(t) e^{e^{\mu_h t}} \geq \frac{\Lambda_h}{\mu_h} e^{\mu_h t} + C \quad (18)$$

$$S_h(t) \geq \frac{\Lambda_h}{\mu_h} + C e^{-\mu_h t} \quad (19)$$

Hence substituting $t = 0$, we have

$$S_h(0) \geq \frac{\Lambda_h}{\mu_h} + C \Rightarrow C \leq S_h(0) - \frac{\Lambda_h}{\mu_h} \quad (20)$$

Which gives,

$$S_h(t) \geq \frac{\Lambda_h}{\mu_h} + (S_h(0) - \frac{\Lambda_h}{\mu_h}) e^{\mu_h t} > 0 \quad (21)$$

From equation (1) also,

$$\frac{dE_h}{dt} = \beta_h S_h I_v - (\alpha_h + \mu_h) E_h \geq -(\alpha_h + \mu_h) E_h \quad (22)$$

$$\int \frac{dE_h}{dt} \geq -\int (\alpha_h + \mu_h) dt \quad (23)$$

Integrating gives:

$$E_h(t) \geq E_h(0) e^{-(\alpha_h + \mu_h)t} > 0 \quad (24)$$

Similarly, from equation (1) we have

$$I_h(t) \geq I_h(0)e^{-(\rho_h+\mu_h)t} > 0$$
(25)
$$T_h(t) \geq T_h(0)e^{-(\gamma_h+\mu_h)t} > 0$$
(26)
$$S_v(t) \geq \frac{\Lambda_v}{\mu_v} + (S_v(0) - \frac{\Lambda_v}{\mu_v})e^{\mu_v t} > 0$$
(27)
$$E_v(t) \geq E_v(0)e^{-(\alpha_v+\mu_v)t} > 0$$
(28)
$$I_v(t) \geq I_v(0)e^{-\mu_v t} > 0$$
(29)

Therefore, all the solutions of the system of equations (1) are positive for all $t > 0$.

2.3 Equilibrium Points of the Model

At equilibrium, we have

$$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dT_h}{dt} = \frac{dS_v}{dt} = \frac{dE_v}{dt} = \frac{dI_v}{dt} = 0$$
(30)

2.3.1 HAT-Free Equilibrium State

Let

$$(S_h, E_h, I_h, T_h, S_v, E_v, I_v) = (S_h^0, E_h^0, I_h^0, T_h^0, S_v^0, E_v^0, I_v^0)$$
(31)

To find the HAT-free equilibrium (HAT-FE) of the given system of differential equations, we set the whole compartment to zero and since the HAT-free equilibrium there are no infected individuals. we set $E_h = I_h = E_v = I_v = 0$ and then solve for the remaining compartments.

Therefore, the system (1) becomes

$$(S_h^0, E_h^0, I_h^0, T_h^0, S_v^0, E_v^0, I_v^0) = \left(\frac{\Lambda_h}{\mu_h}, 0,0,0, \frac{\Lambda_v}{\mu_v}, 0,0\right)$$
(32)

2.3.2 HAT-Endemic Equilibrium State

Let

$$(S_h, E_h, I_h, T_h, S_v, E_v, I_v) = (S_h^*, E_h^*, I_h^*, T_h^*, S_v^*, E_v^*, I_v^*)$$
(33)

and

$$A_1 = (\alpha_h + \mu_h), A_2 = (\rho_h + \delta_h + \mu_h), A_2 = (\gamma_h + \mu_h) \ and \ A_4 = (\alpha_v + \mu_v)$$

Then,

$$(S_h^*, E_h^*, I_h^*, T_h^*, S_v^*, E_v^*, I_v^*) = \begin{pmatrix} \frac{A_1\mu_v}{\beta_h\sigma_v}, \frac{(\Lambda_v\beta_v\sigma_h - \mu_v A_2A_3)A_2}{\sigma_h\beta_v A_2A_3}, \frac{\Lambda_v\beta_v\sigma_h - \mu_v A_2A_3}{\beta_v A_2A_3}, \frac{\gamma_h(\Lambda_v\beta_v\sigma_h - \mu_v A_2A_3)}{\mu_h(\sigma_v\beta_h\mu_v A_1)}, \\ \frac{A_2A_3}{\sigma_h\beta_v}, \frac{(\Lambda_h\beta_h\sigma_v - \mu_h\mu_v A_1)\mu_v}{\sigma_v\beta_h\mu_v A_1}, \frac{\Lambda_h\beta_h\sigma_v - \mu_h\mu_v A_1}{\beta_h\mu_v A_1} \end{pmatrix}$$
(34)

2.4 The Basic Reproduction Number

The basic reproduction number, commonly represented as R_0, is a fundamental concept in

mathematical epidemiology that provides insight into the potential spread of an infectious disease. This threshold parameter is crucial for deter- mining whether an infection will establish itself within a population or fade away.

The computation of R_0 can be performed using the next-generation matrix approach, a method initially introduced by [14] and subsequently refined by[15]. The basic reproduction number serves as a key factor in assessing the stability of the disease-free equilibrium (DFE). Specifically, when $R_0 < 1$ the DFE is locally asymptotically stable, implying that the disease will not persist in the population. Conversely, if $R_0 > 1$, the DFE becomes unstable, allowing for the potential spread of the infection (see Hethcote, 2000).

Mathematically, R_0 is defined as the spectral radius (i.e., the dominant eigen- value) of the matrix product FV^{-1}, expressed as:

$$R_0 = \rho(FV^{-1})$$

(35)

where ρ denotes the spectral radius.

$$f_i(x) = \begin{pmatrix} \beta_h S_h I_v \\ 0 \\ \beta_v S_v I_h \\ 0 \end{pmatrix}$$

(36)

$$V_i(x) = \begin{pmatrix} A_1 E_h \\ -\sigma_h E_h + A_2 \mathrm{I}_h \\ A_4 E_v \\ -\sigma_v E_v + \mu_v I_v \end{pmatrix}$$

(37)

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_h S_h \\ 0 & 0 & 0 & 0 \\ 0 & \beta_v S_v & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{38}$$

$$V = \begin{pmatrix} A_1 & 0 & 0 & 0 \\ -\sigma_h & A_2 & 0 & 0 \\ 0 & 0 & A_4 & 0 \\ 0 & 0 & -\sigma_v & \mu_v \end{pmatrix} \tag{39}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{A_1} & 0 & 0 & 0 \\ \frac{\sigma_h}{A_1 A_2} & \frac{1}{A_2} & 0 & 0 \\ 0 & 0 & \frac{1}{A_4} & 0 \\ 0 & 0 & \frac{\sigma_v}{A_1 \mu_v} & \frac{1}{\mu_v} \end{pmatrix} \tag{40}$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_h S_h \sigma_v}{A_1 \mu_v} & \frac{\beta_h S_h}{\mu_v} \\ 0 & 0 & 0 & 0 \\ \frac{\beta_v S_v \sigma_h}{A_1 A_4} & \frac{\beta_v S_v}{A_2} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{41}$$

$$FV^{-1} - \lambda I = \begin{pmatrix} -\lambda & 0 & \frac{\beta_h S_h \sigma_v}{A_1 \mu_v} & \frac{\beta_h S_h}{\mu_v} \\ 0 & -\lambda & 0 & 0 \\ \frac{\beta_v S_v \sigma_h}{A_1 A_4} & \frac{\beta_v S_v}{A_2} & -\lambda & 0 \\ 0 & 0 & 0 & -\lambda \end{pmatrix} \tag{42}$$

Hence

$$R_0 = \frac{\sqrt{A_1 A_2 A_4 \mu_v \beta_h \beta_v S_h S_v \sigma_h \sigma_v}}{A_1 A_2 A_4 \mu_v} \tag{43}$$

At DFE,

$$R_0 = \sqrt{\frac{\beta_h \beta_v \Lambda_h \Lambda_v \sigma_h \sigma_v}{A_1 A_2 A_4 \mu_h \mu_v^2}} \tag{44}$$

2.5 Stability Analysis

2.5.1 Local Stability of Disease-Free Equilibrium Point

Theorem 2

The disease-free equilibrium point is said to be locally asymptotically stable if all the eigenvalues of the Jacobian matrix at the DFE are negative ($R_0 < 1$) otherwise it is unstable.

Proof: The Jacobian matrix of the system of equation is:

$$J(E^*) = \begin{pmatrix} -\beta_h I_v - \mu_h & 0 & 0 & 0 & 0 & 0 & -\beta_h S_h \\ -\beta_h I_v & -A_1 & 0 & 0 & 0 & 0 & \beta_h S_h \\ 0 & 0 & -A_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_3 & 0 & 0 & 0 \\ 0 & 0 & -\beta_v S_v & 0 & -\beta_v I_h - \mu_v & 0 & 0 \\ 0 & 0 & \beta_v S_v & 0 & -\beta_v I_h & -A_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_v & -\mu_v \end{pmatrix} \tag{45}$$

while at disease-free equilibrium (DFE), all infected compartments (such as I_h *and* I_v) are zero, which will simplify the Jacobian matrix as:

$$J(E^0) = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & -\beta_h S_h \\ 0 & -A_1 & 0 & 0 & 0 & 0 & \beta_h S_h \\ 0 & 0 & -A_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_3 & 0 & 0 & 0 \\ 0 & 0 & -\beta_v S_v & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & \beta_v S_v & 0 & 0 & -A_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_v & -\mu_v \end{pmatrix} \tag{46}$$

Reducing the matrix (46) to upper triangular matrix and the characteristic equation gives

$$J(E^0)-\lambda I=\begin{vmatrix} -\mu_h-\lambda & 0 & 0 & 0 & 0 & 0 & -\beta_h S_h \\ 0 & -A_1-\lambda & 0 & 0 & 0 & 0 & \beta_h S_h \\ 0 & 0 & -A_2-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_3-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_v-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -A_4-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_v-\lambda \end{vmatrix}=0 \qquad (47)$$

The determinant of equation (47) gives:

$$(-\mu_h-\lambda_1)(-A_1-\lambda_2)(-A_2-\lambda_3)(-A_3-\lambda_4)(-\mu_v-\lambda_5)(-A_4-\lambda_6)(-\mu_v-\lambda_7)=0 \qquad (48)$$

Which gives:

$$\lambda_1=-\mu_h \text{ or } \lambda_2=-A_1 \text{ or } \lambda_3=-A_2 \text{ or } \lambda_4=-A_3 \text{ or } \lambda_5=-\mu_v \text{ or } \lambda_6=-A_4 \text{ or } \lambda_7=-\mu_v \qquad (49)$$

Therefore,

$$\lambda_1=-\mu_h,\ \lambda_2=-A_1,\ \lambda_3=-A_2,\ \lambda_4=-A_3,\ \lambda_5=-\mu_v,\ \lambda_6=-A_4,\ \lambda_7=-\mu_v \qquad (50)$$

$$\lambda_1,\lambda_2,\lambda_3,\lambda_4,\lambda_5,\lambda_6,\lambda_7<0$$

Since all the eigenvalues of $J(E^0)$ of the disease-free equilibrium are negative, it shows that the DFE is locally asymptotically stable.

3.0 Optimal Control Formulation

We are examining a system composed of seven nonlinear optimal control models. In this framework, **U₁(t)** represents efforts to increase public awareness and educate the population, aiming to reduce the number of susceptible individuals. **U₂(t)** corresponds to regular screening of at-risk populations and providing prompt treatment to infected individuals. Lastly, **U₃(t)** involves the use of insecticide-treated nets to help control the spread of the disease.

$$\left.\begin{aligned} \frac{dS_h}{dt} &= \Lambda_h+\gamma_h T_h-(1-u_1)\,\beta_h S_h I_v-\mu_h S_h \\ \frac{dE_h}{dt} &= (1-u_1)\beta_h S_h I_v-(\sigma_h+\mu_h)E_h \\ \frac{dI_h}{dt} &= \sigma_h E_h-(\rho_h+\delta_h+\mu_h+u_2)I_h \\ \frac{dT_h}{dt} &= (\rho_h+u_2)I_h-(\gamma_h+\mu_h)T_h \\ \frac{dS_v}{dt} &= \Lambda_v-(1-u_1)\beta_v S_v I_h-\mu_v S_v \\ \frac{dE_v}{dt} &= (1-u_1)\beta_v S_v I_h-(\sigma_v+\mu_v)E_v \\ \frac{dI_v}{dt} &= \sigma_v E_v-(\mu_v+u_3)I_v \end{aligned}\right\} \qquad (51)$$

we define the objective function as

$$J=\min_{(u_1,u_2,u_3)}\int_{t_0}^{t_1}\left(\frac{1}{2}\omega_1 u_1^2+\frac{1}{2}\omega_2 u_2^2+\frac{1}{2}\omega_3 u_3^2+c_1S_h+c_2I_h+c_3E_v+c_4I_v\right) \qquad (52)$$

Subject to the state equations above, with appropriate state initial conditions. $t_0$is the initial time and t_1 is the terminal time. The constant terms $w_1, w_2, w_3, c_1, c_2, c_3, c_4$ represent the weight constants. The terms

$\omega_1 u_1^2$, $\omega_2 u_2^2, \omega_3 u_3^2$ are the costs associated with public awareness and educating individuals, Regular screening of the population at risk with prompt treatment of infected individuals and impact of vector control using insecticide-treated traps respectively. The control set is defined as:

$$U = \{(u_1, u_2, u_3) | 0 \leq u_1 \leq u_{1max}, 0 \leq u_2 \leq u_{2max}, 0 \leq u_3 \leq u_{3max}\} \quad (53)$$

Pontryagin's Principle will be used to determine the necessary and sufficient conditions for our optimal control problem to hold. The principle transforms equations (51) into a pointwise minimization problem of the Hamiltonian (*H*) with respect to the control variables $u_1, u_2 and\ u_3$.

$$H = \frac{1}{2}\omega_1 u_1^2 + \frac{1}{2}\omega_2 u_2^2 + \frac{1}{2}\omega_3 u_3^2 + c_1 S_h + c_2 I_{h1} + c_3 E_v + c_4 I_v + \lambda_1(\Lambda_h + \gamma_h T_h - (1-u_1)\beta_h S_h I_v - \mu_h S_h) + \lambda_2((1-u_1)\beta_h S_h I_v - (\sigma_h + \mu_h)E_h) + \lambda_3(\sigma_h E_h - (\delta_h + \mu_h)I_h - (\tau_h + u_2)I_h) + \lambda_4((\rho_h + u_2)I_h - \gamma_h + \mu_h)T_h) + \lambda_5(\Lambda_v - (1-u_1)\beta_v S_v I_h - \mu_v S_v) + \lambda_6((1-u_1)\beta_v S_v I_h - (\sigma_v + \mu_v)E_v) + \lambda_7(\sigma_v E_v - (u_3 + \mu_v)I_v) \quad (54)$$

Where $\lambda_i, i = 1,2,3, \ldots,7$ are the adjoint variables.

Theorem 4

Given optimal controls $u_1(t), u_2(t), u_3(t)$ and solutions $S_h^*, E_h^*, I_h^*, T_h^*, S_v^*, E_v^*, I_v^*$ of system that optimize $J(u_i)$ over u, then there exist adjoint variables $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7$ satisfying:

$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial S_h}$, $\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial E_h}$, $\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial I_h}$, $\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial T_h}$, $\frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial S_v}$, $\frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial E_v}$, $\frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial I_v}$

$$\frac{d\lambda_1}{dt} = \lambda_1\big((1-u_1)\beta_h I_v + \mu_h\big) - \lambda_2(1-u_1)\beta_h I_v - c_1 \quad (55)$$

$$\frac{d\lambda_2}{dt} = \lambda_2(\alpha_h + \mu_h) - \lambda_3\alpha_h \quad (56)$$

$$\frac{d\lambda_3}{dt} = C_2 + \lambda_3(\sigma_h E_h - 2(\tau_h + u_2)I_h - \delta_h - \mu_h) + \lambda_4(\tau_h + u_2) - \lambda_5(1-u_1)\beta_v S_v + \lambda_6(1-u_1)\beta_v S_v \quad (57)$$

$$\frac{d\lambda_4}{dt} = -\lambda_4(\gamma_h + \mu_h) + \lambda_1\gamma_h \quad (58)$$

$$\frac{d\lambda_5}{dt} = C_3 - \lambda_5\big((1-u_1)\beta_v I_h + \mu_h\big) + \lambda_6(1-u_1)\beta_v I_h \quad (59)$$

$$\frac{d\lambda_6}{dt} = -\mu_v\lambda_6 + \sigma_v\lambda_7 \quad (60)$$

$$\frac{d\lambda_7}{dt} = c_4 - \lambda_1(1-u_1)\beta_h S_h + \lambda_2(1-u_1)\beta_h S_h - (u_3 + \mu_v)\lambda_7 \quad (61)$$

And with transversality conditions:

$$\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = \lambda_5 = \lambda_6 = \lambda_7 = 0 \quad (62)$$

And the optimal control u^*that satisfies the optimality conditions:

$$\frac{\partial H}{\partial u_i} = 0, i = 1,2,3$$

$$u_1^* = max\left\{0, min\left(\frac{\beta_h S_h I_v(\lambda_2 - \lambda_1)}{\omega_1}\right), 1\right\} \quad (63)$$

$$u_2^* = max\left\{0, min\left(\frac{\beta_v S_v I_h(\lambda_7 - \lambda_6) + I_{h1}(\lambda_3 - \lambda_4)}{\omega_2}\right), 1\right\} \quad (64)$$

$$u_3^* = max\left\{0, min\left(\frac{\lambda_1 I_v}{\omega_3}\right), 1\right\} \quad (65)$$

4.0 Numerical Simulation

In this section, we'll look at some real-world examples to better understand how different control strategies—both used individually and combined—can influence the outcomes of our model. We'll begin with starting values such as $S_h(0) = 900$, $E_h(0) = 50$, $I_h(0) = 30$, $R_h(0) = 15$, $S_v(0) = 1500$, $E_v(0) = 500$, and $I_v(0) = 300$. To make the results easier to grasp, we've included visual graphs below that illustrate how the model behaves under different scenarios**.**

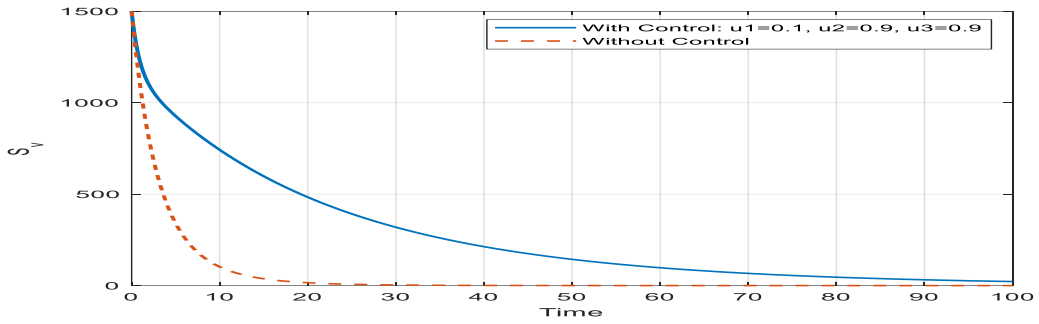

Figure 2: The dynamics of susceptible vector with and without control

This plot of susceptible vectors vividly illustrates HAT dynamics over time—with and without control measures—and highlights how timely interventions can dramatically curb transmission compared to an uncontrolled outbreak.

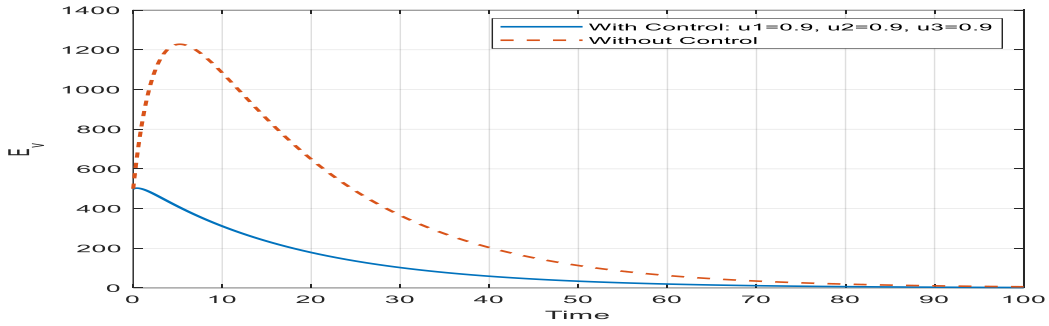

Figure 3: Dynamics of exposed vector combine with control

This graph effectively illustrates the impact of control when a community is exposed to HAT outbreak, this show that with proper control efforts, outbreaks can be managed more efficiently compared to when it is without control.

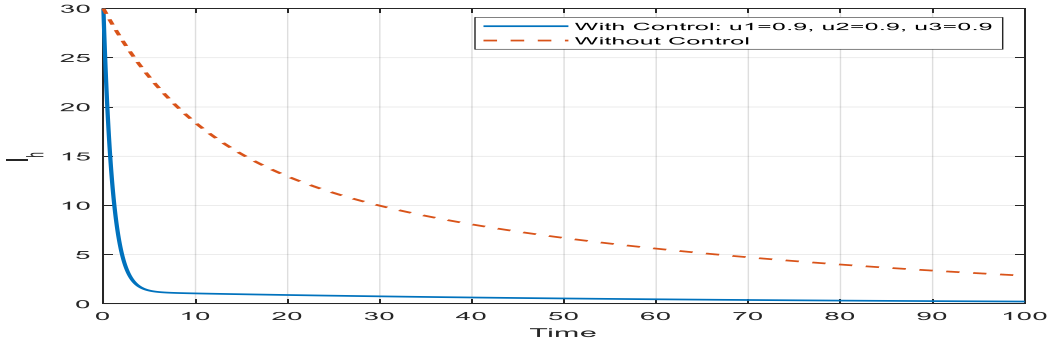

Figure 4: The dynamics of infected human with and without control

This graph of the infected human population highlights how control measures alter HAT's trajectory—demonstrating that, with well-designed interventions, the disease can be contained far more rapidly and effectively than during an uncontrolled outbreak.

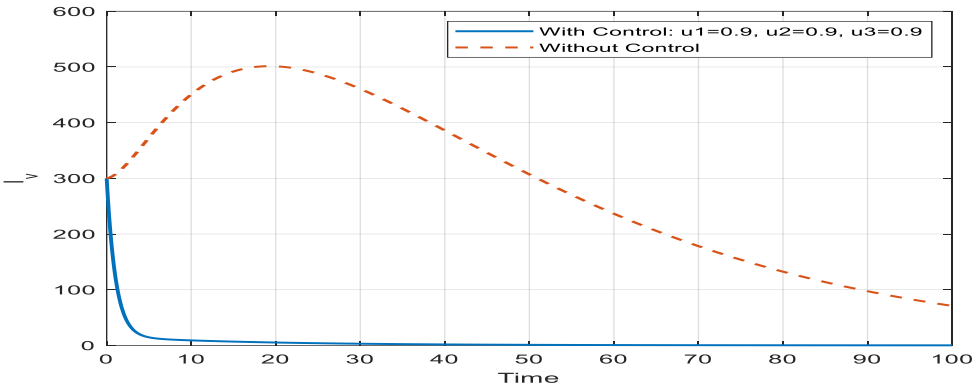

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Figure 5: The dynamics of infected tsetse fly with and without control

This plot of infected vector population shows the impact of control on the spread of HAT in the population, show that with proper control strategies, HAT spread can be more easily and efficiently controlled compared to when it the disease spreads without control.

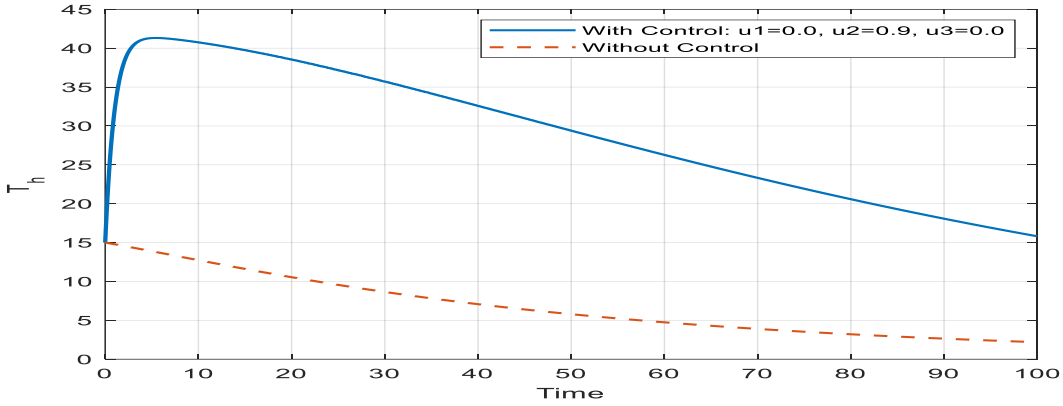

Figure 6: The dynamics of treatment combine with control

This graph depicting the effect of treatment with control measures in human population, this can effectively slow down the spread and accelerate the elimination of HAT, compared to scenarios without intervention.

CONCLUSION

The findings from our model highlight the predictable dynamics of Human African Trypanosomiasis (HAT) transmission between humans and tsetse flies. The boundedness and positivity of the solution were discussed to show that the model is mathematically consistent and biologically meaningful. The equilibria points (HAT-free and HAT-endemic equilibrium) were calculated. Stability analysis identifies HAT-free and endemic equilibria, with the basic reproduction number R_0 derived to assess transmission potential. The optimal control frame- work was introduced to the model. From our analysis, we discovered that the intervention strategies: public awareness, regular screening and prompt treatment, and effective vector control are essential to reducing the disease burden. However, the rapid loss of immunity after recovery points to a critical need for long-term solutions, particularly vaccine development. The plots and the analysis results establish that strategically timed, integrated approaches such as public awareness, regular screening and prompt treatment, prioritizing vector reduction, and relapse- aware

healthcare prove most effective. This study provides good insights for policymakers, advocating for adaptive, resource efficient strategies and research interventions to protect vulnerable populations and move toward the eventual elimination of HAT.

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