

Global Stability and Optimal Control Analysis of Human African Trypanosomiasis Transmission dynamics with Two stages Infection

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ABSTRACT A Mathematical model describing the transmission dynamics and optimal control of Human African Trypanosomiasis (HAT) is developed and analyzed. It is a neglected tropical disease transmitted by tsetse flies, continues to be a public health problem in sub-Saharan Africa. This study proposes an eight-compartmental mathematical model incorporating human and vector dynamics, with a relapse. The boundedness and positivity of the solution were discussed to show that the model is both mathematically consistent and biologically meaningful. The equilibria points (disease-free and endemic equilibrium) were calculated. The global stability analysis identifies disease-free and endemic equilibria, with the basic reproduction number R_0 of both derived to assess transmission were discussed. An optimal control framework is applied to optimize intervention strategies, three time-dependent controls were integrated: public awareness, regular screening and prompt treatment to mitigate further spread, and vector control using insecticide-treated traps. Pontryagin's Maximum Principle was used for the analysis. The results demonstrate 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 to accelerate HAT elimination.

KEYWORDS: Compartmental model; Optimal control; Pontryagin's maximum principle; Stability; Transmission Dynamics.

I. INTRODUCTION

Human African trypanosomiasis (HAT), commonly known as sleeping sickness, remains a serious public health concern across sub-Saharan Africa. Caused by *Trypanosoma brucei* parasites and transmitted by tsetse flies, the disease significantly impacts affected communities. HAT progresses in two distinct stages: an early hemolymphatic phase characterized by mild, nonspecific symptoms (e.g., fever, headaches, swollen lymph nodes), and a second, more severe meningoencephalitic phase, marked by neurological disturbances such as psychiatric disorders and sleep cycle disruption. If untreated, this stage is often fatal. Notably, individuals may remain infectious for years, serving as asymptomatic reservoirs and sustaining transmission. Understanding HAT's progression is vital for timely diagnosis and treatment, which can prevent neurological damage and reduce the risk of further spread. Studies such as [1] and [2] have provided detailed insights into the clinical features and pathophysiology of both disease stages, emphasizing the need for stage-specific interventions. Mathematical modeling plays a central role in capturing the transmission

dynamics of HAT and evaluating control strategies. Early work by [3] introduced a deterministic model exploring interactions between humans, cattle, and tsetse flies, offering a rigorous derivation of the basic reproduction number, R_0 , and showing that the disease-free equilibrium is stable when $R_0 < 1$. Building on foundational models like the SIR framework introduced by [4], researchers have expanded the modeling landscape to incorporate disease complexities and intervention measures. In addition, [5] developed a fractional-order model incorporating time-dependent control using data from Ghana. Their analysis demonstrated that combining prevention and treatment controls is both effective and cost-efficient. Similarly, [6]; [7] and [8] analyzed seven-compartmental models incorporating optimal control and cost-effectiveness, evaluating the impact of treatment, vector control, and public education in reducing HAT prevalence. Further contributions by [9] presented a comprehensive nine-class model that incorporated disease dynamics in humans, livestock, and vectors. Their simulations showed how host-vector interactions and treatment measures influence transmission. More recently, [10] employed Pontryagin's Maximum Principle to optimize control strategies, highlighting that integrated approaches education, treatment, and vector management offer the best outcomes for disease containment. [7, 8], both developed seven compartmental models: the SEIR model for humans and the SEI model for vectors to study the dynamics of the disease. They also employed optimal control models to evaluate the effectiveness of various intervention strategies in reducing disease prevalence and transmission. In this work, we propose and analyze an eight-compartmental model (SEI_1I_2R) for humans, capturing the two human infectious stages of HAT, with relapse, and an SEI model for vectors. By integrating empirical data, we aim to investigate transmission drivers and assess the effectiveness of various control strategies. 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, basic reproduction number R_0 and global stability via disease free and endemic equilibrium. In Section 3 we considered the optimal control model and provides numerical simulations of the model using a set of data. Finally, Section 4 concludes with a discussion of findings and policy implications.

II. MATERIALS AND METHODS

A. Model Formation

A mathematical model that describes the effect of vector-borne diseases on hosts and vectors was developed by dividing the total human population into five compartments: Susceptible human class $S_h(t)$ exposed human class $E_h(t)$ stage one infected human class $I_{h1}(t)$, stage two infected human class $I_{h2}(t)$ and recovered human class $R_h(t)$. 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 parameters used in the schematic diagram of the model in Figure 1.

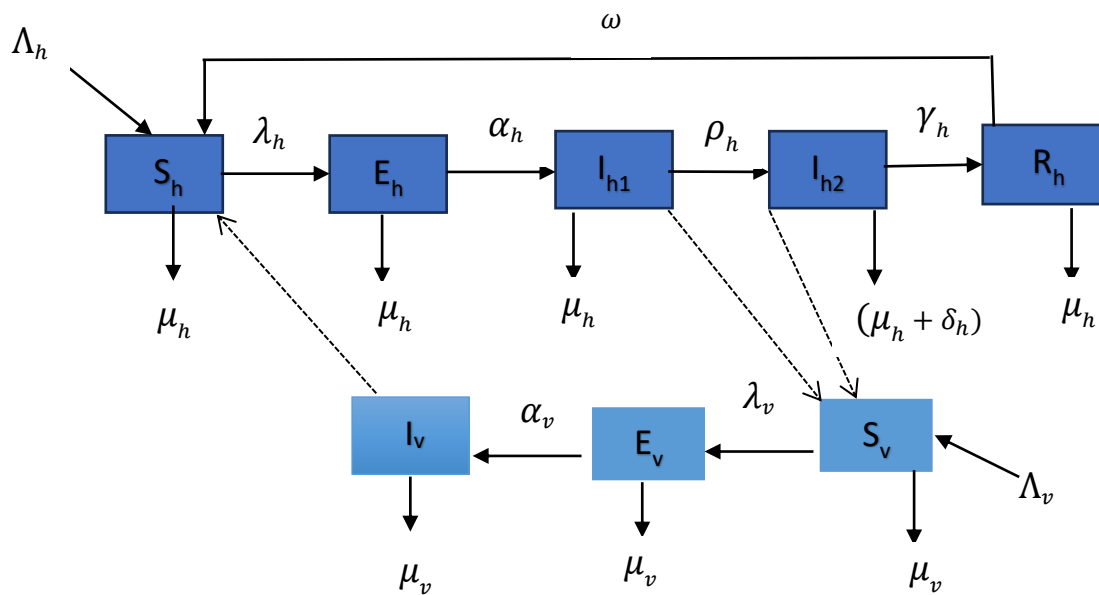


Figure 1: Schematic Diagram of the Proposed Model

$$\left. \begin{aligned} \frac{dS_h}{dt} &= \Lambda_h + \omega R_h - \beta_h S_h I_v - \mu_h S_h \\ \frac{dE_h}{dt} &= \beta_h S_h I_v - (\alpha_h + \mu_h) E_h \\ \frac{dI_{h1}}{dt} &= \alpha_h E_h - (\rho_h + \mu_h) I_{h1} \\ \frac{dI_{h2}}{dt} &= \rho_h I_{h1} - (\gamma_h + \delta_h + \mu_h) I_{h2} \\ \frac{dR_h}{dt} &= \gamma_h I_{h2} - (\omega + \mu_h) R_h \\ \frac{dS_v}{dt} &= \Lambda_v - \beta_v S_v (I_{h1} + I_{h2}) - \mu_v S_v \\ \frac{dE_v}{dt} &= \beta_v S_v (I_{h1} + I_{h2}) - (\alpha_v + \mu_v) E_v \\ \frac{dI_v}{dt} &= \alpha_v E_v - \mu_v I_v \end{aligned} \right\} \quad (1)$$

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

$I_{h1}(t)$	Number of infectious human stage 1 at time t
$I_{h2}(t)$	Number of infectious human stage 2 at time t
$R_h(t)$	Number of recovered humans at time t
$S_v(t)$	Number of susceptible tsetse flies at time t
$E(t)$	Number of exposed tsetse flies at time t
$I_v(t)$	Number of infectious tsetse flies at time t
$U_1(t)$	The effect of public awareness and educating the population to reduce the susceptible individuals
$U_2(t)$	Regular screening of the population at risk and prompt treatment of infected individuals
$U_3(t)$	Use of insecticides treated nets
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	The rate of at which susceptible humans get exposed to the disease.
β_v	The rate at which susceptible tsetse fly gets exposed to the disease
α_h	The rate at which human Progresses from exposed to stage 1 of infection
α_v	The rate at which the exposed tsetse fly gets infected
ρ_h	The rate at which human Progresses from stage 1 of infection to stage 2
γ_h	The recovery rate of humans
ω	The rate at which the recovered human loss immunity and return to susceptible

Taking the Human sub model

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

The vector sub model

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

Invariant Region of the Model

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

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_{h2}}{dt} + \frac{dI_{h1}}{dt} + \frac{dR_h}{dt} = \Lambda_h - (\delta_h + \mu_h)N_h \quad (4)$$

and

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_{v2}}{dt} + \frac{dI_{v1}}{dt} + \frac{dR_v}{dt} = \Lambda_v - \mu_v N_v \quad (5)$$

Theorem 1

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

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

Proof

Let

$$\Omega = (S_h, E_h, I_{h1}, I_{h2}, R_h, S_v, E_v, I_v) \in \mathbb{R}_+^8 \quad (6)$$

with non-negative initial conditions using the differential inequality theorem, [11] on equation (4). In the absence of the disease induced death in the human population, equations (4) become:

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

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,

$$\Lambda_v - \mu_v N_v \geq K e^{-\mu_v t}$$

Where K is the constant

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

$$\Omega = \{(S_h, E_h, I_{h1}, I_{h2}, R_h) \in \mathcal{R}_+^5 : S_h, E_h, I_{h1}, I_{h2}, R_h \geq 0, N_h \leq \Lambda_h - \mu_h N_h\} \quad (10)$$

$$\Omega = \{(S_v, E_v, I_v) \in \mathcal{R}_+^3 : S_v, E_v, I_v \geq 0, N_v \leq \Lambda_v - \mu_v N_v\} \quad (11)$$

Thus, the feasible set of the model is given by

$$\Omega = \{(S_h, E_h, I_{h1}, I_{h2}, R_h, S_v, E_v, I_v) \in \mathcal{R}_+^8 : S_h, E_h, I_{h1}, I_{h2}, R_h, S_v, E_v, I_v \geq 0; N_h \leq \frac{\Lambda_h}{\mu_h}, N_v \leq \frac{\Lambda_v}{\mu_v}\} \quad (12)$$

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

B. 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

$$\{(S_h(0), E_h(0), I_{h1}(0), I_{h2}(0), R_h(0), S_v(0), E_v(0), I_v(0)) \geq 0\} \in \Omega \quad (13)$$

Then, the solution set

$\{S_h(t) + E_h(t) + I_{h1}(t) + I_{h2}(t) + R_h(t) + S_v(t) + E_v(0) + I_v(t)\}$ of the system (1) is positive for all $t > 0$.

Proof

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

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

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

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

using integrating factor, we have:

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

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

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

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

$$I_{h1}(t) \geq I_{h1}(0)e^{-(\rho_h + \mu_h)t} > 0 \quad (25)$$

$$I_{h2}(t) \geq I_{h2}(0)e^{-(\gamma_h + \delta_h + \mu_h)t} > 0 \quad (26)$$

$$R_h(t) \geq R_h(0)e^{-(\omega + \mu_h)t} > 0 \quad (27)$$

$$S_v(t) \geq \frac{\Lambda_v}{\mu_v} + (S_v(0) - \frac{\Lambda_v}{\mu_v})e^{\mu_v t} > 0 \quad (28)$$

$$E_v(t) \geq E_v(0)e^{-(\alpha_v + \mu_v)t} > 0 \quad (29)$$

$$I_v(t) \geq I_v(0)e^{-\mu_v t} > 0 \quad (30)$$

Therefore, all the solutions of the system of equations (1) are positive for all $t > 0$. This ensure the model predictions are physically meaningful and realistic.

C. Equilibrium Points of the Model

At equilibrium, we have

$$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_{h1}}{dt} = \frac{dI_{h2}}{dt} = \frac{dR_h}{dt} = \frac{dS_v}{dt} = \frac{dE_v}{dt} = \frac{dI_v}{dt} = 0 \quad (31)$$

HAT-Free Equilibrium State

Let

$$(S_h, E_h, I_{h1}, I_{h2}, R_h, S_v, E_v, I_v) = (S_h^0, E_h^0, I_{h2}^0, I_{h1}^0, R_h^0, S_v^0, E_v^0, I_v^0) \quad (32)$$

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 $E_h = I_{h1} = I_{h2} = E_v = I_v = 0$ and then solve for the remaining compartments, using Maple 2020.

Therefore, the system (1) becomes

$$(S_h^0, E_h^0, I_{h2}^0, I_{h1}^0, R_h^0, S_v^0, E_v^0, I_v^0) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right) \quad (33)$$

HAT-Endemic Equilibrium State

Let

$$(S_h, E_h, I_{h1}, I_{h2}, R_h, S_v, E_v, I_v) = (S_h^*, E_h^*, I_{h2}^*, I_{h1}^*, R_h^*, S_v^*, E_v^*, I_v^*) \quad (34)$$

and

$$A_1 = (\alpha_h + \mu_h), A_2 = (\rho_h + \mu_h), A_3 = (\gamma_h + \delta_h + \mu_h), A_4 = (\omega + \mu_h), A_1 = (\alpha_v + \mu_v)$$

Then,

$$\left. \begin{aligned} S_h^* &= A_1 A_2 A_3 A_5 \mu_v \left(\frac{A_1 A_2 A_3 A_4 \mu_v + A_3 A_4 \beta_v \Lambda_h \alpha_h + A_4 \rho_h \beta_v \Lambda_h \alpha_h - \mu_v \omega \rho_h \gamma_h \alpha_h}{(A_1 A_2 A_3 A_4 A_5 \mu_v \mu_h + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v - \beta_h \omega \rho_h \Lambda_v \alpha_h \alpha_v \gamma_h) \beta_v \alpha_h (A_3 + \rho_h)} \right), \\ E_h^* &= A_2 A_3 A_5 \mu_v \left(\frac{A_1 A_2 A_3 A_4 \mu_h \mu_v^2 + A_3 \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v + \rho_h \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v}{(A_1 A_2 A_3 A_4 A_5 \mu_v \mu_h + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v - \beta_h \omega \rho_h \Lambda_v \alpha_h \alpha_v \gamma_h) \beta_v \alpha_h (A_1 + \rho_h)} \right), \\ I_{h1}^* &= A_3 A_4 \left(\frac{A_1 A_2 A_3 A_4 \mu_h \mu_v^2 + A_3 \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v + \rho_h \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v}{(A_1 A_2 A_3 A_4 A_5 \mu_v \mu_h + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v - \beta_h \omega \rho_h \Lambda_v \alpha_h \alpha_v \gamma_h) \beta_v \alpha_h (A_3 + \rho_h)} \right), \\ I_{h2}^* &= A_2 \rho_h \left(\frac{A_1 A_2 A_3 A_4 \mu_h \mu_v^2 + A_3 \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v + \rho_h \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v}{(A_1 A_2 A_3 A_4 A_5 \mu_v \mu_h + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v - \beta_h \omega \rho_h \Lambda_v \alpha_h \alpha_v \gamma_h) \beta_v \alpha_h (A_1 + \rho_h)} \right), \\ R_h^* &= A_2 \rho_h \left(\frac{A_1 A_2 A_3 A_4 \mu_h \mu_v^2 + A_3 \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v + \rho_h \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v}{(A_1 A_2 A_3 A_4 A_5 \mu_v \mu_h + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v - \beta_h \omega \rho_h \Lambda_v \alpha_h \alpha_v \gamma_h) \beta_v \alpha_h (A_1 + \rho_h)} \right), \\ S_v^* &= \left(\frac{A_1 A_2 A_3 A_4 A_5 \mu_h \mu_v + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v - \omega \rho_h \gamma_h \alpha_h \beta_h \Lambda_v \alpha_v}{\beta_h \alpha_v (A_1 A_2 A_3 A_4 \mu_v + A_3 A_4 \beta_v \Lambda_h \alpha_h + A_4 \rho_h \beta_v \Lambda_h \alpha_h - \mu_v \omega \rho_h \Lambda_v \alpha_h \gamma_h)} \right), \\ E_v^* &= A_4 \left(\frac{A_1 A_2 A_3 A_5 \mu_v \mu_v^2 + A_3 \beta_v \alpha_h \Lambda_h \beta_h \Lambda_v \alpha_v + \rho_h \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v}{\beta_h \alpha_v (A_1 A_2 A_3 A_4 \mu_v + A_3 A_4 \beta_v \Lambda_h \alpha_h + A_4 \rho_h \beta_v \Lambda_h \alpha_h - \mu_v \omega \rho_h \Lambda_v \alpha_h \gamma_h) A_5} \right), \\ I_v^* &= A_4 \left(\frac{A_1 A_2 A_3 A_4 A_5 \mu_h \mu_v^2 + A_1 A_2 A_3 A_4 \beta_h \Lambda_v \alpha_v + \rho_h \beta_h \beta_v \Lambda_h \Lambda_v \alpha_h \alpha_v}{\beta_h \mu_v (A_1 A_2 A_3 A_4 \mu_v + A_3 A_4 \beta_v \Lambda_h \alpha_h + A_4 \rho_h \beta_v \Lambda_h \alpha_h - \mu_v \omega \rho_h \Lambda_v \alpha_h \gamma_h) A_5} \right) \end{aligned} \right\} \quad (35)$$

D. 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 determining 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 [12] and subsequently refined by [13]. 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. Mathematically, R_0 is defined as the spectral radius (i.e., the dominant eigenvalue) of the matrix product FV^{-1} , expressed as:

$$R_0 = \rho(FV^{-1}) \quad (36)$$

where ρ denotes the spectral radius, F is the appearance of new infections and V is the progression in and out of the compartment.

$$f_i(x) = \begin{pmatrix} \beta_h S_h I_v \\ 0 \\ \beta_v S_v (I_{h1} + I_{h2}) \\ 0 \\ 0 \end{pmatrix} \quad (37)$$

$$v_i(x) = \begin{pmatrix} A_1 E_h \\ -\alpha_h E_h + A_2 I_{h1} \\ -\rho_h I_{h1} + A_3 I_{h2} \\ A_5 E_v \\ -\alpha_v E_v + \mu_v I_v \end{pmatrix} \quad (38)$$

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & S_h \beta_h \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & S_v \beta_v & S_v \beta_v & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (39)$$

$$V = \begin{pmatrix} A_1 & 0 & 0 & 0 & 0 \\ -\alpha_h & A_2 & 0 & 0 & 0 \\ 0 & -\rho_h & A_3 & 0 & 0 \\ 0 & 0 & 0 & A_5 & 0 \\ 0 & 0 & 0 & -\alpha_v & \mu_v \end{pmatrix} \quad (40)$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{S_h \beta_h \alpha_v}{A_3 \mu_v} & \frac{S_h \beta_h}{\mu_v} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{S_v \beta_v \alpha_h}{A_1 A_2} + \frac{S_v \beta_v \alpha_h \rho_h}{A_1 A_2 A_3} & \frac{S_v \beta_v}{A_2} + \frac{S_v \beta_v \rho_h}{A_2 A_3} & \frac{S_v \beta_v}{A_3} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (41)$$

$$\text{Det} |FV^{-1} - \lambda I| = R_0 = \sqrt{\frac{A_1 A_2 A_3 A_5 \mu_v S_v \beta_v \alpha_h (A_3 + \rho_h) S_h \beta_h \alpha_v}{A_1 A_2 A_3 A_5 \mu_v}} \quad (42)$$

E. Global Stability

To show that system (1) is globally asymptotically stable, we use the Lyapunov function theory for both the HAT-free equilibrium and the HAT-present equilibrium.

Global Stability of the HAT-Free Equilibrium

First, we present the global stability of the HAT-free equilibrium E^0 when $R_0 \leq 1$.

Theorem 2: The HAT-free equilibrium E^0 of the model (1) is globally asymptotically stable (GAS) in Ω if $R_0 \leq 1$ and unstable otherwise.

Proof

Using Castillo-Chavex theorem [14]. Let $X(t)$ denotes the uninfected population $X = (S_h, R_h, S_v)$, and $Y(t)$ represents the infected, $Y = (E_h, I_{h1}, I_{h2}, E_v, I_v)$. Thus, system (1) can be written as

$$\frac{dX}{dt} = F(X, Y), \frac{dY}{dt} = G(X, Y); G(X, 0) = 0. \quad (43 \& 44)$$

Where F and G are corresponding right hand side of system (1). According to [14], to establish the global asymptotic stability of the disease free equilibrium, the following two conditions (D1) and (D2) must be satisfied for $R_0 < 1$.

- (D1) $X^0 = (1,0,1)^T$ is globally asymptotically stable for $\frac{dX}{dt} = F(X, 0)$.
- (D2) $G \geq 0$ where $G(X, Y) = AY - G(X, Y)$ and $A = D_Y G(X^0, 0)$ is Metzler matrix $\forall (X, Y) \in \Omega$

First Condition, that is globally asymptotically stability of X^0 , we have

$$\frac{dX}{dt} = F(X, 0) = \begin{pmatrix} \Lambda_h + \omega R_h - \mu_h S_h \\ -(\omega + \mu_h) R_h \\ \Lambda_v - \mu_v S_v \end{pmatrix} \quad (45)$$

by solving the above system, we have the behavior of each compartment as follows

$$X^0 \begin{pmatrix} S_h \\ R_h \\ S_v \end{pmatrix} = \begin{pmatrix} 1 + S_h(0)e^{-\mu_h t} \\ R_h(0)e^{-(\omega + \mu_h)t} \\ 1 + S_v(0)e^{-\mu_v t} \end{pmatrix} \quad (46)$$

Now since $\lim_{t \rightarrow \infty} X(t) = X^0$ satisfying the first condition. For the second condition (D2)

$$A = \begin{pmatrix} -A_1 & 0 & 0 & 0 & 0 \\ \alpha_h & -A_2 & 0 & 0 & 0 \\ 0 & \rho_h & -A_3 & 0 & 0 \\ 0 & S_v \beta_v & S_v \beta_v & -A_5 & 0 \\ 0 & 0 & 0 & \alpha_v & -\mu_v \end{pmatrix} \quad (47)$$

Then we compute

$$G(X, Y) = AY - G(X, Y) = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (48)$$

It's obvious that $G(X, Y) = 0$, thus $G(X, Y) \geq 0$ in (D2) is satisfied. Hence, E^0 is globally asymptotically stable, provided that $R_0 \leq 1$. HAT die out. This completes the proof.

F. Global Stability of the HAT-present Equilibrium

The result of the global stability of E^* in this section is as follows.

Theorem 3: The HAT-present equilibrium point E^* is globally asymptotically stable (GAS) if $R_0 > 1$.

Proof.

Consider the Lyapunov function

$$L = c_1 S_h + c_2 E_h + c_3 I_{h1} + c_4 I_{h2} + c_5 R_h + c_6 S_v + c_7 E_v + c_8 I_v \quad (49)$$

Where c_i for $i = 1, 2, 3, \dots, 8$ are constants to be chosen during the proof are defined. The derivative of L along the solution of (1) is given by

$$\frac{dL}{dt} = c_1 \frac{dS_h}{dt} + c_2 \frac{dE_h}{dt} + c_3 \frac{dI_{h1}}{dt} + c_4 \frac{dI_{h2}}{dt} + c_5 \frac{dR_h}{dt} + c_6 \frac{dS_v}{dt} + c_7 \frac{dE_v}{dt} + c_8 \frac{dI_v}{dt} \quad (50)$$

gives:

$$\begin{aligned} \frac{dL}{dt} = & c_1 (\Lambda_h + \omega R_h - \beta_h S_h I_v - \mu_h S_h) + c_2 (\beta_h S_h I_v - (\alpha_h + \mu_h) E_h) + c_3 (\alpha_h E_h - (\rho_h + \\ & \mu_h) I_{h1}) + c_4 (\rho_h I_{h1} - (\gamma_h + \delta_h + \mu_h) I_{h2}) + c_5 (\gamma_h I_{h2} - (\omega + \mu_h) R_h) + c_6 (\Lambda_v - \beta_v S_v (I_{h1} + \\ & I_{h2}) - \mu_v S_v) + c_7 (\beta_v S_v (I_{h1} + I_{h2}) - (\alpha_h + \mu_v) E_v) + c_8 (\alpha_v E_v - \mu_v I_v) \end{aligned} \quad (51)$$

$$\begin{aligned} \frac{dL}{dt} = & c_1(\Lambda_h - \mu_h S_h) + (c_2 - c_1)\beta_h S_h I_v + (c_3 - c_2)\alpha_h E_h - c_2\mu_h E_h + (c_4 - c_4)\rho_h I_{h1} - c_3\mu_h I_{h1} + \\ & (c_5 - c_4)I_{h1}I_{h1} - c_4\gamma_h I_{h2} - c_4\delta_h I_{h2} - \mu_h I_{h2} + (c_5 - c_1)\omega R_h + c_6(\Lambda_v - \mu_v S_v) + (c_7 + c_6)\beta_v S_v (I_{h1} + \\ & I_{h2}) - c_5\mu_h R_h + (c_8 - c_7)(\alpha_v E_v - c_7\mu_v E_v - c_8\mu_v I_v \end{aligned} \quad (52)$$

Taking $c_1, c_2, c_3, c_4, c_5, c_6, c_7$ and c_8 such that $c_1 = c_2 = c_3 = c_4 = c_5 = c_6 = c_7 = c_8, \Lambda_h - \mu_h S_h = 0$

And $\Lambda_v - \mu_v S_v = 0$ gives

$$\frac{dL}{dt} = c_2\mu_h E_h - c_3\mu_h I_{h1} - c_4\delta_h I_{h2} - c_4\mu_h I_{h2} - c_5\mu_h R_h - c_7\mu_v E_v - c_8\mu_v I_v \quad (53)$$

It follows that L is positive definite, and dL/dt is negative definite. Therefore, the function L is a Lyapunov function for model system (1), and by Lyapunov asymptotic stability theorem [15], the endemic equilibrium E^* is globally asymptotically stable in the invariant Ω . HAT persisted.

G. Optimal Control Formulation

We are examining a system composed of eight nonlinear optimal control models. In this framework, $U_1(t)$ represents efforts to increase public awareness and educate the population, aiming to reduce the number of susceptible individuals. $U_2(t)$ corresponds to regular screening of at-risk populations and providing prompt treatment to infected individuals. Lastly, $U_3(t)$ involves the use of insecticide-treated nets to help control the spread of the disease.

$$\frac{dS_h}{dt} = \Lambda_h + \omega R_h - (1 - u_1)\beta_h S_h I_v - \mu_h S_h \quad (54)$$

$$\frac{dE_h}{dt} = (1 - u_1)\beta_h S_h I_v - (\alpha_h + \mu_h)E_h \quad (55)$$

$$\frac{dI_{h1}}{dt} = \alpha_h E_h - (\rho_h + \mu_h)I_{h1} - u_2 I_{h1} \quad (56)$$

$$\frac{dI_{h2}}{dt} = (\rho_h + u_2)I_{h1} - (\gamma_h + \delta_h + \mu_h)I_{h2} \quad (57)$$

$$\frac{dR_h}{dt} = \gamma_h I_{h2} - (\omega + \mu_h)R_h \quad (58)$$

$$\frac{dS_v}{dt} = \Lambda_v - (1 - u_1)\beta_v S_v (I_{h1} + I_{h2}) - \mu_v S_v \quad (59)$$

$$\frac{dE_v}{dt} = (1 - u_1)\beta_v S_v I_h - (\alpha_h + \mu_v)E_v \quad (60)$$

$$\frac{dI_v}{dt} = \alpha_v E_v - \mu_v I_v \quad (61)$$

we define the objective function as

$$J = \min_{(u_1, u_2, u_3)} \int_{t_0}^{t_1} \left(\frac{1}{2}\omega_1 u_1^2 + \frac{1}{2}\omega_2 u_2^2 + \frac{1}{2}\omega_3 u_3^2 + c_1 S_h + c_2 I_{h1} + c_3 E_v + c_4 I_v \right) \quad (62)$$

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:

$$U = \{(u_1, u_2, u_3) | 0 \leq u_1 \leq u_{1max}, 0 \leq u_2 \leq u_{2max}, 0 \leq u_3 \leq u_{3max}\} \quad (63)$$

Pontryagin's Maximum Principle will be used to determine the necessary and sufficient conditions for our optimal control problem to hold. The principle transforms equations (54) to (61) into a pointwise minimization problem of the Hamiltonian (H) with respect to the control variables u_1, u_2 and u_3 .

$$H = \frac{1}{2}\omega_1 u_1^2 + \frac{1}{2}\omega_1 u_1^2 + \frac{1}{2}\omega_1 u_1^2 + c_1 S_h + c_2 I_{h1} + c_3 E_v + c_4 I_v + \lambda_1 (\Lambda_h + \omega R_h - (1 - u_1) \beta_h S_h I_v - \mu_h S_h) + \lambda_2 ((1 - u_1) \beta_h S_h I_v - (\alpha_h + \mu_h) E_h) + \lambda_3 (\alpha_h E_h - (\rho_h + \mu_h) I_{h1} - u_2 I_{h1}) + \lambda_4 ((\rho_h + u_2) I_{h1} - (\gamma_h + \delta_h + \mu_h) I_{h2}) + \lambda_5 (\gamma_h I_{h2} - (\omega + \mu_h) R_h) + \lambda_6 (\Lambda_v - (1 - u_1) \beta_v S_v (I_{h1} + I_{h2}) - \mu_v S_v) + \lambda_7 ((1 - u_1) \beta_v S_v I_h - (\alpha_h + \mu_v) E_v) + \lambda_8 (\alpha_v E_v - \mu_v I_v) \quad (64)$$

Where $\lambda_i, i = 1, 2, 3, \dots, 8$ are the adjoint variables.

Given optimal controls $u_1(t), u_2(t), u_3(t)$ and solutions $S_h^*, E_h^*, I_{h2}^*, I_{h1}^*, R_h^*, S_v^*, E_v^*, I_v^*$ of system that optimize $J(u_i)$ over u , then there exist adjoint variables $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8$ satisfying:

$$\begin{aligned} \frac{d\lambda_1}{dt} &= -\frac{\partial H}{\partial S_h}, & \frac{d\lambda_2}{dt} &= -\frac{\partial H}{\partial E_h}, & \frac{d\lambda_3}{dt} &= -\frac{\partial H}{\partial I_{h1}}, & \frac{d\lambda_4}{dt} &= -\frac{\partial H}{\partial I_{h2}}, & \frac{d\lambda_5}{dt} &= -\frac{\partial H}{\partial R_h}, & \frac{d\lambda_6}{dt} &= -\frac{\partial H}{\partial S_v}, \\ \frac{d\lambda_7}{dt} &= -\frac{\partial H}{\partial E_v}, & \frac{d\lambda_8}{dt} &= -\frac{\partial H}{\partial I_v} \end{aligned}$$

$$-\frac{d\lambda_1}{dt} = c_1 + \lambda_1(-(1 - u_1)\beta_h I_v - \mu_h) + \lambda_2(1 - u_1)\beta_h I_v \quad (65)$$

$$-\frac{d\lambda_2}{dt} = \lambda_2(-\alpha_h - \mu_h) + \lambda_3 \alpha_h \quad (66)$$

$$-\frac{d\lambda_3}{dt} = \lambda_3(-\rho_h - u_2) + \lambda_4(\rho_h + u_2) \quad (67)$$

$$-\frac{d\lambda_4}{dt} = \lambda_4(-\gamma_h - \delta_h - \mu_h) + \lambda_4 \gamma_h \quad (68)$$

$$-\frac{d\lambda_5}{dt} = \lambda_1 \omega + \lambda_4(-\omega - \mu_h) \quad (69)$$

$$-\frac{d\lambda_6}{dt} = c_3 + \lambda_6(-(1 - u_2)\beta_v I_h - \mu_v) + \lambda_7(1 - u_2)\beta_v I_h \quad (70)$$

$$-\frac{d\lambda_7}{dt} = -\mu_v \lambda_7 + \alpha_v \lambda_4 \quad (71)$$

$$-\frac{d\lambda_8}{dt} = c_4 - \lambda_1(1 - u_1)\beta_h S_h + \lambda_2 \rho_h - u_2(1 - u_1)\beta_h S_h + (-u_3 - \mu_v) \quad (72)$$

and with transversality conditions:

$$\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = \lambda_5 = \lambda_6 = \lambda_7 = \lambda_8 = 0 \quad (73)$$

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

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

$$u_1^* = \max \left\{ 0, \min \left(\frac{\beta_h S_h I_v (\lambda_2 - \lambda_1)}{\omega_1}, 1 \right) \right\} \quad (74)$$

$$u_2^* = \max \left\{ 0, \min \left(\frac{\beta_v S_v I_h (\lambda_7 - \lambda_6) + I_{h1} (\lambda_3 - \lambda_4)}{\omega_2}, 1 \right) \right\} \quad (75)$$

$$u_3^* = \max \left\{ 0, \min \left(\frac{\lambda_1 I_v}{\omega_3}, 1 \right) \right\} \quad (76)$$

III. RESULTS AND DISCUSSIONS

In this section, we'll explore some numerical examples of model (1) to see how different control strategies—both individually and combined—impact the outcomes. We'll start with initial values like $S_h(0) = 900$, $E_h(0) = 50$, $I_{h1}(0) = 30$, $I_{h2}(0) = 20$, $R_h(0) = 15$, $S_v(0) = 1500$, $E_v(0) = 500$, and $I_v(0) = 300$. To help visualize the results, we've included graphical representations of the model in the Figures below:

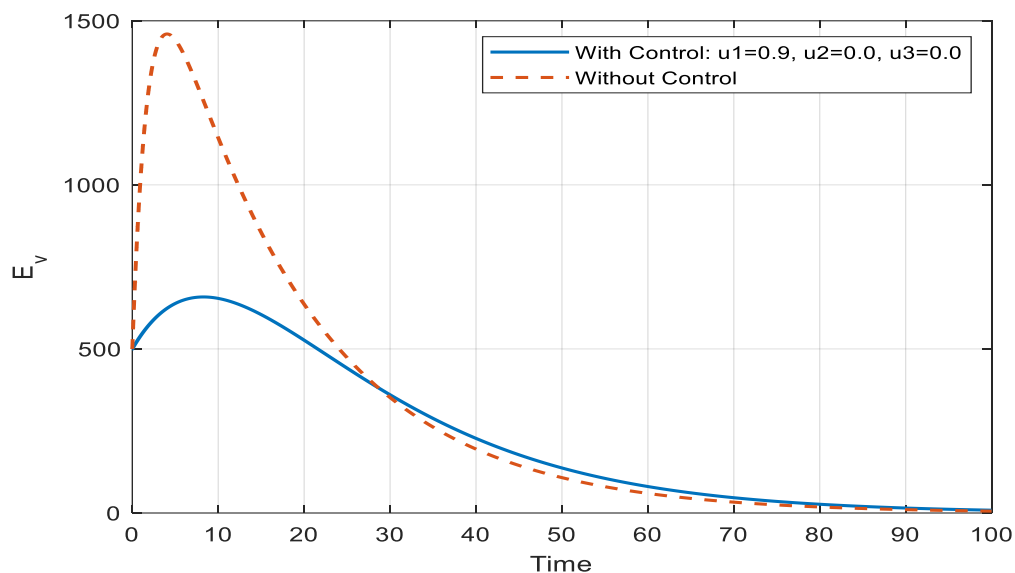


Figure 1: Dynamics of Exposed Vector E_v with Control Combination

The image above shows that initially, the number of exposed vectors rises sharply, but over time, it gradually decreases back toward zero. On the other hand, when control measures are put into place, the number of exposed vectors drops more steadily and consistently toward zero. This really underscores how important these control efforts are in managing and reducing vector populations effectively.

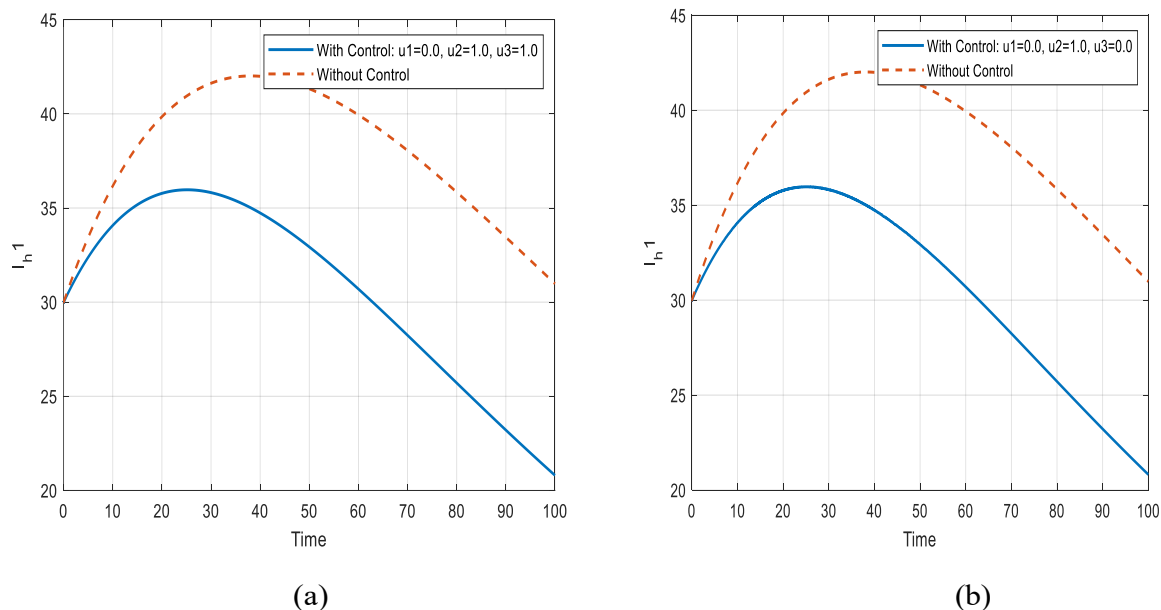


Figure 2: Plots of the Dynamics of the First Stage of Human Infected Class

Figure 2 illustrates how the early stage of the infected population evolves under two different scenarios: with and without control measures. It clearly shows that implementing control measures can effectively slow down the spread of the infection, preventing or delaying the progression to the second stage.

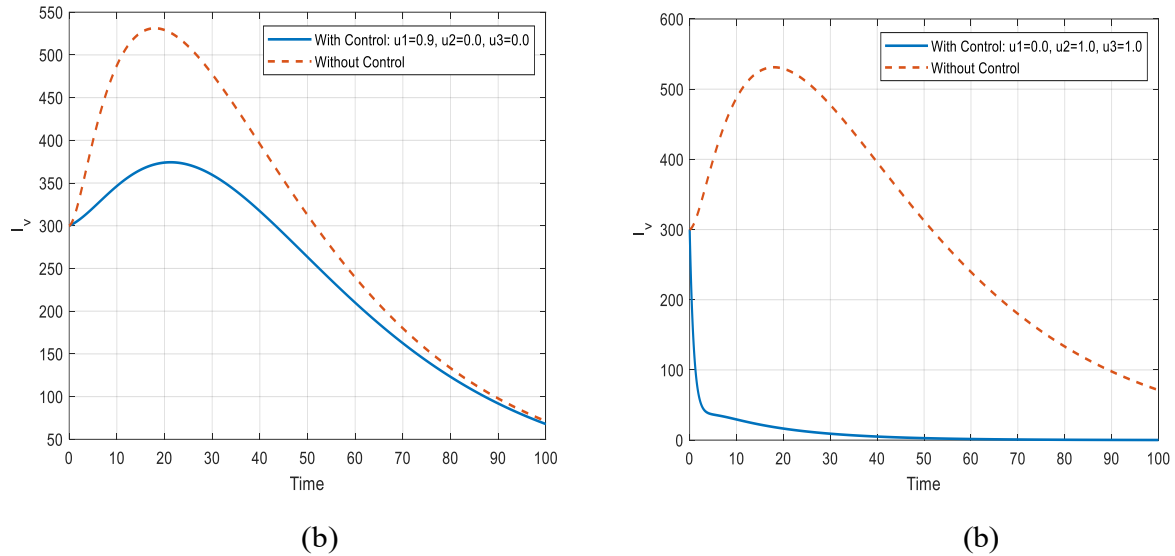


Figure 3: Dynamics of Infected Vector

Figure 3 illustrates that, without any control measures, the number of infected vectors slowly rises over the first nineteen days. After this point, the infections begin to decline. When control measures are put in place, the number of infections still decreases gradually in scenario (a), but in scenario (b), the decline is much more effective, approaching zero more quickly. This clearly underscores how crucial these control measures are in effectively managing and reducing infected vectors.

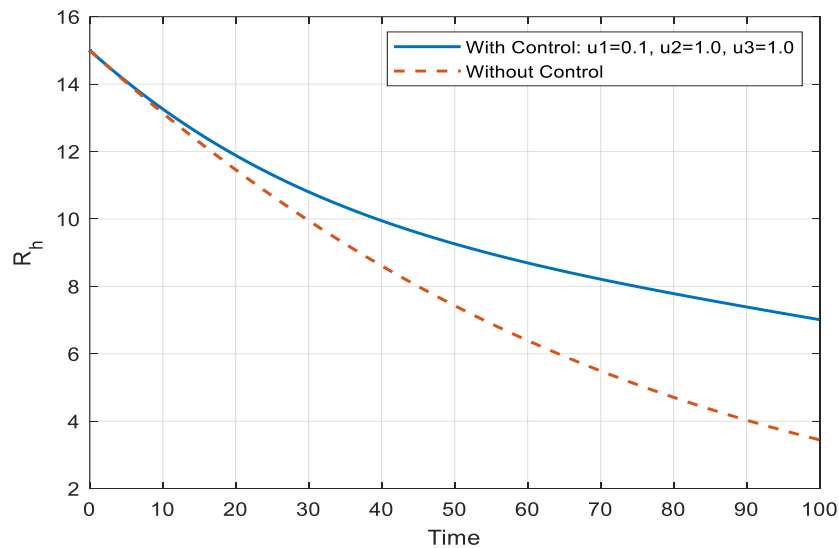


Figure 4: Dynamics of Recovered Humans

Figure 4 shows some interesting trends regarding recovered humans, with and without control.

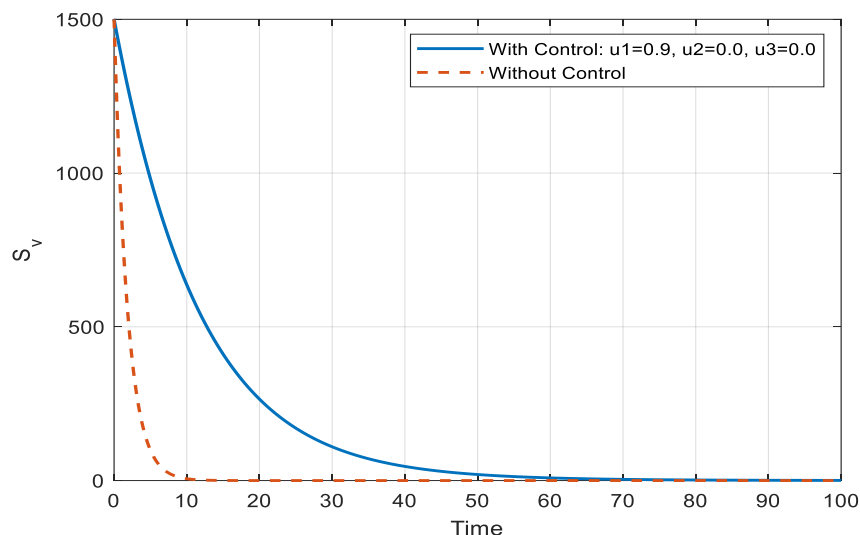


Figure 5: Dynamics of Susceptible Vector

Figure 5 illustrates a sudden drop in the number of susceptible vectors during the first ten days when no control measures are implemented. Once control measures are introduced, a portion of these susceptible vectors are prevented from becoming infected over the next sixty days. As a result, the number of infected vectors begins to decrease.

IV. 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 framework 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 results demonstrate 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. In the future, it would be valuable to explore how the changing patterns of HAT affect people's lives, especially in terms of their social and economic well-being.

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