

Mathematical Analysis and Numerical Simulation of the Integrated HIV/TB Co-Infection Model

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ABSTRACT

In this study, we examined an integrated model of HIV and TB co-infection. Specifically, we proposed a system of nonlinear differential equations to describe the co-dynamics of both diseases and conducted a comprehensive stability analysis of its critical points alongside numerical simulations. The stability analysis of the equilibrium points revealed that the disease-free equilibrium is globally asymptotically stable when the basic reproduction number, R_0 , is less than one ($R_0 < 1$). This result indicates that, under these conditions, both diseases can be eradicated from the population over time. A local sensitivity analysis was performed to identify parameters with the greatest influence on disease dynamics. The analysis demonstrated that the parameter ω_1 is a key driver for tuberculosis (TB) transmission, as evidenced by its high sensitivity index in the TB-specific reproduction number, R_T . From a biological perspective, ω_1 represents a control measure related to reducing aerosol transmission, highlighting the critical importance of respiratory hygiene, such as covering one's mouth when coughing or sneezing. A similar analysis for HIV indicated that the parameter ω_2 exerts the most significant influence on HIV transmission, as shown by its impact on the HIV-specific reproduction number, R_H . Biologically, ω_2 encapsulates behavioral controls, emphasizing the necessity of promoting safe sexual practices and preventing contact with infected bodily fluids to curb HIV spread. Numerical simulations supported our analytical findings. They demonstrated that in a co-infection setting, the disease with the higher basic reproduction number tends to dominate the epidemiological landscape. Furthermore, the simulations illustrated that targeted increases in the control measures ω_1 and ω_2 lead to a substantial reduction in the number of co-infection cases within the community, underscoring the effectiveness of combined, disease-specific intervention strategies.

Keywords: HIV, basic reproduction number, stability analysis, numerical simulation, tuberculosis

INTRODUCTION

Human Immunodeficiency Virus (HIV) is a lentivirus that causes acquired immunodeficiency syndrome (AIDS), a condition marked by the progressive failure of the immune system [1]. HIV spreads through blood, semen, vaginal fluids, and breast milk, either as free particles or inside infected immune cells [2]. The virus primarily attacks CD4⁺ T lymphocytes (helper T cells), macrophages, and dendritic cells [3]. Through direct killing and triggering apoptosis (programmed cell death) of infected and nearby uninfected cells, HIV steadily depletes CD4⁺ T cells [3]. When their count falls below a critical threshold, the immune system collapses, leaving the body vulnerable to opportunistic infections. According to the U.S. Centers for Disease Control and Prevention (CDC), about 20 such infections are classified as AIDS-defining illnesses [4]. An HIV-positive person developing any of these is diagnosed with AIDS, regardless of their CD4⁺ count. Among these, tuberculosis

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(TB) is one of the most common and deadly co-infections. Worldwide, about one-third of the population is infected with *Mycobacterium tuberculosis*, with roughly 8.7 million active TB cases and over 1.4 million deaths annually [4]. HIV and TB form a dangerous syndemic: HIV-induced immunosuppression increases the risk of both new TB infection and reactivation of latent TB, while active TB accelerates HIV replication and disease progression. This vicious cycle is worsened by drug-resistant TB strains, weak health systems, delayed diagnosis, and slow vaccine development, leading to a global resurgence of both diseases.

Numerous models have explored HIV and TB separately, considering both immunological [5] and epidemiological perspectives [6]. Several co-infection models have also been proposed, with and without treatment interventions [7]. Integrative frameworks linking within-host immunological dynamics to population-level epidemiological spread have been introduced [8]. However, modeling HIV/TB co-infection remains challenging due to the complex interactions between the pathogens, host immunity, treatment strategies, and healthcare access. Existing models often simplify these dynamics, frequently omitting factors such as stratified healthcare availability, drug resistance, and comprehensive qualitative analysis, including equilibrium stability and sensitivity assessments. Despite progress in HIV and TB modeling, there is a clear need for a more robust analytical framework that incorporates key biological and epidemiological features, performs stability and sensitivity analysis, and accounts for realistic treatment and healthcare access scenarios. This study aims to (1) develop a mathematical model describing HIV and TB co-infection dynamics, (2) perform numerical simulations and equilibrium stability analysis to explore the behavior of the system, and (3) conduct sensitivity analysis to identify key parameters that most influence disease transmission and progression. By addressing the limitations of existing models, the findings from this research are expected to enhance understanding of HIV/TB syndemic dynamics and provide actionable insights for public health policy and intervention strategies. Improved modeling of co-infection dynamics can support targeted resource allocation, optimize treatment strategies, and ultimately reduce mortality associated with HIV/TB co-infection.

LITERATURE REVIEW

Infectious diseases are disorders caused by pathogenic microorganisms, including bacteria, viruses, fungi, and parasites. These diseases constitute a major global public health burden, affecting millions of individuals annually and encompassing conditions such as influenza, HIV/AIDS, tuberculosis (TB), and pneumonia [9]. Among these, HIV/AIDS remains one of the most significant global health challenges, having infected approximately 70 million individuals and contributing substantially to global morbidity and mortality [10]. Sub-Saharan Africa bears a disproportionate share of this burden, accounting for more than two-thirds of all people living with HIV [11]. Human Immunodeficiency Virus (HIV) is a retrovirus that integrates its genetic material into host immune cells, primarily targeting CD4⁺ T lymphocytes. Progressive depletion of these cells, in the absence of effective treatment, leads to severe immunosuppression, culminating in acquired immunodeficiency syndrome (AIDS), the advanced stage of infection [12]. HIV transmission occurs through exposure to infected body fluids, including unprotected sexual contact, sharing of contaminated needles, and vertical transmission from mother to child during pregnancy, delivery, or breastfeeding [13]. According to the U.S. Centers for Disease Control and Prevention (CDC), untreated HIV infection typically progresses through three clinical stages: acute infection, clinical latency, and AIDS.

A critical challenge in HIV management is the high prevalence of co-infection with other pathogens, particularly *Mycobacterium tuberculosis*, the causative agent of tuberculosis. Co-infection refers to the simultaneous presence of multiple infectious agents within a single host. Individuals living with HIV are significantly more susceptible to developing active tuberculosis, a risk that persists even among those receiving antiretroviral therapy (ART) [14]. The interaction between HIV and TB is synergistic and deleterious: HIV-induced immunosuppression facilitates the reactivation or progression of latent TB infection, while active TB accelerates HIV disease progression. This bidirectional interaction exacerbates disease severity and contributes substantially to mortality, particularly in resource-limited settings where both infections are endemic. A comprehensive understanding of HIV–TB co-infection dynamics is therefore essential for the development of effective integrated public health interventions. Mathematical modelling provides a powerful framework for understanding the transmission dynamics and control of infectious diseases. Such models enable the simulation of disease spread, evaluation of intervention strategies, and support evidence-based public health decision-

making. A substantial body of literature has focused on modelling HIV and TB as separate entities. For instance, [10] developed a sex-structured model to investigate heterosexual transmission dynamics of HIV, highlighting the importance of targeted intervention strategies. Similarly, other studies have examined optimal control strategies for HIV, demonstrating that combined approaches involving antiretroviral therapy (ART) and pre-exposure prophylaxis (PrEP) are essential for effective disease control [14]. However, the simultaneous modelling of HIV and TB co-infection presents considerably greater complexity than single-disease frameworks. Co-infection models must adequately capture nonlinear interactions between pathogens, host immune response dynamics, and the effects of concurrent treatment strategies. Existing models often rely on simplifying assumptions such as homogeneous mixing, absence of drug resistance, and uniform access to healthcare services, which may limit their realism and applicability. Furthermore, many studies do not provide comprehensive analyses of key dynamical properties, including stability (disease persistence or elimination) and sensitivity (identification of influential parameters). The present study seeks to address these limitations by developing and analysing a more realistic mathematical model for HIV–TB co-infection. The objective is to identify key epidemiological drivers of disease dynamics and provide insights to inform more effective prevention and treatment strategies for these interacting infections.

Dynamical Systems and Linearization

Consider a system of autonomous ordinary differential equations (ODEs) defined by a vector field $f: U \subset \mathbb{R}^n \rightarrow \mathbb{R}^n$:

$$\dot{x} = f(x), \quad x(0) = x_0 \in U, \quad (1)$$

where $x \in U$ represents the state variables, and $U = \mathbb{R}_+^n = \{x \in \mathbb{R}^n: x_i \geq 0, i = 1, \dots, n\}$ denotes the biologically feasible region corresponding to non-negative population compartments [15]. A point $x^* \in U$ is called an equilibrium (or critical point) of the system if it satisfies $f(x^*) = 0$. The local behavior of the system near an equilibrium x^* is governed by its linearization. Let $y = x - x^*$ denote a perturbation around the equilibrium. The linearized system is then given by

$$\dot{y} = Ay, \quad (2)$$

where $A = Df(x^*) \in \mathbb{R}^{n \times n}$ is the Jacobian matrix of f evaluated at x^* . The eigenvalues of A determine the local stability of the equilibrium x^* .

Stability and Lyapunov Theory

A set $V \subset U$ is called an invariant set for the flow of equation (1) if $x(0) \in V$ implies $x(t) \in V$ for all $t \in \mathbb{R}$. If this condition holds only for all $t \geq 0$, then V is called a positive invariant set [15]. The equilibrium x^* of the linear system (2) are said to be (stable in the sense of Lyapunov) if all eigenvalues λ_i of A satisfy $\text{Re}(\lambda_i) \leq 0$, with those having zero real parts being simple. The equilibrium is asymptotically stable if $\text{Re}(\lambda_i) < 0$ for all i .

For nonlinear systems, Lyapunov's direct method provides a more general criterion for stability. A continuously differentiable function $V: U \rightarrow \mathbb{R}$ is called a Lyapunov function for an equilibrium $x^* \in U$ if it is positive definite, that is, $V(x) > 0$ for $x \neq x^*$ and $V(x^*) = 0$, and its derivative along the trajectories of the system,

$$\dot{V}(x) = \nabla V(x) \cdot f(x), \quad (3)$$

is negative semi-definite (for stability) or negative definite (for asymptotic stability). If, in addition, $V(x) \rightarrow \infty$ as $\|x\| \rightarrow \infty$, that is, V is radially unbounded, then the equilibrium x^* is globally asymptotically stable [16].

The Basic Reproduction Number

The basic reproduction number, denoted R_0 , is a threshold quantity in epidemiology. It represents the average number of secondary infections caused by a single infected individual introduced to a wholly susceptible

population [17]. For compartmental models with multiple infected states, R_0 is efficiently computed using the next-generation matrix (NGM) approach [18]. The system of ODEs is decomposed into new infection terms (F) and all other transition terms (V). Linearizing these matrices at the disease-free equilibrium (DFE), x_0 , yields the matrices F and V . The NGM is $K = FV^{-1}$, and the spectral radius of this matrix gives R_0 :

$$R_0 = \rho(FV^{-1}). \quad (4)$$

For the epidemiological model described, the disease-free equilibrium x_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$ [19].

Sensitivity Analysis

To identify the parameters that most critically influence R_0 and thus disease dynamics, a local sensitivity analysis is performed. The normalized forward sensitivity index of R_0 with respect to a parameter p is defined as [18],

$$Y_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}. \quad (5)$$

This index quantifies the proportional change in R_0 resulting from a proportional change in p . Parameters with large absolute sensitivity indices warrant particular attention in data estimation and intervention design.

Numerical Methods

As analytical solutions for nonlinear co-infection models are often intractable, numerical integration is essential. The fourth-order Runge-Kutta (RK4) method provides a robust and accurate scheme for solving the system (1). Given a step size $h > 0$, the approximation proceeds from $y_i \approx y(t_i)$ to $y_{i+1} \approx y(t_i + h)$ via:

$$\begin{aligned} k_1 &= f(t_i, y_i), \\ k_2 &= f\left(t_i + \frac{h}{2}, y_i + \frac{h}{2}k_1\right), \\ k_3 &= f\left(t_i + \frac{h}{2}, y_i + \frac{h}{2}k_2\right), \\ k_4 &= f(t_i + h, y_i + hk_3), \\ y_{i+1} &= y_i + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4). \end{aligned}$$

All simulations in this study were performed using MATLAB, which leverages its optimized ODE solvers that implement adaptive variants of the Runge-Kutta method [20]. Specifically, we employed the ode45 solver, which is based on an explicit Runge-Kutta (4,5) formula, the Dormand-Prince pair. The solver uses adaptive step-size control with a relative tolerance of 10^{-6} and an absolute tolerance of 10^{-8} . The integration interval was set to $[0, 5]$ months for all simulations, with initial conditions specified in Section 4.

METHODS

Model formulation

A deterministic compartmental model is formulated to capture the co-circulation dynamics of Human Immunodeficiency Virus (HIV) and Tuberculosis (TB) within a homogeneously mixing population of total size $N(t)$. The population is stratified into eight mutually exclusive compartments:

- $S(t)$: Susceptible individuals
- $I_t(t)$: Individuals with active TB infection

- $I_{ht}(t)$: Individuals co-infected with HIV and active TB
- $T_t(t)$: TB-infected individuals undergoing treatment
- $T_{ht}(t)$: HIV/TB co-infected individuals undergoing treatment
- $I_a(t)$: Individuals with AIDS
- $I_{at}(t)$: Individuals with AIDS and concurrent active TB
- $R_t(t)$: Individuals who have recovered from TB

The model incorporates key epidemiological and clinical realities, notably that HIV infection substantially increases susceptibility to TB, with an estimated 80% of people living with HIV being vulnerable to TB infection or reactivation [21]. HIV-positive individuals may access antiretroviral therapy (ART), which stabilizes their infection and halts progression to AIDS; those untreated progress to the AIDS class. TB-infected individuals can receive effective treatment, after which they recover and re-enter the susceptible pool, as recovery does not confer lasting immunity. Co-infected individuals experience elevated disease-induced mortality, reflecting the synergistic pathogenesis of the two diseases, while a constant natural mortality rate, μ , applies to all compartments. Transmission follows a standard incidence formulation. The effective contact rate for TB, α , is reduced by a control parameter ω_1 ($0 \leq \omega_1 \leq 1$), representing the efficacy of interventions such as respiratory hygiene campaigns and infant BCG vaccination. The complete system of nonlinear differential equations describing the co-infection model is given by:

$$\begin{cases} \frac{dS}{dt} = B - \alpha(1 - \omega_1) \frac{SI_t}{N} - \sigma(1 - \omega_2) \frac{SI_{ht}}{N} + \delta R_t - \mu S, \\ \frac{dI_t}{dt} = \alpha(1 - \omega_1) \frac{SI_t}{N} - b(1 - \omega_3) \frac{SI_t I_{ht}}{N} - (c + \beta_1 + \mu) I_t, \\ \frac{dI_{ht}}{dt} = \sigma(1 - \omega_2) \frac{SI_{ht}}{N} + b(1 - \omega_3) \frac{SI_t I_{ht}}{N} - (1 - a) I_{ht} - (\beta_2 + a + \mu + \gamma_2) I_{ht}, \\ \frac{dT_t}{dt} = c I_t - (\gamma_1 + \beta_3 + \mu) T_t, \\ \frac{dT_{ht}}{dt} = a I_{ht} + \gamma_3 I_{at} - (\beta + \mu + \beta_4) T_{ht}, \\ \frac{dI_{at}}{dt} = \gamma_2 I_{ht} - (\gamma_3 + \mu + \beta_6) I_{at}, \\ \frac{dI_a}{dt} = \beta T_{ht} + (1 - a) I_{ht} - (\mu + \beta_5) I_a, \\ \frac{dR_t}{dt} = \gamma_1 T_t - (\delta + \mu) R_t. \end{cases} \quad (6)$$

where the total population is $N(t) = S(t) + I_t(t) + I_{ht}(t) + T_t(t) + T_{ht}(t) + I_{at}(t) + I_a(t) + R_t(t)$.

Figure 1 illustrates the schematic diagram of the model, showing all transitions between the epidemiological compartments.

Figure 1: Transmission dynamics of the model.

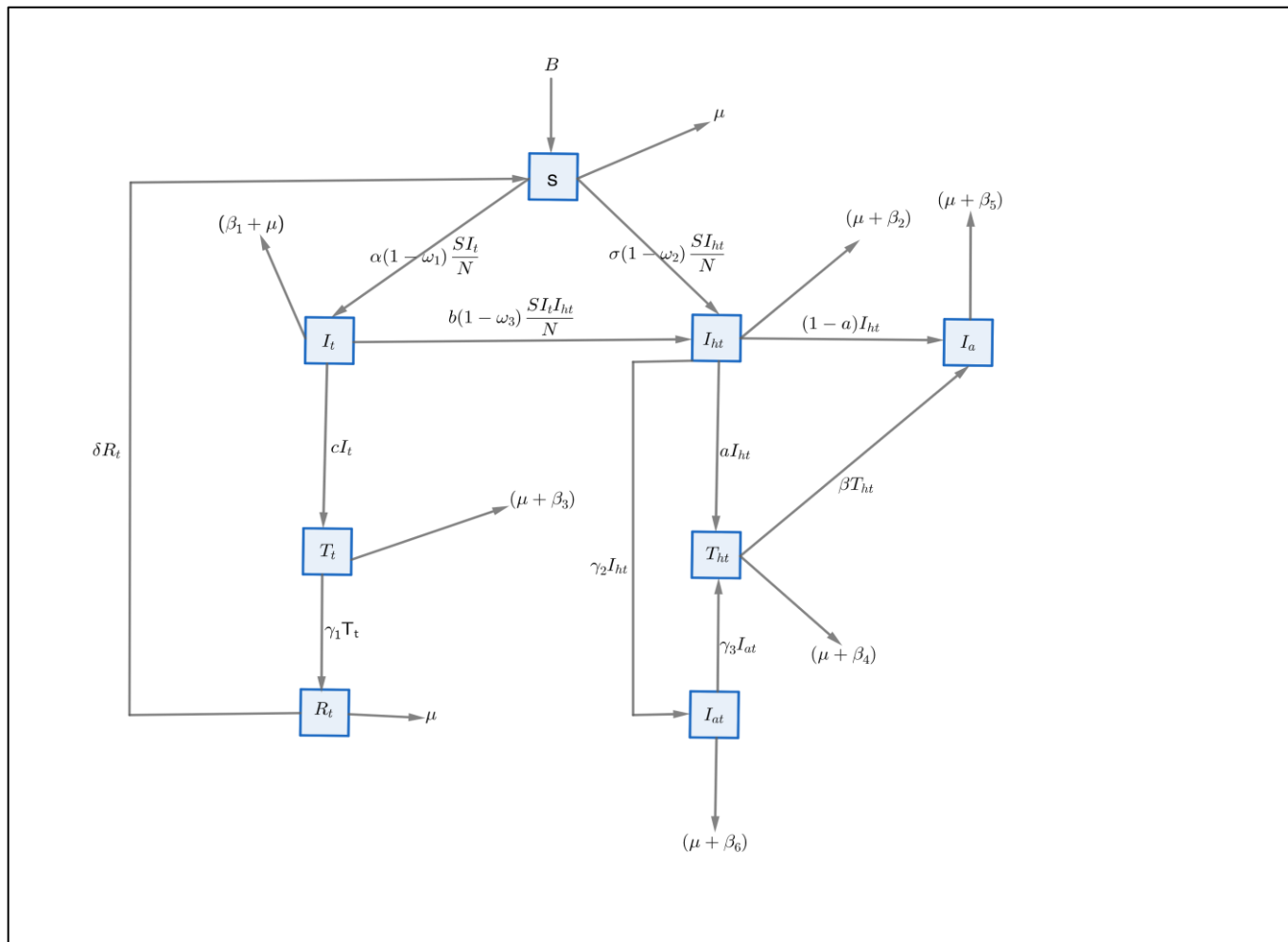


Table 1 represents the description of the model parameters and their corresponding values.

Table 1: Model Parameters and Descriptions

Parameter	Description	Value	Unit	Biological Interpretation	Source
α	Susceptible–TB contact rate	0.1	Month ⁻¹	Rate at which susceptible individuals acquire TB infection through contact with infectious TB cases	[1], [7]
σ	Susceptible–HIV contact rate	0.1	Month ⁻¹	Rate at which susceptible individuals acquire HIV infection through risky behavior	[1], [10]
b	TB-infected to HIV transmission rate	0.07	Month ⁻¹	Additional HIV acquisition rate for individuals already infected with TB	[7], [21]
ω_1	TB prevention efficacy (respiratory hygiene)	0.8	Dimensionless (0–1)	Proportion of TB transmission prevented through educational campaigns, mask-wearing, and cough etiquette	[1], [9]
ω_2	HIV prevention efficacy (safe practices)	0.8	Dimensionless (0–1)	Proportion of HIV transmission prevented through condom use, safe-sex education, and avoiding infected fluids	[10], [14]
ω_3	TB-infected HIV prevention efficacy	0.8	Dimensionless (0–1)	Proportion of HIV transmission prevented among TB-infected individuals through targeted counseling	[7], [21]
a	HIV treatment rate (ART uptake)	0.8	Month ⁻¹	Rate at which HIV-infected individuals initiate antiretroviral therapy	[14], [17]

c	TB treatment rate	0.7	Month ⁻¹	Rate at which TB-infected individuals initiate and complete TB treatment	[1], [7]
δ	Recovery to susceptible rate	0.2	Month ⁻¹	Rate at which recovered TB individuals lose immunity and return to the susceptible pool	[7], [21]
β	HIV treatment failure rate	0.3	Month ⁻¹	Rate at which HIV-infected individuals on ART progress to AIDS due to treatment failure	[14], [17]
μ	Natural mortality rate	0.013	Month ⁻¹	Background mortality rate applicable to all compartments	[1], [7]
β_1	TB disease-induced death rate	0.08	Month ⁻¹	Additional mortality due to active TB infection	[1], [7]
β_2	HIV/TB co-infection death rate	0.1	Month ⁻¹	Additional mortality due to the synergistic interaction of HIV and TB	[7], [21]
β_3	TB treatment death rate	0.03	Month ⁻¹	Mortality during TB treatment, for example due to drug toxicity or late presentation	[1], [7]
β_4	HIV/TB treatment death rate	0.5	Month ⁻¹	Mortality during concurrent HIV/TB treatment	[7], [21]
β_5	AIDS disease death rate	0.8	Month ⁻¹	Mortality due to AIDS-related complications	[14], [17]
β_6	Pre-AIDS TB death rate	0.52	Month ⁻¹	Mortality due to TB in HIV-positive individuals who have not yet progressed to AIDS	[7], [21]
γ_1	TB recovery rate	0.7	Month ⁻¹	Rate of successful TB treatment and recovery	[1], [7]
γ_2	HIV/TB pre-AIDS progression rate	0.8	Month ⁻¹	Rate at which HIV/TB co-infected individuals progress to the pre-AIDS TB class	[7], [21]
γ_3	Pre-AIDS TB treatment uptake rate	0.15	Month ⁻¹	Rate at which pre-AIDS TB co-infected individuals initiate treatment	[7], [21]
B	Recruitment rate	0.2	Month ⁻¹	Rate of new individuals entering the susceptible population through births and immigration	[1], [9]

Model analysis

Positivity of the solutions

The system of equations (6) governs the dynamics of the human population. To guarantee biological realism, all state variables and parameters are assumed to be non-negative. By adopting the methodology [22] and considering the first equation of system (6), we obtain

$$\begin{aligned}\frac{dS}{dt} &= B - \alpha(1 - \omega_1)\frac{SI_t}{N} - \sigma(1 - \omega_2)\frac{SI_{ht}}{N} + \delta R_t - \mu S, \\ &= B + \delta R_t - \left(\alpha(1 - \omega_1)\frac{I_t}{N} + \sigma(1 - \omega_2)\frac{I_{ht}}{N} + \mu\right)S.\end{aligned}\quad (7)$$

Let's suppose that,

$$\frac{dS}{dt} \geq -\left(\alpha(1 - \omega_1)\frac{I_t}{N} + \sigma(1 - \omega_2)\frac{I_{ht}}{N} + \mu\right)S, \quad (8)$$

integrating both sides of this equation, we get

$$\int \frac{dS}{S} \geq -\int \left(\alpha(1 - \omega_1)\frac{I_t}{N} + \sigma(1 - \omega_2)\frac{I_{ht}}{N} + \mu\right) dt. \quad (9)$$

$$\ln S \geq -\left(\alpha(1-\omega_1)\frac{I_t}{N} + \sigma(1-\omega_2)\frac{I_{ht}}{N} + \mu\right) \Rightarrow S(t) \geq e^{-\left(\alpha(1-\omega_1)\frac{I_t}{N} + \sigma(1-\omega_2)\frac{I_{ht}}{N} + \mu\right)t+k}, \quad (10)$$

$$\text{hence } S(t) \geq ke^{-\left(\alpha(1-\omega_1)\frac{I_t}{N} + \sigma(1-\omega_2)\frac{I_{ht}}{N} + \mu\right)t}. \quad (11)$$

By utilizing the initial condition at $t = 0$, where $S(0) \geq k$, yield

$$S(t) \geq S(0)e^{-\left(\alpha(1-\omega_1)\frac{I_t}{N} + \sigma(1-\omega_2)\frac{I_{ht}}{N} + \mu\right)t} \geq 0, \text{ since } \left(\alpha(1-\omega_1)\frac{I_t}{N} + \sigma(1-\omega_2)\frac{I_{ht}}{N} + \mu\right) > 0. \quad (12)$$

The remaining equations in the system of equations (6) undergo the same procedure, with the following outcomes

$$\begin{cases} S(t) \geq S(0)e^{-\left(\alpha(1-\omega_1)\frac{I_t}{N} + \sigma(1-\omega_2)\frac{I_{ht}}{N} + \mu\right)t} \geq 0, \\ I_t(t) \geq I_t(0)e^{\left(\alpha(1-\omega_1)\frac{S}{N} - b(1-\omega_3)\frac{S I_{ht}}{N} - (c+\beta_1+\mu)\right)t} \geq 0, \\ I_{ht}(t) \geq I_{ht}(0)e^{\left(\sigma(1-\omega_2)\frac{S}{N} + b(1-\omega_3)\frac{S I_t}{N} - (1+\beta_2+\mu+\gamma_2)\right)t} \geq 0, \\ T_t(t) \geq T_t(0)e^{-(\gamma_1+\beta_3+\mu)t} \geq 0, \\ T_{ht}(t) \geq T_{ht}(0)e^{-(\beta+\mu+\beta_4)t} \geq 0, \\ I_{at}(t) \geq I_{at}(0)e^{-(\gamma_3+\mu+\beta_6)t} \geq 0, \\ I_a(t) \geq I_a(0)e^{-(\mu+\beta_5)t} \geq 0, \\ R_t(t) \geq R_t(0)e^{-(\delta+\mu)t} \geq 0, \end{cases} \quad (13)$$

with

$$\{S(t), I_t(t), I_{ht}(t), T_t(t), T_{ht}(t), I_a(t), I_{at}(t), R_t(t)\} \geq 0. \quad (14)$$

As a result, all the model equations' solutions (6) are positive.

Positive invariant region

A positively invariant region is a subset of the state space such that every solution of the system that starts within this region remains there for all future time. Establishing the existence of such a region ensures that the model is mathematically well-posed and that all state variables remain biologically meaningful. To determine the positively invariant region for the system of equations (6), we begin by summing all the equations of the system, which yields

$$N(t) = S(t) + I_t(t) + I_{ht}(t) + T_t(t) + T_{ht}(t) + I_a(t) + I_{at}(t) + R_t(t), \quad (15)$$

$$\begin{aligned} \frac{dN(t)}{dt} &= \frac{dS}{dt} + \frac{dI_t}{dt} + \frac{dI_{ht}}{dt} + \frac{dT_t}{dt} + \frac{dT_{ht}}{dt} + \frac{dI_a}{dt} + \frac{dI_{at}}{dt} + \frac{dR_t}{dt}, \\ &= B - \mu N - \beta_1 I_t - \beta_2 I_{ht} - \beta_3 T_t - \beta_6 I_{at} - \beta_4 T_{ht} - \beta_5 I_a. \end{aligned} \quad (16)$$

In a stable condition, when the system is free of infections, equation (16) transforms into

$$\frac{dN(t)}{dt} = B - \mu N \leq 0, \Rightarrow B - \mu N \leq 0, \Rightarrow N \leq \frac{B}{\mu}, \quad (17)$$

where

$$\frac{dN(t)}{dt} + \mu N \leq B. \quad (18)$$

The integrating factor for equation (18) is $e^{\mu t}$. Multiply both sides of equation (18) by $e^{\mu t}$ gives

$$e^{\mu t} \frac{dN}{dt} + e^{\mu t} N\mu \leq B e^{\mu t} \Rightarrow d(Ne^{\mu t}) \leq B e^{\mu t} dt, \quad (19)$$

integrating both sides, yields

$$N(t)e^{\mu t} = \frac{B e^{\mu t}}{\mu} + c \Rightarrow N(t) = \frac{B}{\mu} + c e^{-\mu t}. \quad (20)$$

Applying the initial condition $t = 0$; $N(0) = N_0$ gives

$$N_0 \leq \frac{B}{\mu} + c \Rightarrow N_0 - \frac{B}{\mu} \leq c \Rightarrow N(t) \leq \left(N_0 - \frac{B}{\mu}\right) e^{-\mu t} + \frac{B}{\mu}. \quad (21)$$

Therefore, as $t \rightarrow \infty$ in equation (21), the human population N approaches $\frac{B}{\mu}$ (That is, $N \rightarrow K = \frac{B}{\mu}$, the parameter K is called the carrying capacity). Hence, all feasible solution sets of the human population of the system equations (6) enter the region D ,

where

$$D = \{(S(t), I_t(t), I_{ht}(t), T_t(t), T_{ht}(t), I_a(t), I_{at}(t), R_t(t)) \in \mathbb{R}_+^8 : S(t) \geq 0, I_t(t) \geq 0, I_{ht}(t) \geq 0, T_t(t) \geq 0, T_{ht}(t) \geq 0, I_a(t) \geq 0, I_{at}(t) \geq 0, R_t(t) \geq 0, N \leq \frac{B}{\mu}\}. \quad (22)$$

Thus, region D serves as a positively invariant set under the dynamics described by the system equations (6), ensuring all solutions remain within its boundary.

Basic reproduction number R_0

The following is a presentation of the model equations, which are prepared beginning with the recently infectious classes.

$$\begin{cases} \frac{dI_t}{dt} = \alpha(1 - \omega_1) \frac{SI_t}{N} - b(1 - \omega_3) \frac{SI_t I_{ht}}{N} - x_1 I_t, \\ \frac{dI_{ht}}{dt} = \sigma(1 - \omega_2) \frac{SI_{ht}}{N} + b(1 - \omega_3) \frac{SI_t I_{ht}}{N} - x_2 I_{ht}, \\ \frac{dT_t}{dt} = c I_t - x_3 T_t, \\ \frac{dT_{ht}}{dt} = a I_{ht} + \gamma_3 I_{at} - x_4 T_{ht}, \\ \frac{dI_{at}}{dt} = \gamma_2 I_{ht} - x_5 I_{at}, \\ \frac{dI_a}{dt} = \beta T_{ht} + (1 - a) I_{ht} - x_6 I_a, \end{cases} \quad (23)$$

where

$$\begin{aligned} x_1 &= c + \beta_1 + \mu, & x_2 &= 1 + \beta_2 + \mu + \gamma_2, & x_3 &= \gamma_1 + \beta_3 + \mu, \\ x_4 &= \beta + \mu + \beta_4, & x_5 &= \gamma_3 + \mu + \beta_6, & x_6 &= \mu + \beta_5, & x_7 &= \mu + \delta. \end{aligned}$$

To achieve the co-infection model equations' disease-free equilibrium (6), set

$$I_t = I_{ht} = T_t = T_{ht} = I_{at} = I_a = 0.$$

Therefore, disease-free equilibrium of model equations (6) is presented as follows

$$x_0 = \left(\frac{B}{\mu}, 0, 0, 0, 0, 0, 0\right).$$

The next-generation matrix is presented as follows

$$\mathcal{F}_i(x_0) = \begin{pmatrix} \alpha(1-\omega_1)\frac{SI_t}{N} - b(1-\omega_3)\frac{SI_t I_{ht}}{N} \\ \sigma(1-\omega_2)\frac{SI_{ht}}{N} + b(1-\omega_3)\frac{SI_t I_{ht}}{N} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, F = \begin{pmatrix} \alpha(1-\omega_1) & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma(1-\omega_2) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$\mathcal{V}_i(x_0) = \begin{pmatrix} x_1 I_t \\ x_2 I_{ht} \\ -c I_t + x_3 T_t \\ -a I_{ht} - \gamma_3 I_{at} + x_4 T_{ht} \\ -\gamma_2 I_{ht} + x_5 I_{at} \\ -\beta T_{ht} - (1-a) I_{ht} + x_6 I_a \end{pmatrix}, V = \begin{pmatrix} x_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & x_2 & 0 & 0 & 0 & 0 \\ -c & 0 & x_3 & 0 & 0 & 0 \\ 0 & -a & 0 & x_4 & -\gamma_3 & 0 \\ 0 & \gamma_2 & 0 & 0 & x_5 & 0 \\ 0 & -(1-a) & 0 & -\beta & 0 & x_6 \end{pmatrix}.$$

After calculation we get

$$V^{-1} = \begin{pmatrix} \frac{1}{x_1} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{x_2} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{x_3} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{x_4} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{x_5} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{x_6} \end{pmatrix}, FV^{-1} = \begin{pmatrix} \frac{\alpha(1-\omega_1)}{x_1} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\sigma(1-\omega_2)}{x_2} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

Therefore, the number of new cases caused by an infectious disease, denoted as the basic reproduction number according to equation (4), is expressed as follows

$$R_0 = \max\{R_H, R_T\}, \quad \text{where} \quad R_T = \frac{\alpha(1-\omega_1)}{c+\beta_1+\mu}, \quad R_H = \frac{\sigma(1-\omega_2)}{1+\beta_2+\mu+\gamma_2}. \quad (24)$$

Here, R_T is the TB basic reproduction number, and R_H is for HIV. In cases when both R_T and R_H exceed one, the disease spreads within the population. However, the disease dies over time if they are kept below one.

The overall basic reproduction number is defined as the maximum of the disease-specific reproduction numbers because the two diseases interact competitively within the host population. In a co-infection setting, the disease with the higher reproduction number will dominate the epidemiological landscape, as it can sustain transmission even when the other disease is controlled. This is consistent with the findings of [7] and [8], who demonstrated that in co-infection models, the overall R_0 is determined by the dominant disease. The use of the maximum function rather than a sum or product reflects the biological reality that each disease has independent transmission pathways, and the threshold for disease persistence is determined by the disease with the greatest transmission potential.

Stability analysis of disease-free equilibrium (DFE)

Theorem 1 (Local Asymptotic Stability of DFE): The disease-free equilibrium $x_0 = \left(\frac{B}{\mu}, 0, 0, 0, 0, 0\right)$ of the co-infection model (6) is locally asymptotically stable if $R_0 < 1$ (i.e., $R_T < 1$ and $R_H < 1$) and unstable if $R_0 > 1$.

The Jacobian matrix of system (6) evaluated at the DFE is:

$$J_0 = \begin{pmatrix} -\mu & -\alpha(1-\omega_1) & -\sigma(1-\omega_2) & 0 & 0 & 0 & 0 & \delta \\ 0 & [\alpha(1-\omega_1) - x_1] & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & [\sigma(1-\omega_2) - x_2] & 0 & 0 & 0 & 0 & 0 \\ 0 & c & 0 & -x_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & a & 0 & -x_4 & \gamma_3 & 0 & 0 \\ 0 & 0 & \gamma_2 & 0 & 0 & -(\gamma_3 + \mu + \beta_6) & 0 & 0 \\ 0 & 0 & -(1-a) & 0 & \beta & 0 & -(\mu + \beta_5) & 0 \\ 0 & 0 & 0 & \gamma_1 & 0 & 0 & 0 & -(\mu + \delta) \end{pmatrix}.$$

Expressing J_0 in terms of R_T and R_H , we obtain

$$(J_0 - I\lambda) = \begin{pmatrix} -\mu & -\alpha(1-\omega_1) & -\sigma(1-\omega_2) & 0 & 0 & 0 & 0 & \delta \\ 0 & x_1(R_T - 1) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & x_2(R_H - 1) & 0 & 0 & 0 & 0 & 0 \\ 0 & c & 0 & -x_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & a & 0 & -x_4 & \gamma_3 & 0 & 0 \\ 0 & 0 & \gamma_2 & 0 & 0 & -(\gamma_3 + \mu + \beta_6) & 0 & 0 \\ 0 & 0 & -(1-a) & 0 & \beta & 0 & -(\mu + \beta_5) & 0 \\ 0 & 0 & 0 & \gamma_1 & 0 & 0 & 0 & -(\mu + \delta) \end{pmatrix}.$$

After calculation, the aforementioned Jacobian matrix's eigenvalues are

$$\lambda_1 = -\mu, \quad \lambda_2 = x_1(R_T - 1), \quad \lambda_3 = x_2(R_H - 1), \quad \lambda_4 = -(\gamma_1 + \beta_3 + \mu), \\ \lambda_5 = -(\beta + \mu + \beta_4), \quad \lambda_6 = -(\gamma_3 + \mu + \beta_6), \quad \lambda_7 = -(\mu + \beta_5), \quad \lambda_8 = -(\mu + \delta).$$

If $R_T < 1$ and $R_H < 1$, then $\lambda_2 < 0$ and $\lambda_3 < 0$. All other eigenvalues have negative real parts since all parameters are positive. Therefore, all eigenvalues have negative real parts, and the DFE is locally asymptotically stable. Conversely, if $R_T > 1$ or $R_H > 1$, then at least one eigenvalue has a positive real part, and the DFE is unstable.

Theorem 2 (Global Asymptotic Stability of DFE): The disease-free equilibrium x_0 of the co-infection model (6) is globally asymptotically stable in the region D if $R_0 < 1$.

Consider the Lyapunov function:

$$V = I_t + I_{ht} + T_t + T_{ht} + I_{at} + I_a.$$

Differentiating along the trajectories of system (6):

$$\frac{dV}{dt} = \alpha(1-\omega_1) \frac{SI_t}{N} - b(1-\omega_3) \frac{SI_{ht}}{N} - x_1 I_t + \sigma(1-\omega_2) \frac{SI_{ht}}{N} + b(1-\omega_3) \frac{SI_{ht}}{N} - x_2 I_{ht} \\ + cI_t - x_3 T_t + aI_{ht} + \gamma_3 I_{at} - x_4 T_{ht} + \gamma_2 I_{ht} - x_5 I_{at} + \beta T_{ht} + (1-a)I_{ht} - x_6 I_a.$$

Since $S \leq N$ in D , we have:

$$\frac{dV}{dt} \leq \alpha(1 - \omega_1)I_t - x_1I_t + \sigma(1 - \omega_2)I_{ht} - x_2I_{ht} + cI_t - x_3T_t + aI_{ht} + \gamma_3I_{at} - x_4T_{ht} + \gamma_2I_{ht} - x_5I_{at} + \beta T_{ht} + (1 - a)I_{ht} - x_6I_a.$$

Simplifying:

$$\frac{dV}{dt} \leq x_1(R_T - 1)I_t + x_2(R_H - 1)I_{ht} - x_3T_t - x_4T_{ht} - x_5I_{at} - x_6I_a.$$

If $R_T < 1$ and $R_H < 1$, then all coefficients are negative, and $\frac{dV}{dt} < 0$ for all $(I_t, I_{ht}, T_t, T_{ht}, I_{at}, I_a) \neq (0, 0, 0, 0, 0, 0)$. Moreover, $V = 0$ only at the DFE. By LaSalle's invariance principle [25], the DFE is globally asymptotically stable in D .

3.2.5 Analysis of the endemic equilibrium point's stability (EEP)

At the state of endemic equilibrium, both HIV and TB exist in the population. This equilibrium point is achieved when

$$(S(t) = I_t(t) = I_{ht}(t) = T_t(t) = T_{ht}(t) = I_a(t) = I_{at}(t) = R(t)) = 0. \quad (25)$$

The co-infection model equations (6) have the following endemic equilibrium points

$$E^* = (S^*, I_t^*, I_{ht}^*, T_t^*, T_{ht}^*, I_a^*, I_{at}^*, R_t^*),$$

where

$$I_t^* = \frac{(1 + \mu + \gamma_2 + \beta_2)(R_H - 1)}{b(1 - \omega_3)}, I_{ht}^* = \frac{(c + \mu + \beta_1)(R_T - 1)}{b(1 - \omega_3)},$$

$$T_t^* = \frac{c(1 + \mu + \beta_2)(R_H - 1)}{b(1 - \omega_3)(\gamma_1 + \mu + \beta_3)},$$

$$T_{ht}^* = \frac{a(c + \mu + \beta_1)(R_T - 1)}{b(1 - \omega_3)(\beta + \mu + \beta_4)}, R_t^* = \frac{\gamma_1 c(1 + \mu + \gamma_2 + \beta_2)(R_H - 1)}{b(1 - \omega_3)(\gamma_1 + \beta_3 + \mu)(\delta + \mu)},$$

$$I_a^* = \frac{(1 - a)(c + \mu + \beta_1)(R_T - 1)}{b(1 - \omega_3)(\beta + \mu + \beta_4)(\beta_5 + \mu)} + \frac{\beta a(c + \mu + \beta_1)(R_T - 1)}{b(1 - \omega_3)(\beta + \mu + \beta_4)(\beta_5 + \mu)},$$

$$I_{at}^* = \frac{\gamma_2(c + \mu + \beta_1)(R_T - 1)}{b(1 - \omega_3)(\gamma_3 + \mu + \beta_6)},$$

$$S^* = \frac{B - \alpha(1 + \mu + \gamma_2 + \beta_2)(R_H - 1)}{b\mu(1 - \omega_3)} + \frac{b(1 - \omega_2)(x_1)(R_T - 1)}{b\mu(1 - \omega_3)} + \frac{\delta\gamma_1 c(1 + \beta_2 + \mu + \gamma_2)(R_H - 1)}{b\mu(1 - \omega_3)(\gamma_1 + \mu + \beta_3)(\delta + \mu)}.$$

For the model equations (6), the endemic equilibrium point E^* occurs and is unique if and only if $R_H > 1$ and $R_T > 1$. The positivity of I_t^* , T_t^* , and R_t^* , holds when $R_H > 1$. Similarly, the positivity of I_{ht}^* , T_{ht}^* , I_{at}^* , and I_a^* , holds when $R_T > 1$. Additionally, S^* is positive when $R_T > 1$ and $R_H > 1$.

Theorem 3 (Global Asymptotic Stability of EEP): The endemic equilibrium E^* of the co-infection model (6) is globally asymptotically stable in the region D if $R_0 > 1$ (i.e., $R_T > 1$ or $R_H > 1$) and $X < Y$, where X and Y are defined in equations (28) and (29) below.

Consider the Lyapunov function:

$$V = (S - S^* - S^* \ln \frac{S}{S^*}) + (I_t - I_t^* - I_t^* \ln \frac{I_t}{I_t^*}) + (I_{ht} - I_{ht}^* - I_{ht}^* \ln \frac{I_{ht}}{I_{ht}^*}) + (T_t - T_t^* - T_t^* \ln \frac{T_t}{T_t^*}) \\ + (T_{ht} - T_{ht}^* - T_{ht}^* \ln \frac{T_{ht}}{T_{ht}^*}) + (I_{at} - I_{at}^* - I_{at}^* \ln \frac{I_{at}}{I_{at}^*}) + (I_a - I_a^* - I_a^* \ln \frac{I_a}{I_a^*}) + (R_t - R_t^* - R_t^* \ln \frac{R_t}{R_t^*}). \quad (26)$$

A Lyapunov function in this form has been described in [24]. The derivative of V is now expressed as

$$\frac{dV}{dt} = (1 - \frac{S^*}{S})S' + (1 - \frac{I_t^*}{I_t})I_t' + (1 - \frac{I_{ht}^*}{I_{ht}})I_{ht}' + (1 - \frac{T_t^*}{T_t})T_t' + (1 - \frac{T_{ht}^*}{T_{ht}})T_{ht}' + (1 - \frac{I_{at}^*}{I_{at}})I_{at}' \\ + (1 - \frac{I_a^*}{I_a})I_a' + (1 - \frac{R_t^*}{R_t})R_t' \\ = (1 - \frac{S^*}{S})[B - \alpha(1 - \omega_1)\frac{SI_t}{N} - \sigma(1 - \omega_2)\frac{SI_{ht}}{N} + \delta R_t - \mu S] + (1 - \frac{R_t^*}{R_t})[\gamma_1 T_t - (\delta + \mu)R_t] \\ + (1 - \frac{I_t^*}{I_t})[\alpha(1 - \omega_1)\frac{SI_t}{N} - b(1 - \omega_3)\frac{SI_{ht}}{N} - (c + \beta_1 + \mu)I_t] \\ + (1 - \frac{I_{ht}^*}{I_{ht}})[\sigma(1 - \omega_2)\frac{SI_{ht}}{N} + b(1 - \omega_3)\frac{SI_{ht}}{N} - (1 + \beta_2 + \mu + \gamma_2)I_{ht}] \\ + (1 - \frac{T_t^*}{T_t})[cI_t - (\gamma_1 + \beta_3 + \mu)T_t] + (1 - \frac{T_{ht}^*}{T_{ht}})[aI_{ht} + \gamma_3 I_{at} - (\beta + \mu + \beta_4)T_{ht}] \\ + (1 - \frac{I_{at}^*}{I_{at}})[\gamma_2 I_{ht} - (\gamma_3 + \mu + \beta_6)I_{at}] + (1 - \frac{I_a^*}{I_a})[\beta T_{ht} + (1 - a)I_{ht} - (\mu + \beta_5)I_a] \\ = [B - \alpha(1 - \omega_1)\frac{SI_t}{N} - \sigma(1 - \omega_2)\frac{SI_{ht}}{N} + \delta R_t] - \frac{S^*}{S}[B - \alpha(1 - \omega_1)\frac{SI_t}{N} - \sigma(1 - \omega_2)\frac{SI_{ht}}{N} + \delta R_t] \\ + [\gamma_1 T_t - (\delta + \mu)R_t] - \frac{R_t^*}{R_t}[\gamma_1 T_t - (\delta + \mu)R_t] + [\alpha(1 - \omega_1)\frac{SI_t}{N} - b(1 - \omega_3)\frac{SI_{ht}}{N} - (x_1)I_t] \\ - \frac{I_t^*}{I_t}[\alpha(1 - \omega_1)\frac{SI_t}{N} - b(1 - \omega_3)\frac{SI_{ht}}{N} - (\beta_1 + \mu)I_t] + [\sigma(1 - \omega_2)\frac{SI_{ht}}{N} + b(1 - \omega_3)\frac{SI_{ht}}{N} - (x_2)I_{ht}] \\ - \frac{I_{ht}^*}{I_{ht}}[\sigma(1 - \omega_2)\frac{SI_{ht}}{N} + b(1 - \omega_3)\frac{SI_{ht}}{N} - (x_2)I_{ht}] + [cI_t - (\gamma_1 + \beta_3 + \mu)T_t] - \frac{T_t^*}{T_t}[cI_t - (x_3)T_t] \\ + [aI_{ht} + \gamma_3 I_{at} - (x_4)T_{ht}] - \frac{T_{ht}^*}{T_{ht}}[aI_{ht} + \gamma_3 I_{at} - (x_4)T_{ht}] + [\gamma_2 I_{ht} - (\gamma_5 + \mu + \beta_6)I_{at}] \\ - \frac{I_{at}^*}{I_{at}}[\gamma_2 I_{ht} - (\gamma_5 + \mu + \beta_6)I_{at}] + [\beta T_{ht} + (1 - a)I_{ht} - (x_6)I_a] - \frac{I_a^*}{I_a}[\beta T_{ht} + (1 - a)I_{ht} - (\mu + \beta_5)I_a]. \quad (27)$$

Thus $\frac{dV}{dt} = X - Y$, where

$$X = B + \delta R_t + \alpha S^*(1 - \omega_1)\frac{I_t}{N} + \sigma S^*(1 - \omega_2)\frac{I_{ht}}{N} + \mu S^* + \alpha(1 - \omega_1)\frac{SI_t}{N} + bI_t^*(1 - \omega_3)\frac{SI_{ht}}{N} + cI_t \\ + (c + \beta_1 + \mu)I_t^* + \sigma(1 - \omega_2)\frac{SI_{ht}}{N} + b(1 - \omega_3)\frac{SI_{ht}}{N} + (1 + \beta_2 + \mu + \gamma_2)I_{ht}^* + (\gamma_2 + \mu + \beta_6)I_{at}^* \\ + \beta T_{ht} + (1 - a)I_{ht} + (\mu + \beta_5)I_a^*. \quad (28)$$

$$Y = \alpha(1 - \omega_1)\frac{SI_t}{N} + \sigma(1 - \omega_2)\frac{SI_{ht}}{N} + \mu S + B\frac{S^*}{S} + \delta R_t\frac{S^*}{S} + (c + \beta_1 + \mu)I_t + \sigma(1 - \omega_1)\frac{SI_t^*}{N} \\ + (\gamma_1 + \beta_3 + \mu)T_t + c\frac{T_t^* I_t}{T_t} + (\beta + \mu + \beta_4)T_{ht} + a\frac{T_{ht}^* I_{ht}}{T_{ht}} + \gamma_3\frac{T_{ht}^* I_{at}}{T_{ht}} + (\gamma_3 + \mu + \beta_6)I_{at} + \gamma_2\frac{I_{at}^* I_{ht}}{I_{at}} \\ + (\mu + \beta_5)I_a + \beta\frac{I_a^* T_{ht}}{I_a} + (1 - a)\frac{I^* I_{ht}}{I_a}. \quad (29)$$

Given that X and Y are positive, if $X < Y$, then $\frac{dV}{dt} = X - Y < 0$, and when $S = S^*$, $I_t = I_t^*$, $I_{ht} = I_{ht}^*$, $T_t = T_t^*$, $T_{ht} = T_{ht}^*$, $I_{at} = I_{at}^*$, $I_a = I_a^*$, and $R_t = R_t^*$ in D , then $\frac{dV}{dt} = 0$. Therefore, the largest compact invariant set in $\{(S^*, I_t^*, I_{ht}^*, T_t^*, T_{ht}^*, I_{at}^*, I_a^*, R_t^*) \in D : \frac{dV}{dt} = 0\}$ is the singleton E^* . By LaSalle's invariance principle [25], it follows that E^* is globally asymptotically stable on D if $X < Y$.

Sensitivity analysis

We examine the basic reproduction numbers in equation (24) and the sensitivity indices with respect to the model's parameters. From equation (5), we obtain the sensitivity of R_T and R_H respectively, using the numerical values of the parameters presented in Table 1.

To determine the sensitivity indices of R_T , we set:

$$\begin{aligned} \Upsilon_{\alpha}^{R_T} &= \frac{\alpha}{R_T} \frac{\partial R_T}{\partial \alpha} = \left(\frac{\alpha(c + \beta_1 + \mu)}{\alpha(1 - \omega_1)} \right) \left(\frac{1 - \omega_1}{c + \beta_1 + \mu} \right) = 1, \\ \Upsilon_{\omega_1}^{R_T} &= \frac{\omega_1}{R_T} \frac{\partial R_T}{\partial \omega_1} = \left(\frac{\omega_1(c + \beta_1 + \mu)}{\alpha(1 - \omega_1)} \right) \left(\frac{-\alpha}{(c + \beta_1 + \mu)} \right) = \frac{-\omega_1}{1 - \omega_1}, \\ \Upsilon_c^{R_T} &= \frac{c}{R_T} \frac{\partial R_T}{\partial c} = \left(\frac{c(c + \beta_1 + \mu)}{\alpha(1 - \omega_1)} \right) \left(\frac{-\alpha(1 - \omega_1)}{(c + \beta_1 + \mu)^2} \right) = \frac{-c}{c + \beta_1 + \mu}. \end{aligned}$$

Similarly, for R_H :

$$\Upsilon_{\sigma}^{R_H} = 1, \Upsilon_{\omega_2}^{R_H} = \frac{-\omega_2}{1 - \omega_2}, \Upsilon_a^{R_H} = \frac{-a}{a + \beta_2 + \mu + \gamma_2}.$$

Using the parameter values from Table 1 and equation (24), we obtain:

$$\begin{aligned} R_T &= \frac{0.1 \times (1 - 0.8)}{0.7 + 0.08 + 0.013} = \frac{0.02}{0.793} = 0.02522, \\ R_H &= \frac{0.1 \times (1 - 0.8)}{0.8 + 0.1 + 0.013 + 0.8} = \frac{0.02}{1.713} = 0.01168. \end{aligned}$$

Therefore, the basic reproduction number is:

$$R_0 = \max(0.02522, 0.01168) = 0.02522.$$

Table 2: Sensitivity Indices of R_T

Parameter	Sensitivity Index	Sign
α	+1.000	Positive
ω_1	-4.000	Negative
c	-0.883	Negative
β_1	-0.101	Negative
μ	-0.016	Negative

Table 3: Sensitivity Indices of R_H

Parameter	Sensitivity Index	Sign
σ	+1.000	Positive
ω_2	-4.000	Negative
a	-0.467	Negative

β_2	-0.058	Negative
γ_2	-0.467	Negative

The results illustrate the impact of the chosen parameters on R_T and R_H are depicted in Figure 2 and Figure 3, respectively.

Figure 2: The positive (resp. negative) impact of the parameter α (a) and ω_1 (b) on R_T .

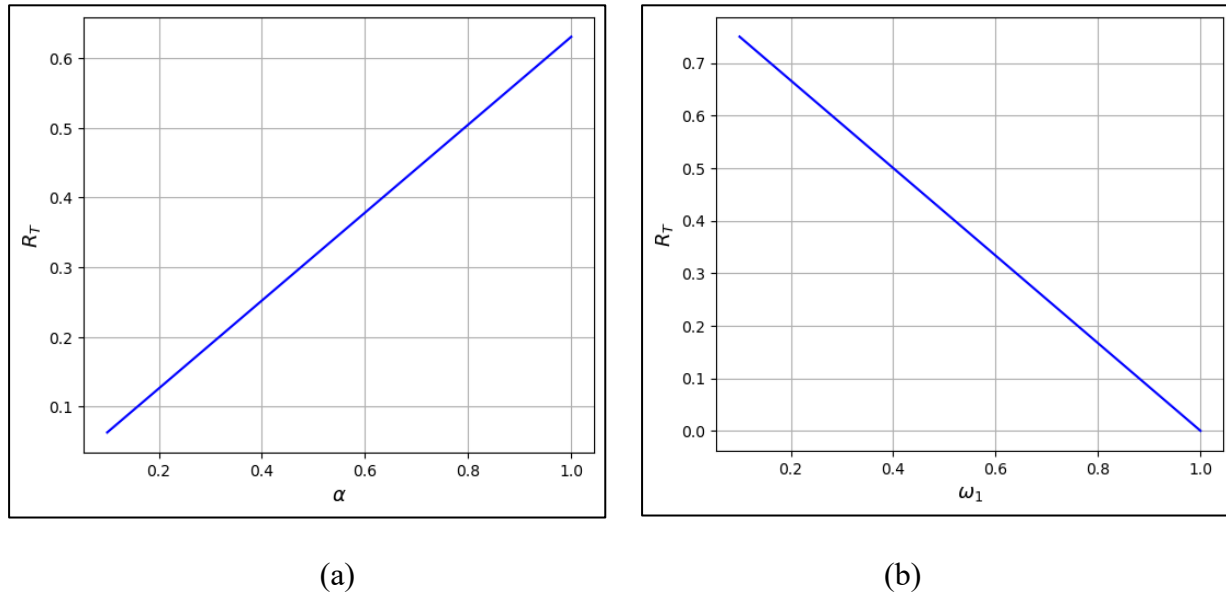
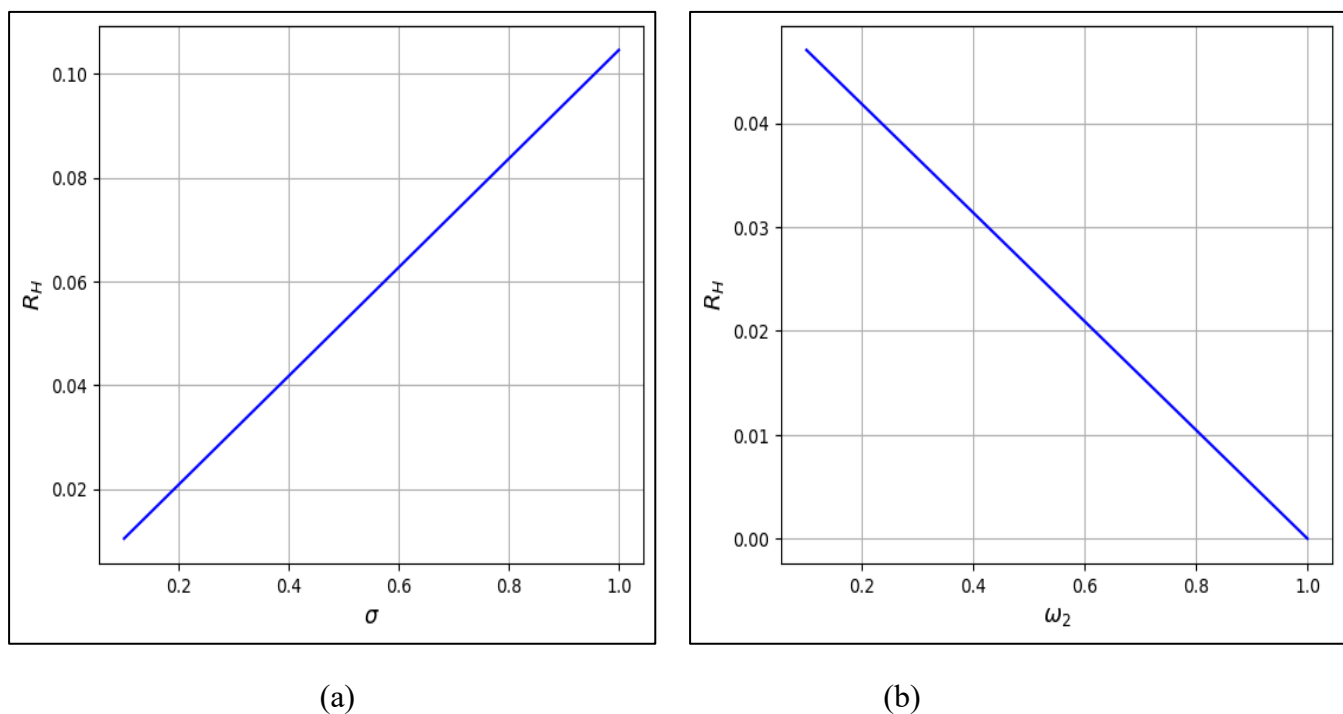


Figure 3: The positive (resp. negative) impact of the parameter σ (a) and ω_2 (b) on R_H .



The impact of α and ω_1 on the susceptible and TB-infected population, as well as σ and ω_2 on the same variables, is illustrated in Figure 4 and Figure 5, respectively.

Figure 4: Trend of susceptible and TB-infected population for different values of α (a) and ω_1 (b).

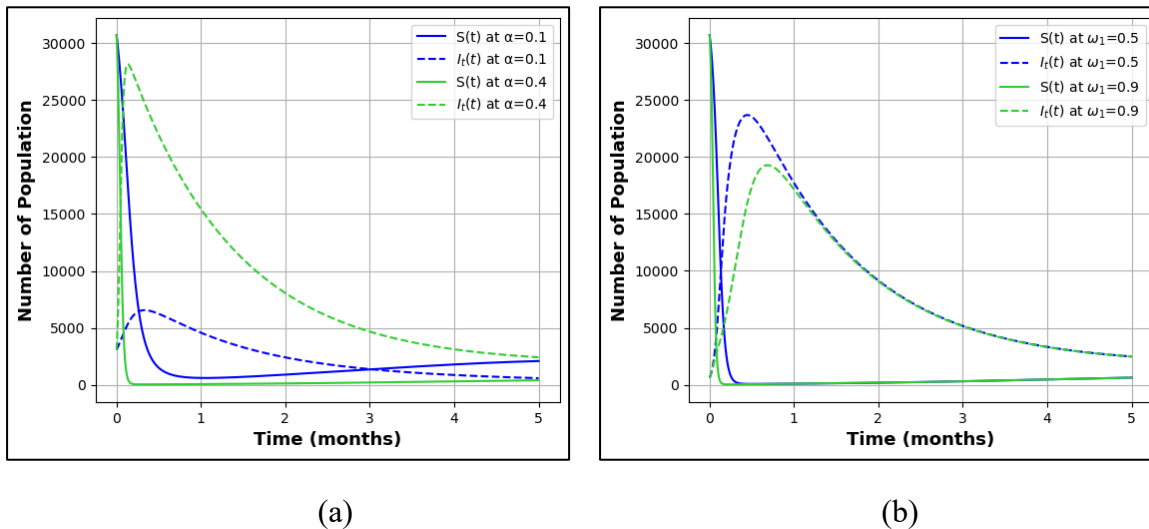
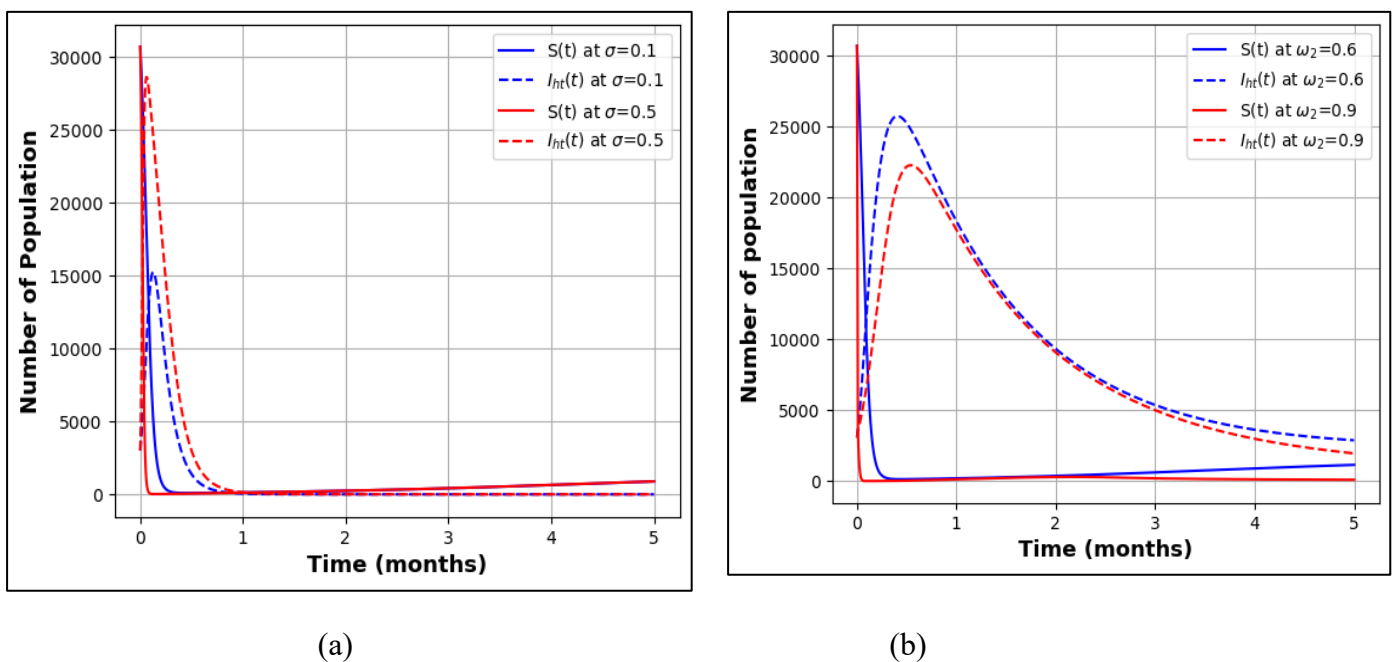
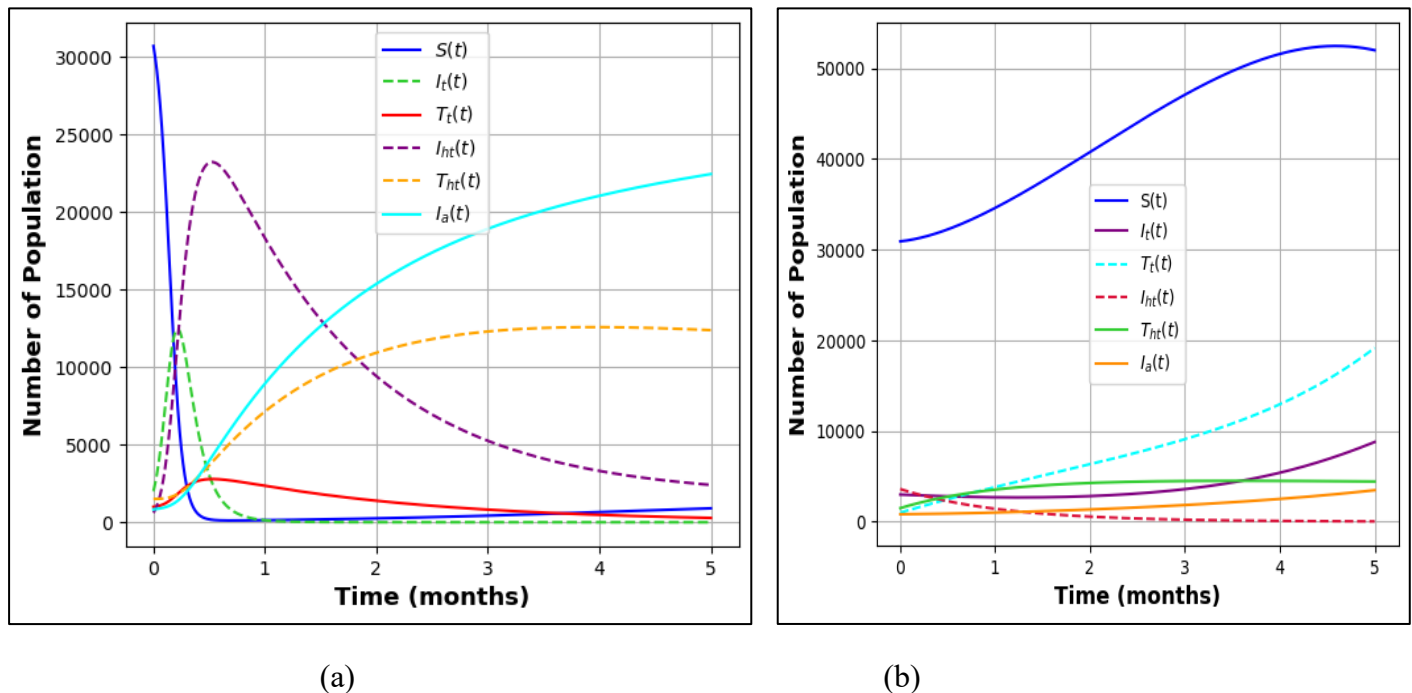


Figure 5: Trend of susceptible and co-infected population for different values of σ (a) and ω_2 (b).



To ensure the instability and stability of the mathematical model, the results of numerical simulation using the values of the parameters given in Table 1 are depicted in Figure 6 (a). Meanwhile, Figure 6 (b) displays the numerical simulation's results using the parameter values settings $\alpha = 0.009$, $\sigma = 0.009$, $\omega_1 = 0.9$, $\omega_2 = 0.87$, $a = 0.9$, $c = 0.94$, $\mu = 0.038$, $B = 0.49$, $\delta = 0.08$, $\gamma_1 = 0.6$, $\omega_3 = 0.9$, $\beta_1 = 0.7$, $\beta_2 = 0.001$, $\beta_3 = 0.05$, $\beta_4 = 0.03$, and $\beta_5 = 0.06$.

Figure 6: Variation of the model variables involves utilizing the parameter values outlined in Table 1 (a) alongside additional set parameter values (b).



NUMERICAL SIMULATION RESULTS

Simulation Setup

All numerical simulations were performed using MATLAB's ode45 solver, which implements an explicit Runge-Kutta (4,5) formula (the Dormand-Prince pair) with adaptive step-size control. The relative tolerance was set to 10^{-6} , and the absolute tolerance was set to 10^{-8} . The integration interval was $[0, 5]$ months for all simulations. Initial conditions were set as follows:

$$S(0) = 10000, I_t(0) = 100, I_{ht}(0) = 50, T_t(0) = 20, \\ T_{ht}(0) = 10, I_{at}(0) = 5, I_a(0) = 5, R_t(0) = 0.$$

These initial conditions represent a scenario where a small number of infectious individuals are introduced into a largely susceptible population.

Results

Figure 2 demonstrates the sensitivity of R_T to changes in α and ω_1 . Panel (a) shows that increasing α from 0.1 to 1.0 increases R_T linearly from approximately 0.03 to 0.63. Panel (b) shows that increasing ω_1 from 0.1 to 1.0 decreases R_T from approximately 0.75 to 0. This confirms that ω_1 is a critical control parameter. Figure 3 shows the corresponding results for R_H . Panel (a) shows that increasing σ increases R_H , while panel (b) shows that increasing ω_2 decreases R_H . Figure 4 illustrates the temporal dynamics of the susceptible and TB-infected populations for different values of α and ω_1 . Panel (a) shows that when $\alpha = 0.1$, the susceptible population declines slowly while the TB-infected population rises to a peak of approximately 8,000 before declining. When $\alpha = 0.4$, the susceptible population declines more rapidly, and the TB-infected population peaks at approximately 12,000. Panel (b) shows that when $\omega_1 = 0.5$, the TB-infected population peaks at approximately 24,000, while when $\omega_1 = 0.9$, the peak is reduced to approximately 19,000.

Figure 5 shows the temporal dynamics of the susceptible and co-infected populations for different values of σ and ω_2 . Panel (a) shows that increasing σ from 0.1 to 0.5 leads to a more rapid decline in the susceptible population and a higher peak in the co-infected population. Panel (b) shows that increasing ω_2 from 0.6 to 0.9 reduces the co-infected population. Figure 6 shows the overall population dynamics under two different parameter scenarios. Panel (a), using the parameter values in Table 1, shows an initial increase in TB-infected and co-infected populations accompanied by a decline in the susceptible population, while the number of AIDS-infected individuals increases steadily. Panel (b), using parameter values corresponding to disease stability, shows an increase in the susceptible population and a decline in the TB-infected, AIDS-infected, and co-infected populations.

DISCUSSION

Sensitivity analysis results presented in Table 2 indicate that parameters with positive sensitivity indices contribute to increased disease endemicity, whereas parameters with negative sensitivity indices reduce endemicity. As shown in Table 2, parameters with negative sensitivity indices play a crucial role in reducing the basic reproduction number for tuberculosis, R_T , to values below unity. Consequently, these parameters should be prioritized in designing intervention strategies aimed at controlling TB transmission within the study population. Among these, the parameter ω_1 , which represents the effectiveness of respiratory hygiene practices such as covering the mouth while coughing or sneezing, emerges as the most influential. As indicated in Table 2, ω_1 has the largest negative sensitivity index (-4.0000), highlighting its substantial impact on reducing TB transmission. This finding underscores the importance of educational campaigns promoting proper respiratory hygiene as a key public health intervention.

Similarly, Table 3 identifies parameters with negative sensitivity indices that significantly influence the HIV basic reproduction number, R_H . These parameters are essential for reducing R_H below unity and, hence, for limiting HIV transmission. Notably, the parameter ω_2 , which represents adherence to safe sexual practices and avoidance of contact with infected body fluids, exhibits the highest negative sensitivity index (-4.0000). This result emphasizes the critical role of targeted interventions and awareness programs in mitigating the HIV disease burden. The effects of these parameters on R_T and R_H are further supported by numerical simulations, as illustrated in Figures 2 and 3. Figure 2 demonstrates that increases in the transmission-related parameters α and σ lead to higher values of R_T and R_H , thereby promoting disease persistence. In contrast, Figure 3 shows that increasing the control-related parameters ω_1 and ω_2 results in a decline in both reproduction numbers, suggesting the potential for disease elimination within the community.

Figure 4 illustrates the impact of variations in α and ω_1 on the transmission dynamics from susceptible individuals to those infected with TB. As shown in Figure 4(a), increasing α from 0.1 to 0.4 leads to a reduction in the susceptible population and a corresponding increase in the TB-infected population. Conversely, Figure 4(b) indicates that increasing ω_1 from 0.5 to 0.9 results in a decrease in the number of TB-infected individuals. The effects of the parameters σ and ω_2 , which govern transmission from susceptible individuals to co-infected individuals, are depicted in Figure 5. Figure 5(a) shows that as σ increases from 0.1 to 0.5, the susceptible population declines while the number of co-infected individuals rises. In contrast, Figure 5(b) demonstrates that increasing ω_2 from 0.6 to 0.9 leads to a reduction in the co-infected population.

Finally, Figure 6 illustrates the overall population dynamics under different parameter scenarios. Figure 6(a), which uses the parameter values listed in Table 1, shows an initial increase in TB-infected and co-infected populations accompanied by a decline in the susceptible population, while the number of AIDS-infected individuals increases steadily. In contrast, Figure 6(b), which employs parameter values corresponding to disease stability, shows an increase in the susceptible population and a decline in the TB-infected, AIDS-infected, and co-infected populations.

Limitations

This study has several limitations that should be acknowledged. First, the model assumes homogeneous mixing, which may not accurately reflect real-world contact patterns. Second, the parameter values used in the

simulations are based on published literature and may not be representative of all settings. Third, the model does not account for drug resistance, treatment adherence, demographic structure, or healthcare access, all of which can significantly influence disease dynamics. Fourth, the assumption of a constant recruitment rate B may not hold in populations with significant migration or demographic changes. Future work should address these limitations by incorporating more realistic features such as age structure, spatial heterogeneity, and behavioral heterogeneity.

CONCLUSIONS

In this study, we formulated and analyzed an eight-compartment nonlinear ordinary differential equation model to investigate the co-dynamics of tuberculosis (TB) and HIV transmission. The model was shown to be well-posed, with positive and bounded solutions. Analysis of the co-infection model yielded basic reproduction numbers for TB (R_T) and HIV (R_H). We established that when the overall reproduction number satisfies $R_0 < 1$ (implying $R_T < 1$ and $R_H < 1$), the disease-free equilibrium is globally asymptotically stable, indicating that both diseases would eventually be eliminated from the population. Conversely, when $R_0 > 1$ (implying $R_T > 1$ or $R_H > 1$), an endemic equilibrium exists and is globally asymptotically stable, signifying persistent disease circulation. Sensitivity analysis identified ω_1 as the most influential parameter for reducing R_T , and ω_2 as the most influential for reducing R_H . Numerical simulations supported these findings, demonstrating that reducing contact rates with infectious individuals and enhancing control measures ω_1 and ω_2 significantly lowers infection incidence. These results underscore the potential effectiveness of targeted educational campaigns in controlling TB and HIV. Future work could extend this analysis by formulating and evaluating optimal control strategies to further quantify the impact of such interventions.

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