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Application of Optimal Control to the SEITRS Mathematical Model of Tuberculosis Transmission with Control Variables Socialization and Therapy

Muhammad Rafiq[1*], Sri Wigantono[2], Indriasri Raming[2]

[1] *Study Program of Mathematics, Universitas Mulawarman, Indonesia.*
[*] *Corresponding author: sriwigantono@fmipa.unmul.ac.id*

A B S T R A C T

Tuberculosis (TB) is an infectious disease caused by *Mycobacterium tuberculosis*, which remains a serious public health concern. The objective of this study is to develop, analyze, and propose an optimal control strategy for the transmission dynamics of TB using an SEITRS mathematical model. The model consists of five population compartments: Susceptible (S), Exposed (E), Infected (I), Treatment (T), and Recovered (R). The methodology involves constructing the SEITRS model, determining the equilibrium points, and analyzing their stability under different conditions of the basic reproduction number. The model has two equilibrium points, namely the non-endemic and endemic equilibrium. If the basic reproduction number is less than one and certain conditions are satisfied, the non-endemic equilibrium is locally asymptotically stable. Conversely, if the basic reproduction number is greater than one and specific conditions are met, the endemic equilibrium becomes locally asymptotically stable. Furthermore, this study provides optimal control strategies in the SEITRS model. We use two control variables in this model, namely socialization and therapy, to reduce the number of infected individuals. The sufficient conditions for the existence of optimal controls are derived using Pontryagin's Maximum Principle. Numerical simulations are then conducted to examine the impact of applying these controls on the system. The simulation results indicate that the simultaneous implementation of socialization and therapy controls is effective in reducing the number of TB-infected individuals.

Keywords: Numerical simulation, optimal control, stability analysis, SEITRS mathematical model, tuberculosis.

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1. Introduction

Tuberculosis (TB) remains a significant public health issue, particularly in developing countries. Despite being a long-known disease, TB continues to pose a serious threat due to its high transmission and mortality rates. The COVID-19 pandemic has exacerbated the situation by shifting healthcare priorities, causing disruptions in early detection, case reporting, and treatment programs. According to the *Global Tuberculosis Report 2023*, the pandemic contributed to a rise in undiagnosed TB cases, increasing the risk of transmission and mortality [1].

Caused by *Mycobacterium tuberculosis*, TB is the second deadliest infectious disease after COVID-19 [2]. It is primarily spread through airborne droplets from infected individuals, though other transmission routes such as unpasteurized milk or skin lesions also exist. Common symptoms include prolonged cough, hemoptysis, chest pain, and persistent fever [3]. WHO data from 2022 indicates that Indonesia had approximately 969,000 TB cases, placing it second globally after India, with an incidence rate of 354 per 100,000 population and an estimated 150,000 deaths [4].

Environmental and behavioral factors—such as close contact with TB patients, poor ventilation, smoking, and overcrowding—contribute to TB spread [5]. Although vaccination helps reduce susceptibility, its effectiveness decreases with age, requiring treatment to prevent further transmission [6]. Latent infections can also reactivate under certain conditions [1].

Mathematical models play an essential role in understanding disease dynamics. The classical SIR model, introduced by Kermack and McKendrick in 1927, divides populations into susceptible, infected, and recovered groups [7]. Various studies have extended this model. Saleng *et al.* (2022) proposed an MSEITR TB model incorporating vaccination and treatment [8], while Faruk (2016) added treatment rates to a SEIR model, showing faster infection reduction with higher therapy intensity [9]. Puspitasari *et al.* (2019) and Khan *et al.* (2018) explored vaccination, therapy, and socialzation in TB and Hepatitis B models, respectively [3, 10].

This study modifies the MSEITR model from Saleng *et al.* by removing the maternal antibody compartment and adding a reinfection parameter (δ), forming an SEITRS model. The exclusion of the *Maternal Antibody* compartment is justified by the study's focus on a general heterogeneous population characterized by variations in age groups, immune status, and risk factors. Epidemiological evidence indicates that approximately 90% of tuberculosis (TB) cases occur among adults, rendering maternal immunity irrelevant in this context [4]. Furthermore, the introduction of the parameter δ is essential to represent the risk of TB reactivation within the adult population, as recovery from infection does not confer permanent immunity. The analysis includes equilibrium points, the basic reproduction number, and stability. Optimal control strategies socialzation and therapy are applied using Pontryagin's Maximum Principle. Numerical simulations via the fourth order Runge-Kutta method are conducted to compare system behavior with and without control interventions.

2. Research Methods

This study employs a mathematical modeling approach to analyze the transmission dynamics of tuberculosis (TB) using a modified SEITRS model. The research procedure consists of four main stages: model formulation, model analysis, optimal control formulation, and numerical simulation.

2.1. Model Formulation

The proposed SEITRS model is developed by modifying the MSEITR model introduced by Saleng *et al.* (2022). The maternal antibody compartment is removed, and a reinfection parameter is added to represent the recurrence of TB infection. The total human population $N(t)$ is divided into five compartments: susceptible (S), exposed (E), infected (I), treatment (T), and recovered (R).

A system of nonlinear ordinary differential equations is constructed to describe the rate of change for each compartment. The parameters used in the model, such as transmission rate, recovery rate, and natural death rate, are adopted from relevant epidemiological literature to ensure biological relevance.

2.2. Model Analysis

The equilibrium points of the SEITRS model are determined to identify the disease-free and endemic states. The equilibrium points are obtained by solving the system under the steady-state condition $F(X^*) = 0$. To investigate the system's behavior in the neighborhood of these points, the

Jacobian matrix is constructed as

$$J(X) = \left[\frac{\partial F_i}{\partial x_j}\right]$$

and subsequently evaluated at each equilibrium to derive the characteristic polynomial, which serves as the basis for the local stability analysis [11].

The basic reproduction number is derived using the *next-generation matrix* approach by defining the new infection matrix F and the transition matrix V, leading to

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

where $\rho(\cdot)$ denotes the spectral radius of the resulting matrix [12].

Local stability is assessed using the Routh–Hurwitz criterion by examining the signs of the Hurwitz determinants associated with the characteristic polynomial

$$P(\lambda) = \lambda^n + a_1\lambda^{n-1} + a_2\lambda^{n-2} + \cdots + a_n = 0. \tag{1}$$

Each root may be either real or complex and must satisfy $P(\lambda_i) = 0$, for all $i = 1, 2, \ldots, n$. The Hurwitz matrix is denoted by H_n, which consists of the coefficients a_i from the characteristic equation (1). Each of the n Hurwitz matrices can be expressed as [8]

$$H_1 = (a_1),$$

$$H_2 = \begin{pmatrix} a_1 & 1 \\ 0 & a_2 \end{pmatrix},$$

$$H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{pmatrix},$$

$$\vdots$$

$$H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \cdots & 0 & 0 \\ a_3 & a_2 & a_1 & 1 & \cdots & 0 & 0 \\ a_5 & a_4 & a_3 & a_2 & \ddots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & a_{n-1} & a_{n-2} \\ 0 & 0 & 0 & 0 & \cdots & 0 & a_n \end{pmatrix}.$$

In general, the disease-free equilibrium is locally stable when $R_0 < 1$, whereas an endemic equilibrium exists and becomes locally stable when $R_0 > 1$.

2.3. Optimal Control Formulation

To reduce the number of infected individuals, two time-dependent control variables are introduced: $u_1(t)$ representing socialization, and $u_2(t)$ representing therapy. The objective functional is formulated to minimize the number of infected individuals while considering the costs of implementing control measures. The necessary conditions for optimality are derived using Pontryagin's Maximum Principle, leading to a system of state and adjoint equations with transversality conditions [13].

2.4. Numerical Simulation

Numerical simulations are carried out using the fourth-order Runge–Kutta method. Simulations are conducted under three scenarios : without control, with a single control, and with both controls simultaneously. The results are used to evaluate the effects of socialization and therapy efforts on TB transmission dynamics and to compare the outcomes in terms of infection reduction and control efficiency.

3. Result and Discussion

3.1. Mathematical Model of Tuberculosis Transmission

The population in this model refers to the study by [8], and the compartmental diagram of the model is shown in Figure 1 below.

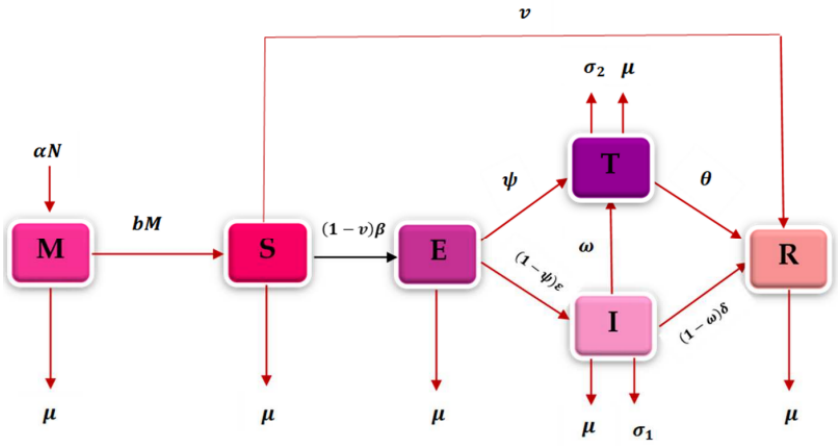

Figure 1. Diagram transmission of the MSEITR TB Model

Based on the transmission diagram in Figure 1, a system of differential equations is formulated as follows :

$$\frac{dM}{dt} = \alpha N - bM - \mu M, \tag{2}$$

$$\frac{dS}{dt} = bM - (1-\nu)\beta\frac{I}{N}S - \nu S - \mu S, \tag{3}$$

$$\frac{dE}{dt} = (1-\nu)\beta\frac{I}{N}S - (1-\psi)\varepsilon E - \psi E - \mu E, \tag{4}$$

$$\frac{dI}{dt} = (1-\psi)\varepsilon E - (1-\omega)\delta I - (\omega + \mu + \sigma_1)I, \tag{5}$$

$$\frac{dT}{dt} = \omega I + \psi E - (\theta + \sigma_2)T - \mu T, \tag{6}$$

$$\frac{dR}{dt} = \theta T + \nu S + (1-\omega)\delta I - \mu R. \tag{7}$$

Next, the model expressed in Equations (2) to (7) is modified by removing the *M (Maternal Antibody)* compartment, adjusting several notations, and adding a parameter for the rate of immunity loss (δ) in the *R (Recovered)* compartment. The assumptions used in constructing the mathematical model of TB transmission without control are as follows :

1. New individuals enter the population through birth (Λ) and die naturally at a rate μ is same,
2. Susceptible individuals (S) become infected through contact with infectious individuals (I) at a rate β, and move to the exposed compartment (E),
3. Individuals in E progress to active infection (I) at a rate ε, or directly enter treatment (T) at a rate ψ,
4. Individuals in I move to T at a rate ω, or die naturally (μ),

5. Individuals in T recover to R at a rate θ, or die naturally μ,
6. Individuals in R may lose immunity and return to S at a rate δ, and
7. Susceptible individuals (S) can be vaccinated and move to R at a rate ρ.

Based on these assumptions, the TB transmission diagram without control is illustrated in Figure 2.

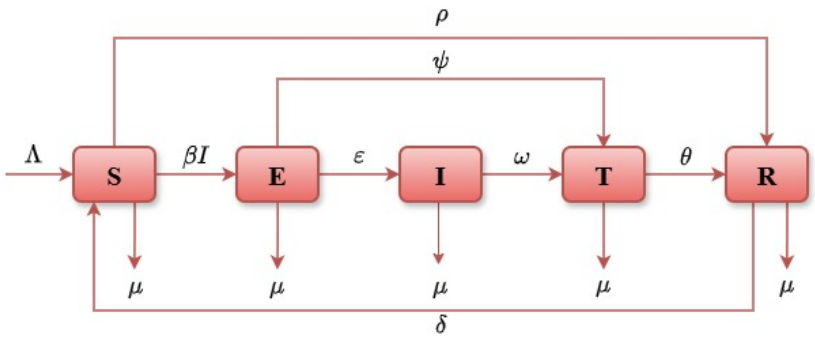

Figure 2. Diagram transmission of the SEITRS TB Model

Based on the transmission diagram in Figure 2, a system of differential equations is formulated as follows :

$$\frac{dS}{dt} = \Lambda N - \beta \frac{I}{N} S - \mu S - \rho S + \delta R, \tag{8}$$

$$\frac{dE}{dt} = \beta \frac{I}{N} S - (\varepsilon + \mu + \psi) E, \tag{9}$$

$$\frac{dI}{dt} = \varepsilon E - (\mu + \omega) I, \tag{10}$$

$$\frac{dT}{dt} = \omega I - (\mu + \theta) T + \psi E, \tag{11}$$

$$\frac{dR}{dt} = \theta T - (\mu + \delta) R + \rho S, \tag{12}$$

with parameter descriptions given in Table 1

Table 1. Description of Parameters in the TB Transmission Model without Control

Parameter	Description	Unit	Condition
Λ	Birth rate	1/month	$\Lambda > 0$
μ	Natural death rate	1/month	$\mu > 0$
ρ	Vaccination rate	1/month	$\rho > 0$
β	TB infection rate	1/month	$\beta > 0$
ω	Treatment rate for the infected class	1/month	$\omega > 0$
θ	Recovery rate through treatment	1/month	$\theta > 0$
ε	Incubation rate	1/month	$\varepsilon > 0$
ψ	Treatment rate for the exposed class	1/month	$\psi > 0$
δ	Immunity waning rate	1/month	$\delta > 0$

Let

$$s = \frac{S}{N}, \quad e = \frac{E}{N}, \quad i = \frac{I}{N}, \quad t_h = \frac{T}{N}, \quad r = \frac{R}{N},$$

and satisfying the relation $n = s + e + i + t_h + r = 1$.
Thus, the new system of differential equations is obtained as follows :

$$\frac{ds}{dt} = \Lambda - \beta si - \mu s - \rho s + \delta r, \tag{13}$$
$$\frac{de}{dt} = \beta si - (\mu + \varepsilon + \psi)e, \tag{14}$$
$$\frac{di}{dt} = \varepsilon e - (\mu + \omega)i, \tag{15}$$
$$\frac{dt_h}{dt} = \omega i - (\mu + \theta)t_h + \psi e, \tag{16}$$
$$\frac{dr}{dt} = \theta t_h - (\mu + \delta)r + \rho s. \tag{17}$$

3.1.1. Non-Endemic Equilibrium Point

The equilibrium point of Equations (13) to (17) is a solution that can be obtained by solving the system from $\frac{ds}{dt} = 0$, $\frac{de}{dt} = 0$, $\frac{di}{dt} = 0$, $\frac{dt_h}{dt} = 0$, and $\frac{dr}{dt} = 0$. At the equilibrium point, the growth rate of the system remains constant. In other words, there is no change in population size (steady state). Let $b_1 = \mu+\varepsilon+\psi$, $b_2 = \mu+\omega$, $b_3 = \mu+\theta$ and $b_4 = \mu+\delta$. Furthermore, Equations (13) to (17) can be written as follows :

$$\Lambda - \beta si - \mu s - \rho s + \delta r = 0, \tag{18}$$
$$\beta si - b_1 e = 0, \tag{19}$$
$$\varepsilon e - b_2 i = 0, \tag{20}$$
$$\omega i - b_3 t_h + \psi e = 0, \tag{21}$$
$$\theta t_h - b_4 r + \rho s = 0. \tag{22}$$

Next, from Equations (18) to (22), the non-endemic equilibrium point will be determined.

The non-endemic equilibrium point is a condition where there is no disease transmission in the population, so that $i = 0$. By solving Equations (18)-(22), the non-endemic equilibrium point is obtained as

$$E_0 = (s, e, i, t_h, r) = \left(\frac{b_4\Lambda}{b_4\mu + b_4\rho - \delta\rho}, 0, 0, 0, \frac{\rho\Lambda}{b_4\mu + b_4\rho - \delta\rho}\right).$$

All infection-related compartments, namely e, i, and t_h, are equal to zero, indicating that there is no active disease transmission within the population.

3.1.2. Endemic Equilibrium Point

The endemic equilibrium point is a condition where disease transmission occurs within the population, so that $i \neq 0$. By solving Equations (18)-(22), the endemic equilibrium point is obtained as $E_1 = (s^*, e^*, i^*, t_h^*, r^*)$ with

$$s^* = \frac{b_1 b_2}{\beta\varepsilon},$$
$$e^* = \frac{b_2 b_3(\Lambda\beta\varepsilon b_4 - \mu b_1 b_2 b_4 - \rho b_1 b_2 b_4 + \delta\rho b_1 b_2)}{\beta\varepsilon(b_1 b_2 b_3 b_4 - \delta\theta(\psi b_2 + \omega\varepsilon))},$$
$$i^* = \frac{\Lambda\beta\varepsilon b_3 b_4 - \mu b_1 b_2 b_3 b_4 - \rho b_1 b_2 b_3 b_4 + \delta\rho b_1 b_2 b_3}{\beta(b_1 b_2 b_3 b_4 - \delta\theta(\psi b_2 + \omega\varepsilon))},$$

$$t_h^* = \frac{(\psi b_2 + \omega\varepsilon)(\Lambda\beta\varepsilon b_4 - \mu b_1 b_2 b_4 - \rho b_1 b_2 b_4 + \delta\rho b_1 b_2)}{\beta\varepsilon(b_1 b_2 b_3 b_4 - \delta\theta(\psi b_2 + \omega\varepsilon))},$$
$$r^* = \frac{b_1 b_2(b_1 b_2 b_3\rho - \mu b_2\psi\theta - \mu\omega\theta\varepsilon - \rho b_2\psi\theta - \rho\omega\theta\varepsilon) + \Lambda\beta\varepsilon b_2\psi\theta + \Lambda\beta\varepsilon^2\omega\theta}{\beta\varepsilon\,(b_1 b_2 b_3 b_4 - \delta\theta(\psi b_2 + \omega\varepsilon))}.$$

All infection-related compartments, namely e, i, and t_h, are not equal to zero, indicating that there is active disease transmission within the population.

3.1.3. Basic Reproduction Number

The basic reproduction number ($\mathcal{R}$,) is calculated using the Next Generation Matrix (NGM) method based on the infective compartments E and I. The infection matrix (F) and the transition matrix (V) at the point E_0 are given by

$$F = \begin{bmatrix} 0 & \frac{b_4\beta\Lambda}{b_4(\mu+\rho)-\delta\rho} \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} b_1 & 0 \\ -\varepsilon & b_2 \end{bmatrix}.$$

The inverse of matrix V is

$$V^{-1} = \begin{bmatrix} \frac{1}{b_1} & 0 \\ \frac{\varepsilon}{b_1 b_2} & \frac{1}{b_2} \end{bmatrix}.$$

Hence, the next generation matrix is

$$K = FV^{-1} = \begin{bmatrix} \frac{b_4\beta\varepsilon\Lambda}{b_1 b_2(b_4(\mu+\rho)-\delta\rho)} & \frac{b_4\beta\Lambda}{b_2(b_4(\mu+\rho)-\delta\rho)} \\ 0 & 0 \end{bmatrix}.$$

The value of R_0 is obtained from the dominant eigenvalue of matrix K, which is

$$R_0 = \frac{b_4\beta\varepsilon\Lambda}{b_1 b_2(b_4(\mu+\rho)-\delta\rho)}.$$

Describes the combined effects of the infection rate (β), progression rate from exposed to infectious (ε), recruitment rate (Λ), and other demographic parameters on disease transmission. The basic reproduction number (R_0) is the average number of new cases generated by a single infected individual when the entire population is still susceptible. If $R_0 < 1$, each infectious individual produces less than one new infection on average, indicating that tuberculosis will eventually die out and the disease-free equilibrium E_0 is stable. Conversely, if $R_0 > 1$, the disease can invade and persist within the population, leading to the existence of the endemic equilibrium E_1.

3.1.4. Stability Analysis of the Non-Endemic Equilibrium Point

To analyze the stability of the equilibrium point, the Jacobian matrix is used to determine the eigenvalues of the system. Based on Equations (13)–(17), the general form of the Jacobian matrix is obtained as

$$J(s,e,i,t_h,r) = \begin{bmatrix} -(\beta i+\mu+\rho) & 0 & -\beta s & 0 & \delta \\ \beta i & -b_1 & \beta s & 0 & 0 \\ 0 & \varepsilon & -b_2 & 0 & 0 \\ 0 & \psi & \omega & -b_3 & 0 \\ \rho & 0 & 0 & \theta & -b_4 \end{bmatrix}. \tag{23}$$

By substituting the non-endemic equilibrium point $E_0 = \left(\frac{b_4\Lambda}{b_4\mu + b_4\rho - \delta\rho}, 0, 0, 0, \frac{\rho\Lambda}{b_4\mu + b_4\rho - \delta\rho}\right)$ into (23), the Jacobian matrix at the point E_0 is obtained as

$$J(E_0) = \begin{bmatrix} -\mu-\rho & 0 & -\frac{\beta b_4\Lambda}{b_4\mu + b_4\rho - \delta\rho} & 0 & \delta \\ 0 & -b_1 & \frac{\beta b_4\Lambda}{b_4\mu + b_4\rho - \delta\rho} & 0 & 0 \\ 0 & \varepsilon & -b_2 & 0 & 0 \\ 0 & \psi & \omega & -b_3 & 0 \\ \rho & 0 & 0 & \theta & -b_4 \end{bmatrix}.$$

The characteristic equation is

$$|J(E_0) - \lambda I| = (\lambda + \mu + \rho)(\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4) = 0.$$

Thus, one eigenvalue is $\lambda_1 = -\mu - \rho$, and the remaining eigenvalues satisfy

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

with

$$\begin{aligned} a_1 &= b_1 + b_2 + b_3 + b_4 > 0, \\ a_2 &= b_1b_2(1 - R_0) + b_1b_3 + b_1b_4 + b_2b_3 + b_2b_4 + b_3b_4 > 0, \quad \text{for } R_0 < 1, \\ a_3 &= b_1b_2(b_3 + b_4)(1 - R_0) + b_1b_3b_4 + b_2b_3b_4 > 0, \quad \text{for } R_0 < 1, \\ a_4 &= (1 - R_0)b_1b_2b_3b_4 > 0, \quad \text{for } R_0 < 1. \end{aligned}$$

Therefore, λ_3 and λ_4 will be negative if $R_0 < 1$. Based on the Routh-Hurwitz criteria, the characteristic roots of equation above have negative real parts, the endemic equilibrium point is locally asymptotically stable if $R_0 < 1$ and fulfill specific conditions.

3.1.5. Stability Analysis of the Endemic Equilibrium Point

By substituting the endemic equilibrium point into the Jacobian matrix in Equation (10), we obtain

$$J(E_1) = \begin{bmatrix} -(\beta\left(\frac{d_3}{\beta}\right) + \mu + \rho) & 0 & -\beta\left(\frac{d_1}{\beta\varepsilon}\right) & 0 & \delta \\ \beta\left(\frac{d_3}{\beta}\right) & -b_1 & \beta\left(\frac{d_1}{\beta\varepsilon}\right) & 0 & 0 \\ 0 & \varepsilon & -b_2 & 0 & 0 \\ 0 & \psi & \omega & -b_3 & 0 \\ \rho & 0 & 0 & \theta & -b_4 \end{bmatrix},$$

with $d_1 = b_1b_2$, $d_3 = \dfrac{(b_1b_2b_3)(b_4\mu + b_4\rho - \delta\rho)(R_0 - 1)}{(A - B)}$, $A = b_1b_2b_3b_4$, and $B = \delta\theta(\psi b_2 + \omega\varepsilon)$

Based on the Jacobian matrix, the characteristic equation can be determined as

$$|J(E_1) - \lambda I| = \lambda^5 + c_1\lambda^4 + c_2\lambda^3 + c_3\lambda^2 + c_4\lambda + c_5 = 0$$

with

$c_1 = C + (d_3 + \mu + \rho)$

$$= \left(\frac{1}{A-B}\right)\Big((C+\mu+\rho)(A-B) + (A(\mu+\rho) - b_1b_2b_3\delta\rho)(R_0-1)\Big) > 0, \text{ for } R_0 > 1,$$

$$c_2 = D + C(d_3+\mu+\rho) - \delta\rho,$$

$$= \left(\frac{1}{A-B}\right)\Big((D + C(\mu+\rho) - \delta\rho)(A-B) + C(A(\mu+\rho) - b_1b_2b_3\delta\rho)(R_0-1)\Big) > 0, \text{ for } R_0 > 1,$$

$$c_3 = E + D(d_3+\mu+\rho) + d_3(b_1b_2) - \delta\rho(b_1+b_2+b_3),$$

$$= \left(\frac{1}{A-B}\right)\Big(\big(E + D(\mu+\rho) - \delta\rho(b_1+b_2+b_3)\big)(A-B) + (D+b_1b_2)(A(\mu+\rho) - b_1b_2b_3\delta\rho)(R_0-1)\Big) > 0, \text{ for } R_0 > 1.$$

$$c_4 = E(d_3+\mu+\rho) + d_3(b_1b_2b_3 + b_1b_2b_4) - \delta\rho(b_1b_3 + b_2b_4),$$

$$= \left(\frac{1}{A-B}\right)\Big((E(\mu+\rho) - \delta\rho(b_1b_3 + b_2b_3))\,(A-B) + (E + b_1b_2b_3 + b_1b_2b_4)(A(\mu+\rho) - b_1b_2b_3\delta\rho)(R_0-1)\Big) > 0, \text{ for } R_0 > 1,$$

$$c_5 = Ad_3 - Bd_3,$$

$$= A(\mu+\rho - b_1b_2b_3\delta\rho)(R_0-1) > 0, \text{ for } R_0 > 1.$$

and $C = (b_1+b_2+b_3+b_4)$, $D = (b_1b_3 + b_1b_4 + b_2b_3 + b_2b_4 + b_3b_4)$, and $E = (b_1b_3b_4 + b_2b_3b_4)$.

From the characteristic equation above, it can be seen that the values of λ_2, λ_3, λ_4, and λ_5 will be negative if $R_0 > 1$ and other conditions are met. Based on the Routh-Hurwitz criteria eigenvalues λ_1, λ_2, λ_3, λ_4, and λ_5 are negative, the endemic equilibrium point is locally asymptotically stable under the condition $R_0 > 1$.

3.1.6. Simulation of the Non-Endemic Equilibrium Point Analysis

In this section, we do the numerical simulations. The equilibrium analysis simulation in the non-endemic state was performed using the parameter values and initial values for each population as shown in Table 2 and Table 3.

Table 2. Parameter Values in the Non-Endemic Point

Parameter	Description	Value	Unit
Λ	Birth rate	0.0015	1/month
μ	Natural death rate	0.0015	1/month
ρ	Vaccination rate	0.0001	1/month
β	TB infection rate	0.011	1/month
ω	Treatment rate in infected population class	0.07	1/month
θ	Recovery rate through treatment process	0.036	1/month
ε	Incubation rate	0.99	1/month
ψ	Treatment rate in exposed population class	0.03	1/month
δ	Immunity loss rate	0.004	1/month

Table 3. Initial Values of Each Population

Population at $t = 0$ (Proportion)	Value
s	0.5
e	0.3
i	0.15
t_h	0.05
r	0

Next, the simulation in the non-endemic state with equal values of Λ and μ is shown in Figure 3 below.

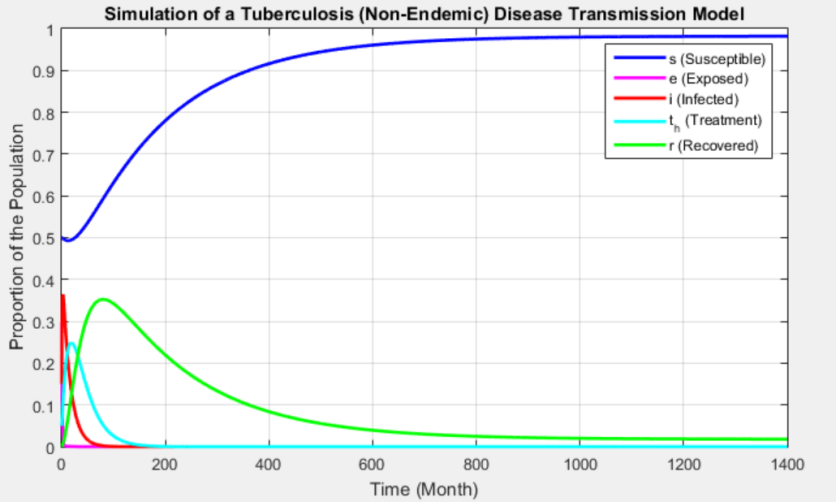

Figure 3. Simulation graph of the non-endemic equilibrium point

Figure 3 illustrates the dynamics of each compartment over time. The susceptible population initially declines until approximately the 20th month, reaching a value of 0.492739, before increasing and stabilizing at 0.982134. The exposed and infected populations decrease and eventually converge to zero after showing a brief increase in the early months. The treatment population rises until the 18th month, then declines with some fluctuations before converging to zero. Meanwhile, the recovered population increases initially, then gradually decreases and stabilizes at 0.017866.

These simulation results confirm that the non-endemic equilibrium point $E_0 = (s, e, i, t_h, r) = (0.982134, 0, 0, 0, 0.017866)$ is locally asymptotically stable. This implies that when the system starts from an initial condition sufficiently close to E_0, the deviations of all state variables converge to this equilibrium point as time progresses. In other words, small perturbations around E_0 decay over time, causing the system to return to equilibrium. This finding is consistent with the analytical condition $R_0 < 1$, indicating that tuberculosis tends to die out within the population.

3.1.7. Simulation of the Endemic Equilibrium Point Analysis

Numerical simulation in the endemic state is carried out using the initial values in Table 3 and parameter values in Table 4 below. We give the value of incubation in higher number because the transition rate is a mathematical quantity and does not represent the biological latent period, so a high value of ε remains consistent with the structure of a simplified TB model.

Table 4. Parameter Values in the Endemic State

Parameter	Description	Value	Unit
Λ	Birth rate	0.0015	1/month
μ	Natural death rate	0.0015	1/month
ρ	Vaccination rate	0.0001	1/month
β	TB infection rate	0.22	1/month
ω	Treatment rate in infected population class	0.07	1/month
θ	Recovery rate through treatment process	0.036	1/month
ε	Incubation rate	0.99	1/month
ψ	Treatment rate in exposed population class	0.03	1/month
δ	Immunity loss rate	0.004	1/month

Next, the simulation in the endemic point with equal values of Λ and μ is shown in Figure 4 below.

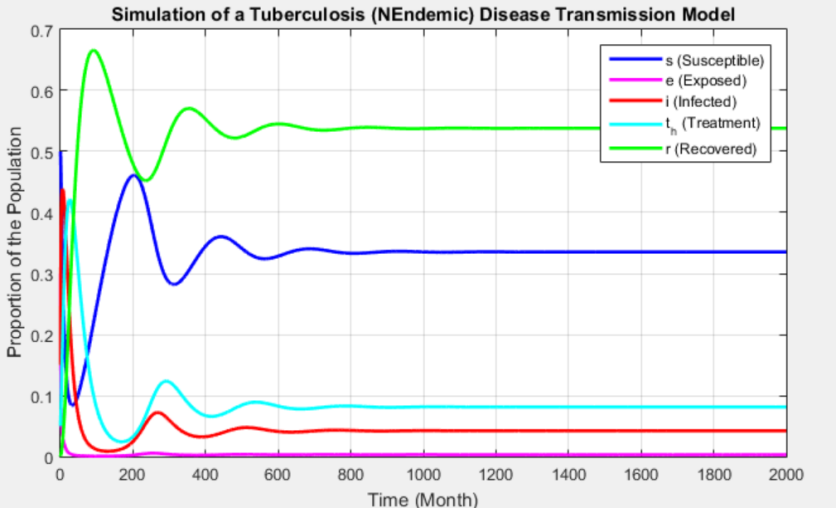

Figure 4. Simulation graph of the endemic equilibrium point

Figure 4 shows that the susceptible population initially declines, then increases, and eventually decreases again, converging to 0.335341, which indicates ongoing disease transmission. The exposed population increases at first, then decreases, rises again, and stabilizes at 0.003050. Similarly, the infected population grows initially, drops significantly, fluctuates, and ultimately converges to 0.042235. The treatment population also follows a similar pattern—initial increase, sharp decrease, then fluctuations before stabilizing at 0.081278. The recovered population increases early on, then gradually declines and converges to 0.538096.

These simulation results indicate that the endemic equilibrium point $E_1 = (s^*, e^*, i^*, t_h^*, r^*) = (0.335341, 0.003050, 0.042235, 0.081278, 0.538096)$ is locally asymptotically stable. This supports the analytical finding that when $R_0 > 1$, the endemic equilibrium point remains stable, and tuberculosis persists within the population.

3.2. Optimal Control Analysis

The model to be discussed in the optimal control problem is the model defined in Equations (13) to (17). Furthermore, the model is extended by introducing control variables in the form of socialization

(u_1) and therapy (u_2). Thus, the mathematical model of Tuberculosis disease transmission involving control through scialization (u_1) and therapy (u_2) is defined as follows :

$$\frac{ds}{dt} = \Lambda - \beta(1-u_1)si - \mu s - \rho s + \delta r, \tag{24}$$
$$\frac{de}{dt} = \beta(1-u_1)si - (\mu + \varepsilon + \psi)e, \tag{25}$$
$$\frac{di}{dt} = \varepsilon e - (\mu + \omega + u_2)i, \tag{26}$$
$$\frac{dt_h}{dt} = (\omega + u_2)i - (\mu + \theta)t_h + \psi e, \tag{27}$$
$$\frac{dr}{dt} = \theta t_h - (\mu + \delta)r + \rho s. \tag{28}$$

u_1 and u_2 are control variables with $0 \leq u_i(t) \leq 1, \forall i = 1, 2$.

In this study, the main objective is to minimize the number of infected individuals as well as reduce the costs arising from the implementation of socialzation (u_1) and therapy (u_2), hence the objective function can be formulated as follow

$$J = \min_{u_1,u_2} \int_{t_0}^{t_f} \left(Qi + \frac{1}{2}R_1u_1^2 + \frac{1}{2}R_2u_2^2 \right) dt$$

subject to the constraints :

$$\begin{aligned}
\frac{ds}{dt} &= \Lambda - \beta(1-u_1)si - \mu s - \rho s + \delta r,\\
\frac{de}{dt} &= \beta(1-u_1)si - (\mu + \varepsilon + \psi)e,\\
\frac{di}{dt} &= \varepsilon e - (\mu + \omega + u_2)i,\\
\frac{dt_h}{dt} &= (\omega + u_2)i - (\mu + \theta)t_h + \psi e,\\
\frac{dr}{dt} &= \theta t_h - (\mu + \delta)r + \rho s,\\
0 \leq& t \leq t_f,\\
0 \leq& u_1 \leq 1,\\
0 \leq& u_2 \leq 1.
\end{aligned}$$

where t_f is the final time, the coefficient Q represents the weight of loss or the level of concern toward the existence of infected individuals in the system, R_1 is the weight of socialzation costs, and R_2 is the weight of therapy costs. By solving the objective function, the optimal controls u_1 and u_2 will be obtained.

According to Pontryagin's Maximum Principle, the first step is to construct the Hamiltonian function as stated. The Hamiltonian function for the mathematical model of Tuberculosis disease transmission with controls in the form of socialzation and therapy is as follow

$$H(x, u, \lambda, t) = L(x, u, t) + \lambda^T(f(x, u, t))$$

namely

$$H = Qi + \frac{1}{2}R_1u_1^2 + \frac{1}{2}R_2u_2^2 + \lambda_1(\Lambda - \beta(1-u_1)si - \mu s - \rho s + \delta r) + \\ \lambda_2(\beta(1-u_1)si - (\mu+\varepsilon+\psi)e) + \lambda_3(\varepsilon e - (\mu+\omega+u_2)i) + \\ \lambda_4((\omega+u_2)i - (\mu+\theta)t_h + \psi e) + \lambda_5(\theta t_h - (\mu+\delta)r)$$

where $\lambda_i, \forall i = 1, \ldots, 5$ are the co-state variables. To obtain the optimal condition of $H(x, u, \lambda, t)$, the stationary condition of $H(x, u, \lambda, t)$ must be satisfied

$$\frac{\partial H}{\partial u} = \left(\frac{\partial H}{\partial u_1}, \frac{\partial H}{\partial u_2}\right) = 0$$

with $\dfrac{\partial H}{\partial u_1} = R_1u_1 + \lambda_1\beta si - \lambda_2\beta si = 0$ and $\dfrac{\partial H}{\partial u_2} = R_2u_2 - \lambda_3 i + \lambda_4 i = 0$. Thus, we obtain

$$u_1 = \frac{(\lambda_2 - \lambda_1)\beta si}{R_1},$$
$$u_2 = \frac{(\lambda_3 - \lambda_4)i}{R_2}.$$

Given the boundary condition for u_1 and u_2 namely $0 \le u_1 \le 1$ and $0 \le u_2 \le 1$, the optimal controls u_1^* and u_2^* are expressed as
For control $u_1(t)$

$$u_1^*(t) = \begin{cases} 0, & \text{if } \dfrac{(\lambda_2 - \lambda_1)\beta si}{R_1} < 0 \\ \dfrac{(\lambda_2 - \lambda_1)\beta si}{R_1}, & \text{if } 0 \le \dfrac{(\lambda_2 - \lambda_1)\beta si}{R_1} \le 1 \\ 1, & \text{if } \dfrac{(\lambda_2 - \lambda_1)\beta si}{R_1} > 1 \end{cases}$$

or

$$u_1^*(t) = \min\left\{\max\left(0, \frac{(\lambda_2 - \lambda_1)\beta si}{R_1}\right), 1\right\}.$$

For control $u_2(t)$

$$u_2^*(t) = \begin{cases} 0, & \text{if } \dfrac{(\lambda_3 - \lambda_4)i}{R_2} < 0 \\ \dfrac{(\lambda_3 - \lambda_4)i}{R_2}, & \text{if } 0 \le \dfrac{(\lambda_3 - \lambda_4)i}{R_2} \le 1 \\ 1, & \text{if } \dfrac{(\lambda_3 - \lambda_4)i}{R_2} > 1 \end{cases}$$

or

$$u_2^*(t) = \min\left\{\max\left(0, \frac{(\lambda_3 - \lambda_4)i}{R_2}\right), 1\right\}.$$

In the form of controls u_1^* and u_2^*, the state variables s and i, and the co-state variables $\lambda_1, \lambda_2, \lambda_3$ and λ_4 are still present. Therefore, the state and co-state equations must be solved to obtain those variables. Let $x = (s, e, i, t_h, r)$, then the state equations are given by

$$\frac{\partial H}{\partial \lambda} = \left(\frac{\partial H}{\partial \lambda_1}, \frac{\partial H}{\partial \lambda_2}, \frac{\partial H}{\partial \lambda_3}, \frac{\partial H}{\partial \lambda_4}, \frac{\partial H}{\partial \lambda_5}\right).$$

Thus, we obtain

$$\dot{x} = \begin{pmatrix} \frac{\partial H}{\partial \lambda_1} \\ \frac{\partial H}{\partial \lambda_2} \\ \frac{\partial H}{\partial \lambda_3} \\ \frac{\partial H}{\partial \lambda_4} \\ \frac{\partial H}{\partial \lambda_5} \end{pmatrix} = \begin{pmatrix} \Lambda - \beta(1-u_1)si - \mu s - \rho s + \delta r \\ \beta(1-u_1)si - (\mu + \varepsilon + \psi)e \\ \varepsilon e - (\mu + \omega + u_2)i \\ (\omega + u_2)i - (\mu + \theta)t_h + \psi e \\ \theta t_h - (\mu + \delta)r + \rho s \end{pmatrix}.$$

The equation $\dot{x}$ represents the system's state dynamics, describing the rate of change of the compartments (s, e, i, t_h, r) over time. It illustrates how the population transitions between compartments due to infection, recovery, and the effects of control variables u_1 and u_2.

The co-state equations can be solved from

$$\dot{\lambda} = -\frac{\partial H}{\partial x} = -\left(\frac{\partial H}{\partial s}, \frac{\partial H}{\partial e}, \frac{\partial H}{\partial i}, \frac{\partial H}{\partial t_h}, \frac{\partial H}{\partial r}\right).$$

Thus, we obtain

$$\begin{pmatrix} \dot{\lambda_1} \\ \dot{\lambda_2} \\ \dot{\lambda_3} \\ \dot{\lambda_4} \\ \dot{\lambda_5} \end{pmatrix} = \begin{pmatrix} \lambda_1(\beta(1-u_1)i + \mu + \rho) - \lambda_2\beta(1-u_1)i - \lambda_5\rho \\ \lambda_2(\mu + \varepsilon + \psi) - \lambda_3\varepsilon - \lambda_4\psi \\ -Q + \lambda_1\beta(1-u_1)s - \lambda_2\beta(1-u_1)s + \lambda_3(\mu + \omega + u_2) - \lambda_4(\omega + u_2) \\ \lambda_4(\mu + \theta) - \lambda_5\theta \\ -\lambda_1\delta + \lambda_5(\mu + \delta) \end{pmatrix}$$

with transversality condition

$$\lambda_i(t_f) = 0, \quad \text{for } i = 1, 2, \ldots, 5.$$

The equation $\dot{\lambda} = (\dot{\lambda_1}, \dot{\lambda_2}, \dot{\lambda_3}, \dot{\lambda_4}, \dot{\lambda_5})$ represents the dynamics of the co-state variables, which indicate the sensitivity of the objective function to each state variable. These variables assess how small changes in the compartments s, e, i, θ_h, r affect the optimization goal and play a crucial role in determining the optimal control strategy.

From the state and co-state equations, the values of the state and co-state variables will be obtained, which are then substituted into the controls u_1^* and u_2^*. Then, the expressions for u_1^* and u_2^* are substituted into the state equations to obtain the optimal solution form. The model under optimal control u_1^* and u_2^* is as follows.

$$\begin{aligned}
\dot{s} &= \Lambda - \beta\left(1 - \left(\min\left\{\max\left(0, \frac{(\lambda_2 - \lambda_1)\beta si}{R_1}\right), 1\right\}\right)\right)si - \mu s - \rho s + \delta r, \\
\dot{e} &= \beta\left(1 - \left(\min\left\{\max\left(0, \frac{(\lambda_2 - \lambda_1)\beta si}{R_1}\right), 1\right\}\right)\right)si - (\mu + \varepsilon + \psi)e, \\
\dot{i} &= \varepsilon e - \left(\mu + \omega + \left(\min\left\{\max\left(0, \frac{(\lambda_3 - \lambda_4)i}{R_2}\right), 1\right\}\right)\right)i, \\
\dot{t_h} &= \left(\omega + \left(\min\left\{\max\left(0, \frac{(\lambda_3 - \lambda_4)i}{R_1}\right), 1\right\}\right)\right)i - (\mu + \theta)t_h + \psi e, \\
\dot{r} &= \theta t_h - (\mu + \delta)r + \rho s.
\end{aligned}$$

To obtain $\dot{s}, \dot{e}, \dot{i}, \dot{t_h}$, and $\dot{r}$ from the optimal form u^*, the nonlinear state and co-state equations must be solved numerically.

3.2.1. Numerical simulation

The best solution of the system controlled by u_1 and u_2 will be described through numerical simulation. In addition, this simulation will also present the behavior between the system solution with optimal control and without control. To calculate the solutions $x(t)$ and $\lambda(t)$, the *forward-backward* method is used. The process of determining these values is divided into the following stages [14].

1. The time interval $[t_0, t_f]$ is divided into points $t_0 = b_1, b_2, \ldots, b_n = t_f$.
2. The control function u_i is defined at discrete points as $u_{ij} = u_i(b_j), \quad i = 1, 2, \ j = 1, \ldots, n$.
3. The control value u_i is updated using a convex combination between the old control value and the new control value, namely
$$u = \frac{u_{\text{old}} + u_{\text{new}}}{2}$$
where u_{new} is obtained from the optimality condition $\frac{\partial H}{\partial u} = 0$ with
$$u_1^*(t) = \min\left\{\max\left(0, \frac{(\lambda_2 - \lambda_1)\beta si}{R_1}\right), 1\right\},$$
$$u_2^*(t) = \min\left\{\max\left(0, \frac{(\lambda_3 - \lambda_4)i}{R_2}\right), 1\right\}.$$
4. The convergence of the optimal solution u is tested with the following convergence condition:
$$\frac{\|u - u_{\text{old}}\|}{\|u\|} \leq 10^{-4} \text{ or } 10^{-4}\|u\| - \|u - u_{\text{old}}\| \geq 0.$$

The simulation is carried out in the time range $t \in [0, 50]$ months, or the initial time $t_0 = 0$ to the final time $t_f = 50$ months using the initial conditions as listed in Table 3 and parameter values in the endemic state. The weights used in the objective function calculation are set as $Q = 1.5$, $R_1 = 0.1$, and $R_2 = 0.2$. The simulation results of the model without control and with control can be seen in Figures 5 to 9 below.

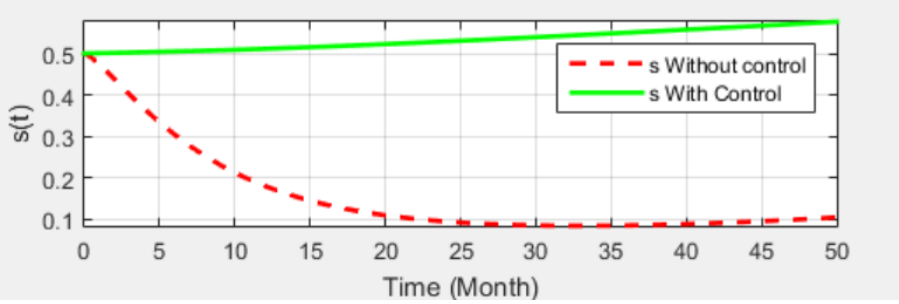

Figure 5. Graph of TB disease spread model in the susceptible population

Figure 5 illustrates the dynamics of the susceptible population (s) to tuberculosis infection over a 50-month period. Without control, the proportion of susceptible individuals declines exponentially from 0.5 to 0.1 during the first 20 months, reflecting active transmission in the absence of preventive measures. Under the application of optimal control, the susceptible proportion increases from 0.5 to 0.576621, indicating that the socialzation control (u_1) effectively retains more individuals in the susceptible compartment by reducing exposure risk.

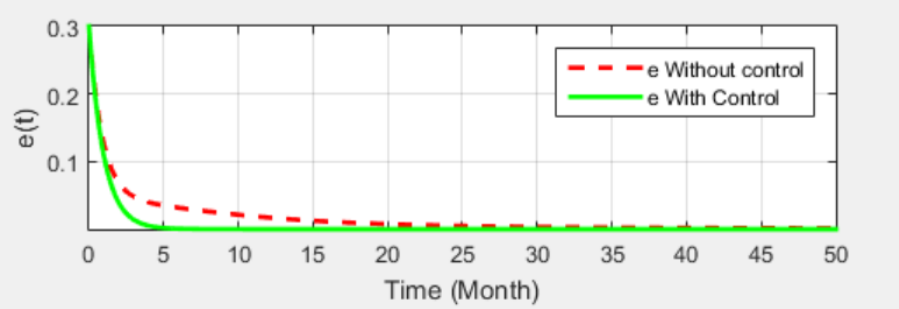

Figure 6. Graph of TB disease spread model in the exposed population

Figure 6 presents the dynamics of the exposed population (e) over a 50-month period. With the implementation of control, the proportion of exposed individuals decreases rapidly and reaches near-zero levels more quickly than in the uncontrolled scenario. For instance, at month 3, the proportion under control is 0.013900, compared to 0.046924 without control. This demonstrates that the socialzation control effectively disrupts the latent transmission chain.

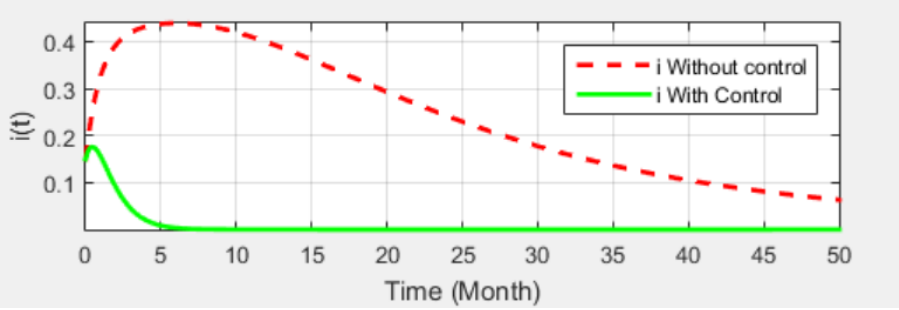

Figure 7. Graph of TB disease spread model in the infected population

Figure 7 illustrates the dynamics of the infected population (i) over a 50-month period. In the absence of control, the proportion of infected individuals rises sharply, peaking at 0.437191 around the fifth month, and then gradually declines. In contrast, under control, the infection peaks earlier—around 0.5 months—at a lower value of 0.175807, followed by a significant decrease. This demonstrates that the implementation of treatment control (u_2) effectively reduces the infected population more rapidly and limits the emergence of new cases.

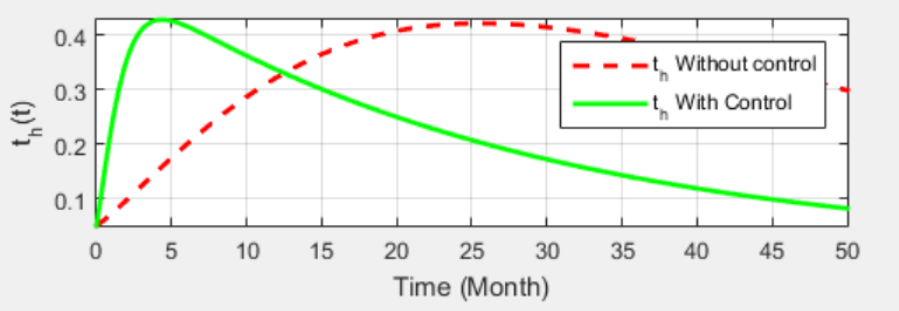

Figure 8. Graph of TB disease spread model in the treatment population

Figure 8 displays the dynamics of the treatment compartment (t_h) over a 50-month period. In the uncontrolled scenario, the proportion of individuals receiving treatment gradually increases, peaking at 0.421408 around the 25th month, and then slowly declines. Under control, the treatment proportion

rises more rapidly, reaching a peak of 0.425679 as early as the fifth month. This indicates that the implementation of control facilitates a faster transition of infected individuals into treatment, reflecting more effective and timely utilization of healthcare services.

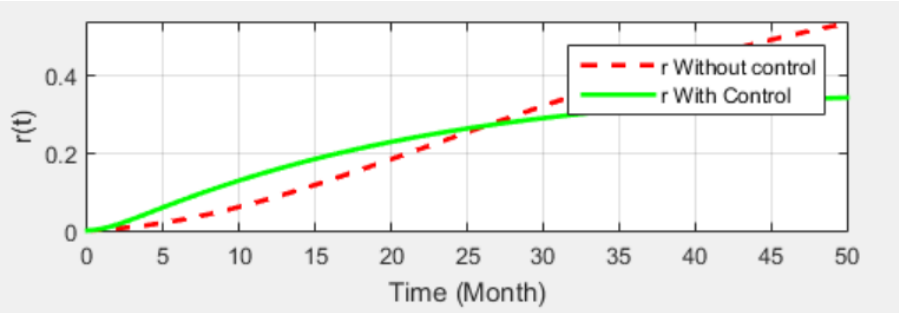

Figure 9. Graph of TB disease spread model in the recovered population

Figure 9 shows the dynamics of the recovered population (r) over a 50-month period. Without control, the proportion of recovered individuals increases at a slower rate compared to the controlled scenario. For example, at month 15, the recovered proportion is 0.116909 without control, whereas it reaches 0.184421 with control. This highlights the significant role of socialzation and therapy controls in accelerating the recovery process.

Next, the graph of the control function over the simulation time range is presented in Figure 10 below.

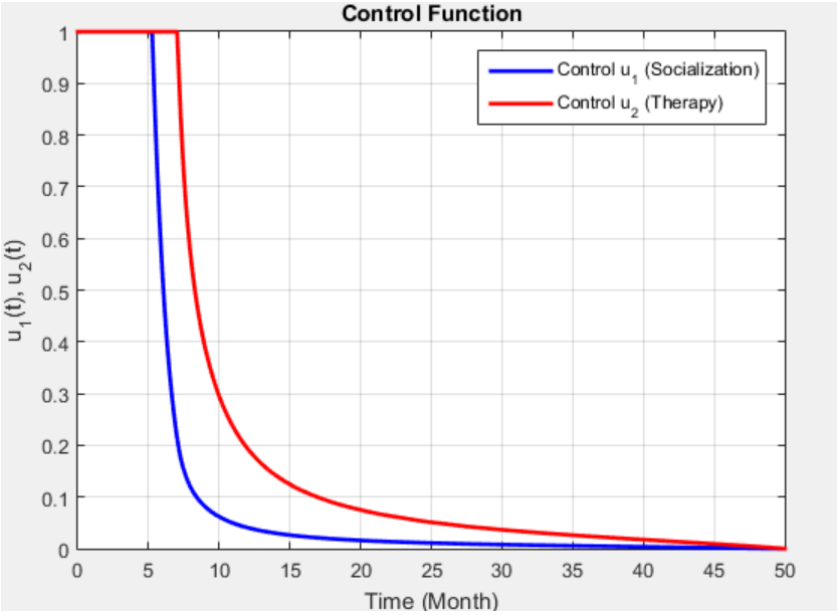

Figure 10. Graph of control functions in Tuberculosis transmision

Figure 10 presents the optimal control profiles for socialization (u_1) and therapy (u_2) in managing the spread of tuberculosis. Both controls begin at high intensity and gradually decline over time, reflecting an aggressive initial intervention strategy. The therapy control (u_2) consistently maintains higher values than socialization (u_1), indicating its dominant role in disease mitigation. This pattern suggests that control efforts are most effective during the early phase of the epidemic and taper off as the number of infections decreases.

4. Conclusions

This study presented a nonlinear SEITRS model to describe the transmission dynamics of tuberculosis (TB), dividing the total population into five compartments: susceptible, exposed, infected, under treatment, and recovered. The model incorporates key epidemiological processes such as population inflow, reinfection, treatment, and relapse, allowing it to capture realistic TB dynamics both with and without control interventions. Analytical results showed that the disease-free equilibrium is locally asymptotically stable when the basic reproduction number satisfies $R_0 < 1$ under specific parameter conditions, implying that TB will die out in the population. Conversely, the endemic equilibrium is stable when $R_0 > 1$, indicating the persistence of infection. An optimal control problem was formulated by introducing two control variables, namely socialization and therapy, which were derived using Pontryagin's Maximum Principle. Simulation results demonstrated that the simultaneous implementation of socialization and therapy controls significantly reduces the number of exposed and infected individuals, accelerates recovery, and decreases the overall disease burden compared to the uncontrolled scenario. Future research may extend this work by considering external factors such as spatial mobility, heterogeneity in population behavior, and early screening interventions to enhance the model's applicability in real-world public health planning.

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