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Modelling the Impact of Control Measures on Tuberculosis Transmission

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Abstract
Tuberculosis (TB) is a global health pandemic which spreads through the air and is caused by *Mycobacterium tuberculosis* (MTB) which is a major contributor of illness and death worldwide. The ease of Tuberculosis transmission in closed environments makes it exposure becomes difficult to prevent, which do result in cases of Infection with and without symptoms. Moreso, Inadequate treatment of Tuberculosis cases often leads to antibiotic resistance, resulting in relapses even after apparent recovery. This study introduces a modified (S, E, I_1, I_2, T, R) model which analyze Tuberculosis transmission and optimal control strategies. Four key intervention strategies were examined: awareness-based interventions, tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment. Qualitative analysis and optimal control analysis were conducted to validate the model. The model's system of differential equations was solved numerically using finite difference methods and simulated in MATLAB. Optimal control analysis using Pontryagin's Maximum Principle demonstrated how gradual control strategies reduce infections and flatten the transmission curve. Results from the study showed that combination of the strategies are most effective for controlling Tuberculosis. These findings can guide policymakers in developing comprehensive and evidence-based decision to combat Tuberculosis spread.

Keywords: Mathematical Model, Tuberculosis cases, Awareness Based Intervention, Tuberculosis examination, Essential Treatment.

Introduction

Tuberculosis is an infectious disease which is caused by the organism mycobacterium tuberculosis (MTB) that affects both humans and animals. The tuberculosis bacillus bacteria currently infect one-third of the world's population, according to the World Health Organization (WHO, 2019). It is one of the most prevalent infectious illnesses that can be contracted through close contact with infected people. In 2019, 87% of tuberculosis deaths took place in low and middle income nations with Asia accounting for 44% of projected cases and Africa for 24%. The Western Pacific area accounted for a substantial amount of (18%) cases, followed by the Eastern Mediterranean region (8%), Europe (3%) and the Americas (3%) each of which made a little contribution (Kuddus et al., 2021). According to Wu et al. (2020) the number of tuberculosis cases has been decreasing over the past 20 years with between 10,000 and 20,000 new cases reported per year and the case fatality rate in America ranges from 0.05 to 0.07. However, since the year 2000, over sixty-six million lives have been saved due to global efforts to combat tuberculosis (Inayaturohmat et al., 2022). Mycobacterium tuberculosis (MTB) typically affects the lungs of infected individuals (Wu et al., 2020). The bacteria get into the air when an infected person coughs, sneezes, shouts or spits (Malik et al., 2018). This tiny mycobacterium tuberculosis (MTB) may remain in the atmosphere for a long period and continue to move (Wu et al., 2020). This infection can be transmitted by inhaling air or saliva droplets released by an infected patient (Adeleke et al., 2025). The most common method that tuberculosis spreads is by direct contact with someone susceptible by an infected person. As a result, contact with an infected person can cause an individual to contact the disease, either with or without presenting symptoms. In actuality, not every tuberculosis patient will

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have symptoms at the same time and the incubation time for some individuals with tuberculosis ranges from one year to many years (Loyinmi, 2025; Wu et al., 2020). Once infected, a person may have the infection for many years and potentially for the rest of their lives. Infection with tuberculosis can cause either latent tuberculosis, in which the bacteria do not spread and the person is not infectious or active tuberculosis in which the bacteria are active and the person can infect others (Alfiniyah et al., 2024). Individuals who live close to people who have an active tuberculosis infection, those with weakened immune systems due to age or other health issues and those who reside in areas where the prevalence of tuberculosis infection is higher than usual are all at a heightened risk of contracting the disease. The Symptoms of tuberculosis illness varies on the part of the body the tuberculosis bacteria exist. A patient's medical history, current health situation and physical examination can all be used to determine if a certain symptom is indicative of tuberculosis. Tuberculosis may be diagnosed by several methods which include sputum analysis, skin tests and chest X-rays. When diagnosing active tuberculosis, further tests are performed such as detecting the causative organism which is mycobacterium tuberculosis in the sputum. In actuality, not every tuberculosis patient will have symptoms at the same time. Due to the lack of accurate tuberculosis tests many infections go undetected these days which has been making tuberculosis management more challenging (Wu et al.,2020). Early illness identification, patient follow-up and clinical care are the main obstacles to the elimination of tuberculosis according to (Bhadauria et al., 2023). The high cost of treating tuberculosis which places a significant burden on the general people is among the possible factor contributing to inadequate treatment. The course of treatment which often lasts for six months or more and sometimes for up to twenty-four months along with drug-resistant strains which proliferate if treatment is not completed has significantly been making treatment more challenging. According to the World Health Organization (WHO, 2019) significant advancements in the treatment and cure of tuberculosis have led to a consistent decline in incident cases and fatalities in recent years. According to (Wu et al., 2020) most active and latent tuberculosis can be successfully treated based on decades of technology and expertise. However, the course of remedies must run at least six months for the treatment of tuberculosis to be successful. Thus, it is essential to provide a comprehensive model for the spread of tuberculosis and identify critical factors that influence the disease's which will assist in determining the best strategies to be implemented to bring tuberculosis under control across the globe (Loyinmi, 2025; Bhadauria et al., 2023).

One way that Mathematics plays a crucial part in modeling a medical condition epidemic phenomenon is by using a deterministic model to understand disease's propagation (Loyinmi et al., 2023; Inayaturohmat et al., 2022). Over the years, mathematical models have been used to examine a variety of infectious illnesses among which are Nipah virus, measles, Lassa fever, influenza, chicken pox and rubella (Gbodogbe., 2025; Loyinmi et al., 2025; Adeleke et al., 2025; Loyinmi, 2024). So, for public health sectors to make well-informed policy choices, epidemiological models of disease transmission are a crucial resource for evaluating the possible effects of each innovative treatments. Due to ethical, logistical and practical considerations clinical trials would not be viable without the use of mathematical models to simulate various treatment and "what if" situations (Kuddus et al., 2021; Loyinmi et al., 2024). Optimal control is one of the ongoing control measures which has been consider to lower the number of tuberculosis patients. According to (Anisa'Maulina & Imron, 2024; Loyinmi et al., 2025; Gbodogbe, 2025) optimal control is a model that is required to help make judgments about reaching a goal while simultaneously minimizing or maximizing the number of system performance variables. According to Kim, S et al., (2020) the optimal control theory applied to tuberculosis models is of interest to modelers because it offers public health professionals' useful information for decision and policy-making.

However, there are variety of models have been formulated, mathematically analyzed and applied to numerous infectious diseases (Loyinmi & Ijaola, 2024). The study by Bhadauria et al. (2023) presents a five-dimensional mathematical model to analyze tuberculosis (TB) dynamics in India. It categorizes TB cases into drug-sensitive (DS), multi-drug-resistant (MDR), and isolated classes. The model includes calculations of the reproduction number, equilibrium points, and stability analysis. Numerical simulations project TB trends from 2018 to 2035, aiming for possible eradication by 2035. The findings emphasize that high treatment success and effective isolation of MDR cases are crucial for eradication. The study's projections rely heavily on optimistic assumptions particularly achieving a 95% treatment success rate and isolating 50% of MDR-TB cases which may not be feasible in real-world settings due to healthcare and operational constraints. In the research conducted by (Ogbaji et al., 2019) a mathematical model was proposed for vaccination and treatment strategy to eradicate tuberculosis with absent of emigration effect. In which an existing model was modified by incorporating the immigrants effect, efficacy of vaccination, treatment and new babies were considered 100% vaccinated. The existence and uniqueness of solution

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of the modified model was carried out. Moreso, the stability analysis of the disease-free equilibrium shows that the disease-free equilibrium (DFE) is locally asymptotically stable. The effective reproductive number(R_e) was computed under different conditions. In the case where there is treatment and vaccination effective reproductive number(R_e) is 0.2527. The results show that mycobacterium tuberculosis can be eradicated if mass vaccination and treatment actions are properly initiated and enforced. (Alfiniyah et al., 2020) developed a mathematical model that captured the dynamics of Tuberculosis transmission specifically among smokers, incorporating the effects of case detection. Their innovative approach lied in the integration of smoking behavior as a key factor in tuberculosis transmission dynamics, which has been underexplored in previous models. They analyzed the existence and stability of the tuberculosis model equilibrium based on the basic reproduction number. Additionally, parameter sensitivity analysis was conducted to identify the most influential factors in the spread of the disease. Furthermore, their study investigated the effectiveness of various control strategies, including social distancing for smokers, tuberculosis screening in high-risk populations and tuberculosis treatment in low-income communities. By employing the pontryagin maximum principle, they solved the optimal control problems to determine the most effective combination of interventions. Simulation results demonstrated that a targeted combination of control measures can effectively reduce the number of tuberculosis infected individuals.

This study will consider using a compartmental modeling approach to study the transmission dynamics of a modified (S, E, I_1, I_2, T, R) tuberculosis model at two levels of infection. This study will also discuss how mathematical modeling can be used in determining the impacts of optimal control strategies such as awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment on the spread of the disease. This vaccine's efficacy in the treatment of the disease varies and it still does not offer complete protection against the illness, particularly for people with pulmonary tuberculosis. Therefore, it becomes essential to create more effective strategies which will be a more effective solution is also crucial to limiting the spread of tuberculosis. Therefore, the controls strategies use in this study are variables whose value are determined by using Pontryagin's Minimum Principle in optimal control theory. Stability and sensitivity analyses of the mathematical model of tuberculosis transmission were also examined in this study. The model is numerically simulated using the fourth-order Runge-kutta in MATLAB, and its numerical solution is determined using the finite difference method.

Materials and Methods

To understand the transmission dynamics tuberculosis this study utilizes a proposed (S, E, I_1, I_2, T, R) model. This model aims to implore multiple compartments defined with Six (6) compartmental state variables and parameters for the population of humans with an outbreak of tuberculosis. First, it starts with the recruitment rate (Λ) of people into the susceptible class once a contact is made between an infected person with symptoms (I_1) or an infected person without symptoms (I_2). The patients go into an exposed class (E) where a fraction of humans exposed become infected with symptoms (I_1) while the second fraction of humans exposed becomes infected without symptoms (I_2) as the third fraction of humans exposed recover due to their immune systems. Thereafter, humans who are infected with symptoms (I_1) and those who are infected without symptoms (I_2) proceed to the treatment class (T). Once humans have been considered for treatment, they then proceed to recovered class (R). At this stage, due to the loss of immunity and the possibility of reinfection, an individual may be susceptible to contacting tuberculosis. However, the transmission dynamics of the model are primarily been determined by the human recruitment rate which is a key parameter that affects population growth over time: The disease progression is determined by the human-to-human contact rate (β) between humans in the susceptible class and infected humans with or without symptoms. An exposed individual can become infected with symptoms at a rate $(a\gamma)$ and also become infected without symptoms at a rate $(b\gamma)$ as well becomes recover at a rate $\gamma(1-a-b)$. An infected individual with symptoms can be treated at a rate (r) while an infected individual without symptoms will become treated at a rate (q). Once treatment has been completed a patient will proceed to recovery class at a rate(η). Due to some mitigating factors, a patient who has recovered may become susceptible at a rate (ρ). To further represent the effect of tuberculosis on human population dynamics, the model includes the mortality rate (μ) of human and disease-induce death rate (δ) of human who die of tuberculosis. However, through the integration of each state's variables and parameters. The mathematical model presents a thorough framework for the analysis and simulation of tuberculosis transmission dynamics, which yields important information for strategies related to optimal controls.

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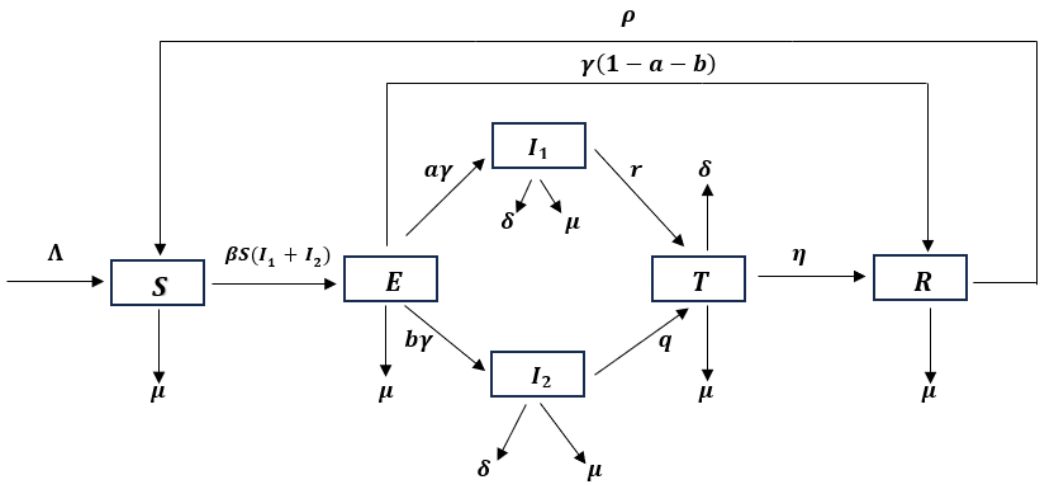

Figure1: Tuberculosis Schematic Diagram

Table 1: Description of each compartment

Compartments	Descriptions
S	Susceptible Human Class
E	Exposed Human Class
I_1	Infected Human with symptoms Class
I_2	Infected Human without symptoms Class
T	Treated Human Class
R	Recovered Class

Table 2: Descriptions of the parameters

Parameters	Descriptions
Λ	Humans recruitment rate
μ	Humans natural death rate
β	Humans to humans contact rate
$a\gamma$	Exposed human class to infected human class with symptoms rate
$b\gamma$	Exposed human class to infected human class without symptoms rate
$\gamma(1-a-b)$	Exposed human class to recovered human class rate
δ	Disease induced death rate
r	Rate of Infected human class with symptoms to Treated Humans class rate
q	Rate of Infected human class without symptoms to Treated human class
η	Human recovery rate after treatments
ρ	Relapse rate from Recovered humans class to susceptible humans class

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The Model Systems of Equation

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \rho R - (\beta(I_1 + I_2) + \mu)S \\
\frac{dE}{dt} &= \beta S(I_1 + I_2) - (\gamma + \mu)E \\
\frac{dI_1}{dt} &= a\gamma E - (r + \delta + \mu)I_1 \\
\frac{dI_2}{dt} &= b\gamma E - (q + \delta + \mu)I_2 \\
\frac{dT}{dt} &= rI_1 + qI_2 - (\eta + \delta + \mu)T \\
\frac{dR}{dt} &= \gamma(1 - a - b)E + \eta T - (\rho + \mu)R
\end{aligned}\right\} \quad (1)$$

with the initial conditions:
$S(0) = S_0, E(0) = E_0, I_1(0) = I_{1_0}, I_2(0) = I_{2_0}, T(0) = T_0, R(0) = R_0.$
The total population of individuals can be expressed as follows: $S(t) + E(t) + I_1(t) + I_2(t) + T(t) + R(t) = N(t)$

Qualitative Analysis of the Model
Existence and Uniqueness of Solution
By using the Lipschitz condition from the systems of eqn. (1)

$$\left.\begin{aligned}
\boldsymbol{B_1} &= \Lambda + \rho R - (\beta(I_1 + I_2) + \mu)S \\
\boldsymbol{B_2} &= \beta S(I_1 + I_2) - (\gamma + \mu)E \\
\boldsymbol{B_3} &= a\gamma E - (r + \delta + \mu)I_1 \\
\boldsymbol{B_4} &= b\gamma E - (q + \delta + \mu)I_2 \\
\boldsymbol{B_5} &= rI_1 + qI_2 - (\eta + \delta + \mu)T \\
\boldsymbol{B_6} &= \gamma(1 - a - b)E + \eta T - (\rho + \mu)R
\end{aligned}\right\} \quad (\mathbf{2})$$

Theorem 1: If A is a region in $0 \leq x \leq M$, then the systems if ***eqn (2)*** is said to possess a unique solution if and only if $\frac{\partial B_i}{\partial h_j}$ are continuous and bounded in B for $\neq j$.

Proof

We need to establish the partial derivative (2) with respect to the state variables which yields:

$\left|\frac{\partial B_1}{\partial S}\right| = |\beta S(I_1 + I_2) + \mu| < \infty$; $\left|\frac{\partial B_1}{\partial E}\right| = |0| < \infty$; $\left|\frac{\partial B_1}{\partial I_1}\right| = |-\beta S| < \infty$;

$\left|\frac{\partial B_1}{\partial I_2}\right| = |-\beta S| < \infty$; $\left|\frac{\partial B_1}{\partial T}\right| = |0| < \infty$; $\left|\frac{\partial B_1}{\partial R}\right| = |\rho| < \infty$;

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$\left|\frac{\partial B_2}{\partial S}\right| = |\beta(I_1 + I_2)| < \infty$; $\left|\frac{\partial B_2}{\partial E}\right| = |-(\gamma + \mu)| < \infty$ $\left|\frac{\partial B_2}{\partial I_1}\right| = |\beta S| < \infty$;

$\left|\frac{\partial B_2}{\partial I_2}\right| = |\beta S| < \infty$; $\left|\frac{\partial B_2}{\partial T}\right| = |0| < \infty$; $\left|\frac{\partial B_2}{\partial R}\right| = |0| < \infty$;

$\left|\frac{\partial B_3}{\partial S}\right| = |0| < \infty$; $\left|\frac{\partial B_3}{\partial E}\right| = |a\gamma| < \infty$; $\left|\frac{\partial B_3}{\partial I_1}\right| = |-(r + \delta + \mu)| < \infty$;

$\left|\frac{\partial B_3}{\partial I_2}\right| = |0| < \infty$; $\left|\frac{\partial B_3}{\partial T}\right| = |0| < \infty$; $\left|\frac{\partial B_3}{\partial R}\right| = |0| < \infty$;

$\left|\frac{\partial B_4}{\partial S}\right| = |0| < \infty$; $\left|\frac{\partial B_4}{\partial E}\right| = |b\gamma| < \infty$; $\left|\frac{\partial B_4}{\partial I_1}\right| = |0| < \infty$;

$\left|\frac{\partial B_4}{\partial I_2}\right| = |-(q + \delta + \mu)| < \infty$; $\left|\frac{\partial B_4}{\partial T}\right| = |0| < \infty$; $\left|\frac{\partial B_4}{\partial R}\right| = |0| < \infty$;

$\left|\frac{\partial B_5}{\partial S}\right| = |0| < \infty$; $\left|\frac{\partial B_5}{\partial E}\right| = |0| < \infty$; $\left|\frac{\partial B_5}{\partial I_1}\right| = |r| < \infty$;

$\left|\frac{\partial B_5}{\partial I_2}\right| = |q| < \infty$; $\left|\frac{\partial B_5}{\partial T}\right| = |-(\delta + \mu + \eta)| < \infty$; $\left|\frac{\partial B_5}{\partial R}\right| = |0| < \infty$;

$\left|\frac{\partial B_6}{\partial S}\right| = |0| < \infty$; $\left|\frac{\partial B_6}{\partial E}\right| = |\gamma(1 - a - b)| < \infty$; $\left|\frac{\partial B_6}{\partial I_1}\right| = |0| < \infty$;

$\left|\frac{\partial B_6}{\partial I_2}\right| = |0| < \infty$; $\left|\frac{\partial B_6}{\partial T}\right| = |\eta| < \infty$; $\left|\frac{\partial B_6}{\partial R}\right| = |-(\rho + \mu)| < \infty$;

Positivity and Boundedness of the systems

The differential equation for the population:

$$N(t) = S(t) + E(t) + I_1(t) + I_2(t) + T(t) + R(t)$$

Is given by:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dT}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = \Lambda - \mu(S + E + I_1 + I_2 + T + R) - \delta(I_1 + I_2 + T)$$

$$= \Lambda - \mu N - \delta(I_1 + I_2 + T) \qquad (3)$$

Theorem 2:

Let (S, E, I_1, I_2, T, R) be the solution of the tuberculosis equation with the initial condition condition in a biologically feasible region. Where **Γ** is a non-negative invariant:

If $\boldsymbol{\Gamma} = (S, E, I_1, I_2, T, R)\, \boldsymbol{\epsilon}\, R_+^6 \;\therefore\; N \leq \frac{\Lambda}{\mu}$

By using the method of Integrating factor.

Recall from $eqn(3)$ that:

$$\frac{dN}{dt} = \Lambda - \mu N - \delta(I_1 + I_2 + T)$$

In a DFE (Disease Free Equilibrium), δ (disease induce death) = 0

So,

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$$\frac{dN}{dt} = \Lambda - \mu N$$

$$N'(\mathrm{t}) + \mu N = \Lambda$$

Multiply through by the integrating factor and integrate

$$\int (e^{\mu t} N'(\mathrm{t}) + \mu N e^{\mu t}) dt = \int (\Lambda e^{\mu t}) dt$$

$$N(t)e^{\mu t} = \frac{\Lambda e^{\mu t}}{\mu} + C$$

$$\lim_{t \to \infty} N(t) = \frac{\Lambda}{\mu} + Ce^{-\mu t}$$

$$\therefore \ N \leq \frac{\Lambda}{\mu} \tag{4}$$

This shows that the equation is well posed and the solutions of different component are non-negative at time (t).

Existence of tuberculosis equilibrium state

The existence of the tuberculosis equilibrium state refers to a specific condition in the mathematical model where tuberculosis is not present or has been eradicated.

$$S^{\circ} \neq 0, E^{\circ} = 0, {I_1}^{\circ} = 0, {I_2}^{\circ} = 0, T^{\circ} = 0, R^{\circ} = 0$$

Then;

$$\frac{dN}{dt} = \Lambda - \mu N - \delta(I_1 + I_2 + T) \tag{3}$$

$$\Lambda - \mu S^{\circ} = 0$$

These give

$$S^{\circ} = \frac{\Lambda}{\mu}$$

We have the DFE point to be

$$\left(S^0, E^0, {I_1}^0, {I_2}^0, T^0, R^0\right) = \left(\frac{\Lambda}{\mu}, 0{,}0, 0{,}0, 0\right) \tag{5}$$

Basic Reproduction Number

The basic reproduction number for the tuberculosis system of equation is obtained via the method of next generation matrix formulated in 1990, by Diekman and Heesterbeek.

Using $R_n = \rho(FV^{-1})$ the new infection term $\boldsymbol{F}$ and the transition terms $\boldsymbol{V}$ of the system of $\boldsymbol{eqn}\,(\mathbf{1})$ are respectively given as:

$$F = \begin{bmatrix} \beta S(I_1 + I_2) \\ 0 \\ 0 \end{bmatrix} \qquad V = \begin{bmatrix} -(\gamma + \mu)E \\ a\gamma E - (r + \delta + \mu)I_1 \\ b\gamma E - (r + \delta + \mu)I_2 \end{bmatrix}$$

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$$F = \begin{bmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I_1} & \frac{\partial f_1}{\partial I_2} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I_1} & \frac{\partial f_2}{\partial I_2} \\ \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I_1} & \frac{\partial f_3}{\partial I_2} \end{bmatrix} = \begin{bmatrix} 0 & \beta S & \beta S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} \frac{\partial v_1}{\partial E} & \frac{\partial v_1}{\partial I_1} & \frac{\partial v_1}{\partial I_2} \\ \frac{\partial v_2}{\partial E} & \frac{\partial v_2}{\partial I_1} & \frac{\partial v_2}{\partial I_2} \\ \frac{\partial v_3}{\partial E} & \frac{\partial v_3}{\partial I_1} & \frac{\partial v_3}{\partial I_2} \end{bmatrix} = \begin{bmatrix} -(\gamma+\mu) & 0 & 0 \\ a\gamma & -(r+\delta+\mu) & 0 \\ b\gamma & 0 & -(q+\delta+\mu) \end{bmatrix}$$

$$|V| = -(\gamma+\mu)(r+\delta+\mu)(q+\delta+\mu)$$

$$FV^{-1} = \begin{bmatrix} \frac{-[a\gamma\beta S(q+\delta+\mu)+b\gamma\beta S(r+\delta+\mu)]}{(\gamma+\mu)(r+\delta+\mu)(q+\delta+\mu)} & \frac{-\beta S}{(r+\delta+\mu)} & \frac{-\beta S}{(q+\delta+\mu)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$R_0 = \left| \frac{-[a\gamma\beta S(q+\delta+\mu)+b\gamma\beta S(r+\delta+\mu)]}{(\gamma+\mu)(r+\delta+\mu)(q+\delta+\mu)} \right|$$

$$R_0 = \frac{[a\gamma\beta S(q+\delta+\mu)+b\gamma\beta S(r+\delta+\mu)]}{(\gamma+\mu)(r+\delta+\mu)(q+\delta+\mu)} \quad (6)$$

Local Stability Analysis at Disease Free Equilibrium

Proof:

We establish the above theorem by calculating the Jacobian matrix of the system in *eqn (1)* at DFE point. In which eqn (5) ;

$$E_0 = \left(S^0, E^0, {I_1}^0, {I_2}^0, T^0, R^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right)$$

It is necessary for the computation of local stability analysis $J(S, E, I_1, I_2, T, R)$ of the system which is given as:

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$$J = \begin{bmatrix} \frac{\partial B_1}{\partial S} & \frac{\partial B_1}{\partial E} & \frac{\partial B_1}{\partial I_1} & \frac{\partial B_1}{\partial I_2} & \frac{\partial B_1}{\partial T} & \frac{\partial B_1}{\partial R} \\ \frac{\partial B_2}{\partial S} & \frac{\partial B_2}{\partial E} & \frac{\partial B_2}{\partial I_1} & \frac{\partial B_2}{\partial I_2} & \frac{\partial B_2}{\partial T} & \frac{\partial B_2}{\partial R} \\ \frac{\partial B_3}{\partial S} & \frac{\partial B_3}{\partial E} & \frac{\partial B_3}{\partial I_1} & \frac{\partial B_3}{\partial I_2} & \frac{\partial B_3}{\partial T} & \frac{\partial B_3}{\partial R} \\ \frac{\partial B_4}{\partial S} & \frac{\partial B_4}{\partial E} & \frac{\partial B_4}{\partial I_1} & \frac{\partial B_4}{\partial I_2} & \frac{\partial B_4}{\partial T} & \frac{\partial B_4}{\partial R} \\ \frac{\partial B_5}{\partial S} & \frac{\partial B_5}{\partial E} & \frac{\partial B_5}{\partial I_1} & \frac{\partial B_5}{\partial I_2} & \frac{\partial B_5}{\partial T} & \frac{\partial B_5}{\partial R} \\ \frac{\partial B_6}{\partial S} & \frac{\partial B_6}{\partial E} & \frac{\partial B_6}{\partial I_1} & \frac{\partial B_6}{\partial I_2} & \frac{\partial B_6}{\partial T} & \frac{\partial B_6}{\partial R} \end{bmatrix} \tag{7}$$

$$J = \begin{bmatrix} -\mu & 0 & -\beta\frac{\Lambda}{\mu} & -\beta\frac{\Lambda}{\mu} & 0 & 0 \\ 0 & -(\gamma+\mu) & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} & 0 & 0 \\ 0 & a\gamma & -(r+\delta+\mu) & 0 & 0 & 0 \\ 0 & b\gamma & 0 & -(q+\delta+\mu) & 0 & 0 \\ 0 & 0 & r & q & -(\eta+\delta+\mu) & 0 \\ 0 & \gamma(1-a-b) & 0 & 0 & \eta & -(\rho+\mu) \end{bmatrix} \tag{8}$$

$$|J-\lambda I| = \begin{bmatrix} -\mu-\lambda & 0 & -\beta\frac{\Lambda}{\mu} & -\beta\frac{\Lambda}{\mu} & 0 & 0 \\ 0 & -(\gamma+\mu)-\lambda & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} & 0 & 0 \\ 0 & a\gamma & -(r+\delta+\mu)-\lambda & 0 & 0 & 0 \\ 0 & b\gamma & 0 & -(q+\delta+\mu)-\lambda & 0 & 0 \\ 0 & 0 & r & q & -(\eta+\delta+\mu)-\lambda & 0 \\ 0 & \gamma(1-a-b) & 0 & 0 & \eta & -(\rho+\mu)-\lambda \end{bmatrix}$$

$$\lambda_1 = -\mu \tag{9}$$

$$J_0 = \begin{bmatrix} -(\gamma+\mu)-\lambda & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} & 0 & 0 \\ a\gamma & -(r+\delta+\mu)-\lambda & 0 & 0 & 0 \\ b\gamma & 0 & -(q+\delta+\mu)-\lambda & 0 & 0 \\ 0 & r & q & -(\eta+\delta+\mu)-\lambda & 0 \\ \gamma(1-a-b) & 0 & 0 & \eta & -(\rho+\mu)-\lambda \end{bmatrix}$$

$$\lambda_2 = -(\rho+\mu) \tag{10}$$

$$J_1 = \begin{bmatrix} -(\gamma+\mu)-\lambda & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} & 0 \\ a\gamma & -(r+\delta+\mu)-\lambda & 0 & 0 \\ b\gamma & 0 & -(q+\delta+\mu)-\lambda & 0 \\ 0 & r & q & -(\eta+\delta+\mu)-\lambda \end{bmatrix}$$

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$$\lambda_3 = -(\eta + \delta + \mu) \tag{11}$$

$$J_2 = \begin{bmatrix} -(\gamma+\mu) - \lambda & \beta\frac{\Lambda}{\mu} & \beta\frac{\Lambda}{\mu} \\ a\gamma & -(r+\delta+\mu) - \lambda & 0 \\ b\gamma & 0 & -(q+\delta+\mu) - \lambda \end{bmatrix}$$

$$\lambda_4 = -(\gamma + \mu) \tag{12}$$

$$J_3 = \begin{bmatrix} \frac{a\gamma\beta S - (\gamma+\mu)(r+\delta+\mu)}{(\gamma+\mu)} - \lambda & \frac{a\gamma\beta S}{(\gamma+\mu)} \\ \frac{b\gamma\beta S}{(\gamma+\mu)} & \frac{b\gamma\beta S - (\gamma+\mu)(q+\delta+\mu)}{(\gamma+\mu)} - \lambda \end{bmatrix}$$

$$\lambda_5 = \frac{a\gamma\beta S - (\gamma+\mu)(r+\delta+\mu)}{(\gamma+\mu)} \tag{13}$$

$$\lambda_6 = \frac{b\gamma\beta S - (\gamma+\mu)(q+\delta+\mu)}{(\gamma+\mu)} \tag{14}$$

From the basic reproduction number R_0 in eqn (6)

$$|R_0| = \frac{[a\gamma\beta S(q+\delta+\mu) + b\gamma\beta S(r+\delta+\mu)]}{(\gamma+\mu)(r+\delta+\mu)(q+\delta+\mu)}$$

$$R_0 = \frac{a\gamma\beta S}{(\gamma+\mu)(r+\delta+\mu)} + \frac{b\gamma\beta S}{(\gamma+\mu)(q+\delta+\mu)}$$

$$R_0 = R_{0I_1} + R_{0I_2} \tag{15}$$

If,

$$R_{0I_1} = \frac{a\gamma\beta S}{(\gamma+\mu)(r+\delta+\mu)} \tag{16}$$

Then

$$\lambda_5 \leq -(r+\delta+\mu)\left[1 - R_{0I_1}\right] \tag{17}$$

Also if,

$$R_{0I_2} = \frac{b\gamma\beta S}{(\gamma+\mu)(q+\delta+\mu)} \tag{18}$$

Then

$$\lambda_6 \leq -(q+\delta+\mu)\left[1 - R_{0I_2}\right] \tag{19}$$

Global Stability of Disease-Free Equilibrium (D F E)

The positive equilibrium point of the model in *eqn (1)* is globally asymptotically stable if $R_0 < 1$

Proof:

To establish the global stability of this equilibriums E_0, we construct the following Lyapunov function following the method of Lyapunov function.

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$$G(S,E,I_1,I_2,T,R) = \left(S - S^0 - S^0 log\frac{S^0}{S}\right) + \left(E - E^0 - E^0 log\frac{E^0}{E}\right) + \left(I_1 - {I_1}^0 - {I_1}^0 log\frac{{I_1}^0}{I_1}\right) + \left(I_2 - {I_2}^0 - {I_2}^0 log\frac{{I_2}^0}{I_2}\right) + \left(T - T^0 - T^0 log\frac{T^0}{T}\right) + \left(R - R^0 - R^0 log\frac{R^0}{R}\right) \quad (20)$$

By direct calculation and solving for the derivative of G along the system path of the model (1) we obtain,

$$\frac{dG}{dt} = \left(\frac{S - S^0}{S}\right)\frac{dS}{dt} + \left(\frac{E - E^0}{E}\right)\frac{dE}{dt} + \left(\frac{I_1 - {I_1}^0}{I_1}\right)\frac{dI_1}{dt} + \left(\frac{I_2 - {I_2}^0}{I_2}\right)\frac{dI_2}{dt} + \left(\frac{T - T^0}{T}\right)\frac{dT}{dt} + \left(\frac{R - R^0}{R}\right)\frac{dT}{dt} \quad (21)$$

So, by expanding $eqn\ (1)$ which involve representing the positive and negative terms with X and Y respectively we have:

$$\frac{dG}{dt} = X - Y$$

$$X = \left(1 - \frac{S^0}{S}\right)(\Lambda + \rho R) + \left(1 - \frac{E^0}{E}\right)(\beta S(I_1 + I_2)) + \left(1 - \frac{{I_1}^0}{I_1}\right)a\gamma E + \left(1 - \frac{{I_2}^0}{I_2}\right)b\gamma E + \left(1 - \frac{T^0}{T}\right)(rI_1 + qI_2) + \left(1 - \frac{R^0}{R}\right)(\gamma(1 - a - b)E + \eta T) \quad (22)$$

Similarly,

$$Y = \frac{(S - S^0)^2}{S}(\beta S(I_1 + I_2) + \mu) + \frac{(E - E^0)^2}{E}(\gamma + \mu) + \frac{(I_1 - {I_1}^0)^2}{I_1}(r + \delta + \mu) + \frac{(I_2 - {I_2}^0)^2}{I_2}(q + \delta + \mu) + \frac{(T - T^0)^2}{T}(\eta + \delta + \mu) + \frac{(R - R^0)^2}{R}(\rho + \mu) \quad (23)$$

If $X < Y$, then $\frac{dG}{dt}$ will be negative definite along the system (solution) path. So, it means that only at Tuberculosis Bacteria free system (Eo) would $\frac{dG}{dt} \leq 0$. This indicate that the system is globally stable at the tuberculosis bacteria disease free system.

Existence of Endemic Equilibrium Point

The presence of endemic equilibrium point refers to the process of stable solution in the model where the tuberculosis bacteria is positively present in the population. These equilibrium point represent the stable disease state where the number of infected individuals and other compartment reaches a steady state. These equilibrium point represent the stable solution of model where the disease persist over time. Moreso, it provides an important clue about the long-term dynamics of the tuberculosis disease in the population.

The endemic equilibrium point is defined as $(S^*(t), 0, 0, 0, 0, 0)$ which satisfy

$$(S' = E' = {I_1}' = {I_2}' = T = R = 0)$$

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$$\left.\begin{aligned}
&\Lambda + \rho R - (\beta(I_1 + I_2) + \mu)S = 0\\
&\beta S(I_1 + I_2) - (\gamma + \mu)E = 0\\
&a\gamma E - (r + \delta + \mu)I_1 = 0\\
&b\gamma E - (q + \delta + \mu)I_2 = 0\\
&rI_1 + qI_2 - (\eta + \delta + \mu)T = 0\\
&\gamma(1 - a - b)E + \eta T - (\rho + \mu)R = 0
\end{aligned}\right\} \quad (24)$$

By further simplification we have

$$S = \frac{\Lambda + \rho R}{\beta S(I_1 + I_2) + \mu} \quad (25)$$

$$E = \frac{\beta S(I_1 + I_2)}{\gamma + \mu} \quad (26)$$

$$I_1 = \frac{a\gamma E}{r + \delta + \mu} \quad (27)$$

$$I_2 = \frac{b\gamma E}{q + \delta + \mu} \quad (28)$$

$$T = \frac{rI_1 + qI_2}{\eta + \delta + \mu} \quad (29)$$

$$R = \frac{\gamma(1 - a - b)E + \eta T}{\rho + \mu} \quad (30)$$

Sensitivity Analysis of the Model

We verify different parameters within their respective plausible ranges, and the model response is observed. This variation can be done individually or collectively for multiple parameters simultaneously. As a result, we analyze the reproduction number of the model where we look at the variation and the impact of each parameter's value on the reproduction number.

The normalized forward-sensitivity index of the variable U, which is dependent on a parameter V is as follows:

$$X_V^U = \frac{\partial U}{\partial V} \cdot \frac{V}{U} \quad (31)$$

However, with regard to this model we will calculate the sensitivity indices for the fundamental reproduction number R_0 in (**6**),

$$R_0 = \frac{[a\gamma\beta S(q + \delta + \mu) + b\gamma\beta S(r + \delta + \mu)]}{(\gamma + \mu)(r + \delta + \mu)(q + \delta + \mu)} = \frac{[a\gamma\beta\Lambda(q + \delta + \mu) + b\gamma\beta\Lambda(r + \delta + \mu)]}{\mu(\gamma + \mu)(r + \delta + \mu)(q + \delta + \mu)} \quad (32)$$

The normalized forward sensitivity index of B is given by:

$$X_\beta^{R_0} = \frac{\partial R_0}{\partial \beta} \cdot \frac{\beta}{R_0} \quad (33)$$

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Parameter	Value	Source
a	0.0333	Assumed
b	0.015	Assumed
γ	0.11	Alfiniyah et al., 2024.
β	0.369	Assumed
Λ	0.7	Assumed
q	0.035	Assumed
δ	0.0240	Ogbaji et al., 2019.
μ	0.0124	Ogbaji et al., 2019.
r	0.06	Alfiniyah et al., 2024.

Table 3: Basic reproduction numbers parameters and values

$$R_0 = \frac{[a\gamma\beta\Lambda(q+\delta+\mu)+b\gamma\beta\Lambda(r+\delta+\mu)]}{\mu(\gamma+\mu)(r+\delta+\mu)(q+\delta+\mu)} = 1.88$$

The normalized forward sensitivity index approach was used to find the sensitivity index of all parameters in the reproduction number.

Sensitivity index of a

The normalized forward sensitivity index of a, is given by:

$$X_a^{R_0} = \frac{\partial R_0}{\partial a}\cdot\frac{a}{R_0} = +2.953 \quad (34)$$

Sensitivity index of b

The normalized forward sensitivity index of b, is given by:

$$X_b^{R_0} = \frac{\partial R_0}{\partial b}\cdot\frac{b}{R_0} = +1.3889 \quad (35)$$

Sensitivity index of γ

The normalized forward sensitivity index of γ, is given by:

$$X_\gamma^{R_0} = \frac{\partial R_0}{\partial \gamma}\cdot\frac{\gamma}{R_0} = +1 \quad (36)$$

Sensitivity index of β

The normalized forward sensitivity index of β, is given by:

$$X_\beta^{R_0} = \frac{\partial R_0}{\partial \beta}\cdot\frac{\beta}{R_0} = +1 \quad (37)$$

Sensitivity index of Λ

The normalized forward sensitivity index of Λ, is given by:

$$X_\Lambda^{R_0} = \frac{\partial R_0}{\partial \Lambda}\cdot\frac{\Lambda}{R_0} = +1 \quad (38)$$

Sensitivity index of q

The normalized forward sensitivity index of q, is given by:

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$$X_q^{R_0} = \frac{\partial R_0}{\partial q} \cdot \frac{q}{R_0} = +8.215 \quad (39)$$

Sensitivity index of δ

The normalized forward sensitivity index of $\boldsymbol{\delta}$, is given by:

$$X_\delta^{R_0} = \frac{\partial R_0}{\partial \delta} \cdot \frac{\delta}{R_0} = -0.2819 \quad (40)$$

Sensitivity index of μ

The normalized forward sensitivity index of $\boldsymbol{\mu}$, is given by:

$$X_\mu^{R_0} = \frac{\partial R_0}{\partial \mu} \cdot \frac{\mu}{R_0} = -0.00000000142 \quad (41)$$

Sensitivity index of r

The normalized forward sensitivity index of $\boldsymbol{r}$, is given by:

$$X_\mu^{R_0} = \frac{\partial R_0}{\partial \mu} \cdot \frac{\mu}{R_0} = -0.38703 \quad (42)$$

The sensitivity indices of all other parameters in the context of the fundamental reproduction number are given below:

Table 4: Basic Reproduction number parameters and sensitivity index values

Parameter	Value	Source	Index Sign	Sensitivity Index Value
a	0.0333	Assumed	+	2.953
b	0.015	Assumed	+	1.3889
γ	0.11	Alfiniyah et al., 2024.	+	1
β	0.369	Assumed	+	1
Λ	0.7	Assumed	+	1
q	0.035	Assumed	+	8.215
δ	0.0240	Ogbaji et al., 2019.	−	0.2819
μ	0.0124	Ogbaji et al., 2019.	−	0.00000000142
r	0.06	Alfiniyah et al., 2024.	−	0.38703

Table 4: Sensitivity indices with the basic reproduction number parameter value

Tuberculosis Optimal Control Analysis

Let

$$K_1 = \text{Awareness based Intervention (ABI)}$$

$$K_2 = \text{Conducting Tuberculosis Examination}$$

$$K_3 = \text{Provision of Essential Treatment}$$

$$K_4 = \text{Tuberculosis diagnosis with Treatment}$$

$$\rho = \text{Probability of Infection}$$

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From eqn (1)

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \rho R - (\beta(I_1 + I_2) + \mu)S \\
\frac{dE}{dt} &= \beta S(I_1 + I_2) - \mu E - a\gamma E - b\gamma E - \gamma(1 - a - b)E \\
\frac{dI_1}{dt} &= a\gamma E - (r + \delta + \mu)I_1 \\
\frac{dI_2}{dt} &= b\gamma E - (r + \delta + \mu)I_2 \\
\frac{dT}{dt} &= rI_1 + qI_2 - (\eta + \delta + \mu)T \\
\frac{dR}{dt} &= \gamma(1 - a - b)E + \eta T - (\rho + \mu)R
\end{aligned}\right\} \quad (1)$$

By introducing the control strategies

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \rho(1 - K_1)R - \mu S - \beta(I_1 + I_2)\sigma\,(1 - K_1)S \\
\frac{dE}{dt} &= \beta(I_1 + I_2)\sigma\,(1 - K_1)S - \mu E - a\gamma(1 - K_2)E - b\gamma(1 - K_2)E - \gamma(1 - a - b)(1 - K_2)E \\
\frac{dI_1}{dt} &= a\gamma(1 - K_2)E - (r + \delta + \mu)I_1 - K_3 I_1 \\
\frac{dI_2}{dt} &= b\gamma(1 - K_2)E - (q + \delta + \mu)I_2 - K_3 I_2 \\
\frac{dT}{dt} &= rI_1 + K_3 I_1 + qI_2 + K_3 I_1 - (\eta + \delta + \mu)T - K_4 T \\
\frac{dR}{dt} &= \gamma(1 - a - b)(1 - K_2)E + K_4 T + \eta T - \mu R - \rho(1 - K_1)R
\end{aligned}\right\} \quad (43)$$

Mathematical analysis of the model with optimal control strategies

By using pontryagin's maximum principle, an objective functional is formulated in presenting the existence of the optimal control measures. The objective function (G) establishes the most effective of the strategies which were presented. These measures are analytically presented to be feasible in minimizing the transmission of tuberculosis in a finite time interval $[O, T]$ with $K = \{(K_1, K_2, K_3, K_4)\epsilon K\}$Lebesgue measurable on $[0,1]$, $0 \leq K_i(t) \leq 1\,\epsilon\,[O, T], i = 1,2,3,4.$

The objective functional (G) is defined by:

$$G\big((K_1, K_2, K_3, K_4)\big) = \int_0^T (M_1 E + M_2 I_1 + M_3 I_2 + \frac{1}{2}(W_1 K_1^2 + W_2 K_2^2 + W_3 K_3^2 + W_4 K_4^2))dt \quad (44)$$

Which is subjected to the system of $eqn(1)$ with:

$S(0) > 0, E(0) > 0, I_1(0) > 0, I_2(0) > 0, T(0) = 0, R(0).$

Where K_1 is Awareness based Intervention, K_2 is conducting Tuberculosis examination, K_3 is the provision of essential treatment and K_4 is conducting tuberculosis examination with the provision of essential. Moreso, W_1, W_2,

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W_3 are weight contanst corresponding to each control, while M_1, M_2, M_3 are weight constants corresponding to the minimized population E, I_1 and I_2. Optimal control timeout is at an interval $0 \leq t \leq T$.

To achieve the objective of the control problem, we implement the function such that: $G\left(K_1^*(t), K_2^*(t), K_3^*(t), K_4^*(t)\right) = min\{G(K_1, K_2, K_3, K_4), K_1, K_2, K_3, K_4 \epsilon K\}$ (45)

Existence of an optimal control

Theorem 2: If we consider the objective functional $G(K_1, K_2, K_3, K_4)$ in eqn (44) where the set control K is measureable with the initial condition which is given at $t = 0$. Then there exists an optimal control.

$$K^* = \left(K_1^*(t), K_2^*(t), K_3^*(t), K_4^*(t)\right). \quad (46)$$

Such that:

$$G\left(K_1^*(t), K_2^*(t), K_3^*(t), K_4^*(t)\right) = min\{G(K_1, K_2, K_3, K_4), K_1, K_2, K_3, K_4 \epsilon K\}$$

Proof: Using the convexity of the integral of G to optimize control (K_1, K_2, K_3, K_4) the positive region of the model, boundedness of the solution Lipchitz property of the system which contain the state variables (S, E, I_1, I_2, T, R), thus the optimal control of the model exists.

We need to establish the Hamiltonian (M) and Langrangian (L) for the control problem.

The Langrangian can be written as:

$$L = M_1E + M_2I_1 + M_3I_2 + \frac{1}{2}(W_1K_1^2 + W_2K_2^2 + W_3K_3^2 + W_4K_4^2) \quad (47)$$

And the Hamiltonian function for the system is:

$$H = M_1E + M_2I_1 + M_3I_2 + \frac{1}{2}(W_1K_1^2 + W_2K_2^2 + W_3K_3^2 + W_4K_4^2) + \alpha_L S[\Lambda + \rho(1 - K_1)R - \mu S - \beta(I_1 + I_2)\sigma\,(1 - K_1)S] + \alpha_L E[\beta(I_1 + I_2)\sigma\,(1 - K_1)S - \mu E - a\gamma(1 - K_2)E - b\gamma(1 - K_2)E - \gamma(1 - a - b)(1 - K_2)E] + \alpha_L I_1[a\gamma(1 - K_2)E - (r + \delta + \mu)I_1 - K_3I_1] + \alpha_L I_2[b\gamma(1 - K_2)E - (q + \delta + \mu)I_2 - K_3I_2] + \alpha_L T[rI_1 + K_3I_1 + qI_2 + K_3I_2 - (\eta + \delta + \mu)T - K_4T] + \alpha_L R[\gamma(1 - a - b)(1 - K_2)E + K_4T + \eta T - \mu R - \rho(1 - K_1)R] \quad (48)$$

Where, $\alpha_L i, i \in (S, E, I_1, I_2, T, R)$ are the disjointed variables. We need to apply the required conditions to the Hamiltonian in the above theorem.

Theorem 3: By considering an optimal control $K^* = \left(K_1^*(t), K_2^*(t), K_3^*(t), K_4^*(t)\right)$ and a solution $Z^* = (S^*, E^*, I_1{}^*, I_2{}^*, T^*, R^*)$.

The state variable in K_1^*, K_2^*, K_3^* and K_4^* are obtained by solving the state equation.

$$\dot{x} = \frac{\partial G}{\partial f} \quad (49)$$

While the Lagrange multiplier for the controls K_1^*, K_2^*, K_3^* and K_4^* are obtained by solving the lagrange equation.

$$\dot{x} = -\frac{\partial G}{\partial x} \quad (50)$$

In which the state variable and the Lagrange multiplier will be substituted. The optimal solution to the mathematical model of tuberculosis will be determined by the substitution of the control in to the state equation.

Thus, the controller forms of K_1^*, K_2^*, K_3^* and K_4^* depends on the state and costate variables.

The state equations are given as follows:

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$$\left.\begin{aligned}
\frac{dS}{dt} &= \frac{\partial G}{\partial f_1} = \Lambda + \rho(1-K_1)R - \mu S - \beta(I_1+I_2)\sigma\,(1-K_1)S \\
\frac{dE}{dt} &= \frac{\partial G}{\partial f_2} = \beta(I_1+I_2)\sigma\,(1-K_1)S\; - \mu E - \; a\gamma(1-K_2)E - b\gamma(1-K_2)E \\
&\qquad -\gamma(1-a-b)(1-K_2)E \\
\frac{dI_1}{dt} &= \frac{\partial G}{\partial f_3} = a\gamma(1-K_2)E - (r+\delta+\mu)I_1 - K_3 I_1 \\
\frac{dI_2}{dt} &= \frac{\partial G}{\partial f_4} = b\gamma(1-K_2)E - (q+\delta+\mu)I_2 - \; K_3 I_2 \\
\frac{dT}{dt} &= \frac{\partial G}{\partial f_5} = rI_1 + K_3 I_1 + qI_2 + K_3 I_1 - (\eta+\delta+\mu)T - K_4 T \\
\frac{dR}{dt} &= \frac{\partial G}{\partial f_6} = \gamma(1-a-b)(1-K_2)E + K_4 T + \eta T - \mu R - \rho(1-K_1)R
\end{aligned}\right\} \tag{51}$$

However, the costate equations are given below:

$$\begin{aligned}
\frac{d\alpha_L S}{dt} = -\frac{\partial G}{\partial S} &= -[\alpha_L S[-\beta(I_1+I_2)\sigma\,(1-K_1)] - \mu] + \alpha_L E[\beta(I_1+I_2)\sigma\,(1-K_1)\,]] \\
&= \alpha_L S[\beta(I_1+I_2)\sigma\,(1-K_1) - \mu] - \alpha_L E[\beta(I_1+I_2)\sigma\,(1-K_1)\,]
\end{aligned} \tag{52}$$

$$\begin{aligned}
\frac{d\alpha_L E}{dt} = -\frac{\partial G}{\partial E} &= -\big[M_1 + \alpha_L E[-a\gamma(1-K_2) - b\gamma(1-K_2)\; - \gamma(1-a-b)(1-K_2) - \mu] + \alpha_L I_1[a\gamma(1-K_2)] \\
&\quad + \alpha_L I_2[b\gamma(1-K_2)] + \alpha_L R[\gamma(1-a-b)(1-K_2)]\big] \\
&= -M_1 + \alpha_L E[a\gamma(1-K_2) + b\gamma(1-K_2) + \gamma(1-a-b)(1-K_2) + \mu] - \alpha_L I_1[a\gamma(1-K_2)] - \\
&\quad \alpha_L I_2[b\gamma(1-K_2)] - \alpha_L R[\gamma(1-a-b)(1-K_2)]
\end{aligned} \tag{53}$$

$$\begin{aligned}
\frac{d\alpha_L I_1}{dt} = -\frac{\partial G}{\partial I_1} &= -\big[M_2 + \alpha_L S[-\beta\sigma\,(1-K_1)S] + \alpha_L E[\beta\sigma\,(1-K_1)S\,] + \alpha_L I_1[-(r+\delta+\mu)I_1 - K_3\,] \\
&\quad + \alpha_L T[r+K_3]\big] \\
&= -M_2 + \alpha_L S[\beta\sigma\,(1-K_1)S] - \alpha_L E[\beta\sigma\,(1-K_1)S\,] + \alpha_L I_1[(r+\delta+\mu) + K_3\,] - \\
&\quad \alpha_L T[r+K_3]
\end{aligned} \tag{54}$$

$$\frac{d\alpha_L I_2}{dt} = -\frac{\partial G}{\partial I_2} = -\big[M_3 + \alpha_L S[-\beta\sigma\,(1-K_1)S] + \alpha_L E[\beta\sigma\,(1-K_1)S\,] + \alpha_L I_2[-(q+\delta+\mu)I_2 - \; K_3] + \alpha_L T[q+K_3]\big]$$

$$= -M_3 + \alpha_L S[\beta\sigma\,(1-K_1)S] - \alpha_L E[\beta\sigma\,(1-K_1)S\,] + \alpha_L I_2[(q+\delta+\mu)I_2 + K_3] - \alpha_L T[q+K_3] \tag{55}$$

$$\begin{aligned}
\frac{d\alpha_L T}{dt} = -\frac{\partial G}{\partial T} &= -\big[\alpha_L T[-(\eta+\delta+\mu) - K_4] + \alpha_L R[K_4+\eta]\big] \\
&= \alpha_L T[(\eta+\delta+\mu) - K_4] - \alpha_L R[K_4+\eta]
\end{aligned} \tag{56}$$

$$\frac{d\alpha_L R}{dt} = -\frac{\partial G}{\partial R} = -\big[\alpha_L S[\rho(1-K_1)] + \alpha_L R[-\rho(1-K_1) - \mu]\big] \tag{57}$$

The optimal conditions are obtained when the Hamiltonian function satisfies the following stationary conditions:

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$$\frac{\partial G}{\partial K_1} = 0, \frac{\partial G}{\partial K_2} = 0, \frac{\partial G}{\partial K_3} = 0, \frac{\partial G}{\partial K_4} = 0, \tag{58}$$

So, differentiating the control parameters K_1, K_2, K_3 and K_4.

We obtain.

$$\frac{dK_1}{dt} = W_1K_1 + \alpha_L S[-\rho R + \beta(I_1 + I_2)\sigma S] - \alpha_L E[\beta(I_1 + I_2)\sigma S] + \alpha_L R[\rho R] = 0$$

$$K_1 = \frac{\alpha_L S[\rho R - \beta(I_1+I_2)\sigma S] + \alpha_L E[\beta(I_1+I_2)\sigma S] - \alpha_L R[\rho R]}{W_1} \tag{59}$$

$$\frac{dK_2}{dt} = W_2K_2 + \alpha_L E[a\gamma E + b\gamma E + \gamma(1-a-b)E] - \alpha_L I_1[a\gamma E] - \alpha_L I_2[b\gamma E] - \alpha_L R[\gamma(1-a-b)E] = 0$$

$$K_2 = \frac{-\alpha_L E[a\gamma E + b\gamma E + \gamma(1-a-b)E] + \alpha_L I_1[a\gamma E] + \alpha_L I_2[b\gamma E] + \alpha_L R[\gamma(1-a-b)E]}{W_2} \tag{60}$$

$$\frac{dK_3}{dt} = W_3K_3 + \alpha_L I_1[-I_1] + \alpha_L I_2[-I_2] + \alpha_L T[I_1 + I_2]$$

$$K_3 = \frac{\alpha_L I_1[I_1] + \alpha_L I_2[I_2] - \alpha_L T[I_1+I_2]}{W_3} \tag{61}$$

$$\frac{dK_4}{dt} = W_4K_4 + \alpha_L T[-T] + \alpha_L R[T] = 0$$

$$K_4 = \frac{\alpha_L T[T] - \alpha_L R[T]}{W_4} \tag{62}$$

This provides the required optimization measures and putting boundary conditions of each of this control gives:

$$K_1^* = \min\{1, max\{0, C_1\}\}$$

$$K_2^* = \min\{1, max\{0, C_2\}\}$$

$$K_3^* = \min\{1, max\{0, C_3\}\}$$

$$K_4^* = \min\{1, max\{0, C_4\}\}$$

$$K_1 = \frac{\alpha_L S[\rho R - \beta(I_1 + I_2)\sigma S] + \alpha_L E[\beta(I_1 + I_2)\sigma S] - \alpha_L R[\rho R]}{W_1}$$

$$K_2 = \frac{-\alpha_L E[a\gamma E + b\gamma E + \gamma(1-a-b)E] + \alpha_L I_1[a\gamma E] + \alpha_L I_2[b\gamma E] + \alpha_L R[\gamma(1-a-b)E]}{W_2}$$

$$K_3 = \frac{\alpha_L I_1[I_1] + \alpha_L I_2[I_2] - \alpha_L T[I_1 + I_2]}{W_3}$$

$$K_4 = \frac{\alpha_L T[T] - \alpha_L R[T]}{W_4}$$

Numerical Solution

Finite Difference Scheme (FSD) is used to obtain the numerical solution of the set of differential equations presented in ***eqn* 1**, in order to achieve numerical values.

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$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \rho R - (\beta(I_1 + I_2) + \mu)S \\
\frac{dE}{dt} &= \beta S(I_1 + I_2) - (\gamma + \mu)E \\
\frac{dI_1}{dt} &= a\gamma E - (r + \delta + \mu)I_1 \\
\frac{dI_2}{dt} &= b\gamma E - (r + \delta + \mu)I_2 \\
\frac{dT}{dt} &= rI_1 + qI_2 - (\eta + \delta + \mu)T \\
\frac{dR}{dt} &= \gamma(1 - a - b)E + \eta T - (\rho + \mu)R
\end{aligned}\right\} \tag{63}$$

$$S(t) \geq 0, E(t) \geq 0, I_1(t) \geq 0, I_2(t) \geq 0, T(t) \geq 0, R(t) \geq 0$$

By using the finite difference scheme as stated earlier ***eqn* 1** can be decomposed as;

$$\left.\begin{aligned}
\frac{S_{(J+1)} - S_J}{h} &= \Lambda + \rho R_k - (\beta(I_1 + I_2) + \mu)S_k \\
\frac{E_{(J+1)} - E_J}{h} &= \beta S_k\left(I_{1_k} + I_{2_k}\right) - (\gamma + \mu)E_k \\
\frac{I_{1_{(J+1)}} - I_{1_J}}{h} &= a\gamma E_k - (r + \delta + \mu)I_{1_k} \\
\frac{I_{2_{(J+1)}} - I_{2_J}}{h} &= b\gamma E_k - (r + \delta + \mu)I_{2_k} \\
\frac{T_{(J+1)} - T_J}{h} &= rI_{1_k} + qI_{2_k} - (\eta + \delta + \mu)T_k \\
\frac{R_{(J+1)} - R_J}{h} &= \gamma(1 - a - b)E_k + \eta T_k - (\rho + \mu)R_k
\end{aligned}\right\} \tag{64}$$

Which can alternatively be written as;

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$$\left.\begin{aligned} S_{(J+1)} &= S_J + h\,(\,\Lambda + \rho R_k - (\beta(I_1 + I_2) + \mu)S_k\,) \\ E_{(J+1)} &= E_J + h\,(\,\beta S_k(I_{1_k} + I_{2_k}) - (\gamma + \mu)E_k\,) \\ I_{1_{(J+1)}} &= I_{1_J} + h\,(a\gamma E_k - (r + \delta + \mu)I_{1_k}\,) \\ I_{2_{(J+1)}} &= I_{2_J} + h\,(\,b\gamma E_k - (r + \delta + \mu)I_{2_k}\,) \\ T_{(J+1)} &= T_J + h\,(\,rI_{1_k} + qI_{2_k} - (\eta + \delta + \mu)T_k\,) \\ R_{(J+1)} &= T_J + h\,(\,\gamma(1 - a - b)E_k + \eta T_k - (\rho + \mu)R_k\,) \end{aligned}\right\} \quad (65)$$

In which $J = 0,1,2,3,4, \ldots, h$ is the step size

Numerical Simulation

In this section, we deploy a means to observe the propagation of the disease with respect to time and evaluating the effectiveness of the control strategies. The state variables' initial conditions are as follows;
$S = 10000, E = 500, I_1 = 2500, I_2 = 2000,\ T = 1500\ and\ R = 1000$.

Discussion

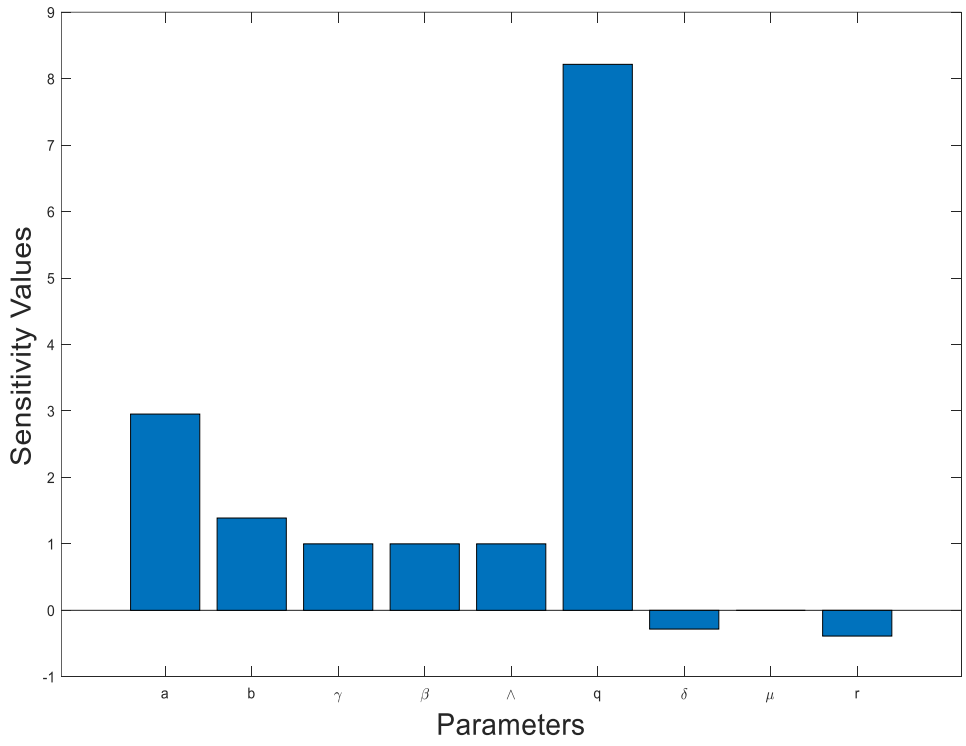

Figure 2: Graph of Sensitivity Index values and parameters

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In **Figure 2**, The parameters and the reproduction number have an inverse connection, as indicated by the negative sensitivity index. A positive sensitivity index, on the other hand suggests that a larger reproduction number results from a rise in the parameter value. This analysis aids in determining which parameter have the most impact on the analysis's outcome.

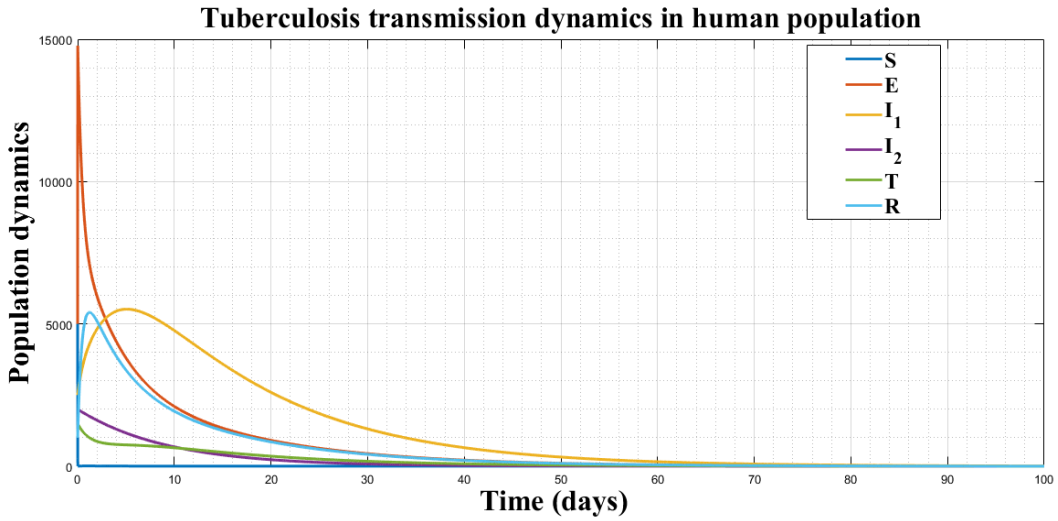

Figure 3: Trajectories solution of the Tuberculosis model

Figure 3, displays every compartment of the tuberculosis model, which are crucial for comprehending and examining the dynamics of the disease's transmission. Additionally, this strengthens the model's ability to explain, facilitates the spread of tuberculosis and assists in the decision-making process for disease prevention and control.

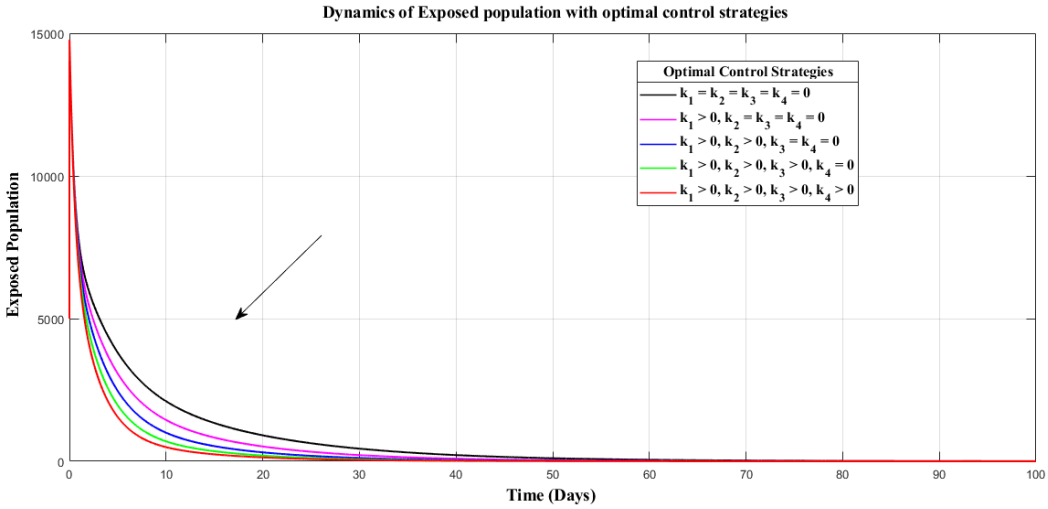

Figure 4: Trajectories solution for optimizing exposed population

Figure 4, illustrates the beneficial implications of the control measures that were put in place in the exposed population of the mathematical modeling of tuberculosis. These include awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment. The population in the exposed compartment has significantly decreased as a result of this strategies. This decrease in the number of exposed humans shows how well the control measures are working to curb the bacteria's spread. Additionally, it shows that efforts to reduce the spread of tuberculosis bacteria have been successful.

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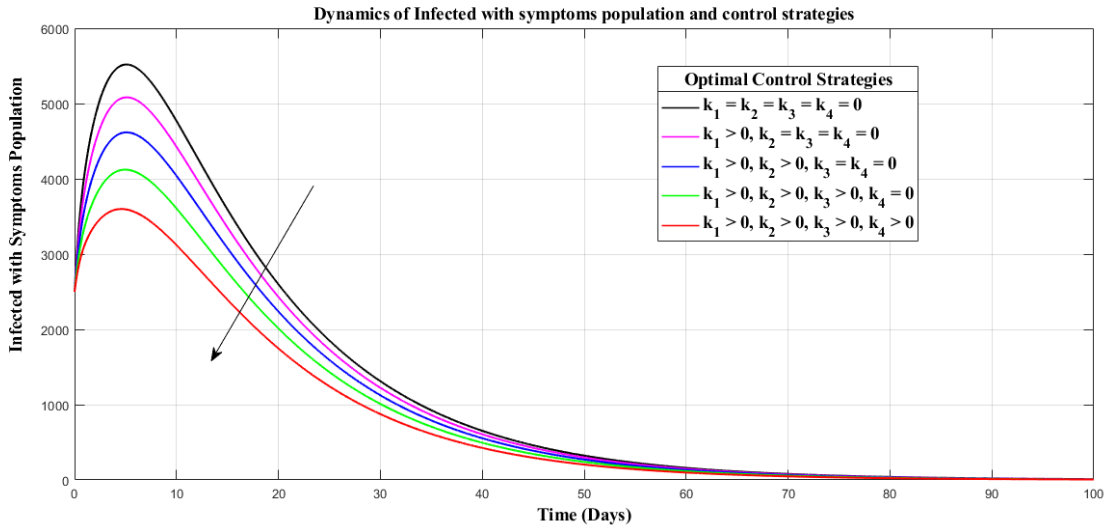

Figure 5: Trajectories solution for optimizing infected with symptoms population

Figure 5, illustrates the decline in the number of people displaying signs of a tuberculosis infection. In addition to demonstrating the beneficial effects of these control methods on public health, the decrease in tuberculosis infection within a group of individuals exhibiting symptoms also suggests the possibility of successfully preventing or controlling the disease outbreaks. By using control strategies such as awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment. It will improve the ability to protect the populations from tuberculosis infection, which will result in symptoms and subsequently lower the risks of tuberculosis on human health.

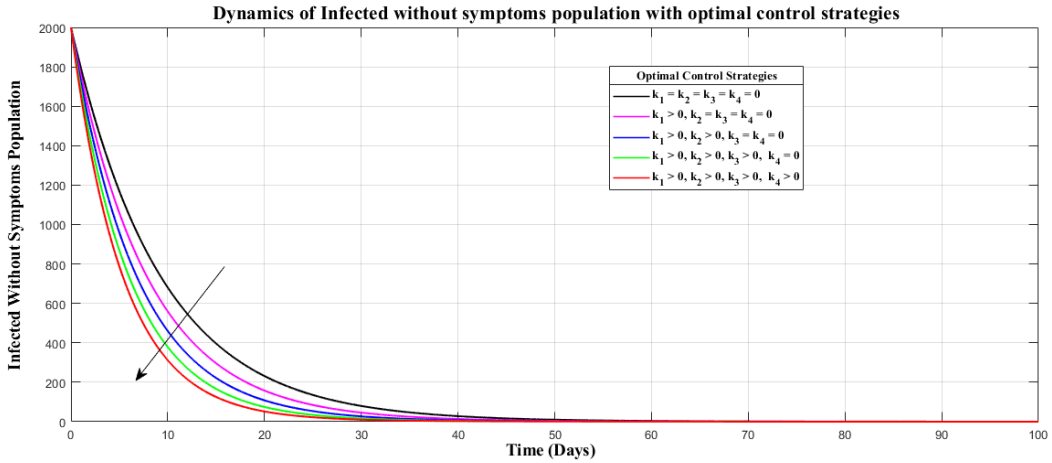

Figure 6: Trajectories solution for optimizing infected without symptoms population

A decrease in the number of people with tuberculosis who do not exhibit symptoms is seen in **Figure 6**. The impact of these control strategies: awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment are symbolized by the decline in tuberculosis infection within this group. This improve the ability to protect the populations who are infected without symptoms from tuberculosis infection which subsequently lowers the hazard of tuberculosis on human health. If implemented, the interventions will help public health authorities identify individuals who may be infectious of tuberculosis but for specific reasons may have not been aware. Thereby, reducing interactions with infected individuals who would have become informed of their present health condition may be achieved by putting control strategies like as awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and

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tuberculosis diagnosis with treatment into practice. In order to minimize the number of people who are at risk to having tuberculosis to the minimum.

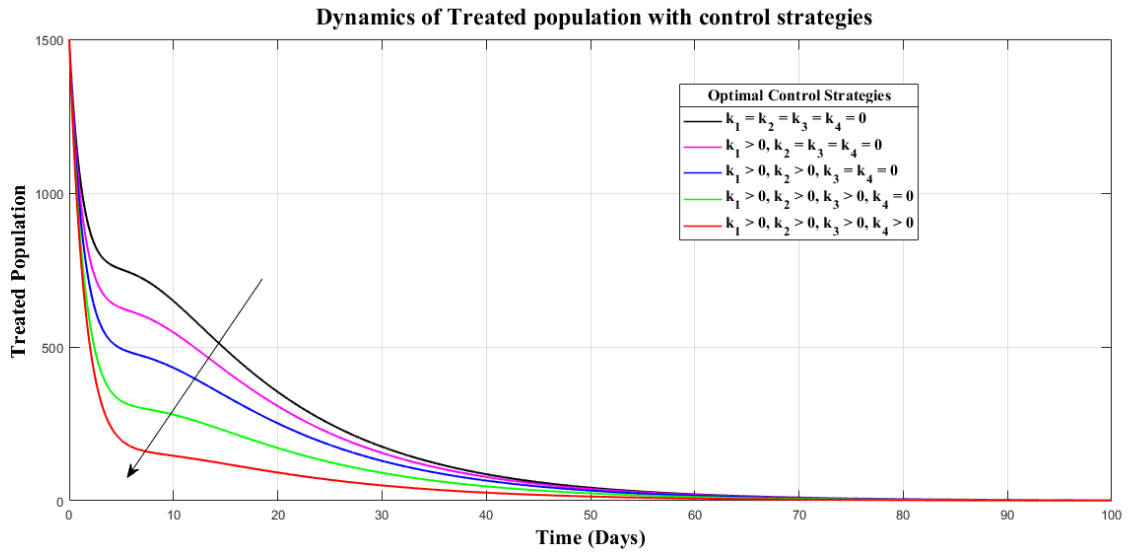

Figure 7: Trajectories solution for optimizing Treated population

The dynamic efficiency of these procedures in managing the epidemic is the positive consequence of the control strategies shown in **Figure 7** for the dynamics of tuberculosis in treated population. When the optimal control of treated persons reaches a particular level, it means that a proactive treatment strategy is working. Additionally, this suggests that awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment are all significantly contributing to the survival and recovery of the human population. The decrease in the number of people receiving treatment indicates that these control measures are effectively halting the bacterial spread. This is demonstrated by the fall in the human treated class after reaching specific thresholds. Because that illustrates awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment all assisted to lessen the severity of tuberculosis infections and stop the disease from spreading. In the end, the model's dynamic nature emphasizes how flexible and successful these control measures are in reducing tuberculosis outbreaks.

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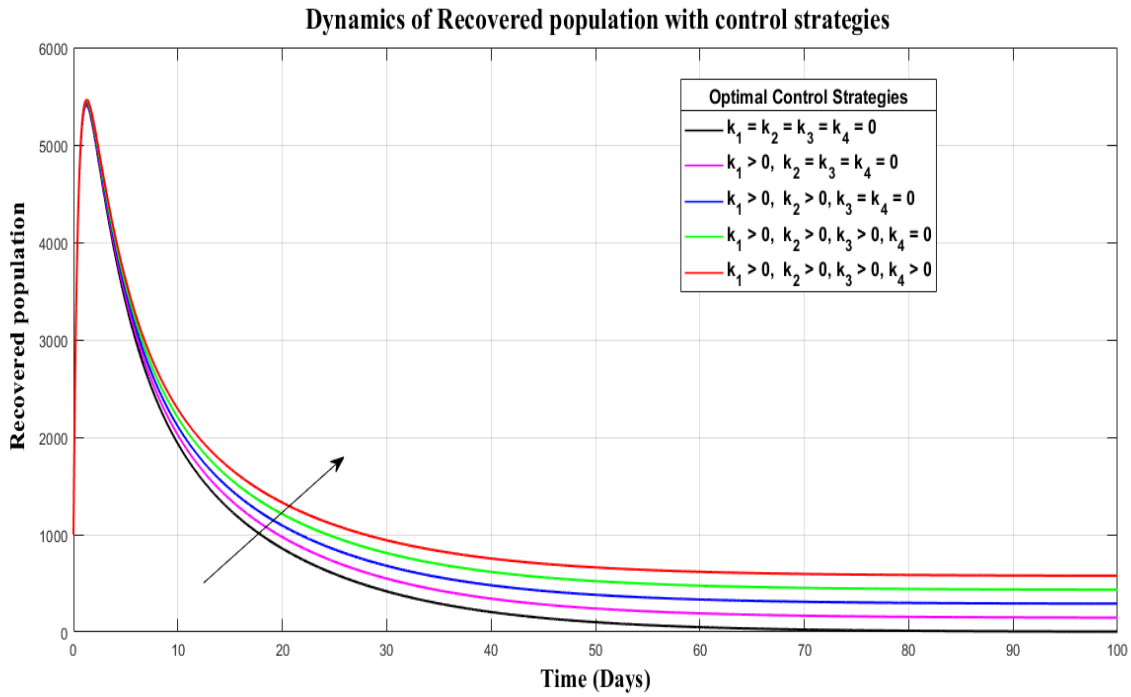

Figure 8: Trajectories solution for optimizing Recovered population.

Figure 8, shows the rise in the recovered population as a result of control measures, particularly awareness-based interventions (ABI), conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment. First of all, it shows how well these control measures work to stop the bacteria's spread and help the exposed and treated populations recover from their infections. Diagnosing tuberculosis at the right time using techniques such as molecular diagnostic testing, culture testing, sputum smear microscopy, chest X-rays and clinical examination. Alongside treatment services include anti-tuberculosis drugs, short-term, directly witnessed therapy, treatment monitoring and supportive health care services. Secondly, in order to increase the odds of recovery and survival for both the symptomatic and asymptomatic populations, tuberculosis diagnosis with treatments must be prioritized. This results in a greater percentage of people moving into the recovered class, which is good for slowing the spread of the disease in human population. Overall, the observable rise in the recovered population shows how effective these control measures are in reducing the negative effects of tuberculosis on human health and emphasizes the significance of ongoing efforts to implement and promote the control strategies.

Conclusion

The study reveals how the four control strategies which are awareness-based interventions, conducting tuberculosis examinations, provision of essential treatments and tuberculosis diagnosis with treatment help to reduce the spread of tuberculosis. The analysis presented in the study demonstrate the beneficial effects of the control strategies in lowering the incidence, severity, duration and susceptibility of tuberculosis cases. Susceptibility, exposure, infection with or without symptoms, treatment and recovery instances of tuberculosis in humans are all decreased by the control strategies. Thereby, producing better results for public health practitioners and policy makers. Therefore, these approaches provide confidence in the ongoing efforts to reduce tuberculosis by reducing the effect of the outbreaks.

Recommendations

1. The study makes an insightful contribution to the field of mathematical epidemiology, particularly through its detailed classification of tuberculosis infections and evaluation of various control measures.
2. It is recommended that the tuberculosis model to be adopted and further explored in both the public health sector and academic research.

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3. The findings from the study offer a solid foundation for designing targeted tuberculosis control interventions and developing predictive tools for outbreak management.
4. Moreso, researchers and health authorities are encouraged to apply this model to various epidemiological settings to validate its effectiveness and provide a support based evidence towards tuberculosis prevention and control.

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