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Mathematical Modelling Of Tuberculosis Transmission Dynamics With Control Measures

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Abstract
Background
Tuberculosis is a global endemic that claims millions of lives every year. It is an infectious airborne disease caused by bacteria and is transmitted through coughing and sneezing.
Materials and Methods
The study utilized secondary data obtained from the Ministry of health through the National TB Program. The study developed a six compartmental SVLATR mathematical model that incorporated TB transmission dynamics with control measures to assess their impact on TB spread . The threshold quantity called basic reproduction number R_0, that determines whether the disease persists and spreads or gets eliminated was computed using the Next Generation Matrix (NGM) . Stability properties of the system were analyzed using the Jacobian Matrix and eigenvalue analysis.
Findings
The basic reproduction number was found to be $R_0 \approx 1.73$. The model was used to predict future trends in TB and projected a decline in TB incidence, prevalence and mortality for the next 5 years but insufficient to fully eliminate TB. The Disease-Free Equilibrium (DFE) is stable if $R_0 < 1$, since $R_0 > 1$ it was found to be unstable while Endemic Equilibrium (EE) is locally asymptotically stable.
Conclusion
A reproduction number $R_0 \approx 1.73 > 1$ indicates persistence of the endemic and can't be eliminated. Extreme measures should be taken to lower R_0 below unity to eliminate TB. This research provides policy makers with recommendations for improving Tuberculosis control.
Keywords: *Tuberculosis, Mathematical Modelling, Control Measures, Stability Analysis, Reproduction Number, Compartmental model*

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I. Introduction

Tuberculosis is an infectious airborne disease caused by a bacterium called Mycobacterium tuberculosis [1]. It is transmitted through inhaling of the bacteria released from an infected person after coughing or sneezing as presented by [2]. The bacteria gets into the lungs and in the long run affects other body organs [3]. It is one of major causes of fatalities in developing countries claiming millions of lives every year as demonstrated by [4]. However, it can be prevented through vaccination of the susceptible population and immunization of the infants as highlighted by [5,6]. Though tuberculosis is curable, there still exists variations in diagnosis, prevention and efficacy in treatment of the disease as presented by [7].

Tuberculosis can be diagonized based on the medical history, physical examination and tests before or after showing symptoms of the infection. Conditions that increases chances to contracting tuberculosis include: Diabetes, weakened immune system, smoking of tobacco among others [7,8]. Tuberculosis is more rampant in elderly individuals, children and risk factor groups such as health care workers, smokers and drug addicts [8].

Tuberculosis can be prevented by avoiding overcrowded public places, observing good hygiene at all times and being in properly ventilated places [14]. The disease can also be prevented by immunization of the infants using BCG vaccine [15,16] .

The common symptoms of tuberculosis include: weight loss, fatigue, fever, night sweats, chest pain among others [16]. Delayed and poor diagnosis may lead to the development of the disease to stages that might be difficult to control [17].Therefore, early detection is highly recommended for successful prevention measures [15]. Vaccination of susceptible population has been proven to reduce the disease burden if it is properly done [5]. However, TB can be successfully treated as there is cure for Tuberculosis [17].

Treatment of infected persons with tuberculosis can be done using antibiotics and it can be successfully cured by proper adherence to medication [17]. However, poor medication may result into prolonged treatment hence leading to poor treatment outcomes [15]. Though the disease can be fully cured, there are some bacteria that resists treatment of tuberculosis leading to poor response to treatment [17]. Drug resistance bacteria have been shown to undergo mutation to form strains that are resistant to medication [17].

II. Materials And Methods

Model Formulation and Assumptions

A deterministic compartmental model was developed in order to capture the natural history of tuberculosis transmission. Six epidemiologically meaningful compartments were developed from the stratified total population $N(t)$: Susceptible (S), Vaccinated (V), Latent (L), Active TB population (A), Treatment (T), and Recovered (R). The transitions and interactions between compartments are modeled using differential equations. Epidemiological data and literature used to establish parameter values. The population is assumed to mix homogeneously, meaning every susceptible individual has an equal chance of contacting an infectious TB individual. Transmission of TB occurs when susceptible individuals come into effective contact with individuals who have active TB infection. Sus-ceptible individuals can move to the vaccinated class through vaccination, which reduces their probability of infection. Individuals who become infected progress to the active TB class, where they are capable of transmitting the disease to susceptible individuals. Individuals diagnosed with TB may move from the infected class to the treatment class, where medical intervention reduces the infectious period. Those receiving treatment may recover and move to the recovered class. People with active tuberculosis may die due to TB related complications at a disease induced death rate. All compartments experience natural mortality, which is unrelated to tuberculosis. Individuals who recover from TB are assumed to gain temporary or partial immunity, reducing their risk of immediate re-infection. Model parameters such as transmission rate, vaccination rate, treatment rate, and recovery rate are assumed to remain constant over the study period.

The expression for the population identification is:

$$N(t) = S(t) + V(t) + L(t) + A(t) + T(t) + R(t)$$

Consequently, a six-compartment SV LATR model was generated by extending the modified SEIR framework. These compartments serve as the foundation used to construct the differential equations system that characterizes the dynamics of tuberculosis under various preventative and control measures.

Schematic Diagram for Transmission of Tuberculosis

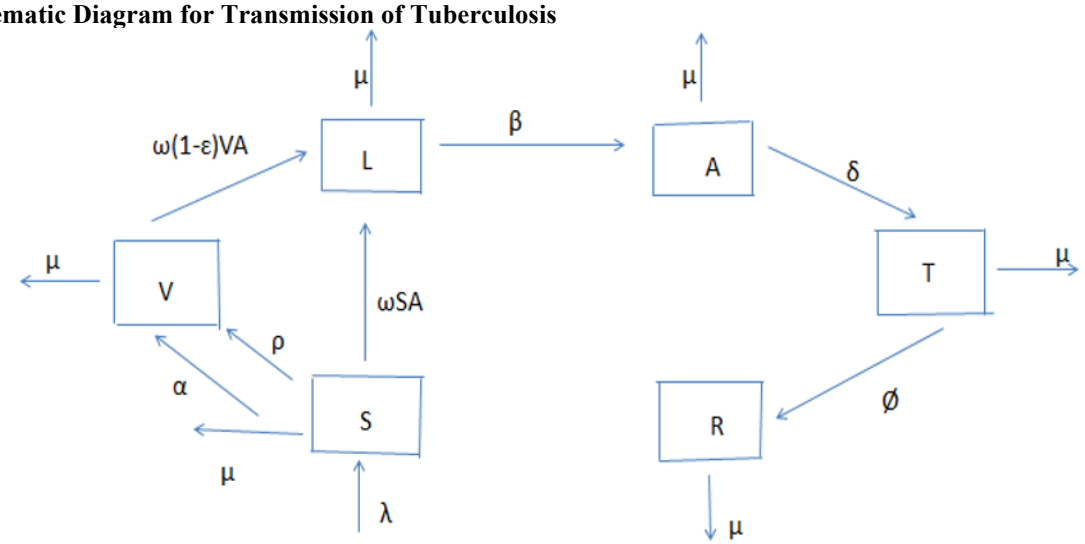


Figure 1: Flow Diagram of the Model

Figure 1 illustrates the suggested TB transmission model. The schematic diagram's parameterized arrows illustrate the well-defined epidemiological processes that govern the transitions within various compartments. New members first join the vulnerable class, and recruitment into the population happens at a constant rate λ Vaccinated individuals may become infected, albeit at a lower risk, due to imperfect efficacy (ε), while vaccine-induced immunity wanes at a rate ρ, returning individuals to the susceptible pool. At rates ωSA and $(1 - \varepsilon)VA$, respectively, transmission occurs when susceptibles or vaccinated individuals effectively come into contact with infectious cases. This in turn leading to movement into the latent class. Latently infected

individuals become infectious when they develop active TB at a rate β. Active cases may commence treatment at rate δ, entering the treatment compartment. A proportion recover at rate ϕ , moving into the recovered class. Mortality is incorporated in two forms: all individuals experience natural death at rate μ, while active TB cases face an additional disease-induced mortality rate σ.

Model Equations

The model consists of six compartments: Susceptible (S), Vaccinated (V), Latent (L), Active TB (A), Treatment (T), and Recovered (R). The dynamics of each compartment are governed by the following system of ordinary differential equations.

Susceptible Population

Births at the rate λ and waning immunity from the vaccinated class at the rate ρ are the two main ways that the susceptible population gets recruited. It declines at the rates of infection (ωSA), vaccination (α), and natural death (μ). Consequently, the equation is:

$$\frac{dS}{dt} = \lambda + \rho V - \omega SA - (\mu + \alpha)S \quad (1)$$

Vaccinated Population

By vaccinating susceptibles at a rate αS, the vaccinated class increases. The rate of infection is $(1 - \varepsilon)$, the rate of waning is ρ, and the rate of natural deaths is μ. Consequently,

$$\frac{dV}{dt} = \alpha S - \omega(1 - \varepsilon)VA - (\mu + \rho)V \quad (2)$$

Latent Population

New infections from susceptible and vaccinated individuals populate the latent class. As active TB progresses, it declines at a rate of β and natural deaths μ. Therefore,

$$\frac{dL}{dt} = \omega SA + \omega(1 - \varepsilon)VA - (\beta + \mu)L \quad (3)$$

Active TB Population

At rate βL, the active TB class rises as latency progresses. Natural deaths μ, TB-related deaths σ, and treatment at rate δ all contribute to its decline. Consequently,

$$\frac{dA}{dt} = \beta L - (\mu + \sigma + \delta)A \quad (4)$$

Treatment Population

By starting therapy for active TB patients at rate δA, the treatment class grows. Recovery at rate ϕ and natural deaths μ cause it to decline. Consequently,

$$\frac{dT}{dt} = \delta A - (\mu + \phi)T \quad (5)$$

Recovered Population

The recovered class is populated through recovery of treated individuals at rate ϕT and diminishes through natural deaths μ. Therefore,

$$\frac{dR}{dt} = \phi T - \mu R \quad (6)$$

Summary of the Model

The complete system of equations can be summarized as follows:

$$\frac{dS}{dt} = \lambda + \rho V - \omega SA - (\mu + \alpha)S \quad (7)$$

$$\frac{dV}{dt} = \alpha S - \omega(1 - \varepsilon)VA - (\mu + \rho)V \quad (8)$$

$$\frac{dL}{dt} = \omega SA + \omega(1 - \varepsilon)VA \ - (\beta + \mu)L \quad (9)$$

$$\frac{dA}{dt} = \ \beta L - (\mu + \sigma + \delta)A \quad (10)$$

$$\frac{dT}{dt} = \delta A - (\mu + \phi)T \quad (11)$$

$$\frac{dR}{dt} = \phi T - \mu R \tag{12}$$

III. Findings

Model Analysis

Positivity and Boundedness

Biological validity of the tuberculosis model requires that the system's solutions remain non–negative and bounded for all t >0. We verify the invariance and positivity of solutions in this section.

Positivity of Solutions

Lemma 1. Let the initial conditions satisfy

$$S(0) \geq 0, \quad V(0) \geq 0, \quad L(0) \geq 0, \quad A(0) \geq 0, \quad T(0) \geq 0, \quad R(0) \geq 0.$$

Then the solutions $S(t), V(t), L(t), A(t), T(t), R(t)$ remain non–negative for all $t > 0$.

$Proof$. Consider the governing equations:

$$\frac{dS}{dt} = \lambda + \rho V - \omega SA - (\mu + \alpha)S$$
$$\frac{dV}{dt} = \alpha S - \omega(1-\varepsilon)VA - (\mu + \rho)V$$
$$\frac{dL}{dt} = \omega SA + \omega(1-\varepsilon)VA - (\beta + \mu)L$$
$$\frac{dA}{dt} = \beta L - (\mu + \sigma + \delta)A$$
$$\frac{dT}{dt} = \delta A - (\mu + \phi)T$$
$$\frac{dR}{dt} = \phi T - \mu R$$

Inflows (non-negative terms) less outflows (linear decay terms with positive coefficients) is how each equation is expressed. Therefore, the solution cannot turn negative once any compartment reaches 0 because the derivative at that point is non–negative. Consequently, for all t> 0, solutions remain non–negative.

Invariant Region

Lemma 2. Let $N(t)$ denote the total population

$$N(t) = S(t) + V(t) + L(t) + A(t) + T(t) + R(t).$$

Then the feasible region

$$\Omega = \left\{(S, V, L, A.T.R) \epsilon\, R_+^6 : N(t) \leq \frac{\lambda}{\mu}\right\}$$

is positively invariant.

Adding all the equations gives

$$\frac{dN}{dt} = \lambda - \mu N - \sigma A.$$

Since $-\sigma A \leq 0$, we obtain

$$\frac{dN}{dt} \leq \lambda - \mu N$$

The solution of the comparison equation $\frac{dN}{dt} \leq \lambda - \mu N$ is:

$$N(t) = \frac{\lambda}{\mu} + \left(N(0) - \frac{\lambda}{\mu}\right) e^{-\mu t}$$

Thus, as t $\rightarrow \infty$, $N(t) \leq \frac{\lambda}{\mu}$. This proves that all solutions enter and remain in **Ω**.

Conclusion: The region **Ω** is positively invariant and attracting. Therefore, all solutions of the system with non–negative initial conditions remain positive and bounded for all t > 0. Hence, the model is mathematically and biologically well–posed.

IV. Data Sources: Ministry Of Health Surveillance Data

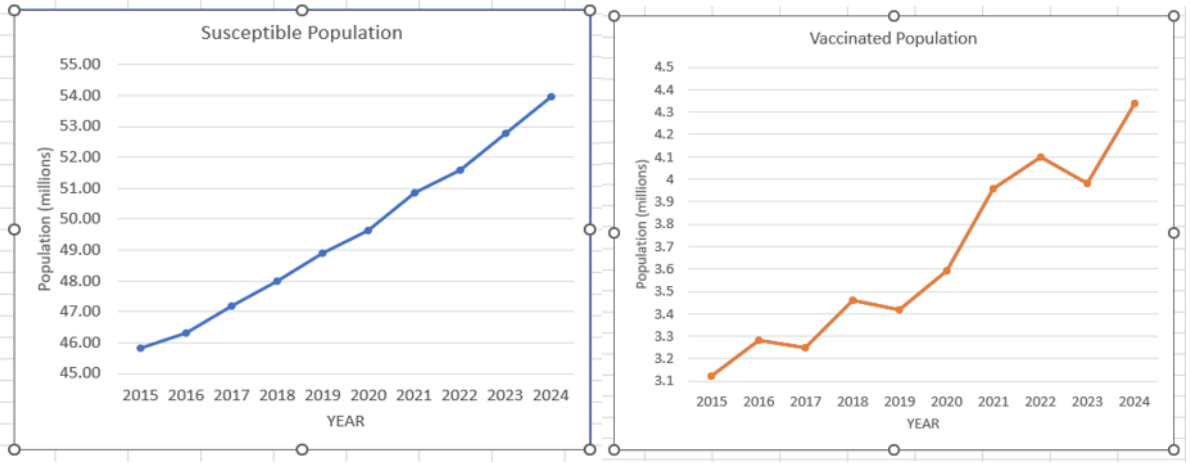
The following table presents Kenyan tuberculosis (TB) epidemiology data from 2015 to 2024, as supplied by the Ministry of Health through the National Tuberculosis Program. The table summarizes the annual population distributions across key TB-related com-partments, including susceptible, vaccinated, active TB cases, individuals undergoing treatment, recovered cases, and TB-related deaths.

Table 1: Epidemiological State Variables for TB in Kenya (2015–2024)

Year	Susceptible	Vaccinated	Active TB	Treatment	Recovered	TB Deaths
2015	45,823,418	3,118,764	175,482	81,518	72,551	4,891
2016	46,372,905	3,284,196	169,247	75,898	65,972	4,887
2017	47,184,362	3,251,487	164,839	85,188	74,965	5,111
2018	48,047,611	3,462,904	157,106	96,478	80,077	6,271
2019	48,891,274	3,418,765	149,892	86,504	72,563	5,615
2020	49,652,189	3,587,142	151,478	72,943	61,272	5,179
2021	50,846,532	3,962,518	141,936	77,854	68,512	4,671
2022	51,611,804	4,104,376	133,214	90,560	79,693	5,434
2023	52,784,916	3,981,744	135,006	97,126	86,442	6,313
2024	53,962,411	4,336,582	128,394	96,865	74,586	5,812

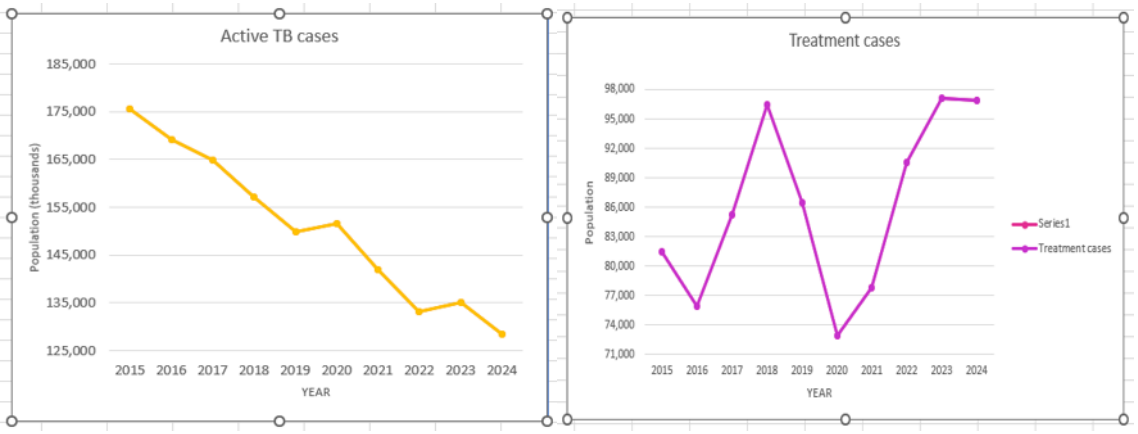
Table 1 shows the trends in TB epidemiology in Kenya over the study period. The data indicate a steady decline in active TB cases and unsteady fluctuation in TB-related deaths, suggesting improvements in case detection, treatment coverage, and treatment outcomes. There is also a slight increase in the number of vaccinated individuals, reflecting ongoing immunization efforts. Overall, the table illustrates progress in TB control attributable to strengthened prevention and treatment strategies implemented by the National Tuberculosis Program.

V. Trends Of TB In Kenya 2015 -2024



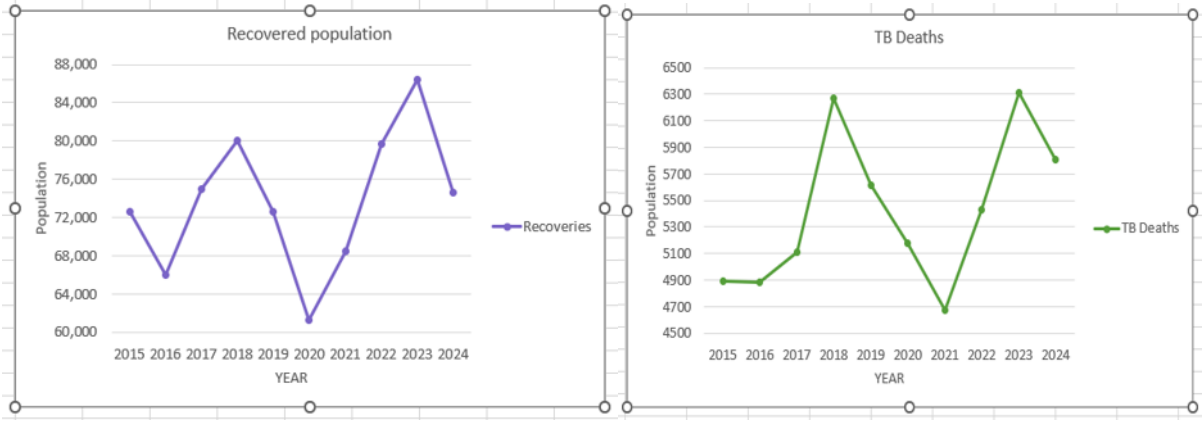
(a) Susceptible Population (b) Vaccinated Population

Figure 2: Susceptible and Vaccinated Population



(a) Active TB Cases (b) Treatment Population

Figure 3: Active TB population and Treatment Population



(a) Recovered Population (b) TB Deaths

Figure 4: Recovered Population and TB Deaths

Figures 2,3 and 4 shows graphs with clear contrasting trends from 2015 to 2024. The susceptible population steadily increases from about 46 million to roughly 54 million, indicating a growing pool of individuals at risk. The vaccinated population also rises overall from just above 3.1 million to about 4.3 million though with minor fluc-tuations around 2017, 2019, and 2023. This suggests a gradual but somewhat inconsistent improvements in vaccination coverage; meanwhile, active TB cases display a consistent downward trend from approximately 175,000 to about 128,000. Despite a slight temporary rise around 2020 and a small uptick in 2023. Graphs of Treatment, recoveries and TB deaths shows greater fluctuations over time with a lot of unpredictable inconsistencies. Together, these patterns suggest that while the population at risk is expanding, increased vaccination and possibly other interventions are contributing to a general reduction in active TB cases over time.

VI. Parameter Values And Sources

Table 2: Descriptions and Numerical Values of the Model Parameters

Parameter	Description	Value	Units	Source
λ	Recruitment rate into susceptible	0.0148	year^{-1}	Modeled
ρ	Vaccine waning rate	0.067, 0.1	year^{-1}	Assumed
α	Rate of vaccination of susceptible	0.5	year^{-1}	Modeled
ω	Adequate contact rate	5	year^{-1}	Modeled
ε	Vaccine efficacy	0.5	year^{-1}	Modeled
β	Progression from latent to active TB	0.02768	year^{-1}	Assumed
δ	Treatment rate for TB persons	0.1	year^{-1}	Modeled
ϕ	Recovery rate of treated persons	0.01	year^{-1}	Modeled
μ	Natural death rate	0.007	year^{-1}	Modeled
σ	TB-induced death rate	0.1	year^{-1}	Modeled

Table 2 presents the key parameters used in the mathematical modeling of tuberculosis transmission and control, together with their descriptions, numerical values, units, and sources. The parameters represent essential demographic processes such as recruitment into the susceptible population and natural mortality. They also explore core epidemiological mechanisms including vaccination coverage and waning immunity, effective contact rates, progression from latent to active tuberculosis, treatment initiation, recovery, and TB-induced mortality. Most parameter values were obtained through modeling and were informed by established theoretical and empirical studies, while some parameters were as-sumed to facilitate the exploration of plausible intervention scenarios. Collectively, these parameters define the structure and dynamic behavior of the model and form the quantitative foundation for the analytical investigations, stability analysis, and bifurcation analysis carried out to assess tuberculosis prevention and control strategies.

VII. Equilibrium Points

Disease Free Equilibrium (DFE)

At the disease–free state, there is no active infection or latent infection:

$$A^* = 0, \quad L^* = 0, \quad T^* = 0, \quad R^* = 0.$$

We set $S = V = 0^{\cdot}$ with $A^* = 0$ and solve:

$$0 = \lambda + \rho V^* - (\mu + \alpha)S^* \quad (1)$$
$$0 = \alpha S^* - (\mu + \rho)V^*. \quad (2)$$

From (2):

$$V^* = \frac{\alpha}{\mu + \rho} S^*.$$

Substitute into (1):

$$(\mu + \alpha)S^* = \lambda + \rho \frac{\alpha}{\mu + \rho} S^*$$

Hence,

$$S^* \left[(\mu + \alpha) - \frac{\rho\alpha}{\mu + \rho}\right] = \lambda,$$

so

$$S^* = \frac{\lambda(\mu + \rho)}{(\mu + \alpha)(\mu + \rho) - \rho\alpha}$$

Then,

$$V^* = \frac{\alpha}{\mu + \rho} S^* = \frac{\alpha\lambda}{(\mu + \alpha)(\mu + \rho) - \rho\alpha}.$$

Thus, the Disease-Free Equilibrium (DFE) is

$$E_0 = (S^*, V^*, L^* = 0, A^* = 0, T^* = 0, R^* = 0)$$

There is no TB when the population is at the disease-free equilibrium (DFE), and the population is made up only of susceptible and vaccinated individuals. Recruitment (λ), vaccination rate (α), vaccine waning (ρ), and natural death (μ) each determine their proportions. Specifically, if $\alpha = 0$ (no vaccination), the vaccinated class vanishes ($V^* = 0$) and the susceptible class equals the natural replacement level ($S^* = {}^{\lambda}/_{\mu}$; if $\rho = 0$(no fading), on the other hand the size of the vaccinated pool rises as vaccination rates increase. Minor infection introductions cease and the equilibrium is stable when $R_0 < 1$; if $R_0 > 1$, the equilibrium is unstable and the disease may continue endemically. The basic reproduction number (R_0) is calculated and analyzed using the DFE, denoted by the notation E_0, as the baseline equilibrium.

Endemic Equilibrium ($A^* > 0$)

If an endemic equilibrium exists, then $A^* > 0$. We first express $L\hat{}*, T\hat{}*, R\hat{}*$ in terms of A^*.

From $\dot{A} = 0$:

$$0 = \beta L^* - (\mu + \sigma + \delta)A^* \quad \Rightarrow$$
$$L^* = \frac{\mu + \sigma + \delta}{\beta} A^*.$$

From$\dot{T} = 0$:

$$T^* = \frac{\delta}{\mu + \phi} A^*$$

From $\dot{R} = 0$

$$R^* = \frac{\phi}{\mu} T^* = \frac{\phi\delta}{\mu(\mu + \phi)} A^*.$$

Now use $\dot{L} = 0$

$$0 = \omega S^* A^* + \omega(1 - \varepsilon)V^* A^* - (\beta + \mu)L^*.$$

Divide by A^* (since $A^* > 0$) and substitute L^*:

$$\omega(S^* + (1 - \varepsilon)V^*) = \frac{(\beta + \mu)(\mu + \sigma + \delta)}{\beta} := K.$$

Thus,

$$S^* + (1 - \varepsilon)V^* = \frac{K}{\omega}. \quad ()$$

Next, use $\dot{S} = 0, \dot{V} = 0$:

$$S^*(\mu + \alpha + \omega A^*) = \lambda + \rho V^*, \quad (1')$$
$$V^*(\mu + \rho + \omega(1 - \varepsilon)A^*) = \alpha S^* \quad (2')$$

From (2′):

$$V^* = \frac{\alpha S^*}{\mu + \rho + \omega(1 - \varepsilon)A^*} \quad (3)$$

Substitute (3) into (1′):

$$S^* = \frac{\lambda(\mu + \alpha + \omega A^*) - \rho\alpha S^*}{\mu + \rho + \omega(1-\varepsilon)A^*}$$

So explicitly:

$$S^* = \frac{\lambda(\mu + \alpha + \omega A^*) - \rho\alpha}{\mu + \rho + \omega(1-\varepsilon)A^*} \quad (4).$$

Now substitute (3) into relation ():

$$S^*\left[1 + \frac{(1-\varepsilon)\alpha}{\mu + \rho + \omega(1-\varepsilon)A^*}\right] = \frac{K}{\omega}. \quad (5)$$

Nonlinear Equation for A^*

Combining (4) and (5), eliminating S^*, yields a single nonlinear equation in A^*:

$$F(A^*) := \frac{\lambda(\mu + \alpha + \omega A^*) - \rho\alpha}{\mu + \rho + \omega(1-\varepsilon)A^*}\left[1 + \frac{(1-\varepsilon)\alpha}{\mu + \rho + \omega(1-\varepsilon)A^*}\right] - \frac{K}{\omega} = 0.$$

Any positive root $A^* > 0$ of $F(A^*) = 0$ yields an endemic equilibrium, with S^*, V^* from (4)–(3), and L^*, T^*, R^* from the linear relations above.

Existence Condition

Since $\lambda > 0$, the disease-free equilibrium E_0 always exists. Whenever the basic reproduction number $R_0 > 1$, which is associated with the effective susceptible pool $S^* + (1-\varepsilon)V^*$at DFE, there is usually an endemic equilibrium with $A^* > 0$. The next-generation method can calculate the exact value R_0, and the existence of a positive solution to $F(A^*) = 0$ is indicated by the condition $R_0 > 1$.

The exact value of R_0 can be calculated, and the existence of a positive solution to $F(A^*) = 0$ is indicated by the condition $R_0 > 1$.

VIII. Basic Reproduction Number (R_0)

Infectious Subsystem

We focus on the infected compartments:

$$L = Latent\ TB,$$
$$A = Active\ TB$$

The system of differential equations for these compartments is:

$$\dot{L} = \omega S_{\text{eff}} A - (\beta + \mu)L,$$
$$\dot{A} = \beta L - (\delta + \mu + \sigma)A,$$

Where;

$$S_{\text{eff}} = S^* + (1-\varepsilon)V^*$$

Define F (New Infections) and V (Transitions)

New infections occur when susceptible individuals come into contact with active TB cases:

$$F_L = \omega S_{\text{eff}} A, \quad F_A = 0.$$

Thus, the new infection matrix is:

$$F = \begin{bmatrix} 0 & \omega S_{\text{eff}} \\ 0 & 0 \end{bmatrix}$$

The transition matrix representing movement between compartments (excluding new infections) is:

$$V_L = (\beta + \mu)L, \quad V_A = -\beta L + (\delta + \mu + \sigma)A.$$

Or in matrix form:

$$V = \begin{bmatrix} \beta + \mu & 0 \\ -\beta & \delta + \mu + \sigma \end{bmatrix}$$

Step 2: Compute V^{-1}

The inverse of the transition matrix V is:

$$V^{-1} = \frac{1}{(\beta + \mu)(\delta + \mu + \sigma)}\begin{bmatrix} \beta + \mu & 0 \\ -\beta & \delta + \mu + \sigma \end{bmatrix}$$

Next Generation Matrix K

The Next Generation Matrix (NGM) is defined as:

$$K = FV^{-1}.$$

Multiplying the matrices F and V^{-1}gives:

$$K = \begin{bmatrix} 0 & \omega S_{\text{eff}} \\ 0 & 0 \end{bmatrix} \frac{1}{(\beta+\mu)(\delta+\mu+\sigma)} \begin{bmatrix} \delta+\mu+\sigma & 0 \\ \beta & \beta+\mu \end{bmatrix}$$

$$= \frac{1}{(\beta+\mu)(\delta+\mu+\sigma)} \begin{bmatrix} \omega S_{\text{eff}}\beta & \omega S_{\text{eff}}(\beta+\mu) \\ 0 & 0 \end{bmatrix}$$

Thus, the Next Generation Matrix is:

$$K = \frac{1}{(\beta+\mu)(\delta+\mu+\sigma)} \begin{bmatrix} \omega S_{\text{eff}}\beta & \omega S_{\text{eff}}(\beta+\mu) \\ 0 & 0 \end{bmatrix}$$

Including the scalar factor yields

$$K = \begin{bmatrix} \dfrac{\omega S_{\text{eff}}\beta}{(\beta+\mu)(\delta+\mu+\sigma)} & \dfrac{\omega S_{\text{eff}}(\beta+\mu)}{(\beta+\mu)(\delta+\mu+\sigma)} \\ 0 & 0 \end{bmatrix} \tag{4.27}$$

Cancelling the common factor (β + μ) gives the simplified NGM

$$K = \begin{bmatrix} \dfrac{\omega S_{\text{eff}}\beta}{\delta+\mu+\sigma} & \dfrac{\omega S_{\text{eff}}}{\delta+\mu+\sigma} \\ 0 & 0 \end{bmatrix} \tag{4.28}$$

Eigenvalue Analysis

The eigenvalues of K satisfy

$$\det(K - \lambda I) = 0. \tag{4.29}$$

Thus,

$$K - \lambda I = \begin{bmatrix} \dfrac{\omega S_{\text{eff}}\beta}{\delta+\mu+\sigma} - \lambda & \dfrac{\omega S_{\text{eff}}}{\delta+\mu+\sigma} \\ 0 & -\lambda \end{bmatrix} \tag{4.30}$$

Since $\det(K - \lambda I)$ is upper triangular, its determinant is

$$\det(K - \lambda I) = \left(\frac{\omega S_{\text{eff}}\beta}{\delta+\mu+\sigma} - \lambda\right)(-\lambda) \tag{4.31}$$

Solving $\det(K - \lambda I) = 0$ gives the eigenvalues:

$$\lambda_1 = \frac{\omega S_{\text{eff}}\beta}{(\beta+\mu)(\delta+\mu+\sigma)}, \quad \lambda_2 = 0 \tag{4.32}$$

Hence, the spectral radius of the NGM, which defines the basic reproduction number, is

$$R_0 = \rho(K) = \frac{\omega S_{\text{eff}}\beta}{(\beta+\mu)(\delta+\mu+\sigma)}. \tag{4.33}$$

By definition, the basic reproduction number is the largest eigenvalue:

$$R_0 = \frac{\omega S_{\text{eff}}\beta}{(\beta+\mu)(\delta+\mu+\sigma)}.$$

Interpretation

R_0 represents the average number of secondary TB cases generated by a single infectious individual in a population that is mostly susceptible (adjusted for vaccination). If $R_0 < 1$, the infection will eventually die out; if $R_0 > 1$, TB can invade and persist. This expression clearly shows the influence of the effective susceptible population (S_{eff}), contact rate (ω), progression rate from latent to active TB (β), treatment rate (δ), and mortality (μ, σ) on TB transmission potential.

Plug in Fitted Values

$$R_0 \approx 1.73.$$

Epidemiological Interpretation of R_0 in TB Context

The expected number of new TB cases produced by a single infectious individual introduced into a perfectly susceptible (but vaccinated) population at equilibrium is represented by the basic reproduction number $R_0 \approx 1.73$. Since $R_0 > 1$, the disease can survive and spread across the community because, on average, each case of active TB results in more than one secondary case. According to epidemiology, this threshold result suggests that TB transmission persists and cannot be eradicated with the existing levels of vaccination coverage, treatment, and natural recovery rates.

Interventions that lower R_0 below unity are therefore required for effective control; these could include increased vaccine coverage and efficacy, better treatment rates, or transmission reductions through preventive efforts. Infection introductions will not cease until $R_0 < 1$, which will stabilize the disease-free equilibrium. This value draws into focus inadequacies in the current control measures from a policy view. The expression for R_0 indicates that the effective pool of susceptibles (S_{eff}) and transmission intensity (ω) are the main drivers of

new infections, whereas progression rates from latent to active TB (β) also play a significant role. Conversely, self-recovery (ϕ), treatment initiation (δ), and natural mortality (μ) all work to lower R_0.

IX. Stability Analysis

We consider the system

$$\dot{S} = \lambda + \rho V - \omega SA - (\mu + \alpha)S, \tag{4.34}$$
$$\dot{V} = \alpha S - (1 - \varepsilon)VA - (\mu + \rho)V, \tag{4.35}$$
$$\dot{L} = \omega SA + (1 - \varepsilon)VA - (\beta + \mu)L, \tag{4.36}$$
$$\dot{A} = \beta L - (\mu + \sigma + \delta)A, \tag{4.37}$$
$$\dot{T} = \delta A - (\mu + \phi)T, \tag{4.38}$$
$$\dot{R} = \phi T - \mu R. \tag{4.39}$$

All parameters $\lambda, \rho, \omega, \alpha, \mu, \varepsilon, \beta, \sigma, \delta, \phi$ are strictly positive. Equilibrium values are denoted by a star, e.g., $S^*, V^*,$

Stability Analysis of the Disease-Free Equilibrium (DFE)

The disease-free equilibrium (DFE) of the SVLATR model is given by

$$E_0 = (S^*, V^*, L^* = 0, A^* = 0, T^* = 0, R^* = 0),$$

where

$$S^* = \frac{\lambda(\mu + \rho)}{(\mu + \alpha)(\mu + \rho) - \rho\alpha}, V^* = \frac{\alpha\lambda}{(\mu + \alpha)(\mu + \rho) - \rho\alpha}.$$

Using the parameter values in Table 4.2:
we obtain

$$S^* \approx 0.373, \quad V^* \approx 1.742$$

Jacobian Matrix of the Infectious Subsystem

Focusing on the infectious compartments (L, A), the Jacobian matrix evaluated at the DFE is

$$J_{\text{infectious}} = \begin{bmatrix} -(\beta + \mu) & \omega S_{\text{eff}} \\ \beta & -(\delta + \mu + \sigma) \end{bmatrix}$$

where

$$S_{\text{eff}} = S^* + (1 - \varepsilon)V^*.$$

Eigenvalues of the Jacobian Matrix at the DFE

The Jacobian matrix evaluated at the disease–free equilibrium (DFE) is

$$J_{\text{infectious}} = \begin{bmatrix} -(\beta + \mu) & \omega S_{\text{eff}} \\ \beta & -(\delta + \mu + \sigma) \end{bmatrix} \tag{4.40}$$

The eigenvalues λ are obtained from the characteristic equation

$$\det(J_{\text{infectious}} - \lambda I) = 0. \tag{4.41}$$

Thus,

$$J_{\text{infectious}} - \lambda I = \begin{bmatrix} -(\beta + \mu) & \omega S_{\text{eff}} \\ \beta & -(\delta + \mu + \sigma) \end{bmatrix} \tag{4.42}$$

Computing the determinant gives

$$\begin{aligned} \det(J_{\text{infectious}} - \lambda I) &= [-(\beta + \mu) - \lambda][-(\delta + \mu + \sigma) - \lambda] - \beta\,\omega S_{\text{eff}} \\ &= (\lambda + \beta + \mu)(\lambda + \delta + \mu + \sigma) - \beta\,\omega S_{\text{eff}} \end{aligned} \tag{4.43}$$

Hence, the characteristic polynomial is

$$\lambda^2 + (\beta + \delta + 2\mu + \sigma)\lambda + (\beta + \mu)(\delta + \mu + \sigma) - \beta\,\omega S_{\text{eff}} = 0 \tag{4.44}$$

Solving the quadratic equation yields the eigenvalues

$$\lambda_{1,2} = \frac{-(\beta + \delta + 2\mu + \sigma) \pm \sqrt{(\beta + \delta + 2\mu + \sigma)^2 - 4[(\beta + \mu)(\delta + \mu + \sigma) - \beta\,\omega S_{\text{eff}}]}}{2} \tag{4.45}$$

Using $\varepsilon = 0.5$, we compute

$$S_{\text{eff}} = 0.187 + (1 - 0.5)(0.813) = 0.187 + 0.4065 = 0.5935.$$

Numerical Jacobian Matrix

Substituting the remaining parameter values

$$\beta = 0.02768, \quad \mu = 0.007, \quad \delta = 0.1, \quad \sigma = 0.1, \quad \omega = 5,$$

the Jacobian becomes

$$J_{\text{infectious}} = \begin{bmatrix} -0.04248 & 2.9675 \\ 0.02768 & -0.2148 \end{bmatrix}$$

Eigenvalue Computation

The characteristic equation is

$$\det(J - \lambda I) = 0.$$

which gives

$$\lambda^2 + 0.2573\lambda - 0.0731 = 0.$$

Solving, the eigenvalues are

$$\lambda_1 \approx 0.198, \quad \lambda_2 \approx -0.370.$$

Stability Conclusion

Since one eigenvalue is positive and the other is negative, the disease-free equilibrium is unstable. This result is consistent with the basic reproduction number

$$R_0 = 1.73 > 1$$

which confirms that tuberculosis can successfully invade the population in the absence of effective control measures.

Therefore, the disease-free equilibrium of the $SVLATR$ model is locally unstable, highlighting the necessity of implementing prevention and treatment strategies to reduce transmission.

Summary Table

Table 3: Stability of the Disease-Free Equilibrium based on R_0

Condition	Stability of DFE
$R_0 < 1$	Locally asymptotically stable
$R_0 = 1$	Critically stable (neutral)
$R_0 > 1$	Unstable

Local Stability of the DFE

Stability of E_0

According to the standard next-generation theorem, E_0 is unstable if $R_0 > 1$and locally asymptotically stable if $R_0 < 1$. (The remaining eigenvalues, which are related to the (S, V) and (T, R) blocks, form linear, dissipative subsystems at the DFE, therefore they have negative real parts regardless of infection.

Stability Analysis of the Endemic Equilibrium (EE)

An endemic equilibrium (EE) exists when tuberculosis persists in the population, that is, $A^* > 0$.

From the steady–state conditions of the $SVLATR$ model, the remaining compartments can be expressed in terms of, A^* as

$$L^* = \frac{\mu + \sigma + \delta}{\beta} A^*, \qquad T^* = \frac{\delta}{\mu + \phi} A^*, \qquad R^* = \frac{\phi\delta}{\mu(\mu + \phi)} A^*.$$

The susceptible and vaccinated compartments satisfy

$$0 = \lambda + \rho V^* - (\mu + \alpha + \omega A^*) S^*$$
$$0 = \alpha S^* - (\mu + \rho) V^*.$$

From the second equation,

$$V^* = \frac{\alpha}{\mu + \rho} S^*.$$

Jacobian Matrix at the Endemic Equilibrium

The Jacobian matrix evaluated at the endemic equilibrium is given by

$$J = \begin{bmatrix} -(\mu + \alpha + \omega A^*) & \rho & 0 & -\omega S^* & 0 & 0 \\ \alpha & -(\mu + \rho) & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\beta + \mu) & \omega S^* & 0 & 0 \\ 0 & 0 & \beta & -(\delta + \mu + \sigma) & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\mu + \phi) & 0 \\ 0 & 0 & 0 & 0 & \phi & -\mu \end{bmatrix}$$

This matrix is block triangular; hence, its eigenvalues consist of eigenvalues of the diagonal blocks.

Eigenvalues of the (S, V) block

$$J_{SV} = \begin{bmatrix} -(\mu + \alpha + \omega A^*) & \rho \\ \alpha & -(\mu + \rho) \end{bmatrix}$$

The characteristic equation is

$$\lambda^2 + \lambda(2\mu + \alpha + \rho + \omega A^*) + [(\mu + \alpha + \omega A^*)(\mu + \rho) - \alpha\rho] = 0.$$

Thus, the eigenvalues are

$$\lambda_{1,2} = \frac{-(2\mu + \alpha + \rho + \omega A^*) \mp \sqrt{(2\mu + \alpha + \omega A^*)^2 - 4\,[(\mu + \alpha + \omega A^*)(\mu + \rho) - \alpha\rho]}}{2}$$

Since all parameters are positive, both eigenvalues satisfy

$$\lambda_{\{1,2\}} < 0.$$

Eigenvalues of the infectious (L, A) block

$$J_{LA} = \begin{bmatrix} -(\beta + \mu) & \omega S^* \\ \beta & -(\delta + \mu + \sigma) \end{bmatrix}$$

The characteristic equation is

$$\lambda^2 + \lambda(\beta + \delta + 2\mu + \sigma) + [(\beta + \mu)(\delta + \mu + \sigma) - \beta\omega S^*] = 0$$

Hence, the eigenvalues are

$$\lambda_{3,4} = \frac{-(\beta + \delta + 2\mu + \sigma) \pm \sqrt{(\beta + \delta + 2\mu + \sigma)^2 - 4[(\beta + \mu)(\delta + \mu + \sigma) - \beta\omega S^*]}}{2}.$$

Define the basic reproduction number as

$$R_0 = \frac{\beta\omega S^*}{(\beta + \mu)(\delta + \mu + \sigma)}.$$

Then,

$\lambda_{3,4} < 0$ if and only if $R_0 < 1$

Eigenvalues of the (T, R) block

$$J_{TR} = \begin{bmatrix} -(\mu + \phi) & 0 \\ \phi & -\mu \end{bmatrix}$$

The eigenvalues are

$$\lambda_5 = -(\mu + \phi), \quad \lambda_6 = -\mu.$$

which are always negative.

Eigenvalues from the Non-Infectious Subsystems

From the diagonal entries, the following eigenvalues are obtained:

$$\lambda_1 = -(\mu + \rho), \qquad \lambda_2 = -(\mu + \phi), \qquad \lambda_3 = -\mu$$

Using parameter values in Table 2:

$$\lambda_1 = -0.107, \quad \lambda_2 = -0.017, \quad \lambda_3 = -0.007,$$

which are all strictly negative.

Infected Subsystem Eigenvalues

The infected subsystem (L, A) is described by the 2 × 2 matrix

$$J_{infected} = \begin{bmatrix} -(\beta + \mu) & \omega S^* \\ \beta & -(\delta + \mu + \sigma) \end{bmatrix}$$

The characteristic equation is

$$\lambda^2 + (\beta + \delta + 2\mu + \sigma)\lambda + (\beta + \mu)(\delta + \mu + \sigma) - \beta\omega S^* = 0$$

Substituting parameter values

$$\beta = 0.02768, \quad \mu = 0.007, \quad \delta = 0.1, \quad \sigma = 0.1,$$

Yields

$$\text{Trace} = -(\beta + \mu + \delta + \mu + \sigma) = -0.2417 < 0.$$

At the endemic equilibrium, the effective reproduction number satisfies

$$R_0^* = \frac{\beta\omega S^*}{(\beta + \mu)(\delta + \mu + \sigma)} > 1,$$

which implies

$$(\beta + \mu)(\delta + \mu + \sigma) - \beta\omega S^* > 0.$$

Hence, the determinant of J_{infected} is positive:

$$\det(J_{\text{infected}}) > 0.$$

By the Routh–Hurwitz criteria, both eigenvalues of the infected subsystem satisfy

$$\text{Re}(\lambda_4), \text{Re}(\lambda_5) < 0.$$

X. Discussion

Pursuant to this study, the basic reproduction number, or R_0., should be lowered to a value less than unity in order to develop effective tuberculosis (TB) control programs. It takes a variety of tactics to do this. First, the effective susceptible population, (S_{eff}), can be significantly reduced by increasing vaccination

coverage (α) and enhancing vaccine effectiveness (ε). Second, in order to shorten the infectious period and reduce transmission, treatment programs must be reinforced to raise the rate of transition from active TB to treatment (δ). Third, promoting treatment completion and adherence improves recovery outcomes (ϕ), which lowers the reservoir of infection. Lastly, the reduction of exposure and subsequent transmission is greatly aided by preventive measures such as active case detection through screening, infection control measures, and treatments targeted at lowering contact rates (ω).

XI. Conclusion

Stability Classification of the Endemic Equilibrium

Since all eigenvalues of the Jacobian matrix evaluated at the endemic equilibrium have strictly negative real parts, the endemic equilibrium is locally asymptotically stable.

Conclusion on Stability

The endemic equilibrium exists only if $R_0 > 1$. The endemic equilibrium is locally asymptotically stable, meaning small perturbations will return to EE. Persistent infection is maintained in the population at equilibrium.

Summary Table

Table 4: Stability of Disease-Free and Endemic Equilibria

Equilibrium	Condition	Stability Type
Disease-Free Equilibrium (DFE)	$R_0 < 1$	Locally asymptotically stable
DFE	$R_0 > 1$	Unstable
Endemic Equilibrium (EE)	$R_0 > 1, A^* > 0$	Locally asymptotically stable

Jacobian and Local Stability Summary

Let $X = (S, V, L, A, T, R)^{>}$. The Jacobian $J(X)$ has the block form

$$J = \begin{bmatrix} J_{SV\,LA} & 0 \\ J_{21} & J_{TR} \end{bmatrix}$$

where the infection–free treatment block does not feed back to (S, V, L, A):

$$J_{TR} = \begin{bmatrix} -(\mu+\phi) & 0 \\ \phi & -\mu \end{bmatrix}, \qquad J_{21} = \begin{bmatrix} 0 & 0 & 0 & \delta \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Hence the spectrum of J is the union of the spectra of $J_{SV\,LA}$ and J_{TR}. Since J_{TR} has strictly negative real–part eigenvalues, local stability is determined by $J_{SV\,LA}$:

$$J_{SV\,LA} = \begin{bmatrix} -(\mu+\alpha)-\omega A & \rho & 0 & -\omega S \\ \alpha & -(\mu+\rho)-(1-\varepsilon)A & 0 & -(1-\varepsilon)V \\ \omega A & (1-\varepsilon)A & -(\beta+\mu) & \omega S+(1-\varepsilon)V \\ 0 & 0 & \beta & -(\delta+\mu+\sigma) \end{bmatrix}$$

DFE

At E_0 we have $A = 0$ and the infection block reduces, via the next–generation method, to the threshold R_0 given above. Therefore: E_0 is locally asymptotically stable if $R_0 < 1$; E_0 is unstable (one eigenvalue > 0) if $R_0 > 1$.

Endemic Equilibrium

Assume $R_0 > 1$ and let E^*be a solution of (??). Then $J_{SV\,LA}(E^*)$ is a Metzler, irreducible matrix of the standard SEIR–type bilinear model. Under these conditions (no re-infection from R or T into S, V , mass–action incidence, and positivity of parameters), the endemic equilibrium E^* is locally asymptotically stable.

TB Interpretation

The term $\omega S^* + (1-\varepsilon)V^*$is the effective susceptible pool at equilibrium; vaccination efficacy ϵ and movement into V both reduce it.

R_0 increases with transmission ω and decreases with progression/treatment clearance $(\beta+\mu)$ and $(\delta+\mu+\sigma)$

Bringing R_0 below unity (by increasing $\varepsilon, \alpha, \delta$, or reducing ω) stabilizes the DFE; otherwise the system settles at the endemic E^*.

Recommendations for Future Research

Future research should build on this work by addressing several limitations of the current study and extending the modeling framework to capture additional complexities of tuberculosis transmission. While the

SVLATR model successfully represents core TB dynamics and intervention effects, it assumes homogeneous mixing and does not explicitly account for heterogeneous contact patterns across age groups, social settings, or high-risk populations, which may influence transmission intensity. Further studies should incorporate structured contact networks, spatial heterogeneity, and setting-specific transmission to improve realism. In addition, drug resistance was not explicitly modeled; extending the framework to include drug-sensitive and drug-resistant TB strains, treatment failure, and resistance amplification would provide more detailed insights for control planning. The current analysis also relied on aggregated surveillance data, which may mask under-reporting and diagnostic delays; future research should prioritize high-resolution, longitudinal data, including treatment adherence, relapse, and reinfection information.

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Conflict of Interest

No conflict of interest has been declared by any of the authors on the publication process.

Data Availability

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Consent to Participate

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