

A Compartmental Model of Tuberculosis Incorporating Latent, Subclinical, and Active States with Detection, Diagnosis and Treatment Success-Failure Dynamics

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ABSTRACT

This study analyzes the dynamics of a tuberculosis (TB) model with latent, subclinical, and active stages, incorporating detection, diagnosis, and treatment success-failure dynamics. Results show that when the basic reproduction number $R_0 < 1$, the disease cannot invade, and thus the disease-free equilibrium point is stable. When $R_0 > 1$, transmission persists and spreads, resulting in an unstable disease-free equilibrium point. Optimal control analysis identifies improved treatment u_3 as the most effective strategy, remaining at high levels across different cost scenarios. Early detection u_1 and improved diagnosis u_2 provide additional benefits but are applied at lower levels, especially as costs increase. Simulation results further show that combining controls, particularly those including treatment, leads to greater reduction in infection, increased recovery, and better preservation of the total population. These findings highlight that in high-transmission settings, prioritizing treatment alongside targeted detection and diagnosis provides an effective and cost-efficient approach to reducing TB burden.

Keywords - basic reproduction number, compartmental model, optimal control, subclinical tuberculosis, tuberculosis treatment

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I. INTRODUCTION

Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains a leading global infectious disease despite decades of progress in diagnosis, treatment, and control. Transmission occurs primarily via airborne droplets produced by people with pulmonary TB, making the disease highly transmissible in close-contact and congregate settings. Persistent delays in diagnosis, interruptions in treatment, and a substantial pool of undiagnosed infections sustain transmission and impede elimination efforts [1], [2].

Two often underappreciated components of the TB epidemic are latent TB infection (LTBI) and subclinical TB. LTBI denotes persistent, viable infection without clinical disease or infectiousness; globally, roughly one-quarter of people are estimated to harbor LTBI [3], [4]. Subclinical TB refers to bacteriologically or radiologically confirmed disease in individuals who do not report classical TB symptoms. This stage is epidemiologically significant because symptom-

based screening can miss cases that still contribute to transmission [5], [6], [7].

Mathematical modelling serves as an essential tool for studying transmission patterns and evaluating control strategies. Existing models have examined latent infection, active disease, diagnosis, quarantine, treatment and asymptomatic/subclinical stages [8], [9], [10], [11], [12]. However, models that simultaneously incorporate latent, subclinical and active infectious stages together with detection, diagnosis, and treatment success-failure dynamics remain important for evaluating integrated TB control strategies.

This paper formulates and analyzes a deterministic compartmental TB model. The objectives are to establish well-posedness and boundedness, compute the basic reproduction number, analyze the local stability of the disease-free equilibrium, and formulate an optimal-control problem involving early detection, improved diagnosis, and improved treatment success.

II. MODEL FORMULATION

The proposed model incorporates latent infection, subclinical and active infectious TB, detection and diagnosis processes, and treatment outcomes. The total population at time t , denoted by $N(t)$, is partitioned into seven classes: susceptible $S(t)$, latent $L(t)$, subclinical infectious $I_S(t)$, active infectious $I_A(t)$, latent under treatment $T_L(t)$, infectious under treatment $T_I(t)$, and recovered $R(t)$. The total infectious population is $I(t) = I_S(t) + I_A(t)$.

Susceptible individuals are recruited at rate Λ , and every compartment experiences natural death at rate μ . Susceptible individuals acquire infection through contact with subclinical infectious individuals I_S and active infectious individuals I_A at transmission rates β_1 and β_2 , respectively. Latent individuals progress to the subclinical infectious class at rate k_1 , while subclinical infectious individuals progress to the active infectious class at rate k_2 . Detection moves latent individuals to T_L at rate σ_1 and subclinical infectious individuals to T_I at rate σ_2 . Diagnosis moves active infectious individuals to T_I at rate σ_3 . Treatment successes move individuals from T_L and T_I to R , while treatment failures return them to infected compartments.

The model is governed by the following system of ordinary differential equations:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \left(\beta_1 \frac{I_S}{N} + \beta_2 \frac{I_A}{N}\right) S - \mu S, \\ \frac{dL}{dt} = \left(\beta_1 \frac{I_S}{N} + \beta_2 \frac{I_A}{N}\right) S - (k_1 + \sigma_1 + \mu)L + (1 - \gamma_1)\delta_1 T_L, \\ \frac{dI_S}{dt} = k_1 L - (k_2 + \sigma_2 + d_1 + \mu)I_S + (1 - \gamma_2)\alpha\delta_2 T_I, \\ \frac{dI_A}{dt} = k_2 I_S - (\sigma_3 + d_2 + \mu)I_A + (1 - \gamma_2)(1 - \alpha)\delta_2 T_I, \\ \frac{dT_L}{dt} = \sigma_1 L - (\delta_1 + \mu)T_L, \\ \frac{dT_I}{dt} = \sigma_2 I_S + \sigma_3 I_A - (\delta_2 + \mu)T_I, \\ \frac{dR}{dt} = \gamma_1 \delta_1 T_L + \gamma_2 \delta_2 T_I - \mu R. \end{cases}$$

The biologically feasible region consists of all nonnegative state variables satisfying $0 < N \leq \Lambda/\mu$.

Table 1 gives the parameter descriptions and values used in the numerical simulations.

Parameter	Description	Value
Λ	Recruitment rate	793 persons/year [13]
μ	Natural death rate	0.0143 per year [14]
β_1	Transmission rate from I_S	2.00 per year, estimated
β_2	Transmission rate from I_A	3.00 per year [15]
k_1	Progression rate from L to I_S	0.25 per year [16]
k_2	Progression rate from I_S to I_A	0.69 per year [16]
σ_1	Detection rate of L leading to treatment initiation	0.40 per year [17]
σ_2	Detection rate of I_S leading to treatment initiation	0.80 per year [17]
σ_3	Diagnosis rate of I_A leading to treatment initiation	0.70 per year [17]
δ_1	Treatment completion rate for T_L	0.06 per year [18]
δ_2	Treatment completion rate for T_I	1.50 per year [18]
γ_1	Treatment success fraction for T_L	0.50 [19]
γ_2	Treatment success fraction for T_I	0.2906 [19]

Parameter	Description	Value
α	Fraction of T_I failures returning to I_S	0.20 [17]
d_1	Disease-induced death rate for I_S	0.365 per year [18]
d_2	Disease-induced death rate for I_A	0.22 per year [18]

Table 1. Description, values, and references of model parameters.

III. MODEL ANALYSIS

The model is epidemiologically meaningful on Ω because all state variables remain nonnegative and bounded for nonnegative initial conditions. In particular, summing the equations gives

$$\frac{dN}{dt} = \Lambda - \mu N - d_1 I_S - d_2 I_A \leq \Lambda - \mu N,$$

which implies $0 < N(t) \leq \Lambda/\mu$ for solutions initiated in the feasible region. Therefore, the model is positively invariant and bounded in Ω .

The disease-free equilibrium is

$$P_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0 \right).$$

Using the next-generation matrix approach [20], [21], the basic reproduction number is

$$R_0 = \frac{k_1[\beta_1(\mu + \sigma_3 + d_2) + \beta_2 k_2]}{(k_1 + \mu + \sigma_1)(d_1 + k_2 + \mu + \sigma_2)(d_2 + \mu + \sigma_3)}.$$

The disease-free equilibrium P_0 is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. This threshold result means that reducing transmission, increasing detection and diagnosis, or improving treatment-related transitions can lower the capacity of TB to invade the population.

IV. OPTIMAL CONTROL PROBLEM

Three time-dependent controls are incorporated to mitigate TB transmission and improve treatment

The control objective is to minimize infected compartments and intervention costs. The objective functional is

$$J(u_1, u_2, u_3) = \int_{t_0}^{t_f} \left(L(t) + I_S(t) + I_A(t) + \sum_{i=1}^3 \frac{1}{2} C_i u_i^2(t) \right) dt,$$

where $C_i > 0$ are implementation-cost weights. By Pontryagin's Minimum Principle [22], optimal controls exist over the admissible set

$$\Gamma = \{(u_1, u_2, u_3): 0 \leq u_i(t) \leq 1, u_i \in \mathcal{L}^2(t_0, t_f), i = 1, 2, 3\}.$$

The optimality system yields bounded control characterizations obtained by setting the Hamiltonian derivatives with respect to the controls equal to zero and then projecting the resulting expressions onto the admissible interval $[0, 1]$. These

outcomes. The early-detection control u_1 represents active case finding, intensified community screening, contact tracing and mobile health campaigns. The improved-diagnosis control u_2 represents faster and more accurate TB detection through better facilities, reliable laboratory tests and trained personnel. The treatment-success control u_3 represents strengthened follow-up, adherence counselling, reliable drug supply and reduction of barriers to treatment completion. For all $t \geq 0$, the controls satisfy $0 \leq u_i(t) \leq 1$.

projected controls balance reductions in disease burden against the relative costs of intervention.

V. NUMERICAL SIMULATIONS AND DISCUSSION

Numerical simulations were performed over a 30-year horizon. The results support the analytical threshold behavior of the model. When

$R_0 < 1$, the infected compartments L , I_S and I_A eventually decrease toward zero, while treatment compartments also diminish because they depend on the presence of infection. This confirms the local asymptotic stability of the disease-free equilibrium and the positivity and boundedness of solutions.

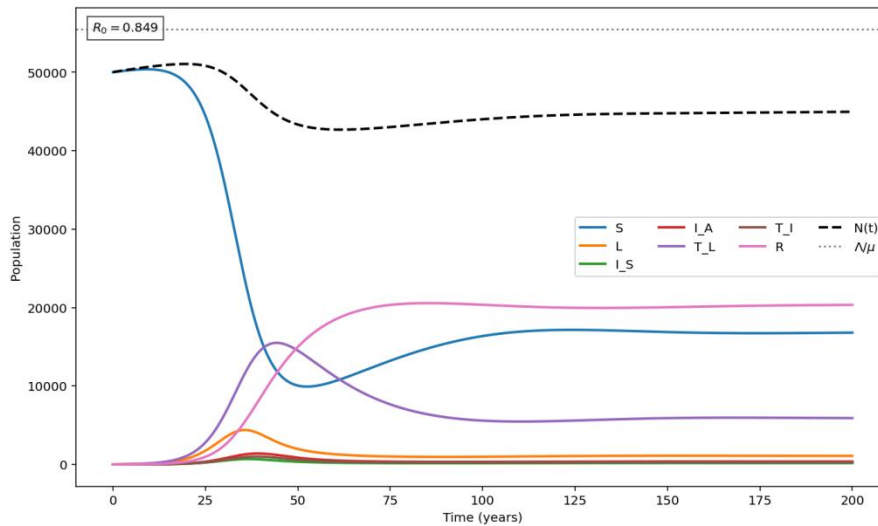


Figure 1. Stability of the disease-free equilibrium with positive and bounded solutions.

Under optimal control, treatment improvement u_3 remains close to its upper bound for most of the simulation horizon, especially in low- and moderate-cost cases. Early detection u_1 and improved diagnosis u_2 are used more moderately and tend to decrease earlier as implementation cost increases. This indicates that treatment improvement is the most influential and cost-effective control in the simulated high-transmission setting.

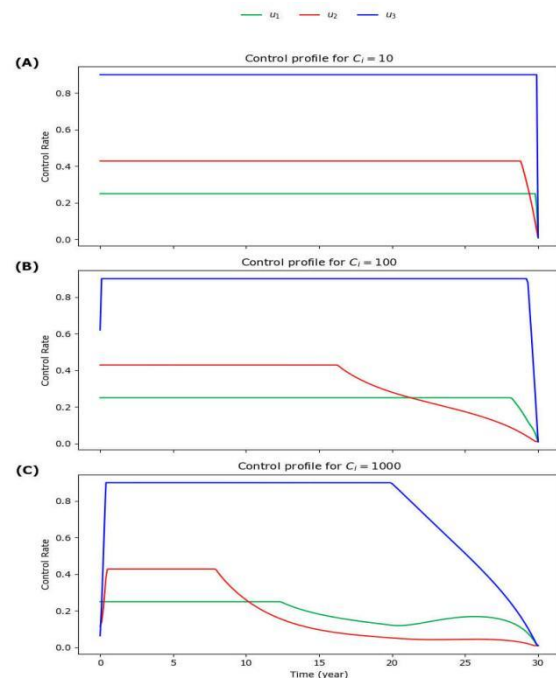


Figure 2. Optimal controls under three cost scenarios.

At $t = 30$ years, the total infected population drops from 1484 with no control to 54, 96 and 399 under low-, moderate- and high-cost control scenarios, respectively. The reduction is strongest under low-cost implementation, but all full-control scenarios substantially reduce infected populations.

At $t = 30$ years	No control	$C_i = 10$	$C_i = 10^2$	$C_i = 10^3$
L	971	42	78	298
I_S	160	5	8	41
I_A	353	7	10	60
Total	1484	54	96	399

Table 2. Values of infected compartments at $t = 30$ years for different control-cost scenarios.

Pairwise-control simulations show that combinations including treatment improvement outperform the combination of early detection and improved diagnosis alone. Under low-cost paired interventions, the total infected population at year 30 is 1093 for (u_1, u_2) , 65 for (u_1, u_3) , and 68 for (u_2, u_3) .

At $t = 30$ years	u_1 and u_2	u_1 and u_3	u_2 and u_3
L	748	49	52
I_S	116	6	7
I_A	229	10	9
Total	1093	65	68

Table 3. Comparison of infected compartment values at $t = 30$ years for low-cost pairwise control combinations.

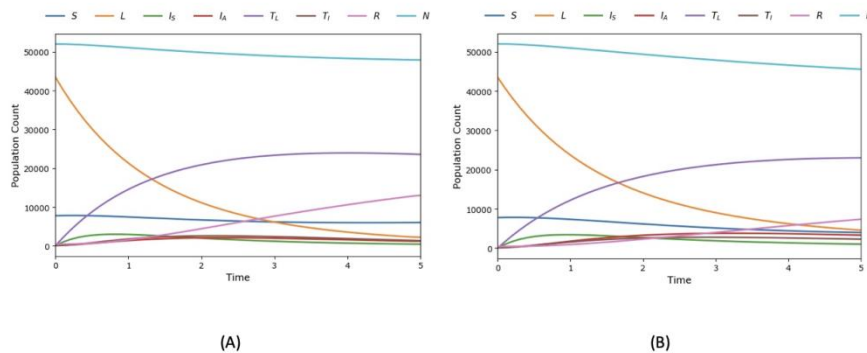


Figure 3. Dynamics of the system with and without controls at $t = 5$.

Overall, the use of control reduces infection, increases recovery, and better preserves the total population. These results emphasize the importance of strategies that improve treatment completion while being supported by early detection and improved diagnosis.

VI. CONCLUSION AND RECOMMENDATIONS

This study developed and analyzed a TB compartmental model incorporating latent, subclinical and active infectious stages, detection, diagnosis, and treatment success–failure dynamics. The model is mathematically well posed in a positively invariant and bounded feasible region. The disease-free equilibrium is locally

asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

The optimal-control analysis indicates that treatment improvement is the most effective strategy, while early detection and improved diagnosis provide additional benefits, particularly when implementation costs are low to moderate. In high-transmission settings, combined control measures that prioritize treatment and are supported by detection and diagnosis are essential for reducing TB burden in a cost-efficient manner.

Future studies may include local stability analysis of the endemic equilibrium, bifurcation analysis, and sensitivity analysis.

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