MATHEMATICAL MODELLING OF TUBERCULOSIS TRANSMISSION DYNAMICS

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Abstract
In this study, a mathematical modeling of Tuberculosis (TB) infection was developed by incorporating vaccination, isolation and treatment. The Basic reproductive number was computed using the next generation matrix method. Analysis of the model at disease free equilibrium state shows that whenever the basic reproductive number is less than unity at the disease free equilibrium state and locally and globally asymptotically stable whenever the basic reproductive number is greater than unity. The equilibrium states of the model were analysed, including the Disease-Free and Endemic Equilibria, were determined and analyzed for stability using the basic reproduction number (R_0) derived via the next generation matrix method. Numerical simulations were performed using MAPLE software, our results shows that increasing vaccination coverage, maintaining effective isolation of infectious individuals, and improving treatment rates significantly reduce the prevalence of TB over time.

Keywords: Local stability, Mathematical model, Reproduction number, Simulation, Tuberculosis and treatment

Introduction

Tuberculosis (TB) remains one of the world's most pressing public health challenges. It is an airborne infectious disease caused by *Mycobacterium tuberculosis*, which primarily attacks the lungs (pulmonary TB) but can also affect other organs in extrapulmonary forms (WHO, 2024). Transmission occurs when individuals with active pulmonary TB release tiny infectious droplets into the air through coughing, sneezing, or even speaking. The likelihood of transmission depends on several factors, including the duration and closeness of contact, the infectiousness of the source case, and the surrounding environment (WHO, 2023; CDC, 2022).

In most cases, the immune system can suppress the bacteria after initial exposure, resulting in latent TB infection. However, when immunity is weakened—such as through HIV co-infection or other illnesses—latent TB may progress into active disease, which is the only stage capable of spreading infection. Despite major advances in public health, TB continues to rank among the leading causes of death globally, particularly in regions of sub-Saharan Africa where HIV prevalence is high (WHO, 2025).

Tuberculosis is a potentially severe infectious disease primarily affecting the lungs, caused by Mycobacterium tuberculosis. It spreads through the air when individuals with active pulmonary TB expel bacteria, typically through coughing (Ryckman *et al.,* 2022). While TB most commonly affects the lungs (pulmonary TB), it can also impact other parts of the body (extrapulmonary TB). The infection can remain latent for years before progressing to active disease, especially in people with weakened immune systems (Ismail *et al.,* 2023).

The symptoms of active TB disease can vary depending on which area of the body is affected. Classic symptoms of pulmonary TB include a persistent cough that lasts more than three weeks, chest pain, and coughing up blood or sputum. Systemic symptoms are also common, including fever, night sweats, fatigue, loss of appetite, and unexplained weight loss. In extrapulmonary TB, symptoms are often site-specific and may include joint pain in skeletal TB or neurological symptoms in TB meningitis (Okunoghea *et al.,* 2021).
Diagnosing TB involves a comprehensive approach. Initially, healthcare providers consider the

patient's medical history, including potential exposure to TB and risk factors such as HIV infection or recent travel to high-prevalence areas (Maulina *et al.,* 2024). A physical examination is performed, focusing on the lungs but also checking for signs of extrapulmonary involvement. Chest X-rays are typically ordered to look for characteristic lung abnormalities. However, imaging alone is not sufficient for a definitive diagnosis (Kalyan *et al.,* 2021).

Laboratory tests play a crucial role in confirming TB diagnosis. Rapid molecular tests, such as the Xpert MTB/RIF Ultra and Truenat assays, have revolutionized TB diagnosis by detecting the presence of M. tuberculosis DNA and potential drug resistance within hours (Cohen *et al.,* 2007). Mycobacterial culture remains the gold standard for diagnosis, but it can take several weeks to yield results. The tuberculin skin test (TST) or interferon-gamma release assay (IGRA) can detect latent TB infection by measuring the immune system's response to TB antigens.

In cases of suspected extrapulmonary TB, additional tests may be necessary. These can include biopsies of affected tissues, lumbar punctures for cerebrospinal fluid analysis in suspected TB meningitis, or specialized imaging studies like CT scans or MRIs. Diagnosing extrapulmonary TB can be challenging due to the wide variety of presentations and the often paucibacillary nature of these infections (Singh & Chatterjee, 2021).

The treatment of active TB disease is complex and prolonged, aimed at curing the patient, preventing the development of drug resistance, and reducing transmission. For drug-susceptible TB, the standard first-line treatment regimen consists of a two-month intensive phase followed by a four-month continuation phase (Starshinova *et al.,* 2024). The intensive phase includes four drugs: isoniazid, rifampicin, ethambutol, and pyrazinamide. This is followed by the continuation phase with isoniazid and rifampicin. This regimen has been shown to be highly effective when adhered to properly (Somma *et al.,* 2017).

Model Formulation

This section outlines the development of a mathematical model to aid the analysis of the transmission dynamics of tuberculosis. Using a compartmental approach, a model is formulated whose total host populace can be partitioned into seven compartments in accordance to their epidemiological state. The classes are defined as the Susceptible S(t), Vaccinated V(t), Latent L(t), Infected I(t), Isolated $I_s(t)$, Treated T(t) and Recovered R(t) individuals respectively at the time variable $t \geq 0$. The model incorporates vaccination, isolation and treatment as control strategies to explore and analyze their impact on the outbreak.

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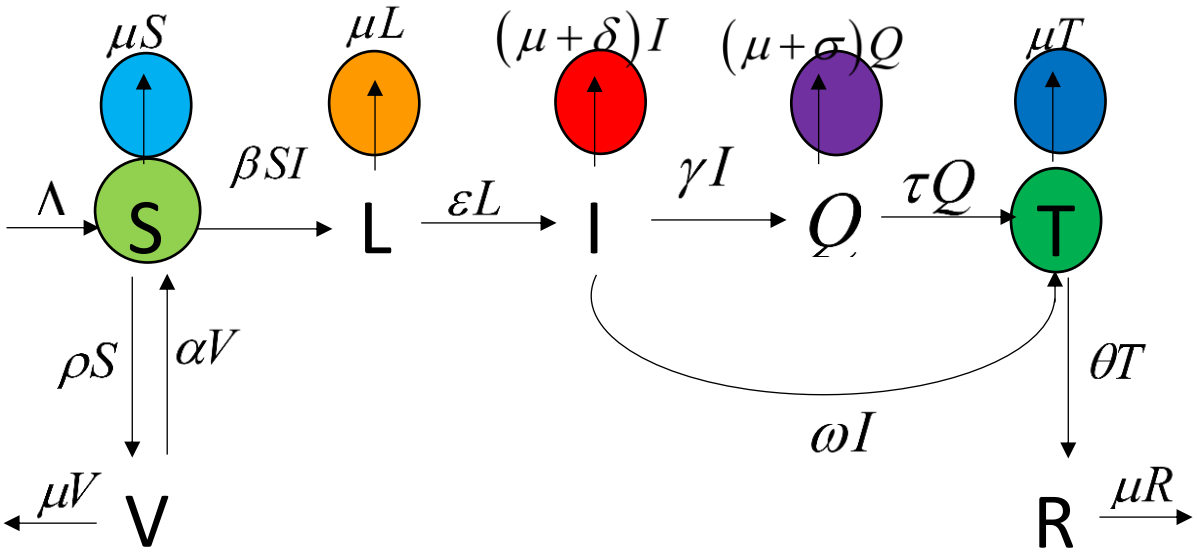

: Schematic representation of the model

Model equations:

$$\frac{dS}{dt} = \Lambda + \alpha V - \beta SI - (\mu + \rho)S \quad (1)$$

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

$$\frac{dL}{dt} = \beta SI - (\mu + \varepsilon)L \quad (3)$$

$$\frac{dI}{dt} = \varepsilon L - (\mu + \delta + \omega + \gamma)I \quad (4)$$

$$\frac{dQ}{dt} = \gamma I - (\mu + \sigma + \tau)Q \quad (5)$$

$$\frac{dT}{dt} = \tau Q - \omega I - (\mu + \theta)T \quad (6)$$

$$\frac{dR}{dt} = \theta T - \mu R \quad (7)$$

Table 1: Variables and parameters of the model

Symbol	Description
$S(t)$	Susceptible individuals at time t
$V(t)$	Vaccinated individuals at time t
L(t)	Latent individuals at time t
$I(t)$	Infected individuals at time t
$Q(t)$	Isolated individuals at time t
$T(t)$	Treated Individuals at time t
$R(t)$	Recovered individuals at time t
Λ	Rate of recruitment
β	Contact rate
ε	Progression rate from the Latent class to Infected class
ρ	Rate of progression from Susceptible class to Vaccinated class
α	Waning rate of vaccine
γ	Rate of progression from Infected class to Isolated class
τ	Treatment rate from Isolated class to Treated class
θ	Recovery rate
μ	Natural death rate
δ	Death rate due to infection
σ	Death rate for Isolated individuals
ω	Treatment rate

Positivity of Solution of the Model

Based on the equations (1) to (7) that represents the population in each compartment, it shows that all model parameters are positive (Bako *et al.,* 2024).

Theorem 1: If the initial value of the model is given as $\{S(0) \geq V(0) \geq L(0) \geq I(0) \geq Q(0) \geq T(0) \geq R(0) \geq\} \in \Omega$ the solution set $\{S(t), V(t), L(t), I(t), Q(t), T(t), R(t)\}$ of the model equations are all positive for all time $t > 0$

Proof:

From equation (1)

$$\frac{dS}{dt} = \Lambda + \alpha V - \beta SI - (\mu + \rho)S$$

Then, $\frac{dS}{dt} \geq -(\mu + \rho)S$

By the method of separation of variable, we obtain

$$\frac{dS}{dt} \geq -K_1 dt$$

Integrating both sides, we have;

$$\ln S(t) \geq -\int K_1 dt$$

$$\ln S(t) \geq -K_1 t$$

Taking exponent of both side, we obtain;

$S(t) \geq S(0)e^{-K_1 t}$

Using the same approach to other equations in the model equation, we have

$V(t) \geq V(0)e^{-K_2 t}, L(t) \geq L(0)e^{-K_3 t}, I(t) \geq I(0)e^{-K_4 t}$

$Q(t) \geq Q(0)e^{-K_6 t}, T(t) \geq T(0)e^{-K_6 t}, R(t) \geq R(0)e^{-\mu t}$

Therefore, the solution of the model equations is positive for all time $t > 0$

Basic Reproduction Numbers $R_\circ$

The basic reproduction number is characterized as the quantity of secondary cases that an infected individual can create in a completely susceptible populace. In biomathematics, it is utilized for future prediction of an epidemic in a population (Bako *et al.,* 2024). If $R_\circ < 1$ the infection will be wiped out. But if $R_\circ > 1$ the infection will spread in the population. $R_\circ$ can be computed by using the next generation matrix approach.

$R_\circ = \rho(FV^{-1}),$

Where, $\rho(FV^{-1})$ describe the spectral radius of FV^{-1}

Applying the next generation matrix, (Bako *et al.,* 2017) we consider the infectious compartments in the model system, which includes:

$$\frac{dL}{dt} = \beta SI - K_3 L$$

$$\frac{dI}{dt} = \varepsilon L - K_4 I$$

$$\frac{dI_s}{dt} = \gamma I - K_5 I_s$$

Let F be the rate of emerging infections in the population and let V be the rate of transfer of infection in the population.

It can be deduced that

$$F_i = \begin{bmatrix} \beta SI \\ 0 \\ 0 \end{bmatrix}$$

$$V_i = \begin{bmatrix} K_3 L \\ -\varepsilon L + K_4 I \\ -\gamma I + K_5 I_s \end{bmatrix}$$

The Jacobian matrix of F evaluated at the disease free equilibrium point is given as;

$$F = \frac{\partial F_i(E_o)}{\partial I} = \begin{bmatrix} 0 & \frac{\beta K_2 \Lambda}{K_1 K_2 - \rho\alpha} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (8)$$

$$V = \frac{\partial V_i(E_o)}{\partial I} = \begin{bmatrix} K_3 & 0 & 0 \\ -\varepsilon & K_4 & 0 \\ 0 & -\gamma & K_5 \end{bmatrix} \quad (9)$$

So, therefore $V^{-1} = \begin{bmatrix} \frac{1}{K_3} & 0 & 0 \\ \frac{\varepsilon}{K_3K_4} & \frac{1}{K_4} & 0 \\ \frac{\varepsilon\gamma}{K_3K_4} & \frac{\gamma}{K_4K_5} & \frac{1}{K_5} \end{bmatrix}$ (10)

Computing the product of F and V^{-1}

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta K_2\Lambda}{K_1K_2 - \rho\alpha} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{K_3} & 0 & 0 \\ \frac{-\varepsilon}{K_3K_4} & \frac{1}{K_4} & 0 \\ \frac{\varepsilon\gamma}{K_3K_4} & \frac{-\gamma}{K_4K_5} & \frac{1}{K_5} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta\varepsilon K_2\Lambda}{K_3K_4(K_1K_2 - \rho\alpha)} & \frac{\beta K_2\Lambda}{K_4(K_1K_2 - \rho\alpha)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (11)$$

The characteristics equation is given as;

$$\left|FV^{-1} - \lambda I\right| = 0 \quad (12)$$

$$\begin{vmatrix} \frac{\beta\varepsilon K_2\Lambda}{K_3K_4(K_1K_2 - \rho\alpha)} - \lambda & \frac{\beta K_2\Lambda}{K_4(K_1K_2 - \rho\alpha)} & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{vmatrix} = 0$$

$$\left(\frac{\beta\varepsilon K_2\Lambda}{K_3K_4(K_1K_2 - \rho\alpha)} - \lambda\right)(-\lambda)(-\lambda) = 0$$

$$\lambda = 0 \text{ (2 times)}$$

$$\frac{\beta\varepsilon K_2\Lambda}{K_3K_4\left(K_1K_2-\rho\alpha\right)}-\lambda=0$$

$$\frac{\beta\varepsilon K_2\Lambda}{K_3K_4\left(K_1K_2-\rho\alpha\right)}-\lambda=0$$

Therefore,

$$R_{\mathrm{o}}=\frac{\beta\varepsilon K_2\Lambda}{K_3K_4\left(K_1K_2-\rho\alpha\right)} \tag{13}$$

Equation (13) is the Basic Reproduction number for the model system.

Numerical Simulations

In this section the numerical simulation study of the model equation is done. For the study, values and sources of variables and parameters for simulating the model is given. Table 1 illustrates the referenced and the assumed estimated values for the model variables. Table 2 shows the reference and the assumed estimate value for the model parameters.

Table 1: Variable values used for the model

Variables	Values	Sources
S	160,840,589	OSMAN *et al.*
V	8,000,000	OSMAN *et al.*
L	1,700,000	OSMAN *et al.*
I	1,800,000	Assumed
Q	1,100,000	Assumed
T	1109,000	OSMAN *et al.*
R	800,000	Assumed

Table 2: Parameter values used for the model

Parameter	Values	Sources
∅	36.61	Assumed
A	0.0082	Assumed
E	0.007	Assumed
Γ	0.00375	Assumed
T	0.004	Assumed
P	0.022	Assumed
B	0.02	OSMAN *et al.*
Ω	0.02	Assumed
Θ	0.021	Assumed
δ_1	0.70	Assumed
δ_2	0.1	OSMAN *et al.*
M	0.018	OSMAN *et al.*

Graphical Representation of Solutions of Model Equations

The graphical representation from the Homotopy perturbation method of the analytical solutions of the model equations. The maple software is used to plot the graphs, using the values given in the table 1 and table 2

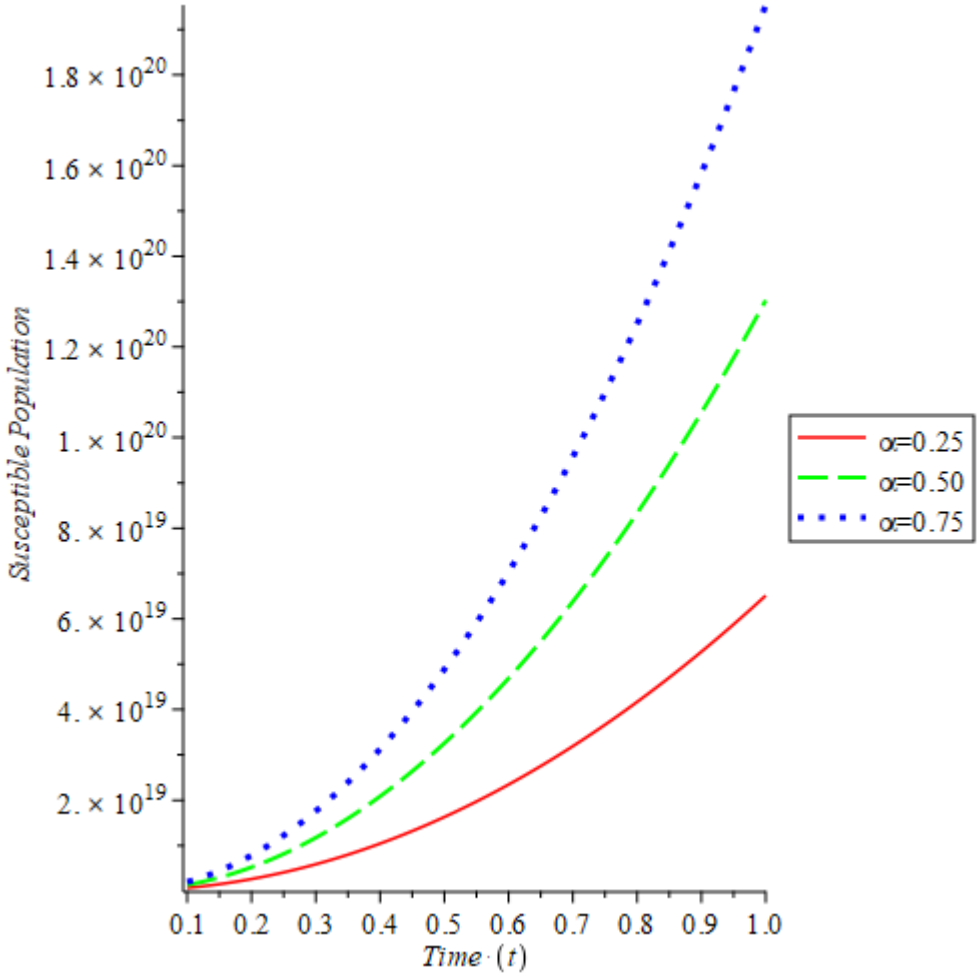

Figure 2: Effect of Infection Rate on the Susceptible Population

Figure 2 is the graph of the susceptible individuals against time for the different values of the infection rate. The graph shows that an increase in the infection rate will lead to a corresponding increase in the susceptible population. This implies that; in an ecosystem, more people will be susceptible to the TB disease if the rate of infection is on the increase.

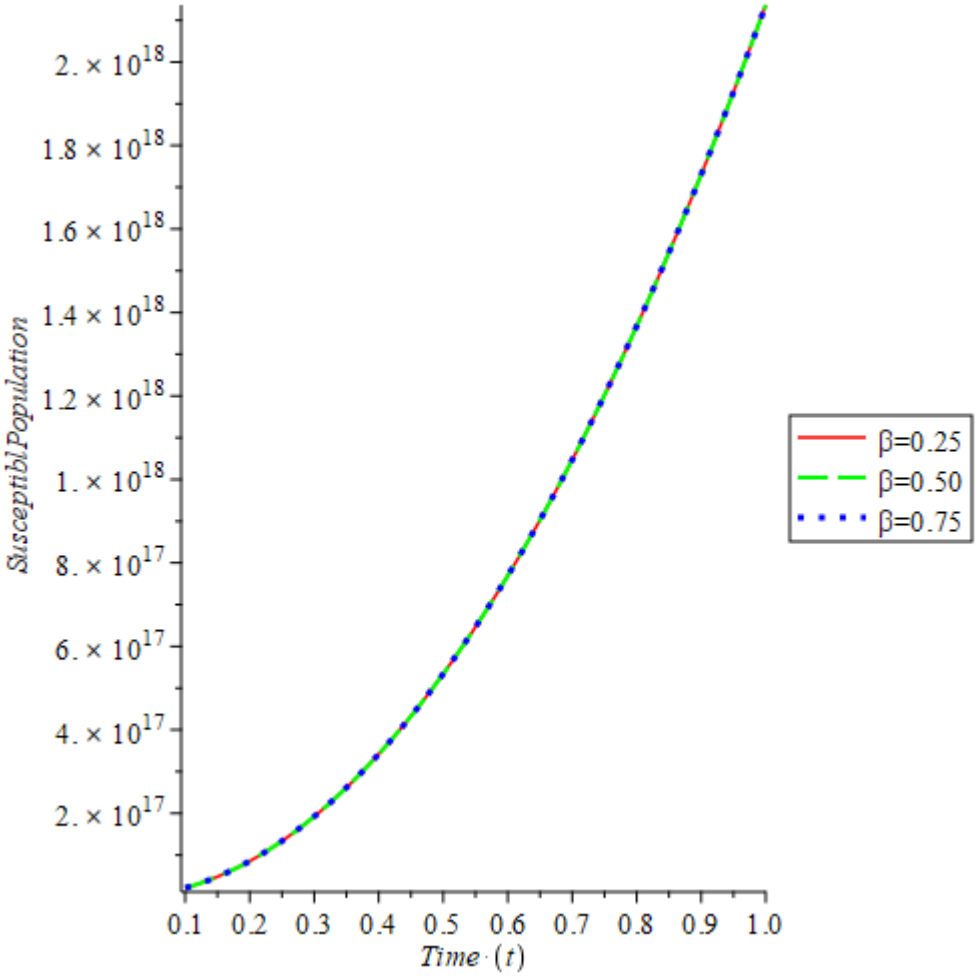

Figure 3: Effect of Waning Rate on the Susceptible Population

Figure 3 is the graph of the susceptible individuals against time for the different values of the vaccine waning rate. Carrying out simulations by varying the waning rate of vaccine. It is observed that for the different level of the waning rate, the TB infection continue to persist in the susceptible population. This further illustrates that an increase in the vaccine waning rate results to a corresponding increase of the susceptible individuals.

Conclusion

A thorough mathematical model of the dynamics of tuberculosis (TB) transmission, including treatment, isolation, and vaccine plans, is presented in this work. The disease-free equilibrium is stable, as shown by an analysis of these equilibria with respect to the effective reproduction number. Interestingly, when the fundamental reproduction number (R_0) is smaller than 1, the TB-free equilibrium shows both local and global asymptotic stability. In order to evaluate the effects of different treatments, both separately and in combination, the researchers computed the effective reproduction number and used the Homotopy Perturbation Method to obtain analytical answers.

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