



Mathematical Modeling of Tuberculosis Dynamics with Vaccination, Public Awareness and Treatment

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ABSTRACT

Tuberculosis (TB) is a bacterial infection caused by Mycobacterium tuberculosis that typically affects the lungs, but can also impact other organs. Globally, tuberculosis (TB) remains a significant cause of mortality from infectious diseases. In this paper, we developed a nonlinear deterministic model which incorporates vaccination, treatment and public awareness to study the dynamics of tuberculosis. The analysis of the model establishes the boundedness and positivity of the solutions, TB free equilibrium is shown to be both locally and asymptotically stable when $R_c < 1$ and unstable when $R_c > 1$. Conversely, the endemic globally equilibrium is globally asymptotically stable when $R_c > 1$ and unstable when $R_c < 1$. The most sensitive parameters for controlling TB transmission are identified using the forward normalized sensitivity index method and found that vaccination and treatment are the most sensitive parameters for decreasing TB transmission. Numerical simulations that shows vaccination, public awareness and treatment of infected individuals are crucial strategies for the effective control of tuberculosis in the population.

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INTRODUCTION

Tuberculosis (TB) is a bacterial infection caused by Mycobacterium tuberculosis that typically affects the lungs, but can also impact other organs (WHO, 2023). Globally, tuberculosis (TB) remains a significant cause of mortality from infectious diseases. According to the latest report from the World Health Organization (WHO), TB has once again become the top infectious disease killer in 2023, exceeding COVID-19 due to a notable increase in cases (Suvvari, 2025). Despite being preventable and curable, tuberculosis (TB) remains a devastating global health problem, claiming the lives of 1.5 million people every year and causing illness in an estimated 10 million people worldwide. This makes TB the top infectious killer on the planet, despite the availability of effective treatments and interventions to prevent the spread of the disease (WHO, 2023). In 2023, an estimated 10.8 million people globally contracted tuberculosis (TB), with 6 million men, 3.6 million women, and 1.3 million children affected. This disease continues to have a significant presence in every nation, emphasizing the need for continued efforts in diagnosing, treating, and preventing this deadly disease (WHO, 2023). The global TB data for 2023 showed that 6.1% of new cases were among people living with HIV. Regionally, nearly half (45%) of all TB cases were reported in South-East Asia, followed by Africa (24%) and the Western Pacific (17%), with the remaining cases distributed across the Eastern Mediterranean (8.6%), the Americas (3.2%), and Europe (2.1%) (WHO, 2025).

Tuberculosis (TB) is primarily transmitted from a person with infectious (active) tuberculosis to susceptible (and potentially latently infected) individuals through airborne droplets produced when the person with active TB coughs or sneezes. These droplets can remain suspended in the air for a prolonged period, allowing for the transmission of the disease to others through inhalation (Castillo-Charvez and Song, 2004). Approximately 25% of the global population is

thought to be infected with TB bacteria. However, only 5 – 10% of those infected will progress to active TB disease, exhibiting symptoms (WHO, 2023). TB symptoms can be mild, making it easy to unknowingly infect others. Some patients with TB may not exhibit symptoms. Common symptoms of TB include prolonged coughing, often with blood, chest pain, weakness, fatigue, fever, and night sweats (WHO, 2023).

Tuberculosis (TB) is largely a preventable disease. TB preventive treatment (TPT) can be administered to individuals at risk, preventing progression from TB infection to disease. Additional Population-based measures, such as TB screening, addressing poverty, malnutrition, HIV, diabetes, and tobacco use, can reduce the burden of TB (WHO, 2025). The currently licensed vaccine, BCG, provides protection against severe forms of TB and mortality associated with TB, particularly in young children (WHO, 2025).

Over the years, researchers have utilized mathematical models to study the dynamics of tuberculosis and develop effective strategies for its control and eradication. Some of these models includes. Study by (Ojo *et al.*, 2023) suggested that reducing effective contact with infected individuals, as well as increasing vaccination rates for susceptible individuals with high efficacy vaccines, will effectively lower the incidence of tuberculosis within a population. (Oshinubi *et al.*, 2023) indicated that by increasing the availability of vaccination, especially for infected individuals, and increasing treatment availability, the prevalence and burden of tuberculosis on the human population can be effectively reduced. (Athithan, and Ghosh, 2013) demonstrated that sustained treatment strategies and improved case detection could significantly increase the effectiveness of tuberculosis control efforts. (Simorangkir *et al.*, 2021) revealed that even though vaccination is more effective in reducing the basic reproduction number compared to observed treatment.

Considering the aforementioned studies, in this research we proposed a mathematical model to assess the impact of vaccination, awareness campaign and treatment in the dynamics of tuberculosis

MATERIALS AND METHODS

Model Description

The dynamics of tuberculosis was study in this model by incorporating vaccination, treatment and awareness. The total population denoted by $N(t)$ is divided into six mutually exclusive compartments. Susceptible $S(t)$, this class comprises all individuals who are at risk of acquire the infection. Vaccinated $V(t)$, comprises all individuals that are vaccinated but few can acquire the infection slower rate. Exposed $E(t)$, this class comprises of susceptible and vaccinated individuals come in contact with infectious individuals. Infected $I(t)$, this class consists of individuals who developed active TB symptoms. Infected under treatment $I_t(t)$, this class comprises of all receiving treatment.

Recovered compartment $R(t)$ those are individuals who acquired TB immunity.

The recruitment into susceptible compartment is by birth at a constant rate π . The class is decline through the infection rate λ and vaccination rate ϕ . Vaccinated compartment is created via vaccination rate ψ , the class is decline through the vaccine wine rate ψ and infection rate λ . Exposed population is created through the infection rate λ , the class is diminish via progression rate σ to $I(t)$ and $I_t(t)$, it further decrease by recovery rate τ_1 . Infected population is created via progression rate σ , the class is further decline to $I_t(t)$ at rate γ . Treatment population is generated through progression and treatment rate γ , the class is diminish by recovery rate τ_2 . Recovered population is created through treatment rates of $I(t)$ and $I_t(t)$. The TB induced death rate δ_1, δ_2 only occurs in infected and treated compartments, while the natural death occurs in all the six compartments μ . The total population will therefore be $N(t) = S(t) + V(t) + E(t) + I(t) + I_t(t) + R(t)$.

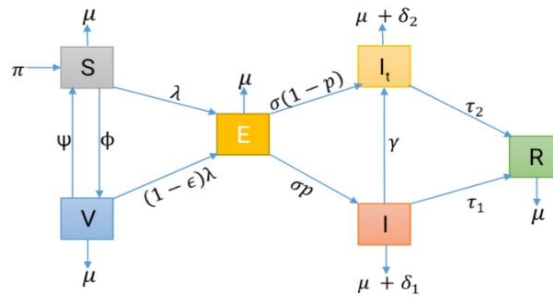


Figure 1: Schematic Diagram of the Model (1)

$$\begin{aligned}
 \frac{dS}{dt} &= \pi + \psi V - (\lambda + \phi + \mu)S, & \frac{dI_t}{dt} &= \sigma p E + \gamma I - (\tau_2 + \mu + \delta_2)I_t, \\
 \frac{dV}{dt} &= \phi S - (1 - \epsilon)\lambda V - (\psi + \mu)V, & \frac{dR}{dt} &= \tau_1 I + \tau_2 I_t - \mu R. \\
 \frac{dE}{dt} &= \lambda S + (1 - \epsilon)\lambda V - (\sigma + \mu)E, & \text{Where} & \\
 \frac{dI}{dt} &= \sigma(1 - p)E - (\gamma + \tau_1 + \mu + \delta_1)I, & \lambda &= \frac{\beta(1-\theta)(1+\xi I_t)}{N}
 \end{aligned}
 \tag{1}$$

Table 1: Interpretation of the State Variables and Parameters Used in the Model (1)

Variables	Descriptions
N	Total population
S	Susceptible individuals
V	Vaccinated individuals
E	Exposed individuals
I	Infected individuals
I _t	Infected under treatment individuals
R	Recovered individuals
Parameters	Descriptions
π	Recruitment of susceptible individuals
p	Treatment rate of exposed individuals
μ	Natural mortality rate
β	Effective contact rate
τ_1	Recovery rate of Infected individuals
τ_2	Recovery rate of treated individuals
ϕ	Vaccine rate
ϵ	Reduction rate of infection due to vaccine
σ	Progression rate
θ	Awareness rate
ψ	Vaccine reversion rate
γ	Treatment rate
ξ	Parameter for decreasing the infection of treated individuals
δ_1, δ_2	TB induced death rate

Theoretical Analysis of the Model

Boundedness and Positivity of Solution

The solution of model (1) is restricted to a manifold (or state space) Ω , denoted by

$$\Omega = \{s(t), v(t), E(t), I(t), I_t(t), R(t) \in R_+^6 : N \leq \frac{\pi}{\mu}\}, \quad (2)$$

Theorem 3.1

The set Ω is positively invariant and acts as an attracting region.

Proof

Our objective is to show that R_+^6 is positively invariant, which means that all solutions to system (1) that start inside Ω always stay inside Ω . Suppose $R(0) > 0$ and that $S(0), V(0), E(0), I(0)$, and $I_t(0) > 0$. If $S(0)$ and $V(0)$ are not both positive, then $S(t) > 0$ and $V(t) > 0$ for $t \in [0, \tilde{t}]$ exist at some time $\tilde{t} > 0$ and $S(t) = V(t) = 0$. Using the system (1) third, fourth, and fifth equations, we now get,

$$\frac{dE}{dt} \geq -(\sigma + \mu)E(t) \quad \text{for } t \in [0, \tilde{t}], \quad (3)$$

$$\frac{dI}{dt} \geq -(\tau_1 + \gamma + \mu + \delta_1)I(t) \quad \text{for } t \in [0, \tilde{t}],$$

$$\frac{dI_t}{dt} \geq -(\tau_2 + \mu + \delta_2)I_t(t) \quad \text{for } t \in [0, \tilde{t}],$$

Thus, $E(0) > 0, I(0) > 0$ and $I_t(0) > 0$ for $t \in [0, \tilde{t}]$. As a result, using the system (1) first and second equations, we've obtained

$$\frac{dS}{dt} \geq -(\lambda + \phi + \mu)S(t) \quad \text{for } t \in [0, \tilde{t}],$$

$$\frac{dV}{dt} \geq -((1 - \epsilon)\lambda + \psi + \mu)V(t) \quad \text{for } t \in [0, \tilde{t}].$$

It is evident that $S(0) > 0$ and $V(0) > 0$, which defy our presumption that $S(\tilde{t}) = V(\tilde{t}) = 0$. $S(t)$ and $V(t)$ are hence positive. As an alternative, we may think about a subsystem of (1) that does not include the first and second equations. This subsystem can be represented in matrix form and clearly shows that the remaining state variables in the model are positive.

$$\frac{dX(t)}{dt} = MY(t) + B(t) \quad (4)$$

With $Y(t) = (E, I, I_t, R)^T$

$$M = \begin{pmatrix} -k_3 & n & n\xi & 0 \\ \sigma(1-p) & -k_4 & 0 & 0 \\ \sigma p & \gamma & -k_5 & 0 \\ 0 & \tau_1 & \tau_2 & -\mu \end{pmatrix} \quad (5)$$

Where,

$n = \frac{\beta(S+(1-\epsilon)V)}{N}, k_3 = \sigma + \mu, k_4 = \gamma + \tau_1 + \mu + \delta_1, k_5 = \tau_2 + \mu + \delta_2$. Subsystem (4) is a monotone system since both $S(t)$ and $V(t)$ are non-negative, indicating that M is a Metzler matrix (Ibrahim et al., 2025). R_+^4 is hence invariant under the flow of subsystem (4). Consequently, under the flow of the system, R_+^6 becomes positively invariant (1).

TB Free Equilibrium Point

The model (1) has a TB free equilibrium, ϵ^0 which occurs when $E = I = I_t = 0$. Mathematically, it can be expressed as:

$$\epsilon^0 = (S^0, V^0, E^0, I^0, I_t^0, R^0) \quad (6)$$

$$S^0 = \frac{\pi k_2}{k_1 k_2 - \phi \psi} \quad (7)$$

$$V^0 = \frac{\pi \phi}{k_1 k_2 - \phi \psi} \quad (8)$$

$$E^0, I^0, I_t^0, R^0 = (0, 0, 0, 0). \quad (9)$$

Where

$$k_1 = \phi + \mu \quad \text{and} \quad k_2 = \psi + \mu$$

Basic Reproduction Number

Following (Abubakar et al., 2025, Andrawus et al., 2024 and Ibrahim et al., 2025) the basic reproduction number R_0 is obtain via the next-generation approach $\rho(N_1 N_2^{-1})$, where N_1 captures for new infection terms linearized at the TB free equilibrium and N_2 describes all other transition within the system:

$$N_1 = \begin{bmatrix} 0 & \frac{\beta(1-\theta)(S^0+(1-\epsilon)V^0)}{N^0} & \frac{\beta(1-\theta)\xi(S^0+(1-\epsilon)V^0)}{N^0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, N_2 = \begin{bmatrix} k_3 & 0 & 0 \\ -\sigma(1-p) & k_4 & 0 \\ -\sigma p & -\gamma & k_5 \end{bmatrix}, \quad (9)$$

And

$$N_2^{-1} = \begin{bmatrix} \frac{1}{k_3} & 0 & 0 \\ \frac{\sigma(1-p)}{k_3 k_4} & \frac{1}{k_4} & 0 \\ \frac{\sigma(\mu p + p\tau_1 + p\delta_1 + \gamma)}{k_3 k_4 k_5} & \frac{\gamma}{k_4 k_5} & \frac{1}{k_5} \end{bmatrix}, \quad (10)$$

Multiplying N_1 and N_2^{-1} , we have

$$N_1 N_2^{-1} = \begin{bmatrix} \frac{\beta\sigma(1-\theta)(S^0+(1-\epsilon)V^0)((1-p)(k_5+\gamma\xi)+p\xi k_4)}{N^0 k_3 k_4 k_5} & \frac{\beta(1-\theta)(\gamma\xi+k_5)(S^0+\theta(1-\epsilon)V^0)}{N^0 k_4 k_5} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (11)$$

We obtained the eigenvalues of $N_1 N_2^{-1}$ as

$$\begin{bmatrix} 0 \\ 0 \\ \frac{\beta\sigma(1-\theta)(S^0+(1-\epsilon)V^0)((1-p)(k_5+\gamma\xi)+p\xi k_4)}{N^0 k_3 k_4 k_5} \end{bmatrix}, \quad (12)$$

The dominant eigenvalue from (12) is

$$\rho(N_1 N_2^{-1}) = \frac{\beta\sigma(1-\theta)(S^0+(1-\epsilon)V^0)((1-p)(k_5+\gamma\xi)+p\xi k_4)}{N^0 k_3 k_4 k_5} \quad (13)$$

Substituting S, V and N at TB free equilibrium in (13), we obtained control reproduction number

$$R_c = \frac{\beta\sigma(1-\theta)[(1-\epsilon)\phi\xi+k_2][(1-p)(k_5+\gamma\xi)+\xi p k_4]}{k_3 k_4 k_5 (\phi+k_2)} \quad (14)$$

Where

$$k_2 = \psi + \mu, k_3 = \sigma + \mu, k_4 = \gamma + \tau_1 + \mu + \delta_1 \quad \text{and} \quad k_5 = \tau_2 + \mu + \delta_2.$$

Interpretation of Control Reproduction Number R_c

The control reproduction number R_c is the number of new tuberculosis cases generated by TB infected individuals in a population comprises of susceptible and vaccinated individuals.

When there is no vaccination, treatment and awareness in the environment (*i.e.* $\phi = \psi = \gamma = \theta = 0$) we obtained the basic reproduction number as

$$R_0 = \frac{\beta\sigma[(1-p)(\tau_2+\mu+\delta_2)+p\xi(\tau_1+\mu+\delta_1)]}{(\sigma+\mu)(\tau_1+\mu+\delta_1)(\tau_2+\mu+\delta_2)} \quad (15)$$

Interpretation of Basic Reproduction Number R_0

The basic reproduction number R_0 is the number of new tuberculosis cases generated by tuberculosis infected individuals in a population with no vaccination, awareness and treatment in the community.

Local Asymptomatic Stability of TB Free Equilibrium

Theorem 3.2

TB free equilibrium (DFE) of the model (1) ϵ^0 is locally-asymptotically stable (GAS) in Ω if $R_c < 1$, and unstable if $R_c > 1$.

Proof

$$J(\epsilon^0) = \begin{bmatrix} -k_1 & 0 & 0 & -\beta(1-\theta)G_1 & -\beta(1-\theta)\xi G_1 & 0 \\ \phi & -k_2 & 0 & -\beta(1-\theta)(1-\epsilon)G_2 & -\beta\xi(1-\theta)(1-\epsilon)G_2 & 0 \\ 0 & 0 & -k_3 & \beta(1-\theta)(G_1 + (1-\epsilon)G_2) & \beta\xi(1-\theta)(G_1 + (1-\epsilon)G_2) & 0 \\ 0 & 0 & \sigma(1-p) & -k_4 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -k_5 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -\mu \end{bmatrix} \quad (16)$$

Where:

$$G_1 = \frac{\mu k_2}{k_1 k_2 - \phi \psi}, G_2 = \frac{\mu(1-\epsilon)\phi\xi}{k_1 k_2 - \phi \psi},$$

$$a_{11} = -k_1, a_{14} = -\beta(1-\theta)G_1,$$

$$a_{15} = -\beta(1-\theta)\xi G_1, a_{21} = \phi, a_{22} = -k_2,$$

$$a_{24} = -\beta(1-\theta)(1-\epsilon)G_2,$$

The following procedures can be used to linearize system (1) and calculate the Jacobian matrix at TB-free equilibrium:

$$a_{25} = -\beta\xi(1-\theta)(1-\epsilon)G_2, a_{33} = -k_3,$$

$$a_{34} = \beta(1-\theta)(G_1 + (1-\epsilon)G_2),$$

$$a_{35} = \beta\xi(1-\theta)(G_1 + (1-\epsilon)G_2),$$

$$a_{43} = \sigma(1-p), a_{44} = -k_4, a_{53} = \sigma p, a_{54} = \gamma,$$

$$a_{55} = -k_5, a_{64} = \tau_1, a_{65} = \tau_2, a_{66} = -\mu.$$

Reducing equation (16) into row echelon yield

$$J(\epsilon^0) = \begin{bmatrix} a_{11} & 0 & 0 & \frac{a_{14}}{a_{11}a_{24}-a_{14}a_{21}} & \frac{a_{15}}{a_{11}a_{25}-a_{15}a_{21}} & 0 \\ 0 & a_{22} & 0 & \frac{a_{11}}{a_{11}a_{24}-a_{14}a_{21}} & \frac{a_{11}}{a_{11}a_{25}-a_{15}a_{21}} & 0 \\ 0 & 0 & a_{33} & \frac{a_{34}}{a_{33}a_{44}-a_{34}a_{43}} & \frac{a_{35}}{a_{33}a_{44}-a_{34}a_{43}} & 0 \\ 0 & 0 & 0 & \frac{a_{33}}{a_{33}a_{44}-a_{34}a_{43}} & \frac{a_{33}}{a_{33}a_{44}-a_{34}a_{43}} & 0 \\ 0 & 0 & 0 & 0 & \frac{a_{33}a_{44}a_{55}-a_{34}a_{43}a_{55}+a_{43}a_{54}a_{35}-a_{44}a_{53}a_{35}}{a_{33}a_{44}-a_{34}a_{43}} & 0 \\ 0 & 0 & 0 & 0 & 0 & a_{66} \end{bmatrix} \quad (17)$$

The eigenvalues are ascertained as follows using Maple software:

$$\left[\begin{array}{l} \lambda_1 = a_{66} \\ \lambda_2 = a_{33} \\ \lambda_3 = \frac{a_{33}a_{44}-a_{34}a_{43}}{a_{33}} \\ \lambda_4 = \frac{a_{33}a_{44}a_{55}-a_{34}a_{43}a_{55}+a_{43}a_{54}a_{35}-a_{44}a_{53}a_{35}}{a_{33}a_{44}-a_{34}a_{43}} \\ \lambda_5 = a_{22} \\ \lambda_6 = a_{11} \end{array} \right] \quad (18)$$

Clearly $\lambda_1, \lambda_2, \lambda_5$ and λ_6 are all negatives from (18) and for the remaining after simplification, we have.

$$\lambda_3 \text{ is negative if and only if } \frac{-k_3 k_4 + \beta \sigma(1-p)(G_1 + (1-\epsilon)G_2)}{k_3} < 0 \quad (19)$$

$$\Leftrightarrow -k_3 k_4 + \beta \sigma(1-p)(G_1 + (1-\epsilon)G_2) < 0 \quad (20)$$

$$\Leftrightarrow \beta \sigma(1-p)(G_1 + (1-\epsilon)G_2) < k_3 k_4 \quad (21)$$

$$\Leftrightarrow \frac{\beta \sigma(1-p)(G_1 + (1-\epsilon)G_2)}{k_3 k_4} < 1 \quad (22)$$

$$\lambda_4 \text{ is also negative } \Leftrightarrow \frac{-k_3 k_4 k_5 + \beta \sigma(1-\theta)[G_1 + (1-\epsilon)G_2][(1-p)(k_5 + \gamma\xi) + \xi p k_4]}{k_3 k_4 - \beta \sigma(1-\theta)(1-p)[G_1 + (1-\epsilon)G_1]} < 0 \quad (23)$$

$$\Leftrightarrow -k_3 k_4 k_5 + \beta \sigma(1-\theta)[G_1 + (1-\epsilon)G_2][(1-p)(k_5 + \gamma\xi) + \xi p k_4] < 0 \quad (24)$$

$$\Leftrightarrow \beta \sigma(1-\theta)[G_1 + (1-\epsilon)G_2][(1-p)(k_5 + \gamma\xi) + \xi p k_4] < k_3 k_4 k_5 \quad (25)$$

$$\Leftrightarrow \frac{\beta \sigma(1-\theta)[G_1 + (1-\epsilon)G_2][(1-p)(k_5 + \gamma\xi) + \xi p k_4]}{k_3 k_4 k_5} < 1 \quad (26)$$

$$\text{Substituting } G_1 \text{ and } G_2 \text{ in (26) } \lambda_3 \text{ is also negative } \Leftrightarrow \frac{\beta \sigma(1-\theta)[(1-\epsilon)\phi\xi + k_2][(1-p)(k_5 + \gamma\xi) + \xi p k_4]}{k_3 k_4 k_5 (\phi + k_2)} = R_c < 1 \quad (27)$$

This demonstrates that if R_c is less than 1, all of the eigenvalues are negative and unstable otherwise. The proof of Theorem (3.2) is now complete.

Interpretation of Theorem (3.2)

Epidemiologically, Theorem (3.2) shows that if the control reproduction number R_c is smaller than 1, a community will continue to be free of endemic tuberculosis despite a small number of sick people. This suggests that tuberculosis may be

efficiently controlled and kept from becoming endemic if the number of TB cases is minimal and R_c is maintained below 1.

Global Stability of TB Free Equilibrium

Theorem 3.3

TB free equilibrium (DFE) of the model (1) ϵ^0 is globally-asymptotically stable (GAS) in Ω if $R_c < 1$, and unstable if $R_c > 1$.

Proof

Assuring that requirements (Q1) and (Q2) as stated in (Castillo-Charez and Son, 2004) hold true when $R_c < 1$. One way to express the model (1) is as follows:

$$\frac{dQ_1}{dt} = F(Q_1, Q_2),$$

$$\frac{dQ_2}{dt} = G(Q_1, Q_2); G(Q_1, 0) = 0, \quad (28)$$

Where $Q_1 = (S^0, V^0, R^0)$ and $P_2 = (E^0, I^0, 1_t^0)$ where $Q_1 \in R_+^3$ is represent the uninfected compartments and $Q_2 \in R_+^3$ represent the infected compartments. The TB free equilibrium is now denoted as, $M^0 = (Q_1^*, 0)$.

Where,

$Q_1^* = (N^0, 0)$ Now the first requirement (GAS) of Q_1^* gives

$$\frac{dQ_1}{dt} = F(Q_1, 0) = \begin{bmatrix} \pi + \psi V^0 - (\phi + \mu)S^0 \\ \phi S^0 - (\psi + \mu)V^0 \\ -\mu R^0 \end{bmatrix} \quad (29)$$

A linear ODE solving gives,

$$S^0(t) = \frac{\pi + \psi V^0}{(\phi + \mu)} - \frac{\pi + \psi V^0}{(\phi + \mu)} e^{-(\phi + \mu)t} + S_u^0(0) e^{-(\phi + \mu)t} \quad (30)$$

$$V^0(t) = \frac{\phi S^0}{\mu + \psi} - \frac{\phi S^0}{\mu + \psi} e^{-(\psi + \mu)t} + V^0(0) e^{-(\psi + \mu)t} \quad (31)$$

$$R^0(t) = \frac{1}{\mu} - \frac{1}{\mu} e^{-\mu t} + R^0(0) e^{-\mu t} \quad (32)$$

Now, obviously from system (1) we have, $S^0(t) + V^0(t) + R^0(t) \rightarrow N^0(t) \rightarrow$ as $t \rightarrow \infty$ regardless of the value of $S^0(t), V^0(t)$ and $R^0(t)$. Thus, $Q_1^* = (N^0, 0)$ is GAS.

Next, for the second requirement, that is $\tilde{G}(Q_1, Q_2) = A Q_2 - G(Q_1, Q_2) \geq 0$

$$A = \begin{pmatrix} -(\sigma + \mu) & \frac{\beta(S+(1-\epsilon)V)}{N} & 0 \\ \sigma(1-p) & -(\gamma + \tau_1 + \mu + \delta_1) & 0 \\ \sigma p & \gamma & -(\tau_2 + \mu + \delta_2) \end{pmatrix} \quad (33)$$

Matrix A is of Metzler type, since its off-diagonal elements are nonnegative.

$$G(Q_1, Q_2) = \begin{pmatrix} \frac{\beta S^0}{N^0} + \frac{(1-\epsilon)\beta V^0}{N^0} - (\sigma + \mu)E^0 \\ \sigma(1-p)E^0 - (\gamma + \tau_1 + \mu + \delta_1)I^0 \\ \sigma p E^0 + \gamma I^0 - (\tau_2 + \mu + \delta_2)I_t^0 \end{pmatrix} \quad (34)$$

Thus,

$$\tilde{G}(Q_1, Q_2) = A Q_2 - G(Q_1, Q_2) = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} \quad (35)$$

That is

$$\tilde{G}(Q_1, Q_2) = [0 \quad 0 \quad 0]^T \quad (37)$$

It is obvious that $\tilde{G}(Q_1, Q_2) = 0$.

TB Endemic Equilibrium Point

The model (1) has a TB endemic equilibrium, ϵ^{**} which occurs when E, I, I_t are all nonzero. Mathematically, it can be expressed as:

$$\begin{aligned} \epsilon^{**} &= (S^{**}, V^{**}, E^{**}, I^{**}, I_t^{**}, R^{**}) \\ S^{**} &= \frac{\pi(\lambda(1-\epsilon) - k_2)}{(1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda + \phi\psi - k_1k_2} \\ V^{**} &= \frac{\pi\phi}{(1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda - \phi\psi - k_1k_2} \\ E^{**} &= \frac{\lambda\pi(\lambda(\epsilon-1) + \phi(\epsilon-1) - k_2)}{k_3((1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda + \phi\psi - k_1k_2)} \\ I^{**} &= \frac{\lambda\pi\sigma(1-p)(\lambda(\epsilon-1) + \phi(\epsilon-1) - k_2)}{k_3k_4((1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda + \phi\psi - k_1k_2)} I_t^{**} \\ I_t^{**} &= \frac{\lambda\pi\sigma(\lambda(1-\epsilon) + \phi(\epsilon-1) - k_2)(\gamma(p-1) - pk_4)}{k_3k_4k_5((1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda + \phi\psi - k_1k_2)} \\ R^{**} &= \frac{\lambda\pi\sigma\tau_1(1-p)(\lambda(\epsilon-1) + \phi(\epsilon-1) - k_2)}{\mu k_3k_4((1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda + \phi\psi - k_1k_2)} + \frac{\lambda\pi\sigma\tau_2(1-p)(\lambda(\epsilon-1) + \phi(\epsilon-1) - k_2)}{\mu k_3k_4k_5((1-\epsilon)\lambda^2 + (k_1(\epsilon-1) - k_2)\lambda + \phi\psi - k_1k_2)} \end{aligned} \quad (38)$$

Where

$$k_1 = \phi + \mu, k_2 = \psi + \mu, k_3 = \sigma + \mu, k_4 = \gamma + \tau_1 + \mu + \delta_1 \text{ and } k_5 = \tau_2 + \mu + \delta_2$$

Existence of TB Endemic Equilibrium Point

The Descartes rule of sign was used to confirm the existence of the endemic equilibrium point of the suggested model (1). According to this rule, the number of positive roots in a polynomial equation with real coefficients and degree $n \geq 2$ is either equal to or less than the number of sign changes in the equation's terms. In this instance, the endemic state's force of infection was symbolized by:

$$\lambda^{**} = \frac{\beta(1-\theta)(I^{**} + \xi I_t^{**})}{N^{**}} \quad (39)$$

Where

$$N^{**} = S^{**} + V^{**} + E^{**} + I^{**} + I_t^{**} + R^{**}.$$

When (38) is substituted into (39), the following quadratic equation in terms of λ^{**} is obtained: When $R_c < 1$, the TB free equilibrium of equation (6) is equivalent to $\lambda^{**} = 0$

$$\lambda^{**2} A_1 + A_2 \lambda^{**} + A_3 = 0. \quad (40)$$

Where

$$A_1 = (\epsilon - 1)[\mu k_3 k_4 + \sigma(1-p)\mu k_4 + \sigma((p-1)\gamma - pk_4) + (1-p)(\tau_1 k_5 + \tau_2)]$$

$$\begin{aligned} A_2 &= \mu k_3 k_4 k_5 + \phi((\epsilon - 1) - k_2) + \mu k_4 k_5 + \sigma(1-p) \\ &\quad - pk_4 \mu + (1-p)(\tau_1 k_5 + \tau_2) \\ A_3 &= k_3 k_4 k_5 [1 - R_c]. \end{aligned} \quad (41)$$

Obviously, $A_1 > 0$ since all the parameters are positive with $0 < p < 1$. So there are four cases to be considered depending on the sign of A_2 and A_3 .

Theorem 3.4

The system (1) has:

- i. No endemic equilibrium if $A_2 > 0$ and $A_3 > 0 \Leftrightarrow R_c < 1$.
- ii. A unique endemic equilibrium if $A_2 < 0$ and $A_3 < 0 \Leftrightarrow R_c > 1$.
- iii. A unique endemic equilibrium if $A_2 > 0$ and $A_3 < 0 \Leftrightarrow R_c > 1$.
- iv. Two positive equilibrium if $A_2 < 0$ and $A_3 > 0 \Leftrightarrow R_c < 1$ and $A_2^2 - 4A_1A_3$

The following theorem was established based on items (ii) and (iii) of theorem (3.4) for reference

Theorem 3.5

The system (1) has a unique positive endemic equilibrium if $R_c > 1$.

Global Stability of TB Endemic Equilibrium

Theorem 3.7

Assuming vaccination rate, treatment rate of infected individuals and TB induced mortality rate are all negligible, then the endemic equilibrium is globally asymptotically stable while $R_c > 1$ and unstable when $R_c < 1$.

Proof

The Lyapunov function of Goh-Volterra type F can be defined as follows.

$$\begin{aligned} F &= \left(S - S^{**} - S^{**} \ln \frac{S^{**}}{S} \right) + \left(V - V^{**} - V^{**} \ln \frac{V^{**}}{V} \right) + \\ &\quad \left(E - E^{**} - E^{**} \ln \frac{E^{**}}{E} \right) + \frac{(\sigma+\mu)}{\sigma} \left(I - I^{**} - I^{**} \ln \frac{I^{**}}{I} \right) + \\ &\quad + \frac{(\sigma+\mu)(\gamma+\mu)}{\sigma\gamma} \left(I_t - I_t^{**} - I_t^{**} \ln \frac{I_t^{**}}{I_t} \right). \end{aligned} \quad (42)$$

Differentiating (44) with respect to time yields

$$\begin{aligned} \dot{F} &= \left(1 - \frac{S^{**}}{S} \right) \dot{S} + \left(1 - \frac{V^{**}}{V} \right) \dot{V} + \left(1 - \frac{E^{**}}{E} \right) \dot{E} + \frac{(\sigma+\mu)}{\sigma} \left(1 - \frac{I^{**}}{I} \right) \dot{I} \\ &\quad + \frac{(\sigma+\mu)(\gamma+\mu)}{\sigma\gamma} \left(1 - \frac{I_t^{**}}{I_t} \right) \dot{I}_t \end{aligned} \quad (43)$$

With

$$N = \frac{\pi}{\mu}$$

As the infection's force is altered, we have

$$\bar{\lambda} = \bar{\beta}(I + \xi I_t)$$

Where

$$\bar{\beta} = \bar{\beta} \frac{\pi}{\mu}$$

When (1) is substituted with (43), we obtain

$$\begin{aligned} \dot{F} &= \left(1 - \frac{S^{**}}{S} \right) (\pi - \lambda S - (\phi + \mu)S) \\ &\quad + \left(1 - \frac{V^{**}}{V} \right) (\phi S - (1-\epsilon)\lambda V - \mu V) \\ &\quad + \left(1 - \frac{E^{**}}{E} \right) (\lambda S + (1-\epsilon)\lambda V - (\sigma + \mu)E) \\ &\quad + \frac{(\sigma + \mu)}{\sigma} \left(1 - \frac{I^{**}}{I} \right) (\sigma E - (\gamma + \mu)I) \\ &\quad + \frac{(\sigma + \mu)(\gamma + \mu)}{\sigma\gamma} \left(1 - \frac{I_t^{**}}{I_t} \right) (\gamma I - (\tau_2 + \mu)I_t) \end{aligned} \quad (44)$$

With relationships

$$\pi = \lambda^{**} S^{**} + (\phi + \mu) S^{**}$$

$$\phi S^{**} = \theta \lambda^{**} V^{**} + \mu V^{**},$$

$$(\sigma + \mu)E^{**} = \lambda^{**}S^{**} + (1 - \epsilon)\lambda^{**}V^{**}, \tag{45}$$

$$\begin{aligned} (\gamma + \mu)I^{**} &= \sigma E^{**}, \\ (\tau_2 + \mu)I_t^{**} &= \gamma I^{**}, \end{aligned}$$

The relations in (45) can be changed to (44).

$$\begin{aligned} \dot{F} \leq (\phi + \mu)S_u^{**} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) + \mu S_a^{**} \left(2 - \frac{V}{V^{**}} - \frac{V^{**}}{V}\right) \\ + \lambda S^{**} \left(5 - \frac{S^{**}}{S} - \frac{SE^{**}}{S^{**}E} - \frac{EI^{**}}{E^{**}I} - \frac{\Pi_t^{**}}{I^{**}I_t} - \frac{I_t}{I_t^{**}}\right) \\ + (1 - \epsilon)\lambda V^{**} \left(5 - \frac{V^{**}}{V} - \frac{VE^{**}}{V^{**}E} - \frac{EI^{**}}{E^{**}I} - \frac{\Pi_t^{**}}{I^{**}I_t} - \frac{I_t}{I_t^{**}}\right) \end{aligned} \tag{46}$$

Applying the relation of geometric to arithmetic means to each yield the inequality

$$\begin{aligned} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S}\right) \leq 0, \left(2 - \frac{V}{V^{**}} - \frac{V^{**}}{V}\right) \leq 0, \\ \left(5 - \frac{S^{**}}{S} - \frac{SE^{**}}{S^{**}E} - \frac{EI^{**}}{E^{**}I} - \frac{\Pi_t^{**}}{I^{**}I_t} - \frac{I_t}{I_t^{**}}\right) \leq 0, \end{aligned} \tag{47}$$

$$\left(5 - \frac{V^{**}}{V} - \frac{VE^{**}}{V^{**}E} - \frac{EI^{**}}{E^{**}I} - \frac{\Pi_t^{**}}{I^{**}I_t} - \frac{I_t}{I_t^{**}}\right) \leq 0.$$

Hence,

$\dot{F} \leq 0$. Only at $S = S^{**}$, $V = V^{**}$, $E = E^{**}$, $I = I^{**}$ and $I_t = I_t^{**}$ is strict equality $\dot{F} = 0$ true. The endemic equilibrium ϵ^* is the only invariant set shown in the model (1). Applying Lasalle invariance principle (Lasalle, 1976) the TB endemic equilibrium is (GAS).

RESULTS AND DISCUSSION

Sensitivity Analysis

In this section, we examined the suggested tuberculosis model in connection to the reproduction number R_c with respect to the biological factors included in the model using the forward sensitivity index approach. This procedure was used to determine each parameter's sign; negative values indicate that the parameter is most susceptible to lowering R_c , while positive values indicate that the parameter is most sensitive to raising R_c (Abubakar et al., 2025, Andrawus et al., 2024).

The normalized local sensitivity index of the R_c with respect to the parameters is given by,

$$\alpha_{\omega}^{R_c} = \frac{\omega}{R_c} \times \frac{\partial R_c}{\partial \omega}$$

Table 2: Forward Normalized Sensitivity Indices

Parameters	Elasticity Indices	Values of the Elasticity Index
p	$\alpha_p^{R_c}$	0.4521
θ	$\alpha_{\theta}^{R_c}$	-0.7641
ϕ	$\alpha_{\phi}^{R_c}$	-0.7413
Ψ	$\alpha_{\psi}^{R_c}$	0.2421
σ	$\alpha_{\sigma}^{R_c}$	0.3143
β	$\alpha_{\beta}^{R_c}$	0.9935
γ	$\alpha_{\gamma\gamma}^{R_c}$	-0.6462
τ_1	$\alpha_{\tau_1}^{R_c}$	-0.3614
τ_2	$\alpha_{\tau_2}^{R_c}$	-0.4501
δ_1	$\alpha_{\delta_1}^{R_c}$	0.3211
δ_2	$\alpha_{\delta_2}^{R_c}$	0.2045
ϵ	$\alpha_{\epsilon}^{R_c}$	-0.1753
ξ	$\alpha_{\xi}^{R_c}$	0.5790

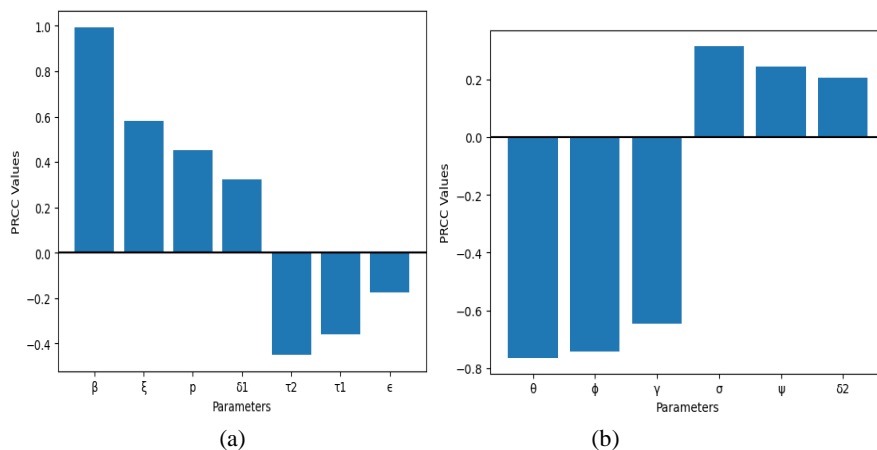


Figure 2: Figure Showing the PRCC Values of R_c Versus the Parameters of Model (1)

Numerical Simulation

Numerical simulations of the proposed model (1) provide valuable insights into the transmission dynamics of tuberculosis by capturing the interactions among key factors influencing its spread and control. Some model parameters

are derived from existing literature, while others are assumed, and two are control variables bounded between 0 and 1. These simulations enable visualization of how parameter variations affect the transmission and control of tuberculosis within the population.

Table 3: Ranges and Baseline Values of Parameters of Model (1)

Parameters	Ranges (Baseline)	Unit	Reference
λ	0.0006	per year	Fitted
π	0.0012	per year	Fitted
ρ	0.06	per year	Fitted
θ	0.76521	per year	Fitted
ϕ	0.2007	per year	Fitted
ψ	0.20	per year	(Kuddus et al., 2025)
σ	0.01432	per year	Fitted
β	0.55	per year	Fitted
γ	0.0002	per year	Fitted
τ^1	0.129	per year	(Peter et al., 2025)
τ^2	0.2	per year	Fitted
δ^1	0.055	per year	Fitted
δ^2	0.0353	per year	Fitted
μ	0.0142	per year	(Peter et al., 2025)
ϵ	0.7914	per year	Fitted

Vaccination Strategy

The impact of vaccination on the susceptible and vaccinated compartments is shown below. Figure 3(a) illustrates the effect of vaccination on susceptible individuals. As the vaccination rate increases, the number of susceptible individuals decreases significantly. This shows that vaccination has a strong impact on reducing the susceptible

population and limiting disease transmission. Figure 3(b) illustrates the effect of vaccination on the vaccinated population. As the vaccination rate increases, the number of vaccinated individuals rises substantially. This indicates that vaccination has a significant positive impact on the vaccinated compartment.

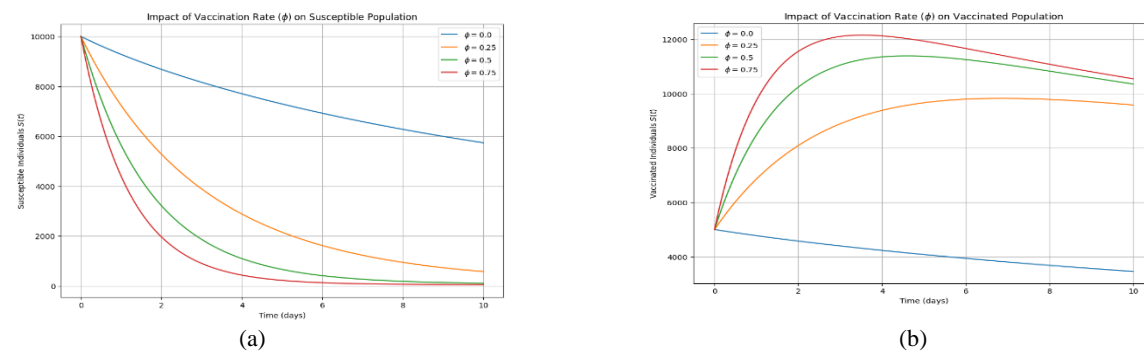
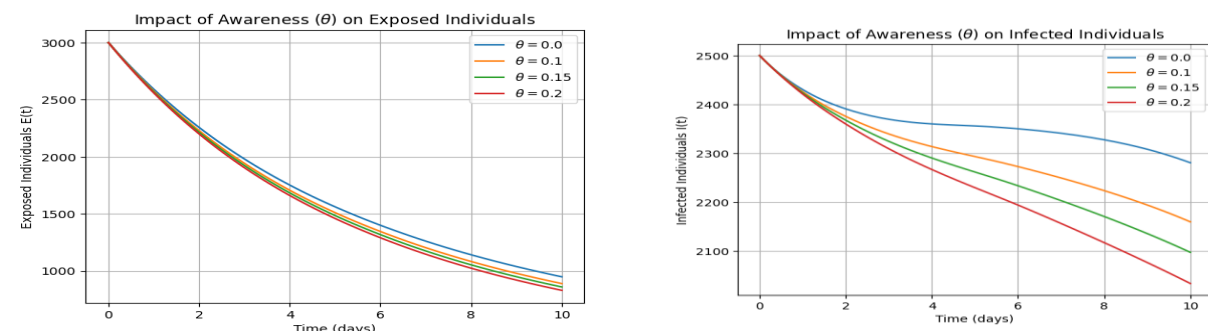


Figure 3: Figure Illustrating the Impact Vaccination on Susceptible Individuals (a) and Vaccinated Individuals (b) with Different Values of ϕ

Public Awareness Campaign Strategy

The impact of public awareness on the exposed, infected, and infected-under-treatment compartments is shown below. Figure 4(a) depicts the effect of public awareness on exposed individuals. Increasing the level of awareness slightly decreases the number of exposed individuals. This shows that public awareness has a measurable impact on reducing the exposed population. Figure 4(b) depicts the effect of public awareness on infected individuals. The number of infected

individuals decreases as the level of public awareness increases. This shows that public awareness has a significant impact on reducing the infected population. Figure 4(c) show the impact of public awareness on infected individuals under treatment. The plot clearly shows that increasing the levels of awareness campaign slightly decrease the number of treated individuals. This shows that public awareness has an impact on treated individuals.



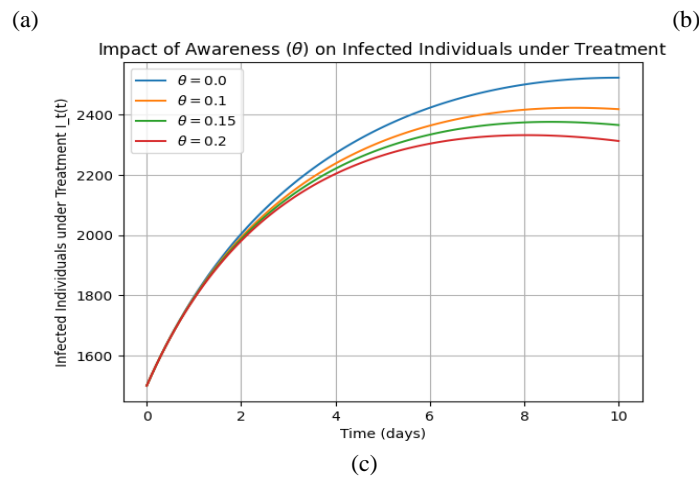


Figure 4: Figure Illustrating the Impact Public Awareness on Exposed Individuals (a), Infected Individuals (b) and Infected Individuals Under Treatment (c) with Different Values of θ

Treatment Strategy

The impact of treatment on the infected and infected-under-treatment compartments is shown below. Figure 5(a) depicts the effect of treatment on infected individuals. The results show that increasing the treatment rate leads to a significant decrease in the number of infected individuals. This indicates that treatment has a strong impact on reducing the infected

population. Figure 5(b) shows the impact of treatment on infected individuals under treatment. The plot clearly shows that increasing the rate, the number of infected individuals under treatment greatly increases. This shows that treatment has a strong impact on infected individuals under treatment.

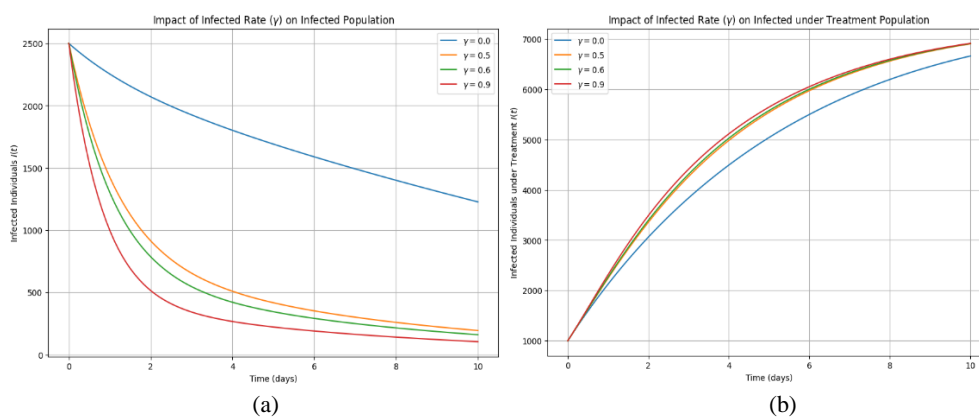


Figure 5: Figure Illustrating the Impact Treatment on Infected Individuals (a) and Infected Individuals Under Treatment (b) with Different Values of γ

CONCLUSION

In this paper, we developed a nonlinear deterministic model which incorporates vaccination, treatment and public awareness for the dynamics of tuberculosis. The analysis of the model demonstrated solution boundedness and positivity have been ascertained, TB free equilibrium is found to be both locally and globally asymptotically stable when $R_c < 1$ and unstable when $R_c > 1$. Conversely, the endemic equilibrium is globally asymptotically stable when $R_c > 1$ and unstable when $R_c < 1$. The most sensitive parameters for the control of TB transmission are identified using the forward normalized sensitivity index method and found that vaccination and treatment rate of infected individuals are the most sensitive parameters for decreasing TB transmission. Numerical simulations that shows vaccination, public awareness and treatment of infected individuals are very important parameters to control tuberculosis in a society.

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