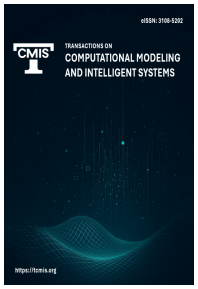

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RESEARCH ARTICLE

Stability analysis of the disease-free equilibrium of tuberculosis transmission dynamics incorporating drug resistance and vaccination

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Abstract

Tuberculosis (TB) remains a major public health challenge, particularly in high-burden countries such as Nigeria. In this study, a deterministic compartmental model was developed and analyzed to investigate the transmission dynamics of TB incorporating vaccination, first-line and second-line treatment, and drug-resistant TB. The human population is stratified into eight compartments namely: susceptible, vaccinated, latent, infectious, first-line treatment, drug-resistant, second-line treatment, and recovered. The model captures key biological processes, including progression from latent to active TB, treatment failure leading to resistance, waning immunity, relapse, and TB-induced mortality. Analysis is performed to establish the positivity and boundedness of solutions, ensuring biological feasibility. The disease-free equilibrium (DFE) is derived, and the basic reproduction number (R_0) is computed using the next-generation matrix approach. Local stability of the DFE is analyzed using the Jacobian matrix and eigenvalue analysis, while global asymptotic stability is established using a Lyapunov function, showing that TB can be eliminated whenever $R_0 < 1$. Numerical simulations, calibrated with secondary data from the WHO and Nigeria's National TB Programme (2020–2023), are used to show the model dynamics under different intervention scenarios. Results show that scaling up first-line and second-line treatment substantially reduces TB prevalence, infectious burden, drug-resistant TB, and cumulative mortality. Vaccination provides preventive intervention. Sensitivity analysis using partial rank correlation coefficients reveals that treatment-related parameters, particularly second-line treatment, exert the strongest influence on R_0. Overall, the findings highlight the necessity of integrated TB control strategies that combine effective treatment and vaccination to achieve sustained TB reduction and eventual elimination in Nigeria.

Keywords: Tuberculosis, mathematical modeling, drug resistance, basic reproduction number, epidemiology

1. Introduction

Tuberculosis (TB), caused by Mycobacterium tuberculosis, is one of the leading infectious diseases worldwide, posing a major threat to public health. According to the World Health Organization's Global Tuberculosis Report 2024, approximately 10.8 million people fell ill with TB in 2023, with

1.25 million deaths attributed to the disease [1]. The report also indicates that 8.2 million new cases were diagnosed in 2023, marking the highest number on record and highlighting the ongoing rise in incidence. The problem is made worse by drug-resistant TB, especially multidrug-resistant (MDR) and rifampicin-resistant (RR) strains, which are predicted to cause 150,000 fatalities in 2023 [2]. The emergence of drug resistance is often linked to incomplete treatment adherence, genetic mutations in the bacteria, and inadequate healthcare infrastructure [3]. Standard first-line treatments fail in approximately 3.6% of new cases and 18% of previously treated cases [2]. Vaccination with Bacillus Calmette-Guérin (BCG) offers partial protection against severe forms in children but limited efficacy against pulmonary TB in adults [4].

Mathematical modeling provides a powerful tool to dissect transmission dynamics, evaluate interventions, and inform policy [5]. It has become an indispensable tool for understanding the transmission dynamics of TB and evaluating intervention strategies. Early models focused on basic susceptible-infected-recovered (SIR) frameworks, but advancements have incorporated complexities such as latency, vaccination, and drug resistance [6]. For instance, SEIR models have been extended to include latent stages and treatment compartments to better capture TB's prolonged incubation period [7]. Recent reviews highlight the role of models in assessing the impact of diagnostics, vaccines, and control measures on TB epidemiology [6]. Models addressing drug-resistant TB emphasize the need to integrate bacterial fitness costs and resistance acquisition rates, though many lack detailed bacterial dynamics [8]. Vaccination, particularly with the Bacillus Calmette-Guérin (BCG) vaccine, is modeled with waning immunity, as its protective efficacy diminishes over time [9]. Vaccination strategies, preventive (pre-exposure) versus active treatment differ in impact. Preventive approaches reduce susceptible pools, while treatment curtails infectious periods [10].

2. Literature review

The study by [11] revealed that R0 was estimated to be 1.0623 in Kaduna Metropolis, indicating that TB remains endemic. The sensitivity analysis revealed that the transmission rate and contact rate significantly affect the spread of TB, while recovery rates can help reduce transmission overtime. Early detection, quarantine, treatment and public enlightenment campaign are essential preventive efforts to stop diseases' spread [12].

A (SLIR) compartmental model was developed by [13] for the control of TB in Nigeria to analyze equilibrium states, assess the stability of the disease-free state and perform numerical simulations using maple software. The result shows that disease-free equilibrium state is stable; however, complete eradication of TB in Nigeria is challenging with current methods. A recommendation for latent TB treatment was suggested. The model did not account for all real-life complexities of TB transmission and control, such as socio-economic factors and drug resistance. Therefore, further research is needed to incorporate additional variables affecting TB dynamics and explore alternative control strategies beyond the current methods.

[14] employed a mathematical modeling approach to evaluate the efficacy of TB management strategies in Nigeria. The aim of the study is to compare the dynamics of treated and untreated populations, as well as the impact of vaccination on TB transmission. A comprehensive dynamic model was developed to assess TB treatment and vaccination strategies. Simulations were conducted using Berkeley Madonna Software to analyze the outcomes of different treatment scenarios and their effects on TB transmission. The treated population was found to be greater than the untreated population. Vaccination exercise significantly contributed to preventing the spread of TB, with the disease-free equilibrium indicating that the disease is under control. Also, chronic patients showed an initial increase in numbers, followed by a decline and subsequent rise, while treated individuals demonstrated a continuous decrease.

A tuberculosis model with three infected classes (latent, active infectious and drug-resistant population) was developed by [15] to characterize the dynamics of TB, focusing on the impact of optimal control strategies for prevention and early treatment, particularly during the latent stage of the disease. A five-system first order nonlinear differential equation model was used to analyze TB dynamics, incorporating the three distinct infected classes. Numerical simulations

and sensitivity analysis was used to assess the effectiveness of various control strategies on disease containment. The findings indicate that early detection and treatment of TB at the latent stage can reduce the latent population by about 50% at the peak of infection compared to uncontrolled cases. The study also showed that optimal control strategies could significantly enhance recovery rates and reduce the transmission potential of TB. The study has limitations related to the assumptions made in the mathematical model, such as the simplification of human behavior and environmental factors influencing TB trans mission. There is a gap in addressing the socio-economic factors and their influence on TB dynamics, as well as the integration of real-world data to validate the model's predictions. Further research is needed to explore the long-term effects of the proposed control strategies in diverse settings and populations.

Based on a careful analysis of the provided literature review on mathematical modeling of TB transmission, the following research key research gaps have been identified. Inadequate validation, real world testing, and collection of empirical data. The majority of models frequently make assumptions that are not empirically verified against actual data, which limits their generalizability, forecast accuracy, and applicability. To close this gap, model validation was done using actual data from WHO and National TB programs. Also, few models fully account for drug-resistant TB. Extended compartmental model ($S\ V\ L\ I\ T_1\ D_R\ T_2\ R$) was formulated to fill in this gap.

3. Methodology

This study used deterministic compartmental modeling approach to explain the transmission dynamics of TB in a human population. This is an extended SEIR model due to the incorporation of vaccination and drug resistance compartment. Eight compartments such as susceptible (S), vaccinated (V), latent (L), infectious (I), first-line treatment (T_1), drug-resistant (D_R), second-line therapy (T_2), and recovered (R) were created. Model parameters such as birth rate, transmission rate, vaccination rate, waning of immunity, development to active disease, first-line of treatment, treatment failure rate, recovery rate, natural death rate, and TB-induced mortality rate were all represented. A system of ordinary differential equations (ODEs) was used to explain the interactions between these compartments. The model's qualitative characteristics were examined using analytical methods. Biological feasibility of the model was established by examining the positivity and boundedness of solutions. The DFE was derived by equating all infectious compartment to zero. Also, R_0 was calculated using the next–generation matrix method. The local stability of the DFE for $R_0 < 1$ was analyzed using the Jacobian matrix and its corresponding eigenvalues.. Sensitivity analysis was performed to evaluate the impact of preventive vaccination (ρ) and active treatment initiation (τ_1) on R. This provides insights on the effectiveness of different intervention strategies in controlling TB and drug resistant TB dynamics. A secondary data gotten from WHO from 2020 to 2023 was use for the numerical simulation. Statistical tool, R software version 4.4.2 with packages such as desolve, dplyr and ggplot2 were used for the numeric simulation.

3.1. Model formation

The model is formulated as a system of ordinary differential equations (ODEs) capturing the transitions between compartments. The schematic diagram (Figure 1) illustrates the flow.

The parameters used in the model are defined in Table 1.

Differential equations of the model

$$\frac{dS}{dt} = \Lambda - \psi SI - \rho S + \omega V + \theta R - \mu S \tag{1}$$

$$\frac{dV}{dt} = \rho S - \omega V - \mu V \tag{2}$$

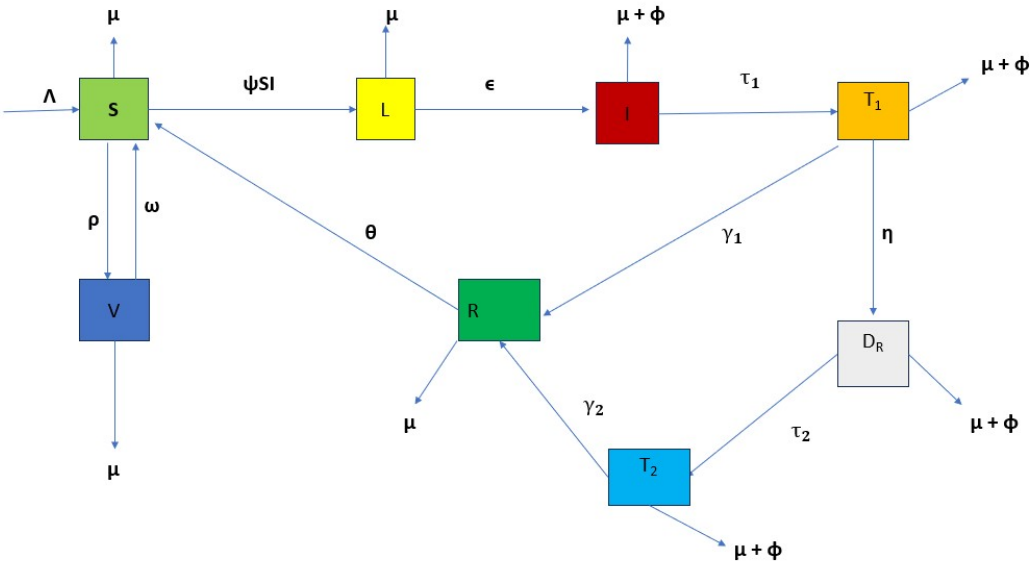

Figure 1. The schematic diagram of the model.

Table 1. Parameter Definition

Parameter	**Definition**
ψ	Transmission rate of TB
ρ	Vaccination rate
ω	Rate of waning immunity from vaccination
ϵ	Progression rate from latent to active TB
τ_1	Rate of entering first-line treatment
η.	Rate of failure of first-line treatment (into D_R)
Λ	Recruitment (birth or immigration) rate
τ_2	Rate of second-line treatment from D_R
γ_1, γ_2	Recovery rates from T_1, T_2, respectively
θ	Loss of immunity or relapse from R
μ	Natural death rate
ϕ	TB induced death rate

$$\frac{dL}{dt} = \psi SI - \epsilon L - \mu L \tag{3}$$
$$\frac{dI}{dt} = \epsilon L - \tau_1 I - (\mu + \phi)I \tag{4}$$
$$\frac{dT_1}{dt} = \tau_1 I - \eta T_1 - \gamma_1 T_1 - (\mu + \phi)T_1 \tag{5}$$
$$\frac{dD_R}{dt} = \eta T_1 - \tau_2 D_R - (\mu + \phi)D_R \tag{6}$$
$$\frac{dT_2}{dt} = \tau_2 D_R - \gamma_2 T_2 - (\mu + \phi)T_2 \tag{7}$$
$$\frac{dR}{dt} = \gamma_1 T_1 + \gamma_2 T_2 - (\theta + \mu)R \tag{8}$$

3.2. Model analysis

Positivity of solutions

To ensure epidemiological relevance, all state variables must remain non-negative for $t > 0$. Consider

$$t_f = \sup\{\, t > 0 : S(t) > 0 \,\}.$$

By contradiction, assume $t_f < \infty$.

From equation (1):

$$\frac{dS}{dt} = \Lambda + \omega V + \theta R - (\rho + \mu)S - \psi SI. \tag{9}$$

Let $\alpha = \rho + \mu$.

Equation (9) becomes

$$\frac{dS}{dt} = \Lambda + \omega V + \theta R - \alpha S - \psi SI.$$

Dropping the positive terms $\omega V + \theta R$,

$$\frac{dS}{dt} \;\geq\; \Lambda - \alpha S - \psi SI. \tag{10}$$

Let

$$p(t) = \psi I + \alpha.$$

Then equation (10) can be written as:

$$\frac{dS}{dt} + p(t)S \;\geq\; \Lambda. \tag{11}$$

The equation (11) is a first order linear differential inequality, where S is the dependent variable and t is the independent variable. Using the integrating factor (I.F) method to solve the differential inequality.

$$I.F = \exp\left(\int_0^t p(t)\,dt\right) = \exp\left(\int_0^t \big(\psi I(t) + \alpha\big)\,dt\right) = \exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right) \tag{12}$$

Multiply both sides of equation (9) with the integrating factor:

$$\begin{aligned}\exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right)\,\frac{dS}{dt} + \big(\psi I(t) + \alpha\big)\,\exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right) S \\ \geq \Lambda \exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right).\end{aligned} \tag{13}$$

Regrouping equation (13), we get:

$$\frac{d}{dt}\left[S(t)\,\exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right)\right] \;\geq\; \Lambda \exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right)$$

Integrating both sides of (12) with respect to t:

$$S(t)\,\exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right) \;\geq\; \int_0^t \Lambda \exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right)\,dt + S(0). \tag{14}$$

Making $S(t)$ the subject in (14):

$$S(t) \;\geq\; \exp\left(-\alpha t - \int_0^t \psi I(t)\,dt\right)\left[\int_0^t \Lambda \exp\left(\alpha t + \int_0^t \psi I(t)\,dt\right)\,dt + S(0)\right]. \tag{15}$$

Since $\Lambda > 0$, $S(0) > 0$, and all exponential terms are positive, the integral is positive. So, the right-hand side of (15) is strictly positive.

Suppose $t_f < \infty$ (that is, $S(t)$ hits 0 at t_f). Evaluating at $t = t_f$:

$$S(t_f) \geq \exp\left(-\alpha t_f - \int_0^{t_f} \psi I(t)\, dt\right) \left[\int_0^{t_f} \Lambda \exp\left(\alpha t + \int_0^t \psi I(t)\, dt\right) dt + S(0)\right] > 0. \tag{16}$$

The inequality (16) shows $S(t_f) > 0$, contradicting the definition of t_f (where $S(t_f) \leq 0$). Thus, $t_f = \infty$ and $S(t) > 0$ for all $t > 0$.

Therefore, the positivity of the state variable S for all $t > 0$ is proved.

Similarly, the positivity of V, L, I, T_1, T_2, D_R, and R can also be established. Hence, all solutions of the TB model remain positive for any positive initial conditions.

Boundedness of solutions

Making use of a Lyapunov-like approach, we show positive invariance of the region

$$\Omega = \left\{(S, V, L, I, T_1, T_2, D_R, R) \in \mathbb{R}_+^8 : N \leq \tfrac{\Lambda}{\mu}\right\},$$

where

$$N = S + V + L + I + T_1 + T_2 + D_R + R.$$

The total population satisfies:

$$\frac{dN}{dt} = \Lambda - \mu N - \phi(I + T_1 + D_R + T_2) \tag{17}$$

Since $-\phi(I + T_1 + D_R + T_2) \leq 0$, it follows that

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

Solving the inequality gives:

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \tfrac{\Lambda}{\mu}\right) e^{-\mu t}. \tag{18}$$

As $t \to \infty$, we have

$$N(t) \leq \frac{\Lambda}{\mu}.$$

Therefore, solutions are bounded in the region

$$\Omega = \left\{(S, V, L, I, T_1, T_2, D_R, R) \in \mathbb{R}_+^8 : N \leq \tfrac{\Lambda}{\mu}\right\}.$$

Hence, all solutions are bounded for all $t \geq 0$, and the model is biologically realistic (the total population does not explode).

Disease-free equilibrium (DFE)

The disease-free equilibrium (DFE) given by equation ((1) to (8)) represents a steady state where the disease is absent, meaning the compartments associated with infection and treatment are zero. For the given system, we need to identify the disease-related compartments and set them to zero, then solve for the non-disease compartments (susceptible, vaccinated, and recovered) such that all time derivatives are zero.

At the disease-free equilibrium (DFE), we have

$$L = I = T_1 = D_R = T_2 = 0.$$

From equation (8), it follows that

$$R = 0.$$

From equation (3), we obtain

$$V^* = \frac{\Lambda\rho}{\mu(\omega + \rho + \mu)}. \tag{19}$$

Hence,

$$S^* = \frac{\Lambda(\omega + \mu)}{\mu(\omega + \rho + \mu)}. \tag{20}$$

Therefore, the DFE is

$$\left(S^*, V^*, L^*, I^*, T_1^*, DR^*, T_2^*, R^*\right) = \left(\frac{\Lambda(\omega+\mu)}{\mu(\omega+\rho+\mu)},\ \frac{\Lambda\rho}{\mu(\omega+\rho+\mu)},\ 0,0,0,0,0,0\right), \tag{21}$$

provided that the denominator is strictly positive.

3.3. Basic reproduction number R_0

The basic reproduction number (R_0) represents the average number of secondary infections caused by a single infected individual in a completely susceptible population. If $R_0 > 1$, the disease can spread; if $R_0 < 1$, the disease dies out. For TB model above, we will use the next-generation matrix approach to compute R_0. This method involves identifying the infection and transition rates for the disease compartments and constructing the matrices F(new infections) and V(transitions out of compartments), then calculating R_0 as the dominant eigenvalue of FV^1. The next-generation matrix method helps us calculate this by looking at how infections start and how people move out of being infectious (e.g., through recovery or death). The infected groups are L(latent), I(active infected), T_1 (first line of treatment for TB), D_R (drug-resistant infected), and T_2 (treatment for resistant TB). We focus only on these groups to build F and V.

The model assumes that only two compartments are infectious namely: I (active untreated) and D_R (drug resistant), whereas people on effective first or second line treatment (T_1, T_2) have greatly reduced or zero transmission. All new infections enter the latent compartment L.

The model assumes that D_R has the same transmissibility as I. Therefore, the rate at which new infections appear in the system per unit time $\psi = \beta\ \mathrm{S}\ \mathrm{I} + \beta\ \mathrm{S}\ D_R$.

Take partial derivatives with respect to each infected compartment, evaluated at the disease-free equilibrium (DFE) At the DFE: $\mathrm{L} = \mathrm{I} = T_1 = D_R = T_2 = 0$

Therefore,

$$F = \begin{bmatrix} 0 & \beta S & 0 & \beta S & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{22}$$

Matrix V (Transitions out)

V shows how people leave the infected compartments (e.g recovering, progression, dying or moving to another stage). We look at the terms that reduce the number in each compartment. The transition terms are the negative of the derivatives of the right-hand sides of the infected compartment equations with respect to their own compartments, evaluated at the DFE.

V also includes inflows from one compartment to another (Off- diagonal terms). The partial derivatives of these inflows are:

Therefore,

$$V = \begin{bmatrix} \epsilon+\mu & 0 & 0 & 0 & 0 \\ -\epsilon & \tau_1+\mu+\phi & 0 & 0 & 0 \\ 0 & -\tau_1 & \eta+\gamma_1+\mu+\phi & 0 & 0 \\ 0 & 0 & -\eta & \tau_2+\mu+\phi & 0 \\ 0 & 0 & 0 & -\tau_2 & \gamma_2+\mu+\phi \end{bmatrix}. \tag{23}$$

Let " a, b, c, d, e" denote the diagonal elements for simplicity. $\mathrm{a} = \epsilon + \mu$
$\mathrm{b} = \tau_1 + \mu + \phi$
$\mathrm{c} = \eta + \gamma_1 + \mu + \phi$
$\mathrm{d} = \tau_2 + \mu + \phi$
$\mathrm{e} = \gamma_2 + \mu + \phi$

Then,

$$V = \begin{bmatrix} a & 0 & 0 & 0 & 0 \\ -\epsilon & b & 0 & 0 & 0 \\ 0 & -\tau_1 & c & 0 & 0 \\ 0 & 0 & -\eta & d & 0 \\ 0 & 0 & 0 & -\tau_2 & e \end{bmatrix}. \tag{24}$$

Since all diagonal elements are positive and non-zero, so V is invertible.

$$V^{-1} = \begin{bmatrix} \frac{1}{a} & 0 & 0 & 0 & 0 \\ \frac{\epsilon}{ab} & \frac{1}{b} & 0 & 0 & 0 \\ \frac{\tau_1\epsilon}{abc} & \frac{\tau_1}{bc} & \frac{1}{c} & 0 & 0 \\ \frac{\eta\tau_1\epsilon}{abcd} & \frac{\eta\tau_1}{bcd} & \frac{\eta}{cd} & \frac{1}{d} & 0 \\ \frac{\eta\tau_1\tau_2\epsilon}{abcde} & \frac{\eta\tau_1\tau_2}{bcde} & \frac{\eta\tau_2}{cde} & \frac{\tau_2}{de} & \frac{1}{e} \end{bmatrix}. \tag{25}$$

$$FV^{-1} = \left[\beta S\left(\tfrac{\varepsilon}{ab} + \tfrac{\varepsilon\tau_1\eta}{abcd}\right),\ 0,\ 0,\ 0,\ 0\right]$$

$$R_0 = \frac{\beta\varepsilon\Lambda(\omega+\mu)}{\mu(\omega+\rho+\mu)(\varepsilon+\mu)(\tau_1+\mu+\phi)}\left(1 + \frac{\tau_1\eta}{(\eta+\gamma_1+\mu+\phi)(\tau_2+\mu+\phi)}\right). \tag{26}$$

3.4. Local stability of the DFE

Local stability analysis ascertains whether a dynamical system returns to an equilibrium point after minor disturbances. This achieved by concentrating on the behavior of the system of that fixed point and using methods like Jacobian matrices and eigenvalues to see if perturbations shrink (stable) or grow (unstable).

In this section, the Jacobian matrix is used to determine the local stability of the system. Substituting the value of S from equation (19),

$$S = S^* = \frac{\Lambda(\omega+\mu)}{\mu(\omega+\rho+\mu)}$$

Theorem 1. *The disease-free equilibrium of the model is Locallylly Asymptotically Stable (LAS) if* $R_0 < 1$.

Proof. The Jacobian matrix for the equation ((1) - (8)) is given by:

$$J = \begin{bmatrix} -\beta I-\rho-\mu & \omega & 0 & -\beta S & 0 & 0 & 0 & \theta \\ \rho & -\omega-\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta I & 0 & -\epsilon-\mu & \beta S & 0 & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -\tau_1-\mu-\phi & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & -\eta-\gamma_1-\mu-\phi & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta & -\tau_2-\mu-\phi & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 & -\gamma_2-\mu-\phi & 0 \\ 0 & 0 & 0 & 0 & \gamma_1 & 0 & \gamma_2 & -(\theta+\mu) \end{bmatrix}, \tag{27}$$

and $I = 0$ into the Jacobian matrix (27).

$$J_{DFE} = \begin{bmatrix} -\rho-\mu & \omega & 0 & -\beta S^* & 0 & 0 & 0 & \theta \\ \rho & -\omega-\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\epsilon-\mu & \beta S^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -\tau_1-\mu-\phi & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & -\eta-\gamma_1-\mu-\phi & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta & -\tau_2-\mu-\phi & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 & -\gamma_2-\mu-\phi & 0 \\ 0 & 0 & 0 & 0 & \gamma_1 & 0 & \gamma_2 & -(\theta+\mu) \end{bmatrix}. \tag{28}$$

To determine the stability, we require the eigenvalues (roots of the characteristic equation $\det(J_{\mathrm{DFE}} - \lambda I) = 0$). Since the matrix is not triangular matrix due to an off-diagonals βS term, so the eigenvalues cannot simply be the diagonal entries. Gaussian elimination method was used to solve for the eigenvalues.

$$J_{DFE} - \lambda I = \begin{bmatrix} -\rho-\mu-\lambda & \omega & 0 & -\beta S^* & 0 & 0 & 0 & \theta \\ \rho & -\omega-\mu-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\epsilon-\mu-\lambda & \beta S^* & 0 & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -\tau_1-\mu-\phi-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & -\eta-\gamma_1-\mu-\phi-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta & -\tau_2-\mu-\phi-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 & -\gamma_2-\mu-\phi-\lambda & 0 \\ 0 & 0 & 0 & 0 & \gamma_1 & 0 & \gamma_2 & -(\theta+\mu)-\lambda \end{bmatrix}. \quad (29)$$

The last column of matrix above (3.29) has a diagonal term – ($\theta + \mu$) - λ only. Therefore, λ I = – ($\theta + \mu$) Eliminate the last row and the last column in (3.29), a new matrix J_1 is formed:

$$J_1 = \begin{bmatrix} -\rho-\mu-\lambda & \omega & 0 & -\beta S^* & 0 & 0 & 0 \\ \rho & -\omega-\mu-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\epsilon-\mu-\lambda & \beta S^* & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -\tau_1-\mu-\phi-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & -\eta-\gamma_1-\mu-\phi-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta & -\tau_2-\mu-\phi-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 & -\gamma_2-\mu-\phi-\lambda \end{bmatrix}. \quad (30)$$

Independent blocks are identified and split into independent subsystems; Gaussian elimination method gives room for each block to be treated separately.

Vaccinated & Susceptible subsystem

$$\begin{pmatrix} -\rho-\mu-\lambda & \omega \\ \rho & -\omega-\mu-\lambda \end{pmatrix}$$

The determinant of the matrix is given by

$$\det = (-\rho-\mu-\lambda)(-\omega-\mu-\lambda) - \rho\omega = 0. \quad (31)$$

Equation (31) can be rewritten as

$$(\lambda+\rho+\mu)(\lambda+\omega+\mu) - \rho\omega = 0.$$

Expanding gives

$$\lambda^2 + \lambda(2\mu+\rho+\omega) + \mu(\mu+\rho+\omega) = 0.$$

This quadratic equation is in the standard form

$$(\lambda+a)(\lambda+b) = \lambda^2 + (a+b)\lambda + ab,$$

where

$$a = \mu, \qquad b = \rho+\omega+\mu.$$

Hence,

$$a+b = \rho+\omega+2\mu, \qquad ab = \mu(\rho+\omega+\mu).$$

Therefore,

$$(\lambda+\mu)(\lambda+\rho+\omega+\mu) = \lambda^2 + (\rho+\omega+2\mu)\lambda + \mu(\rho+\omega+\mu) = 0.$$

Hence, the full characteristic equation is given by

$$(\lambda+\mu)(\lambda+\rho+\omega+\mu) = 0.$$

Setting each factor equal to zero gives:

$$\lambda+\mu = 0 \quad \Rightarrow \quad \lambda_2 = -\mu,$$
$$\lambda+\rho+\omega+\mu = 0 \quad \Rightarrow \quad \lambda_3 = -(\rho+\omega+\mu).$$

Since all parameters are positive, it follows that

$$\lambda_2 < 0 \quad \text{and} \quad \lambda_3 < 0.$$

The eigenvalues of the Vaccinated and Susceptible subsystem are all negative. This implies that the subsystem is stable.

Latent & Infected subsystem

$$\begin{pmatrix} \epsilon - \mu - \lambda & \beta S \\ \epsilon & \tau_1 - \mu - \phi - \lambda \end{pmatrix}$$

$$\det = (\lambda + \varepsilon + \mu)(\lambda + \tau_1 + \mu + \phi) - \varepsilon\beta S = 0. \tag{32}$$

From equation (20), the disease–free equilibrium value of the susceptible population is

$$S = \frac{\Lambda(\omega + \mu)}{\mu(\omega + \rho + \mu)}.$$

Substituting S into the determinant yields

$$(\lambda + \varepsilon + \mu)(\lambda + \tau_1 + \mu + \phi) - \varepsilon\beta\frac{\Lambda(\omega + \mu)}{\mu(\omega + \rho + \mu)} = 0. \tag{33}$$

Expanding equation (33) gives

$$\lambda^2 + (\tau_1 + 2\mu + \phi + \varepsilon)\lambda + (\mu + \varepsilon)(\tau_1 + \mu + \phi) - \varepsilon\beta\frac{\Lambda(\omega + \mu)}{\mu(\omega + \rho + \mu)} = 0.$$

This gives the quadratic characteristic equation

$$\lambda^2 + a_1\lambda + a_0 = 0,$$

where

$$a_1 = \tau_1 + 2\mu + \phi + \epsilon,$$

and

$$a_0 = (\mu + \epsilon)(\tau_1 + \mu + \phi) - \epsilon\beta\frac{\Lambda(\omega + \mu)}{\mu(\omega + \rho + \mu)}.$$

By the Routh–Hurwitz criterion for a second-degree polynomial,

$$p(\lambda) = \lambda^2 + a_1\lambda + a_0, \qquad a_0, a_1 \in \mathbb{R},$$

all roots of $p(\lambda)$ have negative real parts if and only if

$$a_1 > 0 \quad \text{and} \quad a_0 > 0.$$

Since all model parameters are positive, it follows that

$$\lambda_4 < 0 \quad \text{and} \quad \lambda_5 < 0.$$

Hence, all eigenvalues of this subsystem have negative real parts, and the latent and infected subsystem is locally asymptotically stable.

Treatment & Resistance block

$$\begin{pmatrix} -\eta - \gamma_1 - \mu - \phi - \lambda & 0 & 0 \\ \eta & -\tau_2 - \mu - \phi - \lambda & 0 \\ 0 & \tau_2 & -\gamma_2 - \mu - \phi - \lambda \end{pmatrix}$$

This subsystem has an upper triangular Jacobian matrix.

Therefore, the eigenvalues are given by

$$\lambda_6 = -(\eta + \gamma_1 + \mu + \phi), \qquad \lambda_7 = -(\tau_2 + \mu + \phi), \qquad \lambda_8 = -(\gamma_2 + \mu + \phi).$$

Since all parameters are positive, it follows that

$$\lambda_6 < 0, \quad \lambda_7 < 0, \quad \lambda_8 < 0.$$

Hence, all eigenvalues of this subsystem are strictly negative.

However, the full stability includes all the subsystems considered, which were analyzed separately and all have negative eigenvalues.

Since all eigenvalues are negative, the disease-free equilibrium is locally asymptotically stable (LAS), infection dies out. Therefore, $R_0 < 1$ □

3.5. Global stability of the DFE

Global stability of epidemiological model is necessary and makes the model predictable as it guarantees that the eradication of TB infection is independent of the initial size of the population. Global asymptotic stability (GAS) of an epidemiological model can be established by constructing appropriate Lyapunov function.

Theorem 2. *The disease-free equilibrium of the model is Globally Asymptotically stable (GAS) if* $R_0 < 1$.

Proof. The force of infection is defined as

$$\psi = \beta I + \beta D_R.$$

Hence, new infections occur at the rate

$$\psi S = \beta S(I + D_R).$$

To establish the global asymptotic stability (GAS) of the model, we consider the disease–free equilibrium (DFE) of the system given by equations (1)–(8). The infectious compartments are I and D_R; however, infection progresses through the chain

$$L \longrightarrow I \longrightarrow T_1 \longrightarrow D_R \longrightarrow T_2.$$

We therefore consider the infected subsystem

$$(L, I, T_1, D_R, T_2).$$

From the model equations, the dynamics of the infected subsystem are given by

$$\begin{aligned}
\frac{dL}{dt} &= \beta S(I + D_R) - (\epsilon + \mu)L, \\
\frac{dI}{dt} &= \epsilon L - (\tau_1 + \mu + \phi)I, \\
\frac{dT_1}{dt} &= \tau_1 I - (\eta + \gamma_1 + \mu + \phi)T_1, \\
\frac{dD_R}{dt} &= \eta T_1 - (\tau_2 + \mu + \phi)D_R, \\
\frac{dT_2}{dt} &= \tau_2 D_R - (\gamma_2 + \mu + \phi)T_2.
\end{aligned}$$

Let $c = \beta S$.

Choose a linear Lyapunov function defined by

$$F = a_1 L + a_2 I + a_3 T_1 + a_4 D_R + a_5 T_2, \qquad a_i > 0,\ i = 1, 2, \ldots, 5. \tag{34}$$

The time derivative of F, where a dot denotes differentiation with respect to time t, is given by

$$\dot{F} = a_1 \dot{L} + a_2 \dot{I} + a_3 \dot{T_1} + a_4 \dot{D_R} + a_5 \dot{T_2}. \tag{35}$$

Substituting each equation of the infected subsystem into $\dot{F}$:

$$\begin{aligned}
\dot{F} = a_1\big[c(I + D_R) - (\varepsilon + \mu)L\big] + a_2\big[\varepsilon L - (\tau_1 + \mu + \phi)I\big] \\
+ a_3\big[\tau_1 I - (\eta + \gamma_1 + \mu + \phi)T_1\big] + a_4\big[\eta T_1 - (\tau_2 + \mu + \phi)D_R\big] \\
+ a_5\big[\tau_2 D_R - (\gamma_2 + \mu + \phi)T_2\big].
\end{aligned} \tag{36}$$

Collecting like terms in equation (36):

$$\begin{aligned}
\dot{F} = L\big[-a_1(\epsilon + \mu) + a_2\epsilon\big] + I\big[a_1 c - a_2(\tau_1 + \mu + \phi) + a_3\tau_1\big] \\
+ T_1\big[-a_3(\eta + \gamma_1 + \mu + \phi) + a_4\eta\big] \\
+ D_R\big[a_1 c - a_4(\tau_2 + \mu + \phi) + a_5\tau_2\big] - a_5(\gamma_2 + \mu + \phi)T_2.
\end{aligned} \tag{37}$$

Let $a_1 = 1$ and $a_5 > 0$.

$$-a_1(\epsilon + \mu) + a_2\epsilon = 0,$$

Therefore

$$a_2 = \frac{\epsilon + \mu}{\epsilon}.$$

Next, from

$$a_1 c - a_2(\tau_1 + \mu + \phi) + a_3\tau_1 = 0,$$

$$a_3 = \frac{a_2(\tau_1 + \mu + \phi) - c}{\tau_1} = \frac{(\epsilon + \mu)(\tau_1 + \mu + \phi) - c}{\epsilon\,\tau_1}.$$

Similarly, from the coefficient of T_1,

$$-a_3(\eta + \gamma_1 + \mu + \phi) + a_4\eta = 0,$$

$$a_4 = a_3\frac{\eta + \gamma_1 + \mu + \phi}{\eta} = \frac{(\eta + \gamma_1 + \mu + \phi)(\epsilon + \mu)(\tau_1 + \mu + \phi) - c}{\epsilon\,\eta\,\tau_1}.$$

Substituting the values of a_i in equation (3.37) and canceling:

$$\dot{F} = D_R\big[a_1 c - a_4(\tau_2 + \mu + \phi) + a_5\tau_2\big] - a_5(\gamma_2 + \mu + \phi)T_2. \tag{38}$$

Since $a_5 > 0$, $(\gamma_2 + \mu + \phi) > 0$, and $T_2 \geq 0$, it follows that

$$-a_5(\gamma_2 + \mu + \phi)T_2 < 0.$$

Moreover, since $D_R \geq 0$, the sign of the first term depends entirely on

$$a_1 c - a_4(\tau_2 + \mu + \phi).$$

Using $a_1 = 1$ and the expression for a_4, the equation becomes:

$$a_1 c - a_4(\tau_2 + \mu + \phi) = c - a_3\frac{(\eta + \gamma_1 + \mu + \phi)(\tau_2 + \mu + \phi)}{\eta}. \tag{39}$$

Substituting the expression for a_3 in equation (3.39) yields

$$= c - \frac{\big[a_2(\tau_1 + \mu + \phi) - c\big](\eta + \gamma_1 + \mu + \phi)(\tau_2 + \mu + \phi)}{\eta\,\tau_1}. \tag{40}$$

Let

$$A = \frac{(\eta + \gamma_1 + \mu + \phi)(\tau_2 + \mu + \phi)}{\eta\,\tau_1}.$$

Substituting A in equation (40):

$$= c - A\big[a_2(\tau_1 + \mu + \phi) - c\big].$$

Expanding the bracket, we obtain

$$= c - Aa_2(\tau_1 + \mu + \phi) + Ac.$$

$$\dot{F} = c(1 + A) - Aa_2(\tau_1 + \mu + \phi) \tag{41}$$

Substituting

$$a_2 = \frac{\varepsilon + \mu}{\varepsilon}$$

into equation (4):

$$a_1 c - a_4(\tau_2 + \mu + \phi) = c(1 + A) - \frac{\epsilon + \mu}{\epsilon}(\tau_1 + \mu + \phi)A. \tag{42}$$

The basic reproduction number of the model is given by

$$R_0 = c\,\frac{\epsilon}{\epsilon + \mu}\,\frac{1}{\tau_1 + \mu + \phi}\left(1 + \frac{\tau_1\eta}{(\eta + \gamma_1 + \mu + \phi)(\tau_2 + \mu + \phi)}\right). \tag{43}$$

Therefore,

$$1 + \frac{1}{A} = 1 + \frac{\tau_1 \eta}{(\eta + \gamma_1 + \mu + \phi)(\tau_2 + \mu + \phi)}.$$

Hence, the basic reproduction number can be written as

$$R_0 = c\,\frac{\epsilon}{\epsilon + \mu}\,\frac{1}{\tau_1 + \mu + \phi}\left(1 + \frac{1}{A}\right). \tag{44}$$

Rearranging gives

$$c(1 + A) = \frac{\epsilon + \mu}{\epsilon}(\tau_1 + \mu + \phi)\,A\,R_0. \tag{45}$$

Substituting equation (45) into equation (42), we obtain

$$a_1 c - a_4(\tau_2 + \mu + \phi) = \frac{\epsilon + \mu}{\epsilon}(\tau_1 + \mu + \phi)\,A\,(R_0 - 1).$$

Hence,

$$a_1 c - a_4(\tau_2 + \mu + \phi) = K(\tau_1 + \mu + \phi)(R_0 - 1),$$

where

$$K = \frac{\epsilon + \mu}{\epsilon}\frac{\eta + \gamma_1 + \mu + \phi}{\tau_1 \eta} > 0.$$

If $R_0 < 1$, then

$$K(\tau_1 + \mu + \phi)(R_0 - 1) < 0.$$

Since $D_R \geq 0$, $T_2 \geq 0$, and all model parameters are positive, it follows that

$$\dot{F} \leq 0 \quad \text{for all states in the feasible region.}$$

Moreover, $\dot{F} = 0$ at DFE points implies $D_R = 0$ and $T_2 = 0$. From the model equations, this further implies that

$$T_1 = I = L = 0.$$

By LaSalle's Invariance Principle, the disease–free equilibrium of the model is globally asymptotically stable whenever

$$R_0 < 1.$$

□

3.6. Numerical simulations

Given the focus of the study on DFE stability analysis, R_0, vaccination, treatment and drug resistance, a deterministic forward simulation of the ODE model was used to illustrate the time evolution of TB compartments under intervention scenarios and to numerically validate the analytical results. This is to quantify how vaccination and treatment initiation affect TB prevalence, drug-resistance prevalence, total infectious burden and TB mortality. This analysis is done using parameter values gotten from WHO and Nigeria National TB Programme data and other related literatures. Initial conditions are $S(0) = 150000000$, $V(0) = 10000000$, $L(0) = 50000000$, $I(0) = 500000$, $T_1(0) = 200000$, $D_R(0) = 20000$, $T_2(0) = 10000$, $R(0) = 7270000$. The model parameter values are taken as Table 2.

The Figure 2 shows the simulation of active tuberculosis (TB) prevalence in Nigeria over a 5-year period under different combinations of treatment and vaccination strategies.

High treatment rate produces the lowest TB prevalence. Although TB cases initially rise, the curve stabilizes at a significantly lower level (around 6-7 million cases). The high first-line treatment rate (τ_1) ensures rapid detection and cure of TB cases and the high second-line treatment rate (τ_2) effectively reduces the pool of drug-resistant TB cases, preventing prolonged infectiousness. This combination strongly suppresses TB transmission and progression. On the other hand, low treatment rate produces the highest TB burden. This implies that delayed or inadequate treatment leads to prolonged infectiousness and accumulation of active cases. High vaccination reduces the number of individuals progressing to active TB while low vaccination increases the susceptible population. When vaccination coverage is low, treatment efforts alone are insufficient to reduce long term TB prevalence.

Table 2. Model parameter values.

Descriptin	Symbol	Value	Source
Transmission rate of TB	ψ	2.5338×10^{-5}	[17]
Vaccination rate	ρ	0.74	[18]
Rate of waning immunity from vaccination	ω	0.25	[17]
Progression rate from latent to active TB	ϵ	0.05	[19]
Rate of entering first-line treatment	τ_1	1.5	[17]
Rate of failure of first-line treatment	$\eta.$	0.14	[20]
Recruitment (birth or immigration) rate	Λ	6.7	[16]
Rate of second-line treatment from D_R	τ_2	1.0	Assumed
Recovery rate from T_1	γ_1	1.86	[20]
Recovery rate from T_2,	γ_2	0.71	[21]
Loss of immunity or relapse from R	θ	0.1	[22]
Natural death rate	μ	0.0087	[23]
TB induced death rate	ϕ	0.365	[19]

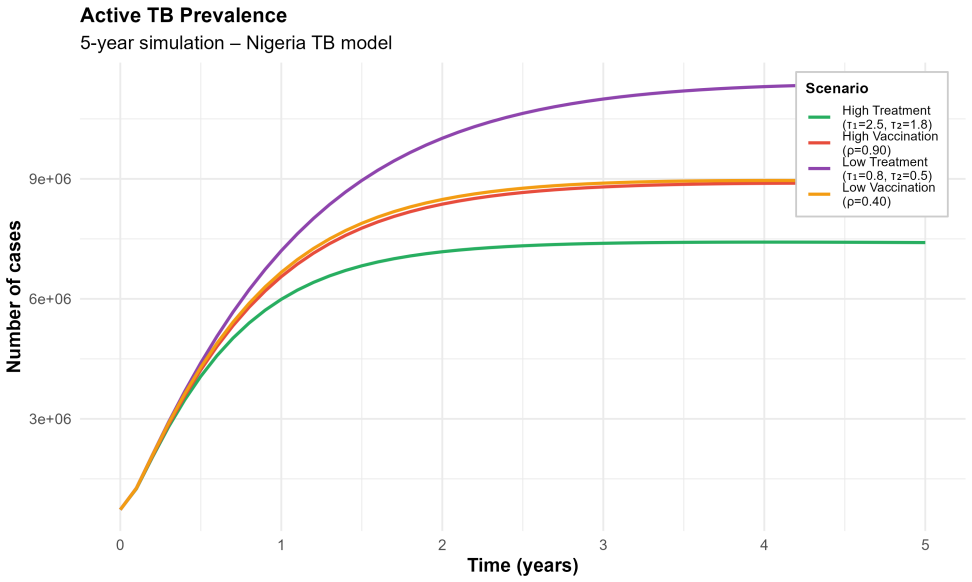

Figure 2. Active TB prevalence.

Overall, the simulation demonstrates that scaling up of first and second line TB treatment in combination with high vaccination coverage would greatly reduce active TB prevalence over time. In contrast, low treatment and vaccination levels lead to persistently high TB burden. These findings emphasize the importance of integrated TB control strategies in high burden settings such as Nigeria.

The Figure 3 illustrates the simulated dynamics of the total infectious TB burden in Nigeria.

This scenario yields the lowest infectious burden throughout the simulation, stabilizing at approximately 3.5 million cases. High first line treatment rate (τ_1) rapidly transfers infectious individuals into treatment, reducing the time spent in the untreated infectious class while high second line treatment (τ_2) limits prolonged infectiousness associated with drug-resistant TB. This demonstrates that aggressive treatment minimizes the pool of infectious individuals, thereby reducing community level transmission potential. In the case of low treatment rate results in the highest infectious burden, exceeding 8 million cases by year 5. This represents a worst case control scenario, characterized by persistent transmission and a large reservoir of infectious TB, posing a substantial public health risk. For high vaccination coverage rate, the infectious burden

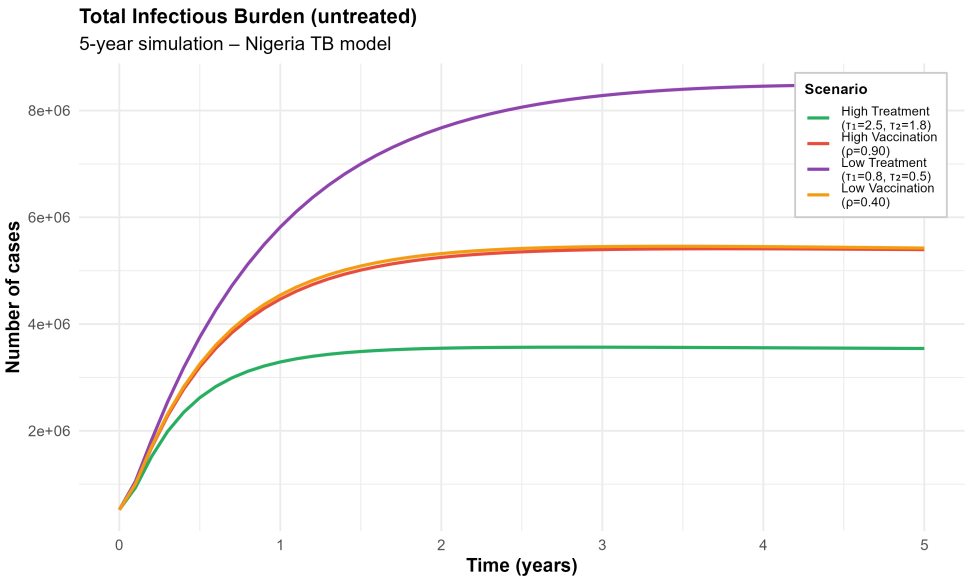

Figure 3. Total infectious TB burden.

stabilizes at a higher level (5.3 million cases) compared to the high-treatment scenario. It reduces susceptibility and disease progression. High vaccination alone cannot adequately suppress the infectious pool without effective treatment scale-up, especially in high burden settings. In contrast, low vaccination produces an infectious burden similar to, but slightly higher than, the high vaccination case. It increases the susceptible population, leading to sustained inflow into the infectious class. When vaccination coverage is low, treatment alone struggles to prevent the build-up of infectious cases, reinforcing the importance of preventive interventions.

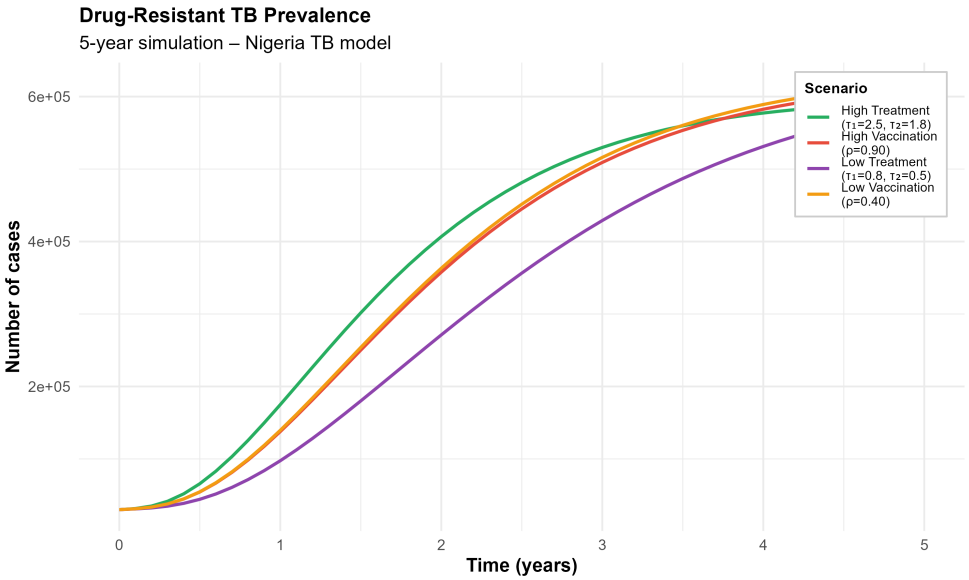

Figure 4. Drug-resistant TB prevalence.

The Figure 4 presents the simulated evolution of drug-resistant tuberculosis (D_R-TB) prevalence in Nigeria. High treatment rate shows a rapid early increase in detected drug-resistant TB cases, followed by stabilization at a moderate level. This scenario shows a rapid early increase in detected D_R-TB cases, followed by stabilization at a moderate level. Although drug-resistant TB prevalence rises initially, effective second-line treatment prevents uncontrolled growth, leading to a lower long term burden. Low treatment scenario shows a slower early increase but reaches a

high drug resistance prevalence. This represents a structurally weak TB control setting, where D_R-TB accumulates over time due to poor treatment and prevention. High vaccination rate steadily increases the drug-resistant TB prevalence and stabilizes at a slightly higher level than in the high treatment scenario. Vaccination reduces new infections but has limited direct impact on existing resistant cases which makes it difficult to control the resistant cases without adequate second line treatment capacity. Drug-resistant has the highest prevalence in low vaccination scenario. It increases overall TB transmission, thereby enlarging the pool of individuals at risk of developing resistance.

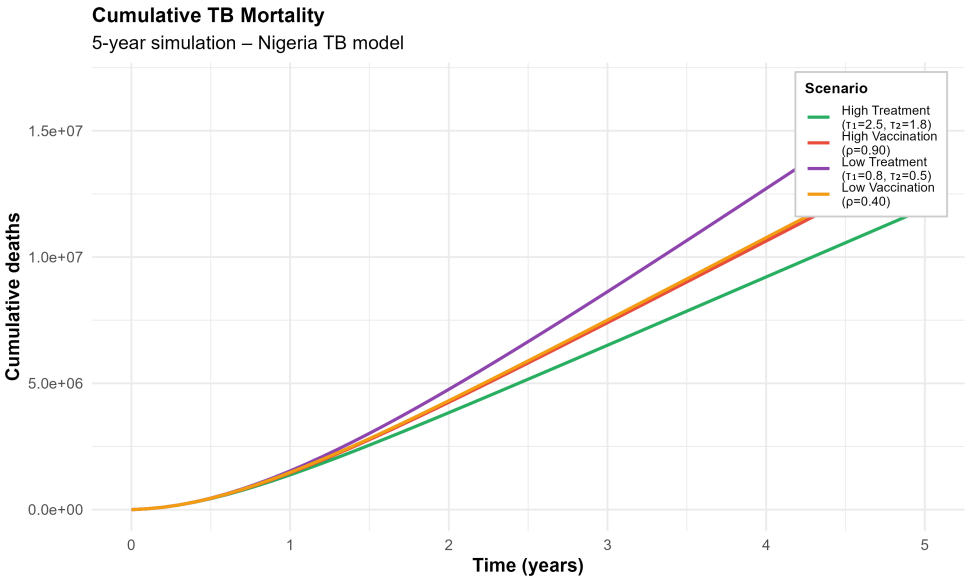

Figure 5. Cumulative TB mortality.

Figure 5 presents the cumulative number of TB related deaths in Nigeria. Treatment rate plays a very dominant role in shaping mortality trajectories. The lowest cumulative mortality is observed in the high treatment scenario while the highest cumulative mortality occurs under low treatment coverage. Effective first-line treatment rapidly reduces infectiousness and progression to severe disease while second-line treatment is critical for managing treatment failure and drug-resistant TB, thereby preventing excess deaths. The widening gap between the high- and low-treatment curves over time indicates that delayed or inadequate treatment has compounding long-term mortality effects. Vaccination modifies mortality indirectly by reducing susceptibility and slowing disease progression. It is an important preventive intervention. However, vaccination alone does not compensate for weak treatment systems. Treatment scale-up yields a stronger short- to medium-term mortality reduction in high-burden settings.

In Figure 6, second-line treatment (τ_2) shows the largest positive PRCC, followed closely by first-line treatment (τ_1). This indicates that changes in treatment-related parameters would greatly influence R_0 in the model. The high sensitivity of R_0 to (τ_1) and (τ_2) underscores the critical role of treatment availability, adherence, and effectiveness, especially in managing drug-resistant TB cases. Recovery rate (γ_1) has a moderately large positive PRCC, indicating a strong association with R_0. This parameter typically represents recovery or removal from the infectious class through treatment or immune clearance. Small variations in recovery-related processes significantly alter R_0, which highlight the importance of treatment success rates, duration of infectiousness, and patient outcomes. Vaccination Coverage (ρ) exhibits a positive PRCC of moderate magnitude. This shows that vaccination plays a meaningful role in shaping R_0, though its influence is weaker than treatment-related parameters. Progression from latent to active TB (ϵ) and treatment failure or relapse rate (η) show negative PRCC values. These negative correlations indicate that increasing these parameters leads to a reduction in R_0 within the model structure. This reflects that increased progression or relapse may shift individuals into compartments with higher mortality.

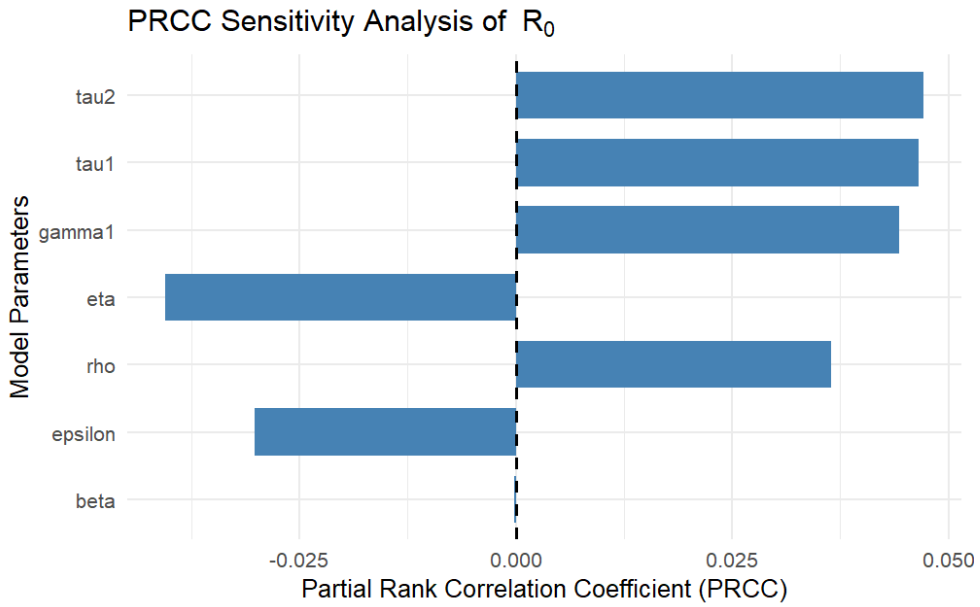

Figure 6. Sensitivity Analysis of R_0

4. Results and discussion

The TB transmission model was shown to be biologically and mathematically determined, as all solutions were proven to be positive and bounded for all time. This guarantees epidemiological realism, ensuring that population compartments remain non-negative and finite. The DFE was derived and analyzed using the next generation matrix approach. Both local and global stability analyses demonstrate that the DFE is locally and globally asymptotically stable whenever $R_0 < 1$, implying that TB can be eliminated regardless of initial conditions if control efforts are sufficient to keep transmission below this threshold.

Numerical simulations, calibrated using WHO and Nigeria National TB Programme data. Results show that treatment-related parameters dominate TB dynamics. High first-line treatment (τ_1) rapidly reduces active TB prevalence by shortening the infectious period, while high second-line treatment (τ_2) effectively limits the accumulation and persistence of drug-resistant TB. In contrast, low treatment coverage leads to sustained transmission, higher active TB prevalence, and a growing reservoir of infectious and drug-resistant cases. Vaccination reduces susceptibility and progression to active disease, thereby contributing to lower prevalence and infectious burden. However, simulations consistently show that vaccination alone is insufficient to control TB in high-burden settings. Its impact is strongest when combined with effective treatment scale-up, highlighting the complementary roles of preventive and curative interventions. Cumulative TB mortality is most sensitive to treatment intensity, with high treatment scenarios yielding the lowest deaths, while inadequate treatment produces compounding mortality over time.

Sensitivity analysis of R_0 using PRCC confirms that second-line and first-line treatment rates produce the strongest influence on transmission potential, followed by recovery and vaccination parameters. Overall, the results shows that integrated TB control strategies by prioritizing robust multi-line treatment alongside vaccination are essential for reducing TB transmission, mortality, and drug resistance in Nigeria and similar high-burden settings.

5. Conclusion

This study developed and analyzed a deterministic compartmental model for tuberculosis transmission incorporating vaccination, first-line and second-line treatment, and drug-resistant TB.

Rigorous qualitative analysis established the positivity and boundedness of solutions, and both local and global stability of the disease-free equilibrium were proven when the basic reproduction number $R_0 < 1$. Numerical simulations, calibrated with WHO and national TB data, demonstrated that treatment-related parameters, particularly first-line and second-line treatment rates are the primary drivers of TB control. While vaccination reduces susceptibility and progression to active disease, its impact is complementary and insufficient in isolation. The results highlight that inadequate treatment coverage leads to sustained transmission, increased drug resistance, and higher cumulative mortality. Overall, the findings show the necessity of integrated TB control strategies that combine effective vaccination with robust, timely, and comprehensive treatment programs to achieve meaningful reductions in TB burden and support progress toward TB elimination in high-burden settings such as Nigeria.

6. Recommendation

Based on the findings of this study, TB control efforts should prioritize the scale-up of effective first-line and second-line treatment to reduce infectiousness, prevent treatment failure, and limit the emergence and transmission of drug-resistant TB. Strengthening early diagnosis, treatment adherence, and access to quality second-line therapy is particularly critical in high-burden settings such as Nigeria, where drug-resistant TB poses a growing threat. Vaccination programs should be sustained and expanded as a complementary preventive measure, as vaccination alone is insufficient to control TB without robust treatment systems. Overall, integrated TB control strategies that combine timely diagnosis, effective multi-line treatment, and sustained vaccination coverage are essential for reducing TB prevalence, mortality, and drug resistance, and for achieving long-term progress toward TB elimination.

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Conflict of interest

There is no conflict of interest to disclose.

Author contributions

Leona Concord Diala: Methodology, Conceptualization, Validation, Data curation, Writing - Original draft. **Abah Roseline Toyin:** Review & Editing, Supervision. **Azuaba Emmanuel:** Review & Editing, Supervision.

Declaration of using AI tools

The authors declare that they have not used any type of generative artificial intelligence for the writing of this manuscript, nor for the creation of images, graphics, tables, or their corresponding captions.

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