



**Original Research Article**

# Unraveling the Spread and Control Nexus with Knowledge, Treatment, and Reinfection in Tuberculosis Dynamics

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## ABSTRACT

The study provides stability assessment both locally and globally and analyzes how the fundamental reproduction number impacts the spread of disease. The tuberculosis control through awareness, early detection, and treatment improves cure rates and reduces transmission. Sensitivity analysis of the parameters of basic reproduction number reveals critical to tuberculosis dynamics. By using homotopy perturbation method in a novel way, the research integrates rigorous mathematical analyses with numerical simulations to provide a deep understanding of the intricate interactions between treatment techniques, knowledge distribution, and reinfection dynamics in tuberculosis. The result shows that treatment through vaccination combined with early detection and patient monitoring, improves cure rates and reduces transmission, highlighting the need for focus, efficient control methods in global eradication of tuberculosis.

**Keywords:** Tuberculosis, Reinfection Treatment, Basic reproduction number, Stability analysis, Homotopy Perturbation Method

## INTRODUCTION

Tuberculosis remains a threat across the globe as a medical problem necessitating a comprehensive understanding of the intricate mechanisms driving both its spread and management (Carlos and Baojun, 2014). In the framework of tuberculosis dynamics, this research explores the intricate connections among treatment approaches, reinfection, and knowledge dissemination (Bisuta et al., 2018). A detailed analysis of how treatment approaches, overall comprehension, and the phenomenon of reinfection all influence the course of tuberculosis transmission must be conducted as we traverse the complexity of this infectious disease (Brian et al. 2013). By separating this relationship, we want to offer details that will help develop targeted

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and effective methods of reducing the negative effects of tuberculosis on public health (Daniel, 2020, Dauda et al., 2020, Egonmwan et al., 2019). Combining these components not only improves our knowledge of the dynamics of tuberculosis but also creates the groundwork for new treatments and approaches to reduce the spread of the disease. Studying how the public's awareness of tuberculosis, the effectiveness of various therapies, and the risk of reinfection all contribute to the infectious disease's tenacity and changes becomes vital as it deals with fresh issues [Ibrahim et al., 2017]. Our study goes beyond conventional frameworks to discover the small but important aspects that shape the course of tuberculosis transmission. To improve our theoretical understanding of the complexities of tuberculosis while developing beneficial consequences for public health interventions by navigating this complex spread and control nexus (Intan et al., 2020, Khajanchi et al., 2018). The combination of treatment techniques, information, and dynamics of reinfection creates new opportunities for focused and creative ways of reducing tuberculosis's detrimental impact on communities around the world. An enormous amount of research has been done in the past couple of decades to comprehend the complexities of tuberculosis, including different aspects of its pathogenesis, approaches to treatment, and treatment protocols (Latifat et al., 2020). The collaborative effort has been carefully recorded in various kinds of research papers, textbooks, and scientific publications, to other sources. This extensive study of tuberculosis has not only enhanced our awareness of the illness but also made a substantial impact on the progress of diagnostic and treatment approach (Liao, 2023). Investigations in science involve a wide range of topics, from the growth of new diagnostic and therapeutic methods to the research of the essential part that immune responses in the host play in the course of disease (Lakstimikan et al., 1989). At the same time, drug-resistant strains of *Mycobacterium tuberculosis*, the tuberculosis-causing agent, have become prevalent compelling researchers to examine ways of resistance and potential intervention strategies (LaSalle, 1976). The combined efforts of the research community have been crucial in helping us comprehend tuberculosis, providing important insights that go beyond simple theoretical knowledge. Based on empirical evidence, these insights have led to notable improvements in tuberculosis diagnosis, treatment, and prevention. Through their adept handling of the intricacies involved in tuberculosis research, scientists have cleared the path for more efficacious strategies that exhibit the potential to ameliorate and enhance the impacts of this widespread epidemic (Liu et al., 2020, Mettle et al., 2020). We investigate the significant advances that have resulted from these studies as we explore the area of tuberculosis research, revealing the novel developments in the field and their consequences for public health (Omale, et al., 2019).

This study of tuberculosis dynamics using a powerful numerical tool of homotopy perturbation method to simulate and reveal important factors affecting the spread of the ailments. Our findings underlined the intricate interactions that exist between the efficacy of therapy, knowledge dispersion, and the risk of reinfection (Zhao et al., 2017, Bisuta et al., 2018). Hence we offered substantial implications for enhancing public health initiatives in the fight against tuberculosis, as well as unique insights into tailored control measures of adequate knowledge on its spread and strict adherence on the usage of vaccination.

## **MATERIALS AND METHODS**

### **Model formulation**

A deterministic mathematical model based on the epidemiological status of population of members that describes the dynamics of tuberculosis transmission. The total population  $N(t)$

splits into some compartmental classes for a disease-modification as sub-population into susceptible  $S(t)$ , exposed  $E(t)$ , infected  $I(t)$ , and recovered  $R(t)$  individuals. The transmission probability, rate of disease coefficient and migration/recruitment into the sub-populations that are vulnerable are measured in terms of  $\beta$ ,  $\alpha$  and  $\Lambda$ . The respective classes are subjected to natural death rate  $\mu$ , while exposed individuals have disease-induced mortality rate  $\varepsilon$  and infected persons have recovery rates  $\gamma$ . The set of individuals that are healed from the disease class denoted by  $T$  can also be re-infected after being exposed at a certain rate  $\delta$ . The model formulation flow to depict the aforementioned is given by figure 1.

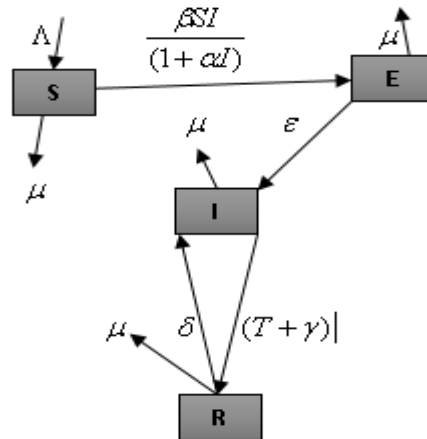


Figure 1: Schematic flow of model description.

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\beta SI}{1 + \alpha I} - \mu S \\ \frac{dE}{dt} &= \frac{\beta SI}{1 + \alpha I} - (\mu + \varepsilon)E \\ \frac{dI}{dt} &= \varepsilon E - (\mu + \gamma)I - TI + \delta R \\ \frac{dR}{dt} &= TI + \gamma I - (\mu + \delta)R \end{aligned} \right\} \quad (1)$$

at an initial condition  $S(t) = s_0 > 0, E(t) = e_0 > 0, I(t) = i_0 > 0, R(t) = r_0 > 0$  over  $0 < T \leq 1$

### Analysis of model solution

#### Positivity and boundedness of model solution

The system (1) describing an epidemic disease in a human population must have nonnegative parameters  $t > 0$ . To ensure mathematical and epidemiological well-being, state variables must be non-negative. This is achieved when the system starts with non-negative initial conditions.

**Theorem 1:**

All solutions of system (1) are bounded in the region space  $\mathfrak{R}_4^+$  at  $t > 0$

**Proof:**

Consider the total population

$$N(t) = S(t) + E(t) + I(t) + R(t) \quad (3)$$

The variation in the total population concerning time is given by:

$$\frac{dN(t)}{dt} = \frac{d}{dt}(S(t) + E(t) + I(t) + R(t)) \quad (4)$$

Such that

$$\frac{dN(t)}{dt} = \Lambda - \mu(S + E + I + R) \Rightarrow \frac{dN(t)}{dt} \leq \Lambda - \mu N$$

Hence, it is obtained that

$$\frac{dN(t)}{dt} + \mu N \leq \Lambda, \text{ leading to } N(t)e^{\mu t} = \frac{\Lambda}{\mu} e^{\mu t} + c \quad (5)$$

Firstly,

$$N(0) = \frac{\Lambda}{\mu} + ce^{-\mu(0)}, \text{ resulting to}$$

$$c = N(0) - \frac{\Lambda}{\mu} \quad (6)$$

Hence, by substituting equation 6 into equation 5 and allowing time to increase progressively, we obtain:

$$\lim_{t \rightarrow \infty} N(t) \leq \lim_{t \rightarrow \infty} \left[ \frac{\Lambda}{\mu} + \left( N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t} \right] = \frac{\Lambda}{\mu} \quad (7)$$

Then  $N(0) \leq \frac{\Lambda}{\mu}$ , then  $N(t) \leq \frac{\Lambda}{\mu}$ . This region is a positively invariant set under the flow defined by equation (2), meaning that no solution trajectory exits through any boundary  $\mathfrak{R}_4^+$ .

Therefore, it is sufficient to analyze the model's dynamics within this domain  $\mathfrak{R}_4^+$ . Within this region, the model is both mathematically and epidemiologically well-posed. This indicates that the total population and each subpopulation  $S(t), E(t), I(t), R(t)$  of the model are bounded and that the solution is unique. Consequently, the model is suitable for studying real-world systems.

Consequently, considering the compartmental disposition.

$$\Psi = \left( (S(t), E(t), I(t), R(t) : N(t) \frac{\Lambda}{\mu}) \right) \text{ it is obtained as;}$$

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta SI}{1 + \alpha I} - \mu S \\
\frac{dS}{dt} &\geq -S(t) \left( \frac{\beta I}{1 + \alpha I} + \mu \right) \\
\frac{dS}{S(t)} &\geq - \left( \frac{\beta I}{1 + \alpha I} + \mu \right) dt \\
\int \frac{dS}{S(t)} &\geq - \int \left( \frac{\beta I}{1 + \alpha I} + \mu \right) dt \\
\ln S(t) &\geq - \left( \frac{\beta I}{1 + \alpha I} + \mu \right) t \\
S(t) &\geq S_0 \ell^{-\left( \frac{\beta I}{1 + \alpha I} + \mu \right) t} > 0 \\
\text{At } t > 0, S(t) &> 0
\end{aligned} \tag{8}$$

In the second compartment,

$$\begin{aligned}
\frac{dE}{dt} &= \frac{\beta SI}{1 + \alpha I} - (\mu + \varepsilon) E(t), \\
\frac{dE}{dt} &\geq -(\mu + \varepsilon) E(t), \\
\frac{dE}{E(t)} &\geq -(\mu + \varepsilon) dt \\
\int \frac{dE}{E(t)} &\geq - \int (\mu + \varepsilon) dt \\
\ln E(t) &\geq -t(\mu + \varepsilon) \\
E(t) &\geq \ell^{-(\mu + \varepsilon)t} \\
E(t) &\geq E_0 \ell^{-t(\mu + \varepsilon)} > 0 \\
\text{At } t > 0, E(t) &> 0
\end{aligned} \tag{9}$$

Thirdly,

$$\begin{aligned}
\frac{dI}{dt} &= \varepsilon E - (\mu + \gamma)I - TI + \delta R \\
\frac{dI}{dt} &\geq -(\mu + \gamma + T)I(t) \\
\frac{dI}{I(t)} &\geq -(\mu + \gamma + T)dt \\
\int \frac{dI}{I(t)} &\geq -\int (\mu + \gamma + T)dt \\
\ln I &\geq -t(\mu + \gamma + T) \\
I(t) &\geq \ell^{-t(\mu + \gamma + T)} \\
I(t) &\geq I_0 \ell^{-t(\mu + \gamma + T)} > 0 \\
\text{At } t > 0, I(t) &> 0
\end{aligned} \tag{10}$$

Lastly,

$$\begin{aligned}
\frac{dR}{dt} &= TI + \gamma I - \mu R - \delta R \\
\frac{dR}{dt} &\geq -(\mu + \delta)R(t) \\
\int \frac{dR}{R(t)} &\geq -\int (\mu + \delta)dt \\
\ln R(t) &\geq -t(\mu + \delta) \\
R(t) &\geq \ell^{-t(\mu + \delta)} \\
R(t) &\geq R_0 \ell^{-t(\mu + \delta)} > 0
\end{aligned} \tag{11}$$

Equation (8) to (11) shows system (1) in the positive quadrant, persisting in the attracting subset  $\Psi$ , which is compact, positively invariant, and influential, with a well-posed, epidemiologically and mathematically represented solution.

#### **Tuberculosis- Non-Infected Equilibrium State**

The equilibrium state representing non-infected individuals with tuberculosis denotes a system free from *Mycobacterium tuberculosis*, where the populations of exposed (E), infected (I), and recovered (R) individuals are all absent.

$$\frac{dN}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \tag{12}$$

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta SI}{1 + \alpha I} - \mu S = 0 \\
\frac{dE}{dt} &= \frac{\beta SI}{1 + \alpha I} - (\mu + \varepsilon)E = 0 \\
\frac{dI}{dt} &= \varepsilon E - (\mu + \gamma)I - TI + \delta R = 0 \\
\frac{dR}{dt} &= TI + \gamma I - \mu R - \delta R = 0
\end{aligned} \quad (13)$$

In the absence of a tuberculosis outbreak, the disease class is expressed as, from equation (12),

$$\Lambda - \frac{\beta SI}{1 + \alpha I} - \mu S = 0, \quad R = 0, \text{ where, } S = \frac{\Lambda}{\mu}$$

Thus, the disease-free equilibrium yields:

$$(S, E, I, R) = \left( S_0 = \frac{\Lambda}{\mu}, E_0 = 0, I_0 = 0, R_0 = 0 \right) \quad (14)$$

### Steady-State Prevalence of Tuberculosis

Highlighting the dynamic nature of tuberculosis prevalence is crucial, especially given its significant role in outbreaks and population dynamics. To analyze the system at steady state, consider the set of equations in (1), from which the equilibrium points can be identified as  $\Phi = (S^*, E^*, I^*, R^*)$  at  $t > 0$

$$\begin{aligned}
I^* &= \frac{(\mu + \delta)(\varepsilon E + \delta R)(T + \gamma)}{(T + \gamma)(\mu + \delta)(\mu + \gamma + T)}, \quad E^* = \frac{(\varepsilon E + \delta R)(T + \gamma)\beta(\mu + \varepsilon)(1 + \alpha)\Lambda}{\beta - \mu(1 + \alpha)(T + \gamma)(\mu + \delta)(\mu + \gamma + T)}, \\
S^* &= \frac{1 + \alpha\Lambda(\mu + \delta)(\varepsilon E + \delta R)(T + \gamma)}{\beta I - \mu(1 + \alpha)(T + \gamma)(\mu + \delta)(\mu + \gamma + T)}, \quad R^* = \frac{(\varepsilon E + \delta R)(T + \gamma)}{(\mu + \delta)(\mu + \gamma + T)}
\end{aligned} \quad (15)$$

### Basic Reproduction Number

The basic reproduction number, denoted  $R_*$ , measures the potential for tuberculosis infections from a single carrier or infected individual in a population with no prior infections. To determine the system (1), we apply the next-generation method, focusing on the infectious classes E and I. This process entails determining the F and V matrices, which represent the rates of new infections and the transitions into and out of the infected compartments, respectively. From the equations in the system (1), we derive these matrices as follows.  $R_* = \rho G$

where  $G = F \times V^{-1}$  and  $\rho$  is the spectral radius of the matrix  $|G - \lambda I|$ .

From the system of equations (1), the matrices F and V are obtained as follows:

$$F_i = \left( \frac{\partial f_i(x_i)}{\partial x_j} \right), \quad V_i = \left( \frac{\partial v_i(x_i)}{\partial x_j} \right) \quad (16)$$

Such that

$$f = \begin{pmatrix} \frac{\beta S}{(1 + \alpha)^2} \\ 0 \end{pmatrix} \quad \text{and} \quad v = \begin{pmatrix} (\mu + \varepsilon)E \\ -\varepsilon E + (\mu + \gamma + T)I \end{pmatrix} \quad (17)$$

then,

$$\begin{aligned}
 F &= \begin{pmatrix} \frac{\beta SI}{(1+\alpha I)^2} \\ 0 \end{pmatrix} V = \begin{pmatrix} (\mu + \varepsilon) & 0 \\ -\varepsilon & (\mu + \gamma + T) \end{pmatrix} \\
 FV^{-1} &= \frac{1}{(\mu + \varepsilon)(\mu + \gamma + T)} \begin{pmatrix} \frac{\beta \Lambda}{\mu(1+\alpha\mu)^2} & 0 \\ 0 & 0 \end{pmatrix} \begin{pmatrix} (\mu + \gamma + T) & 0 \\ \varepsilon & (\mu + \varepsilon) \end{pmatrix} \\
 R_* &= \frac{\beta \Lambda}{\mu(\mu + \varepsilon)(\mu + \gamma + T)} \quad (18)
 \end{aligned}$$

The leading eigenvalue of the non-invariant is the basic reproduction number of the disease model

#### Asymptotic Stability of the Disease-Free State

This section analyzes the stability of the tuberculosis disease-free state by evaluating the influence of the basic reproduction number. When the reproduction number is  $R^* < 1$ , the disease declines, and we determine stability using a Jacobian matrix and a characteristic equation.

#### Theorem 2

The disease-free state of the model is locally asymptotically stable whenever  $R^* < 1$  and unstable if  $R^* > 1$ .

Proof:

The disease-free equilibrium is determined by deriving the Jacobian matrix of the system in equation (1) and evaluating it at the disease-free state using the linearization method.

$$\begin{aligned}
 J_{\ell_0} &= \begin{pmatrix} -\mu & 0 & \beta S & 0 \\ 0 & -(\mu + \varepsilon) & \beta S & 0 \\ 0 & \varepsilon & -(\mu + \gamma + T) & \delta \\ 0 & 0 & T + \gamma & -(\mu + \delta) \end{pmatrix} \\
 J_{\ell_0} &= \begin{pmatrix} -\mu & 0 & \frac{\Lambda\beta}{\mu} & 0 \\ 0 & -(\mu + \varepsilon) & \frac{\Lambda\beta}{\mu} & 0 \\ 0 & \varepsilon & -(\mu + \gamma + T) & \delta \\ 0 & 0 & T + \gamma & -(\mu + \delta) \end{pmatrix} \quad (19)
 \end{aligned}$$

Computing for the eigenvalues,  $|J_{E_1} - \lambda_i I| = 0$

$$\begin{aligned}
 a &= \begin{vmatrix} -(\mu + \gamma + T) - \lambda & \delta \\ T + \gamma & -(\mu + \delta) - \lambda \end{vmatrix} \\
 &= (-(\mu + \gamma + T) - \lambda_3)(-(\mu + \delta) - \lambda_4) = 0, \lambda_4 = -(\mu + \delta), \lambda_3 = -(\mu + \gamma + T) \\
 &\lambda_3 = -(\mu + \gamma + T) - (\mu + \varepsilon) - \lambda_2 |A| = 0 \\
 &\lambda_2 = -(\mu + \varepsilon) \lambda_1 = -\mu \\
 \therefore \lambda_1 &= -\mu, \lambda_2 = -(\mu + \varepsilon), \lambda_3 = -(\mu + \gamma + T), \lambda_4 = -(\mu + \delta)
 \end{aligned}$$



The negativity of the invariants in the region of the system of (1) is subjected to be locally asymptotically stable whenever  $R^* < 1$ .

### Regional Resilience of the Persistent Equilibrium

#### Theorem 3

The persistent equilibrium of the proposed model is locally asymptotically stable within the region if and unstable otherwise in the region of  $\mathfrak{R}^4_+$  if and only if  $R^* > 1$ .

#### Proof:

Suppose,  $S = x + S^*, E = y + E^*, I = z + I^*, R = a + R^*$  (20)

Linearizing equation (1), is then obtained as

$$\left. \begin{aligned} \frac{dx}{dt} &= -2\beta xz(1 + \alpha z)^{-1} - \mu x + \text{higher order} + \text{nonlinear terms...} \\ \frac{dy}{dt} &= 2\beta xz(1 + \alpha z)^{-1} - (\mu + \varepsilon)y + \text{higher order} + \text{nonlinear terms...} \\ \frac{dz}{dt} &= \varepsilon y + (\mu + \gamma)z - Tz + \delta a + \text{higher order} + \text{nonlinear terms...} \\ \frac{da}{dt} &= (T + \gamma)z - (\mu + \delta)a + \text{higher order} + \text{nonlinear terms...} \end{aligned} \right\} \quad (21)$$

Jacobian matrix of the system of (21),

$$\begin{vmatrix} -(2\beta(1 + \alpha)^{-1} + \mu) & 0 & (2\beta(1 + \alpha)^{-1} + \mu) & 0 \\ (2\beta(1 + \alpha)^{-1} + \mu) & -(\mu + \varepsilon) & (2\beta(1 + \alpha)^{-1} + \mu) & 0 \\ 0 & \varepsilon & -(\mu + \gamma + T) & 0 \\ 0 & 0 & (T + \gamma) & -(\mu + \delta) \end{vmatrix} = 0$$

The resulting eigenvalue of the above matrix is obtained as;

$$\left\{ -(2\beta(1 + \alpha)^{-1} + \mu) - \lambda_1, -(\varepsilon + \mu) - \lambda_2, -(T + \gamma + \mu) - \lambda_3, -(\delta + \mu) - \lambda_4 \right\} = 0 \quad (22)$$

If  $a = -(2\beta(1 + \alpha)^{-1}), b = -(\varepsilon + \mu), c = -(T + \gamma + \mu), d = -(\delta + \mu)$  it's therefore obtained that

$$(a - \lambda_1)(b - \lambda_2)(c - \lambda_3)(d - \lambda_4) = 0$$

$$\lambda^4 - [(d + e) + (c + b)]\lambda^3 + [(a + b)(c + d) + ab + cd]\lambda^2 - [abc(c + d) + de(a + b)]\lambda^2 + [ae + ad + bd + ac] + abcde$$

Hence, the persistent resilience of the model in a region is asymptotically stable.

### Global Stability of Disease-Free Equilibrium

We employing the use of Lyapunov's function approach to establish the global asymptotic stability of the model for equation (1) at the disease-free equilibrium, utilizing its algorithm over some constrains to obtain the following set of equations;

$$\text{Define } \Psi(t, S, E, I, R) = C_1 I_1 + C_2 I_2$$

$$\begin{aligned}
\frac{d\Psi}{dt} &= C_1 I_1^* + C_2 I_2^* = C_1 \left( \frac{\beta S_0 I_2}{1 + \alpha S_0} - (\mu + \varepsilon) I_1 \right) + C_2 (\varepsilon I_1 - (\mu + \gamma + T) I_2 + \delta R) \\
\frac{d\Psi}{dt} &\leq (C_2 \varepsilon - C_1 (\mu + \varepsilon)) I_1 - \left( C_1 \frac{\beta S_0}{(1 + \alpha) S_0} - C_2 (\mu + \gamma + T) + \delta R \right) I_2 \\
\frac{d\Psi}{dt} &\leq C_1 (C_2 \varepsilon - C_1 (\mu + \varepsilon)) I_1 - C_2 \left( C_1 \frac{\beta \Lambda}{\mu (1 + \alpha)} - C_2 (\mu + \gamma + T) \right) I_{21} \\
C_1 &= \frac{1}{(\mu + \varepsilon)}, C_2 = \frac{\beta \Lambda}{\mu (\mu + \varepsilon) (1 + \alpha) (\mu + \gamma + T)}, R \leq 0 \\
\frac{d\Psi}{dt} &\leq \left( \frac{\beta \Lambda \varepsilon}{\mu (\mu + \varepsilon) (1 + \alpha) (\mu + \gamma + T)} - \frac{(\mu + \varepsilon)}{(\mu + \varepsilon)} \right) I_1 - \left( \frac{\beta \Lambda}{\mu (1 + \alpha) (\mu + \varepsilon)} - \frac{\beta \Lambda (\mu + \gamma + T)}{\mu (1 + \alpha) (\mu + \varepsilon) (\mu + \gamma + T)} \right) I_2 \\
\frac{d\Psi}{dt} &\leq \left( \frac{\beta \Lambda \varepsilon}{\mu (\mu + \varepsilon) (1 + \alpha) (\mu + \gamma + T)} - 1 \right) I \\
\frac{d\Psi}{dt} &\leq (R_0 - 1)
\end{aligned} \tag{23}$$

It is pertinent to note that when at  $t \rightarrow \infty$ ,  $\frac{d\Psi}{dt} \leq 0$ . Substituting into the model system of equation (1) reveals that, based on LaSalle's invariance principle  $\frac{d\Psi}{dt} = 0$ , is globally asymptotically stable whenever  $R_0 > 1$

#### Global stability for endemic equilibrium

Theorem 4

The model system of equation (1) has no periodic orbits.

**Proof:**

Employing the Dulac's criterion. Let  $X = (S, E, I, R)$  define the Dulac's function  $G = \frac{1}{SI}$ . The following system of equation are obtained;

$$\left. \begin{aligned}
G \frac{dS}{dt} &= \frac{i}{SI} \left\{ \Lambda - \frac{\beta SI}{1 + \alpha I} - \mu S \right\} \\
G \frac{dE}{dt} &= \frac{i}{SI} \left\{ \frac{\beta SI}{1 + \alpha I} - (\mu + \varepsilon) E \right\} \\
G \frac{dI}{dt} &= \frac{i}{SI} \{ \varepsilon E - (\mu + \gamma) I - TI + \delta R \} \\
G \frac{dR}{dt} &= \frac{i}{SI} \{ TI + \gamma I - (\mu + \delta) R \}
\end{aligned} \right\} \tag{24}$$

from above system of equations results to;

$$G \frac{dS}{dt} = \left\{ \frac{\Lambda}{SI} - \frac{\beta}{1 + \alpha} - \frac{\mu}{SI} \right\}$$

$$G \frac{dE}{dt} = \left\{ \frac{\beta}{1+\alpha} - \frac{(\mu+\varepsilon)E}{SI} \right\}$$

$$G \frac{dI}{dt} = \left\{ \frac{\varepsilon E}{SI} - \frac{(\mu+\gamma)}{S} - \frac{T}{S} + \frac{\delta R}{SI} \right\} \quad (25)$$

$$G \frac{dR}{dt} = \left\{ \frac{T}{S} + \frac{\gamma}{S} - \frac{(\mu+\delta)R}{SI} \right\} \quad (26)$$

At  $t > 0$  orbital resolution of the system of equations is given by  $\frac{d(GX)}{dt}$  as obtained below.

$$\frac{d(GX)}{dt} = \frac{\partial}{\partial S} \left\{ G \frac{dS}{dt} \right\} + \frac{\partial}{\partial E} \left\{ G \frac{dE}{dt} \right\} + \frac{\partial}{\partial I} \left\{ G \frac{dI}{dt} \right\} + \frac{\partial}{\partial R} \left\{ G \frac{dR}{dt} \right\}$$

$$\frac{d(GX)}{dt} = \frac{\partial}{\partial S} \left\{ \frac{\Delta}{SI} - \frac{\beta}{1+\alpha} - \frac{\mu}{SI} \right\} + \frac{\partial}{\partial E} \left\{ \frac{\beta}{1+\alpha} - \frac{(\mu+\varepsilon)E}{SI} \right\}$$

$$+ \frac{\partial}{\partial I} \left\{ \frac{\varepsilon E}{SI} - \frac{(\mu+\gamma)}{S} - \frac{T}{S} + \frac{\delta R}{SI} \right\} + \frac{\partial}{\partial R} \left\{ \frac{T}{S} + \frac{\gamma}{S} - \frac{(\mu+\delta)R}{SI} \right\} \quad (27)$$

$$\frac{d(GX)}{dt} = \left\{ -\frac{(\Delta+\beta+\mu)}{S(1+\alpha)} \right\} + \left\{ -\frac{(\mu+\varepsilon)E+\beta}{S(1+\alpha)} \right\}$$

$$+ \left\{ \frac{(\mu+\gamma)-T+\delta R}{SI} \right\} + \left\{ -\frac{T+\gamma+(\mu+\delta)R}{SI} \right\} \quad (28)$$

$$\frac{d(GX)}{dt} = - \left\{ \frac{(\Delta+\beta+\mu)}{S(1+\alpha)} + \frac{(\mu+\varepsilon)E+\beta}{S(1+\alpha)} - \frac{(\mu+\gamma)+T+\delta R}{SI} + \frac{T+\gamma+(\mu+\delta)R}{SI} \right\}$$

$$\frac{d(GX)}{dt} = - \left\{ \frac{(\Delta+\beta+\mu)[(\mu+\varepsilon)+\beta+(\mu+\gamma)+2T+\delta+\gamma+(\mu+\delta)]}{SI} \right\} < 0 \quad (29)$$

This implies that the system has no closed orbit. Epidemiologically, non-existence of a periodic orbit implies that there are fluctuations in the number of infective, which makes it difficult in allocation of resources for the control of the disease.

#### Sensitivity analysis of $R^*$

The main objective is to evaluate the sensitivity of the basic reproduction number by calculating its derivative with respect to all pertinent parameters. This analysis will lead to the determination of the normalized forward sensitivity index, referred to as

$$\left. \begin{aligned} \frac{\partial R_*}{\partial \beta} = \frac{\partial R_*}{\partial \beta} \times \frac{\beta}{R_*} = 0.0112060, \quad \frac{\partial R_*}{\partial \mu} = \frac{\partial R_*}{\partial \mu} \times \frac{\mu}{R_*} = 0.0151427, \quad \frac{\partial R_*}{\partial \Lambda} = \frac{\partial R_*}{\partial \Lambda} \times \frac{\Lambda}{R_*} = 1.0000040 \\ \frac{\partial R_*}{\partial \varepsilon} = \frac{\partial R_*}{\partial \varepsilon} \times \frac{\varepsilon}{R_*} = 1e^{-3}, \quad \frac{\partial R_*}{\partial \gamma} = \frac{\partial R_*}{\partial \gamma} \times \frac{\gamma}{R_*} = 1.0203010, \quad \frac{\partial R_*}{\partial T} = \frac{\partial R_*}{\partial T} \times \frac{T}{R_*} = 1.0326701 \end{aligned} \right\} \quad (30)$$

Table 1: Sensitivity analysis of parameters and indices

Parameter	Sensitivity indices
-----------	---------------------

$\beta$	$\times day^{-1}$
$\mu$	$0.0151427 \times day^{-1}$
$\Lambda$	$1.000040 \times day^{-1}$
$\varepsilon$	$1 e^{-1} \times day^{-1}$
$\gamma$	$1.0203010 \times day^{-1}$
$T$	$1.0326701 \times day^{-1}$
$\alpha$	$0.0212 \times day^{-1}$

The sensitivity analysis of the basic reproduction number  $R_0$  for tuberculosis indicates significant insights into the disease's dynamics and control measures. All parameters of  $R_0$  are positive in their indices, demonstrating that improved knowledge and treatment of tuberculosis have a substantial biological and medical impact. Specifically, the analysis reveals that increasing awareness and accessibility to effective treatment reduces the infection and reinfection rates by 87.27%. This highlights the critical role of education and medical intervention in managing and curbing the spread of tuberculosis. Furthermore, the sensitivity analysis indicates that a significant increase in the level of the susceptible population results in a 12.26% drop in the trend of the infected population. This finding underscores the importance of preventive measures and early detection, which decrease the pool of individuals susceptible to infection. Public health strategies focusing on these aspects can therefore significantly reduce the transmission and prevalence of tuberculosis. Overall, these results emphasize the need for robust public health policies that enhance treatment knowledge and accessibility, promote early detection, and implement preventive measures. Such comprehensive approaches are vital for achieving a substantial reduction in tuberculosis infection rates and advancing towards its eventual eradication.

#### Numerical simulation using Homotopy perturbation method technique

The Homotopy Perturbation Method effectively illustrates the impact of control parameters on tuberculosis spread. Using He's algorithm, we analyzed how increased treatment knowledge reduces tuberculosis transmission. Iterative solutions of Homotopy Perturbation Method demonstrate that improved treatment awareness significantly decreases the infected population while increasing the susceptible, exposed, and recovered populations. Varying data inputs show that better treatment strategies result in a marked reduction in infection rates. This method highlights the critical role of treatment knowledge in tuberculosis control, proving it to be an essential factor in reducing the disease's spread. Overall, Homotopy Perturbation Method provides a robust numerical tool for optimizing tuberculosis management strategies.

$$A(u) - f(r) = 0 \quad r \in \Psi \quad (31)$$

Subject to the boundary condition

$$B(r) = 0 \quad r \in \Gamma \quad (32)$$

Where  $A$  is a general differential operator,  $B$  is a boundary operator,  $f(r)$  is a known analytic function, and  $r$  is the boundary of the domain  $\Psi$ . The operator  $A$  can, generally

speaking, be divided into two parts: a linear part  $L$  and a nonlinear part  $N$ . Equation (1) therefore can be rewritten as follows:

$$L(r) = L_T(u) + N_T(u) \quad (33)$$

Where  $L_T(u), N_T(u)$  represent the linear term, and the nonlinear term of the differential equation respectively. Thus equation (27) becomes

$$L_T(u) + N_T(u) = f(r) \quad r \in \Psi \quad (34)$$

We can construct a Homotopy for (4) so that

$$H(w, p) = (1 - p)[L_T(t) - L_T(u_0)] + p[A(t) - f(r)] = 0 \quad p \in [0, 1], r \in \Psi \quad (35)$$

Simplifying equation (5) to yield this,

$$H(w, p) = L_T(t) - L_T(u_0) + p[L_T(u_0)] + p[N_T(u_0) - f(r)] = 0 \quad (36)$$

Where  $p \in [0, 1]$  and  $u_0$  denotes the initial approximation. Now, as  $p \rightarrow 0$

$$H(w, 0) = L_T(t) - L_T(u_0) = 0$$

And as  $p \rightarrow 1$ ,

$$H(w, 1) = A(t) - f(r) = 0 \quad (37)$$

And we can express the solution of the differential equation as

$$W(t) = W_0(t) + pW_1(t) + p^2W_2(t) + p^3W_3(t) + \dots \quad (38)$$

Substituting (31) into (32), and comparing coefficients of equal powers of  $P$  the resulting equation is solved to obtain the value of  $w_0(t), w_1(t), w_2(t), w_3(t)$

Such that the approximate solution of the differential equation in (32) is

$$\lim_{p \rightarrow 1} W(t) = W_1(t) + W_2(t) + W_3(t) + W_4(t) + \dots \quad (39)$$

The algorithm of the model formulation using the Homotopy Perturbation Method in its simulation is developed as follows;

Table 2: Parameter description and references of the model.

Parameters		Descriptions		
<b>S(t)</b>	Susceptible individuals in the population			
<b>E(t)</b>	Exposed individuals in the population			
<b>I(t)</b>	Infected individuals in the population			
<b>R(t)</b>	Parameters			
Parameters	S(t)	Susceptible individuals in the population	References	
$\Lambda$	E(t)	Exposed individuals in the population	[1]	
$\beta$	I(t)	Infected individuals in the population	[4]	
$\varepsilon$	R(t)	Recovered individuals in the population	[13]	
$T$	Parameters	Descriptions	Values	References

$\gamma$	$\Lambda$	Recruitment rate	$120 \times \text{days}$	[1]
$\mu$	$\beta$	Contact rate of susceptible per unit of time	$0.00124 \times \text{days}$	[4]
$\alpha$	$\varepsilon$	Induced death	$0.0133 \times \text{days}$	[13]
$\delta$	$T$	Treatment rate	$0.0109395 \times \text{days}$	[1]
	$\gamma$	Progression rate from infected to recovered	$4.1301 \times 10^{-2} \text{ days}$	[7]
	$\mu$	Natural death	$0.0766169 \times \text{days}$	[5]
	$\alpha$	Transmission coefficient	$0.11 \times \text{days}$	[16]
	$\delta$	Reinfection	$0.052 \times \text{days}$	[18]

$$\left. \begin{aligned} (1-p) \frac{dS}{dt} + p \left( \frac{dS}{dt} - \left[ \Lambda - \frac{\beta SI}{1+\alpha I} - \mu S \right] \right) &= 0 \\ (1-p) \frac{dE}{dt} + p \left( \frac{dE}{dt} - \left[ \frac{\beta SI}{1+\alpha I} - (\mu + \varepsilon) E \right] \right) &= 0 \\ (1-p) \frac{dI}{dt} + p \left( \frac{dI}{dt} - [\varepsilon E - (\mu + \gamma) I - TI + \delta R] \right) &= 0 \\ (1-p) \frac{dR}{dt} + p \left( \frac{dR}{dt} - [TI + \gamma I - \mu R - \delta R] \right) &= 0 \end{aligned} \right\} \quad (40)$$

The correctional series assumes that the solutions for (1) are such that

$$S(t) = \sum_{k=0}^n p^k s_k(t), E(t) = \sum_{k=0}^n p^k e_k(t), I(t) = \sum_{k=0}^n p^k i_k(t), R(t) = \sum_{k=0}^n p^k r_k(t), \quad (41)$$

When  $p$  approaches 1, this series converges. The following can be obtained by comparing the coefficients of and by evaluating (40) and (41)

At  $n = 0$

$$\frac{dS_o}{dt} = 0, \frac{dE_o}{dt} = 0, \frac{dI_o}{dt} = 0, \frac{dR_o}{dt} = 0 \quad (42)$$

Using the original constraints to solve these equations

$$S_0(t) = s_0, E_0(t) = e_0, I_0(t) = i_0, R_0(t) = r_0$$

Using this procedure results in

$$\begin{aligned} S_1(t) &= (\Lambda + \beta i_0 s_0 - \alpha i_0 - \mu s_0) t \\ E_1(t) &= (\beta i_0 s_0 - \alpha i_0 - (\mu + \varepsilon) e_0) t \\ I_1(t) &= (\varepsilon e_0 - (\mu + \gamma) i_0 - T i_0 - \delta r_0) t \\ R_1(t) &= (T + \gamma) i_0 - (\mu + \delta r_0) t \end{aligned}$$

(43)

For  $n = 2$ 

$$s_2(t) = \frac{1}{2}t^2 \left( \alpha^3 i_0^2 s_0 + \alpha^2 \mu i_0 s_0 + \alpha^2 \beta i_0 s_0 - \alpha^2 \beta i_0 v_0 - \alpha \delta i_0 s_0 + 2\alpha \mu i_0 s_0 + \alpha \rho_0 s_0 - \alpha \beta_0 s_0 + \mu^2 s_0 \right) \\ + 2\mu \beta s_0 - 2\mu \beta v_0 - \beta^2 s_0 - \beta \beta v_0 - \beta^2 v_0$$

$$e_2(t) = -\frac{1}{2}t^2 \left( \alpha^2 i_0^2 s_0 + \alpha \delta i_0 s_0 + 3\alpha \mu i_0 s_0 + \alpha \rho_0 s_0 - \alpha \sigma e_0 s_0 + \alpha \sigma_{10} s_0 + \alpha \beta i_0 s_0 \right) \\ - \alpha \beta i_0 v_0 - \mu^2 e_0 - 2\mu e_0$$
(44)

$$i_2(t) = -\frac{1}{2}t^2 (\alpha i_0 s_0 + \delta^2 i_0 + 2\delta \mu i_0 + 2\delta \rho i_0 - \delta e_0 + \mu^2 i_0 + 2\mu i_0 - \rho e_0 - \varepsilon^2 e_0)$$

$$r_2(t) = -\frac{1}{2}t^2 (\delta T i_0 - \mu^2 r_0 + 2\mu T i_0 + \rho^2 i_0 - \rho T e_0)$$

At  $n = 3$ 

$$S_3(t) = \frac{1}{6}t^3 \left( \mu^2 s_0 + 2\mu \beta s_0 i_0 + \beta^2 s_0 + \mu^2 e_0 i_0 - 2\mu^2 \beta s_0 r_0 + \beta^2 t_0 s_0 + 2\mu^2 \alpha e_0 r_0 - \frac{1}{2}\varepsilon^2 \beta s_0 r_0 \right. \\ \left. 3t^2 s_0 + \mu^2 \varepsilon \alpha e_0 i_0 - 3\mu^2 \beta s_0 r_0 + \varepsilon^2 t_0 s_0 - 2\Lambda^2 \alpha e_0 r_0 - \frac{1}{2}\varepsilon^2 \beta i_0 r_0 + 2\mu^2 \beta s_0 r_0 \right. \\ \left. + \beta^2 t_0 s_0 - \gamma^2 \beta s_0 r_0 + \beta^2 t_0 i_0 - \mu^2 \gamma s_0 r_0 + \beta^2 t_0 e_0 - 2\delta^2 \beta s_0 r_0 + \beta^2 t_0 s_0 \right)$$

$$E_3(t) = -\frac{1}{2}t^3 \left( -\mu^2 e_0 - 2\varepsilon \alpha s_0 i_0 - t^2 \delta e_0 + \mu^2 s_0 i_0 + \mu^2 \beta e_0 i_0 + \beta^2 t_0 s_0 + 3\mu^2 \delta e_0 s_0 - 3\varepsilon^2 \beta s_0 r_0 \right. \\ \left. 2t^2 e_0 + \beta^2 \alpha s_0 i_0 - 2t^2 \beta e_0 r_0 + \varepsilon^2 t_0 s_0 - 2\gamma^2 \varepsilon e_0 i_0 - 3\varepsilon^2 \beta i_0 s_0 \mu^2 \beta s_0 r_0 \right. \\ \left. + \gamma^2 t_0 s_0 - \gamma^2 \beta i_0 r_0 - \beta^2 t_0 s_0 - \mu^2 \beta s_0 i_0 + \beta^2 t_0 e_0 + \delta^2 t s_0 r_0 + \varepsilon^2 t_0 s_0 \right)$$
(45)

$$I_3(t) = \frac{1}{2}t^3 \left( -2t^2 \beta e_0 r_0 + \varepsilon^2 t_0 s_0 - 2\gamma^2 \varepsilon e_0 i_0 - 3\varepsilon^2 \beta i_0 s_0 \mu^2 \beta s_0 r_0 + \beta^2 t_0 s_0 + 2\mu^2 \alpha e_0 r_0 - \frac{1}{2}\varepsilon^2 \beta s_0 r_0 \right. \\ \left. \mu^2 \gamma s_0 r_0 + \beta^2 t_0 e_0 - 2\delta^2 \beta s_0 r_0 - 2\mu^2 \alpha e_0 r_0 + \gamma^2 \beta i_0 r_0 + 2\mu^2 \beta s_0 r_0 \right. \\ \left. + t^2 s_0 e_0 + \beta^2 t_0 e_0 - 2\delta^2 \beta s_0 r_0 + \beta^2 t_0 s_0 \right)$$

$$R_3(t) = \frac{1}{2}t^3 \left( t^2 \delta s_0 r_0 + \beta^2 t_0 e_0 - 2\delta^2 \beta s_0 r_0 + \beta^2 t_0 s_0 + \beta^2 t_0 s_0 \beta^2 t_0 e_0 - 2\delta^2 \beta s_0 r_0 + \beta^2 t_0 s_0 \right. \\ \left. 3t^2 s_0 + \mu^2 \varepsilon \alpha e_0 i_0 + \varepsilon^2 t_0 s_0 - 2\gamma^2 \varepsilon e_0 i_0 + \varepsilon^2 \beta i_0 s_0 + \mu^2 \beta s_0 r_0 + 2\mu^2 \beta s_0 r_0 \right. \\ \left. + \beta^2 t_0 s_0 - \gamma^2 \beta s_0 r_0 + \beta^2 t_0 i_0 - \mu^2 \gamma s_0 r_0 + \beta^2 t_0 e_0 + 2\delta^2 \beta s_0 r_0 + \beta^2 t_0 s_0 \right)$$
(46)

Iteration of this is carried out until it is the required numbers of iterations are obtained.

Thus, every variation of the compartment's raw solutions is obtained as

$$S(t) = \sum_{k=0}^3 s_k(t), E(t) = \sum_{k=0}^3 e_k(t), I(t) = \sum_{k=0}^3 i_k(t), R(t) = \sum_{k=0}^3 r_k(t),$$
(47)

And evaluating these results using the corresponding model parameters of each class given by

$$\left\{ \begin{array}{l} \alpha = 0.0017, \delta = 0.011, \mu = 0.02, \rho = 0.0115, \sigma = 0.0011, \theta = 0.012, \beta_1 = 0.0021, \\ \beta_2 = 0.0013, e_0 = 65, s_0 = 500, i_0 = 23, v_0 = 120, r_0 = 14 \end{array} \right\} \quad \text{We obtained that}$$

$$\left. \begin{array}{l} S(t) = 500 - 30.4320t + 0.7213561075t^2 - 0.0386340409t^3 \\ E(t) = 65 + 18.1785t - 1.171778775t^2 + 0.0415546653t^3 \\ I(t) = 23 - 0.9060t + 0.0292506750t^2 - 0.000844036800t^3 \\ R(t) = 14 - 0.0155t - 0.0050545000t^2 + 0.000145824542t^3 \end{array} \right\} \quad (48)$$

The approximate values for each class are assessed using their corresponding baseline values listed in Table 1. Additionally, we propose the following population dataset as initial values given by  $s_0 = 1000, e_0 = 30, i_0 = 20, a_0 = 40$ . Thus, we derive the following series of results incorporating the parameters whose effects on tuberculosis transmission dynamics are to be examined:

$$s(t) = 1000 + \left( \begin{array}{l} 65.2686 + 1.3362000\alpha \\ -1362.923\alpha^2 - 37.68 \end{array} \right) t + \left( \begin{array}{l} -8.99856418\alpha^2 \\ +5499.838828\alpha^4 \\ 54.775643345 \\ +152.0510083\alpha^2 \\ -0.5381600 \\ +3333.926349 \\ 45.98509816 \\ -3.288025569\alpha \end{array} \right) \frac{t^2}{2} - \left( \begin{array}{l} 11.30828286\alpha^2 \\ -66.76103861\alpha^3 \\ -0.00371982988\alpha^2 \\ +40645.08576\alpha^4 \\ +935.98111186\alpha^2\alpha \\ +56.12092345 \\ -5.923814565\alpha \\ 302.0838612 \\ +36988.74452\alpha^6 \\ -84.74264814\alpha \end{array} \right) \frac{t^3}{6} \quad (49)$$

$$e(t) = 30 + \left( \begin{array}{l} -45.62599000 \\ +1362.924999\alpha^2 \\ -1.336200000\alpha \end{array} \right) t - \left( \begin{array}{l} -69.38980854 \\ -8.998569418\alpha^3 \\ +5499.839928\alpha^4 \\ +152.0510083\alpha^2 \\ -0.09970881600\alpha \\ +5378.993811\alpha^2 \\ +0.0000493608 \\ -5.292993669\alpha \end{array} \right) \frac{t^2}{2} + \left( \begin{array}{l} 11.30828286\alpha^2 - 80.26339203\alpha^3 \\ -105.5276927 - 0.00371982988\alpha\alpha \\ +48897.59557\alpha^4 + 1164.133657\alpha^2 \\ -0.7679873978\alpha + 1431.639314\alpha^4 \\ -1.875838588\alpha^3 + 16753.17626\alpha^2 \\ +0.000384108040 - 16.31203298\alpha \\ +36988.74452\alpha^6 - 84.74264814\alpha^5 \end{array} \right) \frac{t^3}{6}$$



$$i(t) = 20 - 50.40320t - \left( \begin{array}{c} 127.0391180 \\ + 0.681642000\alpha^2 \\ - 0.000668100\alpha \end{array} \right) \frac{t^2}{2} - \left( \begin{array}{c} - 0.004499284\alpha^3 \\ + 320.2194878 \\ + 2.749919964\alpha^4 \\ + 0.0765345416\alpha^2 \\ - 0.0000498544800\alpha \\ + 4.407401276\alpha^2 \\ + 2.46804232 \cdot 10^{-8} \\ - 0.004212705\alpha \end{array} \right) \frac{t^3}{6} \quad (50)$$

$$r(t) = 40 + (46.18360 + 37.68)t - \left( \begin{array}{c} 250.8099123 \\ + 45.9850488 \\ - 2044.386000\alpha^2 \\ + 2.004300000\alpha \end{array} \right) \frac{t^2}{2} + \left( \begin{array}{c} 13.49785413\alpha^3 \\ - 8249.759899\alpha^4 \\ + 727.7734324 \\ - 228.0765125\alpha^2 \\ + 0.1495632240\alpha \\ - 10561.77617\alpha^2 \\ + 56.12053936 \\ + 10.38388802\alpha \end{array} \right) \frac{t^3}{6} \quad (51)$$

## RESULTS AND DISCUSSION

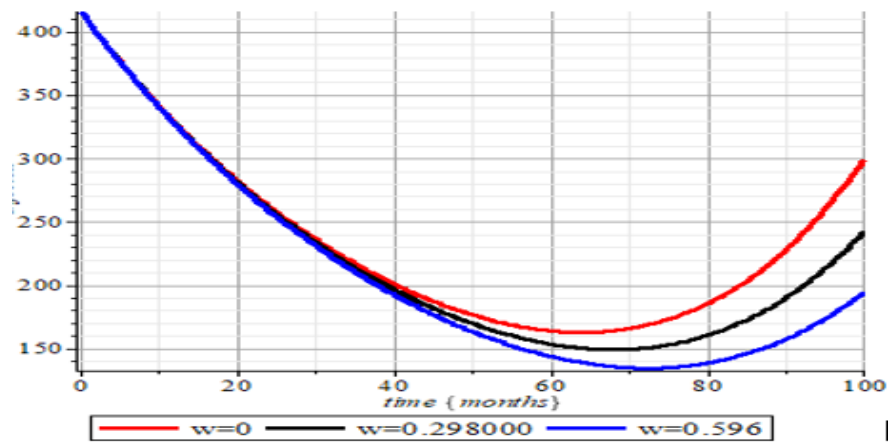


Figure 2: Adverse effect of treatment rate on that of reinfection on the individuals in the recovered population.

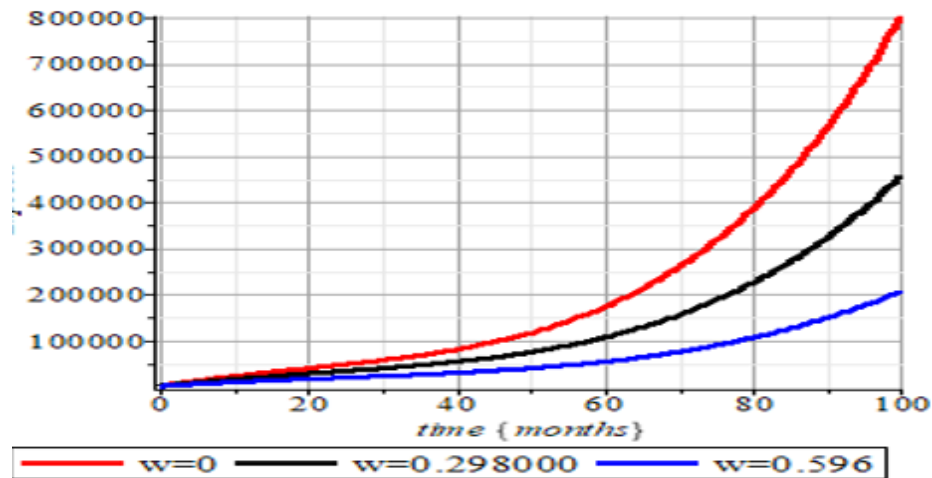


Figure 3: Effect of rapid treatment on the exposed individuals in the population

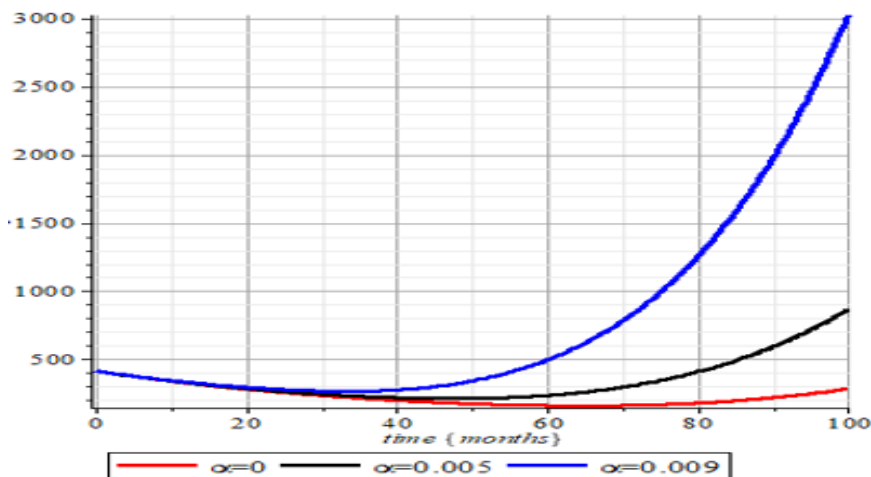


Figure 4: The effect of treatment rate per contact  $\beta$  and transmission coefficient  $\alpha$  on the infected individuals in the population

The result of the simulation have it that fig 2 depicts the impact of treatment rate and reinfection on the recovered population. The results indicate that disturbances from reinfection cause a significant drop in the healed population, ranging from 0.3% to 0.12% over time. However, effective treatment control measures lead to an immediate increase in the recovered population as time progresses. This highlights the adverse effects of reinfection and underscores the importance of continuous treatment and monitoring to sustain recovery rates. For fig.3 the analysis reveals that the disease population is influenced by the incubation period, driven by the induced and recovery rates. Increased awareness and effective treatment strategies medically reduce the latency and spread of tuberculosis. This results in a higher sub-population of susceptible and recovered individuals. The Homotopy Perturbation Method simulation shows that enhanced treatment awareness is crucial for controlling tuberculosis, as it decreases the latent period and minimizes transmission. It is from fig 4 which portrays that contact rate  $\beta$  and transmission coefficient  $\alpha$  are critical factors in the spread of tuberculosis. The simulations

indicate that these can be significantly lowered with increased awareness and implementation of effective treatment control measures. By promoting treatment knowledge and practices, the spread of tuberculosis can be effectively curtailed. Health personalities can leverage these findings to implement strategies that enhance treatment knowledge and reduce tuberculosis spread. This approach is promising for the eradication of tuberculosis, as it emphasizes the importance of continuous education and effective treatment protocols.

## CONCLUSION

The SEIR model, assessing tuberculosis reinfection alongside a control strategy involving efficacy and vaccination, indicates potential for substantial tuberculosis reduction. To achieve this, maintaining the basic reproduction number  $R_0$  below 1 is critical. Key recommendations include intensive awareness campaigns, stigmatizing tuberculosis, ensuring free and accessible testing, and educating on transmission and home care. Discouraging factors like overcrowding, illiteracy, inadequate medical facilities, and high fertility rates is crucial. Additionally, the provision of trained personnel and expanded tuberculosis laboratory services is essential for effective disease management. Implementing these measures offers a comprehensive and informed approach to minimizing tuberculosis incidence in diverse populations.

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### Statement and Declaration

On behalf of all authors the corresponding author states that there is no conflict of interest.

### Data Availability Statement

Data sets generated during the current study are available from the corresponding author on reasonable request.

### Authorship Contribution Statement

Segun R. ADEBAYO: Data preparation, analysis, reviewing

Kazeem A. ODEYEMI: Supervision, analysis, simulation, Conceptualization, methodology

Rasheed G. AYoola: Methodology, computation.

Atinuke B. ADENIJI: Simulation, analysis, qualitative analysis

Aderonke O. OLUWAROTIMI: Writing, computations

Grace O. ADEBAYO: Typesetting and qualitative analysis

Rafiu A. ADELAKUN: Qualitative analysis, reviewing

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