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ORIGINAL RESEARCH

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 the 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, and efficient control methods in the global eradication of tuberculosis

Keywords/phrases: 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 and effective methods of reducing the negative effects of tuberculosis on public health (Daniel, 2020; Dauda et al., 2020; Egonmwan and Okuanghae, 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 general 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 (Itant *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.

2. Materials and method

2.1. Model formulation

A deterministic mathematical model based on the epidemiological status of the 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 β, α and Λ. The respective classes are subjected to natural death rateμ, while exposed individuals have disease-induced mortality rate ε and infected persons have recovery ratesγ.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 δ.

The model formulation flow to depict the aforementioned is given by Fig. 1.

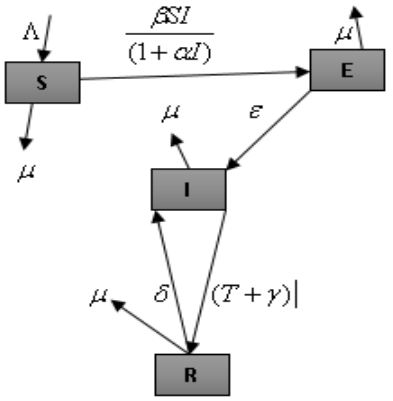

Fig 1: Schematic flow of model description.

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

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

3. 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$\Re_4^+$.at $t > 0$.

Proof:
Consider the total population

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

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

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

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

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

Firstly,

$N(0) = \frac{\Lambda}{\mu} + ce^{-\mu(0)}$, resulting to $c = N(0) - \frac{\Lambda}{\mu}$ (6)

Thus, substituting (6) into (5) as time progressively increases yields:

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

Then $N(0) \leq \frac{\Lambda}{\mu}$, then$N(t) \leq \frac{\Lambda}{\mu}$. This is a positive invariant set under the flow described by (2) so that no solution path leaves through any boundary$\Re_+^4$. Hence, it is sufficient to consider the dynamics of the model in the domain$\Re_+^4$. In this region, the model can be considered has be mathematically and epidemiologically well-posed. This shows that the total population and the subpopulation $S(t), E(t), I(t), R(t)$ of the model are bounded and is a unique solution. Hence, its applicability to studying physical systems is feasible.

Consequently, considering the compartmental disposition.

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

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta SI}{1+\alpha I} - \mu S \\
\frac{dS}{dt} &\geq -S(t)\left(\frac{\beta I}{1+\alpha I} + \mu\right) \\
\frac{dS}{S(t)} &\geq -\left(\frac{\beta I}{1+\alpha I} + \mu\right)dt \\
\int\frac{dS}{S(t)} &\geq -\int\left(\frac{\beta I}{1+\alpha I} + \mu\right)dt \\
InS(t) &\geq -\left(\frac{\beta I}{1+\alpha I} + \mu\right)t
\end{aligned}$$

$S(t) \geq S_0\ell^{-\left(\frac{\beta I}{1+\alpha I}+\mu\right)t} > 0$ (8)

At$t > 0$,$S(t) > 0$

In the second compartment,

$$\frac{dE}{dt} = \frac{\beta SI}{1+\alpha I} - (\mu+\varepsilon)E(t),$$
$$\frac{dE}{dt} \geq -(\mu+\varepsilon)E(t),$$
$$\frac{dE}{E(t)} \geq -(\mu+\varepsilon)dt$$
$$\int \frac{dE}{E(t)} \geq -\int (\mu+\varepsilon)\,dt$$
$$InE(t) \geq -t(\mu+\varepsilon)$$
$$E(t) \geq \ell^{-(\mu+\varepsilon)t}$$
$$E(t) \geq E_0\ell^{-t(\mu+\varepsilon)} > 0 \qquad (9)$$

At$t > 0$,$E(t) > 0$

Thirdly,

$$\frac{dI}{dt} = \varepsilon E - (\mu+\gamma)I - TI + \delta R$$
$$\frac{dI}{dt} \geq -(\mu+\gamma+T)I(t)$$
$$\frac{dI}{I(t)} \geq -(\mu+\gamma+T)dt$$
$$\int \frac{dI}{I(t)} \geq -\int (\mu+\gamma+T)\,dt$$
$$InI \geq -t(\mu+\gamma+T)$$
$$I(t) \geq \ell^{-t(\mu+\gamma+T)}$$
$$I(t) \geq I_0\ell^{-t(\mu+\gamma+T)} > 0 \qquad (10)$$

At$t > 0$,$I(t) > 0$

Lastly,

$$\frac{dR}{dt} = TI + \gamma I - \mu R - \delta R$$
$$\frac{dR}{dt} \geq -(\mu+\delta)R(t)$$
$$\int \frac{dR}{R(t)} \geq -\int (\mu+\delta)dt$$
$$InR(t) \geq -t(\mu+\delta)$$
$$R(t) \geq \ell^{-t(\mu+\delta)}$$
$$R(t) \geq R_0\ell^{-t(\mu+\delta)} > 0 \qquad (11)$$

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

3. 1. Tuberculosis- Non-Infected Equilibrium State

The equilibrium state of non-infected individuals with tuberculosis signifies a system devoid of Mycobacterium tuberculosis, encompassing individuals categorized as infected (I), exposed (E), and recovered (R)$I = E = R = 0$.

$$\frac{dN}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \qquad (12)$$

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

At no outbreak of tuberculosis, the class of the disease is subjected as$t = 0$, from (12),

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

Thus, the disease-free equilibrium yields:

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

3. 2. Steady-State Prevalence of Tuberculosis

Emphasizing the dynamic nature of tuberculosis prevalence, particularly its pivotal role in outbreaks and population is essential. When assessing the system at a steady state. Consider the set equations in (1), where the equilibrium points can be identified as $\Phi = (S^\bullet, E^\bullet, I^\bullet, R^\bullet)$ ar $t > 0$

$$I^\bullet = \frac{(\mu+\delta)(\varepsilon E+\delta R)(T+\gamma)}{(T+\gamma)(\mu+\delta)(\mu+\gamma+T)}, E^*$$
$$= \frac{(\varepsilon E+\delta R)(T+\gamma)\beta(\mu+\varepsilon)(1+\alpha)\Lambda}{\beta-\mu(1+\alpha)(T+\gamma)(\mu+\delta)(\mu+\gamma+T)},$$
$$S^* = \frac{1+\alpha\Lambda(\mu+\delta)(\varepsilon E+\delta R)(T+\gamma)}{\beta I-\mu(1+\alpha)(T+\gamma)(\mu+\delta)(\mu+\gamma+T)}, R^* = \frac{(\varepsilon E+\delta R)(T+\gamma)}{(\mu+\delta)(\mu+\gamma+T)} \qquad (15)$$

3. 3. Basic Reproduction Number

The basic reproduction number, denotedR_*, 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 involves calculating the F and V matrices, representing the rates of new infections and transitions into and out of the infected compartment, 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 equation (1) it is obtained for matrix $F$and$V$:

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

Such that

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

then,

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

$$FV^{-1} = \frac{1}{(\mu+\varepsilon)(\mu+\gamma+T)} \begin{pmatrix} \frac{\beta\Lambda}{\mu(1+\alpha\mu)^2} & 0 \\ 0 & 0 \end{pmatrix} \begin{pmatrix} (\mu+\gamma+T) & 0 \\ \varepsilon & (\mu+\varepsilon) \end{pmatrix}$$

$$R_* = \frac{\beta\Lambda}{\mu(\mu+\varepsilon)(\mu+\gamma+T)} (18)$$

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

3. 4. Asymptotic Stability of the Disease-Free State

This section examines the stability of the disease-free state for tuberculosis by analyzing the basic reproduction number's impact. 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 obtained as the Jacobian matrix of the system of (1) is obtained and evaluated at the disease state using the linearization method

$$J_{\ell_0} = \begin{pmatrix} -\mu 0 \beta S 0 \\ 0 - (\mu+\varepsilon)\beta S 0 \\ 0\varepsilon - (\mu+\gamma+T)\delta \\ 00T + \gamma - (\mu+\delta) \end{pmatrix}$$

$$J_{\ell_0} = \begin{pmatrix} -\mu 0 \frac{\Lambda\beta}{\mu} 0 \\ 0 - (\mu+\varepsilon)\frac{\Lambda\beta}{\mu} 0 \\ 0\varepsilon - (\mu+\gamma+T)\delta \\ 00T + \gamma - (\mu+\delta) \end{pmatrix} \qquad (19)$$

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

$$a = \begin{vmatrix} -(\mu+\gamma+T) - \lambda\delta \\ T + \gamma - (\mu+\delta) - \lambda \end{vmatrix}$$

$$(-(\mu+\gamma+T) - \lambda_3)(-(\mu+\delta) - \lambda_4) = 0, \lambda_4 = -(\mu+\delta), \lambda_3 = -(\mu+\gamma+T)\lambda_3 = -(\mu+\gamma+T) - (\mu+\varepsilon) - \lambda_2|A| = 0$$

$$\lambda_2 = -(\mu+\varepsilon)\lambda_1 = -\mu$$

$$\therefore \lambda_1 = -\mu, \lambda_2 = -(\mu+\varepsilon), \lambda_3 = -(\mu+\gamma+T), \lambda_4 = -(\mu+\delta)$$

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

3. 5. Regional Resilience of the Persistent Equilibrium

Theorem 3

The regional resilience of the persistent equilibrium of the proposed model is locally asymptotically stable if and unstable otherwise in the region of$\Re_+^4$ 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;

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

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

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

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

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

3. 6. 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;

Define $\Psi(t,S,E,I,R)=C_1I_1+C_2I_2$

$$\frac{d\Psi}{dt}=C_1I_1^{\bullet}+C_2I_2^{\bullet}=C_1\left(\frac{\beta S_0I_2}{1+\alpha S_0}-(\mu+\varepsilon)I_1\right)+C_2(\varepsilon I_1-(\mu+\gamma+T)]I_2+\delta R)$$

$$\frac{d\Psi}{dt}\le\left(C_2\varepsilon-C_1(\mu+\varepsilon)\right)I_1-\left(C_1\frac{\beta S_0}{(1+\alpha)S_0}-C_2(\mu+\gamma+T)+\delta R\right)I_2$$

$$\frac{d\Psi}{dt}\le C_1\left(C_2\varepsilon-C_1(\mu+\varepsilon)\right)I_1-C_2\left(C_1\frac{\beta\Lambda}{\mu(1+\alpha)}-C_2(\mu+\gamma+T)\right)I_{2_1}$$

$$C_1=\frac{1}{(\mu+\varepsilon)}, C_2=\frac{\beta\Lambda}{\mu(\mu+\varepsilon)(1+\alpha)(\mu+\gamma+T)}, R\le 0$$

$$\frac{d\Psi}{dt}\le\left(\frac{\beta\Lambda\varepsilon}{\mu(\mu+\varepsilon)(1+\alpha)(\mu+\gamma+T)}-\frac{(\mu+\varepsilon)}{(\mu+\varepsilon)}\right)I_1-\left(\frac{\beta\Lambda}{\mu(1+\alpha)(\mu+\varepsilon)}-\frac{\beta\Lambda(\mu+\gamma+T)}{\mu(1+\alpha)(\mu+\varepsilon)(\mu+\gamma+T)}\right)I_2$$

$$\frac{d\Psi}{dt}\le\left(\frac{\beta\Lambda\varepsilon}{\mu(\mu+\varepsilon)(1+\alpha)(\mu+\gamma+T)}-1\right)I$$

$$\frac{d\Psi}{dt}\le(R_0-1) \quad (23)$$

It is pertinent to note that when at $t\to\infty$, $\frac{d\psi}{dt}\le 0$. 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 $R_0>1$

3. 7 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;

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

from above system of equations results to;

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

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

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

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

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.

3. 8. 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

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

Table 1: Sensitivity analysis of parameters and indices

Parameter	Sensitivity indices
$\boldsymbol{\beta}$	$0.0112060 \times day^{-1}$
$\boldsymbol{\mu}$	$0.0151427\times day^{-1}$
$\boldsymbol{\beta}$	$1.000040\times day^{-1}$
$\boldsymbol{\beta}$	$1e^{-1} \times day^{-1}$
$\boldsymbol{\gamma}$	$1.0203010\times day^{-1}$
$\boldsymbol{T}$	$1.0326701\times day^{-1}$
$\boldsymbol{\alpha}$	$0.0212\times day^{-1}$

The sensitivity analysis of the basic reproduction number R_* for tuberculosis indicates significant insights into the disease's dynamics and control measures. All parameters of R_* 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.

3. 9. 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.

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

Subject to the boundary condition

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

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 Ψ. 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:

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

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

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

We can construct a Homotopy for (4) so that

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

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

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

And as $p \to 1$,

Simplifying equation (5) to yield this,

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

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

And we can express the solution of the differential equation as

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

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

$$\lim_{P \to 1} W(t) = W_1(t) + W_2(t) + W_3(t) + W_4(t) + \ldots \qquad (39)$$

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
S(t)	Susceptible individuals in the population
E(t)	Exposed individuals in the population
I(t)	Infected individuals in the population
R(t)	Recovered individuals in the population

Parameters	Descriptions	Values	References
$\boldsymbol{\Lambda}$	Recruitment rate	$120 \times days$	Carlos and Baojun, 2014
$\boldsymbol{\Lambda}$	Contact rate of susceptible per unit of time	$0.00124 \times days$	Daniel, 2020
$\boldsymbol{\Lambda}$	Induced death	$0.0133 \times days$	Lasalle, 1976
$\boldsymbol{\Lambda}$	Treatment rate	$0.0109395 \times days$	Carlos and Baojun, 2014
$\boldsymbol{\gamma}$	Progression rate from infected to recovered	$4.1301 \times 10^{-2} days$	Ibrahim *et al.*, 2017
$\boldsymbol{\mu}$	Natural death	$0.0766169 \times$ days	Dauda *et al.*, 2020
$\boldsymbol{\alpha}$	Transmission coefficient	$0.11 \times$ days	Omale *et al.*, 2019
$\boldsymbol{\delta}$	Reinfection	$0.052 \times$ days	Bisuta *et al.*, 2018

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

The correctional series assumes that the solutions for (1) are such that $S(t)=\sum_{k=0}^{n}p^k s_k(t), E(t)=\sum_{k=0}^{n}p^k e_k(t), I(t)=\sum_{k=0}^{n}p^k i_k(t), R(t)=\sum_{k=0}^{n}p^k r_k(t),$ (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

$$\begin{aligned}S_1(t)&=(\Lambda+\beta i_0 s_0-\alpha i_0-\mu s_0)t\\E_1(t)&=(\beta i_0 s_0--\alpha i_0-(\mu+\varepsilon)e_0)t\\I_1(t)&=(\varepsilon e_0-(\mu+\gamma)i_0-Ti_0-\delta r_0)t\\R_1(t)&=(T+\gamma)i_0-(\mu+\delta r_0)t\end{aligned} \quad (43)$$

For $n=2$

$$s_2(t)\frac{1}{2}t^2\begin{pmatrix}\alpha^3 i^2{}_0 s_0+\alpha^2\mu i_0 s_0+\alpha^2\beta i_0 s_0-\alpha^2\beta i_0 v_0\\-\alpha\delta i_0 s_0+2\alpha\mu i_0 s_0+\alpha\rho_0 s_0-\alpha\beta_0 s_0\\+\mu^2 s_0+2\mu\beta s_0-2\mu\beta v_0-\beta^2 s_0-\\\beta\beta v_0-\beta^2 v_0\end{pmatrix} \quad (44)$$

$$e_2(t)=-\tfrac{1}{2}t^2\begin{pmatrix}\alpha^2 i_0{}^2 s_0+\alpha\delta i_0 s_0+3\alpha\mu i_0 s_0+\\\alpha\rho_0 s_0-\alpha\sigma e_0 s_0+\alpha\sigma_{1_0} s_0+\\\alpha\beta i_0 s_0-\alpha\beta i_0 v_0-\\\mu^2 e_0-2\mu e_0\end{pmatrix}$$

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

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

At n = 3

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

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

$$I_3(t)=\tfrac{1}{2}t^3\begin{pmatrix}-2t^2\beta e_0 r_0+\varepsilon^2 t_0 s_0-\\2\gamma^2\varepsilon e_0 i_0-3\varepsilon^2\beta i_0 s_0\\\mu^2\beta s_0 r_0+\beta^2 t_0 s_0+\\2\mu^2\alpha e_0 r_0-\frac{1}{2}\varepsilon^2\beta s_0 r_0\\\mu^2\gamma s_0 r_0+\beta^2 t_0 e_0-\\2\delta^2\beta s_0 r_0-2\mu^2\alpha e_0 r_0+\\\gamma^2\beta i_0 r_0+2\mu^2\beta s_0 r_0\\+t^2 s_0 e_0+\beta^2 t_0 e_0-\\2\delta^2\beta s_0 r_0+\beta^2 t_0 s_0\end{pmatrix} \quad (46)$$

$$R_3(t) = \frac{1}{2}t^3 \begin{pmatrix} t^2\delta s_0 r_0 + \beta^2 t_0 e_0 - \\ 2\delta^2\beta s_0 r_0 + \beta^2 t_0 s_0 + \\ \beta^2 t_0 s_0 \beta^2 t_0 e_0 - 2\delta^2\beta s_0 r_0 + \\ \beta^2 t_0 s_0 3t^2 s_0 + \mu^2\varepsilon\alpha e_0 i_0 + \\ \varepsilon^2 t_0 s_0 - 2\gamma^2\varepsilon e_0 i_0 + \varepsilon^2\beta i_0 s_0 + \\ \mu^2\beta s_0 r_0 + 2\mu^2\beta s_0 r_0 + \\ \gamma^2\beta s_0 r_0 + \beta^2 t_0 i_0 - \mu^2\gamma s_0 r_0 + \\ \beta^2 t_0 e_0 + \beta^2 t_0 e_0 + 2\delta^2\beta s_0 r_0 \\ +\beta^2 t_0 s_0 \beta^2 t_0 s_0 \end{pmatrix}$$

Iteration of this is carried out until it is the required number of iterations are obtained. Thus, every variation of the compartment's raw solutions is obtained as We obtained that

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

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

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

We obtained that

$$\left. \begin{array}{c} S(t) = 500 - 30.4320t + \\ 0.7213561075t^2 - 0.03863404097t^3 \\ E(t) = 65 + 18.1785t - 1.171778775t^2 + \\ 0.04155466537t^3 \\ I(t) = 23 - 0.9060t + 0.02925067500t^2 \\ -0.0008440367800t^3 \\ R(t) = 14 - 0.0155t - 0.005054500000t^2 + \\ 0.0001458242542t^3 \end{array} \right\} \quad (48)$$

The approximate results of each class are evaluated using their respective baseline values in Table 1. We also suggest the following population data set as initial values given by $s_0 = 1000, e_0 = 30, i_0 = 20, 4_0 = 40$. Thus we obtain the following series of results embedding the parameters whose influence on the dynamics of tuberculosis transmission are to be analyzed

$$s(t) = 1000 + \begin{pmatrix} 65.2686 + 1.3362000\alpha \\ -1362.923\alpha^2 - 37.68 \end{pmatrix} t + \begin{pmatrix} -8.99856418\alpha^2 \\ +5499.838828\alpha^4 \\ 54.775643345 \\ +152.0510083\alpha^2 \\ -0.5381600 \\ +3333.926349 \\ 45.98509816 \\ -3.288025569\alpha \end{pmatrix} \frac{t^2}{2} - \begin{pmatrix} 11.30828286\alpha^2 \\ -66.76103861\alpha^3 \\ -0.003719829888c^2 \\ +40645.08576\alpha^4 \\ +935.98111186\alpha^2 c \\ +56.12092345 \\ -5.923814565\alpha \\ 302.0838612 \\ +36988.74452\alpha^6 \\ -84.74264814\alpha \end{pmatrix} \frac{t^3}{6} \quad (49)$$

$$e(t) = 30 + \begin{pmatrix} -45.62599000 \\ +1362.924999\alpha^2 \\ -1.336200000\alpha \end{pmatrix} t - \begin{pmatrix} -69.38980854 \\ -8.998569418\alpha^3 \\ +5499.839928\alpha^4 \\ +152.0510083\alpha^2 \\ -0.09970881600\alpha \\ +5378.993811\alpha^2 \\ +0.0000493608 \\ -5.292993669\alpha \end{pmatrix} \frac{t^2}{2} + \begin{pmatrix} 11.30828286\alpha^2 - 80.26339203\alpha^3 \\ -105.5276927 - 0.003719829888\alpha c \\ +48897.59557\alpha^4 + 1164.133657\alpha^2 \\ -0.7679873978\alpha + 1431.639314\alpha^4 \\ -1.875838588\alpha^3 + 16753.17626\alpha^2 \\ +0.000384108040 - 16.31203298\alpha \\ +36988.74452\alpha^6 - 84.74264814\alpha^5 \end{pmatrix} \frac{t^3}{6}$$

$$i(t) = 20 - 50.40320t - \begin{pmatrix} 127.0391180 \\ +0.681642000\alpha^2 \\ -0.000668100\alpha \end{pmatrix} \frac{t^2}{2} - \begin{pmatrix} -0.004499284709\alpha^3 \\ +320.2194878 \\ +2.749919964\alpha^4 \\ +0.0765345416\alpha^2 \\ -0.00004985440800\alpha \\ +4.407401276\alpha^2 \\ +2.46804232 \cdot 10^{-8} \\ -0.004212705\alpha \end{pmatrix} \frac{t^3}{6} \quad (50)$$

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

4. Results and discussion of iteration

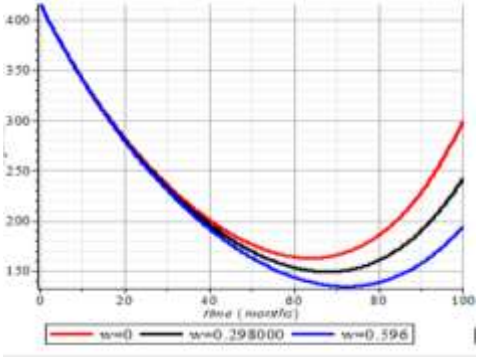

Fig.2: Adverse effect of treatment rate on that of reinfection on the individuals in the recovered population.

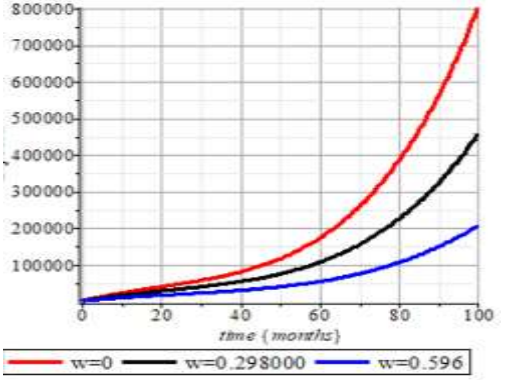

Fig 3: Effect of rapid treatment on the exposed individuals in the population

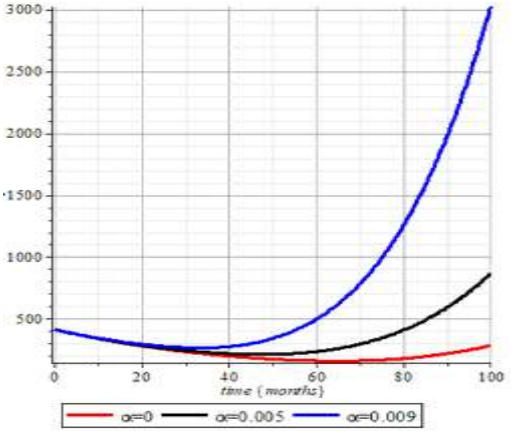

Fig 4: The effect of treatment rate per contactβ and transmission coefficient αa on the infected individuals in the population

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 β 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_* 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

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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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