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A Mathematical Analysis of Tuberculosis Transmission Using a Two-Age Group Compartmental Model

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Abstract

In this study, a two-age-group compartmental model for tuberculosis transmission is developed and analyzed, distinguishing between individuals below 10 years and those above this age. The model includes key epidemiological features such as reinfection within both age groups and the transition of treated individuals back to full susceptibility, representing realistic post-treatment outcomes. Essential analytical properties like non-negativity, existence, and uniqueness of solutions are established to confirm the model's biological validity. Equilibrium analysis identifies both disease-free and endemic steady states, and the basic reproduction number, R_0, is derived using the next-generation matrix method. Local stability analysis indicates that the disease-free equilibrium is asymptotically stable when $R_0 < 1$, suggesting effective disease control, while instability occurs when $R_0 > 1$, resulting in disease persistence. Additionally, the global stability of both equilibrium states is rigorously proven—employing the Metzler matrix method for the disease-free case and a Lyapunov function for the endemic state—demonstrating the model's strong dynamical behavior. Sensitivity analysis of R_0 identifies parameters that significantly impact tuberculosis transmission dynamics, offering insights for targeted intervention strategies. Scenario analyses, supported by three-dimensional plots, illustrate how variations in parameters influence R_0 and the spread of infection. Numerical simulations conducted in Python validate the analytical findings, indicating an increase in immunity as individuals age from under 10 to above 10 years, while higher contact rates among children considerably enhance transmission potential. This study provides a deeper understanding of age-dependent tuberculosis dynamics and offers relevant implications for disease control policies across different age groups.

Keywords: Tuberculosis (TB), Epidemiology, Simulation, Transmission, Two-group.

1. Introduction

Tuberculosis (TB) is a bacterial health issue caused by the Mycobacterium tuberculosis complex (MTBC) [10]. Despite ongoing scientific research and interventions, tuberculosis continues to pose significant risks, resulting in considerable discomfort and mortality

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among both the elderly and young. TB is an airborne disease that spreads through droplets expelled during coughing, sneezing, or spitting [37]. The bacteria can exist in two forms: latent TB, where the bacteria are not contagious, and active TB, where symptoms are present and transmission is possible [6]. Tuberculosis (TB) was the leading infectious cause of death worldwide by a single agent and continues to pose significant challenges to prevention and care efforts worldwide. Historically, the focus has been predominantly on adult TB cases, and surveillance data have often failed to provide breakdowns for adolescents, leading to a neglect of their specific needs in prevention and care policies. A recent estimate indicated that in 2012, approximately 1.8 million adolescents and young adults (aged 10–24) were diagnosed with tuberculosis, accounting for 17% of all new tuberculosis cases worldwide [30]. Adolescence is a critical developmental phase marked by increased susceptibility to TB, along with unique social and developmental factors that increase the risk of transmission, infection, and disease [29], [31]. To effectively combat TB on a global scale, strategies must be implemented that specifically address the needs of adolescents. In children, the majority of TB infections are classified as latent TB [7]. When infected with TB bacteria, children are more susceptible to developing TB disease than adults and tend to become ill more rapidly. In adults, TB disease typically arises from a dormant TB infection that reactivates years later, often when their immune systems are compromised for various reasons [7]. The most prevalent type of TB in children under five years of age is pulmonary TB (PTB), which is frequently not confirmed by bacteriological tests. According to [32], Extra-Pulmonary TB (EPTB) is also common in this age group and can occur alongside pulmonary TB, depending on the child's age. [32, 7] have noted several indicators of TB in children, including reduced playfulness, inadequate weight gain or weight loss, prolonged fever, and persistent cough that does not improve. Adults typically experience these symptoms along with additional signs such as decreased appetite, chest pain, blood in the sputum, swollen lymph nodes, and a cough lasting more than three weeks.

The diagnosis of tuberculosis (TB) in children is a complex task, and the actual prevalence can only be approximated, as noted in [?28, 34]. Contributing factors include a lack of scientific research on childhood TB, uncertainty about its outcomes, and the perception that pediatric TB is not a significant concern for TB control efforts. In 2000, approximately 8.3 million new tuberculosis cases were reported worldwide, approximately 10% involving children under the age of 15 years [34, 17]. The World Health Organization ([36]) estimated that in 2017, there were about 10 million new TB cases globally, with 10% of these occurring in children under 15 years of age. Approximately 1.5 million children under the age of five and adolescents develop TB annually [32]. A community survey conducted in South Africa revealed that children under 13 years of age contribute 14% to the total TB cases, corresponding to an annual incidence of 408 per 100,000 [34]. According to [19], the World Health Organization reported 10 million active TB cases in 2020 and an increase to 10.6 million in 2022, highlighting a rise in active cases over time and underscoring the need for new scientific strategies to combat TB. A detailed investigation of childhood TB by [34] found that previous TB interventions have mainly targeted adults, leaving childhood TB largely overlooked. The authors emphasized that children are at increased risk for severe illness and death after infection and that some adult TB cases may stem from the reactivation of latent infections acquired during childhood. Furthermore, they pointed out that childhood tuberculosis accounts for approximately 14% of all cases, usually contracted by adults with active infections. In support of this observation, [32] identified TB as a significant cause of illness in children under 10 years of age, noting that many cases go undiagnosed and can lead to fatalities, particularly in countries with a high incidence of TB. Ultimately, neglecting childhood TB could lead to future epidemics, highlighting the urgent need to study, understand, and prevent TB infections in children under 10.

Mathematical models play a crucial role in understanding the dynamics of infectious diseases, as highlighted in several studies [13, 24, 16, 11, 25, 18, 1, 2, 3, 9, 5, 4]. For example, [38] developed a mathematical model for tuberculosis that categorizes the population into three age groups: children, middle-aged individuals, and seniors. This model examines how age influences the transmission dynamics of tuberculosis in mainland China. The findings indicate that the impact of tuberculosis varies between different age groups, with recommendations to improve recovery rates in the older age group and reduce the infection rate to mitigate the spread of the disease. In a related investigation, [14] evaluated the epidemiological characteristics and treatment outcomes of children with TB, comparing those under 10 years of age with those aged 10 to 14 in rural Ethiopia, where data on childhood tuberculosis are scarce. Their analysis revealed that more than half of the children studied had pulmonary TB (PTB), with a higher prevalence in those under 10 years of age, while adenitis TB was less common in this age group. They concluded that cases of extrapulmonary tuberculosis (EPTB) were less common among children under 10 years of age compared to their older counterparts. Similarly, [8] examined the transmissibility of tuberculosis between students and non-students, finding that the average effective reproductive number (R_{eff}) for TB in the non-student population was 23.30 times greater than in the students. This highlights the significantly higher transmissibility of TB in nonstudent populations within the regions studied. Continuing this focus on population differences, [33] designed a two-group malaria model structured by age, categorizing individuals aged 10 years and older. They used Latin-Hypercube sampling along with partial rank correlation coefficients to conduct a global sensitivity analysis of the basic reproduction number and vaccination class, among other factors. The results suggested that implementing a combination of personal protection, treatment, and vaccination simultaneously is the most effective strategy for combating malaria epidemics, rather than relying on single or dual interventions. In another study, [?] identified the most sensitive variables pertinent to TB infection control, using the normalized forward sensitivity analysis index to assess the impacts of treatment and vaccination rates. Their findings indicated that increasing vaccination and treatment rates for infected individuals can significantly reduce the prevalence and burden of tuberculosis within the population.

Previous research on tuberculosis has focused primarily on adult infections, with limited attention given to children, particularly regarding transmission dynamics. In contrast, the work of [33] on malaria provides a clearer understanding of the dynamics of the disease in children, including various intervention strategies. We propose a comparable mathematical model for tuberculosis transmission that considers two age groups: children under 10 years of age and those aged 10 and older. This approach aims to improve our understanding of tuberculosis transmission and the interactions between these age groups, ultimately helping to reduce the incidence of intergroup transmission of infection.

The structure of our paper is organized as follows. Section 2 presents the model formulation, including equations and diagrams that illustrate the various compartments of the model. In Section 3, we analyze the model, covering topics such as non-negativity, the existence and uniqueness of solutions, the presence of endemic and disease-free equilibrium points, the basic reproduction number, and both local and global stability. Section 4 details sensitivity and scenario analyses, while Section 5 presents the numerical simulation results. Finally, Section 6 provides the conclusion.

2. Model formulation

The model is an SEIR tuberculosis model of the human population. The total human population of individuals at time t, denoted by $Nh(t)$, is divided into seven compartments comprising Susceptible individuals under 10 years $(Su(t))$, Susceptible individuals over 10 years $(Sv(t))$, Exposed individuals under 10 years $(Eu(t))$, Exposed individuals over 10 years $(Ev(t))$, Infected individuals under 10 years $(Iu(t))$, Infected individuals over 10 years $(Iv(t))$, Recovered under 5 years $(Ru(t))$ and Recovered individuals over 10 years $(Rv(t))$. Hence,

$$Nh(t) = Su(t) + Sv(t) + Eu(t) + Ev(t) + Iu(t) + Iv(t) + Ru(t) + Rv(t)$$

The susceptible under 10 years Su are recruited at a constant rate Λu and can die naturally at the rate μ, or become susceptible over 10 years Sv at the rate ϑ. They are decreased after contact with tuberculosis disease at the rate $\lambda u = \frac{b1\beta u}{Nh} Iu$, where β are the transmission probabilities and b are the rate per capitacontact. When susceptible people under 10 years of age grow, they add up to the number of susceptible people over 10 years (Sv) who are recruited at the rate Λv and can both die naturally at the rate μ, or be exposed Ev at the rate $\lambda v = \frac{b2\beta v}{Nh} Iv$. Individuals exposed less than 10 years (Eu) and over 10 years (Ev) increase by reinfection of recovered individuals ω. The tuberculosis model assumes that infected individuals will become totally susceptible again after treatment r. Exposed individuals become infected at a rate α. In exposed classes, individuals can recover at ψ. Infected individuals under 10 years (Iu) and over 10 years (Iv) can recover from the disease at a rate ρ or die as a result of the disease δ or die naturally μ. As a result of the interaction between the age group, the susceptible compartment after 10 years (Sv) is populated by susceptibles under 10 years of age at (ϑ) and dies naturally. Recovered individuals R are not totally immune to tuberculosis infection, but they do confer some immunity after the primary infection, and since there is no absolute cure for TB, they can be reinfected at a rate ω.

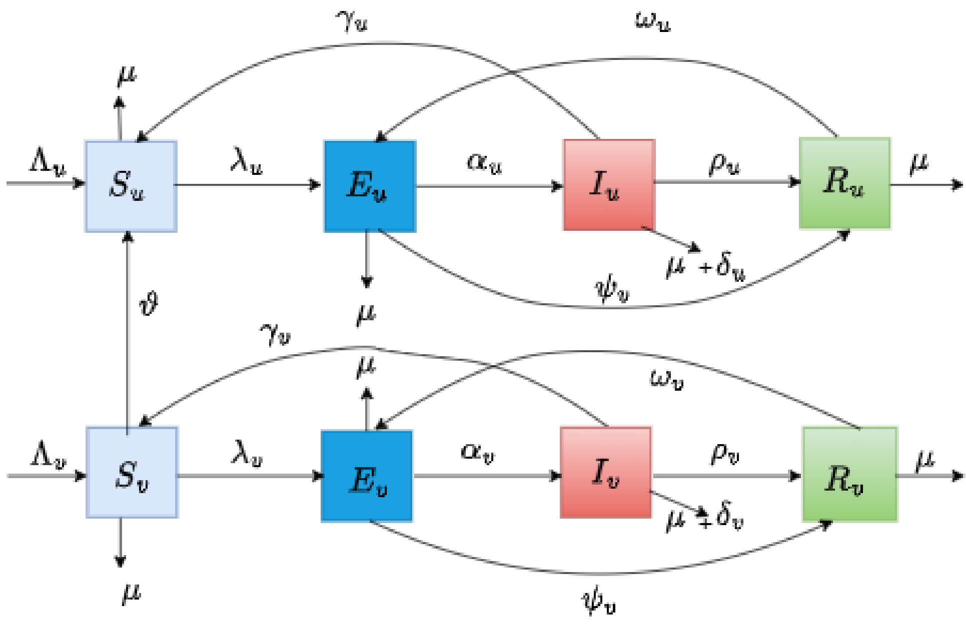


Figure 1: Tuberculosis transmission Model with Control Measures Schematic Diagram

Following the above assumptions, the model equation is given by:

$$
\begin{aligned}
\frac{dS_u}{dt} &= \Lambda u + ruIu - (\vartheta + \lambda_u + \mu)S_u \\
\frac{dS_v}{dt} &= \Lambda v + rvIv + \vartheta Su - (\lambda v + \mu)Sv \\
\frac{dE_u}{dt} &= \lambda uSu + \omega_u Ru - (\alpha u + \psi u + \mu)Eu \\
\frac{dE_v}{dt} &= \lambda vSv + \omega vRv - (\alpha v + \psi v + \mu)Ev \\
\frac{dI_u}{dt} &= \alpha uEu - (\rho u + ru + \mu + \delta u)Iu \\
\frac{dI_v}{dt} &= \alpha vEv - (\rho v + rv + \mu + \delta v)Iv \\
\frac{dR_u}{dt} &= \rho uIu + \psi uEu - (\omega u + \mu)Ru \\
\frac{dR_v}{dt} &= \rho vIv + \psi vEv - (\omega v + \mu)Rv
\end{aligned}
\tag{2.1}
$$

Where,

$$
\lambda u = \frac{b_1 \beta uIu}{N(t)} \tag{2.2}
$$

and

$$
\lambda v = \frac{b_2 \beta vIv}{N(t)} \tag{2.3}
$$

Table 1: Model Variables/parameters and interpretation.

Variables and Parameters	**Descriptions**
S_u	Susceptible under 10 years
S_v	Susceptible over 10 years
E_u	Exposed under 10 years
E_v	Exposed over 10 years
I_u	Infectious under 10 years
I_v	Infectious over 10 years
R_u	Recovered under 10 years
R_v	Recovered over 10 years
Λ_u	Recruitment rate of under 10 years
Λ_v	Recruitment rate of over 10 years
α_u	Progress rate from infected to infectious under 10 years
ρ_u	Recovery rates for infectious under 10 years
δ_u	Disease-induced under 10 years
ϑ	Maturation rate of individuals under 10 years
α_v	Progress rate from exposed to infection for over 10 years
ρ_v	Recovery rate for individuals over 10 years
μ	Natural death rate
δ_v	Diseased-induced death rate of individuals over 10 years
$b1$	Average contact rate under 10 years
$b2$	Average contact rate over 10 years
β_u	Probability of infection under 10 years
β_v	Probability of infection over 10 years
r_u	The treatment rate for the infected individuals under 10 years
r_v	The treatment rate for the infected individuals over 10 years
ω_u	The re-infection of individuals under 10 years
ω_v	The re-infection of individuals over 10 years
ψ_u	Recovery exposed individuals under 10 years
ψ_v	Recovery exposed individuals over 10 years

3. Analysis of the model

3.1. Non-negativity of the solutions

In order for model (2.1) to be epidemiologically meaningful, it is necessary to show that the solutions are non-negative over the passage of time. This result is established

Theorem 3.1. *Given the initial states* $S\sqcap(0) \geqslant 0, Sv(0) \geqslant 0, Eu(0) \geqslant 0, Ev(0) \geqslant 0, Iu(0) \geqslant 0, Iv(0) \geqslant 0, Ru(0) \geqslant 0, Rv(0) \geqslant 0$.
Then the solutions, $(Su(t) \geqslant 0, Sv(t) \geqslant 0, Eu(t) \geqslant 0, Ev(t) \geqslant 0, Iu(t) \geqslant 0, Iv(t) \geqslant 0, Ru(t) \geqslant 0, Rv(t) \geqslant 0)$ of model (1) are non-negative for all time $t > 0$.

Proof:

Let $t_1 = \sup\{t > 0 : \mathcal{S}\sqcap(0) > 0, \mathcal{S}\sqsubseteq(0) > 0, Eu(0)>0, E\nu(0) > 0, Iu(0)>0, I\nu(0) > 0, Ru(0)>0, R\nu(0) > 0\}$.

The 1st equation of (2.1) is given by

$$\frac{dSu}{dt} = \Lambda u + ruIu - (\vartheta + \lambda u + \mu)Su \tag{3.1}$$

$$\frac{dSu}{dt} \geqslant -(\vartheta + \lambda u + \mu)Su$$

$$\int \frac{dSu}{S} u \geqslant - \int (\vartheta + \lambda u + \mu)dt$$

$$\ln Su \geqslant -(\vartheta + \lambda u + \mu)dt + C,$$

where C is arbitrary constant

$$Su(t) \geqslant Ce^{-}(\vartheta + \lambda u + \mu)t$$

At initial condition, $t = 0, Su(t) = Su(0) = C$

$$Su(t) = Su(0)e^{-}(\vartheta + \lambda u + \mu)t \geqslant 0$$

Hence, $Su(t) \geqslant 0$ for all time $t > 0$.
Using the same technique, we have
$S\nu(t) \geqslant 0, Eu(t) \geqslant 0, E\nu(t) \geqslant 0, Iu(t) \geqslant 0, I\nu(t) \geqslant 0, Ru(t) \geqslant 0, R\nu(t) \geqslant 0$
Thus, $Su(t), S\nu(t), Eu(t), E\nu(t), Iu(t), I\nu(t), Ru(t), R\nu(t)$ will remain non-negative for all $t > 0$

3.2. Existence and Uniqueness of Solution of the Model

The general first-order ODE is in the form:

$$x' = f(t, x), x(t_0) = x_0 \tag{3.2}$$

One will be interested in asking the following questions:

1. Under what conditions can we say solution to equation (3.2) exists?
2. Under what conditions can we say there is a unique solution to equation (3.2)

To answers these;
Let:

$$\begin{aligned}
f_1 &= \Lambda u + ruIu - (\vartheta + \lambda u + \mu)Su \\
f_2 &= \Lambda v + rvIv + \vartheta Su - (\lambda v + \mu)Sv \\
f_3 &= \lambda uSu + \omega uRu - (\alpha u + \psi u + \mu)Eu \\
f_4 &= \lambda vSv + \omega vRv - (\alpha v + \psi v + \mu)Ev \\
f_5 &= \alpha uEu - (\rho u + ru + \mu + \delta u)Iu \\
f_6 &= \alpha vEv - (\rho v + rv + \mu + \delta v)Iv \\
f_7 &= \rho uIu + \psi uEu - (\omega u + \mu)Ru \\
f_8 &= \rho vIv + \psi vEv - (\omega v + \mu)Rv
\end{aligned} \tag{3.3}$$

We use the following theorem to established the existence and uniqueness of solution for our model.

Theorem 3.2. *(Uniqueness of Solution)*
Let D *denote the region*

$$\left.\begin{aligned}
|t - t_0| \leqslant a,\ ||x - x_0|| \leqslant b, \\
x = (x_1, x_2, ..., x_n) \\
x_0 = (x_{10}, x_{20}, ..., x_{n0})
\end{aligned}\right\} \tag{3.4}$$

and suppose that $f(t, x)$ satisfies the Lipschitz condition:

$$||f(t, x_1) - f(t, x_2)|| \leqslant K||x_1 - x_2|| \tag{3.5}$$

whenever the pair (t, x_1) and (t, x_2) belong to D, where K is a positive constant. Then there exist a constant $\delta > 0$ such that there exist a unique continuous vector solution $x(t)$ of the equation (3.5) in the interval $|t - t_0| < \delta$.
be continuous and bounded in the domain D.

Lemma 3.3. *If* $f(t, x)$ *has continuous partial derivative* $\frac{\partial f_i}{\partial xj}$ *on a bounded closed convex domain* R *(i.e, convex set of real numbers), where* R *is used to denotes real numbers , then it satisfies a Lipschitz condition in* R. *Our interest is in the domain:*

$$1 \leqslant \epsilon \leqslant R \tag{3.6}$$

So, we look for a bounded solution of the form

$$0 < R < \infty$$

We now prove the following existence theorem.

Theorem 3.4. *(Existence of Solution)*
Let D *denote the domain defined in (3.4) such that (3.5) and (3.6) hold. Then there exist a solution of model of equations (2.1) which is bounded in the domain* D.

Proof. From equation (2.1), let

$$\begin{aligned} f_1 &= \Lambda u + ruIu - (\vartheta + \lambda u + \mu)Su \\ f_2 &= \Lambda v + rvIv + \vartheta Su - (\lambda v + \mu)Sv \\ f_3 &= \lambda uSu + \omega uRu - (\alpha u + \psi u + \mu)Eu \\ f_4 &= \lambda vSv + \omega vRv - (\alpha v + \psi v + \mu)Ev \\ f_5 &= \alpha uEu - (\rho u + ru + \mu + \delta u)Iu \\ f_6 &= \alpha vEv - (\rho v + rv + \mu + \delta v)Iv \\ f_7 &= \rho uIu + \psi uEu - (\omega u + \mu)Ru \\ f_8 &= \rho vIv + \psi vEv - (\omega v + \mu)Rv \end{aligned} \tag{3.7}$$

$\frac{\partial f_i}{\partial x_j}, i, j = 1, 2, 3, 4$ are continuous and bounded. That is, the partial derivatives are continuous and bounded. We explored the following partial derivatives for all the model equations:

□

From the first equation of (3.7)

$$\frac{\partial f_1}{\partial S_u} = -(\vartheta + \lambda_u + \mu) \left|\frac{\partial f_1}{\partial S_u}\right| = |-(\vartheta + \lambda_u + \mu)| = |(\vartheta + \lambda_u + \mu)| < \infty, \frac{\partial f_1}{\partial S_v} = 0, \left|\frac{\partial f_1}{\partial S_v}\right| = |0| < \infty,$$

$$\frac{\partial f_1}{\partial E_u} = 0, \left|\frac{\partial f_1}{\partial E_u}\right| = |0| < \infty, \frac{\partial f_1}{\partial E_v} = 0, \left|\frac{\partial f_1}{\partial E_v}\right| = |0| < \infty, \frac{\partial f_1}{\partial I_u} = r_u, \left|\frac{\partial f_1}{\partial I_u}\right| = |r_u| < \infty,$$

$$\frac{\partial f_1}{\partial I_v} = 0, \left|\frac{\partial f_1}{\partial I_v}\right| = |0| < \infty, \frac{\partial f_1}{\partial R_u} = 0, \left|\frac{\partial f_1}{\partial R_u}\right| = |0| < \infty, \frac{\partial f_1}{\partial R_v} = 0, \left|\frac{\partial f_1}{\partial R_v}\right| = |0| < \infty.$$

Similarly, from the second equation of (3.7), we have

$$\frac{\partial f_2}{\partial S_v} = \vartheta, \left|\frac{\partial f_2}{\partial S_v}\right| = |\vartheta| < \infty, \frac{\partial f_2}{\partial S_v} = -(\lambda v + \mu), \left|\frac{\partial f_2}{\partial S_v}\right| = |(\lambda_v + \mu)| < \infty, \frac{\partial f_2}{\partial E_u} = 0, \left|\frac{\partial f_2}{\partial E_u}\right| = |0| < \infty,$$

$$\frac{\partial f_2}{\partial E_v} = 0, \left|\frac{\partial f_2}{\partial E_v}\right| = |0| < \infty, \frac{\partial f_2}{\partial I_u} = 0, \left|\frac{\partial f_2}{\partial I_u}\right| = |0| < \infty, \frac{\partial f_2}{\partial I_v} = r_v, \left|\frac{\partial f_2}{\partial I_v}\right| = |r_v| < \infty,$$

$$\frac{\partial f_2}{\partial R_u} = 0, \left|\frac{\partial f_2}{\partial R_u}\right| = |0| < \infty, \frac{\partial f_2}{\partial R_v} = 0, \left|\frac{\partial f_2}{\partial R_v}\right| = |0| < \infty.$$

Similarly, from the third equation of (3.7), we have

$$\frac{\partial f_3}{\partial S_u} = \lambda_u, \left|\frac{\partial f_3}{\partial S_u}\right| = |\lambda_u| < \infty, \frac{\partial f_3}{\partial S_v} = 0, \left|\frac{\partial f_3}{\partial S_v}\right| = |0| < \infty,$$

$$\frac{\partial f_3}{\partial E_u} = -(\alpha_u + \psi u + \mu), \left|\frac{\partial f_3}{\partial E_u}\right| = |(\alpha_u + \psi u + \mu)| < \infty, \frac{\partial f_3}{\partial E_v} = 0, \left|\frac{\partial f_3}{\partial E_v}\right| = |0| < \infty,$$

$$\frac{\partial f_3}{\partial I_u} = 0, \left|\frac{\partial f_3}{\partial Iu}\right| = |0| < \infty,$$

$$\frac{\partial f_3}{\partial I_v} = 0, \left|\frac{\partial f_3}{\partial I_v}\right| = |0| < \infty, \frac{\partial f_3}{\partial R_u} = \omega_u, \left|\frac{\partial f_3}{\partial R_u}\right| = |\omega_u| < \infty, \frac{\partial f_3}{\partial R_v} = 0, \left|\frac{\partial f_3}{\partial R_v}\right| = |0| < \infty,$$

Similarly, from the fourth equation of (3.7), we have

$$\frac{\partial f_4}{\partial S_u} = 0, \left|\frac{\partial f_4}{\partial S_u}\right| = |0| < \infty, \frac{\partial f_4}{\partial S_v} = \lambda_v, \left|\frac{\partial f_4}{\partial S_v}\right| = |\lambda_v| < \infty, \frac{\partial f_4}{\partial E_u} = \vartheta, \left|\frac{\partial f_4}{\partial E_u}\right| = |\vartheta| < \infty,$$

$$\frac{\partial f_4}{\partial E_v} = -(\alpha_v + \psi_v + \mu), \left|\frac{\partial f_4}{\partial E_v}\right| = |(\alpha_v + \psi_v + \mu)| < \infty, \frac{\partial f_4}{\partial I_u} = 0, \left|\frac{\partial f_4}{\partial I_u}\right| = |0| < \infty,$$

$$\frac{\partial f_4}{\partial I_v} = 0, \left|\frac{\partial f_4}{\partial I_v}\right| = |0| < \infty,$$

$$\frac{\partial f_4}{\partial R_u} = 0, \left|\frac{\partial f_4}{\partial R_u}\right| = |0| < \infty, \frac{\partial f_4}{\partial R_v} = \omega_v, \left|\frac{\partial f_4}{\partial R_v}\right| = |\omega_v| < \infty.$$

Similarly, from the fifth equation of (3.7), we have

$$\frac{\partial f_5}{\partial S_u} = 0, \left|\frac{\partial f_5}{\partial S_u}\right| = |0| < \infty, \frac{\partial f_5}{\partial S_v} = 0, \left|\frac{\partial f_5}{\partial S_v}\right| = |0| < \infty, \frac{\partial f_5}{\partial E_u} = \alpha_u, \left|\frac{\partial f_5}{\partial E_u}\right| = |\alpha_u| < \infty,$$

$$\frac{\partial f_5}{\partial E_v} = 0, \left|\frac{\partial f_5}{\partial E_v}\right| = |0| < \infty, \frac{\partial f_5}{\partial I_u} = -(\rho_u + r_u + \mu + \delta_u), \left|\frac{\partial f_5}{\partial I_u}\right| = |(\rho_u + r_u + \mu + \delta_u)| < \infty,$$

$$\frac{\partial f_5}{\partial I_v} = 0, \left|\frac{\partial f_5}{\partial I_v}\right| = |0| < \infty, \frac{\partial f_5}{\partial R_u} = 0, \left|\frac{\partial f_5}{\partial R_u}\right| = |0| < \infty, \frac{\partial f_5}{\partial R_v} = 0, \left|\frac{\partial f_5}{\partial R_v}\right| = |0| < \infty.$$

Similarly, from the sixth equation of (3.7), we have

$$\frac{\partial f_6}{\partial S_u} = 0, \left|\frac{\partial f_6}{\partial S_u}\right| = |0| < \infty, \frac{\partial f_6}{\partial S_v} = 0, \left|\frac{\partial f_6}{\partial S_v}\right| = |0| < \infty, \frac{\partial f_6}{\partial E_u} = 0, \left|\frac{\partial f_6}{\partial E_u}\right| = |0| < \infty,$$

$$\frac{\partial f_6}{\partial E_v} = \alpha_v, \left|\frac{\partial f_6}{\partial E_v}\right| = |\alpha_v| < \infty, \frac{\partial f_6}{\partial I_u} = 0, \left|\frac{\partial f_6}{\partial I_u}\right| = |0| < \infty,$$

$$\frac{\partial f_6}{\partial I_v} = -(\rho_v + r_v + \mu + \delta_v), \left|\frac{\partial f_6}{\partial I_v}\right| = |(\rho_v + r_v + \mu + \delta_v)| < \infty, \frac{\partial f_6}{\partial R_u} = 0, \left|\frac{\partial f_6}{\partial R_u}\right| = |0| < \infty,$$

$$\frac{\partial f_6}{\partial R_v} = 0, \left|\frac{\partial f_6}{\partial R_v}\right| = |0| < \infty.$$

Similarly, from the seventh equation of (3.7), we have

$$\frac{\partial f_7}{\partial S_u} = 0, \left|\frac{\partial f_7}{\partial S_u}\right| = |0| < \infty, \frac{\partial f_7}{\partial S_v} = 0, \left|\frac{\partial f_7}{\partial S_v}\right| = |0| < \infty, \frac{\partial f_7}{\partial E_u} = \psi_u, \left|\frac{\partial f_7}{\partial E_u}\right| = |\psi_u| < \infty,$$

$$\frac{\partial f_7}{\partial E_v} = 0, \left|\frac{\partial f_7}{\partial E_v}\right| = |0| < \infty, \frac{\partial f_7}{\partial I_u} = \rho_u, \left|\frac{\partial f_7}{\partial I_u}\right| = |\rho_u| < \infty, \frac{\partial f_7}{\partial I_v} = 0, \left|\frac{\partial f_7}{\partial I_v}\right| = |0| < \infty,$$

$$\frac{\partial f_7}{\partial R_u} = -(\omega_u + \mu), \left|\frac{\partial f_7}{\partial R_u}\right| = |(\omega_u + \mu)| < \infty, \frac{\partial f_7}{\partial R_v} = 0, \left|\frac{\partial f_7}{\partial R_v}\right| = |0| < \infty.$$

Finally, from the final equation (3.7), we have

$$\frac{\partial f_8}{\partial S_u} = 0, \left|\frac{\partial f_8}{\partial S_u}\right| = |0| < \infty, \frac{\partial f_8}{\partial S_v} = 0, \left|\frac{\partial f_8}{\partial S_v}\right| = |0| < \infty, \frac{\partial f_8}{\partial E_u} = 0, \left|\frac{\partial f_8}{\partial E_u}\right| = |0| < \infty,$$

$$\frac{\partial f_8}{\partial E_v} = \psi_v, \left|\frac{\partial f_8}{\partial E_v}\right| = |\psi_v| < \infty, \frac{\partial f_8}{\partial I_u} = 0, \left|\frac{\partial f_8}{\partial I_u}\right| = |0| < \infty, \frac{\partial f_8}{\partial I_v} = \rho_v, \left|\frac{\partial f_8}{\partial I_v}\right| = |\rho_v| < \infty,$$

$$\frac{\partial f_8}{\partial R_u} = 0, \left|\frac{\partial f_8}{\partial R_u}\right| = |0| < \infty, \frac{\partial f_8}{\partial R_v} = -(\omega_v + \mu), \left|\frac{\partial f_8}{\partial R_v}\right| = |(\omega_v + \mu)| < \infty.$$

We have unequivocally demonstrated that all partial derivatives in the system (2.1) are continuous and bounded. As a result, Theorems (2) and (3) ensure the existence and uniqueness of the solution within the region D. This confirms that the model is mathematically sound and reliable for analyzing tuberculosis dynamics in the two age groups.

3.3. Existence Of Endemic Equilibrium Point

The endemic equilibrium point occurs at a point where all the differential equation models (2.1) equal zero, that is,

$$\Lambda_u + r_u I_u - (\vartheta + \lambda_u + \mu)S_u = 0 \tag{3.8}$$

$$\Lambda_v + r_v I_v + \vartheta S_u - (\lambda_v + \mu)S_v = 0 \tag{3.9}$$

$$\lambda_u S_u + \omega_u R_u - (\alpha_u + \psi_u + \mu)E_u = 0 \tag{3.10}$$

$$\lambda v S_v + \omega_v R_v - (\alpha_v + \psi_v + \mu)E_v = 0 \tag{3.11}$$

$$\alpha_u E_u - (\rho_u + r_u + \mu + \delta_u)I_u = 0 \tag{3.12}$$

$$\alpha_v E_v - (\rho_v + r_v + \mu + \delta_v)I_v = 0 \tag{3.13}$$

$$\rho_u I_u + \psi_u E_u - (\omega_u + \mu)R_u = 0 \tag{3.14}$$

$$\rho_v I_v + \psi_v E_v - (\omega_v + \mu)R_v = 0 \tag{3.15}$$

Solving (3.8) to (3.15) gives the equilibrium at

$$\begin{aligned}
S_u^* &= \frac{\Lambda_u + r_u I_u}{(\vartheta + \lambda_u + \mu)} \\
S_v^* &= \frac{\Lambda_u + r_v I_v + \vartheta S_u}{(\lambda_v + \mu)} \\
E_u^* &= \frac{\lambda_u S_u + \omega_u R_u}{(\alpha_u + \psi_u + \mu)} \\
E_v^* &= \frac{\lambda_v S_v + \omega_v R_v}{(\alpha_v + \psi_v + \mu)} \\
I_u^* &= \frac{\alpha_u E_u}{(\rho_u + r_u + \mu + \delta_u)} \\
I_v^* &= \frac{\alpha_v E_v}{(\rho_v + r_v + \mu + \delta_v)} \\
R_u^* &= \frac{\rho_u I_u + \psi_u E_u}{(\omega_u + \mu)} \\
R_v^* &= \frac{\rho_v I_v + \psi_v E_v}{(\omega_v + \mu)}
\end{aligned} \tag{3.16}$$

It is important to note that there is no trivial equilibrium points as long as the natural birth rate of individuals under 10 years and individuals over 10 years Λ_u and Λ_v respectively are not zero. this means that

$$(S^*u, S^*v, E^*u, E^*v, I^*u, I^*v, R^*u, R^*v) \neq (0, 0, 0, 0, 0, 0, 0, 0,)$$

3.4. Disease Free Equilibrium

At the disease-free equilibrium point all the disease compartments are set to be zero, that is, $E_u = E_v = I_u = I_v = R_u = R_v = 0$. We Substitute the above into the model (3.16). Then we have the disease free-equilibrium point 0f the entire model as given as $\epsilon_0 = \left(\frac{\Lambda_u}{\vartheta+\mu}, \frac{\vartheta(\Lambda_v+\Lambda_u)+\Lambda_v\mu}{(\vartheta+\mu)\mu}, 0, 0, 0, 0, 0, 0 \right)$.

3.5. Basic Reproduction Number (R_o) *of the model*

The basic reproduction number is defined as the average number of infections that a single infected individual can cause in a susceptible population during their infectious period. To obtain this,we adapt the next generation matrix method described by Vanden and Watmough (2002).

Let X be the set of the entire disease compartments, that is
$X = (Eu, Ev, Iu, Iv)$. Then our model can be written as

$$\frac{d\mathcal{X}}{dt} = f(x) - v(x) \tag{3.17}$$

where $f(x)$ is the ratio of appearance of new infection into the disease compartments while the $v(x)$ is the rate of transfer of individuals in and out of the disease compartments, it is defined as $v(x) = v^{-}(x) - v^{+}(x)$

$$f(x) = \begin{pmatrix} \frac{b_1\beta uIu}{N} \\ \frac{b_2\beta vIv}{N} \\ 0 \\ 0 \end{pmatrix}, v(x) = \begin{pmatrix} (\psi u + \alpha u + \mu)\ Eu - \omega uRu \\ (\psi v + \alpha v + \mu)\ Ev - \omega vRv \\ (\rho u + ru + \mu + \delta u)Iu - \alpha uEu \\ (\rho v + rv + \mu + \delta v)Iv - \alpha vEv \end{pmatrix}$$

$$F = \begin{pmatrix} 0 & 0 & b_1\beta u & 0 \\ 0 & 0 & 0 & b_2\beta v \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} G_1 & 0 & 0 & 0 \\ 0 & G_2 & 0 & 0 \\ -\alpha u & 0 & G_3 & 0 \\ 0 & -\alpha v & 0 & G_4 \end{pmatrix}$$

$$V^{-}1 = \begin{pmatrix} \frac{1}{G_1} & 0 & 0 & 0 \\ 0 & \frac{1}{G_2} & 0 & 0 \\ \frac{\alpha u}{G_1G_3} & 0 & \frac{1}{G_3} & 0 \\ 0 & \frac{\alpha v}{G_2G_4} & 0 & \frac{1}{G_4} \end{pmatrix}$$

$$V^{-}1.F = \begin{pmatrix} 0 & 0 & \frac{b_1\beta u}{G_1} & 0 \\ 0 & 0 & 0 & \frac{b_2\beta v}{G_2} \\ 0 & 0 & \frac{b_1\alpha u\beta u}{G_1G_3} & 0 \\ 0 & 0 & 0 & \frac{\alpha v b_2\beta v}{G_2G_4} \end{pmatrix}$$

$$F.V^{-}1 = \begin{pmatrix} \frac{b_1\alpha u\beta u}{G_1G_3} & 0 & \frac{b_1\beta u}{G_3} & 0 \\ 0 & \frac{b_2\alpha v\beta v}{G_2G_4} & 0 & \frac{b_2\beta v}{G_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$R_0 = \rho(F.V^{-1})$

$$Ru = \frac{b_1 \alpha u \beta u}{G_1 G_3}, \quad Rv = \frac{b_2 \alpha v \beta u}{G_2 G_4}$$

This implies that

$$\mathcal{R}_0 = \frac{b_1 b_2 \alpha_u \alpha_v \beta_u \beta_v}{G_1 G_2 G_3 G_4}$$

where

$$G_1 = (\alpha_u + \psi_u + \mu), \quad G_2 = (\alpha_v + \psi_v + \mu), \quad G_3 = (\rho_u + r_u + \mu + \delta_u),$$
$$G_4 = (\rho_v + r_v + \mu + \delta_v), \quad G_5 = (\omega_u + \mu), \quad G_6 = (\omega_v + \mu)$$

3.6. Local Stability Analysis of the Disease - Free Equilibrium

The disease-free steady state, ε_0 is locally asymptomatically stable if $R_o < 1$ and unstable if $R_o > 1$.

Proof: The Jacobian matrix of the system evaluated at the disease free equilibrium point.

$$J(E) = \begin{bmatrix} -\vartheta-\mu & 0 & 0 & 0 & ru & 0 & 0 & 0 \\ \vartheta & -\mu & 0 & 0 & 0 & rv & 0 & 0 \\ 0 & 0 & -G_1 & 0 & 0 & 0 & \omega u & 0 \\ 0 & 0 & 0 & -G_2 & 0 & 0 & 0 & \omega v \\ 0 & 0 & \alpha u & 0 & -G_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha v & 0 & -G_4 & 0 & 0 \\ 0 & 0 & \psi u & 0 & \rho u & 0 & -G_5 & 0 \\ 0 & 0 & 0 & \psi v & 0 & \rho v & 0 & -G_6 \end{bmatrix}$$

$$|J - I\lambda| = (-\lambda-\mu)(-\lambda-\mu-\vartheta)\{(\lambda+G_1)(\lambda+G_3)(\lambda+G_5) - (\alpha u \rho u u + (\lambda+G_3)\phi)\omega u\}$$
$$\{(\lambda+G_2)(\lambda+G_4)(\lambda+G_6) - (\alpha v \rho v + (\lambda+G_4)\phi v)\omega v)\} = 0 \quad (3.20)$$

Applying the Routh-Hurwitz criterion, the equation (3.21) will have roots with negative real. Since all the real part of eigen-values are negative, the DFE is locally asymptotically stable if $R_o < 1$.

3.7. Global Asymptotic Stability of the Model

To investigate the global asymptotic stability of the disease free equilibrium of the modeled **??** we use the technique implemented by Castillo-Chavez and Song as described in [32].

To do this, we write the equations in the uninfected class as $\frac{dX}{dt} = F(X, Z)$ and the infected

class as $\frac{dZ}{dt} = G(X, Z)$ where,$X = (Su, Sv, Ru, Rv) \in R^4_+$, denotes the uninfected population.
$Z = (Eu, Ev, Iu, Iv) \in R^4_+$ denotes the infected population and $X_0 = (X^*, 0)$ represents the disease-free equilibrium point of the model (2.1)
Model (2.1) is GAS if it satisfies the following conditions:

1. H_1: $\frac{dX}{dt} = F(X^*, 0), X^*$ is globally asymptotically stable.
2. H_2: $\frac{dZ}{dt} = D_Z G(X^*, 0)Z - \bar{G}(X, Z)$,

   $\bar{G}(X, Z) \geqslant 0$ for all $(X, Z) \in D$

   where $D_Z G(X^*, 0)Z$ represents an M-matrix (characterized by non-negative diagonal elements, also serving as the Jacobian of $G(X, Z)$ and computed for $(X^*, 0)$, the ensuing theorem is applicable, provided the system meets the aforementioned criterion.

Theorem 3.5. *The disease free equilibrium point* $X_O = (X^*, 0)$ *is globally asymptotically stable if* $R_O \leqslant 1$ *and* H_1, H_2 *are satisfied.*

Proof. Let □

$$F(X, Z) = \begin{pmatrix} \Lambda u + ruIu - (\vartheta + \lambda u + \mu)Su \\ \Lambda v + rvIv + \vartheta Su - (\lambda_v + \mu)Sv \\ \rho uIu + \psi uEu - G_5 R_u \\ \rho vIv + \psi vEv - G_6 Rv \end{pmatrix}, G(X, Z) = \begin{pmatrix} \lambda uSu + \omega uRu - G_1 Eu \\ \lambda vSv + \omega vRv - G_2 Ev \\ \alpha uEu - G_3 Iu \\ \alpha vEv - G_4 Iv \end{pmatrix}$$

$$D_Z G(X^*, 0)Z = \begin{pmatrix} b_1 \beta uIu + \omega uRu - G_1 Eu \\ b_2 \beta vIv + \omega vRv - G_2 Ev \\ \alpha uEu - G_3 Iu \\ \alpha vEv - G_4 Iv \end{pmatrix}$$

At the disease free equilibrium point,

$$\begin{aligned} H_1 : \frac{dSu}{dt} &= \Lambda u - \vartheta Su - \mu hSu \\ \frac{dSv}{dt} &= \Lambda v - \vartheta Sv - \mu hSv \\ \frac{dRu}{dt} &= 0 \\ \frac{dRv}{dt} &= 0 \end{aligned} \tag{3.21}$$

$$H_2\!: \bar{G}(X, Z) = D_Z G(X^*, 0)Z - \bar{G}(X, Z),$$

$$\bar{G}(X,Z) = \begin{pmatrix} b_1\beta u I u(1-\frac{Su}{N}) \\ b_2\beta v I v(1-\frac{Sv}{N}) \\ 0 \\ 0 \end{pmatrix}$$

Clearly, $1 \geqslant \frac{Su}{N_h}$ and $1 \geqslant \frac{Sv}{N_h}$ this implies that $\bar{G}(X,Z) \geqslant 0$ Therefore, the disease-free equilibrium of the two-group Tuberculosis model is globally asymptotically stable. In the context of global asymptotic stability, we explore the behavior of equilibrium levels when the disease level undergoes a relatively significant change. If, within a brief time interval, the diseased state reverts to its original equilibrium, we classify it as globally stable. Simply means that the disease can be eradicated whenever the basic reproduction rate is below one.

3.8. Global Stability Analysis For Endemic Equilibrium

Theorem 3.6. *The endemic equilibrium (EE) of the model (2.1) is globally asymptotically stable whenever* $R_0 > 1$

Proof. Let the endemic equilibrium of the model (2.1) be denoted by

$$\epsilon^0 = (S^*u, S^*v, E^*u, E^*v, I^*u, I^*v),$$

$$\begin{aligned} \mathcal{L} = C_1 &\left[Su - S^*u - S^*u\ln\left(\frac{Su}{S^*u}\right) + Eu - E^*u - E^*u\ln\left(\frac{Eu}{E^*u}\right)\right] \\ C_2 &\left[Sv - S^*v - S^*v\ln\left(\frac{Sv}{S^*v}\right) + Ev - E^*v - E^*v\ln\left(\frac{Ev}{E^*v}\right)\right] \\ &+ C_3\left[Iu - I^*u - I^*u\ln\left(\frac{Iu}{I^*u}\right)\right] + C_4\left[Iv - I^*v - I^*v\ln\left(\frac{Iv}{I^*v}\right)\right] \end{aligned} \tag{3.22}$$

Differentiating (3.22), we have

$$\begin{aligned} \mathcal{L} = C_1 &\left[\dot{Su} - \frac{S^*u}{Su}\dot{Su} + \dot{Eu} - \frac{E^*u}{Eu}\dot{Eu}\right] + C_2\left[\dot{Sv} - \frac{S^*v}{Sv}\dot{Sv} + \dot{Ev} - \frac{E^*v}{Ev}\dot{Ev}\right] \\ &+ C_3\left[\dot{Iu} - \frac{I^*u}{Iu}\dot{Iu}\right] + C_4\left[\dot{Iv} - \frac{I^*v}{Iv}\dot{Iv}\right] \end{aligned} \tag{3.23}$$

Where upper dot represent the derivatives of the model (2.1) with respect to time t.

$$\begin{aligned} \mathcal{L} = C_1&\left[\left(1 - \frac{S^*u}{Su}\right)\dot{Su} + \left(1 - \frac{E^*u}{Eu}\right)\dot{Eu}\right] + C_2\left[\left(1 - \frac{S_v^*}{Sv}\right)\dot{Sv} + \left(1 - \frac{E^*v}{Ev}\right)\dot{Ev}\right] \\ &+ C_3\left[\left(1 - \frac{I_u^*}{Iu}\right)\dot{I_u}\right] + C_4\left[\left(1 - \frac{I_v^*}{I_v}\right)\dot{I_v}\right] \end{aligned} \tag{3.24}$$

Substituting the appropriate equation of the model (2.1) in (3.25), we have

$$\begin{aligned}\mathcal{L} = C_1\Bigg[&\left(1-\frac{S_u^*}{S_u}\right)\left(\Lambda_u + r_u I_u - \lambda_u S_u - \vartheta S_u - \mu S_u\right) + \left(1-\frac{E_u^*}{E_u}\right)\left(\lambda_u S_u + \omega_u R_u - G_1 E_u\right)\Bigg]\\
+ C_2\Bigg[&\left(1-\frac{S^* v}{S_v}\right)\left(\Lambda_v + r v I_v + \vartheta Su - \lambda v S_v - \mu S_v\right) + \left(1-\frac{E_v^*}{E_v}\right)\left(\lambda_v S_v + \omega_v R_v - G_2 E_v\right)\Bigg]\\
+ C_3\Bigg[&\left(1-\frac{I_u^*}{I_u}\right)\left(\alpha_u E_u - G_3 I_u\right)\Bigg] + C_4\Bigg[\left(1-\frac{I_v^*}{I_v}\right)\left(\alpha_v E_v - G_4 I_v\right)\Bigg]\end{aligned} \tag{3.25}$$

At steady state of (3.25)

$$\Lambda_u = \lambda_u S_u^* + \vartheta S_u^* + \mu S_u^* - r_u I_u^*$$

$$G_1 = \frac{\lambda_u S_u^* + \omega_u R_u^*}{E_u^*}$$

$$\Lambda_v = \lambda_v S_v^* + \mu S_v^* - r_v I_v^* - \vartheta S_u^*$$

$$G_2 = \frac{\lambda_v S_v^* + \omega_v R_v^*}{E_v^*}$$

$$G_3 = \frac{\alpha_u E_u^*}{I_u^*}$$

$$G_4 = \frac{\alpha_v E_v^*}{I_v^*}$$

Substituting the steady state of Λ_u and Λ_v into (3.25), we have

$$\begin{aligned}\mathcal{L} = C_1\Bigg[&\left(1-\frac{S_u^*}{S_u}\right)\left(\lambda_u S_u^* + \vartheta S_u^* + \mu S_u^* - r_u I_u^* + r_u I_u - \lambda_u S_u - \vartheta S_u - \mu S_u\right)\\
&+\left(1-\frac{E_u^*}{E_u}\right)\left(\lambda_u S_u + \omega_u R_u - G_1 E_u\right)\Bigg]\\
+ C_2\Bigg[&\left(1-\frac{S_v^*}{S_v}\right)\left(\lambda_v S_v^* + \mu S_v^* - r_v I_v^* - \vartheta S_u^* + r_v I_v + \vartheta S_v - \lambda_v S_v - \mu S_v\right)\\
&+\left(1-\frac{E_v^*}{E_v}\right)\left(\lambda_v S_v + \omega_v R_v - G_2 E_v\right)\Bigg]\\
+ C_3\Bigg[&\left(1-\frac{I_u^*}{I_u}\right)\left(\alpha_u E_u - G_3 I_u\right)\Bigg] + C_4\Bigg[\left(1-\frac{I_v^*}{I_v}\right)\left(\alpha_v E_v - G_4 I_v\right)\Bigg]\end{aligned} \tag{3.26}$$

Open the bracket, we have

$$\begin{aligned}L = \quad & C_1\Bigg[\lambda_u S_u^* + \vartheta S_u^* + \mu S_u^* - r_u I_u^* + r_u I_u - \lambda_u S_u - \vartheta S_u - \mu S_u - \frac{\lambda_u S_u^{*2}}{S_u} - \frac{\vartheta S_u^{*2}}{S_u} - \\
&\frac{\mu S_u^{*2}}{S_u} + \frac{r_u I_u^* S_u^*}{S_u}\\
&- \frac{r_u I_u S_u^*}{S_u} + \lambda_u S_u^* + \vartheta S_u^* + \mu S_u^* + \lambda_u S_u + \omega_u R_u - G_1 E_u - \frac{\lambda_u S_u E_u^*}{E_u} - \frac{\omega_u R_u E_u^*}{E_u} + G_1 E_u^*\Bigg]\\
& C_2\Bigg[\lambda_v S_v^* + \mu S_v^* - r_v I_v^* - \vartheta S_v^* + r_v I_v + \vartheta S_u - \lambda_v S_v - \mu S_v - \frac{\lambda_v S_v^{*2}}{S_v} - \frac{\mu S_v^{*2}}{S_v} + \frac{r_v I_v^* S_v^*}{S_v} +\end{aligned}$$

$$\frac{\vartheta S_u^* S_v^*}{S_v}$$
$$- \frac{r_v I_v S_v^*}{S_v} - \frac{\vartheta S_u S_v^*}{S_v} + \lambda_v S_v^* + \mu S_v^* + \lambda_v S_v + \omega_v R_v - G_2 E_v - \frac{\lambda_v S_v E_v^*}{E_v} - \frac{\omega_v R_v E_v^*}{E_v} + G_2 E_v^* \Big]$$
$$+ C_3 \left[\alpha_u E_u - G_3 I_u - \frac{\alpha_u E_u I_u^*}{I_u} + G_3 I_u^* \right] + C_4 \left[\alpha_v E_v - G_4 I_v - \frac{\alpha_v E_v I_v^*}{I_v} + G_4 I_v^* \right]$$

Collect all the terms without dot-stars in the infected classes and equate to zero

$$-G_1 EuC_1 + ruIuC_1 + rvIvC_2 - G_2 EvC_2 + \alpha uEuC_3 - G_3 IuC_3 + \alpha vEvC_4 - G_4 IvC_4$$

This implies that

$$\begin{aligned} (-G_1 C_1 + \alpha u C_3) Eu &= 0 \\ (-G_2 C_2 + \alpha v C_4) Ev &= 0 \\ (ruC_1 - G_3 C_3) Iu &= 0 \\ (-ruC_2 - G_4 C_4) Iv &= 0 \end{aligned}$$

Therefore we have

$$-G_1 C_1 + \alpha u C_3 = 0 \tag{3.27}$$
$$-G_2 C_2 + \alpha v C_4 = 0 \tag{3.28}$$
$$ruC_1 - G_3 C_3 = 0 \tag{3.29}$$
$$-ruC_2 - G_4 C_4 = 0 \tag{3.30}$$

Solving equation (3.27) and (3.29) simultaneously, we recall this

$$\begin{aligned} ax + by &= 0 \\ cx + dy &= 0 \end{aligned}$$

Then

$$x = \frac{b}{ad}, \quad y = \frac{-a}{bc}$$

Where

$$a = -G_1, \quad b = \alpha u \quad c = ru, \quad d = -G_3$$

Hence

$$C_1 = \frac{b}{ad}, \quad C_3 = \frac{-a}{bc}$$

This imply that

$$C_1 = \frac{\alpha u}{G_1 G_3}, \quad C_3 = \frac{G_1}{\alpha uru}$$

Again Solving equation (3.28) and (3.30) simultaneously
In Similarly way, we have

$$C_2 = \frac{\alpha v}{G_2G_4}, \quad C_4 = \frac{-G_2}{\alpha v r v}$$

Now substitute the values of C_1, C_2, C_3, and C_4 in equation (3.28),we have

$$\begin{aligned}
\mathcal{L} = &\frac{\alpha_u}{G_1G_3}\Big[\lambda_u S_u^* + \vartheta S_u^* + \mu S_u^* - r_u I_u^* + r_u I_u - \lambda_u S_u - \vartheta S_u - \mu S_u - \frac{\lambda_u S_u^{*\,2}}{S_u} - \frac{\vartheta S_u^{*\,2}}{S_u} - \frac{\mu S_u^{*\,2}}{S_u} + \\
&\frac{r_u I_u^* S_u^*}{S_u} - \frac{r_u I_u S_u^*}{S_u} + \lambda_u S_u^* + \vartheta S_u^* + \mu S_u^* + \lambda_u S_u + \omega_u R_u - G_1 E_u - \frac{\lambda_u S_u E_u^*}{E_u} - \frac{\omega_u R_u E_u^*}{E_u} + G_1 E_u^*\Big] \\
&\frac{\alpha_v}{G_2G_4}\Big[\lambda_v S_v^* + \mu S_v^* - r_v I_v^* - \vartheta S_v^* + r_v I_v + \vartheta S_u - \lambda_v S_v - \mu S_v - \frac{\lambda_v S_v^{*2}}{S_v} - \frac{\mu S_v^{*2}}{S_v} + \frac{r_v I_v^* S_v^*}{S_v} + \frac{\vartheta S_u^* S_v^*}{S_v} \\
&- \frac{r_v I_v S_v^*}{S_v} - \frac{\vartheta S_u S_v^*}{S_v} + \lambda_v S_v^* + \mu S_v^* + \lambda_v S_v + \omega_v R_v - G_2 E_v - \frac{\lambda_v S_v E_v^*}{E_v} - \frac{\omega_v R_v E_v^*}{E_v} + G_2 E_v^*\Big] \\
&+ \frac{G_1}{\alpha_u r_u}\Big[\alpha_u E_u - G_3 I_u - \frac{\alpha_u E_u I_u^*}{I_u} + G_3 I_u^*\Big] - \frac{G_2}{\alpha_v r_v}\Big[\alpha_v E_v - G_4 I_v - \frac{\alpha_v E_v I_v^*}{I_v} + G_4 I_v^*\Big]
\end{aligned} \tag{3.31}$$

From (3.31) we Collect all the terms with dot-stars including all $\mu S^*u, \mu S^*v$ and $\mu Su, \mu Sv$.

$$\begin{aligned}
\mathcal{L} = &\frac{\alpha_u}{G_1G_3}\Big[2\lambda_u S_u^* + 2\vartheta S_u^* - r_u I_u^* - \frac{\lambda_u S_u^{*\,2}}{S_u} - \frac{\vartheta S_u^{*\,2}}{S_u} - \frac{r_u I_u S_u^*}{S_u} - \frac{\lambda_u S_u E_u^*}{E_u} - \frac{\omega_u R_u E_u^*}{E_u} + G_1 E_u^*\Big] \\
&+ \frac{\alpha_u}{G_1G_3}\Big[2\mu S_u^* - \mu S_u - \frac{\mu S_u^{*\,2}}{S_u}\Big] \\
&+ \frac{\alpha_v}{G_2G_4}\Big[2\lambda_v S_v^* - r_v I_v^* - \vartheta S_v^* - \frac{\lambda_v S_v^{*2}}{S_v} + \frac{r_v I_v^* S_v^*}{S_v} + \frac{\vartheta S_u^* S_v^*}{S_v} - \frac{r_v I_v S_v^*}{S_v} - \frac{\vartheta S_u S_v^*}{S_v} \\
&- \frac{\lambda_v S_v E_v^*}{E_v} - \frac{\omega_v R v E_v^*}{E_v} + G_2 E_v^*\Big] + \frac{\alpha_v}{G_2G_4}\Big[2\mu S_v^* - \mu S_v - \frac{\mu S_v^{*2}}{S_v}\Big] \\
&\frac{G_1}{\alpha_u r_u}\Big[G_3 I_u^* - \frac{\alpha_u E_u I_u^*}{I_u}\Big] - \frac{G_2}{\alpha_v r_v}\Big[G_4 I_v^* - \frac{\alpha_v E_v I_v^*}{I_v}\Big]
\end{aligned} \tag{3.32}$$

Hence, Substitute the steady state of G_1, G_2, G_3 and G_4 in (3.32)

$$\begin{aligned}
\mathcal{L} = &\frac{I_u}{(\lambda_u S_u^* + \omega_u R_u^*)}\Big[2\lambda_u S_u^* + 2\vartheta S_u^* - r_u I_u^* - \frac{\lambda_u S_u^{*\,2}}{S_u} - \frac{\vartheta S_u^{*\,2}}{S_u} - \frac{r_u I_u S_u^*}{S_u} - \frac{\lambda_u S_u E_u^*}{E_u} - \frac{\omega_u R_u E_u^*}{E_u} \\
&+ \lambda_u S_u^* + \omega_u R_u^*\Big] + \frac{I_u}{(\lambda_u S_u^* + \omega_u R_u^*)}\Big[2\mu S_u^* - \mu S_u - \frac{\mu S_u^{*\,2}}{S_u}\Big] \\
&+ \frac{I_v}{(\lambda_v S_v^* + \omega_v R_v^*)}\Big[2\lambda_v S_v^* - r_v I_v^* - \vartheta S_v^* - \frac{\lambda_v S_v^{*2}}{S_v} + \frac{r_v I_v^* S_v^*}{S_v} + \frac{\vartheta S_u^* S_v^*}{S_v} - \frac{r_v I_v S_v^*}{S_v} - \frac{\vartheta S_u S_v^*}{S_v} \\
&- \frac{\lambda_v S_v E_v^*}{E_v} - \frac{\omega_v R_v E_v^*}{E_v} + \lambda_v S_v^* + \omega_v R_v^*\Big] + \frac{I_v}{(\lambda_v S_v^* + \omega_v R_v^*)}\Big[2\mu S_v^* - \mu S_v - \frac{\mu S_v^{*2}}{S_v}\Big] \\
&\frac{(\lambda_u S_u^* + \omega_u R_u^*)}{\alpha_u r_u E_u^*}\Big[\alpha_u E_u^* - \frac{\alpha_u E_u I_u^*}{I_u}\Big] - \frac{(\lambda_v S_v^* + \omega_v R_v^*)}{\alpha_v r_v E_v^*}\Big[\alpha_v E_v^* - \frac{\alpha_v E_v I_v^*}{I_v}\Big]
\end{aligned} \tag{3.33}$$

The above can be simplified to.

$$\begin{aligned}\mathcal{L} = &\frac{I_u S_u^*}{(\lambda_u S_u^* + \omega_u R_u^*) S_u}\left(3\lambda_u S_u + 2\vartheta S_u - \frac{r_u I_u^* S_u}{S_u^*} - \lambda_u S_u^* - \vartheta S_u^* - r_u I_u - \frac{\lambda_u E_u^* {S_u^*}^2}{E_u S_u^*}\right.\\ &\left.- \frac{\omega_u R_u E_u^* S_u}{E_u S_u^*} + \frac{\omega_u R_u^* S_u}{s_u^*}\right) + \frac{I_u \mu S_u^*}{(\lambda_u S_u^* + \omega_u R_u^*)}\left(2 - \frac{S_u}{S_u^*} - \frac{S_u^*}{S_u}\right)\\ &+ \frac{I_v S_v^*}{(\lambda_v S_v^* + \omega_v R_v^*) S_v}\left(3\lambda_v S_v + \vartheta S_v - \frac{r_v I_v^* S_v}{S_v^*} - \lambda_v S_v^* + \vartheta S_u^* + r_v I_v^* - r_v I_v - \vartheta S_u - \frac{\lambda_v E_v^* {S_v^*}^2}{E_v S_v^*}\right.\\ &\left.- \frac{\omega_v R_v E_v^* S_v}{E_v S_v^*} + \frac{\omega_v R_v^* S_v}{s_v^*}\right) + \frac{I_v \mu S_v^*}{(\lambda_v S_v^* + \omega_v R_v^*)}\left(2 - \frac{S_v}{S_v^*} - \frac{S_v^*}{S_v}\right)\\ &\frac{(\lambda_u S_u^* + \omega_u R_u^*)}{r_u}\left(1 - \frac{E_u I_u^*}{I_u E_u^*}\right) - \frac{(\lambda_v S_v^* + \omega_v R_v^*)}{r_v}\left(1 - \frac{E_v I_v^*}{I_v E_v^*}\right)\end{aligned} \tag{3.34}$$

As arithmetic mean is greater that geometric mean, the following inequalities from (3.34) hold:

$$\left(2 - \frac{S_u}{S_u^*} - \frac{S_u^*}{S_u}\right) \leqslant 0, \quad \left(2 - \frac{S_v}{S_v^*} - \frac{S_v^*}{S_v}\right) \leqslant 0,$$

$$\left(3\lambda_u S_u + 2\vartheta S_u - \frac{r_u I_u^* S_u}{S_u^*} - \lambda_u S_u^* - \vartheta S_u^* - r_u I_u - \frac{\lambda_u E_u^* {S_u^*}^2}{E_u S_u^*} - \frac{\omega_u R_u E_u^* S_u}{E_u S_u^*} + \frac{\omega_u R_u^* S_u}{s_u^*}\right) \leqslant 0,$$

$$\left(3\lambda_v S_v + \vartheta S_v - \frac{r_v I_v^* S_v}{S_v^*} - \lambda_v S_v^* + \vartheta S_u^* + r_v I_v^* - r_v I_v - \vartheta S_u - \frac{\lambda_v E_v^* {S_v^*}^2}{E_v S_v^*} - \frac{\omega_v R_v E_v^* S_v}{E_v S_v^*} + \frac{\omega_v R_v^* S_v}{s_v^*}\right) \leqslant 0,$$

$$\left(1 - \frac{E_u I_u^*}{I_u E_u^*}\right) \leqslant 0, \quad \left(1 - \frac{E_v I_v^*}{I_v E_v^*}\right) \leqslant 0$$

Hence, $\dot{\mathcal{L}} \leqslant 0$ for $\bar{R}_0 > 1$. Therefore, $\mathcal{L}$ is a Lyapunov function in D and it concluded that the GAS of EEP is globally asymptotically stable for $\bar{R}_0 > 1$.

3.9. Sensitivity Analysis of Basic Reproduction Number

This study offers an in-depth mathematical analysis of tuberculosis (TB) dynamics in two separate age groups, taking into account factors like reinfection and susceptibility.The aim is to enhance our understanding of the disease's distinct characteristics in children and adolescents, ultimately leading to more effective prevention, and diagnosis. The impact of tuberculosis on children and adolescents is considerable, with millions affected each year globally due to exposure to infectious TB cases and the potential for progression from TB infection to active disease. Our analysis verifies the non-negativity, existence, and uniqueness of the model's solutions, identifying both disease-free and endemic equilibrium states. We assessed the basic reproduction number, R_0, using the next-generation matrix method. The model exhibits local asymptotic stability when R_0 is less than one. By employing the Metzler matrix approach, we established that the disease-free equilibrium is globally asymptotically stable, while a Lyapunov function demonstrated global asymptotic stability of the endemic equilibrium when $R_0 > 1$. Through sensitivity and scenario analyses, supported by Python simulations, we found that the maturity rate of individuals under 10 years shows the highest sensitivity, negatively influencing R_0. This suggests that as maturity increases, susceptibility decreases. Furthermore, factors such as natural death rates and disease-related mortality in this age group also demonstrate an inverse relationship with R_0. In contrast, the average contact rate, probabilities of infection, and progression rates positively correlate with R_0 for both younger and older individuals. Ultimately, improving maturity rates among those under 10 years could significantly reduce R_0, indicating enhanced immunity as children age.

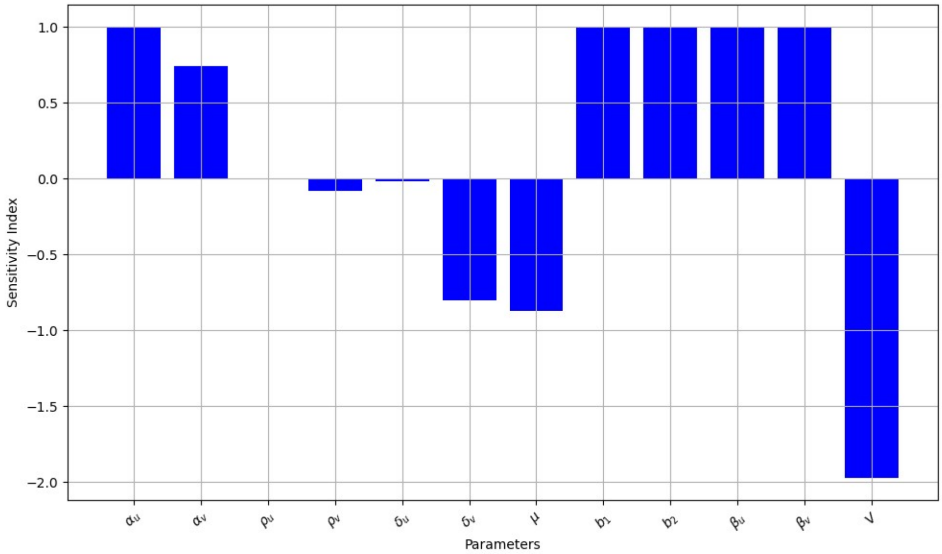


Figure 2: Sensitivity Analysis plot

3.10. Scenario Analysis

Figure 3 displays 3-D plots that demonstrate how different pairs of parameters affect

$$R_0.$$

An increase in b_1 leads to a significant rise in R_0, indicating that higher contact rates among individuals under 10 years old enhance the likelihood of tuberculosis transmission. On the other hand, an increase in α_U, which represents the rate at which exposed individuals under 10 move to the infectious stage, results in a more modest increase in R_0. This underscores the role of quick transitions to infectiousness in heightening the outbreak risk. Moreover, parameters like b_2, β_v, α_v, and β_u also have a positive relationship with R_0, suggesting that higher values of these parameters lead to an increase in R_0. In contrast, the combination of mortality and recovery rates significantly reduces R_0.

4. Numerical Simulation and Results

To obtain the dynamical behaviour of the population, we use the classical fourth-order (RK4) Runge-Kutta method using the initial condition $S_u(0) = 5000$, $S_v(0) = 7000$, $E_u(0) = 900$, $E_v(0) = 4000$, $I_u(0) = 300$, $I_v(0) = 2000$, $R_u(0) = 100$, $R_v(0) = 500$. We chose the time period of 200 days for the simulation with parameter values in table 2. 2.0

Table 2: Parameters Table of Values..

Parameter	**Values (unit)**	**Source**
Λu	4(per person)	Assumed
Λv	5(per day)	[22]
αu	0.005	[35]
αv	0.005	[35]
ρu	0.01(per day)	Assumed
ρv	0.01(per day)	[22]
δu	0.2(per day)	Assumed
δv	0.1(per day)	Assumed
ϑ	3(per day)	Assumed
μ	$\frac{1}{67.6}$(per day)	[22]
b_1	0.1 (per day per person)	Assumed
b_2	0.6501 (per day per person)	[22]
βu	0.8	[26]
βv	0.4	[26]
ru	0.1 (per day)	Assumed
rv	0.1 (per day)	[22]
ωu	0.5	Assumed
ωv	0.79	[12]
ψu	0.01	Assumed
ψv	0.01	[22]

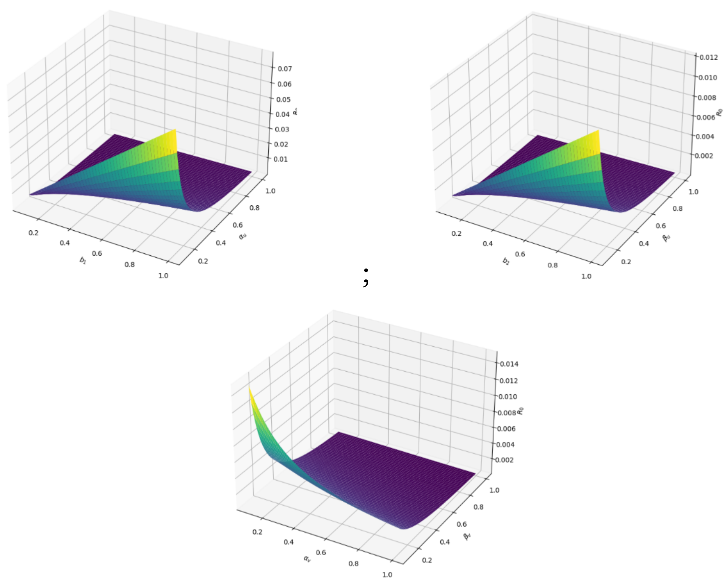


Figure 3: Scenario plots

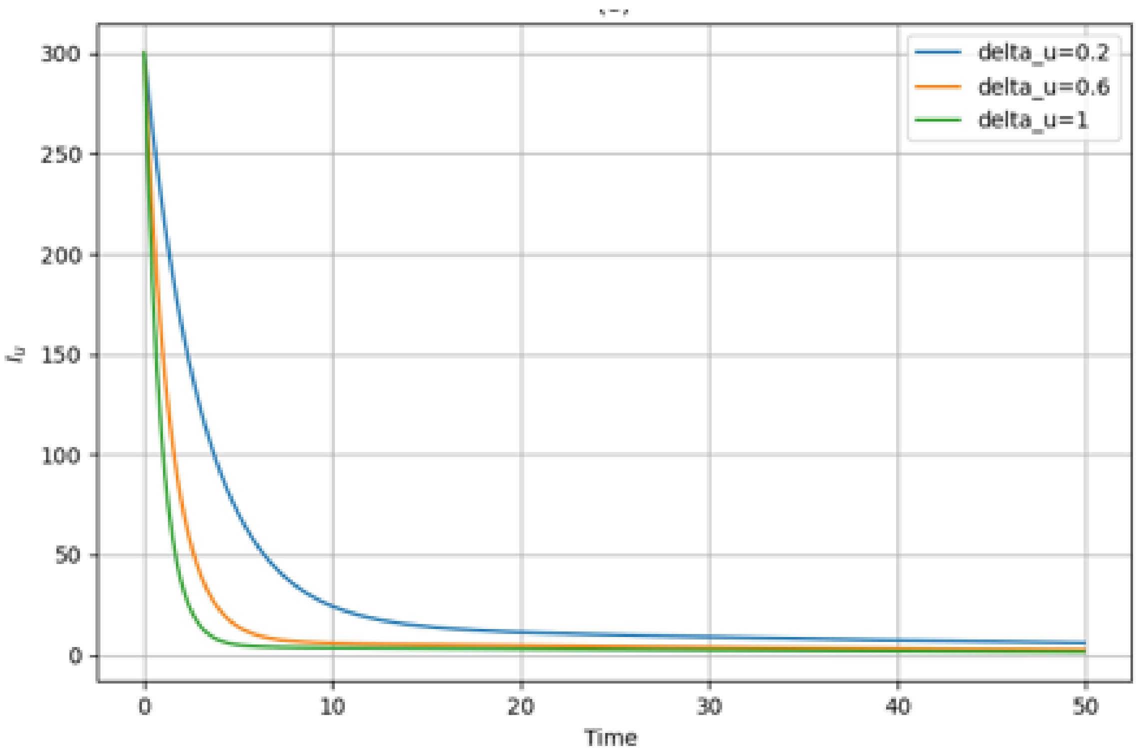
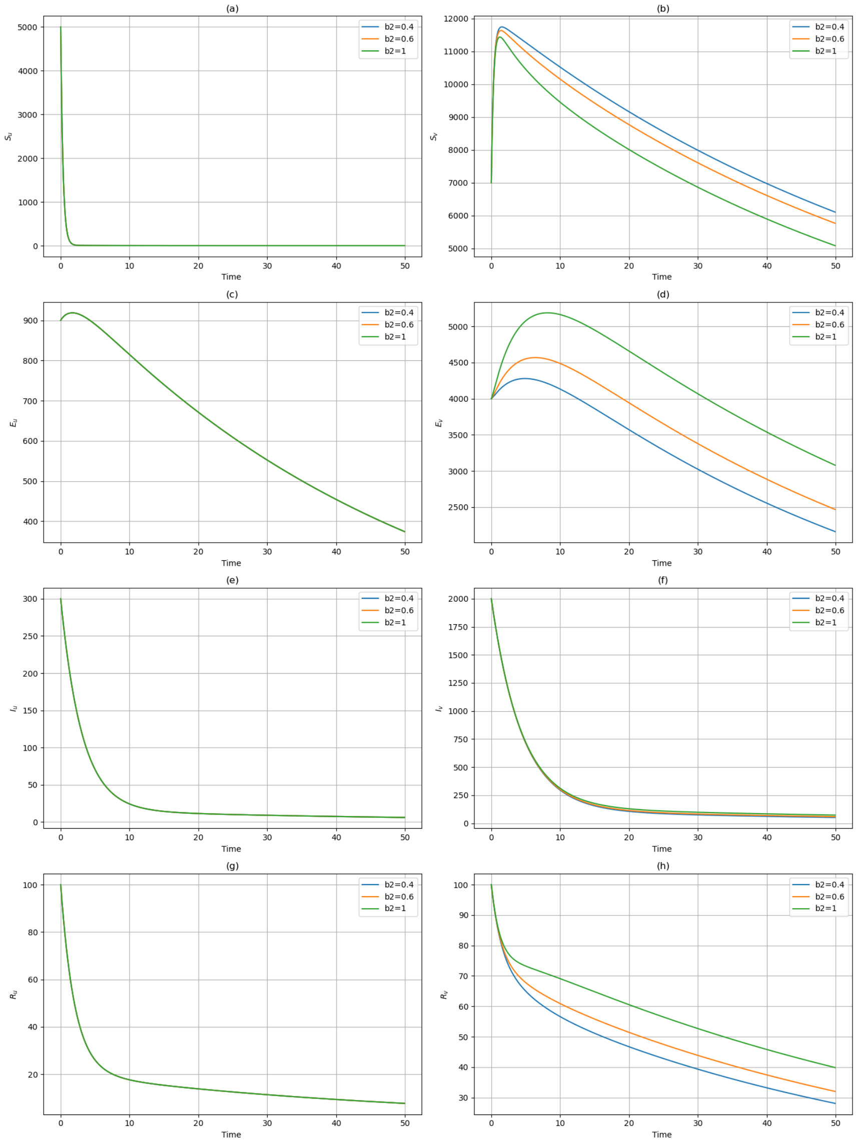
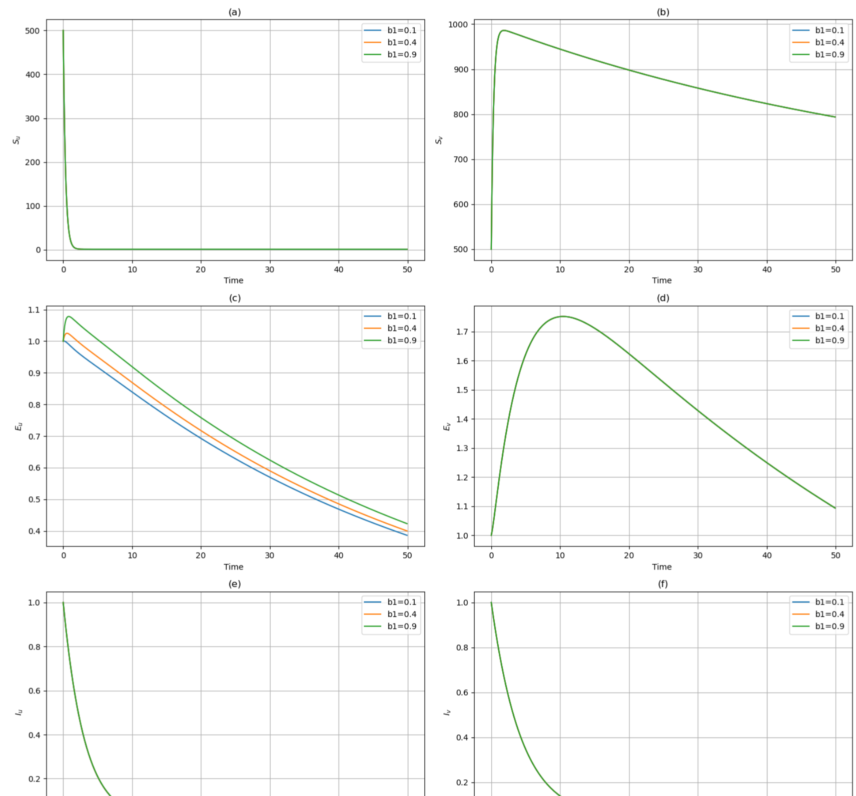


Figure 4: Numerical simulation


(a)
(b)
(c)
(d)
(e)
(f)
(g)
(h)
b2=0.4
b2=0.6
b2=1
Time


(a)
(b)
(c)
(d)
(e)
(f)
b1=0.1
b1=0.4
b1=0.9
Time

4.1. Interpretation of Results

In Figure 4, the analysis elucidates the dynamics of disease progression among children under and over 10 years of age, highlighting significant differences in susceptibility, exposure, and recovery rates. In figure 5 simulations, it duplicate the different simulations For those under 10, all b_2 values show a rapid decline in the susceptible population, suggesting that they are rapidly transitioning to exposed or infected states, reflecting an efficient disease transmission process. The peak in the exposed population and the subsequent decline in the infected population indicate effective recovery pathways despite initial exposure peaks. In contrast, individuals over 10 years show a slower decline in susceptibility, likely due to increased immunity, with increased exposure and infection rates at higher contact rates (b_2). As b_2 increases, the decrease in S_v accelerates. The exposed population for over 10 years initially increases, with more pronounced peaks observed at higher b_2 values, indicating that increased contact rates contribute to a larger number of exposed individuals. The infected population over 10 years follows a similar trend, exhibiting higher values of b_2. Meanwhile, the recovered population over 10 years consistently increases with time, with higher values of b_2. These findings underscore the varying effects of age on disease dynamics and the importance of contact rates in shaping exposure and recovery patterns, particularly highlighting that older individuals benefit from a stabilizing recovery trajectory as the value of b_2 increases.

5. Conclusion

In conclusion, our analysis of the two-group tuberculosis model reveals significant differences in disease dynamics between individuals younger and older than 10 years. By determining the key mathematical properties of our model, including the existence and stability of equilibria, we have gained a thorough understanding of how the disease behaves under various conditions. Sensitivity analyses emphasize the crucial impact of immunity and contact rates on transmission potential, especially among younger populations. These insights are essential for public health strategies aimed at controlling the spread of tuberculosis, indicating that tailored interventions for different age groups could enhance the effectiveness of epidemic management. Our study of the two-group tuberculosis model uncovers important disparities in disease transmission dynamics related to age. By rigorously analyzing the model's mathematical framework, establishing its equilibrium behavior, and assessing its stability, we have enriched our understanding of how tuberculosis transmission varies across age groups.

References

[1] Abbas, M., Kumar, S., & Alshomrani, A. S. (2024). Study of chronic myeloid leukemia with T-cell under fractal-fractional order model. Open Physics, 22(1), 20240032. https://doi.org/10.1515/phys-2024-0032

[2] Abbas, M., Kumar, S.,& Alshomrani, A. S. (2024). Statistical and computational analysis for corruption and poverty model using Caputo-type fractional differential equations. Heliyon, 10(3), e24424. https://doi.org/10.1016/j.heliyon.2024.e24424

[3] Abbas, M., Kumar, S., & Alshomrani, A. S. (2024). Analysis of nonlinear Burgers equation with time fractional Atangana–Baleanu–Caputo derivative. Heliyon, 10(13), e26102. https://doi.org/10.1016/j.heliyon.2024.e26102

[4] Adeyemo, K.M and Adam, U.M and Adeniji, A and Oshinubi, K. (2024).Mathematical Modeling of Two Interacting Populations' Dynamics of Onchocerciasis Disease Spread with Nonlinear Incidence Functions. Mathematics, 12(2), 222. doi:10.3390/math12020222.

[5] Agbaje, J.O., Babasola, O., and Adeyemo, K.M.(2024). Modeling COVID-19 Disease with Deterministic and Data-Driven Models Using Daily Empirical Data in the United Kingdom. Scientific African, 4(2), 289-316. doi:10.3390/covid4020020.

[6] Bandaru, R., Sahoo, D., and Naik, R (2020). Pathogenesis, biology and immunology of tuberculosis. Mediterranean journal of Hematology, 1-25

[7] CDC.(2024).TB Treatment for Children. N. Engl J. Med.

[8] Chen, Q., Yu, S., Rui, J., Guo, Y., Yang, H., Abudurusuli, G., Yang, Z., Liu, H., Luo, L., Wang, M., Lei, Z., Zao, Q., Gavotte. L., Niu, T., Frutos, R., Chen, T (2022).Trasmissibility of tuberculosis among students and non-students: an occupaional-specific mathematical model. Infectious diseases of poverty, 117.

[9] Chiyaka, C., Tchuenche, J.M., Garira, W., and Dube, S (2008). A mathematical analysis of the effects of control strategies on the transmission dynamics of malaria. Applied mathematics and Computation, (195),641-662. publisher:ScienceDirect

[10] Delogu, G., Sali, M., and Fadda, G (2013). The biology of mycobacterium tuberculosis infecttion. Mediterranean journal of Hematology, 5

[11] Egeonu, K. U and Omame, A and Inyama, S.C S (2021). A co-infection model for Two-Strain Malaria and Cholera with Optimal Control. Int J Dyn Cont, doi:10.1007/s40435-020-00748-2.

[12] Horsburgh, C.R and Jo, Y and Nichols, B and Jenkins, H.E and Russell, C.A and White, L.F. (2023).Contribution of Reinfection to Annual Rate of Tuberculosis Infection (ARI) and Incidence of Tuberculosis Disease. Clin Infect Dis Off Publ Infect Dis Soc Am, 76(3),965-972. doi:10.1093/cid/ciac451.

[13] Iboi, E. A., Sharomi, O., Ngonghala, C. N., and Gumel, A. B (2020). Mathematical modeling and analysis of COVID-19 Pandemic in Nigeria. Mathematical Biosciences and Engineering, 10.3934

[14] Jose, M.R., Perez-Butragueno, M., Tesfameriam, A., Reyes, F., Tiziano, G., Endirays, J., Valcha, S., Elala, T., Biru, D., Comeche, B., and Gorgolas, M. (2019). Comparing tuberculosis in children aged under 5 versus 5 to 14 years old in a rural hospital in Southern Ethiopia: an 18-years retrospective cross-sectional study. BMC Public Health, 19(856), springer.

[15] Khan, M.A., and Atangana, A (2020).Modelling the dynamics of novel coronavirus (2019-nCOV) with fractional derivative. Alexandria Eng. J, https://doi.org/10.1016/j.aej

[16] Mandal, S., Sarkar, R. R., and Sinha, S (2011). Mathematical models of malaria ? a review. Malaria journal, 10:202.

[17] Nelson, I.J., and Wells, C.D. (2004). Global epidemiology of childhood tuberculosis. Int. Tuberc lung, (8),636-47.

[18] Nwajeri, U.K., Omame, A., Onyenegha, C.P., and Sinha, S (2021). Mathematical models of malaria ? a review. Results in Physics, 28, https://dx.doi.org/10.1016/j.rinp.2021.104643.

[19] Okolo, P. N and Makama, C. G and Abah, R. T (2023). A mathematical model for tuberculosis transmission dynamics in the presence of testing and therapy, isolation and treatment.FUDMA Journal of Science (FJS), (7),103-116.

[20] Okuonghae, D. (2015).Lyapunov functions and global properties of some tuberculosis models.Journal of Applied Mathematics and Computing, 48(1),421-439. Springer.

[21] Oshinubi, K., Peter, O.J., Addai, E., and other. (2023). Mathematical Modelling of Tuberculosis Outbreak in an East African Country Incorporating Vaccination and Treatment. 11(7),143. doi:10.3390/computation11070143.

[22] Oshinubi, K., Olumuyiwa J. P., Addai, E., Wizerwa, E., Babasola, O., Nwabufo, I.V., Sane, I., Adamm, U.M., Adenizi, A., and Agbaje, J.O (2023). Mathematical modeling of tuberculosis outbreak in a East African country incorporating vaccination and treatment. Computation, 11(143).

[23] Omame, A., Abbas, M., and Onyenegecha, C. P (2022).A fractional order model for the co-interaction of COVID-19 and Hipatitis B virus. Results in Physics, https://doi.org/10.1016/j.rinp.105498.

[24] Omame, A., Abbas, M., and Onyenegecha, C. P (2022).A fractional order model for the co-interaction of COVID-19 and Hipatitis B virus. Results in Physics, https://doi.org/10.1016/j.rinp.105498.

[25] Omame, A., and Okuonghae, D. (2021).A co-infection model for oncogenic Human papillomavirus and tuberculosis with optimal control and cost-effectiveness analysis. Optim Contr Appl Meth, doi:10.1002/oca.2717.

[26] Park, S.Y and Han, S and Kim, Y.M. (2020). Risk of active tuberculosis development in contacts exposed to infectious tuberculosis in congregate settings in Korea. Sci Rep, 10(1), 1306. doi:10.1038/

s41598-020-57697-1.
[27] Perez-Velez, C.M., and Marais, B.J (2012). Tuberculosis in children. N. Engl J. Med, (367),348-61.
[28] Seddon, J., and Shingadia, D (2014). Epidemiology and disease burden of tuberculosis in children: a global perspective. Infect Drung resist, (7),153-65
[29] Seddon, James A., Chiang, Silvia S., Esmail, Hanif., and Coussens, Anna K. (2018). The wonder years: what can primary school children teach us about immunity to Mycobacterium tuberculosis. Frontiers in immunology, 9(2946), Frontiers Media SA.
[30] Snow, Kathryn J., Sismanidis, Charalambos., Denholm, Justin., Sawyer, Susan M., and Graham, Stephen M. (2018).The incidence of tuberculosis among adolescents and young adults: a global estimate. European Respiratory Journal, 51(2), Eur Respiratory Soc.
[31] Snow, Kathryn J., Cruz, Andrea T., Seddon, James A., Ferrand, Rashida A., Chiang, Silvia S., Hughes, Jennifer A., Kampmann, Beate., Graham, Steve M., Dodd, Peter J., Houben, Rein M and others.(2020). Adolescent tuberculosis. The Lancet Child & Adolescent Health, 4(1),68-79. Elsevier.
[32] Stephen, M. Graham., and Jacquie, N. Oliwa. (2023).Diagnosis and management of tuberculosis in children and adolescent: a desk guide for primary Health care worker. Fourth edition, publisher:International Union Against Tuberculosis and Lung Diseases.
[33] Tchoumi, S.Y., Chukwu, C. W., Diagne, M.L., Rwezaura, H., Juga, M.L., and Chuenche, J.M.(2022). Optimal control of a two-group malaria transmission model with vaccination. Network Modeling Analysis in Health Informatics and Bioinformatics, 12(7).
[34] Tsai, K., Chang, H., Chien, S., Chen, K., Chen, K., Mai, M., and Chen, K. (2013). Childhood Tuberculosis, Diagnosis, treatment and vaccination.Pediatrics and Neonatology, (15),295-302.
[35] Usman, T., Aman, M., Khan, O and others. (2020). Construction of partially degenerate Laguerre-Genocchi polynomials with their applications. AIMS Math, (5),4399-4411. doi:10.3934/math.2020280.
[36] WHO (2018). Global Tuberculosis Report. google, doi:WHO/|CDS/TB/2018.20.
[37] WHO.(2024).Global Tuberculosis Report. World Health Organization,
[38] Zhao, Y and Mingtao, L and Yuan, S.(2017). Analysis of transmission and control of tuberculosis in Mainland China, 2015 - 2016, based on age-structure mathematical model. International Journal of Environmental Research and Public Health, 14(1192), doi:10.3390/ijerph14101192.