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Sensitivity Analysis for High and Low Risk Tuberculosis Model

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A B S T R A C T

This study investigates the sensitivity of parameters related to tuberculosis (TB). A proposed model is formulated based on a system of nonlinear ordinary differential equations, and hence a theoretical analysis of the model is conducted. The model is divided into six compartmental classes: susceptible, vaccinated, population which has high risk to become infection by (TB), population which has low risk to become infection by (TB), infectious, recovery individuals.

The Next Generation Matrix (NGM) method is employed to calculate the basic reproduction number, which appeared as a form in several parameters, a sensitivity analysis is conducted to identify the parameters that significantly influence.

1. Introduction

Tuberculosis is an infectious disease caused by the bacterium Mycobacterium tuberculosis (Baqi, 2025). (DAa et al., 2025). TB can be transferred when a person with active TB disease coughs, sneezes, talks, sings, or even, laughs, releasing germs into the air (Olaosebikan & Kolawole, 2024). The spread of TB that using air makes the disease unpredictable and cause new TB cases in some countries (Agbata et al., 2025).

People in close contact with an active TB patient, especially in crowded and poorly ventilated settings, are at higher risk of contracting the infection (Ossaiugbo & Okposo, 2021). A study involving 1,000 patients found that treatment delays averaged 61 days for women and 53 days for men in South Asian countries. Additionally, a significant portion of the population in the region are smokers, which can contribute to the appearance of early symptoms of tuberculosis (TB) (Liu et al., 2025). Approximately 1.7 billion people globally are estimated to carry a latent tuberculosis (TB) infection, placing them at risk of developing and potentially transmitting active TB during their lifetime (Rana & Sharma, 2020).

Latent TB occurs when the body successfully suppresses the bacteria, preventing them from multiplying. In this dormant state, the bacteria remain in the body without causing any symptoms (Rodrigues et al., 2013). It is estimated that around 30% of the global population harbors latent TB, leading to approximately 9 million new active cases each year and 2 million annual deaths, predominantly affecting developing countries (Malek & Hoque, 2024). Those with latent tuberculosis are usually treated on a preventive basis to ensure that the disease does not become active and spread to other areas in the future (Olaosebikan & Kolawole, 2024).

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LTBI can be reactivated by the following conditions: alcoholism, malnutrition, substance abuse, silicosis, renal failure, smoking, steroid therapy, indoor air pollution, and diabetes mellitus. Immunocompromised patients, like HIV infection, and immunosuppressive diseases, such as autoimmune diseases, allergic diseases, and post-organ transplant, have a high risk of developing active tuberculosis disease (Rana & Sharma, 2020). BCG vaccine is given to new born (Chasanah et al., 2019; hasanah et al., 2019). In most cases, it appears as asymptomatic and noninfectious, known as latent TB infection (LTBI), characterized by a lifetime average risk of transformation to active disease estimated at approximately 10% (Diekmann et al., 2010).

The only vaccine currently available is Bacillus Chalmette–Guerin (BCG), though several new vaccines are currently in development, Vaachia & Terna 2022). Even vaccinated persons can become infected with TB at a later stage in life (Chasanah et al. 2019). With a vaccine available and what would appear to be a clear WHO drive to support a coordinated worldwide TB control plan, tuberculosis still is one of the leading causes of mortality resulting from infectious diseases (Baqi 2025). Sensitivity analysis provides information on how variations in model parameters impact the output or results of interest from the model (Olaosebikan & Kolawole, 2024).

Sensitivity analysis tells us how important each parameter is to disease transmission (Rasheed et al., 2024). Researchers can resolve this issue with the help of sensitivity analysis (Peter et al., 2025). The reproductive number R_0 is defined as the expected number of secondary cases that a single infected individual can generate during their infectious period. Olaosebikan and Kolawole (2024) utilized the normalized forward sensitivity index to calculate the sensitivity indices of R_0 following an approach similar to Blower and Dowlatabadi (1994). Ossaiugbo and Okposo (2021) also computed these sensitivity indices for model parameters. A positive sensitivity index indicates that an increase in the parameter will result in a rise in the model's output, whereas a negative value suggests the opposite effect (Olaosebikan & Kolawole, 2024).

The World Health Organization (WHO) has implemented its Global Tuberculosis Eradication Strategy along with the Tuberculosis Action Plan for the European Region (2016-2020) to guide post-2015 efforts. As part of these initiatives, the goal is to achieve a 90% reduction in TB incidence worldwide by 2030 (Yavuz et al., 2023). Addressing TB's trajectory and designing effective control strategies remain critical global health priorities. This underscores the need for sophisticated models that accurately reflect the complexities of TB epidemiology, including factors like latency periods, varying levels of infectiousness, and population diversity (Peter et al., 2024). However, recent advancements in controlling TB face additional challenges due to the emergence and spread of multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB) (Gebre Ergs et al., 2024). These resistant strains have made TB management increasingly difficult.

Traditional six-month treatment regimens are often inadequate, necessitating the development of new therapeutic approaches (Agbata et al., 2025). Moreover, mathematical models that fail to account for bacterial heterogeneity under antibiotic pressure risk oversimplifying key dynamics. This may lead to overlooking critical factors such as competition among strains and varying levels of antibiotic efficacy, which could result in inaccurate predictions regarding the effectiveness of treatment interventions (El-Mesady et al., 2024). To address these complex dynamics, mathematical models have become indispensable tools (Fuller et al., 2024). Considering the persistent global burden of TB, the vulnerability of certain populations, and the limited success of existing control measures in various regions, it is imperative to explore and implement additional preventive strategies (Kozhokaru et al., 2025).

2. Materials and Methods

Taking into consideration that TB exists in two forms latent, which is (low risk), and active, which is (high risk) and also acknowledging that there is only one available vaccine for the disease, as previously mentioned, whose effectiveness decreased over time, it becomes essential to calculate the basic reproduction number (R_0) for both forms of the disease in order to establish a clear and comprehensive understanding of the issue.

In this section, divided deterministic model of TB into six compartments namely:

susceptible $S(t)$, Vaccinated $V(t)$, population which has high risk to become infection by TB E_1, population which has low risk to become infection by TB E_2 ,infection $I(t)$,and recovered $R(t)$,the total population of these six compartments is given $N(t)$.

However, the increase susceptible class $S(t)$ due to births at rate B,recovered class may become susceptible to reinfection over time at rate ξR, and again due to waning efficacy over time at rate θV . The decrease in this class to population high risk at rate $\frac{p\beta SI}{N}$, population low risk $\frac{(1-p)\beta SI}{N}$, vaccination at rate τS, and natural death at rate μS. The vaccinated class $V(t)$ increase due to vaccination from the susceptible class at rate τs.The decrease in this class is natural death at rate μV,pupation high risk at rate $\frac{\omega pSVI}{N}$, and pupation low risk at rate $\frac{\omega(1-p)SVI}{N}$. The pupation high risk becomes infection class E_1, the increase in this due vaccination at rate $\frac{\omega pSVI}{N}$, and susceptible in this class at rate $\frac{p\beta SI}{N}$. The decrease this due to natural death at rate μE_1, progression in this infection $\sigma_1 E_1$, and progression in this class in recovered at rate $\delta_1 E_1$.

The pupation low risk become infection from infection class E_2, the increase in this due vaccination at rate $\frac{\omega(1-p)SVI}{N}$, and susceptible in this class at rate $\frac{(1-p)\beta SI}{N}$. The decrease in this due to natural death at rate μE_2 ,progression in this infection $\sigma_2 E_2$, and progression in this recovered at rate $\delta_2 E_2$.The Infected class $I(t)$, the increase in this due progression to high risk become infection at rate $\sigma_1 E_1$, and in this due progression low risk become infection at rate $\sigma_2 E_2$.The decrease in this due progression to recovered at rate γI , natural death at rate μI,and disease-induced death at rate kI .

The Recovered class $R(t)$, the increase due this in to recovered of infected class at rate γI, progression in this class in recovered at rate $\delta_1 E_1$,and progression in this recovered at rate $\delta_2 E_2$.The decrease recovered class may become susceptible to reinfection over time at rate ξR, natural death at rate μR. These processes can be resented in the following flowchart, Figure 1.

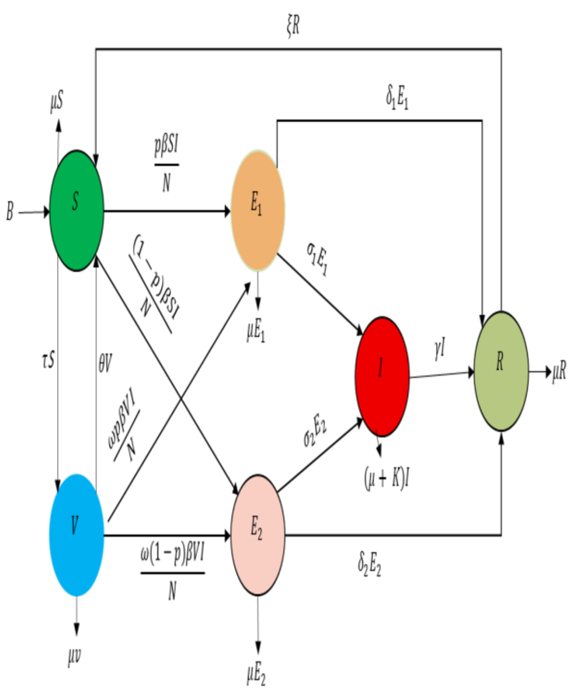

Figure 1. Flowchart for the proposed model

The dynamics depicted in the flowchart can be translated as a system of nonlinear ordinary differential equations:

$$\frac{dS}{dt} = B - \mu S - \frac{(p)\beta SI}{N} - \tau S + \theta V + \xi R$$

$$\frac{dV}{dt} = \tau S - (\mu + \theta)V - \frac{1}{N}\omega\beta VI$$

$$\frac{dE_1}{dt} = \frac{p\beta SI}{N} + \frac{p\omega\beta VI}{N} - (\mu + \sigma_1 + \delta_1)E_1$$

$$\frac{dE_2}{dt} = \frac{(1-p)\beta SI}{N} + \frac{(1-p)\omega\beta VI}{N} - (\mu + \sigma_2 + \delta_2)E_2$$

$$\frac{dI}{dt} = \sigma_1 E_1 + \sigma_2 E_2 - (\gamma + \mu + K)I \quad (1)$$

$$\frac{dR}{dt} = \delta_1 E_1 + \delta_2 E_2 + \gamma I - (\xi + \mu)R$$

The total of pupation is $N(t) = S(t) + V(t) + E_1(t) + E_2 + I(t) + R(t)$

All variables and parameters used in the proposed (TB) transmission model are given in Table 1.

Table 1. Description of variables and parameters

VARIABLES		DEFINTION
$S(t)$		The susceptible
$V(t)$		The Vaccinated
$E_1(t)$		The population which has high risk to become infection by TB
$E_2(t)$		The population which has low risk to become infection by TB
$I(t)$		the infectious
$R(t)$		Recovered
μ		Natural death rate
δ_1		Progression rate from E_1 to$R$
B		Recruitment rate
τ		Rate of Vaccination of Susceptible Individuals
θ		Vaccination wane rate
γ		Transmission rate form I to R
ξ		rate at which recovery individual move to susceptible
β		Transmission rate of infected population
p		Proportion of fast progressors
σ_1		progression rate from E_1 to I
ω		Reduction in the risk of infection due to vaccination
K		Disease induce death
σ_2		progression rate from E_2 to I
δ_2		Progression rata from E_2 to R
N		Total inhabited area

For simplicity we can choose:

$$s=\frac{S}{N} \quad ,v=\frac{V}{N} \quad ,e_1=\frac{E_1}{N} \quad ,e_2=\frac{E_2}{N} \quad ,i=\frac{I}{N} \quad ,r=\frac{R}{N} \quad ,\pi=\frac{B}{N}$$

substituting in system (1) gives

$$\left(\frac{1}{N}\right)\left(\frac{dS}{dt}\right)=\left(\frac{1}{N}\right)\left(B-\mu S-\frac{p\beta SI}{N}-\frac{(1-p)\beta SI}{N}-\tau S+\theta V+\xi R\right)$$

$$\frac{ds}{dt}=\pi-\mu s-\beta si-\tau s+\theta v+\xi r$$

Similarly, for the other variables, we get

$$\frac{dv}{dt}=\tau s-(\mu+\theta)v-\omega\beta vi$$

$$\frac{de_1}{dt}=p\beta si+p\omega\beta vi-(\mu+\sigma_1+\delta_1)e_1 \quad (2)$$

$$\frac{de_2}{dt}=(1-p)\beta si+(1-p)\omega\beta vi-(\mu+\sigma_2+\delta_2)e_2$$

$$\frac{di}{dt}=\sigma_1 e_1+\sigma_2 e_2-(\gamma+\mu+K)i$$

$$\frac{dr}{dt}=\delta_1 e_1+\delta_2 e_2+\gamma i-(\xi+\mu)r$$

with the initial conditions as

$$s(0)\geq 0,\ \ v(0)\geq 0\ \ ,e_1(0)\geq o\ ,e_2(0)\geq 0,\ \ i(0)\geq 0\ \ ,r(0)\geq 0$$

here, also $n=s(t)+v(t)+e_1(t)+e_2(t)+i(t)+r(t)$

1. 2.1 Analysis of the model:

Positivity and boundedness of model solution

Consider the system of equations (2) we have

$$\Omega=\left\{s(t),v(t),e_1(t),e_2(t),i(t),r(t)\in\mathbb{R}_+{}^6 : n\leq\frac{\pi}{\mu}\right\} \qquad (3)$$

The derivative for the total pupation, is given by

$$\frac{dn}{dt}=\frac{ds}{dt}+\frac{dv}{dt}+\frac{de_1}{dt}+\frac{de_2}{dt}+\frac{di}{dt}+\frac{dr}{dt} \qquad (4)$$

$$\frac{dn}{dt}=\pi-\mu(s+v+e_1+e_2+i+r)-ki\leq\pi-\mu n$$

$$\frac{dn}{dt}\leq\pi-\mu n \qquad (5)$$

Solving (5) using Gromwall's inequality gives

$$n(t)\leq n(0)e^{-\mu t}+\frac{\pi}{\mu}[1-e^{-\mu t}] \qquad (*)$$

then at $t\to\infty$, $\ n(t)\leq \max(n(0),\frac{\pi}{\mu})$

Taking limits for both side inequality $(*)$

$$\lim_{t\to\infty} n(t)\leq\lim_{t\to\infty}[n(0)\,e^{-\mu t}+\frac{\pi}{\mu}-\frac{\pi}{\mu}e^{-\mu t}]=\frac{\pi}{\mu}$$

Hencs, $n(t)$ is bounded and represents a physical problem.

Existence and uniqueness of model solution

let

$$f_1=\pi-\mu s-\beta si-\tau s+\theta v+\xi r$$

$$f_2=\tau s-(\mu+\theta)v-\omega\beta vi$$

$$f_3=p\beta si+p\omega\beta vi-(\mu+\sigma_1+\delta_1)e_1 (6)$$

$$f_4=(1-p)\beta si+(1-p)\omega\beta vi-(\mu+\sigma_2+\delta_2)e_2$$

$$f_5=\sigma_1 e_1+\sigma_2 e_2-(\gamma+\mu+K)i$$

$$f_6=e_1+\delta_2 e_2+\gamma i-(\xi+\mu)r$$

Then,

$$\left|\frac{\partial f_1}{\partial s}\right|=\mu+\beta i+\tau<\infty\,,\left|\frac{\partial f_1}{\partial v}\right|=\theta<\infty,\left|\frac{\partial f_1}{\partial e_1}\right|=0<\infty$$

$$\left|\frac{\partial f_1}{\partial e_2}\right|=0<\infty,\left|\frac{\partial f_1}{\partial i}\right|=\beta s<\infty,\left|\frac{\partial f_1}{\partial r}\right|=\ \xi<\infty$$

$$\left|\frac{\partial f_2}{\partial s}\right|=\tau<\infty\,,\left|\frac{\partial f_2}{\partial v}\right|=\mu+\theta+\omega\beta i<\infty,\left|\frac{\partial f_2}{\partial e_1}\right|=0<\infty$$

$$\left|\frac{\partial f_2}{\partial e_2}\right|=0<\infty,\left|\frac{\partial f_2}{\partial i}\right|=\omega\beta v<\infty,\left|\frac{\partial f_2}{\partial r}\right|=0<\infty$$

$$\left|\frac{\partial f_3}{\partial s}\right|=p\beta i<\infty\,,\left|\frac{\partial f_3}{\partial v}\right|=p\omega\beta<\infty,\left|\frac{\partial f_3}{\partial e_1}\right|=\mu+\sigma_1+\delta_1<\infty$$

$$\left|\frac{\partial f_3}{\partial e_2}\right|=0<\infty,\left|\frac{\partial f_3}{\partial i}\right|=p\beta(s+\omega v)<\infty,\left|\frac{\partial f_3}{\partial r}\right|=0<\infty$$

$$\left|\frac{\partial f_4}{\partial s}\right|=(1-p)\beta i<\infty\,,\left|\frac{\partial f_4}{\partial v}\right|=(1-p)\beta\omega i<\infty,\left|\frac{\partial f_4}{\partial e_1}\right|=0<\infty$$

$$\left|\frac{\partial f_4}{\partial e_2}\right| = \mu + \sigma_2 + \delta_1 < \infty, \left|\frac{\partial f_4}{\partial i}\right| = (1-p)\beta(s + \omega v) < \infty, \left|\frac{\partial f_4}{\partial r}\right| = 0 < \infty$$

$$\left|\frac{\partial f_5}{\partial s}\right| = 0 < \infty, \left|\frac{\partial f_5}{\partial v}\right| = 0 < \infty, \left|\frac{\partial f_4}{\partial e_1}\right| = \sigma_1 < \infty$$

$$\left|\frac{\partial f_5}{\partial e_2}\right| = \sigma_2 < \infty, \left|\frac{\partial f_5}{\partial i}\right| = \gamma + \mu + k < \infty, \left|\frac{\partial f_5}{\partial r}\right| = 0 < \infty$$

$$\left|\frac{\partial f_6}{\partial s}\right| = 0 < \infty, \left|\frac{\partial f_6}{\partial v}\right| = 0 < \infty, \left|\frac{\partial f_6}{\partial e_1}\right| = \delta_1 < \infty$$

$$\left|\frac{\partial f_6}{\partial e_2}\right| = \delta_2 < \infty, \left|\frac{\partial f_6}{\partial i}\right| = \gamma < \infty, \left|\frac{\partial f_6}{\partial r}\right| = \xi + \mu < \infty$$

The boundedness of the solution implies that the mathematical model is well-posed within the space, satisfying the criteria of existence, uniqueness, and continuous dependence on initial data.

The existence of disease-free equilibrium point (DFE)

To get the equilibrium points of the system (2), set system equal to zero:

$$\frac{ds}{dt} = \frac{dv}{dt} = \frac{de_1}{dt} = \frac{de_2}{dt} = \frac{di}{dt} = \frac{dr}{dt} = 0$$

Then the (DFE) point defined by

$$(DFE) = (s_0, v_0, e_{10}, e_{20}, i_0, r_0)$$

Where,

$$\dot{s_0} = \frac{\pi}{(\tau + \mu)} = \frac{B}{N(\tau + \mu)}$$

$$\frac{dv}{dt} = \dot{v_0} = \tau s_0 = \frac{\tau B}{N(\tau + \mu)}$$

Then $(DFE) = (\frac{B}{N(\tau+\mu)}, \frac{\tau B}{N(\tau+\mu)}, 0,0,0,0)$

2.3 Computation of Basic Reproduction Number R_0:

The basic reproduction number, R_0, is considered one of the most critical metrics in infectious disease epidemiology (DAa et al., 2025) [5]. The Next Generation Matrix Method (NGM) is a commonly employed approach to calculate R_0(Diekmann et al., 1990 [6]), particularly effective for large systems. This number represents the average number of secondary infections generated by an infected individual within a population, as determined through this method, $R_0 = -FM^{-1}$ as explained as follows

Using Next Generation Matrix Method

The infection sub system of equation (2)

$$\frac{de_1}{dt} = p\beta si + p\omega\beta vi - (\mu + \sigma_1 + \delta_1)e_1$$

$$\frac{de_2}{dt} = (1-p)\beta si + (1-p)\omega\beta vi - (\mu + \sigma_2 + \delta_2)e_2$$

$$\frac{di}{dt} = \sigma_1 e_1 + \sigma_2 e_2 - (\gamma + \mu + K)i$$

which can be composite into:

$$\mathcal{F} = \begin{bmatrix} p\beta si + p\omega\beta vi \\ (1-p)\beta si + (1-p)\omega\beta vi \\ 0 \end{bmatrix}$$

$$\mathcal{M} = \begin{bmatrix} & -(\mu + \sigma_1 + \delta_1)e_1 & \\ & -(\mu + \sigma_2 + \delta_2)e_2 & \\ \sigma_1 e_1 & \sigma_2 e_2 & -(\gamma + \mu + K)i \end{bmatrix}$$

The Jacobians of these two matrices

$$F = \begin{bmatrix} 0 & 0 & p\beta s + p\beta\omega v \\ 0 & 0 & (1-p)\beta s + (1-p)\beta\omega v \\ 0 & 0 & 0 \end{bmatrix}$$

$$M = \begin{bmatrix} -(\mu + \sigma_1 + \delta_1) & 0 & 0 \\ 0 & -(\mu + \sigma_2 + \delta_2) & 0 \\ \sigma_1 & \sigma_2 & -(\mu + k + \gamma) \end{bmatrix}$$

Which at free equilibrium point are and henes

$$M^{-1} = -\begin{bmatrix} \frac{1}{(\mu+\sigma_1+\delta_1)} & 0 & 0 \\ 0 & \frac{1}{(\mu+\sigma_2+\delta_2)} & 0 \\ \frac{\sigma_1}{(\mu+\sigma_1+\delta_1)(\mu+k+\gamma)} & \frac{\sigma_2}{(\mu+\sigma_2+\delta_2)(\mu+k+\gamma)} & \frac{1}{(\mu+k+\gamma)} \end{bmatrix}$$

Since the matrix F it contains two zero columns, the spectral radius of

$$K = -E^T F M^{-1} E$$

$$R_0 = p(K)$$

$$= \frac{1}{2}(trace\ (K) + \sqrt{trace(K)^2 - 4del(K)}$$

Since $del(K) = 0$

Then

$$R_0 = trace\ (K)$$

$$R_0 = (\frac{p\sigma_1}{(\mu+\sigma_1+\delta_1)} + \frac{(1-p)\sigma_2}{(\mu+\sigma_2+\delta_2)}) \times \frac{\beta(s+\omega v)}{(\mu+k+\gamma)}$$

It is clear that the formula of R_0 contains a lot of parameters. The prevailing idea was that if R_0 is less than one ($R_0 < 1$), the disease would fade away and could be overcome. However, it has recently become apparent that the disease can persist even when $R_0 < 1$. This urges us to study the sensitivity of R_0 to each variable and parameter included in R_0 formula.

2.4 Sensitivity analysis of model parameters:

Through sensitivity analysis of the basic reproduction number R_0 parameters, the normalized forward sensitivity index of the basic reproduction number R_0 that depends differentiability index on a parameter u, is defined as a formal [15]

$$\Gamma_u{}^{R_0} = \frac{\partial R_0}{\partial u} \times \frac{u}{R_0}$$

Where,

$$R_0 = (\frac{p\sigma_1}{(\mu+\sigma_1+\delta_1)} + \frac{(1-p)\sigma_2}{(\mu+\sigma_2+\delta_2)}) \times \frac{\beta(s+\omega v)}{(\mu+k+\gamma)}$$

$$s_0 = \frac{\pi}{(\tau+\mu)} \quad , v_0 = \frac{\tau\pi}{(\tau+\mu)}$$

Subsisting $s_0, v_0\ in\ R_0$

$$R_0 = (\frac{p\sigma_1}{(\mu+\sigma_1+\delta_1)} + \frac{(1-p)\sigma_2}{(\mu+\sigma_2+\delta_2)}) \times \frac{\beta\pi(1+\omega\tau)}{(\tau+\mu)(\mu+k+\gamma)}$$

Then we have

$$\Gamma_\beta{}^{R_0} = \left(\frac{p\sigma_1}{(\mu+\sigma_1+\delta_1)} + \frac{(1-p)\sigma_2}{(\mu+\sigma_2+\delta_2)}\right) \times \frac{\pi(1+\omega\tau)}{(\tau+\mu)(\mu+k+\gamma)}$$

$$\times \left(\frac{\beta(\tau+\mu)(\mu+k+\gamma)}{\beta\pi(1+\omega\tau)}\right) \times (\frac{p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)}{(\mu+\sigma_1+\delta_1)(\mu+\sigma_2+\delta_2)}$$

$$\left(\frac{\beta(\mu+k+\gamma)}{\beta\pi(1+\omega\tau)}\right) \times (\frac{(\mu+\sigma_1+\delta_1)(\mu+\sigma_2+\delta_{2)}}{p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)})$$

and henes,

$$\Gamma_\beta{}^{R_0} = 1 > 0$$

Similarly

$$\Gamma_\pi{}^{R_0} = 1 > 0$$

$$\Gamma_k{}^{R_0} = \frac{-k}{(\mu+k+\gamma)} < 0$$

$$\Gamma_\omega{}^{R_0} = \frac{\tau\omega}{(1+\omega\tau)} > 0$$

$$\Gamma_\gamma{}^{R_0} = \frac{-\gamma}{(\mu+k+\gamma)} < 0$$

$$\Gamma_\tau{}^{R_0} = \frac{\tau(\omega\mu-1(}{(\mu+\tau)(\omega+\tau)} < 0 \text{ where } \omega\mu < 1$$

$$\Gamma_\mu{}^{R_0} = -\left[\left(\frac{p\sigma_1}{(\mu+\sigma_1+\delta_1)^2} + \frac{(1-p)\sigma_2}{(\mu+\sigma_2+\delta_2)^2}\right) \times \frac{(\mu+\sigma_1+\delta_1)(\mu+\sigma_2+\delta_2)}{p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)} + (\frac{1}{(\mu+\tau)} + \frac{1}{(\mu+k+\gamma)})\right]\mu < 0$$

$$\Gamma_{\sigma_1}{}^{R_0} = \frac{(\mu+\sigma_2+\delta_2)[p(\mu+\sigma_1+\delta_1)+p\sigma_1]\sigma_1}{(\mu+\sigma_1+\delta_1)[p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)]} > 0$$

$$\Gamma_{\sigma_2}{}^{R_0} = \frac{(\mu+\sigma_1+\delta_1)[(1-p)(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2]\sigma_2}{(\mu+\sigma_2+\delta_2)[p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)]} > 0$$

$$\Gamma_{\delta_1}{}^{R_0} = \frac{-p\sigma_1\delta_1(\mu+\sigma_2+\delta_2)}{(\mu+\sigma_1+\delta_1)[p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)]} < 0$$

$$\Gamma_{\delta_2}^{\ R_0} = \frac{-(1-p)\sigma_2\delta_2(\mu+\sigma_1+\delta_1)}{(\mu+\sigma_2+\delta_2)[p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)]} < 0$$

$$\Gamma_p^{\ R_0} = \frac{[\sigma_1(\mu+\sigma_2+\delta_2)-\sigma_2(\mu+\sigma_1+\delta_1)]p}{p\sigma_1(\mu+\sigma_2+\delta_2)+(1-p)\sigma_2(\mu+\sigma_1+\delta_1)} > 0$$

where $\sigma_1(\mu+\sigma_2+\delta_2) < \sigma_2(\mu+\sigma_1+\delta_1)$

3. Results

Sensitivity indices are obtained by substituting values for each parameter from Table 2.

Sensitivity analysis is a key tool used to understand how changes in input parameters affect the outcomes of a mathematical model. By examining how variations in different parameters influence model results, researchers can identify which factors have the greatest impact. This insight helps direct research and practical efforts toward the parameters that play the most critical role in shaping model behavior. The next figure 1 shows sensitivity index for parameter values contained in table 2. Graphically.

Sensitivity analysis reveals the key factors influencing TB spread. The most impactful parameters include the transmission rate, progression rate, recovery rate. it can be stated that transmission rate of infection population rate, recruitment rate influencing the epidemiological stability of the disease.

Table 2. Sensitivity index for Parameter Values the model

Parameter	Value	Source	Sensitivity index for parameter values
$\boldsymbol{\beta}$	0.00000000655	3	1
$\boldsymbol{\mu}$	0.0204	14	-0.194
$\boldsymbol{\gamma}$	2.50	14	-0.9349
$\boldsymbol{K}$	0.15	14	-0.00561
$\boldsymbol{\tau}$	0.1	14	-0.8203
$\boldsymbol{\omega}$	0.1	14	0.00099
$\boldsymbol{\pi}$	0.005	Estimated	1
$\boldsymbol{p}$	0.14	3	0.01
$\boldsymbol{\delta_1}$	2	Estimated	-0.1384
$\boldsymbol{\delta_2}$	1	Estimated	-0.8335
$\boldsymbol{\sigma_1}$	0.02	Estimated	0.1426
$\boldsymbol{\sigma_2}$	0.01	Estimated	0.8672

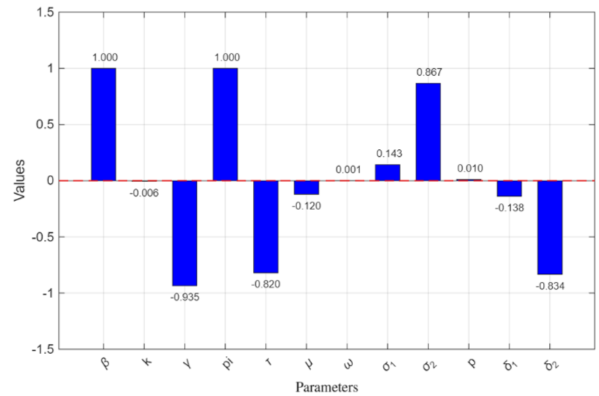

Figure 2. Sensitivity index for Parameter Valus in the model

Numerical Representation: Despite our continued efforts in analyzing the system, we were unable to reach a consistent result or a clear analytical solution. We faced multiple challenges that complicated the results, making it difficult to determine the system's behavior definitively. We therefore intend to resort to numerical methods as an alternative means to explore the system dynamics more precisely. By using numerical solutions, via MATLAB tools, we hope to gain deeper insights into the stability of the system and its behavior of its compartments under different conditions, which may help understand complex phenomena. Figures 3 and 4.

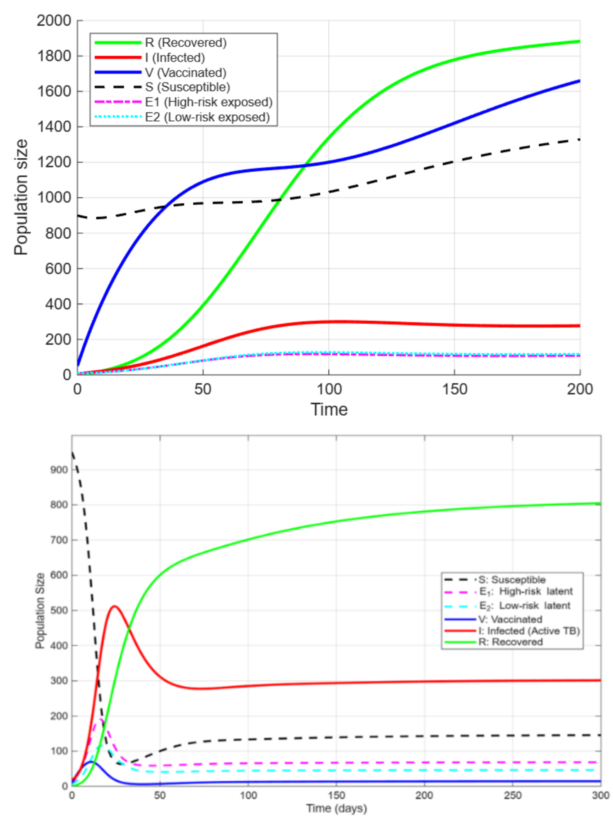

Figure 3. Model dynamics illustrating the global stability

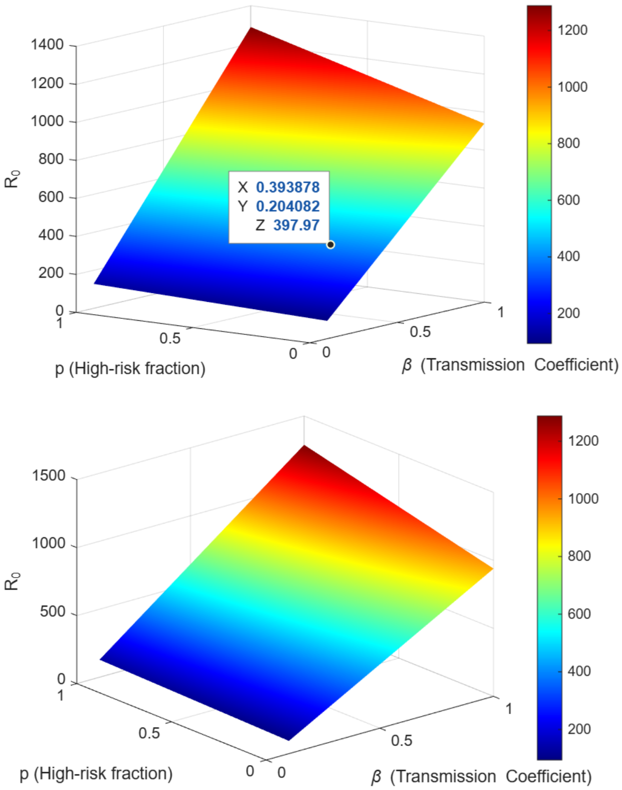

Figure 4. Results graphs of R_0 according to the parameters of model (2)

4. Discussion

The field of mathematical biology has received considerable attention, in-depth research, and continuous development due to its direct connection to people's lives. Starting from simple and straightforward models involving only two differential equations that describe the interaction between two species, such as predator and prey, it has evolved into models that ultimately simulate the latest diseases and pandemics that have swept humanity. This led the systems to include a large number of differential equations, potentially reaching up to ten equations. Consequently, mathematical analysis of these models using simple methods based on linearity and other known techniques became unavailing. Effective methods emerged through calculating basic reproduction number ((R_0), using the Next Generation Matrix method (NGMM), which we have used.

In this study, a mathematical model has been proposed simulating TB epidemic, and hence studied analytically and numerically to find R_0, which is a standard of liner disease or its decay, that is if $R_0$is less than one (R_0<1), the disease would fade away and could be overcome.

All the parameters included in (R_0formula have been examined in order to know impact each of them on R, where they are varying effect. Our goal is to advise the competent authorities for predicting, and controlling the transmission dynamics of this infectious diseases

5. Conclusions

In this study, a tuberculosis (TB) model was analytically studied. A statement of R_0 concluded using next generation matrix method (NGMM), this statement appeared as a form in several parameters, which required sensitivity analysis of the values included

The sensitivity analysis of the tuberculosis transmission model identified several key factors affecting the model's outcomes. R_0 Sensitivity was clarified for all parameters contained therein, for example, the transmission rate between individuals demonstrated a

positive effect β with a value of 1, indicating that increased levels of interpersonal contact are strongly associated with a substantial rise in disease spread, and π with value 1 represent ${}^{B}/_{N}$.

This is followed by the progression rate σ_1, σ_2 from the incubation period to the onset of symptoms with values 0.1426 and 0.8672, respectively, and subsequently the positive value with a weak infect sensitivity p with value 0.01 ω with a value 0.00099.

The negative effect γ with value of -0.934 represents the recovered compartment of the population, Therefore, any increase in the recovery rate leads to a reduction in the basic reproduction numberR_0.

The recovery transform rates δ_1, δ_2 with value -0.1384 and -0.8335, respectively, and subsequently the negative value with a weak infect sensitivity μ with a -0.194 k with a value -0.0056 τ with a value -0.8203

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