Original Research Article

Mathematical Model of Tuberculosis Transmission Dynamics with Vaccination

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ABSTRACT

In Tuberculosis (TB) is an infectious disease caused by bacteria and transmitted through close contact with individuals who have active TB. To better understand its transmission dynamics and control strategies, a deterministic model was developed. The threshold parameter of the model was determined by analyzing its qualitative behavior. The results show that when the effective reproduction number is below one, the tuberculosis-free equilibrium is locally asymptotically stable; otherwise, it becomes unstable. Furthermore, the model's stability and sensitivity were analyzed. Numerical simulations were performed to validate and illustrate the theoretical results. Overall, the study concludes that reducing contact rates among susceptible individuals and enhancing vaccination coverage with highly effective vaccines can significantly decrease the prevalence of tuberculosis within the population.

Keywords: Tuberculosis Model, Basic Reproduction Number, Stability Analysis, Vaccination

INTRODUCTION

Tuberculosis (TB) is an infectious disease caused by bacteria that primarily affect the lungs. The infection spreads through the air when individuals with active TB release bacteria by coughing, sneezing, talking, singing, or even laughing (Bisuta et al., 2018; Brian et al., 2020; Colditz et al., 2019). Only individuals with active pulmonary TB are contagious (Brian et al., 2020). Most people who inhale TB bacteria can resist the infection and prevent bacterial multiplication; in such cases, the bacteria remain dormant, leading to a latent tuberculosis infection (Daniel, 2022).

Although TB is both preventable and curable, it can affect organs beyond the lungs, such as the brain. It is estimated that about 25% of the global population carries TB bacteria. Several drugs have been approved for TB treatment (Dowdy et al., 2021; Egonmwan & Okuonghae, 2022; Fatima et al., 2021; Gomes et al., 2023). Treatment varies depending on whether the infection is latent or active.

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Individuals with latent TB usually undergo preventive therapy to stop the infection from progressing and spreading (Intan et al., 2022). Active TB, on the other hand, often requires a combination of four medications for effective treatment.

Mathematical modeling of infectious diseases serves as a valuable tool for analyzing disease dynamics, predicting epidemic trends, and evaluating intervention strategies (Kaufmann et al., 2024). Such models provide insights into transmission patterns and help identify key factors that can reduce disease

prevalence. Numerous mathematical models describing the transmission dynamics of TB have been developed and analyzed by various researchers (Khajanchi et al., 2023; Pai et al., 2020). For instance, Latifat et al. (2020) formulated a model to examine the role of partial treatment in TB transmission, while Michael et al. (2020), Mojisola et al. (2020), Oguntolu et al. (2023), and Ojo et al. (2022) investigated TB dynamics considering both hospitalized and non-hospitalized infectious individuals. Similarly, Olanrewaju et al. (2022), Pai et al. (2020), Awoke and Kassa (2021), and Ullah et al. (2022) proposed models that

incorporate partial treatment, latent infections, and vaccination of susceptible individuals. Other researchers have explored TB dynamics under various epidemiological contexts (Wazwaz, 2022; WHO, 2018; WHO, 2020; Yang et al., 2021), and further studies on TB modeling can be found in Zhang et al. (2022) and Zumla et al. (2023).

In this study, we propose a new deterministic model to examine the impact of certain control strategies on TB transmission. The findings of this research can serve as a reference for policymakers and public health organizations in developing effective TB control measures. The paper is organized as follows: Section 2 presents the model formulation; Section 3 provides the model analysis; Sections 4 and 5 discuss the numerical simulations and results, respectively; and Section 6 concludes the study.

FORMULATION OF THE MODEL

Study Area Description

In this section, a deterministic model for tuberculosis (TB) transmission is formulated, consisting of five population compartments: susceptible (S(t)), exposed (E(t)), infected (I(t)), vaccinated (V(t)), and recovered (R(t)). Individuals enter the susceptible group through recruitment at a constant rate (B). Susceptible individuals receive vaccination at a specified rate and may lose their vaccine-induced immunity at rate (θ). The force of infection is expressed by (β), while the progression from the exposed to the infectious stage occurs through a saturated incidence term denoted by (α). The recovery rate is given by (γ), and recovered individuals may lose immunity and return to the susceptible compartment at rate (ξ). In addition, individuals may move directly from the exposed to the recovered class at rate (δ).

All population groups experience natural mortality at rate (μ), and those infected face an additional disease-induced death rate (k). The parameter (ω) represents the reduction in infection risk due to vaccination.

The complete set of these interactions is expressed through a system of nonlinear differential equations (Equation 1), and their dynamical flow is illustrated in Figure 1.

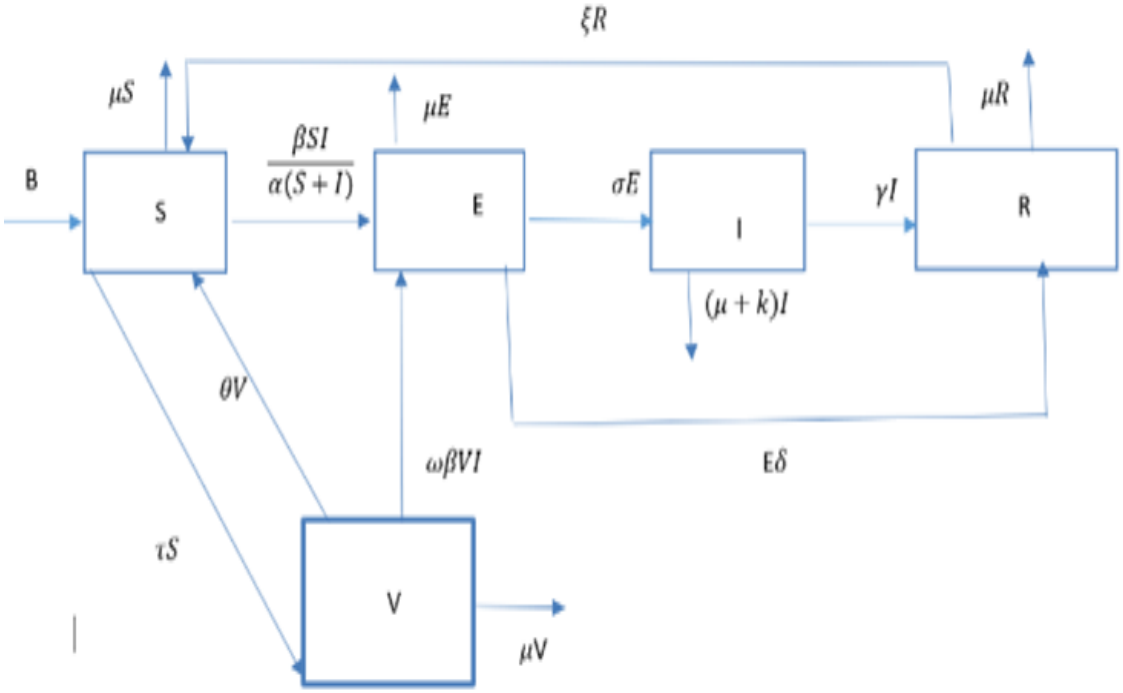

Figure 1. Schematic diagram of the model

$$\left.\begin{aligned}
\frac{dS}{dt} &= B - \frac{\beta SI}{\alpha(S+I)} - \mu S + \xi R + \theta V - \tau S \\
\frac{dV}{dt} &= \tau S - (\omega\beta I + \theta + \mu)V \\
\frac{dE}{dt} &= \frac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta + \mu + \sigma)E \\
\frac{dI}{dt} &= \sigma E - (\gamma + \mu + k)I \\
\frac{dR}{dt} &= \gamma I + \delta E - (\xi + \mu)R
\end{aligned}\right\}$$

Subject to the following initial conditions

$$S(0) = s_0, \; V(0) = v_o, E(0) = e_0, I(0) = i_0, R(0) = r_0 \geq 0. \qquad (2)$$

Table I: Description of parameter

Variables	Definition
$\boldsymbol{S(t)}$	The number of Susceptible individuals at time t
V(t)	The number of Vaccinated individuals at time t
$\boldsymbol{E(t)}$	The number of Exposed individuals at time t
$\boldsymbol{I(t)}$	The number of Infected individuals at time t
$\boldsymbol{R(t)}$	The number of Recovered individuals at time t
Parameter	**Definition**
σ	Progression rate from Latent to infected
$\boldsymbol{k}$	Disease induce death
α	Saturated term
ω	Reduction in the risk of infection due to vaccination
τ	Rate of Vaccination of Susceptible Individuals
θ	Vaccination wane rate
B	Recruitment rate
β	Contact rate
γ	Transmission rate from infected to recovery
μ	Natural death rate
δ	Transmission rate from expose to recovery
ξ	Rate at which recovery individual move to susceptible

Table 2: Values of the model's parameter and references

Parameters	Value	References
$\boldsymbol{B}$	5	(Khajanchi et al., 2023)
β	0.00000000655000,	(Ojo et al., 2023)
σ	0.00050	(Bisuta et al., 2018)
γ	2.50	(Latifat et al., 2020)
δ	1.50-3.5	(Olanrewaju et al.,2022; WHO, 2018)
μ	0.02041	(Gomes et al., 2023)
ξ	1.20	(Intan et al., 2022)

k	0.00050, 0.15	(Dauda et al., 2020)
α	0.03	(Kaufmann et al., 2024)
ω	0.1	(Fatima et al., 2021)
τ	0, 0.1	(WHO, 2020)
θ	0.067	(Pai et al., 2020)

ANALYSIS OF THE MODEL

Positivity and boundedness of model solution

Consider the compartment of the system of equations for case 1 on the population, we have

$$\Omega = \left\{S(t), V(t), E(t), I(t), R(t), \in \Re^5{}_+ : N \leq \frac{B}{\mu}\right\} \qquad (3)$$

The derivatives obtained for the total population at any time t, are given by

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

$$\text{Such that} \quad \frac{dN(t)}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

$$\frac{dN(t)}{dt} = \begin{bmatrix} B - \dfrac{\beta SI}{\alpha(S+I)} - \mu S + \xi R + \theta V - \\ \tau S + \tau S - \omega\beta VI - (\theta + \mu)V + \\ \dfrac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta + \mu + \sigma)E + \\ \sigma E - (\gamma + \mu + k)I + \gamma I + \\ \delta E - (\xi + \mu)R \end{bmatrix}$$

$$\frac{\mathrm{dN(t)}}{\mathrm{dt}} = \mathrm{B} - \mu(\mathrm{S} + \mathrm{V} + \mathrm{E} + \mathrm{I} + \mathrm{R}) - \mathrm{kI} \leq \mathrm{B} - \mu\mathrm{N} \; \frac{\mathrm{dN(t)}}{\mathrm{dt}} \leq \mathrm{B} - \mu\mathrm{N}$$

$$\frac{\mathrm{dN}(t)}{dt} + \mu N \leq B \qquad (5)$$

Solving equation (4) using Gronwall's inequality gives

$$\mathrm{N(t)} \leq \mathrm{N(0)e^{-\mu t}} + \frac{\mathrm{B}}{\mu}[1 - \mathrm{e^{-\mu t}}]$$

This means ast $\rightarrow \infty, N(t) \leq \max\left(N(0), \frac{B}{\mu}\right)$

Taking the initial t and N(t) such that;

$$\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty}\left[\frac{B}{\mu} + \left(N(0) - \frac{B}{\mu}\right)\ell^{-\mu t}\right] = \frac{B}{\mu} \qquad (6)$$

It is said to be bounded from above, hence it represents a physical problem

Existence and uniqueness of model solution

Let:

$$\left.\begin{aligned} f_1 &= B - \frac{\beta SI}{\alpha(S+I)} - \mu S + \xi R + \theta V - \tau S \\ f_2 &= \tau S - \omega\beta VI - (\theta+\mu)V \\ f_3 &= \frac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta+\mu+\sigma)E \\ f_4 &= \sigma E - (\gamma+\mu+k)I \\ f_5 &= \gamma I + \delta E - (\xi+\mu)R \end{aligned}\right\} (7)$$

Then,

$$\left|\frac{df_1}{dS}\right| = \frac{\alpha(\mu+\tau)+\beta}{\alpha} < \infty, \left|\frac{df_1}{dV}\right| = \theta < \infty,$$

$$\left|\frac{df_1}{dE}\right| = 0 < \infty, \left|\frac{df_1}{dI}\right| = \frac{\beta}{\alpha} < \infty, \left|\frac{df_1}{dR}\right| = \xi < \infty$$

$$\left|\frac{df_2}{dS}\right| = \tau < \infty, \quad \left|\frac{df_2}{dV}\right| = (\theta+\mu+\omega\beta) < \infty,$$

$$\left|\frac{df_2}{dE}\right| = 0 < \infty, \left|\frac{df_2}{dI}\right| = \omega\beta < \infty,$$

$$\left|\frac{df_2}{dR}\right| = 0 < \infty, \left|\frac{df_2}{dR}\right| = 0 < \infty$$

$$\left|\frac{df_3}{dS}\right| = \frac{\beta}{\alpha} < \infty, \left|\frac{df_3}{dV}\right| = \omega\beta < \infty$$

$$\left|\frac{df_3}{dE}\right| = (\delta+\mu+\sigma) < \infty, \left|\frac{df_3}{dI}\right| = \frac{\beta(1+\alpha\omega)}{\alpha} < \infty,$$

$$\left|\frac{df_3}{dR}\right| = 0 < \infty (8), \left|\frac{df_4}{dS}\right| = 0 < \infty \;,, \left|\frac{df_4}{dV}\right| = 0 < \infty,$$

$$\left|\frac{df_4}{dE}\right| = \sigma < \infty, \left|\frac{df_4}{dI}\right| = (\gamma+\mu+k) < \infty,$$

$$\left|\frac{df_4}{dR}\right| = 0 < \infty, \left|\frac{df_5}{dS}\right| = 0 < \infty,$$

$$\left|\frac{df5}{dV}\right| = 0 < \infty, \left|\frac{df_5}{dE}\right| = \delta < \infty,$$

$$\left|\frac{df_5}{dI}\right| = \gamma < \infty, \left|\frac{df_5}{dR}\right| = (\xi + \mu) < \infty$$

The solution of the model is bounded and, therefore is well-posed in $\Re^5{}_+$.

Existence of disease-free equilibrium state

At the disease-free equilibrium point, there is no outbreak of disease. Hence,

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

$$\left.\begin{aligned} &B - \tfrac{\beta SI}{\alpha(S+I)} - \mu S + \xi R + \theta V - \tau S = 0 \\ &\tau S - \omega\beta VI - (\theta + \mu)V = 0 \\ &\tfrac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta + \mu + \sigma)E = 0 \\ &\sigma E - (\gamma + \mu + k)I = 0 \\ &\gamma I + \delta E - (\xi + \mu)R = 0 \end{aligned}\right\} \quad (9)$$

Since there is no outbreak out disease, we obtain that

$$\text{I} = \text{V} = 0, \quad \sigma E = (\gamma + \mu + k)I$$

$$E = 0$$

From (9), obtain that

$$(\xi + \mu)R = \gamma I + \delta E$$

$$R = \left(\frac{\gamma I + \delta E}{(\xi + \mu)}\right) = 0$$

Also from (9), we have that

$$B - \frac{\beta SI}{\alpha(S + I)} - \mu S + \xi R + \theta V - \tau S = 0$$

$$(\tau + \mu)S = B$$

$$S_o = \frac{B}{(\tau + \mu)}$$

The Disease Free Equilibrium

$$(\text{DFE})E_1 = \left(S_o, V_o, E_o, I_o, R_o\right) \text{ where } S_o \neq 0 \text{ and I=V= 0 is}$$

$$E_1 = \left(S_O = \frac{B}{(\tau+\mu)}, V_o = 0, E_o = 0, I_o = 0, R_o = 0\right) \quad (10)$$

Endemic equilibrium point

$$\text{Let } E_e = (S^*.V^*, E^*, I^*, R^*)$$

as Endemic Equilibrium where$I \neq 0$.

Consider the system of equation (1) the equilibrium points are:

$$I^* = \frac{\sigma E^*}{(\gamma+\mu+k)} \quad (11)$$

$$R^* = \frac{E^*[\sigma\gamma+\delta(\gamma+\mu+k)]}{(\gamma+\mu+k)(\xi+\mu)} \quad (12)$$

$$V^* = \frac{\tau S^*}{(\omega\beta I^*+\theta+\mu)} \quad (13$$

$$S^* = \frac{\beta-(\delta+\mu+\alpha)E^*+(\omega\beta\sigma I^*+\theta)+\xi E^*[\sigma\gamma+\delta(\gamma+\mu+k)]}{(\gamma+\mu+k)(\xi+\mu)(\tau+\mu)} \quad (14)$$

$$E^* = (\delta+\mu+\sigma)^{-1}\left[\frac{\omega\tau\beta\sigma(\delta+\mu+\alpha)}{(\gamma+\mu+k)^2[(\theta+\mu)+(\gamma+\mu+k)\omega\beta]}\right] \quad (15)$$

Basic reproduction number(R_0)

The model considers two compartments associated with the disease, yet only one is responsible for initiating new infections. Consequently, the exposed and infectious groups are interrelated.

This linkage reflects the expected number of new cases generated by a single infectious person in the population, where $R_o = G = \rho(F \times V^{-1})$

Using Next Generation Matrix approach,

$$\frac{dE}{dt} = \frac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta+\mu+\alpha)E$$
$$\frac{dI}{dt} = \alpha E - (\gamma+\mu)I$$

From the system of equation above, consider the disease compartments where $R_o = G = \rho(F \times V^{-1})$ and $S_o = \frac{B}{(\tau+\mu)}$We have the transition matrix V and F are obtained from the partial derivatives of F and V

with respect to (E, I), evaluated at the disease free equilibrium E ^{1}. Thus,

$$F_i = \left[\frac{\partial f_i(x_i)}{\partial x_j}\right] \text{ and } V_i = \left[\frac{\partial v_i(x_i)}{\partial x_j}\right], i = 1,\ldots,5. \quad (16)$$

$$F = \begin{bmatrix} \frac{\beta S_o}{\alpha} \\ 0 \end{bmatrix}, \text{and } V = \begin{bmatrix} -\omega\beta VI + (\delta+\mu+\sigma)E \\ -\sigma E + (\gamma+\mu+k)I \end{bmatrix}$$

$$\text{And } F = \begin{vmatrix} \frac{\beta B}{\alpha(\tau+\mu)} & 0 \\ 0 & 0 \end{vmatrix}, \text{and}$$

$$|V| = (\delta+\mu+\sigma)(\gamma+\mu+k) - \sigma\omega\beta$$

$$V^{-1} = \begin{vmatrix} \frac{(\gamma+\mu+k)}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} & \frac{\sigma}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \\ \frac{\omega\beta}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} & \frac{(\delta+\mu+\sigma)}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \end{vmatrix}.$$

$$\text{Since } R_o = G = \rho(F \times V^{-1})$$

$$R_o = \begin{vmatrix} \frac{\beta B}{\alpha(\tau+\mu)} & 0 \\ 0 & 0 \end{vmatrix} \begin{vmatrix} \frac{(\gamma+\mu+k)}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} & \frac{\sigma}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \\ \frac{\omega\beta}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} & \frac{(\delta+\mu+\sigma)}{(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \end{vmatrix}$$

$$R_o = \frac{(\gamma+\mu+k)\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \quad (17)$$

The dominant Eigen-value is the Basic reproductive ratio.

Local stability of disease-free equilibrium

The Disease-Free Equilibrium (DFE) of the proposed Epidemic Model is Locally Asymptotically Stable if $R_o < 1$and unstable if $R_o > 1$.

The local stability of the disease-free equilibrium at

$$S_o = \frac{B}{(\tau+\mu)} \text{as } \mathrm{E}^1 = \left(S_o = \frac{B}{(\tau+\mu)}, 0,0,0, R = 0\right)$$

The Jacobian matrix of the system of (1) where $\left|J_{E_1} - \lambda_i I\right| = 0$ as λ_i and I are the Eigen-values and identity matrix respectively. Where i = 1, 2, 3, 4, 5.

Therefore:

$$J_{(E_1)} = \begin{vmatrix} -(\mu+\tau) & \theta & 0 & -\frac{\beta S_o}{\alpha(S_o+1)} & \xi \\ \tau & -(\theta+\mu) & 0 & 0 & 0 \\ 0 & 0 & -(\delta+\mu+\sigma) & \frac{\beta S_o}{\alpha(S_o+1)} & 0 \\ 0 & 0 & \sigma & -(\gamma+\mu+k) & 0 \\ 0 & 0 & \delta & \gamma & -(\xi+\mu) \end{vmatrix} (18)$$

Then, at disease Free Equilibrium; I = V= 0

$$\left|J_{E_1} - \lambda_i I\right| = 0$$

$$\begin{vmatrix} -(\mu+\tau)-\lambda_1 & \theta & 0 & -\frac{\beta S_o}{\alpha(S_o+1)} & \xi \\ \tau & -(\theta+\mu)-\lambda_2 & 0 & 0 & 0 \\ 0 & 0 & -(\delta+\mu+\sigma)-\lambda_3 & \frac{\beta S_o}{\alpha(S_o+1)} & 0 \\ 0 & 0 & \sigma & -(\gamma+\mu+k)-\lambda_4 & 0 \\ 0 & 0 & \delta & \gamma & -(\xi+\mu)-\lambda_5 \end{vmatrix} = 0 (19)$$

We obtain that,

$$\begin{vmatrix} -(\delta+\mu+\sigma)-\lambda & \frac{\beta S_o}{\alpha(S_o+1)} \\ \sigma & -(\gamma+\mu+k)-\lambda \end{vmatrix} = 0$$

$$\lambda = -(\mu+\tau)$$
$$\lambda = -(\xi+\mu)$$

$$\lambda = -(\theta+\mu)$$

for respective Eigen-value , let for respective Eigen-value , we obtain ;

$$a = -(\delta+\mu+\sigma), b = (\gamma+\mu+k), c = \frac{\beta S_o}{\alpha(S_o+1)}$$

$$\lambda^2 - (a+b)\lambda + (ab-c) = 0$$

$$\lambda = \frac{1}{2}abc + \sqrt{a[a-2bc(a+b)] - 3ab(a-c) + 2a(b+c))} - ab - \sqrt{a+2b[c-2ab]} (20)$$

Because the sets are negatively invariant, the system is consequently locally asymptotically stable.

Local Stability of the Endemic Equilibrium

The endemic equilibrium (EE) of the proposed epidemic Model exhibits local asymptotic Stability

if $R_o < 1$ and otherwise unstable.

Let $S = a + S^*, V = b + V^*E = c + E^*, I = d + I^*, R = e + R^*.$ (21)

From system of equation (21),

$$\left.\begin{aligned}
\frac{dS}{dt} &= B - \frac{\beta SI}{\alpha(S+I)} - \mu S + \xi R + \theta V - \tau S \\
\frac{dV}{dt} &= \tau S - (\omega\beta I + \theta + \mu)V \\
\frac{dE}{dt} &= \frac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta + \mu + \sigma)E \\
\frac{dI}{dt} &= \sigma E - (\gamma + \mu + k)I \\
\frac{dR}{dt} &= \gamma I + \delta E - (\xi + \mu)R
\end{aligned}\right\} \qquad (22)$$

By Linearization substituting (21) into (22) above to obtain;

$$\frac{da}{dt} = B - \beta(a + S^*)(d + I^*)\alpha[(a + S^*) + (d + I^*)]^{-1} - \mu(a + S^*)$$
$$+\xi(e + R^*) + \theta(b + V^*) - \tau(a + S^*)$$

$$\frac{db}{dt} = \tau(a + S^*) + \omega\beta(b + V^*)(d + I^*) - (\theta + \mu)(b + V^*)$$

$$\frac{dc}{dt} = \beta(a + S^*)(d + I^*)\alpha[(a + S^*) + (d + I^*)]^{-1}$$
$$+\omega\beta(b + V^*)(d + I^*) - (\delta + \mu + \sigma)(c + E^*)$$

$$\frac{dd}{dt} = \sigma(c + E^*) - (\gamma + \mu + k)(d + I^*)$$

$$\frac{de}{dt} = \gamma(d + I^*) + \delta(c + E^*) - (\xi + \mu)(e + R^*)$$

Therefore, the Jacobian matrix of the system of (23) where $\left|J_{E_1} - \lambda_i I\right| = 0, i = 1, \ldots 5$ (23)

$$J_{(E^*)} = \begin{vmatrix}
-\left[\frac{(\tau+\mu)(d+1)+\beta d\alpha}{(d+1)}\right] & \theta & 0 & -\left[\frac{(\tau+\mu)(a+1)+\beta a\alpha}{(a+1)}\right] & \xi \\
\tau & -[\omega\beta d + (\theta+\mu)] & 0 & -\omega\beta b & 0 \\
\left[\frac{\beta d\alpha}{(d+1)}\right] & \omega\beta d & -(\delta+\mu+\sigma) & \left[\frac{\omega\beta b(a+1)+\beta a\alpha}{(a+1)}\right] & 0 \\
0 & 0 & \sigma & -(\gamma+\mu+k) & 0 \\
0 & 0 & \delta & \gamma & -(\xi+\mu)
\end{vmatrix}$$

$$\begin{vmatrix} -\left[\frac{(\tau+\mu)(d+1)+\beta d\alpha}{(d+1)}\right]-\lambda_1 & \theta & 0 & -\left[\frac{(\tau+\mu)(a+1)+\beta a\alpha}{(a+1)}\right] & \xi \\ \tau & -[\omega\beta d+(\theta+\mu)]-\lambda_2 & 0 & -\omega\beta b & 0 \\ \left[\frac{\beta d\alpha}{(d+1)}\right] & \omega\beta d & -(\delta+\mu+\sigma)-\lambda_3 & \left[\frac{\omega\beta b(a+1)+\beta a\alpha}{(a+1)}\right] & 0 \\ 0 & 0 & \sigma & -(\gamma+\mu+k)-\lambda_4 & 0 \\ 0 & 0 & \delta & \gamma & -(\xi+\mu)-\lambda_5 \end{vmatrix} = 0$$

The resulting Eigen-values are

$$\left(-\left[\frac{(\tau+\mu)(d+1)+\beta d\alpha}{(d+1)}\right]-\lambda\right)(-[\omega\beta d+(\theta+\mu)]-\lambda)$$
$$(-(\delta+\mu+\sigma)-\lambda)(-(\gamma+\mu+k)-\lambda)(-(\xi+\mu)-\lambda)=0 \quad (24)$$

Let

$$A=-\left[\frac{(\tau+\mu)(d+1)+\beta d\alpha}{(d+1)}\right],$$
$$B=-[\omega\beta d+(\theta+\mu)],$$
$$C=-(\delta+\mu+\tau),$$
$$D=-(\gamma+\mu+k),$$
$$E=-(\xi+\mu)$$

Then we have,

$$(A-\lambda)(B-\lambda)(C-\lambda)(D-\lambda)(E-\lambda)=0 \quad (25)$$

$$\lambda^5-[E+(C+D)+(A+B)]\lambda^4+[(A+B)(C+D)+AB+CD](1+E)\lambda^3-$$
$$[AB(C+D)+CD(A+B)](1+E)\lambda^2+E[AB(C+D)+CD(A+B)]\lambda-ABCDE=0.$$

Hence, the system is locally asymptotically stable.

Global Stability of the Disease- Free Equilibrium

To examine the global asymptotic stability of the proposed model in Case 1, we employ the Lyapunov function method. By applying the Lyapunov stability criterion, the global stability of the system at the disease-free equilibrium (DFE) can be established.

$$V(t,S,V,E,I,R) = CI_1 + CI_2 \qquad (26)$$

$$\frac{dV}{dt} = C_1 I_1^{\bullet} + C_2 I_2^{\bullet}$$

$$= C_1\left[\frac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta+\mu+\sigma)E\right] + C_2[\sigma E - (\gamma+\mu+k)I]$$

$$\leq C_1\left[\frac{\beta S_o}{\alpha} I_2 - (\delta+\mu+\sigma)I_1\right] + C_2[\sigma I_1 - (\gamma+\mu+k)I_2]$$

$$= C_1\frac{\beta S_o}{\alpha} I_2 - C_1(\delta+\mu+\sigma)I_1 + C_2\sigma I_1 - C_2(\gamma+\mu+k)I_2$$

$$\leq [C_2\sigma - C_1(\delta+\mu+\sigma)]I_1 + \left[C_1\frac{\beta S_o}{\alpha} - C_2(\gamma+\mu+k)\right] I_2 \leq N, S(27)$$

As $S_O = \frac{B}{(\tau+\mu)}$,

Let $C_1 = \frac{1}{(\delta+\mu+\sigma)}$, $C_2 = \frac{\beta B}{\alpha(\delta+\mu+\sigma)(\tau+\mu)(\gamma+\mu+k)}$

$$\leq \left[\frac{\beta B\sigma}{\alpha(\delta+\mu+\sigma)(\tau+\mu)(\gamma+\mu+k) - \sigma\omega\beta} - 1\right] I_1$$

$$+ \left[\frac{\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)} - \frac{\beta B(\gamma+\mu+k)}{\alpha(\delta+\mu+\sigma)(\tau+\mu)(\gamma+\mu+k)}\right] I_2$$

$$V^* \leq (\gamma+\mu+k)\left[\frac{\beta B\sigma}{\alpha(\delta+\mu+\sigma)(\tau+\mu)(\gamma+\mu+k) - \sigma\omega\beta} - 1\right] I_1$$

$$V^{\bullet} \leq (\gamma+\mu+k)[R_o - 1]I(28)$$

It is imperative to note that $V^{\bullet} = 0$ only when E = 0, the substitution of E = 0 into the model system of equation (1) shows that $S_O = \frac{B}{(\tau+\mu)}$ at $t \to \infty$, $\sigma\omega\beta \leq 1$. Based on LaSalle's invariance principle. Hence E_o = 0 is globally asymptotically stable whenever R_o > 1.

SENSITIVITY ANALYSIS OF R_0

In this section, we compute the sensitivity analysis which determines how sensitive each parameter of basic reproduction is to the disease control. This is obtained by differentiating $\boldsymbol{R_o}$ with respect to all the parameters in$\boldsymbol{R_o}$. The normalized forward sensitivity index is defined:

$$\text{As } R_o = \frac{(\gamma+\mu+k)\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}$$

$$\frac{\partial R_o}{\partial \mu} = \frac{(\gamma+k)\beta B}{\alpha\tau(\delta+\sigma)(\gamma+k)-\sigma\omega\beta} \cdot \frac{\mu\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$

$$= \frac{(\gamma+k)\beta B\mu\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\alpha\tau(\delta+\sigma)(\gamma+k)-\sigma\omega\beta} \quad (29)$$

$$\frac{\partial R_o}{\partial \alpha} = \frac{(\gamma+\mu+k)\beta B}{(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \cdot \frac{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$
$$= \alpha \quad (30)$$

$$\frac{\partial R_o}{\partial \gamma} = \frac{(\mu+k)\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\mu+k)-\sigma\omega\beta} \cdot \frac{\gamma\alpha(\gamma+\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$

$$= \frac{(\mu+k)\gamma\alpha(\gamma+\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\alpha(\tau+\mu)(\delta+\mu+\sigma)(\mu+k)-\sigma\omega\beta} \rightleftarrows \quad (31)$$

$$\frac{\partial R_o}{\partial \beta} = \frac{(\gamma+\mu+k)B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega} \cdot \frac{\beta\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B} = \beta \quad (32)$$

$$\frac{\partial R_o}{\partial k} = \frac{(\gamma+\mu)\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu)-\sigma\omega\beta} \cdot \frac{k\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$
$$= \frac{(\gamma+\mu)k}{(\gamma+\mu+k)} \quad (33)$$

$$\frac{\partial R_o}{\partial B} = \frac{(\gamma+\mu+k)\beta}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \cdot \frac{B\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$
$$= 1 \quad (34)$$

$$\frac{\partial R_o}{\partial \tau} = \frac{(\gamma+\mu+k)\beta B}{\alpha\mu(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta} \cdot \frac{\tau\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)(34)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$
$$= \frac{\tau(\tau+\mu)}{\mu} \quad (35)$$

$$\frac{\partial R_o}{\partial \sigma} = \frac{(\gamma+\mu+k)\beta B}{\alpha(\tau+\mu)(\delta+\mu)(\gamma+\mu+k)-\omega\beta}$$
$$\frac{\sigma\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$
$$= \frac{\sigma\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{\alpha(\tau+\mu)(\delta+\mu)(\gamma+\mu+k)-\omega\beta} \quad (36)$$

$$\frac{\partial R_o}{\partial \omega} = \frac{(\gamma+\mu+k)\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\beta} \cdot \frac{\omega\sigma(\tau+\mu)(\delta+\mu+\sigma)(\gamma+\mu+k)-\sigma\omega\beta}{(\gamma+\mu+k)\beta B}$$
$$= \omega(37)$$

Table 3: Parameter and indices sensitivity analysis

Parameter	Sensitivity
B	0.1778
β	1.56
k	0.05357142857
σ	0.246
γ	0.45
μ	0.003
$\boldsymbol{\delta}$	-0.456
$\boldsymbol{\alpha}$	0.765
$\boldsymbol{\omega}$	0.07142857143
θ	0.03571428571
$\boldsymbol{\tau}$	0.3500000000
ξ	0.3445

The sensitivity analysis provides information about how changes in model parameters influence the model's output or outcomes of interest. A positive sensitivity value indicates that an increase in the

parameter value leads to an increase in the model's output, while a negative sensitivity value indicates the opposite.

In the sensitivity analysis results for the tuberculosis model, several parameters were analyzed to understand their impact on the model's output. The highest positive sensitivity value was found to be 0.765, which corresponds to the contact rate.

On the other hand, the least negative sensitivity value was -0.456, which corresponds to the transmission rate from exposure to recovery. The contact rate represents the rate at which susceptible individuals come into contact with infectious individuals.

A higher contact rate means that individuals are more likely to come into contact with infectious individuals, which can lead to an increase in tuberculosis transmission. The positive sensitivity value of 0.765 suggests that an increase in the contact rate has a strong positive impact on the spread of tuberculosis in the model. This implies that interventions or policies aimed at reducing contact between susceptible and infectious individuals can have a significant impact on reducing tuberculosis transmission.

On the other hand, the transmission rate from exposure to recovery represents the rate at which individuals progress from the exposed state (infected but not yet infectious) to the recovery state. A higher transmission rate in this context means that individuals recover from tuberculosis more quickly after being exposed. The negative sensitivity value of -0.456 indicates that a higher transmission rate from exposure to recovery has a negative impact on the model's output. This suggests that interventions or treatments that reduce the recovery time for individuals in the exposed state can help control the spread of tuberculosis.

NUMERICAL SIMULATIONS

In this section, numerical experiments are carried out to validate the analytical findings obtained earlier. The analysis focuses on examining the impact of three key parameters, selected according to their role in vaccination dynamics.

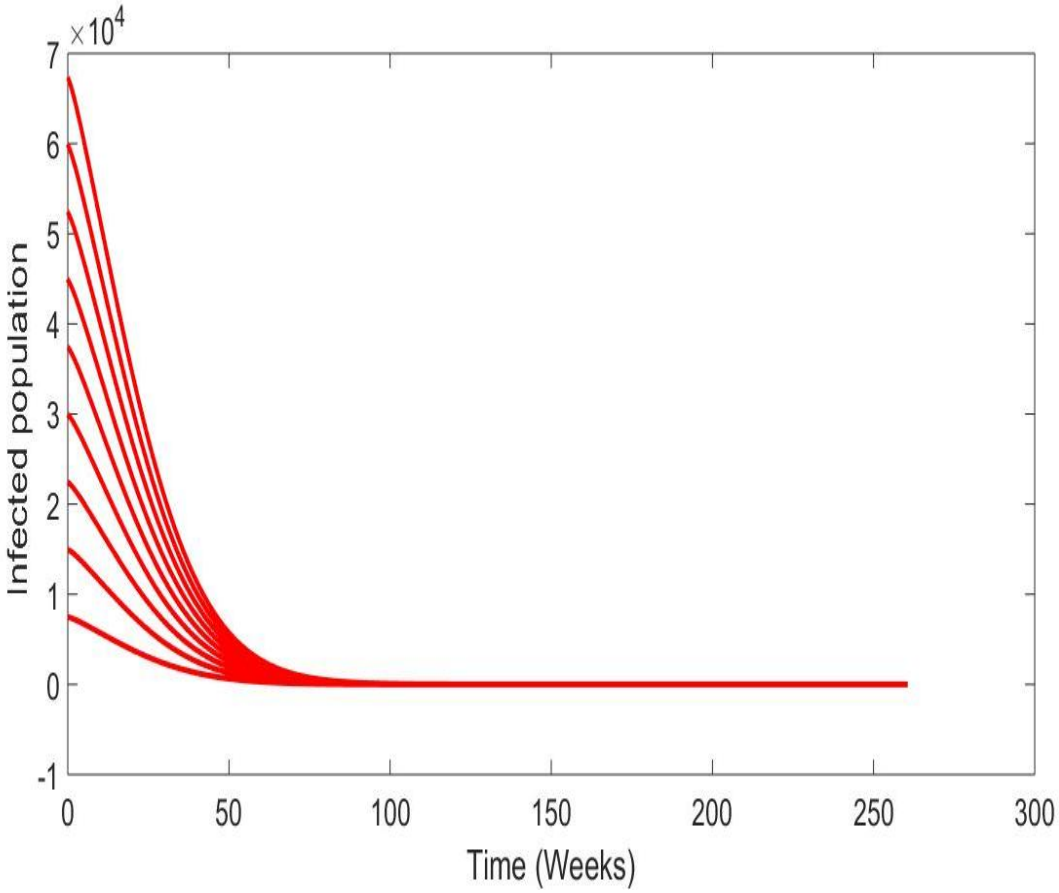

Figure 2: .Convergence of solution trajectories when $R_0<1$

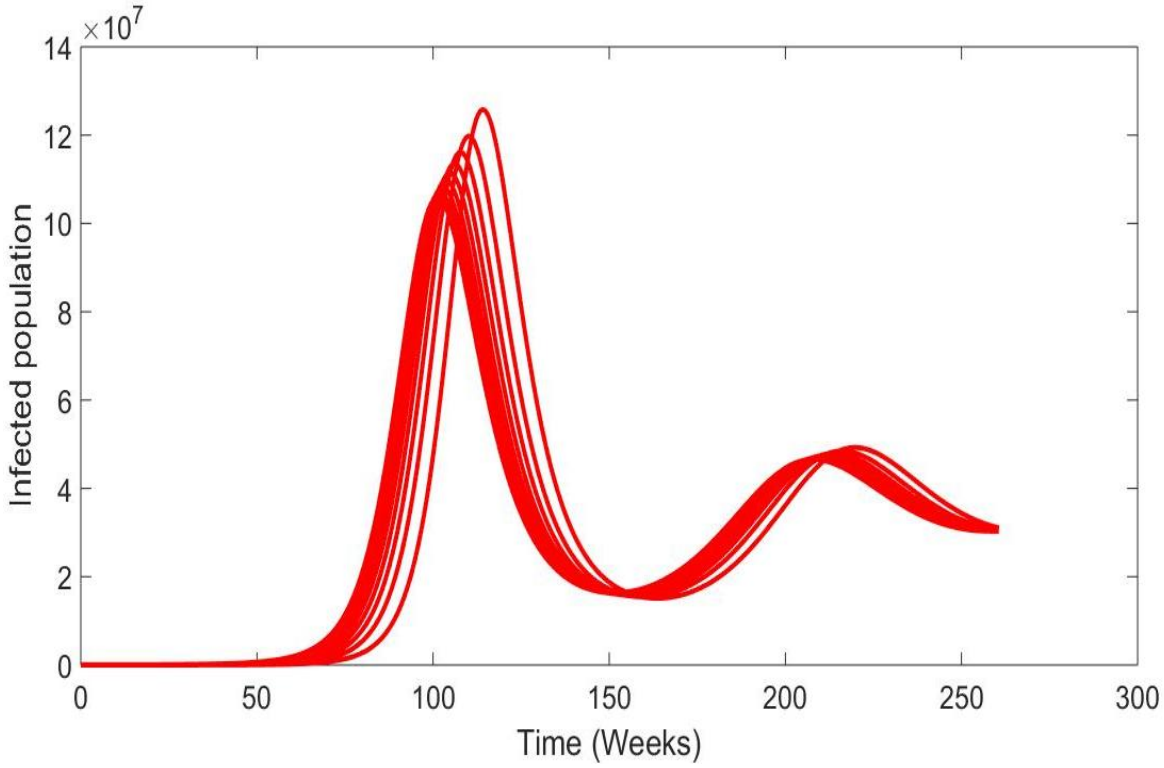

Figure 3.Convergence of solution trajectories when $R_0>1$

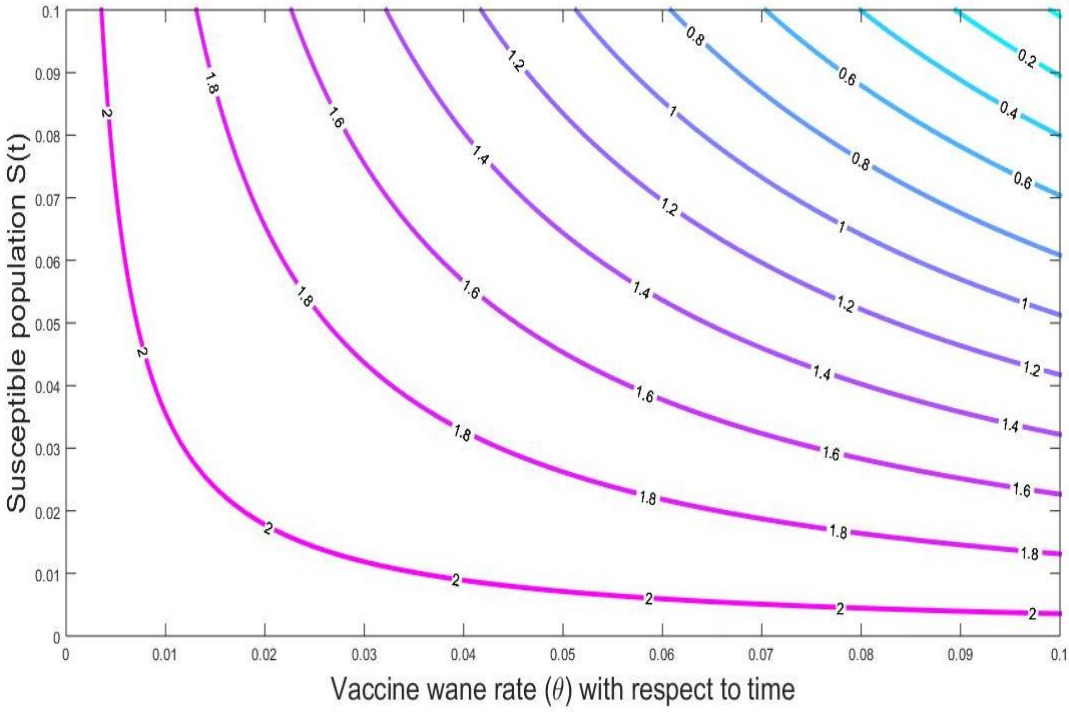

Figure 4 .Effects of vaccine wane rate θ on susceptible S(t) with respect to time

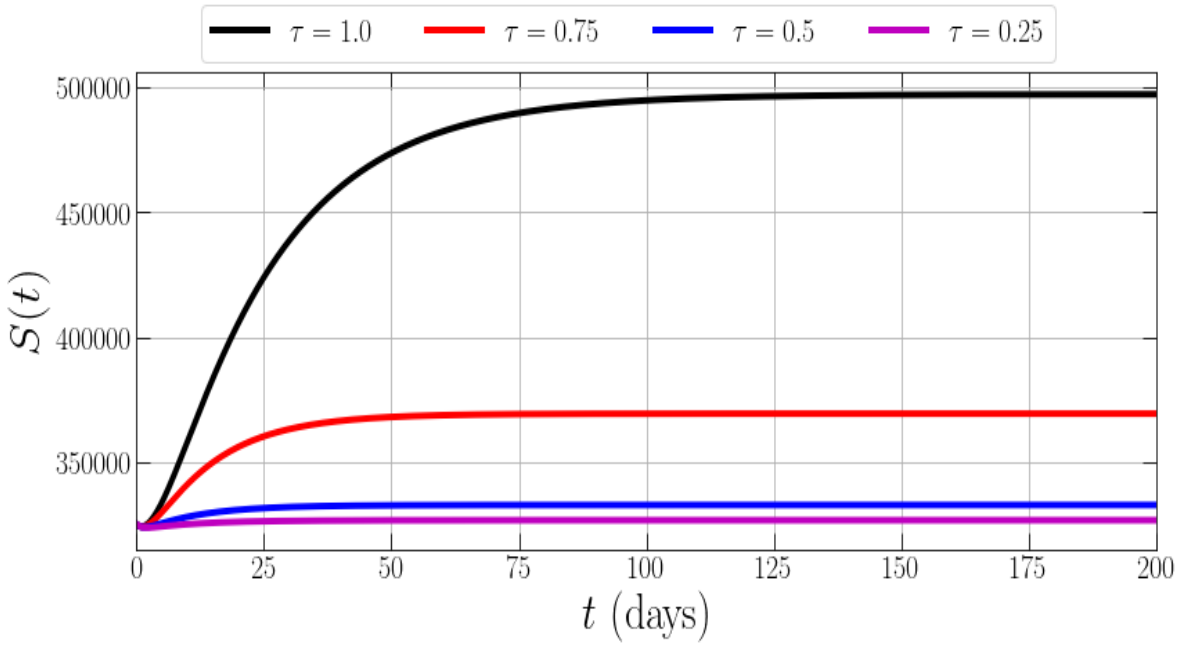

Figure 5. Effect of vaccination rate τ on susceptible population

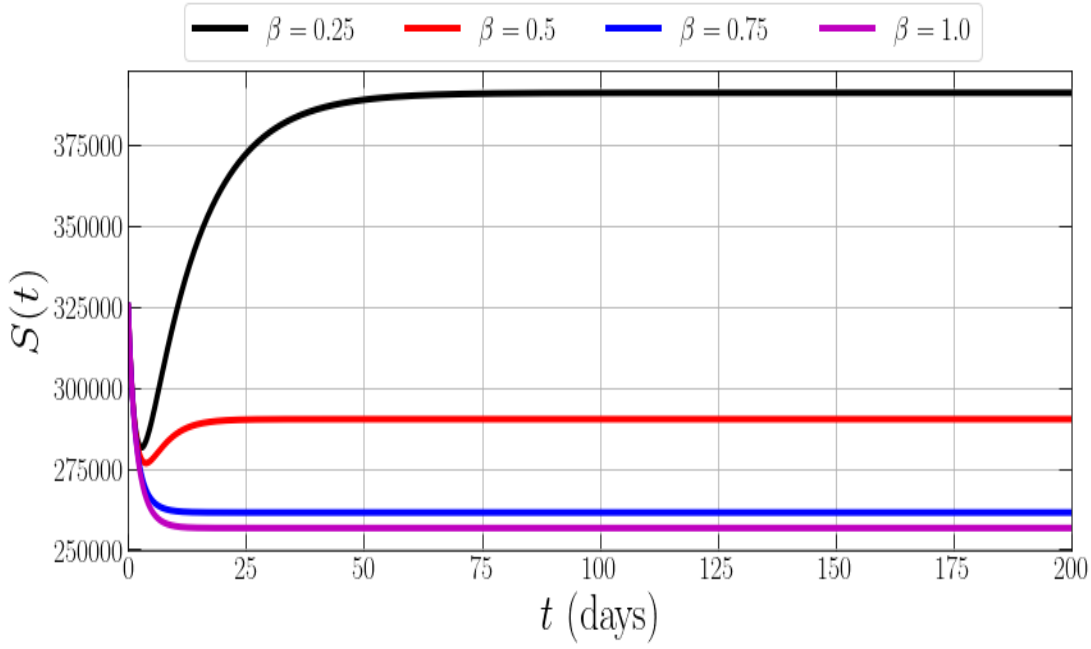

Figure 6.Effect of transmission rate β on susceptible population

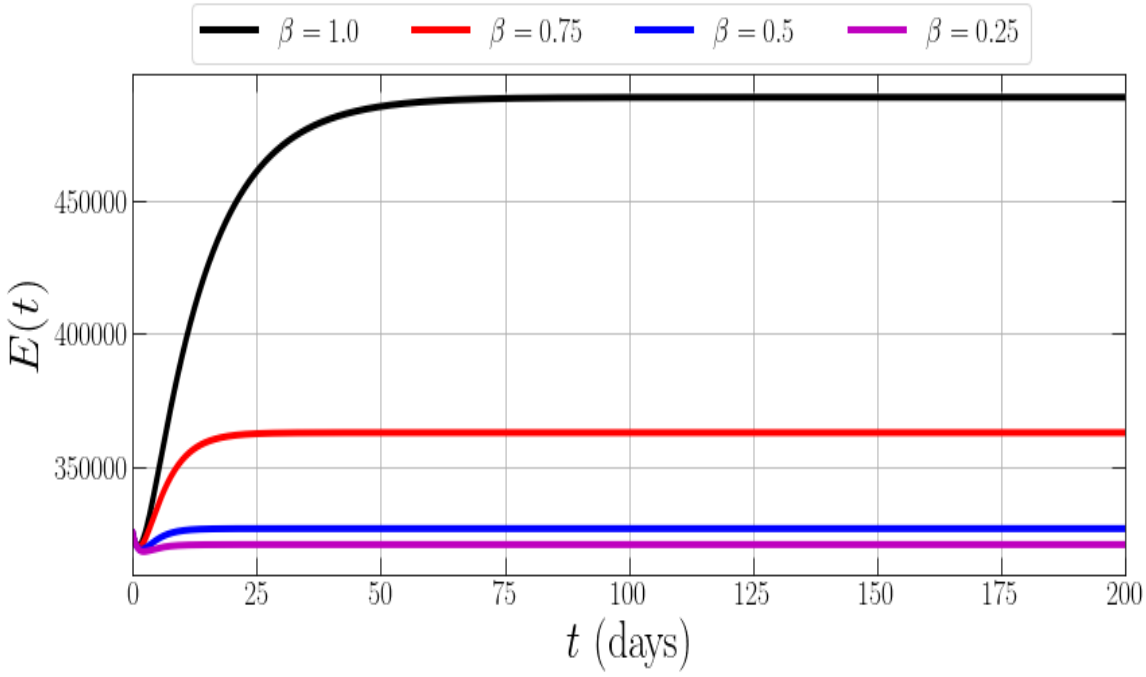

Figure 7.Effect of transmission rate β on exposed population

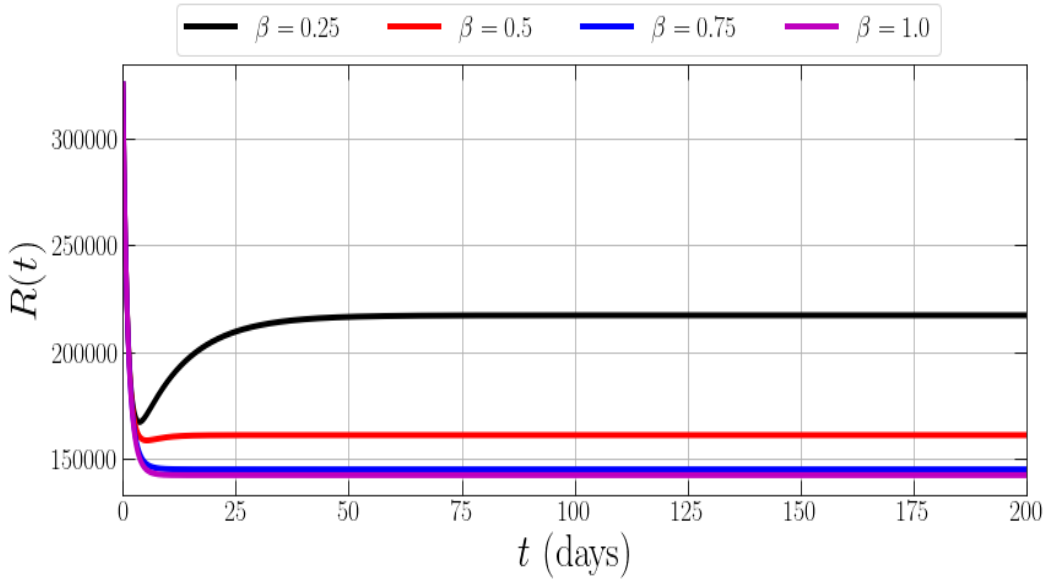

Figure 8.Effect of transmission rate β on recovered population

DISCUSSION

Figure 2 represents the convergence of solution trajectories when R0<1 this implies that the disease transmission will probably wane off because one infectious case will infect less than one person on average. Figure 3. Convergence of solution trajectories when R0>1. In this case, all control strategies must be

implemented to bring the basic reproduction below unity. Figure 4 shows the effects of vaccine wane rate θ on susceptible S(t) with respect to time.

The vaccination tends to wane off with respect to time and individuals who are vaccinated can become susceptible to the disease again. Figure 5 shows the effect of the vaccination rate τ on susceptible populations.

The results show that increasing the vaccination rate will increase the population of the susceptible population. In converse, if the vaccination rate is reduced, more people will be infected thereby, reducing the population of the susceptible individuals. Figure 6 shows the effect of the transmission rate β on susceptible populations. Also, Figure 6.shows that increasing the rate of transmission will reduce the susceptible population that is, more people will be infected. Figure 7. Shows the effect of the transmission rate β on exposed population. Individuals who are exposed to the disease have the tendency to be infected when in contact with the infected individual thereby increasing the exposed population. Figure 8.Shows the effect of transmission rate β on the recovered population as seen in Figure 8, increasing the rate of transmission will reduce the recovered population since recovered individuals are not permanently immune against the disease.

CONCLUSION

In this study, we developed a deterministic model on TB transmission dynamics. The corresponding threshold quantity was discovered by looking at the qualitative behaviors of the model as it was presented. When the effective reproduction number is less than unity, the system's tuberculosis-free equilibrium is considered to be locally asymptotically stable; otherwise, it is unstable. Additionally, we looked at the model's stability analysis and sensitivity analysis. The result of the sensitivity analysis showed that the contact rate was the most sensitive parameter to the disease transmission. It is therefore recommended that, a reduction in the contact rate will reduce the disease burden in the population. The theoretical findings were demonstrated and supported by a numerical simulation. The overall finding indicates that decreasing the contact rate with the susceptible person and increasing the rate of immunizing susceptible persons with highly efficient vaccines will lower the prevalence of tuberculosis in the population.

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