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ORIGINAL RESEARCH

Mathematical Model of Tuberculosis Transmission Dynamics with Vaccination

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Abstract

Tuberculosis (TB) is caused by bacteria. It can spread via close contact with people who have active TB. To gain a better understanding of the disease spread and control, we develop a deterministic model for this purpose. 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 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.

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

Introduction

Tuberculosis (TB) is an infectious illness caused by a kind of bacteria that primarily affects the lungs. It spreads throughout the atmosphere (Bisuta et al., 2018; Brian et al., 2020; Colditz et al., 2019). TB can be transferred when a person with active TB disease coughs, sneezes, talks, sings, or even laughs, releasing germs into the air. Only patients suffering from an active lung infection are infectious in (Brian et al., 2020). The majority of people who breathe in TB bacteria are able to resist it and stop it from multiplying. In these people, the bacterium becomes inactive, resulting in a latent tuberculosis infection in (Daniel, 2022).

Tuberculosis is both preventable and cured. TB most commonly affects your lungs, but it can also damage other organs such as your brain. A 25% of the world's population is thought to be afflicted with tuberculosis bacteria. Several medications have been licensed for the treatment of tuberculosis by (Dowdy et al., 2021; Egonmwan and Okuonghae 2022; Fatima et al., 2021; Gomes et al., 2023). The treatment plan is different depending on whether the person has latent or active tuberculosis. People with latent tuberculosis are typically treated with preventative care to ensure that the disease does not become active

and spread to others in the future (Intan et al., 2022). Active TB usually requires a combination of four medications to treat. Infectious disease mathematical modeling is a tool to analyze the dynamics of infectious diseases through which diseases spread, to forecast the future course of an outbreak, and to assess control methods in (Kaufmann et al., 2024). This can offer helpful information about disease transmission patterns and the identification of factors that can reduce disease in the population. Many mathematical models of the transmission dynamics of TB has been formulated and analyzed by numerous researchers (Khajanchi et al., 2023; Pai et al., 2020). The construction of a model to study the function of partial therapy in tuberculosis disease transmission (Latifat et al.,2020) investigated a dynamical tuberculosis sickness problem that included both hospitalized and non-hospitalized infectious classes (Michael et al., 2020; Mojisola et al., 2020; Oguntolu et al., 2023; Ojo et al., 2022). Also proposed a tuberculosis disease model to predict the impact of treatment and analyze infectious persons to study the stability of tuberculosis with partial treatment (Olanrewaju et al., 2022; Pai et al., 2020; Awoke and Kassa et al., 2021; Ullah et al., 2022). They explored tuberculosis transmission while accounting for the presence of a latent group and vaccination of the susceptible class. Other researchers have also developed TB models under different epidemiological scenarios (Wazwaz, 2022; WHO, 2020; WHO; 2018; Yang et al., 2021). Other studies on the dynamics of TB models can be found in (Zhang et al., 2022; Zumla et al., 2023). In this paper, we develop a new deterministic model by investigating the effects of some control strategies on TB transmission. The outcome of this work and serve as a basis for governmental organization and recommendations for TB control. The rest of the paper is structured as follows; Section two deals with the model formulation, section three deals with the analysis of the model while sections four and five deals with the numerical simulation and discussion of results respectively. Finally, we give the conclusion in section six

2. Formulation of the model

In this section, we develop a deterministic model of TB with five compartments, these are susceptible S(t), exposed E(t), infected I(t), vaccinated V(t), and recovered R(t). The rate of recruitment into the susceptible class is at a rate *B*, susceptible individuals received vaccination at a rate and loss immunity at a rate θ. β Represents the force of infection and the progression rate between the exposed to infected class is denoted by the saturated term α. The recovery rate is represented by γ. The recovered individual lost immunity after recovery and returned to the susceptible class at a rate ξ. There exists a transmission from exposed to recovered class at a rate δ. Natural death rate occurs in all the classes at a rate μ with a disease-induced death rate at a rate *k*. ω is the reduction in the risk of infection due to vaccination. The above descriptions can be represented by a system of nonlinear differential equation 1 and the pictorial illustration is presented in Figure 1.

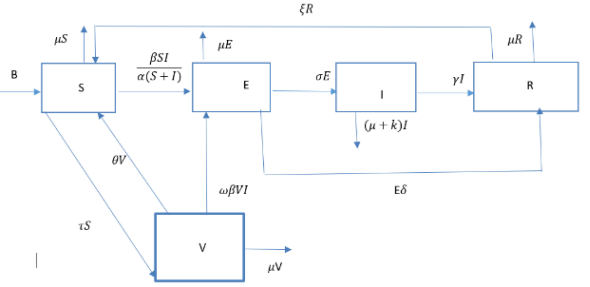

Figure 1. Schematic diagram of the model

$$\left.\begin{aligned}
\frac{dS}{dt} &= B - \frac{\beta SI}{\alpha(S+I)} - \mu S + \xi R + \theta V - \tau S \\
\frac{dV}{dt} &= \tau S - (\omega\beta I + \theta + \mu)V \\
\frac{dE}{dt} &= \frac{\beta SI}{\alpha(S+I)} + \omega\beta VI - (\delta + \mu + \sigma)E \\
\frac{dI}{dt} &= \sigma E - (\gamma + \mu + k)I \\
\frac{dR}{dt} &= \gamma I + \delta E - (\xi + \mu)R
\end{aligned}\right\} \quad (1)$$

Subject to the following initial conditions

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

Table I: Description of parameters

VARIABLES	DEFINITION
$S(t)$	The number of Susceptible individuals at time t
V(T)	The number of Vaccinated individuals at time t
$E(t)$	The number of Exposed individuals at time t
$I(t)$	The number of Infected individuals at time t
$R(t)$	The number of Recovered individuals at time t
PARAMETER	**DEFINITION**
σ	Progression rate from Latent to infected
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
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)

3. Analysis of the model

3.1. Positivity and boundedness of model solution

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

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

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

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

such that

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

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

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

$$\frac{dN(t)}{dt} \leq B - \mu N$$

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

Solving equation (4) using Gronwall's inequality gives

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

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

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

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

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

3.2. Existence and uniqueness of model solution

Let:

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

Then,

$\left|\frac{df_1}{dS}\right| = \frac{\alpha(\mu+\tau)+\beta}{\alpha} < \infty$, $\left|\frac{df_1}{dV}\right| = \theta < \infty$, $\left|\frac{df_1}{dE}\right| = 0 < \infty$, $\left|\frac{df_1}{dI}\right| = \frac{\beta}{\alpha} < \infty$, $\left|\frac{df_1}{dR}\right| = \xi < \infty$

$\left|\frac{df_2}{dS}\right| = \tau < \infty$, $\left|\frac{df_2}{dV}\right| = (\theta + \mu + \omega\beta) < \infty$, $\left|\frac{df_2}{dE}\right| = 0 < \infty$, $\left|\frac{df_2}{dI}\right| = \omega\beta < \infty$, $\left|\frac{df_2}{dR}\right| = 0 < \infty$ $\left|\frac{df_2}{dR}\right| = 0 < \infty$

$\left|\frac{df_3}{dS}\right| = \frac{\beta}{\alpha} < \infty$, $\left|\frac{df_3}{dV}\right| = \omega\beta < \infty$, $\left|\frac{df_3}{dE}\right| = (\delta + \mu + \sigma) < \infty$, $\left|\frac{df_3}{dI}\right| = \frac{\beta(1+\alpha\omega)}{\alpha} < \infty$, $\left|\frac{df_3}{dR}\right| = 0 < \infty$ (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 $\Re^5{}_+$.

3.3. Existence of disease-free equilibrium state

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

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

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

Since there is no outbreak out disease, we obtain that I = V = 0,

$$\sigma E = (\gamma + \mu + k)I$$

$$E = 0$$

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

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

3.4. Endemic equilibrium point

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:

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

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

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

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

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

3.5. Basic reproduction number (R_0)

There are two disease states but only one to create new infection. Hence, Exposed and Infected compartments of the model are connected. This denote the number of secondary infection caused as a result of infected individual in a 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,

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

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

And $F = \begin{vmatrix} \frac{\beta B}{\alpha(\tau+\mu)} & 0 \\ 0 & 0 \end{vmatrix}$, and $|V| = (\delta+\mu+\sigma)(\gamma+\mu+k) - \sigma\omega\beta$

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

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

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

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

The dominant Eigen-value is the Basic reproductive ratio.

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

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

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

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

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

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 ;

$$a = -(\delta+\mu+\sigma), b = (\gamma+\mu+k), c = \frac{\beta S_o}{\alpha(S_o+1)}$$
$$\lambda^2 - (a+b)\lambda + (ab-c) = 0$$

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

Since they are negatively invariant, therefore they are Locally Asymptotically Stable.

3.8. Local stability of endemic equilibrium

The Endemic Equilibrium (DFE) of the proposed Epidemic Model is Locally Asymptotically Stable if $R_o < 1$ and unstable otherwise.
Let $S = a + S^*, V = b + V^* E = c + E^*, I = d + I^*, R = e + R^*.$ (21)

From system of equation (21),

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

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 $|J_{E_1} - \lambda_i I| = 0, i = 1,\ldots 5$ (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

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

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

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

Therefore, they are Locally Asymptotically Stable.

3.9. Global stability of disease free equilibrium

Consider the use of Lyapunov function approach to proceed for the result for global asymptotic stability of the proposed model for case 1, at Disease Free Equilibrium State. Using Lyapunov algorithm;

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

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

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

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

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

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

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

$$\leq \left[\frac{\beta B\sigma}{\alpha(\delta+\mu+\sigma)(\tau+\mu)(\gamma+\mu+k)-\sigma\omega\beta} - 1\right]I_1 + \left[\frac{\beta B}{\alpha(\tau+\mu)(\delta+\mu+\sigma)} - \frac{\beta B(\gamma+\mu+k)}{\alpha(\delta+\mu+\sigma)(\tau+\mu)(\gamma+\mu+k)}\right]I_2$$

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

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

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.

4. 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:

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

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

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

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

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

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

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

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

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

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

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

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
δ	-0.456
α	0.765
ω	0.07142857143
θ	0.03571428571
τ	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.

4. Numerical simulations

In this section, some numerical simulations are performed to confirm the previous analytical results. The influence of three important parameters is chosen based on the effect of vaccination, vaccine wane rate and transmission rate.

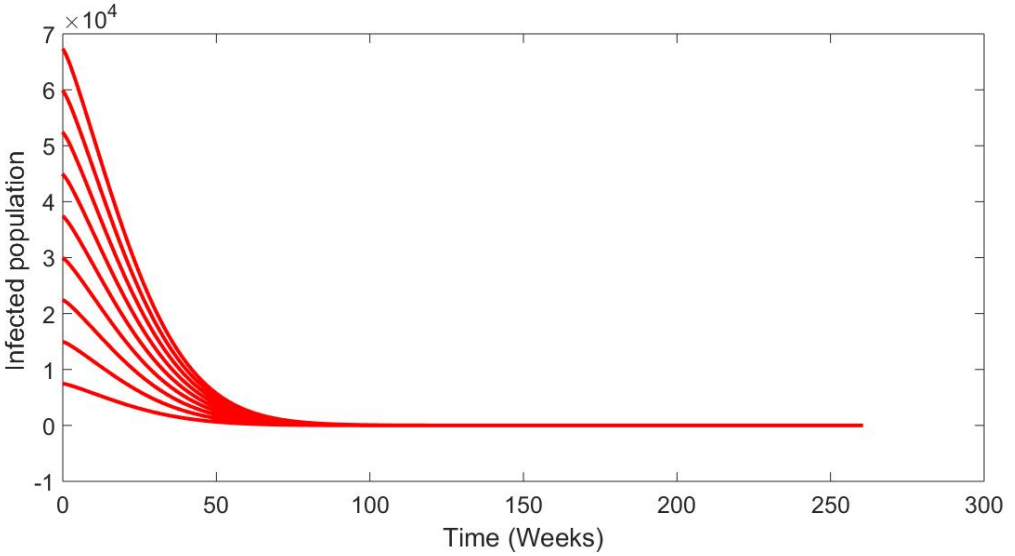

Figure 2. Convergence of solution trajectories when $R_0<1$

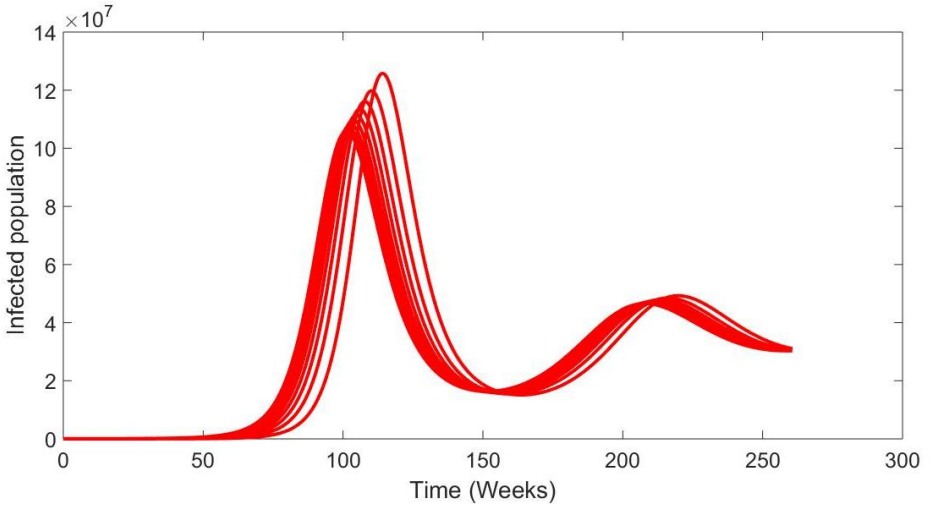

Figure 3. Convergence of solution trajectories when $R_0>1$

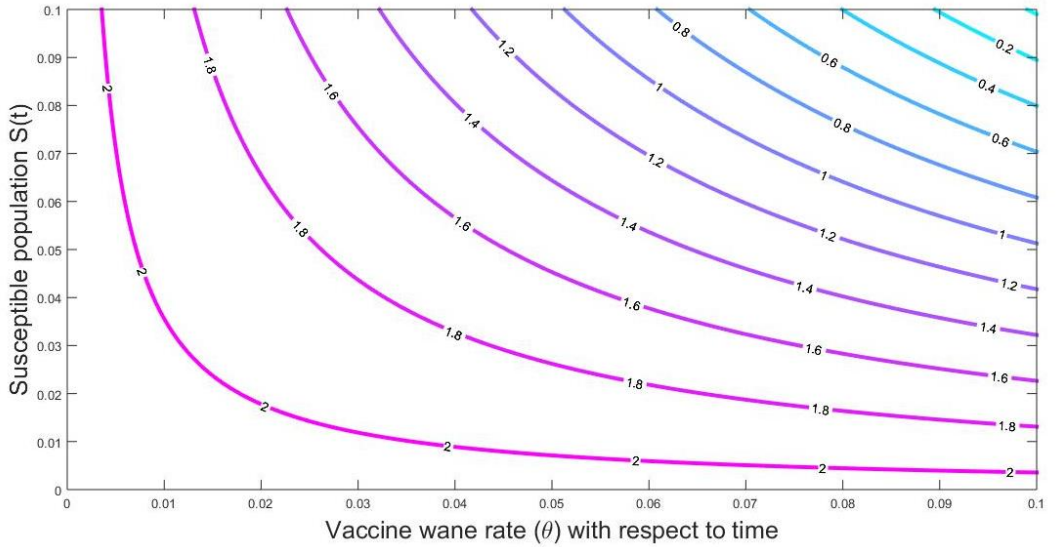

Figure 4. Effects of vaccine wane rate θ on susceptible S(t) with respect to time

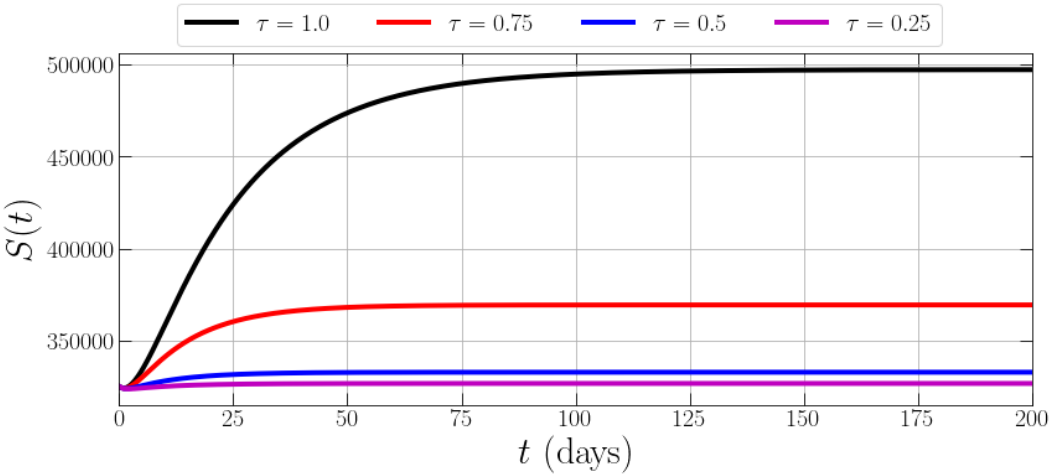

Figure 5. Effect of vaccination rate τ on susceptible population

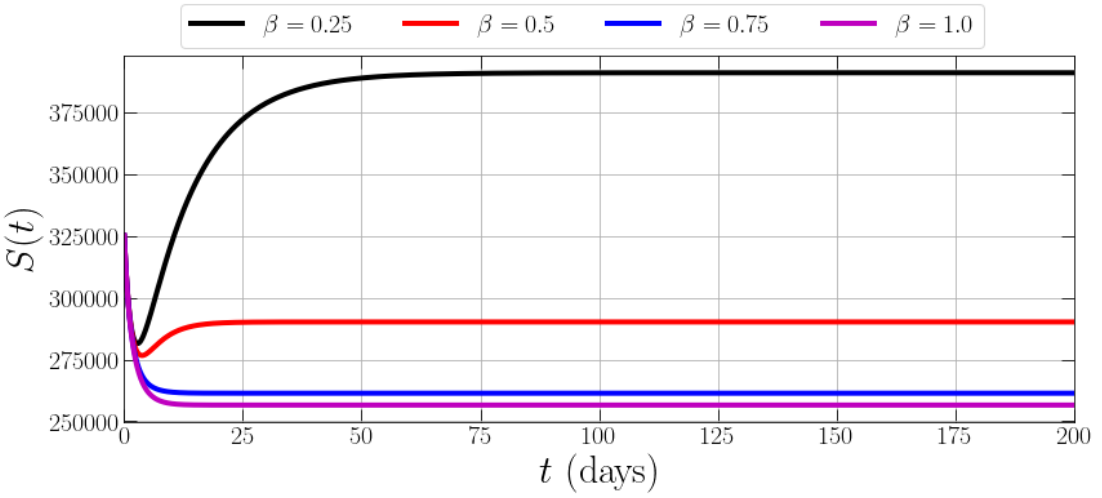

Figure 6. Effect of transmission rate β on susceptible population

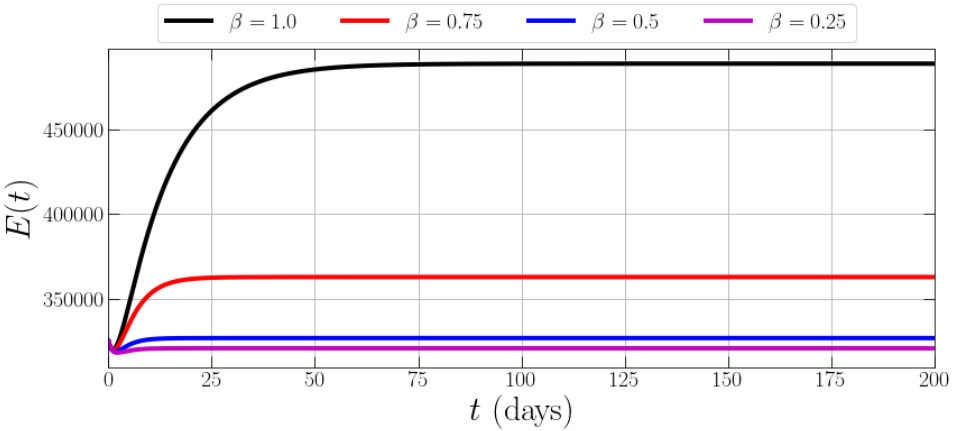

Figure 7. Effect of transmission rate β on exposed population

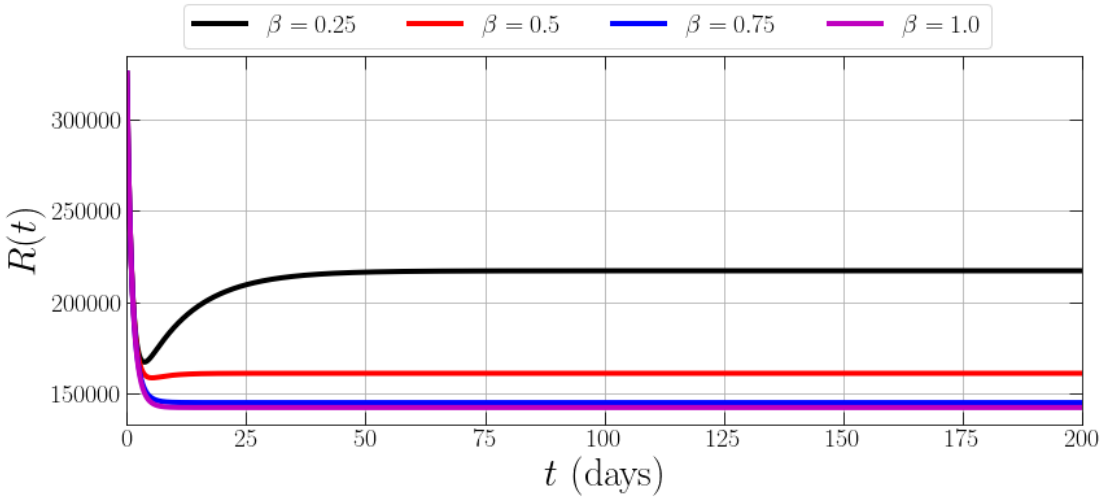

Figure 8. Effect of transmission rate β on recovered population

5. Discussion

Figure 2. represents the convergence of solution trajectories when R_0 <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 R_0 >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.

6. 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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