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STOCHASTIC STABILITY ANALYSIS OF A TUBERCULOSIS EPIDEMIC MODEL WITH RE-INFECTION

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Abstract. The complicated dynamics of tuberculosis (TB), which are impacted by a number of factors such as transmission, immunization, treatment, and re-infection, continue to be a major worldwide health concern. In this work, we develop and examine a mathematical model of tuberculosis transmission that takes treatment, vaccination, and re-infection into account. There are equilibrium points. The model's fundamental reproduction number, R_0, was determined. The equilibrium points were subjected to both local and global stability evaluations. The research indicates that the endemic equilibrium exhibits a locally and globally stable condition when $R_0 > 1$; conversely, when $R_0 < 1$,the disease-free equilibrium is stable both locally and globally. The system experiences a forward bifurcation. The graphical solutions demonstrated how important it is to include vaccination, treatment, and re-infection in a TB transmission model in order to determine the appropriate thresholds for managing and ultimately reducing this illness in the community.The research indicates that the endemic equilibrium exhibits a locally and globally stable condition when $R_0 > 1$; conversely, when $R_0 < 1$,the disease-free equilibrium is stable both locally and globally.

Keywords: Bifurcation analysis, Mycobacterium, Stability analysis, Tuberculosis.

Mathematical Subject Classification: 26A33, 34C60, 65D05, 34H15

1. Introduction

The bacterium responsible for the infectious disease tuberculosis (TB) is called Mycobacterium tuberculosis. The kidneys, brain, spine, and skin can also be impacted, but the lungs are the primary organ afflicted. When TB germs are present in the body but there are no symptoms, it's referred to as latent tuberculosis. Individuals with inactive TB may get ill at any time due to active TB (3, 14, 16). A severe cough, chest pain, blood in the cough, exhaustion, weight loss, lack of appetite, chills, and fever are all signs of active tuberculosis. Once infected, people who are exposed to or physically close to someone who has tuberculosis are more likely to acquire active tuberculosis. Bacillus Calmette Guerin (BCG) immunization, early diagnosis and treatment of TB patients,

and screening those at high risk can all help prevent TB infection and spread (15,8). Antibiotics including isoniazid, rifampin, pyrazinamide, ethambutol, and streptomycin are typically used to treat tuberculosis (7). TB continues to be the world's largest cause of death, especially in low- and middle-income nations (9). An estimated 10.8 million persons globally contracted tuberculosis in 2023 (6). According to estimates, TB killed 1.25 million people worldwide in 2023, including 167,000 HIV-positive individuals (7). To solve this problem, a number of mathematical models have been created. Oshinubi and associates (12) developed a mathematical model that included therapy and immunization for the TB outbreak in Rwanda and Burundi, two countries in East Africa. According to the model study, the prevalence and impact of tuberculosis on the human population can be decreased by expanding immunization and, in particular, treatment options for those who are affected.

A mathematical model for controlling the epidemiology of tuberculosis was created by Ojo et al. (10,17,18,19,20). When the fundamental reproduction number $R_0 < 1$, the model analysis suggests that the disease-free equilibrium is locally asymptotically stable. Additionally, the requirements for the presence of backward bifurcation were determined and the stability of the endemic equilibrium was investigated. The findings indicate that the population's TB burden can be decreased by lowering effective contact with an infected person and increasing the immunization rate of susceptible persons with high vaccine efficacy.

TB has persisted in the human population in spite of these research studies' efforts. In order to properly manage and combat this disease, a mathematical analysis of TB transmission that takes into account vaccine, treatment, and re-infection is pertinent. This paper is divided into the following sections: In section 2, the model is developed. The equilibrium points are assessed, the fundamental reproduction number is calculated, stability assessments are carried out, and the bifurcation analysis is also illustrated in section 3. In section 4, the model is numerically evaluated and confirmed. Lastly, the study ends with a discussion of the findings and recommendations.

2. Mathematical Model Formulation and Assumptions

The developed model divides the human population N(t) into the following compartments: susceptible individuals S(t), vaccinated individuals V(t), exposed individuals E(t), infectious individuals I(t), treated individuals T(t), and recovered individuals R(t). The total population is represented by $N(t) = S(t) + V(t) + E(t) + I(t) + T(t) + R(t)$. The following presumptions form the basis of the study; since everyone has an equal chance of coming into contact with contagious people and receiving a vaccination or treatment, the population is homogeneous. Because vaccination reduces but does not completely eliminate the risk of contracting tuberculosis, there is partial immunity. It is thought that the recruiting rate π is based on birth. βSI is used to express

the force of infection. The infection force for vaccinated individuals is given as $\beta(1-\alpha)$VI because the vaccinated individuals can contract the disease through interaction within the factious TB class at a rate of $(1-\alpha)$. The parameter ζ indicates the vaccination wane rate σ and defines the vaccination rate. Every compartment has a natural mortality rate k. People who have been exposed are moving into the infectious class at a rate of τ. In the meantime, the treatment rate is represented by φ, while the disease-induced death rate is ϵ. The treatment failure rate is represented by γ, and the movement rate from the treated class is represented by ξ. It is assumed that the rate $\gamma\xi$ represents the percentage of treated persons that move into the infectious TB class, whereas the other individuals $(1-\gamma)\,\xi$ shift to the exposed TB compartment as a result of treatment failure. The recovery rate of those who have received treatment is represented by parameter υ. The re-infection rate is expressed as o. The following system of ordinary differential equations can be used to depict the dynamics mentioned above:

$$\begin{aligned}
dS(t) &= \Pi + \zeta V - \beta SI - (\sigma + \kappa - o)S,\\
dV(t) &= \sigma S - \beta(1-\alpha)VI - (\zeta + \kappa)V,\\
dE(t) &= \beta SI + \beta(1-\alpha)VI + (1-\gamma)\xi T - (\tau + \kappa)E,\\
dI(t) &= \tau E + \gamma\xi T - (\kappa + \epsilon + \phi)I,\\
dT(t) &= \phi I - (k + \upsilon + \xi)T,\\
dR(t) &= \upsilon T - (\kappa + o)R
\end{aligned} \tag{2.1}$$

2.1. Invariant Region.

Theorem 2.1. *(bound ness) The region will contain every solution to the system of equations (1).* $\Gamma = \left[(S,V,E,I,T,R)\epsilon R_+^6 : 0 \le S+V+E+I+T+R \le \frac{\Pi}{\kappa}\right]$ *for all positive values* $S(0), V(0), E(0), I(0), T(0), R(0) \in R_+^6$.

Proof. Consider

$$N(t) = \left[(S,V,E,I,T,R)\epsilon R_+^6 : 0 \le S+V+E+I+T+R \in \left[0,\ \frac{\Pi}{\kappa}\right]\right]$$

Now, taking the time derivative of N.

$$\begin{aligned}
\frac{dN}{dt} &= \Pi - \kappa(S+V+E+I+T+R)\\
\frac{dN}{dt} &= \Pi - \kappa N\\
\frac{dN}{dt} &\le \Pi - \kappa N\\
\frac{dN}{dt} + \kappa N &\le \Pi
\end{aligned} \tag{2.2}$$

Integrating equation (2) by the integrating factor then;

$$N(t) \leq \tfrac{\Pi}{\kappa} + Ce^{-kt} \tag{2.3}$$
$$N(0) - \frac{\Pi}{\kappa} = C$$

At t=0, It follow that;

$$N \leq \frac{\Pi}{\kappa} + \left[N(0) - \frac{\Pi}{\kappa}\right] e^{-kt} \tag{2.4}$$

Thus, $t \to \infty$;

$$0 \leq N(t) \leq \frac{\Pi}{\kappa}$$
$$\lim t \to \infty N(t) \leq \frac{\Pi}{\kappa} \tag{2.5}$$

The population is bounded for $t \geq 0$ as it can't grow beyond $\frac{\Pi}{\kappa}$.

Thus, all the solutions of the system of equations (1) enter and remain in the region Γ. □

Theorem 2.2. *All solutions [S,V,E,I,T,R] of the system of equations (1) starting in* $S(0), V(0), E(0), I(0), T(0), R(0) \in R_+^6$ *remain positive* $S(0), V(0), E(0), I(0), T(0), R(0) \forall t > 0$ *in the feasible region* Γ.

Proof. From the system of equations (1), picking on s (t), then

$$dS(t) = \Pi + \zeta v - \beta SI - (\sigma + \kappa - o)S, \tag{2.6}$$

Separating variables;

$$\frac{dS}{S} \geq -(\beta I + \sigma + \kappa - o)dt \tag{2.7}$$

Integrating equation (7) gives;

$$InS(t) \geq -(\beta I + \sigma + \kappa - o)t + InC. \tag{2.8}$$

At, $t = 0; lns(0) = Inc \Rightarrow S(0) = C$

Thus,

$$InS(t) \geq -(\beta I + \sigma + \kappa - o)t + InS(0).$$

This can be reduced to

$$S(t) \geq S(0)e^{-(\beta I + \sigma + \kappa - o)t} \tag{2.9}$$

Thus, $S(t) \geq 0 \forall t \geq 0$.

Performing similar process to the other equations of system (1), then;

$$V(t) \geq V(0)e^{-(\beta(1-\alpha)I + \zeta + \kappa)t}$$
$$E(t) \geq E(0)e^{-(\kappa+\tau)t}$$
$$I(t) \geq I(0)e^{-(\kappa+\epsilon+\phi)t}$$
$$T(t) \geq T(0)e^{-(\kappa+\upsilon+\xi)t}$$

$$R(t) \geq R(0)e^{-(\kappa+o)t} \tag{2.10}$$

Therefore, all the solutions of equations of the system of equations (1) remained positive in the feasible bounded region Γ.

□

3. Mathematical Model Investigation

3.1. Disease Free Equilibrium Point. The steady-state solutions for which there is no illness or infection in the population are known as the disease-free equilibrium point, or E^0 (11). The system of equations (1) is equated to zero, and the solutions of S, V, E, I, T, and R are sought in order to determine the disease-free equilibrium point. Since there are no infections, the variables E, I, T, and R are set to zero. E^0 of the model of the system of equations (1) is consequently obtained as

$$\begin{aligned} E^0 &= S(t)^0, V(t)^0, E(t)^0, I(t)^0, T(t)^0, R(t)^0 \\ &= \left(\tfrac{(\zeta+\kappa)\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, \tfrac{\sigma\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, 0, 0, 0, 0\right) \end{aligned}$$

3.2. Endemic Point of Equilibrium. A steady-state solution characterized by a persistent prevalence of diseases, throughout the population is known as endemic equilibrium, or E* (11). Equations (1) are equal to zero in order to obtain the endemic equilibrium point system and its solutions. The following are the solutions, which are indicated by E*:

$$\begin{aligned} S(t)^* &= \frac{(\beta(1-\alpha)I^* + \zeta + \kappa)}{\sigma} \cdot \frac{\sigma\Pi}{(\beta(1-\alpha)I^* + \kappa)(\beta I^* + \sigma + \kappa - o) - \sigma\zeta}, \\ V(t)^* &= \frac{\sigma\Pi}{(\beta(1-\alpha)I^* + \zeta + \kappa)(\beta I^* + \sigma + \kappa - o) - \sigma\zeta}, \\ E(t)^* &= \frac{1}{\tau}\left(\kappa + \epsilon + \phi - \frac{\gamma\xi\Phi}{\kappa + v + \xi}\right), \\ T(t)^* &= \frac{\Phi}{\kappa + v + \xi} I^*, \\ R(t)^* &= \frac{v\Phi}{(\kappa + o)(\kappa + v + \xi)} I^*. \end{aligned} \tag{3.1}$$

3.3. The Basic Reproduction Number. The number of secondary infections caused by a single primary case in an entirely susceptible population during the course of that case's infectious period is known as the basic reproduction number, or R_0(4). The disease's persistence or extinction in the population is determined by this threshold value. The next generation matrix methodology described in Van (13) was used to calculate the R_0.

From the system of equations (1), the associated matrices are;

$$f = \begin{bmatrix} \beta SI + \beta(1-\alpha)VI \\ 0 \end{bmatrix} \tag{3.2}$$

$$v = \begin{bmatrix} (\tau+\kappa)E \\ -\tau E + (\kappa+\varepsilon+\phi)I \end{bmatrix} \tag{3.3}$$

then differentiating f and v in relation to E and I, respectively;

$$F = \begin{bmatrix} 0 & \beta S^0 I + \beta(1-\alpha)V^0 \\ 0 & 0 \end{bmatrix} \tag{3.4}$$

$$V = \begin{bmatrix} \tau+\kappa & 0 \\ -\tau & \kappa+\varepsilon+\phi \end{bmatrix} \tag{3.5}$$

Substituting the values of S^0 and V^0 respectively into the matrix of f;

$$F = \begin{bmatrix} 0 & \beta\frac{(\zeta+\kappa)\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} + \beta(1-\alpha)\frac{\sigma\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} \\ 0 & 0 \end{bmatrix} \tag{3.6}$$

The inverse of V was computed and obtained as follows;

$$V^{-1} = \begin{bmatrix} \frac{1}{\tau+\kappa} & 0 \\ \frac{\tau}{(\tau+\kappa)(\kappa+\varepsilon+\phi)} & \frac{1}{\kappa+\varepsilon+\phi} \end{bmatrix} \tag{3.7}$$

Hence,

$$FV^{-1} = \frac{1}{\kappa+\varepsilon+\phi}\begin{bmatrix} \frac{\prod\beta\tau((\zeta+\kappa)+(1-\alpha)\sigma)}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} & \frac{\prod\beta((\zeta+\kappa)+(1-\alpha)\sigma)}{((\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma)(\tau+\kappa)} \\ 0 & 0 \end{bmatrix} \tag{3.8}$$

$R_0 = \sigma(M)$is the spectral radius of the matrix FV^{-1} therefore, Hence,

$$R_0 = \frac{\prod\beta\tau((\zeta+\kappa)+(1-\alpha)\sigma)}{((\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma)(\kappa+\varepsilon+\phi)} \tag{3.9}$$

3.4. Local Stability of the Disease –Free Equilibrium (DFE). The stability of the equilibrium point is related to the basic reproduction number R_0 of the model.

Theorem 3.1. *The disease free equilibrium point* $E^0 = \left(\frac{(\zeta+\kappa)\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, \frac{\sigma\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, 0, 0, 0, 0\right)$ *of the system of equations (1) is locally asymptotically stable when* $R_0 < 1$ *and unstable when* $R_0 > 1$ *for any* $t \geq 0$.

Proof. The Jacobian matrix of the system of equations (1) was given by;

$$J = \begin{bmatrix} -\beta I^0 - d & \zeta & 0 & -\beta S^0 & 0 & 0 \\ \sigma & \psi I^0 - (\tau + \kappa) & 0 & \psi V^0 & 0 & 0 \\ \beta I & \psi I^0 & -(\tau + \kappa) & \beta S^0 + \psi V^0 & \delta\xi & 0 \\ 0 & 0 & \tau & -(\kappa + \varepsilon + \phi) & \gamma\xi & 0 \\ 0 & 0 & 0 & \phi & -(\kappa + \upsilon + \xi) & 0 \\ 0 & 0 & 0 & 0 & \upsilon & -e \end{bmatrix} \tag{3.10}$$

Where, $d = (\sigma + \kappa - o),\ e = (\kappa + o),\ \beta(1-\alpha) = \psi$ and $(1-\gamma) = \delta$. Analyzing matrix (19) at the DFE,

$$\begin{aligned} E^0 &= S(t)^0, V(t)^0, E(t)^0, I(t)^0, T(t)^0, R(t)^0 \\ &= \left(\tfrac{(\zeta+\kappa)\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, \tfrac{\sigma\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, 0, 0, 0, 0 \right) \end{aligned} \tag{3.11}$$

$$J = \begin{bmatrix} -d & \zeta & 0 & -\beta \frac{(\zeta+\kappa)\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} & 0 & 0 \\ \sigma & -(\zeta+\kappa) & 0 & -\psi \frac{\sigma\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} & 0 & 0 \\ \beta I & \psi I^0 & -(\tau+\kappa) & \frac{\beta((\zeta+\kappa)\Pi)+\psi(\rho\Pi)}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} & \delta\xi & 0 \\ 0 & 0 & \tau & -(\kappa+\epsilon+\phi+) & \gamma\xi & 0 \\ 0 & 0 & 0 & \phi & -(\kappa+\upsilon+\xi) & 0 \\ 0 & 0 & 0 & 0 & \upsilon & -e \end{bmatrix} \tag{3.12}$$

Where, $d = (\sigma + \kappa - o),\ e = (\kappa + o),\ \beta(1-\alpha) = \psi$ and $(1-\gamma) = \delta$.

The reduced matrices are examined as follows after splitting the matrix because it is block lower triangular and its Eigen values are the union of the Eigen values of the three main diagonal blocks. The uninfected subsystem block (S, V) is a 2x2 matrix with the following Eigen values:

$$A_u = \begin{bmatrix} -d & \zeta \\ \sigma & -(\zeta + \kappa) \end{bmatrix} \tag{3.13}$$

The characteristic equation of the matrix (21) is obtained by finding its determinant as follows;

$$A_u = \begin{vmatrix} -d - \lambda & \zeta \\ \sigma & -(\zeta + \kappa) - \lambda \end{vmatrix} = 0 \tag{3.14}$$

The polynomial can be given as

$$\lambda^2 + (d + \zeta + \kappa)\lambda + d(\zeta + \kappa) - \zeta\sigma = 0$$

All Eigen values are negative if and only if $d(\eta + \mu) > \eta\rho$as shown;

$$\lambda = \frac{-(d + \zeta + \kappa) \pm \sqrt{(d + \zeta + \kappa)^2 - 4[d(\zeta + \kappa) - \zeta\sigma]}}{2}$$

The recovered compartment R

Consider; $\dot{R} = -eR$,

Hence the Eigen value is $\lambda = -e < 0$
The infected subsystem (E, I, T)
The critical block governing infection dynamics is;

$$A_i = \begin{bmatrix} -(\tau+\kappa) & \frac{\beta((\zeta+\kappa)\Pi)+\psi(\sigma\Pi)}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma} & \delta\xi \\ \tau & -(\kappa+\epsilon+\phi) & \gamma\xi \\ 0 & \phi & -(\kappa+\upsilon+\xi) \end{bmatrix} \tag{3.15}$$

Matrix (24) yields:

$$tr(A_i) = (\tau + 3\kappa + \varepsilon + \phi + \upsilon + \xi) \tag{3.16}$$

If 3.16 is negative, then

$$\begin{aligned} det(A_i) = [-(\tau+\kappa)]\,[-(\kappa+\varepsilon+\varphi)(\kappa+\upsilon+\xi) - \phi\gamma\upsilon\xi] \\ - \left(\delta\xi\tau\phi + \frac{\beta((\zeta+\kappa)\Pi)+\psi(\sigma\Pi)}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}\right).\tau[-(\kappa+\upsilon+\xi)] + \delta\xi\tau\phi = 0 \end{aligned} \tag{3.17}$$

If 3.16 is positive, then

$$\frac{\beta((\zeta+\kappa)\Pi)+\psi(\rho\Pi)}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}.\tau(\kappa+\upsilon+\xi) > [(\tau+\kappa)]\,[-(\kappa+\varepsilon+\varphi)(\kappa+\upsilon+\xi) - \phi\gamma\upsilon\xi] \tag{3.18}$$

Therefore; for $R_0 < 1$ all the Eigen vales are negative implying that the DFE is locally asymptotically stable and unstable otherwise.

□

3.5. Local Stability of the Endemic Equilibrium.

Theorem 3.2. *The endemic equilibrium point E*of the system of equations (1) is locally asymptotically stable whenever $R_0 > 1$.*

Proof. The Jacobian matrix of the system of equations (1) at the endemic equilibrium point
$E^* = \left(\frac{(\zeta+\kappa)\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, \frac{\sigma\Pi}{(\sigma+\kappa-o)(\zeta+\kappa)-\zeta\sigma}, 0, 0, 0, 0\right)$

$$J_{EE} = \begin{bmatrix} -\beta I^* - d & \zeta & 0 & -\beta S^* & 0 & 0 \\ \sigma & \psi I^* - g & 0 & \psi V^* & 0 & 0 \\ \beta I^* & \psi I^* & -(\tau+\kappa) & \beta S^* + \psi V^* & \delta\xi & 0 \\ 0 & 0 & \tau & -(\kappa+\upsilon+\xi) & \gamma\xi & 0 \\ 0 & 0 & 0 & \phi & -(\kappa+\upsilon+\xi) & 0 \\ 0 & 0 & 0 & 0 & \upsilon & -e \end{bmatrix} \tag{3.19}$$

Where, $d = (\sigma+\kappa-o)$, $e = (\kappa+o)$, $g = (\kappa+\xi)$, $\beta(1-\alpha) = \psi$ and $(1-\gamma) = \delta$.

Since J_{EE} is blocking lower triangular two Eigen values are directly obtained from the bottom –right 2x2 blocks:

$$\lambda_5 = -(\kappa + \upsilon + \xi),\ \lambda_6 = -(\kappa + o)$$

Which are always negative for positive parameter values.

The stability of E^* thus depends only on the Eigen values of the 4x4 subsystem.

$$A = \begin{bmatrix} -\beta I^* - d & \zeta & 0 & -\beta S^* \\ \sigma & -\beta(1-\alpha)I^* - (\zeta+\kappa) & 0 & -\beta(1-\alpha)V^* \\ \beta I^* & -\beta(1-\alpha)I^* & -(\tau+\kappa) & \beta S^* + \beta(1-\alpha)V^* \\ 0 & 0 & \tau & -e \end{bmatrix} \tag{3.20}$$

□

From matrix(29)

$$Trace(A) = -(\beta I^* + \sigma + \kappa - \zeta) - (\beta(1-\alpha)I^* + \zeta + \kappa) - (\tau + \kappa) - (\kappa + \varepsilon + \phi) \tag{3.21}$$

$$Trace(A) = -(4\kappa + \sigma + \zeta + \tau + \epsilon + \phi - o) - (-\beta I^*(2-\alpha)) \tag{3.22}$$

The determinant of A can be computed as follows;

$$\begin{aligned} A = (\kappa+\epsilon+\phi) det \begin{bmatrix} \beta I^* + \sigma + \kappa - o & -\zeta & -\beta S^* \\ -\sigma & \beta(1-\alpha)I^* + \zeta + \kappa & -\beta(1-\alpha)V^* \\ -\beta I^* & -\beta(1-\alpha)I^* & -(\beta S^* + \beta(1-\alpha)V^*) \end{bmatrix} \\ + \tau det \begin{bmatrix} \beta I^* + \sigma + \kappa - o & -\zeta & -\beta S^* \\ -\sigma & \beta(1-\alpha)I^* - (\tau+\kappa) & \beta(1-\alpha)V^* \\ -\beta I^* & -\beta(1-\alpha)I^* & -(\beta S^* + \beta(1-\alpha)V^*) \end{bmatrix} \end{aligned} \tag{3.23}$$

Hence the determinant of a can be given as

$$\begin{aligned} det(A) = (\kappa+\epsilon+\phi)(\tau+\kappa)\left(\beta^2(1-\alpha)I + \beta I^*(\zeta+\kappa) + (\sigma+\kappa-o)(\zeta+\kappa) - \zeta\sigma\right) \\ -\tau\left((\beta I^* + \sigma + \kappa - o)\beta(1-\alpha)I^* + (\zeta+\kappa)(\beta S^* + \beta(1-\alpha)V^*)\right) \\ -\left((\beta I^* + \zeta + k - o)\beta^2(1-\alpha)^2 I^* V^* - \zeta\sigma(\beta S^* + \beta(1-\alpha)V^*)\right) \\ -\zeta\beta^2(1-\alpha)I^*V^* - \sigma\beta^2(1-\alpha)I^*S^* - \beta^2 I^* S^*\left(\beta(1-\alpha)I^* + \zeta + \kappa\right) \end{aligned} \tag{3.24}$$

From expression (33): $det(A) > 0$

Provided that $(\kappa+\epsilon+\phi)\Omega > \tau\kappa$

Where

$\Omega = -\beta^2(1-\alpha)I^{*2} + \beta I^*(\zeta+\kappa)I^*S^* + (\sigma+\kappa-o)\beta(1-\alpha)I^* + (\sigma+\kappa-o))(\zeta+\kappa) - \kappa\sigma$

And

$$\begin{aligned}\kappa = &((\beta I^* + \sigma + \kappa - o)\beta(1-\alpha)I^* + (\zeta+\kappa)(\beta S^* + \beta(1-\alpha)V^*)) \\ &- \big((\beta I^* + \zeta + k - o)\beta^2(1-\alpha)^2 I^* V^* - \zeta\sigma(\beta S^* + \beta(1-\alpha)V^*)\big) \\ &-\zeta\beta^2(1-\alpha)I^*V^* - \sigma\beta^2(1-\alpha)I^*S^* - \beta^2 I^* S^* \left(\beta(1-\alpha)I^* + \zeta + \kappa\right)\end{aligned}$$

The local asymptotic stability of E^* for the specified parameter values is confirmed by the fact that all Eigen values have negative real components. When $R_0 > 1$, the endemic equilibrium is present and asymptotically stable locally. This suggests that the system will eventually return to the endemic equilibrium as minor disturbances around the endemic state diminish.

3.6. Global Stability Analysis of the Disease Free Equilibrium. The technique by Castillo et. al. (2) is used to analyze the global stability.

Theorem 3.3. *Theorem 3.3 the DFE is globally asymptotically stable in the feasible region gamma whenever* $R_0 < 1$.

Proof. The model is partitioned as:

$$\begin{aligned}\frac{dX}{dt} &= F(X, 0), \\ \frac{dy}{dt} &= B(X, Y),\end{aligned}$$

□

3.6.1. Dynamical of Uninfected Subsystem. When $y = 0$ the subsystem

$$\begin{aligned}\frac{dX}{dt} &= F(X, 0), \\ dS(t) &= \Pi + \xi V - (\sigma + \kappa - o)S, \\ dV(t) &= \sigma S - (\zeta + o)V,\end{aligned}$$

$$dR(t) = -(\kappa + o)R \tag{3.25}$$

The system is linear with recruitment vaccination, loss of immunity and natural death only

This system can be written in matrix form as:

$$\frac{dX}{dt} = AX + b$$

$$A = \begin{bmatrix} -d & \zeta & 0 \\ \sigma & -(\zeta+\kappa) & 0 \\ 0 & 0 & -e \end{bmatrix} \tag{3.26}$$

$$b = \begin{bmatrix} \Pi \\ 0 \\ 0 \end{bmatrix} \tag{3.27}$$

Where $d = \sigma + \kappa - o$ and $\rho = \kappa + o$ at equilibrium, setting:

$$0 = AX_0 + b$$

This yields the system of equations:

$$-dS_0 + \zeta V^0 + \Pi = 0$$

$$\begin{aligned} \sigma S_0 - (\zeta + \kappa)V_0 = 0 \\ -eR_0 = 0 \end{aligned} \tag{3.28}$$

From an additional equation from a system of equations (34), it follows that:

$$R_0 = 0 \tag{3.29}$$

With second equation for system of equations (34) solving for

$$V_0 = \frac{\sigma}{\zeta + \kappa} S_0 \tag{3.30}$$

Substituting equation (39) into the initial equation in the system of equations (34) yields:

$$dS_0 + \zeta(\frac{\sigma}{\zeta + \kappa} S_0) + \Pi = 0$$

Thus:

$$S_0 = \frac{\Pi(\zeta + \kappa)}{(\sigma + \kappa - o)(\zeta + \kappa) - \zeta\sigma} \tag{3.31}$$

Finally, substituting back for

$$V_0 = \frac{\sigma}{\zeta + \kappa} S_0 = \frac{\sigma\Pi}{(\sigma + \kappa - o)(\zeta + \kappa) - \zeta\sigma} \tag{3.32}$$

Consequently, equations (38), (40), and (41) represent the unique steady state. All of matrix A's Eigen values have negative real parts since it is a triangle with strictly negative diagonal entries $-(\sigma + \kappa - o), -(\xi + \kappa), -(\kappa + o)$ The global asymptotic stability of the uninfected subsystem in the absence of infection is thus demonstrated by the fact that every solution of the uninfected subsystem converges to this special disease-free equilibrium. As a result, a unique global asymptotic stable steady state is admitted by the subsystem.

3.6.2. Dynamics of Infected Subsystem. The infected subsystem can be written as:

$$\frac{dY}{dt} = AY - B(X, Y)$$

Where, A is an m- matrix with non-negative off-diagonal elements.

$$\begin{aligned} dE(t) = \beta SI + \psi VI + \delta\xi T - (\tau + \kappa)E, \\ dI(t) = \tau E + \gamma\xi T - (\kappa + \varepsilon + \phi)I \\ dT(t) = \phi I - (\kappa + \upsilon + \xi)T \end{aligned} \tag{3.33}$$

Writing the system of equations (42) in matrix form;

$$A = \begin{bmatrix} -(\tau+\kappa) & 0 & \delta\xi \\ \tau & -(\kappa+\varepsilon+\phi) & \gamma\xi \\ 0 & \phi & -(\kappa+\gamma+\xi) \end{bmatrix} \tag{3.34}$$

$$B(X,Y) = \begin{bmatrix} (\beta SI + \psi VI) \\ 0 \\ 0 \end{bmatrix}$$

and

Matrix A describes the linear transitions among infected compartments and has negative diagonal entries;

$-(\tau+\kappa) \quad -(\kappa+\varepsilon+\phi) \quad -(\kappa+\gamma+\xi)$

These ensure that in absence of new infections, any perturbation decays exponentially to zero.

Observe that B(X, Y) is non-negative $\forall y \geq 0$. With $\psi = \beta(1-\alpha)$ and $0 \leq \alpha < 1$.

This implies that $0 \leq \psi < \beta$.

$$b_1 = \beta SI + \psi VI$$

$$\beta \quad greaterthan \;\; zero$$

$$S, V, I \& \psi greaterthanorequaltozero$$

Each term satisfies: $\beta SI \geq zero, \psi VI \geq zero$

Therefore:

$$b_1 \geq 0$$

$$b_2 = 0$$

$$b_3 = 0$$

Clearly non-negative. Thus $\forall y \geq 0$:

$$B(X,Y) \leq 0.$$

$$B(X,0) = 0 \; for \; Y = 0$$

Next

$$B(X,Y) = \begin{bmatrix} (\beta SI + \psi VI) \\ 0 \\ 0 \end{bmatrix} \tag{3.35}$$

If $y = 0$, then in particular:

$$I = 0 \tag{3.36}$$

$$b_1 = (\beta S.0 + \psi V.0)$$

$$b_2 = 0$$

$$b_3 = 0$$

$$B(X,Y) = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix} \tag{3.37}$$

This confirms that in absence of infection, no new infections are generated. Since $B(X,Y)$ is nonnegative and vanishes when $y = 0$, the unforced dynamics always decay:

$$\frac{dY}{dt} \leq AY. \tag{3.38}$$

As a result, when $R < 1$, the linear section A is stable and there is no endemic equilibrium in the infected subsystem. Using the comparison theorem and conventional arguments in monotone dynamical systems, as shown in (1)

This implies:

$$\lim t \to \infty Y(t) = 0 \tag{3.39}$$

Indeed, the spectral radius of the next generation matrix satisfies:

$$\rho(FV^{(-1)}) = R_0 < 1$$

Therefore any initial infection will eventually vanish, and the infected subsystem converges to the disease – Free State when $R_0 < 1$

Thus, the DFE is globally asymptotically stable in the feasible region Γ whenever $R_0 < 1$.

3.7. Global Stability Analysis of the Endemic Equilibrium.

Theorem 3.4. *The endemic equilibrium point is globally asymptotically stable in the feasible region* Γ *whenever* $R_0 > 1$

Proof. The model is partitioned as:

$$\frac{dX}{dt} = F(x,y),\ \frac{dY}{dt} = G(x,y) \tag{3.40}$$

□

3.7.1. Global Stability of the Uninfected Subsystem (C1). Setting E=I=T=0 the uninfected subsystem becomes;

$$\begin{aligned} dS(t) &= \Pi + \zeta V - (\sigma + \kappa - o)S, \\ dV(t) &= \sigma S - (\zeta + \kappa)V, \\ dR(t) &= 0. \end{aligned} \tag{3.41}$$

Analyzing the subsystem of equations (47) at(S, V) as

$$\begin{aligned} dS(t) &= \Pi + \zeta V - \chi S, \\ dV(t) &= \sigma S - \lambda V, \end{aligned}$$

Where $\chi = \sigma + \kappa - o$ and $\lambda = \zeta + \kappa$.

Setting the derivations in system (48) to zero:

$$0 = \Pi + \zeta V - \chi S,$$
$$0 = \sigma S - \lambda V,$$

Since $dR(t) = 0$ in absence of infection, r remains constant.
Solving for the steady state:

$$V^* = \frac{\sigma}{\lambda} S*,$$
$$S^* = \frac{\Pi}{\chi - \frac{\zeta\sigma}{\lambda}}$$

Provided that

$$\chi* \geq \frac{\zeta\sigma}{\lambda}$$

The Jacobian matrix is:

$$\begin{bmatrix} -\chi & \zeta \\ \sigma & -\lambda \end{bmatrix} \tag{3.42}$$

With the characteristic equation:

$$\lambda^2 + (\chi + \lambda)\lambda + (\chi\lambda - \zeta\sigma) = 0$$

The roots have negative real parts if:

$$Tr(J) = -\chi - \lambda < 0 \;\; det(J) = \chi\lambda - \zeta\sigma > 0. \tag{3.43}$$

As a result, the system is linear and bounded, thus the equilibrium $(S^*, V^*)$is locally asymptotically stable. Therefore, this equilibrium is also globally asymptotically stable in the domain Γ.

3.7.2. Global Stability of the Infected Subsystem (C2). The infected subsystem is:

$$dE(t) = \beta SI + \psi VI + \delta\xi T - (\tau + \kappa)E,$$
$$dI(t) = \tau E + \gamma\xi T - (\kappa + \varepsilon + \phi)I$$
$$dT(t) = \varphi I - (\kappa + v + \xi)T.$$

Define:

$$G(X, Y) = G(X, Y) - G(X, 0).$$

Jacobian of G(X,Y) with respect to y= (E, I, T) is:

$$\begin{bmatrix} -(\tau + \kappa) & \beta S + \xi I & \delta\xi \\ \tau & -(\kappa + \varepsilon + \phi) & \gamma\xi \\ 0 & \psi & -(\kappa + \gamma + \xi) \end{bmatrix} \tag{3.44}$$

Since all of the off-diagonal entries of matrix (53) are non-negative, matrix (53) can be referred to as a Metzler matrix.

System (52) is irreducible as by eliminating the flow of influence among E, I, T the interaction graph contains the cycle $E \to I \to T \to E$

And also the directed edge $T \rightarrow I$ finally system (52) is verified to be cooperative as the elements in the off diagonal position are clearly positive. Since conditions (C1) and (C2) are satisfied, then the endemic equilibrium

$E^* = (S^*, V^*, E^*, I^*, T^*, R^*)$of the tuberculosis model is globally asymptotically stable in the feasible region Γ.

3.8. Bifurcation Analysis. The infected equations are divided into a linear term and a nonlinear remainder in bifurcation analysis. Consequently, the infected subsystem, using the Center Manifold theorem;

$$\frac{dY}{dt} = AY + f(Y, \beta)$$

Near the DFE the dominant infected system is

$$\begin{aligned} dE(t) &= \beta SI + \psi VI + \delta\xi T - (\tau + \kappa)E, \\ dI(t) &= \tau E + \gamma\xi T - (\kappa + \varepsilon + \phi)I + \gamma\zeta T, \\ dT(t) &= \phi I - (\kappa + \upsilon + \xi)T \end{aligned} \tag{3.45}$$

$$A = \begin{bmatrix} -(\tau+\kappa) & F & \delta\xi \\ \tau & -(\kappa+\varepsilon+\phi) & \gamma\xi \\ 0 & \psi & -(\kappa+\upsilon+\xi) \end{bmatrix} \tag{3.46}$$

Where

$$a = \sum_{(k,j,i=1)}^{n} v_k w_i w_j \frac{\partial^2 f_1}{\partial y_i \partial y_j}(0,0), \quad b = \sum_{(k,i=1)}^{n} v_k w_i \frac{\partial^2 f_1}{\partial y_i \partial \beta}(0,0)$$

Hence

$$a = v_1 w_I w_I \frac{\partial^2 f_1}{\partial I^2} = v_1 w_2 w_2(-2\beta c)b = v_1 w_1 \frac{\partial^2 f_1}{\partial I \partial \beta} = v_2 w_2 s_0$$

Computing:

$$a = v_1 w_2 w_2(-2\beta c) < 0, \quad b = v_2 w_2 s_0 > 0$$

This verified a forward bifurcation at $R_0 = 1$ since $a < 0$ and $b > 0$. Infection rises as the transmission rate rises. Therefore, when $R_0 > 1$, the endemic equilibrium appears smoothly. As a result, the bifurcation analysis suggested that in order to ensure disease eradication, control methods should concentrate on making sure that R_0 stays below one. Furthermore, this threshold value is susceptible to changes in vaccination and treatment compliance due to the potential for re-infection and incomplete vaccination.

4. Numerical Simulations

The parameter values in the simulations were mainly derived from (1; 5; 6; 10). The other values $\Pi = 10, \kappa = 0.0079, \alpha = 0.6, \epsilon = 0.02, \xi = 0.03 and \ o = 0.005$ were used. Three scenarios based on the value R_0 and other five control strategy scenarios were considered in order to examine the system's response under different transmission intensities:

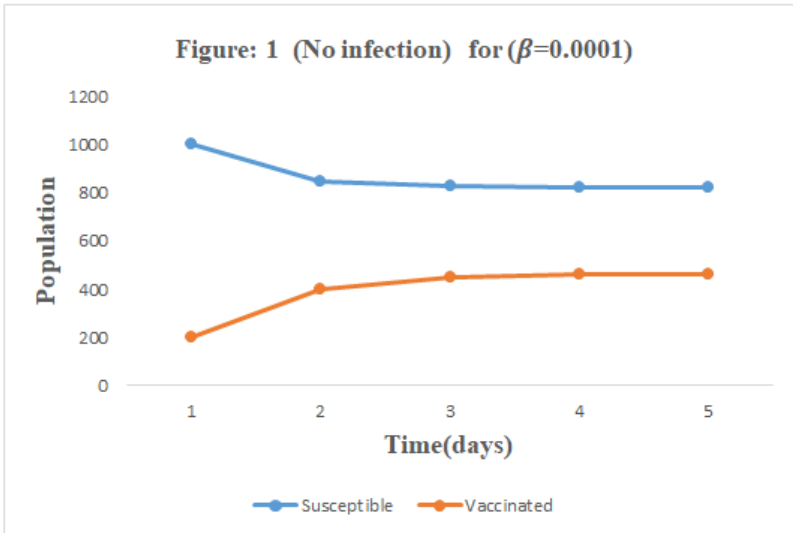
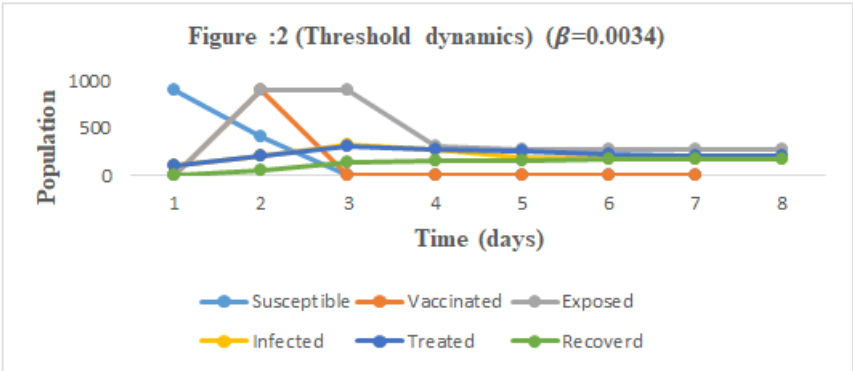
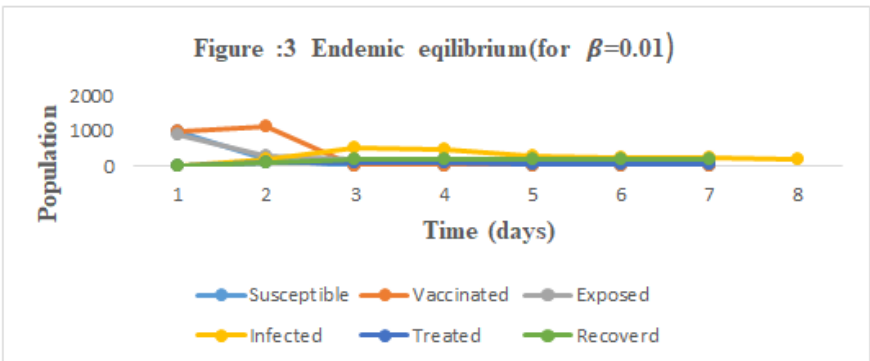


Figure 1 shows that as people become protected through vaccination, the number of susceptible people initially decreases. The curve illustrates how the population in the susceptible subgroup stabilizes with time. The number of vaccinated people rises in the interim, but this eventually levels out. Since there are no infections, the disease-free equilibrium is asymptotically stable both locally and globally when $R_0 < 1$.

Figure 2 suggests that as people receive vaccinations, their susceptibility initially declines. After being exposed, they get an infection. After a slow increase, the infectious compartment steadily levels out. As infectious persons receive treatment, the number of treated individuals rises before stabilizing. Recoveries are steadily increasing as a result of treatment. As a result, the system experiences a trans critical bifurcation for $R_0 = 1$. The stability of the disease-free equilibrium shifts as R_0 approaches one, indicating that the disease does not go extinct right away. This is due to the system reaching a steady state that represents the key threshold when the inflection neither completely vanishes nor increases exponentially.

Figure 3 shows that as more people are exposed following vaccination, the population in the susceptible class rapidly decreases. However, because of the

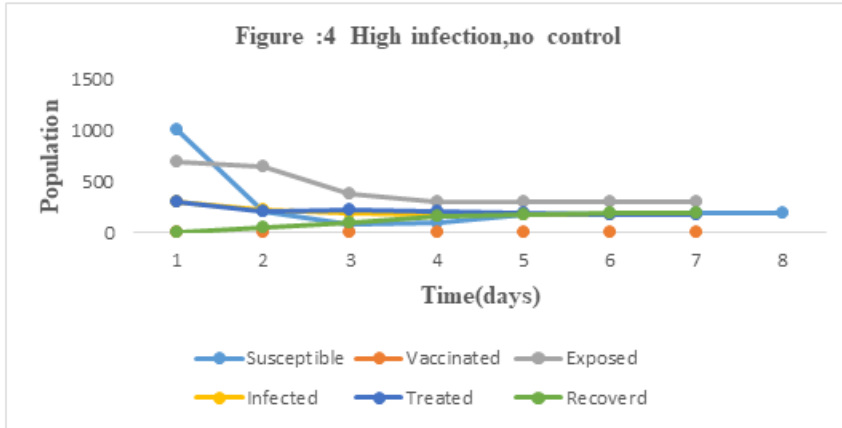


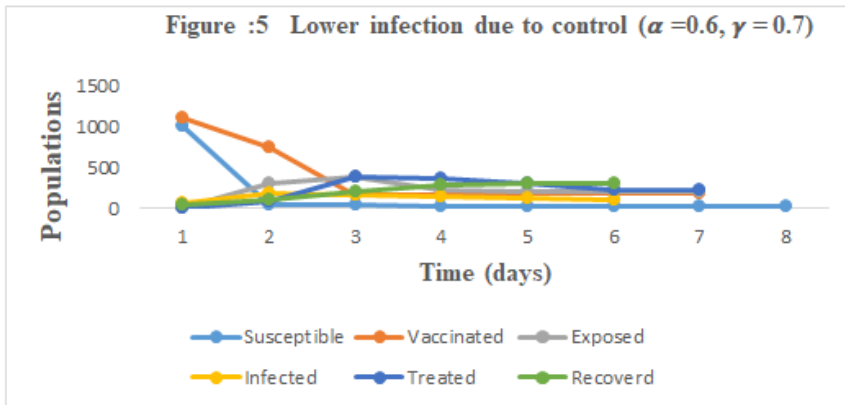


increased contact rate, these people become infected very quickly, which leads to a higher epidemic level. As people receive treatment, recovery rates also steadily increase, but eventually stabilize. As a result, the disease endures in the population and reaches an endemic equilibrium when $R_0 > 1$.

According to Figure 4, the number of individuals in the susceptible compartment declines when vaccination is not taken into consideration as a control approach, and those individuals then immediately enter the exposed class. As they enter the sick class, the number of exposed individuals likewise declines. Over time, the number of recoveries rises as a result of treating these individuals. Because vaccination is a control approach that provides protection, the number of recovered individuals does not increase considerably.

The rising curve in Figure 5 illustrates how the number of recovered individuals tends to rise when high vaccine efficacy and treatment default rates are taken into account. However, effective vaccine and treatment success rates cause the number of illnesses to decrease over time, suggesting the significance of the two control techniques. Lower vaccine efficacy is depicted in Figure 6,

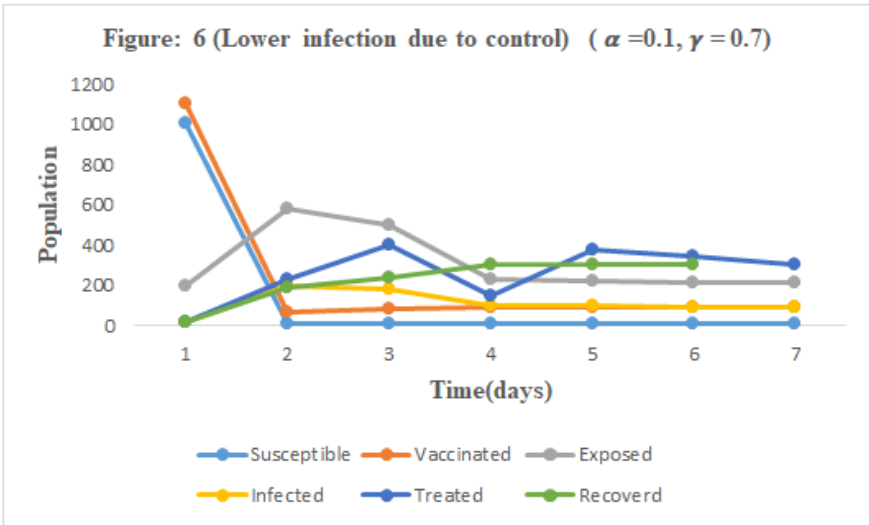


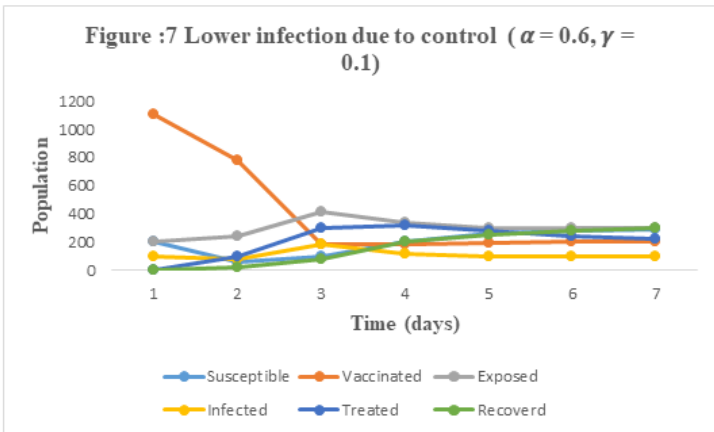


which raises concerns about the safety of those who have recovered. This suggests that the treatment success rate by itself is insufficient since it causes the number of infectious individuals to increase relative to those in the vaccinated compartment.

Figure 7 makes it clear that inadequate treatment causes a sharp decline in the number of people who recover. Therefore, to ensure a steady increase in the number of recoveries, both immunization and treatment must be effective.

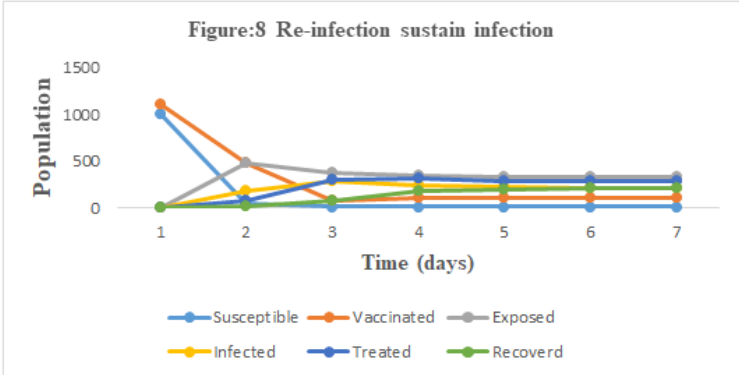
The effects of re-infections taken into account concurrently with the other parameters are depicted in Figure 8. Re-infection lowers recovery rates, which raises the number of exposed and subsequently infected people.





5. Conclusions

To investigate the dynamics of tuberculosis (TB) transmission in a community, a deterministic model was created. The investigation confirmed the biological relevance of the model by demonstrating that all solutions stay positive and limited inside the feasible zone. The next-generation matrix approach was used to determine the basic reproduction number R_0 and to obtain the disease-free equilibrium (DFE) and endemic equilibrium (EE).It was found that whereas $R_0 > 1$ guarantees persistence, $R_0 < 1$ results in the eradication of illness. The DFE is locally and globally asymptotically stable when $R_0 < 1$,



but the EE is stable when $R_0 > 1$, according to stability analysis using the Jacobian matrix. At $R_0 = 1$, a forward bifurcation takes place. The significance of immunization, treatment, and preventing reinfection in limiting the spread of tuberculosis was emphasized using numerical simulations.

6. Future Study

In order to ensure high vaccine efficiency, which can significantly reduce the susceptible population and lower the overall risk of illness, the model suggests increasing coverage. To promote early detection, treatment compliance, and preventive actions, ongoing education and awareness efforts are essential. Concentrating on stochastic modelling, this may readily represent erratic variations in tuberculosis transmission, particularly in low-incidence areas or small populations.

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