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Mathematical Modeling of Mycobacterium Tuberculosis Transmission in Human with Plan for Vaccination after Recovery through Hospital Treatments

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Competing Interests.
The authors declare no competing interests

ABSTRACT

Tuberculosis (TB), a serious public health infection that mainly affects the lungs, is caused by bacteria (Mycobacterium tuberculosis, TB). This research analyzed using a compartmental modelling approach to study the transmission dynamics of TB with different stages of infection. Qualitative analysis of the proposed model reveals that the model exhibits two equilibrium points: the disease-free equilibrium point (DFE) and the endemic equilibrium (EE). The basic reproduction number (R_o) is determined using the next-generation matrix technique, and stability analysis is carried out to show whether the disease can persist or die out in population. With the aid of the sensitivity analysis, we determined the most sensitive parameters of the model to control the spread of TB infection effectively. Our analysis shows that treatment (medication) and campaign awareness coupled with other key control measures, could help bring the spread of MTB infection in human geographical boundaries under control and improve livelihood.

Keywords. Basic reproductive number, Sensitivity analysis, Equilibrium Point

1. Introduction

Tuberculosis (TB) is an infectious disease caused by the Mycobacterium tuberculosis bacillus, remains one of the world's deadliest diseases [36]. According to the World Health Organization (WHO), in 2013, about 9 million people were infected, worldwide, with TB and 1.5 million deaths from the disease were reported, 360,000 of whom were HIV-positive [36]. Tuberculosis is seen to be declining slowly each year and an estimated 37 million lives were saved between 2000 and 2013 through effective diagnosis and treatment (World Health Organization, 2014). On the average, TB incidence fell to about 1.5% per year, between 2000 and 2013, worldwide [36]. Globally, TB mortality rate fell by an estimated 45% between 1990 and 2013 while the prevalence rate dropped by 41% [36].

Even with such positive results achieved within the last 14 years, it is still thought that deaths from tuberculosis are preventable; in fact, the death toll is still considered unacceptably high. Hence, efforts are geared toward accelerating programmes that will result in a reduction in the TB burden globally [within the context of the Millennium Development Goals (MDGs)], and reach the Stop TB Partnership target of a 50% reduction by 2015 [36]. More than half of the approximately 9 million individuals who are infected with tuberculosis in 2013 (56%) were in South-East Asia and Western Pacific Regions. A further one quarter of these infected individuals are in the African Region, which account for the highest rates of TB cases and deaths relative to population [36]. Generally, reducing the incidence and prevalence of TB in a population hinges on successful treatment and high case detection rates. It fact, it is seriously encouraged that effort should be concentrated on ensuring that all TB cases are detected, notified and commence treatment immediately. It is reported that about 6.1 million TB cases were reported to the WHO in 2013 out of which about 5.7 million were individuals were newly diagnosed and another 0.4 million were already on treatment [36]. Tuberculosis notification has stabilized in recent years, with about 64% of the estimated 9 million individuals who developed TB in 2013 were notified as newly diagnosed cases [36]. [8] published a paper titled "A Mathematical Model of a Tuberculosis Transmission Dynamics Incorporating First and Second Line Treatment". Tuberculosis (TB) is a protracted bacterial infectious disease caused by *Mycobacterium tuberculosis* which position

a major health, social and economic burden globally, especially in low and a middle

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country (WHO, 2013). The surge in HIV-TB co -infection and growing emergence of multidrug resistant TB (MDR-TB) and extensively drug resistant TB (XDR-TB) strains has further fueled TB epidemic (WHO,2013; WHO, 2016).

Tuberculosis is a disease which is caused by the bacterium Mycobacterium Tuberculosis. With nearly 3 million deaths each year, tuberculosis has the dubious distinction of being the one and only cause of death from a single infectious agent in the entire world [21]. Analysis of a mathematical model for tuberculosis with diagnosis. Tuberculosis (TB) is a chronic bacterial infectious disease caused by *Mycobacterium tuberculosis* which pose a major health, social and economic burden globally, especially in low- and middle-income countries. It is the second deadliest disease due to a single infectious agent only after HIV/ AIDS. The surge in HIV-TB co-infection and growing emergence of multidrug-resistant TB (MDR-TB) and extensively drug resistant TB (XDR-TB) strains has further fueled TB epidemic. TB usually affects the lungs (pulmonary TB) but it can also affect other sites as well (extra-pulmonary TB) [19]. Analysis of a mathematical model for tuberculosis: What could be done to increase the case detection, Nigeria has been ranked fourth among the 22 countries designated by the WHO as the high-burden countries (HBC) for TB. Nigeria is also said to have the highest number of new TB cases in Africa [36] having about 300,000 of estimated TB cases recorded each year, which result in 30,000 deaths annually. Moreover, the total of the notified cases for TB of all forms increased from 46,473 in 2003 to 59,493 in 2004. According to WHO (2006), the incidences of the detection of smear-positive cases tripled between 1996 and 2004. In 2002, Nigeria had nearly 368,000 new TB cases. Of these, 159,000 were pulmonary sputum smear-positive (SS+) cases. The public health burden posed by TB is becoming increasingly important as the country's HIV epidemic unfolds [26].

Tuberculosis (TB) is a worldwide pandemic disease. According to World Health Organization (WHO), one-third of the world's population is currently infected by the TB bacillus bacteria [28]. Being a disease of poverty, the vast majority of TB deaths are in the developing world with more than half occurring in Asia. The estimated global incidence rate is falling very slowly from the peak of 141 cases per 100,000 population in 2002 to 128 cases per 100,000 population in 2010. The TB death rate has also fallen by 40% since 1990 and the number of deaths is also declining. Globally, the percentage of people successfully treated reached its highest level at 87% in 2009.

Tuberculosis is a bacterial disease with an estimated one third of the world human population as its reservoir [17] . The disease is most commonly transmitted from a person suffering from infectious (active) tuberculosis to susceptible (and possibly latently infected) persons by infected droplets created when the person with active TB coughs or sneezes. Among generally healthy persons, infection with TB is highly likely to be asymptomatic. Data from a variety of sources suggest that the life time risk of developing clinically active TB after being infected is approximately 10% (Hopewell,1994).

Mycobacterium tuberculosis (MTB) is a disease caused by tubercle bacillus or M tuberculosis which belongs to the genus Mycobacterium under the family of Mycobacteriaceae that affect a variety of hosts, including humans, livestock animals (horse, camel, pig) and wild animals (tiger, lion, gorilla), which serve as a reservoir [27].

2. Materials and Methods

The model is a modification of the model proposed by [21]. The total number of population at time, represented by N(t) is categorized in to (5) compartments as follows; : susceptible human, who are at risk of being infected with MTB disease (S(t)), Exposed individuals or latent stage (E(t)), asymptomatic MTB infected individuals with no clinical symptoms of MTB infection (A(t)), MTB infected individuals with clinical symptoms (I (t)), hospitalized individuals, who are under treatment of (H(t)), individuals who recovered from MTB infection (R(t)), and extended the model proposed by [21] by adding a vaccination class suggesting both individuals from susceptible and Recovery class receive vaccination to minimize reinfection rate (V(t)) so that:

$$N(t) = S(t) + E(t) + A(t) + I(t) + H(t) + R(t) + V(t). \quad (1)$$

2.1. Assumption Made in The Formulation of The Model

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There is a natural recovery for the individual who is infected with the diseases.

(i) All population are only recruited in the susceptible class.

(ii) The number of susceptible individuals is reduced through natural death, vaccination and infection due to the virus.

(iii) Individuals that show no clinical symptoms and clinical symptoms can recover as a result of hospital treatment which is denoted by the parameter γ_1 and γ_2 respectively.

(iv) Once an individual recovered, they receive vaccination to minimize reinfection rate due to low immunity denoted by θ.

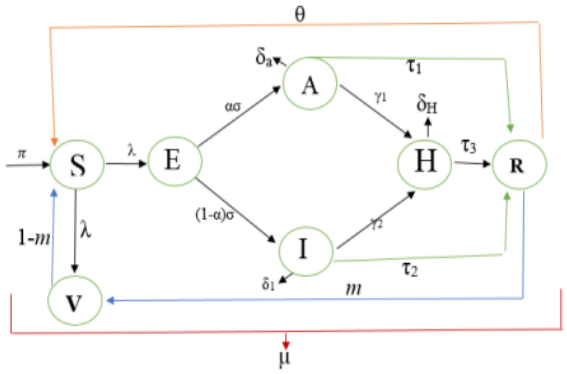

Fig 3.1: schematic diagram of the model

γ_1 = Treatment of no clinical symptoms individuals

γ_2 = Treatment of clinical symptoms individuals

2.2 Model Equation

The model equations for the MTB Dynamic are given as follows;

$$\frac{dS}{dt} = \pi + \theta R + (1-m)V - (\mu - \lambda)s$$

$$\frac{dE}{dt} = \lambda s - (\mu + \sigma)E$$

$$\frac{dA}{dt} = \alpha\sigma E - (\mu + \gamma_1 + \delta_1 + \tau_1)A$$

$$\frac{dI}{dt} = (1-\alpha)\sigma E - (\mu + \gamma_2 + \tau_2 + \delta_2)I \quad (2)$$

$$\frac{dH}{dt} = \gamma_1 A + \delta\gamma_2 I - (\delta_3 + \tau_3 + \mu)\text{H}$$

$$\frac{dR}{dt} = \tau_1 A + \tau_2 I + \tau_3 H - (\theta + m + \mu)R$$

$$\frac{dV}{dt} = mR + \lambda S - (1-m)V - \mu V$$

Where:

$$\lambda = \frac{\beta(\eta_1 E + \eta_2 H + \eta_3 A + \eta_4 V + I)}{N} \quad (3)$$

And β is the effective contact rate, and η_1, η_2 and η_3 are modification for the decrease/ increase of MTB infection.

In susceptible class, human population is recruited through birth or immigration at a level π, diminished to exposed class at rate α and to the vaccination class at the rate *m*.

$$\frac{dS}{dt} = \pi + \theta R + (1-m)V - (\mu - \lambda)s$$

In the exposed class, there is a progression from exposed class to infected class at rate σ, and natural death occur at rate μ

$$\frac{dE}{dt} = \lambda s - (\mu + \sigma)E$$

In the asymptomatic class, there is a progression from vaccinated class to hospitalized class at rate γ_1, the hospitalized individual gets recovered and become susceptible to the disease due to vaccination failure at rate θ and also there is a natural death at rate μ .

$$\frac{dA}{dt} = \alpha\sigma E - (\mu + \alpha_1)A$$

In the recovered class, the individual who got infected with tuberculosis recovered from the disease. There is natural death at rate μ, possibility of returning to the susceptible class

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at the rate of θ, getting vaccination at the rate of m.

$$\frac{dR}{dt} = \tau_1 A + \tau_2 I + \tau_3 H - (\theta + m + \mu)R$$

In the vaccination class there is progression form the susceptible recovery class at the rate of λ and m respectively and possibility of natural death and returning to susceptible class at the rate of μ and (1-m) respectively.

$$\frac{dV}{dt} = mR + \lambda S - (1-m)V - \mu V$$

Table 1: Description of the state variables apply in the system (1)

Variables	Demonstration
N	Total human population
S	Susceptible individuals
E	Latent individuals
A	Asymptomatically infected individuals with no clinical symptoms of MTB infection
I	Symptomatically infected individuals with clinical symptoms of MTB infection
H	Hospitalized individuals
R	Recovered individuals
V	Vaccinated individuals

Table 2: Description of the state parameters apply in the system (1)

Parameters	Demonstration
Π	Recruitment rate of humans
μ	Natural death rate
β	Transmission probability for the MTB infection
σ	Progression of individuals from latent state of MTB infection
α	Proportion of infected individuals who developed symptoms
τi(i = 1,2,3)	Recovery rates of individuals
θ	Loss of immunity
m	Rate at which the vaccinated recovers due to vaccine efficiency
δj(j = 1,2,3)	Death caused by the disease

3. Model Analysis

3.1 Existence and Uniqueness of Solution

Using the [20] theorem we re-write equation (1) as:

$$\begin{aligned}
F_1 &= \pi + \theta R + (1-m)V - (\lambda+\mu)S \\
F_2 &= \lambda S - (\sigma+\mu)E \\
F_3 &= \alpha\sigma E - (\gamma_1+\tau_1+\delta_1+\mu)A \\
F_4 &= (1-\alpha)\sigma E - (\gamma_2+\tau_2+\delta_2+\mu)I \\
F_5 &= \gamma_1 A + \gamma_2 I - (\delta_3+\tau_3+\mu)H \\
F_6 &= \tau_1 A + \tau_2 I + \tau_3 H - (\theta+m+\mu)R \\
F_7 &= mR + \lambda S - (1-m)V - \mu V
\end{aligned} \tag{4}$$

$$\left|\frac{\partial F_1}{\partial S}\right| = |-(\lambda+\mu)| < \infty$$

$$\left|\frac{\partial F_1}{\partial I}\right| = 0 < \infty, \quad \left|\frac{\partial F_1}{\partial E}\right| = |0| < \infty$$

$$\left|\frac{\partial F_1}{\partial H}\right| = 0 < \infty$$

$$\left|\frac{\partial F_1}{\partial A}\right| = O < \infty \qquad \left|\frac{\partial F_1}{\partial R}\right| = |\theta| < \infty$$

$$\left|\frac{\partial F_1}{\partial V}\right| = |1-m| < \infty.$$

$$\left|\frac{\partial F_2}{\partial S}\right| = |\lambda| < \infty, \quad \left|\frac{\partial F_2}{\partial I}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_2}{\partial E}\right| = |-(\sigma+\mu)| < \infty, \quad \left|\frac{\partial F_2}{\partial H}\right| = 0 < \infty$$

$$\left|\frac{\partial F_2}{\partial A}\right| = 0 < \infty, \qquad \left|\frac{\partial F_2}{\partial R}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_2}{\partial V}\right| = 0 < \infty.$$

$$\left|\frac{\partial F_3}{\partial S}\right| = 0 < \infty, \quad \left|\frac{\partial F_3}{\partial I}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_3}{\partial H}\right| = 0 < \infty, \qquad \left|\frac{\partial F_3}{\partial E}\right| = |\alpha\sigma| < \infty$$

$$\left|\frac{\partial F_3}{\partial R}\right| = 0 < \infty \qquad \left|\frac{\partial F_3}{\partial A}\right| = |-(\gamma_1+\tau_1+\delta_1+\mu)|$$

$$\left|\frac{\partial F_3}{\partial V}\right| = 0 < \infty.$$

$$\left|\frac{\partial F_4}{\partial S}\right| = 0 < \infty$$

$$\left|\frac{\partial F_4}{\partial I}\right| = |-(\gamma_2+\tau_2+\delta_2+\mu)| < \infty$$

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$$\left|\frac{\partial F_4}{\partial E}\right| = |(1-\alpha)\sigma| < \infty$$

$$\left|\frac{\partial F_4}{\partial A}\right| = 0 < \infty, \quad \left|\frac{\partial F_4}{\partial R}\right| = 0 < \infty, \quad \left|\frac{\partial F_4}{\partial H}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_4}{\partial V}\right| = 0 < \infty$$

$$\left|\frac{\partial F_5}{\partial S}\right| = 0 < \infty, \quad \left|\frac{\partial F_5}{\partial R}\right| = 0 < \infty, \quad \left|\frac{\partial F_5}{\partial E}\right| = 0,$$

$$\left|\frac{\partial F_5}{\partial A}\right| = |\gamma_1| < \infty$$

$$\left|\frac{\partial F_5}{\partial I}\right| = |\gamma_2| < \infty$$

$$\left|\frac{\partial F_5}{\partial H}\right| = |-(\tau_3 + \delta_3 + \mu)| < \infty, \quad \left|\frac{\partial F_5}{\partial V}\right| = 0 < \infty.$$

$$\left|\frac{\partial F_6}{\partial S}\right| = 0 < \infty, \quad \left|\frac{\partial F_6}{\partial E}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_6}{\partial A}\right| = |\tau_1| < \infty, \quad \left|\frac{\partial F_6}{\partial I}\right| = |\tau_2| < \infty$$

$$\left|\frac{\partial F_6}{\partial H}\right| = |\tau_3| < \infty,$$

$$\left|\frac{\partial F_6}{\partial R}\right| = |-(\theta + m + \mu)| < \infty, \quad \left|\frac{\partial F_6}{\partial V}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_7}{\partial S}\right| = |\lambda| < \infty, \quad \left|\frac{\partial F_7}{\partial I}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_7}{\partial E}\right| = 0 < \infty, \quad \left|\frac{\partial F_7}{\partial H}\right| = 0 < \infty,$$

$$\left|\frac{\partial F_7}{\partial A}\right| = 0 < \infty, \quad \left|\frac{\partial F_7}{\partial R}\right| = |m| < \infty,$$

$$\left|\frac{\partial F_7}{\partial V}\right| = |-(1-m)\mu| < \infty,$$

3.2 Disease free equilibrium Point (DFE)

The population is infection-free at the disease-free equilibrium point (DFE) also known as the steady state solution in the absence of the disease, this means that:

$(E^* = 0,\ A^* = 0,\ I^* = 0,\ R^* = 0,\ H^* = 0,\ V^* = 0)$

and to achieve that, we set equation (2) to be equal to zero

$$\pi + \theta R + (1-m)V - (\lambda + \mu)S = 0$$

$$\lambda S - (\sigma + \mu)E = 0$$

$$\alpha\sigma E - (\gamma_1 + \tau_1 + \delta_1 + \mu)A = 0$$

$$(1-\alpha)\sigma E - (\gamma_2 + \tau_2 + \delta_2 + \mu)I = 0$$

(5)

$$\gamma_1 A + \gamma_2 I - (\delta_3 + \tau_3 + \mu)H = 0$$

$$\tau_1 A + \tau_2 I + \tau_3 H - (\theta + m + \mu)R = 0$$

$$mR + \lambda S - (1-m)V - \mu V = 0$$

$$s = \frac{\pi}{\lambda + \mu}$$

$\therefore$ Therefore, the disease free-equilibrium form denoted as G^* is as follows $:=$

$$G^* = (S^*, E^*, A^*, I^*, H^*, R^*, V^*) = \left(\frac{\pi}{\lambda+\mu}, 0, 0, 0, 0, 0, 0\right)$$

3.3 Endemic Equilibrium (EE)

Suppose S≠0, E≠0, A≠0, I≠0, H≠0, R≠0, V≠0, Solving equation (3) gives

$$s^{**} = \frac{\pi + \theta R^{**} + (1-m)V^{**}}{\lambda + \mu} \quad (6)$$

$$E^{**} = \frac{\lambda S^{**}}{\sigma + \mu} \quad (7)$$

$$A^{**} = \frac{\alpha\sigma E^{**}}{\gamma_1 + \tau_1 + \delta_1 + \mu} \quad (8)$$

$$I^{**} = \frac{(1-\alpha)\sigma E^{**}}{\gamma_2 + \tau_2 + \delta_2 + \mu} \quad (9)$$

$$H^{**} = \frac{\gamma_1 A^{**} + \lambda_2 I^{**}}{\delta_3 + \tau_3 + \mu} \quad (10)$$

$$R^{**} = \frac{\tau_1 A^{**} + \tau_2 I^{**} + \tau_3 H^{**}}{\theta + m + \mu} \quad (11)$$

$$V^{**} = \frac{mR^{**} + \lambda S^{**}}{\mu + (1+m)} \quad (12)$$

$\therefore$ Therefore, the Endemic equilibrium point is at $G^{**}(S^{**}, E^{**}, A^{**}, I^{**}, H^{**}, R^{**}, V^{**})$

3.4 Basic reproduction Number (R_0)

It can be determined using the method of next-generation matrix. The model equation

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(2) can be re-written starting with the new diseases in the system equation.

$$\frac{dE}{dt} = \lambda s - (\mu + \sigma)E$$

$$\frac{dA}{dt} = \alpha\sigma E - (\mu + \gamma_1 + \delta_1 + \tau_1)A$$

$$\frac{dI}{dt} = (1-\alpha)\sigma E - (\mu + \gamma_2 + \tau_2 + \delta_2)I$$

$$\frac{dH}{dt} = \gamma_1 A + \delta\gamma_2 I - (\delta_3 + \tau_3 + \mu)H \quad (13)$$

$$\frac{dR}{dt} = \tau_1 A + \tau_2 I + \tau_3 H - (\theta + m + \mu)R$$

$$\frac{dV}{dt} = mR + \lambda S - (1-m)V - \mu V$$

The matrix $\boldsymbol{F}$ is the rate of appearance of new infection and $\boldsymbol{V}$ is the matrix containing the rest of the terms.

$$F = [\lambda S],$$

$$V = \begin{bmatrix} \alpha\sigma E - (\mu + \gamma_1 + \tau_1 + \delta_1)A \\ (1-\alpha)\sigma E - (\mu + \gamma_2 + \tau_2 + \delta_2)I \end{bmatrix} \quad (14)$$

$$F_{E_0} = = S^0 \begin{bmatrix} \lambda & 0 \\ 0 & 0 \end{bmatrix},$$

$$V_{E_0} = \begin{bmatrix} -(\mu + \gamma_1 + \tau_1 + \delta_1) & 0 \\ 0 & -(\mu + \gamma_2 + \tau_2 + \delta_2) \end{bmatrix}$$

$$V^{-1} = \frac{1}{(\mu+\gamma_1+\tau_1+\delta_1)(\mu+\gamma_2+\tau_2+\delta_2)} \begin{bmatrix} -(\mu + \gamma_2 + \tau_2 + \delta_2) & 0 \\ 0 & -(\mu + \gamma_1 + \tau_1 + \delta_1) \end{bmatrix}$$

$$F^* V^{-1} = \begin{bmatrix} \frac{-\pi\lambda}{(\mu+\lambda)(\mu+\gamma_1+\tau_1+\delta_1)} & 0 \\ 0 & 0 \end{bmatrix}$$

The largest absolute spectra of the matrix above is:

$$\frac{\pi\lambda}{(\mu+\lambda)(\mu+\gamma_1+\tau_1+\delta_1)}$$

It follows that the associated basic reproduction number of the model (1), represented by R_0 is known as:

$$R_0 = \frac{\pi\lambda}{(\mu+\lambda)(\mu+\gamma_1+\tau_1+\delta_1)} \quad (15)$$

The threshold quantity, R_0, is the basic reproduction number for MTB infection, which determine the secondary infection caused when an infected individual is introduced into a susceptible human population.

Using theorem 1, with reproduction number, $R_0 < 1$, the disease-free equilibrium (DFE) point is locally asymptotically stable indicating that the population cannot be invaded by the disease. Hence, the proof the theorem hold as follows:

Theorem 1 The DFE (ϵ_0)**,** of the *MTB* model (1) is locally asymptotically stable (LAS) If $R_0 < 1$, and unstable if $R_0 > 1$ [6]**.**

Proof: The Jacobian around the disease-free equilibrium is given by:

$$J_{E_0} = \begin{pmatrix} -(\mu+\lambda) & 0 & 0 & 0 & 0 & \theta & (1-m) \\ \lambda & -(\mu+\sigma) & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha\sigma & -a_1 & 0 & 0 & 0 & 0 \\ 0 & -(1-\alpha)\sigma & 0 & -a_2 & 0 & 0 & 0 \\ 0 & 0 & \gamma_1 & \delta\gamma_2 & -a_3 & 0 & 0 \\ 0 & 0 & \tau_1 & \tau_2 & \tau_3 & -a_4 & 0 \end{pmatrix}$$

Where $-a_1 = (\mu + \gamma_1 + \tau_1 + \delta_1)$, $-a_2 = (\mu + \gamma_2 + \tau_2 + \delta_2)$, $-a_3 = (\mu + \tau_3 + \delta_3)$,

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$-a_4 = (\square \quad +m+\mu)$. From the matrix above, we have the trace to be negative and hence the disease-free equilibrium will be locally asymptotically stable if the determinant of the matrix is positive.

3.5 Sensitivity Analysis

In this phase, we determine and present which of the parameters in the R_0 will increase or decrease the burden of disease in the population. Consequently, using the technique used by [2] we have:

sensitivity with respect to π: $\frac{\lambda}{(\mu+\lambda)(\mu+\gamma_1+\tau_1+\delta_1)}$

sensitivity with respect to λ: $\frac{\pi}{(\lambda+\mu)(\mu+\gamma_1+\tau_1+\delta_1)} - \frac{\lambda\pi}{(\lambda+\mu)^2(\mu+\gamma_1+\tau_1+\delta_1)}$

sensitivity with respect to μ: - $\frac{\lambda\pi}{(\lambda+\mu)(\mu+\gamma_1+\tau_1+\delta_1)^2} - \frac{\lambda\pi}{(\lambda+\mu)^2(\mu+\gamma_1+\tau_1+\delta_1)}$

sensitivity with respect to δ_1 : - $\frac{\lambda\pi}{(\lambda+\mu)(\mu+\gamma_1+\tau_1+\delta_1)^2}$

sensitivity with respect to τ_1 : - $\frac{\lambda\pi}{(\lambda+\mu)(\mu+\gamma_1+\tau_1+\delta_1)^2}$

sensitivity with respect to γ_1 : - $\frac{\lambda\pi}{(\lambda+\mu)(\mu+\gamma_1+\tau_1+\delta_1)^2}$

sensitivity index with respect to π at π = 0.34, λ =0.8, μ =0.02, δ_1 =0.003, τ_1 =0.049 and γ_1 =0.027: + 40000/4059

sensitivity index with respect to λ at π = 0.34, λ =0.8, μ=0.02, δ_1 =0.003, τ_1 =0.049 and γ_1 =0.027: + 17000/166419

sensitivity index with respect to at π=0.34, λ=0.8, μ=0.02, δ_1 =0.003, τ_1 =0.049 and γ_1 =0.027: 624920000/16475481

sensitivity index with respect to δ_1 at π=0.34, λ =0.8, μ=0.02, δ_1 =0.003, τ_1 =0.049 and γ_1 =0.027: - 13600000/401841

sensitivity index with respect to τ_1 at π=0.34, λ =0.8, μ=0.02, δ_1 =0.003, τ_1 =0.049 and γ_1 =0.027: -13600000/401841

sensitivity index with respect to γ_1 at π=0.34, λ =0.8, μ=0.02, δ_1 =0.003, τ_1 =0.049 and γ_1 =0.027:-13600000/401841

Table 3: Sensitivity indices of the parameters in R_0^{MTB}

Param-eter	Value	Source	Sensitivity sign
	0.34	Assumed	+
Λ	0.8	Assumed	+
	0.02	Assumed	–
δ_1	0.003	Assumed	–
τ_1	0.049	Assumed	–
γ_1	0.027	Assumed	–

4. Results

Numerical simulation is carried out, we will solve and present plots of the system using the classical Runge-kutta numerical method of order 4 forward in time of 40 years via MATLAB. In doing so, we will vary key parameters to see graphically how they affect the spread of TB in the human population.

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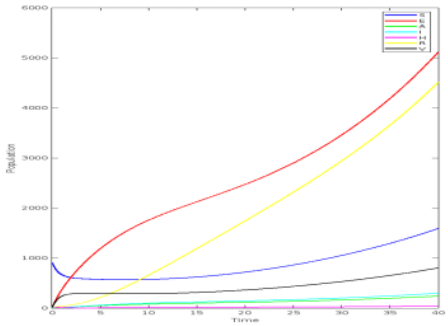

Figure 1: Plot of the State Variables

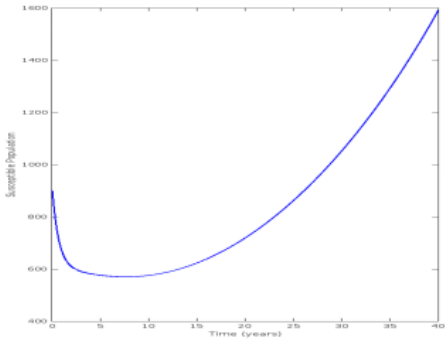

Figure 2: Plot of Susceptible Population

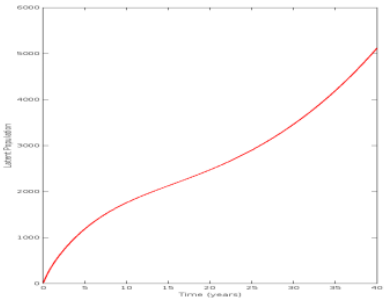

Figure 3: Plot of Latent Population

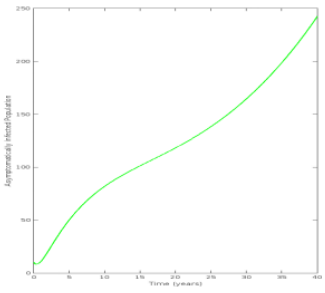

Figure 4: Plot of Asymptomatically Infected Population

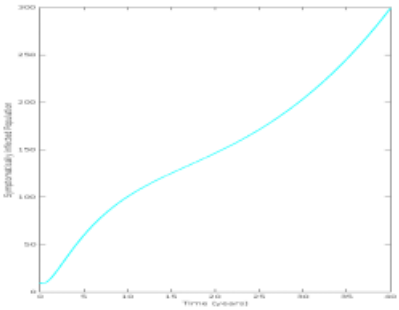

Figure 5: Plot of Symptomatically Infected Population

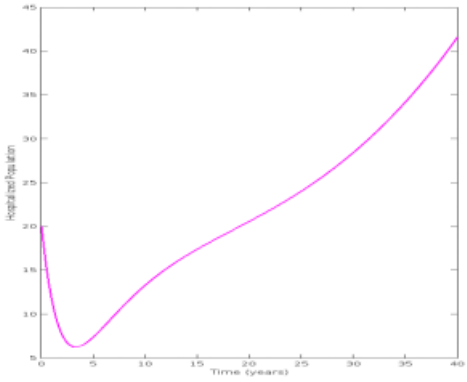

Figure 6: Plot of Hospitalized Population

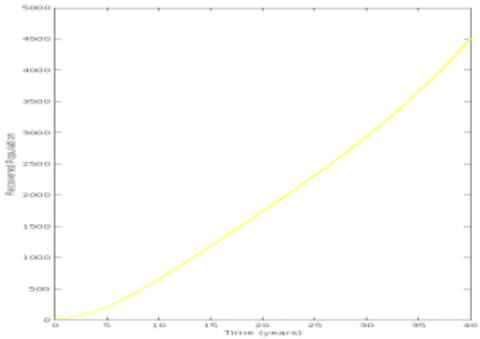

Figure 7: Plot of Recovered Population

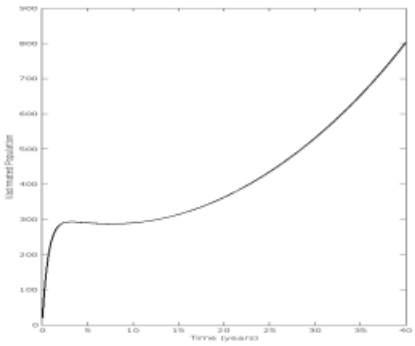

Figure 8: Plot of Vaccinated Population

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5. Discussion

This study builds upon an existing mathematical model of TB by integrating sensitivity analysis. Our findings indicate that individuals who have recovered from TB lose immunity over time as the vaccine's effectiveness wears off, becoming susceptible again. This phenomenon contributes to the decline in the number of recovered individuals, as shown in Figure 8. Moreover, our results show a significant increase in individuals with latent and infectious TB, as well as those who have recovered, as evident in their respective graphs.

First, we proved that our model is mathematically sound by showing it has a unique solution that remains bounded and non-negative over time. This essential property guarantees the model's stability and reliability, enabling us to trust its predictions and simulations. By establishing this foundation, we paved the way for further investigation and evaluation of our model's accuracy and effectiveness.

Our model analysis yields a crucial discovery: the disease-free equilibrium is locally stable, indicating that the disease can coexist with the population in either a dormant or prevalent state. Moreover, our bifurcation analysis reveals that the endemic equilibrium is bi-stable, suggesting that the disease can persist at either a low or high level. This means the population can shift between two states: a low-prevalence state with few cases or a high-prevalence state where the disease is widespread. This bi-stability implies that factors like intervention effectiveness, immune response, and transmission rates can trigger transitions between these states. This insight has vital implications for public health strategies, emphasizing the need for sustained efforts to control the disease and prevent its resurgence.

The sensitivity analysis offers vital insights into TB epidemiology, pinpointing key parameters that significantly impact disease transmission. As shown in Table 3, the analysis reveals a positive correlation between certain parameters and the basic reproductive number, while others exhibit a negative correlation. Specifically, parameters with a positive index directly influence the basic reproductive number, meaning increases in these parameters will boost transmission, and decreases will curb it. Conversely, parameters with a negative index inversely affect the basic reproductive number, indicating increases will reduce transmission, and decreases will enhance it. This understanding is crucial for developing effective TB control and prevention strategies. By identifying the most critical parameters, public health officials can concentrate efforts on key factors like vaccination, contact, and recovery rates to combat TB transmission and ultimately achieve disease elimination.

6. Conclusion

We presented a model to study the transmission dynamics of MTB infection in the human population. The model is focused on the analysis of mycobacterium tuberculosis transmission in humans, the model is analyzed in consideration of different stages of the infection. The system exhibits two equilibrium points: the disease-free equilibrium point (DFE), which is locally asymptotically stable whenever the $R_0 < 1$, otherwise is unstable. The analysis also showed that the treatment, campaign awareness, and other possible measures are vital to control MTB infection in human geographical boundaries.

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