A MODEL OF TUBERCULOSIS WITH EXOGENOUS RE-INFECTION AND VACCINATION

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Abstract

In this study, a continuous mathematical model for the dynamics of Tuberculosis at a varied recruitment rate bN was formulated. In the model, we partitioned the population into Susceptible (S), Vaccinated (M), exposed (E), Infected (I) and recovered (R) individuals. We analyzed a SMEIR compartmental nonlinear deterministic mathematical model of Tuberculosis epidemic in a community with constant population. Analytical studies were carried out on the model using the method of linearized stability. The basic reproductive number R_0 that governs the disease transmission is obtained from the largest eigenvalue of the next-generation matrix. The disease-free equilibrium is computed and proved to be locally asymptotically stable if $R_0 \leq 1$ and unstable if $R_0 > 1$. Finally, we simulate the model system in MATLAB and obtained the graphical behavior of the infected compartment at different levels vaccination and treatment rates. From the simulation, we observed that the Tuberculosis infection was eradicated when $R_0 \leq 1$ while it persist in the environment when $R_0 > 1$.

Keywords: *Tuberculosis, SMEIR Model, Basic reproduction number, Local stability, numerical simulation*

INTRODUCTION

Tuberculosis (TB), also known as the "white death", or historically as consumption (Chambers Dictionary,1998) is an infectious disease usually caused by *Mycobacterium tuberculosis* (MTB) bacteria (WHO, 2020). Tuberculosis generally affects the lungs, but it can also affect other parts of the body (WHO, 2020). Most infections show no symptoms, in which case it is known as latent tuberculosis (WHO, 2020). Around 10% of latent infections progress to active disease which, if left untreated, kill about half of those affected (WHO, 2020).

Tuberculosis may infect any part of the body, but most commonly occurs in the lungs (known as pulmonary tuberculosis) (Adkinson *et. al.*, 2010). The general signs and symptoms include fever, chills, night sweats, loss of appetite, weight loss, and fatigue (WHO, 2020). Significant nail clubbing may also occur (Gibson *et. al.*, 2005). The main cause of TB is *Mycobacterium tuberculosis* (MTB), a small, aerobic, non-motile bacillus (Adkinson *et. al.*, 2010). The high lipid content of this pathogen accounts for many of its unique clinical characteristics (Southwick, 2007). When people with active pulmonary TB cough, sneeze, speak, sing, or spit, they expel infectious aerosol droplets 0.5 to 5.0 μm in diameter. A single sneeze can release up to 40,000 droplets (Cole and Cook, 1998). Each one of these droplets may transmit the disease, since the infectious dose of tuberculosis is very small (the inhalation of fewer than 10 bacteria may cause an infection) (Nicas *et. al.*, 2005).

The most important risk factor globally for developing active TB is concurrent HIV infection; 13% of those with TB are also infected with HIV (WHO, 2011). This is a particular problem in sub-Saharan Africa, where HIV infection rates are high (WHO, 2006) (Chaisson and Martinson, 2008). Of those without HIV infection who are infected with tuberculosis, about 5–10% develops active disease during their lifetimes (Gibson *et. al.*, 2005); in contrast, 30% of those co-infected with HIV develop the active disease (Gibson *et. al.*, 2005). Tuberculosis prevention and control efforts rely primarily on the vaccination of infants and the detection and appropriate treatment of active cases (Lawn and Zumla, 2011). The World Health Organization (WHO) has achieved some success with improved treatment regimens, and a small decrease in case numbers (Lawn and Zumla, 2011). Some countries have legislation to involuntarily detain or examine those suspected to have tuberculosis, or involuntarily treat them if infected (Coker *et. al.*, 2007). The only available vaccine as of 2021 is bacillus Calmette-Guérin (BCG) (McShane, 2011) (CDC, 2021). In children it decreases the risk of getting the infection by 20% and the risk of infection turning into active disease by nearly 60% (Roy *et. al.*, 2007).

It is the most widely used vaccine worldwide, with more than 90% of all children being vaccinated (Lawn and Zumla, 2011). The immunity it induces decreases after about ten years (Lawn and Zumla, 2011).

Treatment of TB uses antibiotics to kill the bacteria. Effective TB treatment is difficult, due to the unusual structure and chemical composition of the mycobacterium cell wall, which hinders the entry of drugs and makes many antibiotics ineffective (Brennan and Nikaido, 1995).

Active TB is best treated with combinations of several antibiotics to reduce the risk of the bacteria developing antibiotic resistance (Lawn and Zumla, 2011). The routine use of rifabutin instead of rifampicin in HIV-positive people with tuberculosis is of unclear benefit as of 2007 (Davies *et. al.*, 2007). Acetylsalicylic acid (aspirin) at a dose of 100 mg per day has been shown to improve clinical signs and symptoms, reduce cavitary lesions, lower inflammatory markers, and increase the rate of sputum-negative conversion in patients with pulmonary tuberculosis (Di Bella *et. al.*, 2022).

MODEL DESCRIPTION AND FORMULATION

$S(t)$ = Number of susceptible individuals at time (t)

$E(t)$ = Number of exposed individuals at time (t)

$I(t)$ = Number of infected individuals at time (t)

$R(t)$ = Number of Recovered individuals at time (t)

$M(t)$ = Number of actively vaccinated individuals at time (t)

$N(t)$ = The total population size at time (t)

bN = Varying recruitment (b is natural birth rate, $b > 0$)

Λ = Constant recruitment rate used in the original model

β = Average number of susceptible individuals per contact per unit time.

σ = Average number of treated individuals infected by one infectious individual per-contact per unit time.

c = Per-capita contact rate

μ = Per capita natural death rate

κ = The rate at which individuals leave the latent class by becoming infectious

d = Per-capita disease-induced death rate

r = Per-capita treatment rate

p = the level of re-infection

θ = the rate at which susceptible individuals leave the susceptible class for vaccinated class M.

ψ = the rate at which vaccinated individuals leave the vaccinated class for susceptible class S.

ASSUMPTIONS OF THE MODEL

The assumptions in the existing model are still valid in our model. The following are the additional assumptions in the model.

i. 90 percent of the susceptible individuals are infants.
ii. the vaccination is strictly on susceptible individuals.
iii. the vaccination of the infants is artificially active.
iv. susceptible individuals move to the vaccinated class through active immunization at a rate θ
v. actively vaccinated individuals move to the susceptible class when they lose immunity at a rate ψ.

FLOW DIAGRAM OF THE MODEL

Partitioning our population size into the models subclasses, we demonstrate the dynamical transfer of the population with the flow diagram as shown in Figure 1.

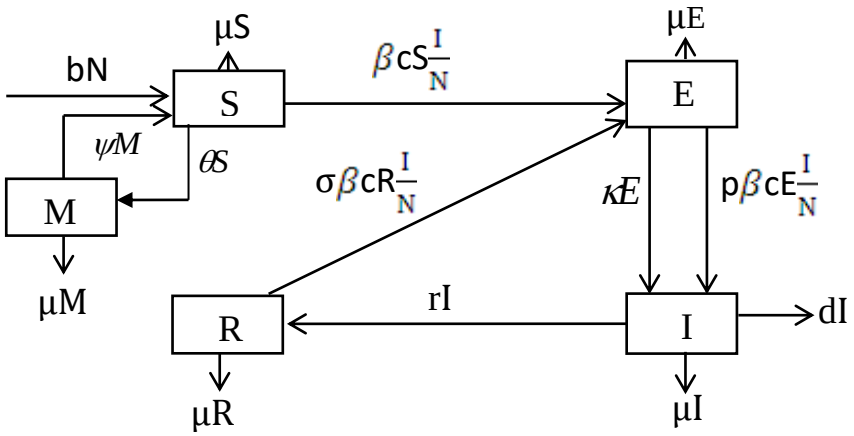


Figure 1: Schematic Diagram of the Model.

EQUATIONS OF THE MODEL

$$\frac{dS}{dt} = bN - \beta cS\frac{I}{N} - \mu S - \theta S + \psi M \quad (1)$$

$$\frac{dE}{dt} = \beta cS\frac{I}{N} - p\beta cE\frac{I}{N} - (\mu + \kappa)E + \sigma\beta cR\frac{I}{N} \quad (2)$$

$$\frac{dI}{dt} = p\beta cE\frac{I}{N} + \kappa E - (\mu + r + d)I \quad (3)$$

$$\frac{dR}{dt} = rI - \sigma\beta cR\frac{I}{N} - \mu R \quad (4)$$

$$\frac{dM}{dt} = \theta S - \mu M - \psi M \tag{5}$$

$$N = S + E + I + R + M \tag{6}$$

THE EXISTENCE OF DISEASE-FREE EQUILIBRIUM STATE OF THE MODEL

We will investigate the existence of the disease-free equilibrium state of the model which we will be denoted as G^*, a state in which there are no exposed and infected individuals. We assume that all the parameters are non-negative.

To find the existence of disease-free equilibrium state of the system, equation $(1)-(5)$ should be equated to zero (i.e. $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dM}{dt} = 0$). The resulting system is solved simultaneously.

$$bN - \beta cS\frac{I}{N} - \mu S - \theta S + \psi M = 0$$

$$\beta cS\frac{I}{N} - p\beta cE\frac{I}{N} - (\mu + \kappa)E + \sigma\beta cR\frac{I}{N} = 0$$

$$p\beta cE\frac{I}{N} + \kappa E - (\mu + r + d)I = 0$$

$$rI - \sigma\beta cR\frac{I}{N} - \mu R = 0$$

$$\theta S - \psi M - \mu M = 0$$

At the disease-free equilibrium state, $E = I = 0$, we have

$$b(S + R + M) - \mu S - \theta S + \psi M = 0$$

$$-\mu R = 0$$

$$\theta S - \psi M - \mu M = 0$$

$$\Rightarrow R = 0$$

$$b(S + M) - \mu S - \theta S + \psi M = 0 \tag{7}$$

$$\theta S = \psi M + \mu M \tag{8}$$

Substitute (8) in (7), we have

$$b(S + M) - \mu S - \mu M - \psi M + \psi M = 0$$

$$b(S + M) - \mu S - \mu M = 0$$

$b(S+M)-\mu(S+M)=0$

$(b-\mu)(S+M)=0$

$b-\mu=0 \Rightarrow b=\mu$ and

$S+M\neq 0$, where $S^*\neq 0$ and $M^*\neq 0$ are arbitrary.

The disease-free equilibrium point is $G^*=(S^*,0,0,0,M^*)$.

BASIC REPRODUCTION NUMBER OF THE DISEASE

The basic reproduction number R_0 is the average number of new infections that one infected case will generate during their entire infectious lifetime (Heffernan *et. al.*, 2005).

It is very important in determining whether the disease persists in the population or die out. We use the next generation matrix to compute the basic reproduction number R_0 which is formulated in (Guerra *et. al.*, 2017). Let us assume that there are n compartments of which the first m compartments correspond to infected individuals.

Let

- $F_i(y)$ be the rate of appearance of new infections in compartment i,
- $V_i^+(y)$ be the rate of transfer of individuals into compartment i by all other means, and
- $V_i^-(y)$ be the rate of transfer of individuals out of compartments i.

It is assumed that each function is continuously differentiable at least twice in each variable. The disease transmission model consists of nonnegative initial conditions together with the following system of equations:

$$\frac{dy_i}{dt}=f_i(y)=F_i(y)-V_i(y),\ i=1,2,3,\ldots,n \tag{9}$$

where $V_i(y)=V_i^-(y)-V_i^+(y)$. (10)

$$R_0=\rho\left(FV^{-1}\right)=\rho\left(\left(\left.\frac{\partial F_i}{\partial y_j}\right|_{E^0}\right)\left(\left.\frac{\partial V_i}{\partial y_j}\right|_{E^0}\right)^{-1}\right), \tag{11}$$

where F are the new infection transfer terms and V is the non-singular matrix of the remaining transfer terms. The basic reproduction number R_0 of the model (1) – (5) is calculated using the

next generation matrix (Guerra *et. al.*, 2017). In using their approach (Guerra *et. al.*, 2017), we have:

Let $f_1 = \frac{dE}{dt} = \beta cS\frac{I}{N} - p\beta cE\frac{I}{N} - (\mu+\kappa)E + \sigma\beta cR\frac{I}{N}$

$f_2 = \frac{dI}{dt} = p\beta cE\frac{I}{N} + \kappa E - (\mu + r + d)I$

$$\frac{d}{dt} = F - V = \begin{pmatrix} \frac{\beta cSI}{N} \\ 0 \end{pmatrix} - \begin{pmatrix} \frac{p\beta cEI}{N} + (\mu+k)E - \frac{\sigma\beta cRI}{N} \\ (\mu+r+d)I - p\beta cEI - kE \end{pmatrix}$$

$$F = \left(\left.\frac{\partial f_i}{\partial y_i}\right|_{E^\circ}\right) = \begin{pmatrix} \left.\frac{\partial f_1}{\partial E}\right|_{E^\circ} & \left.\frac{\partial f_1}{\partial I}\right|_{E^\circ} \\ \left.\frac{\partial f_2}{\partial E}\right|_{E^\circ} & \left.\frac{\partial f_2}{\partial I}\right|_{E^\circ} \end{pmatrix} = \begin{pmatrix} 0 & \frac{\beta cS^\circ}{S^\circ + M^\circ} \\ 0 & 0 \end{pmatrix}$$

$$V = \left(\left.\frac{\partial V_i}{\partial y_i}\right|_{E^\circ}\right) = \begin{pmatrix} \left.\frac{\partial V_1}{\partial E}\right|_{E^\circ} & \left.\frac{\partial V_1}{\partial I}\right|_{E^\circ} \\ \left.\frac{\partial V_2}{\partial E}\right|_{E^\circ} & \left.\frac{\partial V_2}{\partial I}\right|_{E^\circ} \end{pmatrix} = \begin{pmatrix} \mu+k & 0 \\ -k & \mu+r+d \end{pmatrix}$$

The inverse of V is obtained and given by

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu+k} & 0 \\ \frac{k}{(\mu+k)(\mu+r+d)} & \frac{1}{\mu+r+d} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta cS^\circ}{S^\circ + M^\circ} \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\mu+k} & 0 \\ \frac{k}{(\mu+k)(\mu+r+d)} & \frac{1}{\mu+r+d} \end{pmatrix}$$

$$\left|FV^{-1} - \lambda I\right| = \begin{vmatrix} \frac{k\beta cS^\circ}{(S^\circ+M^\circ)(\mu+k)(\mu+r+d)} - \lambda & \frac{\beta cS^\circ}{(S^\circ+M^\circ)(\mu+r+d)} \\ 0 & -\lambda \end{vmatrix} = 0$$

Therefore, the basic reproduction number R_0 which is the dominant eigenvalue of FV^{-1} is λ_2

$$\lambda_2 = R_0 = \frac{k\beta c S^\circ}{\left(S^\circ + M^\circ\right)(\mu + k)(\mu + r + d)}$$

THE STABILITY OF THE DISEASE-FREE EQUILIBRIUM STATE OF THE MODIFIED MODEL

To examine the local stability of the disease-free E° equilibrium, we obtain the Jacobian matrix by differentiating the functions $\left(f_i : i = 1,2,3,4,5\right)$ partially with respect to the variables in the model equations. The Jacobian matrix from the partial derivatives of (1) to (5) at disease-free $\left(J_{E^\circ}\right)$ is given by:

Let $f_1 = \frac{dS}{dt}$, $f_2 = \frac{dE}{dt}$, $f_3 = \frac{dI}{dt}$, $f_4 = \frac{dR}{dt}$, $f_5 = \frac{dM}{dt}$

Computing the Jacobian matrix J of the model

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial M} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} & \frac{\partial f_2}{\partial M} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} & \frac{\partial f_3}{\partial M} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} & \frac{\partial f_4}{\partial M} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial R} & \frac{\partial f_5}{\partial M} \end{bmatrix} \Rightarrow J_{G^{**}} = \begin{bmatrix} -\theta & b & b - \frac{\beta c S}{S+M} & b & b + \psi \\ 0 & -(\mu + \kappa) & \frac{\beta c S}{S+M} & 0 & 0 \\ 0 & \kappa & -(\mu + r + d) & 0 & 0 \\ 0 & 0 & r & -\mu & 0 \\ \theta & 0 & 0 & 0 & -(\psi + \mu) \end{bmatrix}$$

Let $v = b - \frac{\beta c S}{S+M}$, $w = b + \psi$, $x = -(\mu + \kappa)$, $y = -(\mu + r + d)$, $h = \frac{\beta c S}{S+M}$, $z = -(\psi + \mu)$

The characteristics equation is given by

$$\left|J_{G^*} - \lambda I\right| = \begin{vmatrix} -\theta - \lambda & b & v & b & w \\ 0 & x - \lambda & h & 0 & 0 \\ 0 & \kappa & y - \lambda & 0 & 0 \\ 0 & 0 & r & -\mu - \lambda & 0 \\ \theta & 0 & 0 & 0 & z - \lambda \end{vmatrix} = 0$$

$$\Rightarrow (-\theta - \lambda)\left[(x-\lambda)(y-\lambda)(-\mu-\lambda)(z-\lambda) + h\kappa(-\mu-\lambda)(z-\lambda)\right] + + w\left[(x-\lambda)(y-\lambda)(-\theta)(-\mu-\lambda) + h\kappa(-\theta)(-\mu-\lambda)\right] = 0$$

$$\Rightarrow(-\theta-\lambda)(-\mu-\lambda)(z-\lambda)\left[(x-\lambda)(y-\lambda)+h\kappa\right]+w(-\theta)(-\mu-\lambda)\left[(x-\lambda)(y-\lambda)+h\kappa\right]=0$$
$$\Rightarrow\left[(x-\lambda)(y-\lambda)+h\kappa\right]\left[(-\theta-\lambda)(-\mu-\lambda)(z-\lambda)+w(-\theta)(-\mu-\lambda)\right]=0$$

$$\Rightarrow(-\mu-\lambda)\left[(x-\lambda)(y-\lambda)+h\kappa\right]\left[(-\theta-\lambda)(z-\lambda)-w\theta\right]=0$$

$$\Rightarrow(-\mu-\lambda)\left[xy-\lambda x-\lambda y+\lambda^2+h\kappa\right]\left[-\theta z+\lambda\theta-\lambda z+\lambda^2-w\theta\right]=0$$

$$(-\mu-\lambda)\left[\lambda^2-\lambda(x+y)+(xy+h\kappa)\right]\left[\lambda^2-\lambda(z-\theta)-\theta(w+z)\right]=0$$

$$\therefore-\mu-\lambda=0\Rightarrow\lambda_1=-\mu$$

$$\lambda^2-\lambda(x+y)+(xy+h\kappa)=0\equiv a\lambda^2+b\lambda+c=0 \tag{12}$$

$$a=1,b=-(x+y),c=xy+h\kappa$$

Using general formula to solve equation (12), we have

$$\lambda=\frac{(x+y)\pm\sqrt{(x+y)^2-4(xy+h\kappa)}}{2}$$

$$\frac{(x+y)\pm\sqrt{(x+y)^2-4xy(1+\frac{h\kappa}{xy})}}{2}=\frac{(x+y)\pm\sqrt{(x+y)^2-4xy(1+C_1)}}{2}$$

Where $C_1=\frac{h\kappa}{xy}=\frac{h\kappa}{\left[-(\mu+\kappa)\right]\left[-(\mu+r+d)\right]}=\frac{h\kappa}{(\mu+\kappa)(\mu+r+d)}$

$$D=(x+y)^2-4xy(1+C_1)>0$$

$$(x-y)^2>4xyC_1$$

$$C_1<\frac{(x-y)^2}{4xy}$$

$$\frac{h\kappa}{xy}<\frac{(x-y)^2}{4xy}\Rightarrow h\kappa<\frac{(x-y)^2}{4}\Rightarrow h<\frac{(x-y)^2}{4\kappa}$$

Therefore, for $(x+y)^2-4xy(1+C_1)>0$ then $C_1<\frac{(x-y)^2}{4xy}$

$$\Rightarrow\lambda=\frac{(x+y)\pm\sqrt{(x+y)^2}}{2}=\frac{(x+y)\pm(x+y)}{2}$$

$$\Rightarrow \lambda_2 = \frac{(x+y)+(x+y)}{2} = \frac{2(x+y)}{2} = x+y$$

$$\lambda_2 = x+y = -(\mu+\kappa)-(\mu+r+d) = -(\mu+\kappa+\mu+r+d) = -(2\mu+\kappa+r+d)$$

$$\therefore \lambda_2 = -(2\mu+\kappa+r+d)$$

$$\lambda_3 = \frac{(x+y)-(x+y)}{2} = \frac{0}{2} = 0$$

$$\therefore \lambda_3 = 0$$

$$\lambda^2 - \lambda(z-\theta) - \theta(z+w) = 0 \equiv a\lambda^2 + b\lambda + c = 0 \tag{13}$$

$$a = 1, b = -(z-\theta), c = -\theta(z+w)$$

Using general formula to solve equation (13), we have

$$\lambda = \frac{(z-\theta) \pm \sqrt{(z-\theta)^2 + 4\theta(z+w)}}{2(1)} = \frac{-[m+\theta] \pm \sqrt{[(m+\theta]^2 - 4\theta(m-w)}}{2}$$

Where $z = -m = -(\mu+\psi) \Rightarrow m = \mu+\psi$

$$\lambda = \frac{-[m+\theta] \pm \sqrt{[m+\theta]^2 - 4\theta m(1-\frac{w}{m})}}{2}$$

$$\lambda = \frac{-[m+\theta] \pm \sqrt{[m+\theta]^2 - 4\theta m(1-C_2)}}{2}$$

where $C_2 = \frac{w}{m} = \frac{b+\psi}{\mu+\psi}$

$$D = (m+\theta)^2 - 4\theta m(1-C_2) > 0$$

$$(m-\theta)^2 > -4\theta m C_2$$

$$C_2 > -\frac{(m-\theta)^2}{4\theta m} \text{ Or}$$

$$\frac{w}{m} > -\frac{(m-\theta)^2}{4\theta m} \Rightarrow w > -\frac{(m-\theta)^2}{4\theta}$$

Therefore, for $(m+\theta)^2 - 4\theta m(1-C_2) > 0$ then $C_2 > -\frac{(m-\theta)^2}{4\theta m}$

$$\Rightarrow \lambda = \frac{-[m+\theta] \pm \sqrt{[m+\theta]^2}}{2} = \frac{-[m+\theta] \pm [m+\theta]}{2}$$

$$\Rightarrow \lambda_4 = \frac{-[m+\theta]+[m+\theta]}{2} \Rightarrow \lambda_4 = \frac{0}{2} = 0 \therefore \lambda_4 = 0$$

$$\lambda_5 = \frac{-[m+\theta]-[m+\theta]}{2} = \frac{-2[m+\theta]}{2}$$

$$\lambda_5 = -[m+\theta] = -(\psi+\mu+\theta) \therefore \lambda_5 = -(\psi+\mu+\theta)$$

Therefore, if $\lambda_1 < 0, \lambda_2 < 0, \lambda_3 \le 0, \lambda_4 \le 0, \lambda_5 < 0$ where $\beta, c, \mu, \lambda, r, d, k, \theta, \psi$ are positive constant, then it is uniformly stable.

NUMERICAL SIMULATIONS

Table 1: Initial Values of the Variables in the Model

Experiment → 1 Initial Values ↓	2	3	4	5	6
S(0)	13250	13250	13250	13250	13250	13250
E(0)	10500	10500	10500	10500	10500	10500
I(0)	1000	1000	1000	1000	1000	1000
R(0)	250	250	250	250	250	250
M(0)	11925	11925	11925	11925	11925	11925
N(0)	25000	25000	25000	25000	25000	25000

Table 2: Values of the Parameters in the Model

Experiment → 1 Parameters ↓	2	3	4	5	6
β	80	80	80	80	80	80
σ	0.9	0.9	0.9	0.9	0.9	0.9
c	0.1	0.1	0.1	0.1	0.1	0.1

μ	0.0167	0.0167	0.0167	0.0167	0.0167	0.0167
k	0.005	0.005	0.005	0.005	0.005	0.005
d	0.1	0.1	0.1	0.1	0.1	0.1
p	0.4	0.4	0.4	0.4	0.4	0.4
θ	0	1	2	3	4	5
ψ	0.1	0.1	0.1	0.1	0.1	0.1
b	0.1668	0.1668	0.1668	0.1668	0.1668	0.1668
r	0	1	2	3	4	5

THE GRAPHICAL REPRESENTATION OF THE RESULTS OF THE TB-MODEL

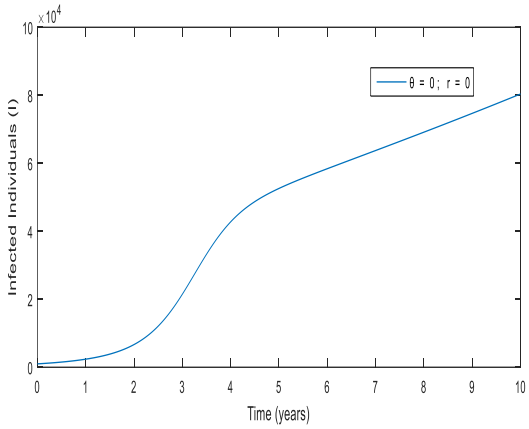
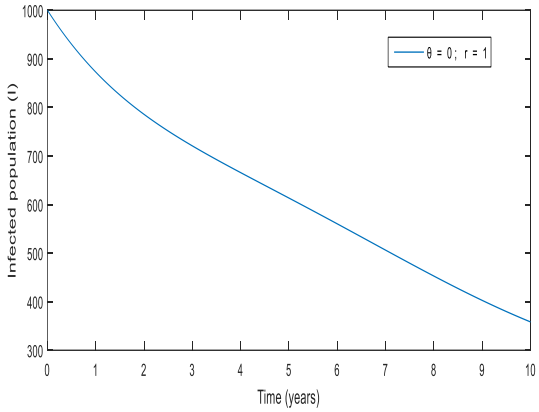


Figure 2(a)



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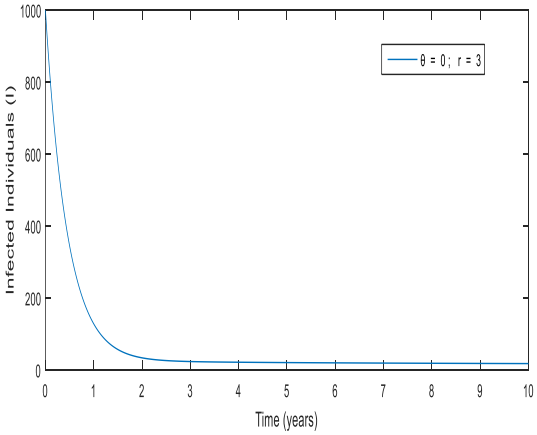


Figure 2(c)

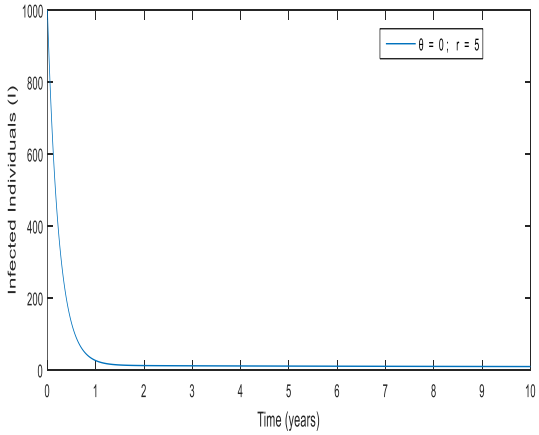
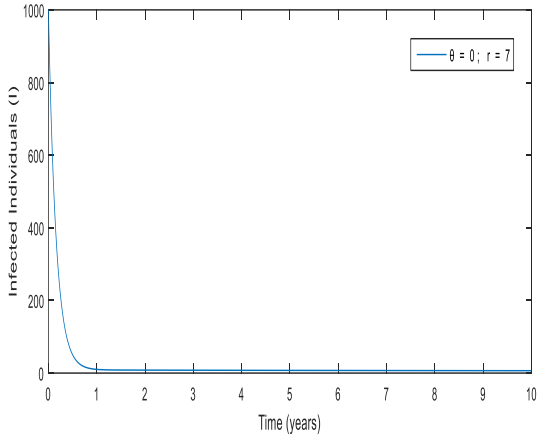


Figure 2(d)



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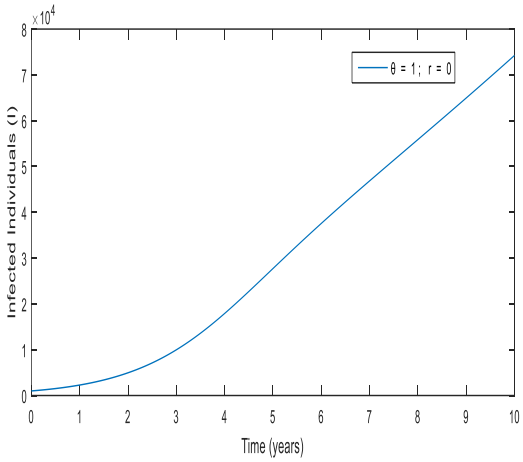

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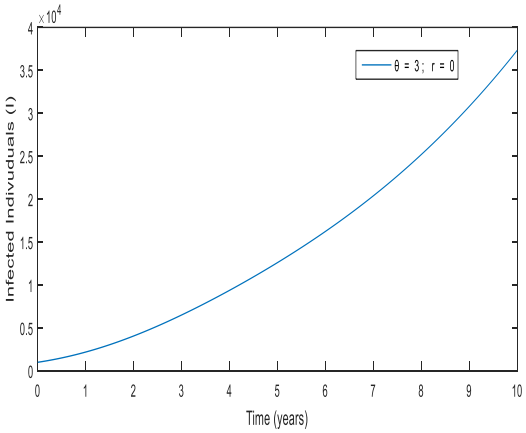

Figure 2(g)

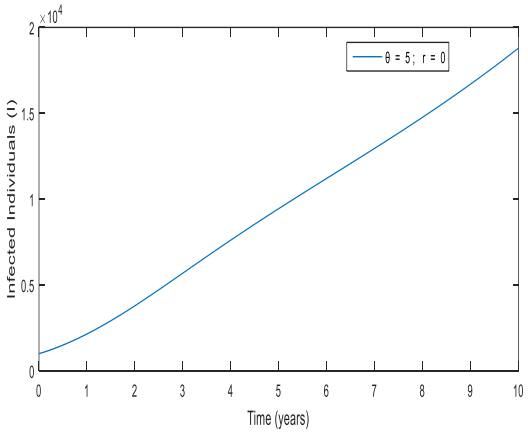

Figure 2(h)

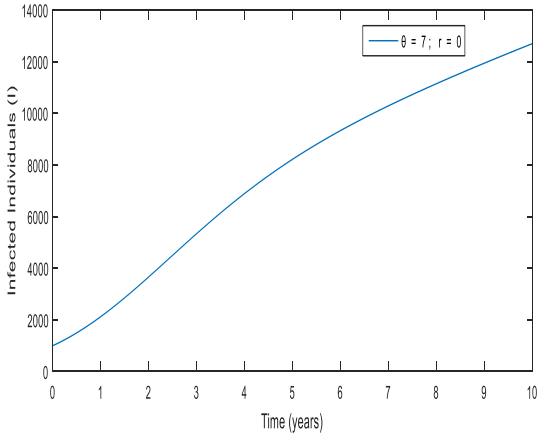

Figure 2(i)

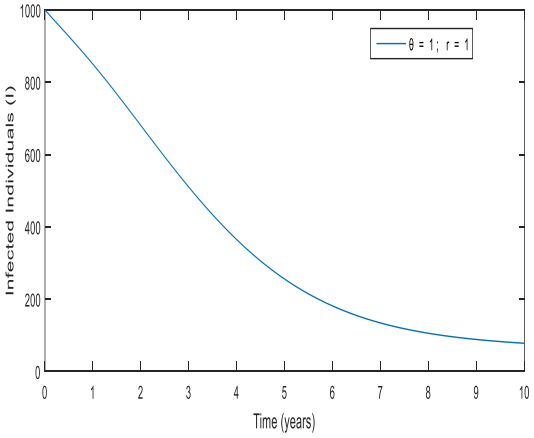

Figure 2(j)

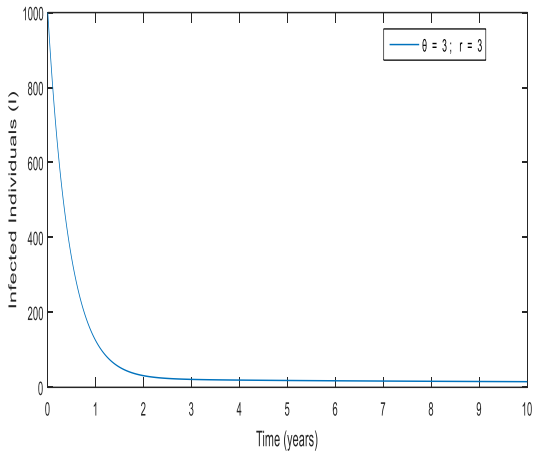

Figure 2(k)

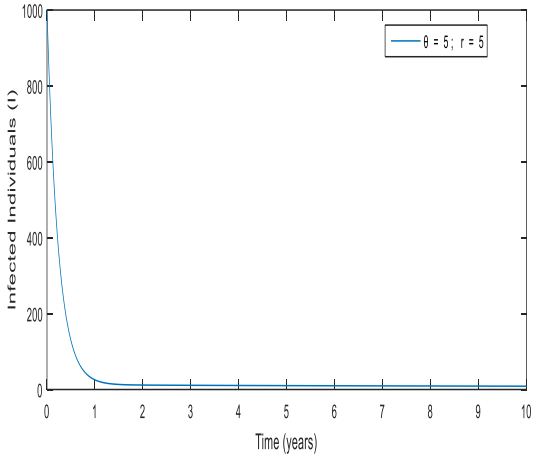

Figure 2(l)

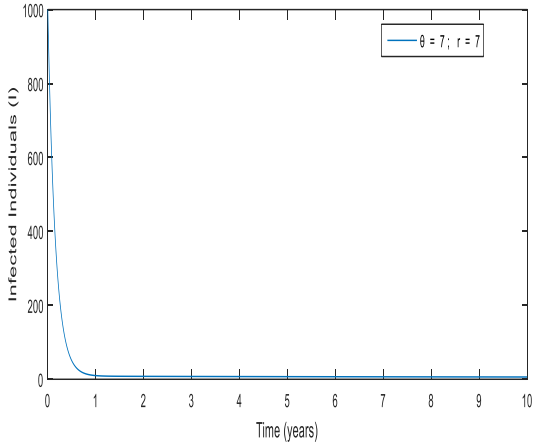

Figure 2(m)

INTERPRETATION OF THE RESULTS OF THE TB-MODEL

In figure 2(a), the dynamics of the disease with vaccination rate $\theta = 0$ and treatment rate $r = 0$ shows that the infectious population will grow from the initial infectious population of 1000 to 8×10^4 for 10years.

In figure 2(b), the dynamics of the disease with vaccination rate $\theta = 0$ and treatment rate $r = 1$ shows that the infectious population will decline from the initial infectious population of 1000 to 300 for 10years.

In figure 2(c), the dynamics of the disease with vaccination rate $\theta = 0$ and treatment rate $r = 3$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 50 after 2 years and remains constant throughout the remaining years.

In figure 2(d), the dynamics of the disease with vaccination rate $\theta = 0$ and treatment rate $r = 5$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 40 after 1 year and remains constant throughout the remaining years.

In figure 2(e), the dynamics of the disease with vaccination rate $\theta = 0$ and treatment rate $r = 7$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 30 after 1 year and remains constant throughout the remaining years.

In figure 2(f), the dynamics of the disease with vaccination rate $\theta = 1$ and treatment rate $r = 0$ shows that the infectious population will increase rapidly from the initial infectious population of 1000 to 7.4×10^4 for 10years.

In figure 2(g), the dynamics of the disease with vaccination rate $\theta = 3$ and treatment rate $r = 0$ shows that the infectious population will increase rapidly from the initial infectious population of 1000 to 3.7×10^4 for 10years.

In figure 2(h), the dynamics of the disease with vaccination rate $\theta = 5$ and treatment rate $r = 0$ shows that the infectious population will increase rapidly from the initial infectious population of 1000 to 1.8×10^4 for 10years.

In figure 2(i), the dynamics of the disease with vaccination rate $\theta = 7$ and treatment rate $r = 0$ shows that the infectious population will increase rapidly from the initial infectious population of 1000 to 1.25×10^4 for 10years.

In figure 2(j), the dynamics of the disease with vaccination rate $\theta = 1$ and treatment rate $r = 1$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 80 for 10years.

In figure 2(k), the dynamics of the disease with vaccination rate $\theta = 3$ and treatment rate $r = 3$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 100 in the first year and then gradually drops from 100 to 50 in the second year and finally remains constant throughout the remaining years.

In figure 2(l), the dynamics of the disease with vaccination rate $\theta = 5$ and treatment rate $r = 5$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 50 in the first year and then gradually drops from 50 to 20 in the second year and finally remains constant throughout the remaining years.

In figure 2(m), the dynamics of the disease with vaccination rate $\theta = 7$ and treatment rate $r = 7$ shows that the infectious population will decline rapidly from the initial infectious population of 1000 to 10 in the first year and finally remains constant throughout the remaining years.

CONCLUSION

In this paper, we observed that the disease free equilibrium (DFE) state of the TB model is $\left(S^*,0,0,0,M^*\right)$ where S^* and M^* are arbitrary. The basic reproduction number of the model is $R_0 = \frac{k\beta cS^\circ}{\left(S^\circ + M^\circ\right)\left(\mu + k\right)\left(\mu + r + d\right)} < 1$. The stability analysis shows that the eigenvalues of the TB model have both negative real parts and zeros. In the numerical experiments, we used a matlab functions which uses the Runge-Kutta scheme of order four (RK4) to solve non-stiff ordinary differential equations. The numerical results show that eradication is possible when treatment rate r are constantly increases.

RECOMMENDATION

In consideration of the findings of this study, we recommend that treatment rate $r \geq 0.9$ should be maintained in order to eradicate Tuberculosis in the population.

Finally, more research on tuberculosis should be done in order to obtain a vaccine that is more effective than *Bacillus Calmette-Guerin* (BCG) as well as increasing the treatment rate r.

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