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Sensitivity and Stability Analysis of Tuberculosis Disease with Infectious Latent

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ABSTRACT – *Tuberculosis (TB) is a dangerous contagious disease which can even lead to death if no control measure is applied. The disease is caused by mycobacterium which generally affects lungs and other related organs such as lymph gland, intestine, kidneys, uterus, bone and brain. The spread of TB occurs via the bacteria contaminated air which is inhaled into the lungs. Cough, chest pain, shortness of breath, appetite loss, weight loss, fever, cold and fatigue are some of the symptoms of TB. However, we proposed a mathematical model to investigate the transmission dynamics of tuberculosis and it is investigated analytically that the endemic equilibrium point is stable with the help of Routh-Hurwitz criteria. The sensitivity analysis shows that there would be an epidemic if and only if* $\beta \approx \beta^1$, *where* $\beta^1 \leq 0.1$. *Finally, using Matlab, it is shown that the disease free equilibrium is unstable which the endemic equilibrium becomes stable beyond 60 days. In addition, the recovered population increased rapidly while the exposed population decreased steeply in the disease-free equilibrium. It is an indication that there will be no outbreak of the tuberculosis infection. Besides, an increased in the effective contact rate increases both the infected population and recovered population. It is equally inferred that the recovered population do not show a trend pattern as* $\propto$ *increases while the susceptible and infected populations increased and decreased respectively as* $\propto$ *is increased. The recovered population showed no response pattern for* $\propto$ *since recovered individuals do not obtain permanent immunity.*

KEY WORDS: Sensitivity, stability, tuberculosis (TB), disease, latent, infection, dynamics, transmission.

1.0 INTRODUCTION

[1] See tuberculosis as an age long disease that still support huge levels of prevalence across the whole world. They identified the main cause of tuberculosis to be mycobacterium, a small, aerobic non motile bacillus. Tuberculosis is spread from person to person via moving air. They equally studied tuberculosis transmission by considering the existence of latent group and vaccine administration to the susceptible group. The model was formulated in SEIR-type and the results suggested that the vaccination rate would lower the transmission rate of tuberculosis disease. [2] were some of the earlier scholars to introduced mathematical model for tuberculosis (TB) dynamics. They divided the population into three categories and constructed the model

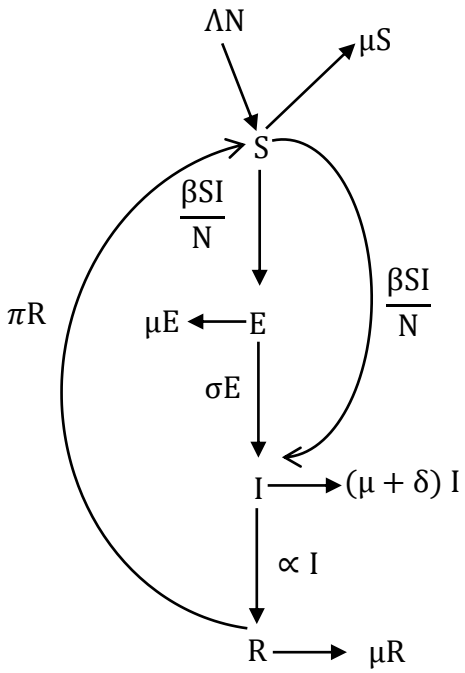
according to the epidemiological characteristics of tuberculosis transmission. A model for tuberculosis with exogenous reinfection was investigated by [3], the analysis showed that exogenous infection has a drastic effect on the qualitative dynamics of tuberculosis. [4] then presented a system of ordinary differential equations modelling the population of dynamics of tuberculosis (TB) with isolation and immigration of infective. [5] who investigated a deterministic tuberculosis model with genetic heterogeneity in susceptibility and disease progression. It was showed that the tuberculosis disease was control under a treatment regime. Meanwhile, most developed countries in the world, see tuberculosis as disease of the past, however in several countries like Nigeria, (especially in Sub-Sahara Africa and South-East Asia), the effect of tuberculosis is still high and devastating till date [6]. [7] defined disease as a disorder in their "stabilizing the steady state solution of lasser fever: problems and prospect. [8], tuberculosis is a contaminated disease which leads to high rate of mortality. The disease is caused by bacteria mycobacterium, tuberculosis usually attacks the lungs and other sensitive organs such as brain, kidney, gland, lymph and intestine. The widespread of tuberculosis via the bacteria contained air which is inhaled into the lungs. Tuberculosis cases increase in the year 2013 to 2015. Nigeria came third behind India and China in the new tuberculosis census [9]. Some of the symptoms of tuberculosis patients includes cough, chest pain, shortness of breath, fever, cold, fatigue, appetite lose. The epidemic of tuberculosis and HIV have continued to grow in countries that are poor, even with the introduction of effective and quality treatment strategies, such as DOTS, for active tuberculosis cases, the incidence of tuberculosis will continue to increase if HIV epidemic is not carefully and urgently checked [10]. Unfortunately, in several resource poor countries with high tuberculosis and HIV burden, antiretroviral therapy is often not commenced on persons infected with HIV until such patients have HIV, as a result, several persons with HIV infection are already infected with tuberculosis unknown to them [10]. Despite the gains achieved by DOTS, the treatment strategy has been limited however by the cost-effectiveness of the programme, the high unpredictability of DOTS interventions together with tuberculosis programmes, and the suitability of the treatment strategy to patients and health care workers in various setting [11]. [12] modifies the work of [13] by incorporating time-dependent control functions and to effectively apply optimal control theory in the resulting equation. The control functions denote the effect of limited-resources on intensive mass media enlightenment campaign and case findings approach, as they impact on the population dynamics of tuberculosis. Tuberculosis is seen to be declining gradually each year and an estimated 37 million lives were saved between 2000 and 2013 via effective diagnosis and treatment [14]. [3] established and analyzed a dynamical model to investigate, the spread of tuberculosis in a community with isolation and incomplete treatment and discovered that with the help of the model that prediction and control of tuberculosis is possible in the future. See also [5]. [15] now construct a model of the spread of MDR type S.I.R, then the results were obtained with the aid of 4^{th} order Range-Kuta method. The results shows clearly that the spread of tuberculosis can be controlled by lowering the rate of epidemic transmission and increasing the rate of recovery. [16] measures, the risk of airborne infections disease. Tuberculosis

transmission was equally investigated by [17] by considering the existence of latent group and vaccine administration to the susceptible group. The model was formulated in SEIR-type and the results suggested that the vaccination rate would lower the transmission rate of tuberculosis disease. Besides, [18] investigated the stability analysis of lasser fever using reproduction number and discovered that if $\mathrm{Re}(\lambda) > 0$, then the disease free equilibrium is unstable which means invasion is always possible and the infection will be able to spread in the population.

Furthermore, [19] investigated, statistical modelling of HIV, Tuberculosis, and Hepatitis B transmission in Ghana. The study applied competing risk techniques on these three diseases by assuming they were the major risks in the study population. Among all opportunistic infections that could also act within HIV infected individuals, tuberculosis has been asserted to be the most predominant. See [20] as well, but in this paper, we consider the sensitivity and stability analysis of tuberculosis disease with infectious latent.

2. MATHEMATICAL FORMULATION

2.1 FLOW DIAGRAM OF THE PROPOSED MODEL



From the flow diagram above, we have considered some basic assumptions:

(i) There is a random mixing of individuals in the population

(ii) Infected and latent individual recover from the symptoms of the disease

(iii) Some of new-born and migrants may be possibly latently infected at time they are born or migrate into the population.

(iv) Recovered is not removed from population and may become susceptible.

2.2 VARIABLES AND PARAMETER OF THE PROPOSED MODEL

Λ = Recruitment rate of new individuals

μ = Natural death rate

β = Effective contact rate

σ = Rate at which exposed individuals become infected

$\propto$ = Recovery rate

π = Rate at which recovered individuals' loose immunity and become susceptible again.

N = Total population

λ = Latent infections rate

E = Exposed latent

S = Susceptible

I = Infected

R = Recovered

3. Method of Solutions

From the flow diagram above, we considered the following model equations

$$\frac{dS}{dt} = \Lambda N - \frac{\beta SI}{N} + \pi - NS - \frac{\beta SI}{N} \quad (3.1)$$

$$\frac{dE}{dt} = \frac{\beta SI}{N} - (N + \sigma)E \tag{3.2}$$

$$\frac{dI}{dt} = \frac{\beta SI}{N} + \sigma N - (\mu + \delta + \propto)I \tag{3.3}$$

$$\frac{dR}{dt} = \propto I - (\mu + \pi)R \tag{3.4}$$

$$N = S + E + I + R \tag{3.5}$$

We scale (3.1) to (3.5)

Let $S = \frac{S}{N}, \quad e = \frac{E}{N}, \quad i = \frac{I}{N}, \quad r = \frac{R}{N}$

$$\left.\begin{array}{l} S = Ns, \quad E = eN, \quad I = iN, \quad R = rN \\ \frac{ds}{dt} = \frac{Nds}{dt}, \quad \frac{dE}{dt} = \frac{Nde}{dt}, \quad \frac{dI}{dt} = \frac{Ndi}{dt}, \quad \frac{dR}{dt} = \frac{Ndr}{dt} \end{array}\right\} \tag{3.6}$$

Substitute (3.6) in (3.1) to (3.5)

$$\frac{ds}{dt} = \Lambda - \beta si + \pi r - Ns - \beta si \tag{3.7}$$

$$\frac{de}{dt} = \beta si - (\mu + \sigma)e \tag{3.8}$$

$$\frac{di}{dt} = \beta si + \sigma e - (\mu + \delta + \propto)i \tag{3.9}$$

$$\frac{dr}{dt} = \propto i - (\mu + \pi)r \tag{3.10}$$

$$S + e + i + r = I$$

Stability Analysis

3.1 ENDEMIC EQUILIBRIUM POINT

In the determination of endemic equilibrium point, we have that;

$$\frac{ds}{dt} = \frac{de}{dt} = \frac{di}{dt} = \frac{dr}{dt} = 0$$

By simplification, it is established that

$$\Lambda - 2\beta si + \pi r - \mu s = 0 \tag{3.11}$$

$$\beta si - (\pi + r)e = 0 \tag{3.12}$$

$$\beta si + \sigma e - (\mu + \delta + \propto)i = 0 \tag{3.13}$$

$$\propto i - (\mu + \pi)r = 0 \tag{3.14}$$

(3.13) – (3.12)

$$\sigma e + (\mu + \sigma)e - (\mu + \delta + \propto)i = 0$$

$$e(2\sigma + \mu) - (\mu + \delta + \propto)i = 0$$

$$e = \frac{(\mu + \delta + \propto)i}{2\sigma + \mu} \tag{3.15}$$

Substituting (3.15) into (3.12), we have

$$S^* = \frac{(\mu + \sigma)(\mu + \delta + \propto)}{\beta(\mu + 2\sigma)} \tag{3.16}$$

From (3.14), we have

$$r = \frac{\propto i}{(\mu + \pi)} \tag{3.17}$$

Put (3.16) and (3.17) into (3.11), we have

$$\Lambda - \frac{2\beta(\mu+\sigma)(\mu+\delta+\sigma)}{\beta(\mu+2\sigma)}i + \frac{\propto \pi i}{(\mu+\pi)} - \frac{\mu(\mu+\sigma)(\mu+\delta+\sigma)}{\beta(\mu+2\sigma)} = 0$$

$$\left(\frac{2(\mu+\sigma)(\mu+\delta+\sigma)}{\mu+2\sigma} + \frac{\propto \pi}{\mu+\pi}\right)i = \frac{\mu(\mu+\sigma)(\mu+\delta+\sigma)}{\beta(\mu+2\sigma)} - \Lambda$$

$$\frac{2(\mu+\sigma)(\mu+\pi)(\mu+\delta+\sigma)}{(\mu+2\sigma)(\mu+\pi)} + \propto \pi(\mu+2\sigma)i - \frac{\mu(\mu+\sigma)(\mu+\delta+\sigma)}{\beta(\mu+2\sigma)} - \Lambda\beta(\mu+2\sigma)$$

$$i = \frac{[\mu(\mu+\sigma)(\mu+\delta+\sigma) - \Lambda\beta(\mu+2\sigma)](\mu+2\sigma)(\mu+\pi)}{\beta(\mu+2\sigma)[2(\mu+\sigma)(\mu+\pi)(\mu+\delta+\sigma) - \propto \pi(\mu+2\sigma)]}$$

On simplifying, we have

$$i^* = \frac{(\mu+\pi)[\mu(\mu+\sigma)(\mu+\delta+\sigma) - \Lambda\beta(\mu+2\sigma)]}{\beta[2(\mu+\sigma)(\mu+\pi)(\mu+\delta+\sigma) - \propto \pi(\mu+2\sigma)]} \tag{3.18}$$

Put (3.18) in (3.17), so that

$$r = \frac{\propto (\mu+\pi)[\mu(\mu+\sigma)(\mu+\delta+\sigma) - \Lambda\beta(\mu+2\sigma)]}{\beta(\mu+\sigma)[2(\mu+\sigma)(\mu+\pi)(\mu+\delta+\sigma) - \propto \pi(\mu+2\sigma)]}$$

$$r^* = \frac{\propto [\mu(\mu+\sigma)(\mu+\delta+\sigma) - \Lambda\beta(\mu+2\sigma)]}{\beta[2(\mu+\sigma)(\mu+\pi)(\mu+\delta+\sigma) - \propto \pi(\mu+2\sigma)]} \tag{3.19}$$

Put (3.18) in (3.15)

$$e^* = \frac{(\mu+\delta+\sigma)(\mu+\sigma)[\mu(\mu+\sigma)(\mu+\delta+\sigma) - \Lambda\beta(\mu+2\sigma)]}{\beta(\mu+2\sigma)[2(\mu+\sigma)(\mu+\pi)(\mu+\delta+\sigma) - \propto \pi(\mu+2\sigma)]}$$

Hence, we have obtained the endemic equilibrium.

Let $\quad a = \mu + \pi$

$$b = \mu + \delta + \sigma$$

$$c = \mu + \sigma$$

$$d = \mu + 2\sigma$$

So that

$$S^* = \frac{bc}{\beta d}, \quad e^* = \frac{ab(\mu bc - \pi\beta d)}{\beta d(2abc - \propto \pi d)}, \quad i^* = \frac{a(\mu bc - \mu\beta d)}{\beta(2abc - \propto \mu d)}$$

$$r^* = \frac{\propto (\mu bc - \pi\beta d)}{\beta(2abc - \propto \pi d)}$$

3.2 DETERMINATION OF THE EIGEN VALUES OF THE CHARACTERISTIC POLYNOMIAL AT EE_P.

$$f_1 = \Lambda - \beta s i + \pi r - \pi s - \beta s i$$

$$f_2 = \beta s i - (\mu + \sigma)e$$

$$f_3 = \beta s i + \sigma e - (\mu + \delta + \sigma) i$$

$$f_4 = \propto i - (\mu + \pi) r$$

$$J = \begin{bmatrix} \frac{\delta f_1}{\delta s} & \frac{\delta f_1}{\delta e} & \frac{\delta f_1}{\delta i} \\ \frac{\delta f_2}{\delta s} & \frac{\delta f_2}{\delta e} & \frac{\delta f_2}{\delta i} \\ \frac{\delta f_3}{\delta s} & \frac{\delta f_3}{\delta e} & \frac{\delta f_3}{\delta i} \end{bmatrix}$$

$$|J - \lambda I| = \begin{bmatrix} -2\beta i - \mu - \lambda & 0 & -2\beta s^* \\ \beta i^* & -(\mu + \sigma) - \lambda & \beta s^* \\ \beta i^* & \sigma & \beta s^* - (\mu + \delta + \sigma) - \lambda \end{bmatrix} = 0$$

By simplification with $k = \mu bc - \Lambda\beta d$ and $m = 2abc - \propto \pi d$, we have

$$\left(\frac{-2ak-\mu m}{m}-\lambda\right)\left(\frac{-bc^2-bc\lambda-\sigma bc}{d}+bc+(b+c)\lambda+\lambda^2\right)$$

$$+\frac{-2abc\sigma k-2abck(c+\lambda)}{md}=0$$

$$\frac{(-2ak-\mu m)(-bc^2-bc\lambda-\sigma bc)}{md}+bc\left(\frac{-2ak-\mu m}{m}\right)+\left(\frac{-2ak-\mu m}{m}\right)(b+c)\lambda$$

$$+\frac{(-2ak-\mu m)}{m}\lambda^2-\lambda\left(\frac{-bc^2-bc\lambda-\sigma bc}{d}\right)-bc\lambda-(b+c)\lambda^2-\lambda^3-\frac{2abc\sigma k}{md}-\frac{2abc^2k}{md}$$

$$-\frac{2abck\lambda}{md}=0$$

$$\frac{2abc^2k+2abck\lambda+2abc\sigma k+\mu mbc^2+\mu mbc\lambda+\mu m\sigma bc}{md}+bc\frac{(2ak+\mu m)}{m}$$

$$-\frac{2abk+2ack+\mu mb+\mu mc}{md}\lambda-\frac{2ak+\mu m}{m}\lambda^2+\frac{bc^2a+bc\lambda^2+\sigma bc\lambda}{d}-bc\lambda-(b+c)\lambda^2$$

$$-\lambda^3-\frac{2abc\sigma k+2abc^2k}{md}-\frac{2abck\lambda}{md}=0$$

$$-\lambda^3+\left(\frac{2ak+\mu m}{m}+\frac{bc}{d}-(b+c)\right)\lambda^2$$

$$+\left(\frac{2abck+\mu mbc}{md}-\frac{2abk+2ack+\mu mb+\mu mc}{m}+\frac{bc^2+\sigma bc}{d}-bc-\frac{2abck}{md}\right)\lambda+$$

$$\frac{2abc^2k+2abc\sigma k+\mu mbc^2+\mu m\sigma bc}{md}-bc\frac{(2ak+\mu m)}{m}=0$$

$$\lambda^3+\left(\frac{2ak+\mu m}{m}-\frac{bc}{d}+b+c\right)\lambda^2+\left(\frac{2abk+2ack+\mu mb+\mu mc}{m}-\frac{\mu mbc}{md}+bc-\right.$$

$$\frac{bc^2+\sigma bc}{d})\lambda \pm \frac{bc(2ak+\mu m)}{m} - \frac{\mu bc^2+\mu bc\sigma}{d} = 0$$

$$\Rightarrow \lambda^3 + Y\lambda^2 + Z\lambda + A = 0$$

Where

$$Y = 2(\mu+\pi)[(\mu+\delta+\propto)(\mu+\sigma) - \Lambda\beta(\mu+2\sigma)] + \mu(2(\mu+\pi)(\mu+\delta+\propto)(\mu+\sigma) - \propto \pi$$

$$(\mu+2\sigma)$$

$$2(\mu+\pi)(\mu+\delta+\propto)(\mu+\sigma) - \propto \pi(\mu+2\sigma) - \frac{(\mu+\delta+\propto)(\mu+\sigma)}{\mu+2\sigma} + \mu+\delta+\propto+\mu+\sigma$$

$$Z = 2(\mu+\pi)(\mu+\delta+\propto)[\mu(\mu+\delta+\propto)(\mu+\sigma) - \Lambda\beta(\mu+2\sigma)] + 2(\mu+\pi)(\mu+\sigma)[\mu$$

$$\frac{(\mu+\delta+\propto)(\mu+\sigma) - \Lambda\beta(\mu+2\sigma)] + \mu[2(\mu+\pi)(\mu+\delta+\propto)(\mu+\sigma) - \propto \pi(\mu+2\sigma)](\mu+\delta+\propto+\mu+\sigma)}{2(\mu+\pi)(\mu+\delta+\propto)(\mu+\sigma) - \propto \pi(\mu+2\sigma)}$$

$$+ (\mu+\delta+\propto)(\mu+\sigma) - \frac{\mu(\mu+\delta\ +\sigma)(\mu+\sigma)}{\mu+2\sigma}$$

$$-\frac{(\mu+\delta\ +\sigma)(\mu+\sigma)^2 + \sigma(\mu+\delta\ +\sigma)(\mu+\sigma)}{\mu+2\sigma}$$

$$A = (\mu+\delta\ +\sigma)(\mu+\sigma)\left[\frac{\begin{matrix}2(\mu+\pi)[\mu(\mu+\delta+\propto)(\mu+\sigma) - \Lambda\beta(\mu+2\sigma)] \\ -\mu[2(\mu+\pi)(\mu+\delta+\propto)(\mu+\sigma) - \propto \pi(\mu+2\sigma)]\end{matrix}}{2(\mu+\pi)(\mu+\delta+\propto)(\mu+\sigma) - \propto \pi(\mu+2\sigma)} - \frac{\mu(\mu+\sigma)+\mu\sigma}{\mu+2\sigma}\right]$$

By Routh-Hurwitz stability criterion, if $Y > 0$, $Z > 0$ and $YZ - A > 0$, then the endemic equilibrium point is stable.

We check:

$$\propto = 0.058,\ \delta = 0.139,\ \mu = 0.0222,\ \sigma = 0.00256,\ \Lambda = 0.05,\ \pi = 0.00256.$$

These parameter values are chosen such that the endemic equilibrium is stable and there is an outbreak of tuberculosis. Sensitivity analysis showed that there would be an epidemic if $\beta \approx \beta^{\iota}$, where $\beta^{\iota} \leq 0.1$.

4. GRAPHICAL RESULTS

We now present the numerical solutions using initial conditions for state variables $s(0) = 0.5,\ e(0) = 0.4,\ i(0) = 0.2$ and $r(0) = 0.1$. The parameter values are chosen in a way such that endemic equilibrium is achieved. The parameter values are given as: $\alpha = 0.2,\ \rho = 0.06,\ \mu = 0.04,\ \sigma = 0.2,\ \beta = 0.3,\ \Lambda = 0.1$ and π=0.0256. We present our results in graphical form as shown below.

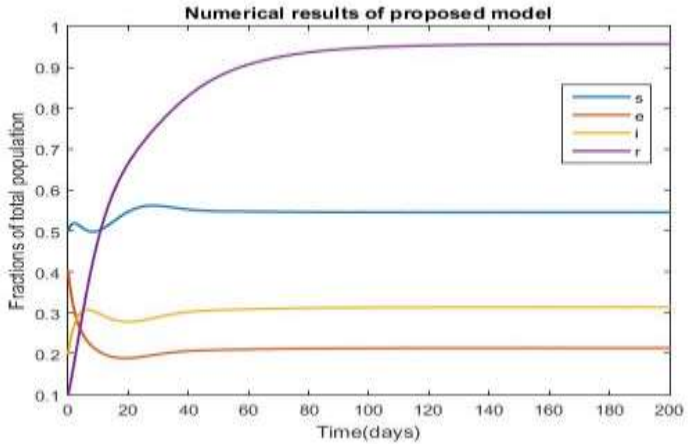
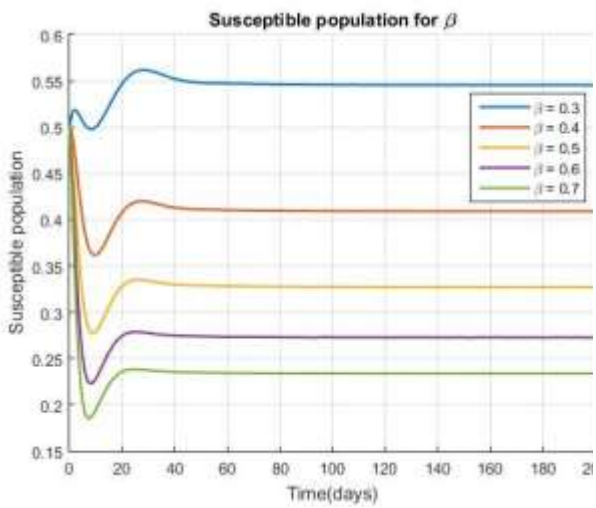
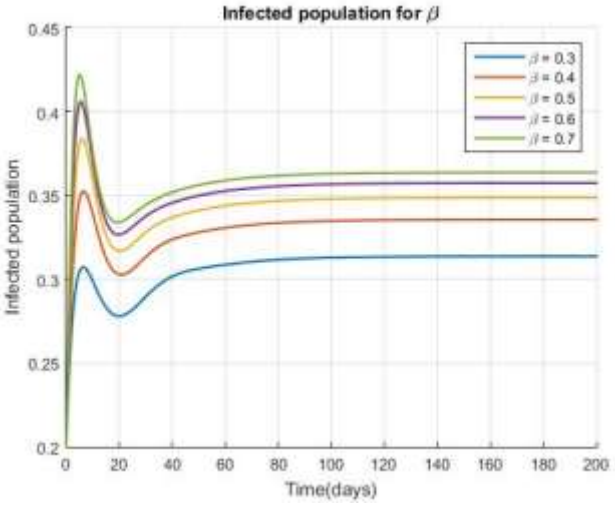
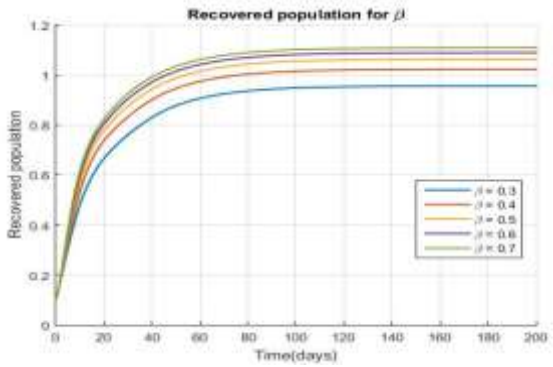


Figure 4.1: Solution curves of the TB proposed model at endemic equilibrium



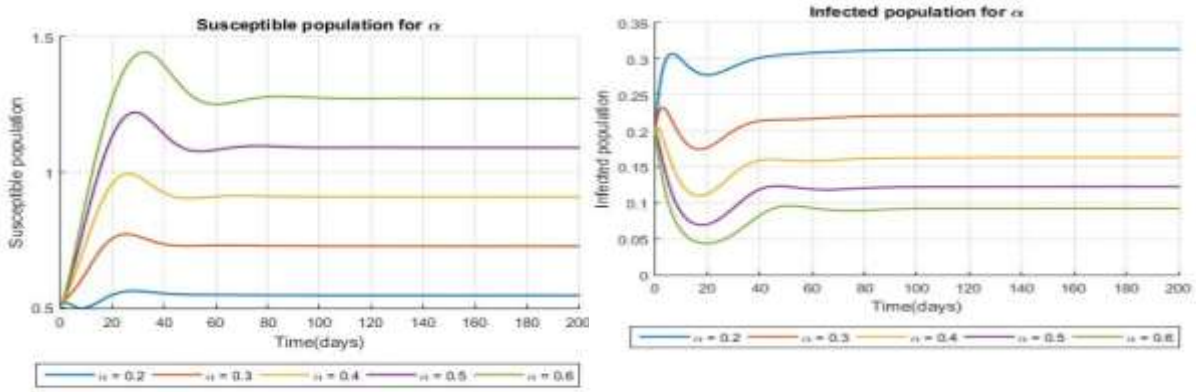


(a) (b)

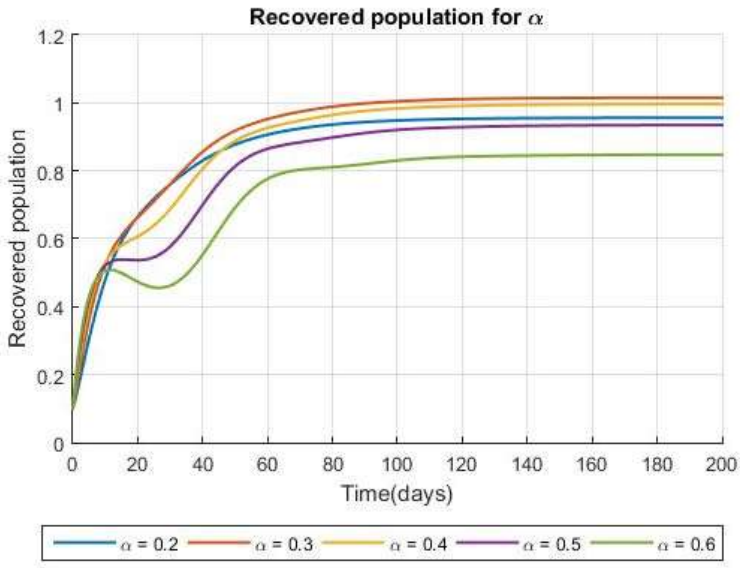


(c)

Figure 4.2: Solution curves for the effect of contact rate β on (a) Susceptible population (b) Infected Population (c) Recovered population.



(a) (b



(c)

Figure 4.3: Solution curves for the effect of recovery rate α on (a) Susceptible population (b) Infected Population (c) Recovered population

5. DISCUSSION

Figure 4.1 shows that the disease free equilibrium is unstable while the endemic equilibrium becomes stable beyond 60 days. In addition, the recovered population increased rapidly while the exposed population decreased steeply in the disease-free equilibrium. This shows that there will be no outbreak of the TB infection.

Beside, an increase in the effective contact rate increases both the infected population and recovered population. The same cannot be said for the susceptible population where an increase in contact rate results in a decrease in the population size. This is true because as more individuals come in contact with infectives the infected class increases and by induction more individuals receive treatment and recover from the disease.

Also, the results from figure 4.3 inferred that the recovered population do not show a trend pattern as α increases while the susceptible and infected populations increased and decreased respectively as α is increased from 0.2 to 0.6. The recovered population showed no response pattern for α because recovered individuals do not obtain permanent immunity and becomes susceptible again.

6. CONCLUSION

In this paper, we have proposed a mathematical model to study the transmission dynamics of tuberculosis. Through analytical studies, we determined the endemic equilibrium point and established the stability of our proposed tuberculosis model at the equilibrium point obtained.

We went further to obtained the numerical results of the proposed model, which we displayed in figure 4.1. Our numerical results were compared with the analytical results in three (3) and we observed that the numerical results agreed with our analytical findings.

Finally, we carried out sensitivity analysis of the model parameter β and α to determine the model response to them, we observed that the recovered population do not show a trend pattern as α is increased from 0.2 to 0.6. The same cannot be said for the parameter β as seen in figure 4.2, we observed that the infected and recovered populations showed an inverse relationship with β while the susceptible population showed a direct relationship with β.

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