ANALYSIS AND MODELING OF TUBERCULOSIS TRANSMISSION DYNAMICS

Abstract

Mycobacterium tuberculosis is the causative agent of Tuberculosis in humans.[1,8] A mathematical model that explains the transmission of Tuberculosis is developed. The model consists of four compartments; the susceptible humans, the infectious humans, the latently infected humans, and the recovered humans. We conducted an analysis of the disease-free equilibrium and endemic equilibrium points. We also computed the basic reproduction number using the next generation matrix approach. The disease-free equilibrium was found to be asymptotically stable if the reproduction number was less than one. The most sensitive parameter to the basic reproduction number was also determined using sensitivity analysis. Recruitment and contact rate are the most sensitive parameter that contributes to the basic reproduction number.Ordinary Differential Equations is used in the formulation of the model equations. The Tuberculosis model is analyzed in order to give a proper account of the impact of its transmission dynamics and the effect of the latent stage in TB transmission. The steady state's solution of the model is investigated. The findings showed that as more people come into contact with infectious individuals, the spread of TB would increase. The latent rate of infection below a critical value makes TB infection to persist. However, the recovery rate of infectious individuals is an indication that the spread of the disease will reduce with time which could help curb TB transmission

Keywords: latent TB, Transmission dynamics, Basic reproduction number, Stability analysis and equilibrium points.

1.0 Introduction

Tuberculosis (TB) is an airborne disease caused by the bacterium *Mycobacterium tuberculosis* [1]. *Mycobacterium* is carried in air particles called droplets nuclei. Depending on the environment, these tiny particles can remain suspended in the air for several hours, potentially infecting anyone who breathes them in. However, not everyone who inhales the bacteria gets sick because some people's immune system immediately kills the bacteria. In others, the bacteria remain in a latent or dormant The bacteria become inactive, but they remain alive in the body. People with latent tuberculosis have no symptoms of TB; they don't feel sick and can't spread the disease to others. Once infected, an individual stays infected for many years possibly latently-infected for life.[1,9]. TB is one of the oldest recorded human and animal diseases. It has been in animals before the existence of human species. Evidence that supports human cases of TB, as well as its role in human mortality, goes back to centuries. It’s not noticeable when the individual is infected and that makes the transmission of the disease easier.[9].

The rate of tuberculosis cases in many countries of sub-Saharan Africa over the past decade is largely attributed to Human immunodeficiency Virus (HIV) and other emerging infections.

Mathematical model of disease transmission within the human population have been acknowledged in helping policymakers and epidemiologists interpret epidemiological trends and understand the dynamics of the disease spread with efficiency of disease prevention and control.

In order to efficiently control and prevent infectious disease like TB one has to be adequately informed about the mechanism of the spread and the transmission dynamics of the disease. This will help our predictions and our strategies to eliminate diseases. The study of epidemic dynamics is an important theoretic approach to investigate the transmission dynamics of infectious diseases since they describe change over time.[1].

From different analysis and numerical simulations, mathematical models can is often used as a tool to understand the spread of infectious diseases and how to control it. Mathematical models developed for the transmission of tuberculosis are numerous.

Authors in [14] proposed a mathematical model that analyzed the study of TB transmission dynamics based on MSLR model. One of the principal attributes of these models is that the force of infection is a function of the number of infectious hosts in the population at any given time t. Other such as the recovery of infectious individuals and the death rate are modelled as linear terms with constant coefficients.

In their paper, the effect of vaccination and treatment on the transmission dynamics of TB was analyzed. The endemic equilibrium state of the model using the basic reproduction number shows that TB can be effectively controlled or eradicated if the total removal rate from both the latent and the infectious classes is usually less than the product of the total contraction and total breakdown of susceptible class. Their model was basically addressing mainly vaccination and treatment as a way of controlling the spread of TB.

Authors in [3], observed and predicted epidemiological models which review earlier study on modelling different aspects of tuberculosis dynamics. They observed that there was an increase in tuberculosis in the 1990s and the emergence of drug-resistant in the first decade of the 21^{st} century. They based their models on various mathematical systems such as systems of ordinary differential equations, simulation models Markov Chain and Monte Carlo method using a statistical analysis of TB patient data sets.

The above authors also extended the same model, Murphy et al. [2003] to form a new model considering how the presence of a genetically susceptible sub-population alters the effects of TB treatment at both latent and active stages. It is assumed that treatment doesn't confer immunity, but instead it moves individuals from actively infected to latently infected. Treatment of latently infected individuals reduces their reactivation rate. Results indicate that the exclusive treatment of latently infected individuals alone is not as effective as a treatment of actively infected individuals alone. Their research focused mainly on the treatment of latently infected as a way of reducing the spread of the disease.

Authors in [5,13], proposed a model by considering that the recovered individuals can only be re-infected by making contact with infectious individuals. They came up with SEIR model. In their research, they developed a model for the transmission of TB disease by considering recurrent infection and vaccination. The DFE and its stability are presented and its relation to

the basic reproduction number and vaccination reproduction number is discussed. Numerical examples show that vaccination is able to prevent the disease from spreading. They recommended that further study can be done for the optimal vaccination level as the function of the recurrent rate of infection. Their main focus was on the reoccurrence of TB .Many of these models address mainly immunization and treatment of the latently infected individuals.

According to [17],on tuberculosis, the Sustainable Development Goals (SDGs) for 2030 adopted by the United Nations in 2015, as one of the targets is to end the global TB epidemic. The WHO End TB strategy approved by the World Health Assembly in 2014, calls for a 90% reduction in TB deaths and an 80% reduction incidence rate by 2030, compared with 2015.

To combat the challenges of TB epidemic, there has been a massive scale-up of both treatment and diagnostic facilities particularly in Africa where TB is endemic.

3.0 The Model

This model is developed from [5.13] who combined immunization with latent TB treatment controlling the spread of TB. From their model, we assume the immunization and treatment of TB and focus on the effect of the latently infected in the transmission of TB. This model developed in this study is an improvement of the one developed by authors [6,8] in that it deals mainly with the general transmission dynamics and isolates the latent stage as the main contributing factor. This model is analysed qualitatively.

The population at a given time t is denoted by $N(t)$. The model divides the population into four epidemiological classes with respect to their disease status in the environment. The total population, represented by N(t), is divided into the subpopulation of susceptible humans (S), infectious humans (I), latent (L), and recovered (R).the total population becomes;

$N(t)=S(t)+L(t)+I(t)+R(t)$, where;

S(t)= the susceptible population that are at risk of developing infection from TB.

I(t)= infectious humans, showing symptoms of the disease.

L(t)= latent population, population having the disease but not showing the symptoms.

R(t)= those that have recovered after treatment and have got temporary immunity.

The susceptible humans enter into the population at a rate Λ . Susceptible humans acquire the disease through ingestion or contact and inhalation of spores. Contact with infectious humans at a rate β and individuals recover from the disease at a rate α . Humans who are infected with the disease die at a rate δ and the recovered human may lose immunity and return to the susceptible compartments at a rate γ . The natural death rate of the entire human compartments is μ . Susceptible humans become latently infected at the rate θ , and latently infected become infectious at the rate γ

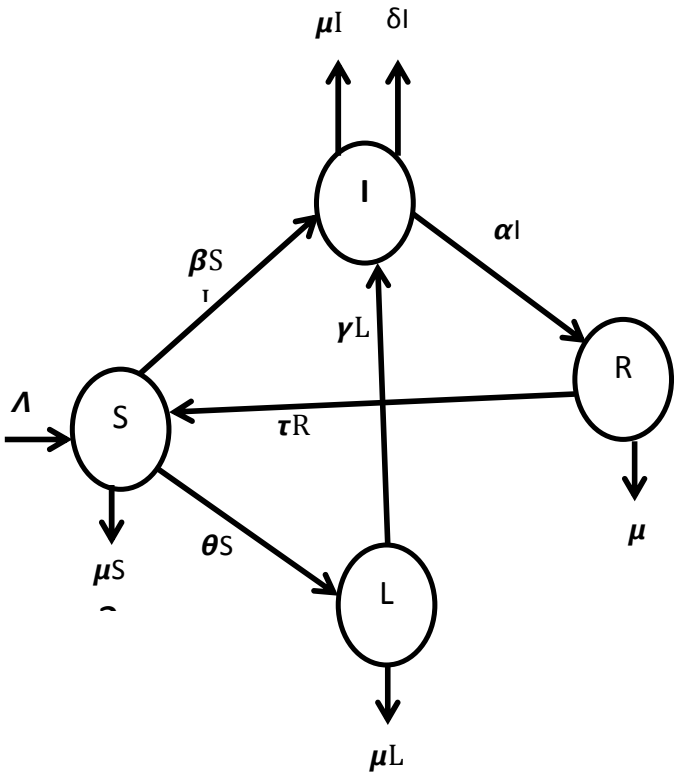

Figure 3.1: Model flow chart showing the compartments

From the figure above, the model equation become;

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI + \tau R - (\mu + \theta) S \\
\frac{dI}{dt} &= \beta SI + \gamma L - (\mu + \delta + \alpha) I \\
\frac{dL}{dt} &= \theta S - (\mu + \gamma) L \\
\frac{dR}{dt} &= \alpha I - (\mu + \tau) R
\end{aligned} \tag{3}$$

3.2 **Assumptions of the model**

From this model;

1. We assume that there is random mixing of individuals in the population.

2. We also assume that infected and latent individuals recover from the symptoms of TB after treatment.

3. We further assume that some of the newborns and migrants may be possibly latently infected at the time they are born or migrate into the population.

4. We also assume that the latently infected will not go back to the susceptible because they already have the bacteria.

3.3 The Disease Free Equilibrium.

The disease free equilibrium points is where there are no infections in the population

At the disease free equilibrium, there is no infection hence no recovery that is; I=L=R=0. Therefore at the equilibrium, we have, $\frac{dS}{dt}=\frac{dI}{dt}=\frac{dL}{dt}=\frac{dR}{dt}=0$ (4)

From equation (1) we have:

$$\Lambda-(\mu+\theta)S=0$$

$$S=\frac{\Lambda}{}\ S=\frac{\Lambda}{\mu+\theta} \tag{5}$$

The disease free equilibrium points from the model is expressed as follows;

$$E(S,I,L,R)=\left(\frac{\Lambda}{\mu+\theta},0,0,0\right) \tag{6}$$

3.4 Stability of the Disease Free Equilibrium

We will determine the stability of the disease-free equilibrium points which is done by linearizing the system of differential equations by obtaining the Jacobian at disease-free equilibrium ; $\left(\frac{\Lambda}{\mu+\theta},0,0,0\right)$ The Jacobian of the system of differential equation 4.1 is as shown below.

$$J=\begin{bmatrix} \frac{\partial S}{\partial S} & \frac{\partial S}{\partial I} & \frac{\partial S}{\partial L} & \frac{\partial S}{\partial R} \\ \frac{\partial I}{\partial S} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial L} & \frac{\partial I}{\partial R} \\ \frac{\partial L}{\partial S} & \frac{\partial L}{\partial I} & \frac{\partial L}{\partial L} & \frac{\partial L}{\partial R} \\ \frac{\partial R}{\partial S} & \frac{\partial R}{\partial I} & \frac{\partial R}{\partial L} & \frac{\partial R}{\partial R} \end{bmatrix} \tag{7}$$

$$J=\begin{bmatrix} -\beta I-(\mu+\theta) & -\beta S & 0 & \tau \\ \beta S & \beta S-(\mu+\delta+\alpha) & \gamma & 0 \\ \theta & 0 & -(\mu+\gamma) & 0 \\ 0 & \alpha & 0 & -(\mu+\tau) \end{bmatrix} \tag{8}$$

But I=0 $S=\frac{\Lambda}{\mu+\theta}$ Therefore the stability will be calculated using the Jacobian Matrix at the disease-free equilibrium by finding the determinant of the matrix

$$J\left(\frac{\Lambda}{\mu+\theta},0,0,0\right)=\begin{bmatrix} -(\mu+\theta) & -\frac{\beta\Lambda}{\mu+\theta} & 0 & \tau \\ 0 & -\frac{\beta\Lambda}{\mu+\theta}-(\mu+\delta+\alpha) & \gamma & 0 \\ \theta & 0 & -(\mu+\gamma) & 0 \\ 0 & \alpha & 0 & -(\mu+\tau) \end{bmatrix}=0$$

$$(J-\lambda I)=\begin{bmatrix} -(\mu+\theta)-\lambda & -\frac{\beta\Lambda}{\mu+\theta} & 0 & \tau \\ 0 & -\frac{\beta\Lambda}{\mu+\theta}-(\mu+\delta+\alpha)-\lambda & \gamma & 0 \\ \theta & 0 & -(\mu+\gamma)-\lambda & 0 \\ 0 & \alpha & 0 & -(\mu+\tau)-\lambda \end{bmatrix}=0$$

Where λ is the Eigen value

Using the Jacobian at the disease free equilibrium we determine the Eigenvalue at the disease-free equilibrium

$$\lambda_1 = -(\mu+\theta), \lambda_2 = -\frac{\beta\Lambda}{\mu+\theta} + (\mu+\delta+\alpha), \lambda_3 = -(\mu+\gamma), \lambda_4 = -(\mu+\tau) \qquad (9)$$

Since all Eigenvalues are negative, then the disease-free equilibrium is locally asymptotically stable.

3.5 The basic reproduction number

The basic reproduction number is defined as the average number of secondary cases arising from an average primary case in an entirely susceptible population over the period of infection [2,11]. The reproduction number is used to predict whether the epidemiological model has a disease-free equilibrium (DFE) at which the population remains in the absence of the disease. If $R_0 < 1$ then the disease-free equilibrium (DFE) is locally asymptotically stable. If $R_0 > 1$ then DFE is unstable. In order to get the reproductive number, we calculate it using the next generation matrix from the model equations $F = \frac{\partial f_i(x_0)}{\partial x_j}$, $V = \frac{\partial v_i(x_0)}{\partial x_j}$

F_i = rate at which new infections enter the compartment

V_i = transfer of individuals out and into the i[th] compartment.

X_0 =DFE

using the second and third equations above, we obtain;

$$F = \begin{bmatrix} \beta SI \\ 0 \end{bmatrix}$$

$$V = \begin{bmatrix} -\gamma I + (\mu+\delta+\alpha) I \\ -\theta S + (\mu+\theta) L \end{bmatrix}$$

$$V = \frac{\partial V}{\partial I}\bigg|_{\frac{\Lambda}{\mu+\theta}} = (\mu+\delta+\alpha) \qquad F(I) = \beta SI$$

$$\frac{\partial V}{\partial L} = \mu+\gamma$$

$$F = \frac{\partial f}{\partial I}\bigg|_{\frac{\Lambda}{\mu+\theta},0,0,0} = \beta$$

$$F = \begin{bmatrix} \beta SI \\ 0 \end{bmatrix}$$

$$DFE = \left(\frac{\Lambda}{\mu + \theta}, 0, 0, 0 \right)$$

Matrix F defines new infections in different compartments, differentiated w.r.t I and L evaluated at the DFE.

$$f_i = \beta SI \quad F = \begin{bmatrix} f_1 \\ f_2 \end{bmatrix} \qquad F = \begin{bmatrix} \beta SI \\ 0 \end{bmatrix}$$

$$V = \begin{bmatrix} \frac{\partial L}{\partial L} & \frac{\partial L}{\partial I} \\ \frac{\partial I}{\partial L} & \frac{\partial I}{\partial I} \end{bmatrix} = \begin{bmatrix} \mu + \gamma & 0 \\ -\gamma & \mu + \delta + \alpha \end{bmatrix}$$

$$V = \frac{1}{(\mu + \delta + \alpha)(\mu + \gamma)} \begin{bmatrix} \mu + \delta + \alpha & 0 \\ \gamma & \mu + \gamma \end{bmatrix}$$

$$F = \begin{bmatrix} \frac{\partial \beta SI}{\partial L} & \frac{\partial \beta SI}{\partial I} \\ 0 & 0 \end{bmatrix} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & \beta \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu + \gamma} & 0 \\ \frac{\gamma}{(\mu + \gamma)(\mu + \delta + \alpha)} & \frac{1}{\mu + \delta + \alpha} \end{bmatrix} = 0$$

Now we calculate the eigenvalues of the matrix to determine the basic reproduction number, R_0 defined as the spectral radius (dominant eigenvalue) of the matrix. This is computed by $|A - I\lambda| = 0$ where A is the matrix and I is the 2x2 identity matrix.

$$\begin{bmatrix} \frac{\beta\gamma}{(\mu+\gamma)(\mu+\delta+\alpha)} - \lambda & \frac{\beta}{\mu+\delta+\alpha} \\ 0 & 0-\lambda \end{bmatrix} = 0$$

$$\lambda = 0$$

$$\lambda = \frac{\beta\gamma}{(\mu+\gamma)(\mu+\delta+\alpha)} \tag{10}$$

FV^{-1} is called the next generation matrix. The spectral radius of FV^{-1} is equal to R_0.

R_0 is the maximum Eigen value of FV^{-1}. Therefore;

$$R_0 = \frac{\beta\gamma}{(\mu+\gamma)(\mu+\delta+\alpha)}$$

We now give the condition necessary to ensure a disease-free population;

Theorem

If $\gamma < \frac{\mu(\mu+\delta+\alpha)}{\beta-(\mu+\delta+\alpha)}$, the disease will not take hold in the population.

Proof

At the disease-free equilibrium, $R_0 = \frac{\beta\gamma}{(\mu+\delta+\alpha)(\mu+\gamma)}$ then $R_0 < 1$

$$\frac{\beta\gamma}{(\mu+\delta+\alpha)(\mu+\gamma)} < 1$$
$$\beta\gamma < (\mu+\delta+\alpha)(\mu+\gamma)$$
$$\beta\gamma < \gamma(\mu+\delta+\alpha) + \mu(\mu+\delta+\alpha) \quad (7)$$
$$\beta\gamma - \gamma(\mu+\delta+\alpha) < \mu(\mu+\delta+\alpha)$$
$$\gamma < \frac{\mu(\mu+\delta+\alpha)}{\beta-(\mu+\delta+\alpha)}$$

Lemma

If $\gamma < \frac{\mu(\mu+\delta+\alpha)}{\beta-(\mu+\delta+\alpha)}$ then the disease will take hold in the Population.

When $R_0 > 1$, then $\frac{\beta\gamma}{(\mu+\delta+\alpha)(\mu+\gamma)} > 1$ (11)

3.5: Endemic equilibrium

The endemic equilibrium points where the disease will co-exist in all compartments of the population, that is, the state where the disease persistent in the population.. In this situation, if $E^*(S^*I^*L^*R) \neq 0$ Using the systems of equations above, we derive the endemic equilibrium point.

$$R = \frac{\alpha I}{\mu+\theta} \qquad L = \frac{\theta S}{\mu+\gamma}$$

$$\beta SI + \gamma\frac{\theta S}{\mu+\gamma} = (\mu+\delta+\alpha)I$$

$$S^* = \frac{(\mu+\delta+\alpha)}{\beta}$$

$$\theta S - (\mu+\gamma)L = 0$$

$$\theta\left[\frac{\mu+\delta+\alpha}{\beta}\right] = (\mu+\gamma)L$$

$$L^* = \frac{\theta(\mu+\delta+\alpha)}{\beta(\mu+\gamma)}$$

$$\Lambda - \beta SI + \frac{\tau\alpha I}{\mu+\theta} = (\mu+\theta)S$$

$$\Lambda - \beta SI\left[\frac{\mu+\delta+\alpha}{\beta}\right] + \frac{\tau\alpha I}{\mu+\tau} = \left(\mu+\theta\left(\frac{\mu+\delta+\alpha}{\beta}\right)\right)$$

$$\Lambda - (\mu+\theta)\left(\frac{\mu+\delta+\alpha}{\beta}\right) = \left[(\mu+\delta+\alpha) + \frac{\tau\alpha}{\mu+\tau}\right]$$

$$I^* = \frac{\Lambda - (\mu+\theta)\dfrac{\mu+\delta+\alpha}{\beta}}{(\mu+\delta+\alpha)\left(\dfrac{\tau\alpha}{\mu+\tau}\right)} = \frac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}$$

$$\alpha I = (\mu+\tau)R \qquad\qquad \alpha I = (\mu+\tau)R$$

$$\alpha\left[\frac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}\right] = (\mu+\tau)R$$

$$R^* = \alpha\left[\frac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha(\mu+\tau)}\right]$$

The endemic equilibrium becomes

$$\left(S^*, I^*, L^*, R^*\right) = \begin{bmatrix} \dfrac{(\mu+\delta+\alpha)}{\beta}, \dfrac{\theta(\mu+\delta+\alpha)}{\beta(\mu+\gamma)}, \dfrac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}, \\ \alpha\left[\dfrac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha(\mu+\tau)}\right] \end{bmatrix} \qquad (12)$$

Stability of the Endemic Equilibrium

3.7 The stability analysis of the endemic equilibrium

If the basic reproduction number is less than one, then the endemic equilibrium is asymptotically stable.

Using the Jacobian matrix,

$$J = \begin{bmatrix} -\beta S^* - (\mu+\theta) & -\beta S^* & 0 & \tau \\ \beta I^* & (\mu+\delta+\alpha) & \gamma & 0 \\ \theta & 0 & -(\mu+\gamma) & 0 \\ 0 & \alpha & 0 & -(\mu+\tau) \end{bmatrix}$$

The characteristic equation of the Jacobian matrix becomes $\left[J - I\lambda\right] = 0$

$$\left[J - I\lambda\right] = \begin{bmatrix} -\beta S^* - (\mu+\theta) - \lambda & -\beta S^* & 0 & \tau \\ \beta I^* & (\mu+\delta+\alpha) - \lambda & \gamma & 0 \\ \theta & 0 & -(\mu+\gamma) - \lambda & 0 \\ 0 & \alpha & 0 & -(\mu+\tau) - \lambda \end{bmatrix} = 0$$

$$= \begin{bmatrix} -(\mu+\delta+\alpha) - (\mu+\theta) - \lambda & -(\mu+\delta+\alpha) & 0 & \tau \\ \beta\left[\dfrac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha) + \tau\alpha}\right] & (\mu+\delta+\alpha) - \lambda & \gamma & 0 \\ \theta & 0 & -(\mu+\gamma) - \lambda & 0 \\ 0 & \alpha & 0 & -(\mu+\tau) - \lambda \end{bmatrix}$$

$$-\left(-(\mu+\delta+\alpha) - (\mu+\theta) - \lambda\right)\begin{bmatrix} (\mu+\delta+\alpha) - \lambda & \gamma & 0 \\ 0 & -(\mu+\gamma) & 0 \\ \alpha & 0 & -(\mu+\tau) - \lambda \end{bmatrix} -$$

$$(\mu+\delta+\alpha)\begin{bmatrix} \beta\dfrac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha) + \tau\alpha} & \gamma & 0 \\ \theta & -(\mu+\gamma) - \lambda & 0 \\ \alpha & 0 & -(\mu+\tau) - \lambda \end{bmatrix} -$$

$$\tau\begin{bmatrix} \beta\left[\dfrac{\beta\Lambda - (\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha) + \tau\alpha}\right] & (\mu+\delta+\alpha) - \lambda & \gamma \\ \theta & 0 & -(\mu+\gamma) - \lambda \\ 0 & \alpha & 0 \end{bmatrix}$$

$$= -\left(\mu+\delta+\alpha-(\mu+\theta)-\lambda\right)\left[(\mu+\delta+\alpha)-\lambda\right]\begin{bmatrix}-(\mu+\gamma)-\lambda & 0\\ 0 & -(\mu+\tau)-\lambda\end{bmatrix}-$$

$$\gamma\begin{bmatrix}\theta & 0\\ \alpha & -(\mu+\tau)-\lambda\end{bmatrix}-(\mu+\delta+\alpha)\left[\beta\frac{\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}\right]\begin{bmatrix}-(\mu+\gamma)-\lambda & 0\\ 0 & -(\mu+\tau)-\lambda\end{bmatrix}$$

$$-\tau\left\{\beta\frac{\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}\begin{bmatrix}\theta & -(\mu+\gamma)-\lambda\\ \alpha & 0\end{bmatrix}-(\mu+\delta+\alpha)-\lambda\begin{bmatrix}\theta & -(\mu+\gamma)-\lambda\\ 0 & 0\end{bmatrix}\right\}+$$

$$\gamma\begin{bmatrix}\theta & 0\\ 0 & \alpha\end{bmatrix}=0$$

Solving and selecting the dominant eigen value, we obtain;

$$\lambda=\frac{[\mu+\delta+\alpha]\left\{\beta\left[\beta\left(\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)(\mu+\gamma)\right)\right]\right\}}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}$$

Therefore; the basic reproduction number of the endemic equilibrium is;

$$R_0=\frac{[\mu+\delta+\alpha]\left\{\beta\left[\beta\left(\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)(\mu+\gamma)\right)\right]\right\}}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}$$

(13)

Whether the disease persists or dies out in the population depends on the magnitude of the basic reproduction number. The DEE is asymptotically stable if $R_0<1$ and unstable if $R_0>1$. On the other hand, the EE is locally asymptotically stable when $R_0>1$ and unstable when $R_0<1$.

Lemma: in order to control the spread of TB in any population, effort must be made to ensure that the EE is unstable, $R_0>1$.

Theorem: If $R_0>1$ then the endemic equilibrium is asymptotically stable.

The characteristic equation is $\left[J-I\lambda\right]$ =0. Taking the dominant eigenvalue,

the basic reproduction number is.

$$R_0 = \frac{[\mu+\delta+\alpha]\left\{\beta\left[\beta\left(\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)(\mu+\gamma)\right)\right]\right\}}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}$$

The Endemic Equilibrium is locally asymptotically stable when $R_0 > 1$ and unstable when $R_0 < 1$.

Proof:

If $R_0 < 1$the disease-endemic e

$$\frac{[\mu+\delta+\alpha]\left\{\beta\left[\beta\left(\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)(\mu+\gamma)\right)\right]\right\}}{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha} < 1$$

$$\gamma < \frac{(\mu+\tau)(\mu+\delta+\alpha)+\tau\alpha}{[\mu+\delta+\alpha]\left\{\beta\left[\beta\left(\beta\Lambda-(\mu+\tau)(\mu+\delta+\alpha)(\mu+\tau)(\mu+\gamma)\right)\right]\right\}} - \mu$$

If $R_0 > 1$ then,

The disease endemic equilibrium is asymptotically stable. Hence the disease will continue to exist in the population. Otherwise, it will die out with time if $R_0 < 1$.

4.0 Conclusion

In this paper, the effect of latently infected population on the transmission of TB was analyzed. Since the population that is latently infected cannot be identified, they can easily spread the disease during their transmission from latent to infectious and if there is a random mixing of individuals in the population. The duration of the latent infection is not known and varies from one individual to another. The endemic equilibrium state of the model using basic reproduction number shows that TB can be effectively controlled if the rate of both the latently and infectious class is always less than one. From the results, as the transmission rate increases or as the recovery rate decreases, $R_0 > 1$ and the disease-free equilibrium is unstable. This indicates that the disease will spread when there is an outbreak. Consequently, as the

transmission rate decreases, or the recovery rate increases. $R_0 < 1$the DFE will be stable hence the disease will not spread

The model gave a basic reproductive number $R_0 > 1$, This means that the disease will persist in the population.

5.0 Recommendations

TB transmission can be minimized in the population if the effort is made to ensure that the endemic equilibrium of the model is never stable. This can be achieved if the following recommendations are considered;

1) People should be enlightened on the mode of TB transmission dynamics and home care strategies of people with TB.
2) The government should intensify the education on TB in the churches, schools, to the individuals in the communities of its existence, free access to medical care and treatment duration.
3) The government should integrate TB programs into other existing health services such as outreach, maternal and child welfare programs among others in order to increase its awareness.

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