

# Mathematical Analysis Of The Endemic Equilibrium Of The transmission Dynamics Of Tuberculosis.

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**Abstract:** In this study, we modeled the effect of vaccination and treatment on the transmission dynamics of Tuberculosis (TB). The analysis of the Endemic equilibrium state of the model, using the Basic Reproduction number,  $R_0$  shows that TB can effectively be controlled or even be eradicated if effort is made to ensure that the total removal rate from both the Latent and the Infectious classes is always less than the product of total contraction and total breakdown of the Susceptible class.

**Index Terms:** Transmission Dynamics, Basic Reproduction Number, Endemic Equilibrium State, Latent Class, Infectious Class, Susceptible Class.

## 1. INTRODUCTION

Tuberculosis or TB (short for Tubercles Bacillus) is an air borne and highly infectious disease caused by infection with the bacteria mycobacterium tuberculosis. [7]. An exposed or Susceptible individual is infected with the disease when he or she inhales the TB germs which are released into the air when infected individuals cough, sneeze, spit or talk. The first period of infection is the period of Latency when individual exhibits no symptoms of the disease and is not infectious to others. Such an individual is said to have Latent TB infection. The second stage is the period of Active TB infection when the individual start to exhibit some or all the symptoms of TB. The Latent period can be extremely variable as a great majority ( $\approx 90\%$ ) may live with the disease as long as possible without it progressing to Active TB whereas a small proportion ( $\approx 10\%$ ) will progress to Active TB infection, falling ill within months or several years after infection.[2]. An individual with Active TB infection may recover without any treatment, may be cured with drugs or may die from the disease. The degree of protection afforded by a previous infection and the mechanism by which individuals with partial immunity are protected are controversial. The highest risk group to acquire TB when exposed to it are children under five years of age, persons who are immuno-compromised (i.e. have weakened immunity), especially those who are HIV-Positive, persons who have diabetes or kidney failure, people that take excessive alcohol and drugs, those with poor nutrition and lack of food, those suffering from stress and those living in poorly ventilated rooms [13].

Tuberculosis usually attacks the lungs but can also attack other parts of the body like the kidney, Spine, brain, bones, joints etc.[4]. The classic symptoms of TB of the lungs are a chronic cough which may result in blood-tinged sputum, fever, night sweats, loss of appetite, weight loss and fatigue. Infection of other organs causes a wide range of symptoms. Pneumonia, lung collapse and enlarged lymph nodes may also occur.[16]. Two forms of tuberculosis that become life- threatening are:

1. Miliary TB, which means the bacteria have spread throughout the lungs and into the bloodstream.
2. TB meningitis (infection of the covering of the spinal cord and /or brain by TB bacteria).

Diagnosis relies on radiology (commonly chest X- ray), a tuberculin skin test, blood tests, as well as microscopic examination and microbiological culture of bodily fluids (such as sputum). Prevention relies on screening programmes and vaccination, usually with Bacillus calmette - Guérin (BCG) vaccine given to infants. The Directly Observed Treatment Short (DOTS) Course is the internationally recommended strategy for the control and cure for TB. Treatment for tuberculosis 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 makes many antibiotics ineffective and hinders the entry of drugs. [11],[1],[5]. The two antibiotics most commonly used are Rifampicin and Isoniazid. However, instead of the short course of antibiotics typically used to cure other bacterial infections, TB requires much longer periods of treatment (around 6 to 24 months) to entirely eliminate mycobacterium from the body. (Center for Disease Control and Prevention [6]. TB has remain a global problem despite many decades of study, the wide spread availability of vaccines, an arsenal of anti-microbial drugs as well as a highly visible World Health Organisation WHO effort to promote a unified global strategy. The World Health Organization (WHO) declared Tuberculosis (TB) a global emergency in 1993 and it remains one of the world's major causes of illness and death. One third of the world's population, two billion people, carry the TB bacteria. More than nine million of these become sick each year with active TB that can be spread to others [15]. TB poses significant challenges to developing economies as it primarily affects people during their most productive years. More than 90% of new TB cases and deaths occur in developing countries. Nigeria

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ranks 10th among the 22 high-burden TB countries in the world. WHO estimates that 210,000 new cases of all forms of TB occurred in the country in 2010, equivalent to 133/100,000 population. There were an estimated 320,000 prevalent cases of TB in 2010, equivalent to 199/100,000 cases[15]. The TB burden is compounded by a high prevalence of HIV in the country which stands at about 4.1% in general population. The age groups commonly affected by TB are the most productive age groups, with the 25 – 34 age group accounting for 33.6% (15,303) of the smear positive cases registered in 2010.[15].

## 2. THE MODEL:

Mathematical models have played a key role in the formulation of TB control strategies and the establishment of interim goals for intervention programme. Most of these models are of the SEIR class, in which the host population is categorized by infection status as Susceptible, Exposed (infected but not yet infectious), Infectious and Recovered. One of the principal attribute of these models is that the force of infection (the rate at which susceptible leave the susceptible class and move into the infected category i.e. become infected) is a function of the number of infectious hosts in the population at any time  $t$  and is thus a non-linear term. Other transitions such as the recovery of infectious individuals and death are modeled as linear terms with constant coefficients. Our model is a deterministic or compartmental, MSEIR- type model where the population is partitioned into 5 components or classes based on the epidemiological state of individuals, and it is assumed that the population size in a compartment is differentiable with respect to time and that the epidemic process is deterministic. Therefore, the TB transmission dynamics between the compartments shall be described by a system of differential equation which shall be solved to obtain both the disease-free equilibrium state and the endemic equilibrium state. The stability analysis of the disease-free equilibrium state shall be carried out using the Routh-Hurwitz criterion while that of the endemic equilibrium state shall be done using the reproduction number,  $R_0$ .

### 2.1 ASSUMPTIONS OF THE MODEL

The model is based on the following assumptions.

1. That the population is heterogeneous. That is, the individuals that make up the population can be grouped into different compartments or groups according to their epidemiological state.
2. That the population size in a compartment is differentiable with respect to time and that the epidemic process is deterministic. In other words, that the changes in population of a compartment can be calculated using only history to develop the model.
3. That a proportion of the population of newborns is immunized against TB infection through vaccination.
4. That the immunity conferred on individuals by vaccination expires after some time at a given rate.
5. That the population mixes homogeneously. That is, all susceptible individuals are equally likely to be infected by infectious individuals in case of contact.

6. That the infection does not confer immunity to the cured and recovered individuals and so they go back to the susceptible class at a given rate.
7. That people in each compartment have equal natural death rate of  $\beta$ .
8. That all newborns are previously uninfected by TB and therefore join either the immunized compartment or the susceptible compartment depending on whether they are vaccinated or not.
9. That there are no immigrants and emigrants. The only way of entry into the population is through new – born babies and the only way of exit is through death from natural causes or death from TB-related causes.

### 2.2 MODEL VARIABLES AND PARAMETERS

The following variables and parameters shall be used in this model.

- $M(t)$ : the number of individuals who are immunized against TB through Vaccination at time  $t$   
 $S(t)$ : the number of susceptible individuals at time  $t$   
 $L(t)$ : the number of latently infected / exposed individuals at time  $t$ .  
 $I(t)$ : the number of infectious individuals at time  $t$ .  
 $R(t)$ : the number of individuals who have been treated and have recovered from the infection at time  $t$ .  
 $\phi$ : the rate of expiration of vaccine efficacy.  
 $\kappa$ : the rate at which susceptible individuals become latently infected by TB.  
 $\mu$ : the rate at which latently infected individuals become actively infected.  
 $\psi$ : the rate at which actively infected individuals recover from TB infection.  
 $q$ : the rate at which individuals who are latently infected recover from TB through treatment.  
 $\pi$ : the rate at which recovered individuals become susceptible to TB Infection again.  
 $\eta$ : the tuberculosis-induced mortality / death rate.  
 $\beta$ : the natural mortality / death rate  
 $P$ : population of new births joining the population  $N$ .  
 $cP$ : the proportion of new births that have been immunized through Vaccination.  
 $N$ : the total population size

### 2.3 MODEL DESCRIPTION

Based on the standard SEIR model, the population is partitioned into 5 compartments or classes namely: Immunized  $M(t)$ , Susceptible  $S(t)$ , Latent  $L(t)$ , Infectious  $I(t)$  and Recovered  $R(t)$  compartments. The Immunized component increases due to the coming in of the immunized newborns into the population, where we assumed that a proportion,  $cP$ , of the incoming individuals are immunized through vaccination. The component reduces due to the progression of people in this class to the Susceptible class as a result of the expiration of the duration of vaccine efficacy at the rate of  $\phi$  and also as a result of natural death at the rate of  $\beta$ . The susceptible component of the population grows due to the coming in of new- born babies not immunized against TB infection into the population at the rate of  $(1-c)P$ , the coming in of some recovered individuals due to the fact that the infection does not confer immunity to recovered individuals, at the rate of  $\pi$  and as a result of the expiration of the efficacy of the

vaccine, at the rate of  $\phi$ . This component decreases due to the latent infection of individuals at the rate of  $\kappa$  and due to death from natural causes at the rate of  $\beta$ . The population of the latent component grows as a result of infection of individuals in the susceptible class at the rate of  $\kappa$ . This class reduces due to the progression of latently infected individuals to active TB infection at the rate of  $\mu$ , the successful treatment and cure of latent TB patients at the rate of  $q$  and as a result of death from natural causes at the rate of  $\beta$ . The infectious compartment increases due to the progression of latently infected individuals to active TB infection at the rate of  $\mu$ . The component reduces as a result of successful cure of infectious TB patients at the rate of  $\psi$ , death as a result of active TB infection at the rate of  $\eta$  and also due to death from natural causes at the rate of  $\beta$ . Lastly, the recovered component grows as a result of successful treatment and cure of latent TB patients at the rate of  $q$  and that of the infectious TB patient at rate of  $\psi$  and decrease due to the fact that recovered individuals are not immune against the infection and so they return to the susceptible class at the rate of  $\pi$  and also as a result of death from natural cause at the rate of  $\beta$ . (Enagi & Ibrahim,2011) The model can be described by a flow chart as shown in figure 3.1.

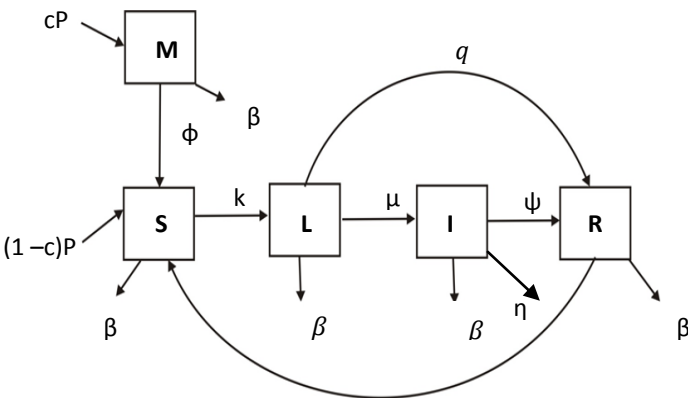


Figure 3.1: A Flow Chart of the model

**2.4 THE MODEL EQUATIONS**

Applying the assumptions and the inter-relations between the variables and the parameters as described in section 3.3, the role of vaccination and treatment on tuberculosis transmission dynamics can be describe by the following differential equations:

$$\frac{dM}{dt} = cP - (\phi + \beta)M \tag{2.1}$$

$$\frac{dS}{dt} = (1 - c)p + \phi M + \pi R - (\kappa I + \beta)S \tag{2.2}$$

$$\frac{dL}{dt} = \kappa SI - (q + \mu + \beta)L \tag{2.3}$$

$$\frac{dI}{dt} = \mu L - (\psi + \eta + \beta)I \tag{2.4}$$

$$\frac{dR}{dt} = qL + \psi I - (\pi + \beta)R \tag{2.5}$$

$$N(t) = M(t) + S(t) + L(t) + I(t) + R(t) \tag{2.6}$$

**2.5 EQUILIBRIUM SOLUTIONS**

Let E (M, S, L, I, R) be the equilibrium point of the system described by the equations (2.1) – (2.6). At the equilibrium state, we have

$$\frac{dM}{dt} = \frac{dS}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

That is,

$$cP - (\phi + \beta)M = 0 \tag{2.7}$$

$$(1 - c)P + \phi M + \pi R - (\kappa I + \beta)S = 0 \tag{2.8}$$

$$\kappa SI - (q + \mu + \beta)L = 0 \tag{2.9}$$

$$\mu L - (\psi + \eta + \beta)I = 0 \tag{2.10}$$

$$qL + \psi I - (\pi + \beta)R = 0 \tag{2.11}$$

**2.6 The Endemic Equilibrium State**

The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. For the disease to persist in the population, the Immunized Class, the susceptible class, the Latently Infected Class, the Infectious class and the Recovered Class must not be zero at equilibrium state. In other words, if  $E^*(M^*, S^*, L^*, I^*, R^*)$  is the endemic equilibrium state, then  $E^*(M^*, S^*, L^*, I^*, R^*) \neq (0,0,0,0,0)$ . In order to obtain the endemic equilibrium state, we solve equations (2.7 – 2. 11) simultaneously taking into consideration the fact the  $E^*(M^*, S^*, L^*, I^*, R^*) \neq (0,0,0,0,0)$ . From Equation (2.7)

$$cP - (\phi + \beta)M = 0$$

$$\Rightarrow M^* = \frac{cP}{\phi + \beta} \tag{2.12}$$

From Equation (2.10),

$$\mu L - (\psi + \beta + \eta)I = 0$$

$$\Rightarrow L = \frac{(\psi + \beta + \eta)I}{\mu} \tag{2.13}$$

Substituting Equation (2.13) into Equation (2.9) gives

$$\kappa SI - (q + \mu + \beta) \frac{(\psi + \beta + \eta)I}{\mu} = 0$$

$$\Rightarrow I \left[ \kappa S - \frac{(q + \mu + \beta)(\psi + \beta + \eta)}{\mu} \right] = 0 \tag{2.14}$$

From Equation (2.14),

$$\left[ \kappa S - \frac{(q + \mu + \beta)(\psi + \beta + \eta)}{\mu} \right] = 0 \tag{2.15}$$

(since  $I \neq 0$ )

$$\text{So, } S^* = \frac{(q + \mu + \beta)(\psi + \beta + \eta)}{\kappa \mu} \tag{2.16}$$

Substituting Equation (2.13) for L into Equation (2.11) gives:

(2.25)

$$q\left[\frac{(\psi+\beta+\eta)I}{\mu}\right] + \psi I - (\pi + \beta)R = 0$$

$$\Rightarrow R = \frac{[q(\psi+\beta+\eta)+\mu\psi]I}{\mu(\pi+\beta)} \quad (2.17)$$

Adding Equation (2.7) and Equation (2.8) we have:

$$cP - (\varphi + \beta)M + (1-c)P + \varphi M + \pi R$$

$$- (kI + \beta)S = 0$$

$$P - \beta M + \pi R - \kappa SI - \beta S = 0$$

$$P - \beta M + \pi R - \beta S = \kappa SI \quad (2.18)$$

Substituting Equation. (2.18) into Equation. (2.9) gives

$$P - \beta M + \pi R - \beta S - (q + \mu + \beta)L = 0 \quad (2.19)$$

Adding Equation (2.19) and Equation (2.10) gives

$$P - \beta M + \pi R - \beta S - (q + \mu + \beta)L + \mu L$$

$$- (\psi + \beta + \eta)I = 0$$

$$\Rightarrow P - \beta M + \pi R - \beta S - (q + \beta)L - (\psi + \beta + \eta)I = 0 \quad (2.20)$$

Adding Equation (2.20) and Equation (2.11) gives

$$P - \beta M + \pi R - \beta S - (q + \beta)L - (\psi + \beta + \eta)I + qL$$

$$+ \psi I - (\pi + \beta)R = 0$$

$$\Rightarrow P - \beta M - \beta S - \beta L - \beta I - \eta I - \beta R = 0$$

$$\text{Or } P - \beta M - \beta S - \beta L - (\beta + \eta)I - \beta R = 0 \quad (2.21)$$

Substitute Equations (2.12), (2.16), (2.13) and (2.17) for M, S, L and R into Equation (2.21) to have

$$P - \beta \left[ \frac{cp}{\varphi + \beta} \right] - \beta \left[ \frac{(q + \mu + \beta)(\psi + \beta + \eta)}{k\mu} \right]$$

$$- \beta \left[ \frac{(\psi + \beta + \eta)}{\mu} \right] - (\beta + \eta)I - \beta \left[ \frac{[q(\psi + \beta + \eta) + \mu\psi]I}{\mu(\pi + \beta)} \right] = 0 \quad (2.22)$$

From equation (2.22) have that

$$I^* = \frac{[k\mu p(\varphi + \beta) - k\mu\beta cP - \beta(\varphi + \beta)(q + \mu + \beta)(\psi + \beta + \eta)](\pi + \beta)}{k(\varphi + \beta)\{(\pi + \beta)[\beta(\psi + \beta + \eta) + \mu(\beta + \eta)] + \beta[q(\psi + \beta + \eta) + \mu\psi]\}} \quad (2.23)$$

Substituting Equation (2.23) for I into Equation (2.13) and Equation (2.17) gives

$$L^* = \frac{(\psi + \beta + \eta)[k\mu p(\varphi + \beta) - k\mu\beta cP - \beta(\varphi + \beta)(q + \mu + \beta)(\psi + \beta + \eta)](\pi + \beta)}{k\mu(\varphi + \beta)\{(\pi + \beta)[\beta(\psi + \beta + \eta) + \mu(\beta + \eta)] + \beta[q(\psi + \beta + \eta) + \mu\psi]\}} \quad (2.24)$$

And

$$R^* = \frac{[q(\psi + \beta + \eta) + \mu\psi][P k\mu(\varphi + \beta) - k\mu\beta cP - \beta(\varphi + \beta)(q + \mu + \beta)(\psi + \beta + \eta)]}{k\mu(\varphi + \beta)\{(\pi + \beta)[\beta(\psi + \beta + \eta) + \mu(\beta + \eta)] + \beta[q(\psi + \beta + \eta) + \mu\psi]\}}$$

Therefore, the endemic equilibrium state is  $E^*(M^*, S^*, L^*, I^*, R^*)$

### 3. STABILITY ANALYSIS OF THE ENDEMIC EQUILIBRIUM STATE

Recall that the Jacobian Matrix of this model is

$$J = \begin{pmatrix} -(\varphi + \beta) & 0 & 0 & 0 & 0 \\ \varphi & -(\kappa I + \beta) & 0 & -\kappa S & \pi \\ 0 & \kappa I & -(q + \mu + \beta) & \kappa S & 0 \\ 0 & 0 & \mu & -(\psi + \beta + \eta) & 0 \\ 0 & 0 & q & \psi & -(\pi + \beta) \end{pmatrix}$$

At the endemic equilibrium state,  $E^*$ , the Jacobian Matrix becomes

$$J^* = \begin{pmatrix} -(\varphi + \beta) & 0 & 0 & 0 & 0 \\ \varphi & -(\kappa I^* + \beta) & 0 & -\kappa S^* & \pi \\ 0 & \kappa I^* & -(q + \mu + \beta) & \kappa S^* & 0 \\ 0 & 0 & \mu & -(\psi + \beta + \eta) & 0 \\ 0 & 0 & q & \psi & -(\pi + \beta) \end{pmatrix}$$

The characteristics equation is  $|J^* - I\lambda| = 0$ .

Computations to obtain the eigen values from the characteristic equation is very complex and so this approach renders conclusion very difficult. Feng *et al.* (2001) described the use of basic reproduction number,  $R_0$  in analyzing the stability of the endemic equilibrium states. According to their work, when  $R_0 > 1$ , the system has a unique endemic equilibrium that is globally asymptotically stable. The same technique shall be adopted in this work to analysis the stability of the endemic equilibrium state.

#### 3.1 THE BASIC REPRODUCTION NUMBER, $R_0$ OF TUBERCULOSIS

Diekmann *et al.* (1990), defined the basic reproduction number,  $R_0$ , as the average number of secondary infections caused by an infectious individual during his / her entire life as an infectious person. Tuberculosis infection and re-infection are always existent in a community due to respiratory contact between the susceptible individuals, treated individuals, and the infectious individuals. Whether the disease becomes persistent or dies out depends on the magnitude of the basic reproductive number,  $R_0$ . Stability of equilibrium points can be analyzed using  $R_0$ . The disease-free equilibrium is locally asymptotically stable if  $0 < R_0 < 1$  and unstable if  $R_0 > 1$ . In other words, when  $0 < R_0 < 1$ , every infectious individual will cause less than one secondary infection and hence the disease will die out and when  $R_0 > 1$ , every infectious individual will cause more than one secondary infection and hence an epidemic will occur. All public health control measures are usually based on methods that, if effective, would lower  $R_0$  to below unity. (Ssematimba *et al.*, 2005). On the other hand, the endemic equilibrium is locally stable when  $R_0 > 1$  and unstable when  $0 < R_0 < 1$ . In order to control the spread of TB in any society effort must be made to ensure that the endemic equilibrium is unstable i.e ( $0 < R_0 < 1$ ) For the case of a single infected compartment,  $R_0$  is simply the product of the infection rate and the mean duration of the infection. However, for more complicated models with several infected compartments, this simple definition of  $R_0$  is



insufficient. For a more general situation we can estimate this parameter by investigating the stability of the infection-free equilibrium. (Okyere, 2007). The expression for  $R_0$  for Tuberculosis, which Blower *et al.* (1995), calculated from their simple model is given by:

$$R_0 = R_0^{FAST} + R_0^{SLOW}$$

Where,

$$R_0^{FAST} = \left(\frac{\beta b}{\mu}\right) \left(\frac{1}{\mu + \mu t}\right) \rho$$

$$R_0^{SLOW} = \left(\frac{\beta b}{\mu}\right) \left(\frac{1}{\mu + \mu t}\right) \left(\frac{(1-\rho)v}{v + \mu}\right)$$

In this model, it is assumed that the infected individuals can develop active TB by either direct progression (the disease develops immediately after infection) or endogenous reactivation (the disease develops after the infection). Because of these different ways of developing the disease, two types of TB must be modeled. These would be denoted as primary progressive TB (which is referred to as FAST Tuberculosis) and reactivation tuberculosis (which is referred to SLOW tuberculosis (Atkins, 2008). The expression for  $R_0$  for TB, which Blower *et al.*(1995), calculated from their more detailed model is given as:

$$R_0 = R_0^{FAST} + R_0^{SLOW} + R_0^{RELAPSE}$$

Where

$$R_0^{FAST} = \left(\frac{\beta b}{\mu}\right) \left(\frac{1}{\mu + \mu \theta + c}\right) \rho f$$

$$R_0^{SLOW} = \left(\frac{\beta b}{\mu}\right) \left(\frac{1}{\mu + \mu t + c}\right) \left(\frac{q(1-\rho)v}{v + \mu}\right)$$

$$R_0^{RELAPSE} = \left(\frac{\beta b}{\mu}\right) \left[ \frac{1}{(\mu + \mu t + c) \left( (\mu + \mu t + c) - \left( \frac{2\omega c}{2\omega + \mu} \right) \right)} \right] \left[ (p + \frac{(1-\rho)v}{v - \mu}) \left( \frac{\omega c}{2\omega + \mu} \right) \right]$$

These equations show that a Tuberculosis epidemic can be seen as a series of linked sub-epidemic (Blower *et al.*; 1995). The value of  $R_0$  in each of the sub-epidemics is determined by the product of three components:

1. The average number of infections that one infectious case causes per unit time.
2. The average time that an individual remains infectious (which is the same for FAST and SLOW TB but different for RELAPSE, and
3. The probability that a latent case will develop into an infectious case (which is different for FAST, SLOW or RELAPSE TB) (Atkins, 2008)

Ssematimba *et al.*, (2005) defined the reproduction number of tuberculosis in a density-dependent model as

$$R_0 = \left(\frac{\Lambda}{A}\right) \left(\frac{(\beta_1 + \beta_2)c}{\mu + d + r_2}\right) \left(\frac{\kappa}{\mu + \kappa + r_1}\right)$$

Where,

$\left(\frac{\Lambda}{A}\right)$  is the density of the susceptible population.

$\beta_1 c$  and  $\beta_2 c$  are the effective transmission rates.

$\left(\frac{1}{\mu + d + r_2}\right)$  is the effective infectious period.

$\left(\frac{(\beta_1 + \beta_2)c}{\mu + d + r_2}\right)$  is the number of latent infections produced by a typical infectious individual during the mean infectious period.

$\left(\frac{\kappa}{\mu + \kappa + r_1}\right)$  is the probability of progression from latent stage into the infectious stage.

Adopting this definition for our model we have that  $R_0$  is given by:

$$R_0 = \left(\frac{(1-c)P + \varphi + \pi}{\beta + k}\right) \left(\frac{k}{q + \mu + \beta}\right) \left(\frac{\mu}{\psi + \beta + \eta}\right)$$

Where

$\left(\frac{(1-c)P + \varphi + \pi}{\beta + k}\right)$  is the average number of individuals in the susceptible class.

$\left(\frac{k}{q + \mu + \beta}\right)$  is the number of latent infections produced by a typical infectious individuals during the mean infectious period.

$\left(\frac{\mu}{\psi + \beta + \eta}\right)$  is the probability of progressing from the latent class into the infectious class. For the endemic equilibrium state to be globally asymptotically stable we must have  $R_0 > 1$ . That is,

$$\left(\frac{(1-c)P + \varphi + \pi}{\beta + k}\right) \left(\frac{k}{q + \mu + \beta}\right) \left(\frac{\mu}{\psi + \beta + \eta}\right) > 1$$

$$\Rightarrow \frac{k\mu[(1-c)P + \pi]}{(\beta + k)(q + \mu + \beta)(\psi + \beta + \eta)} > 1$$

$$\Rightarrow k\mu[(1-c)P + \pi] > (\beta + k)(q + \mu + \beta)(\psi + \beta + \eta)$$

$$\Rightarrow \frac{k\mu[(1-c)P + \varphi + \pi]}{(\beta + k)} > (q + \mu + \beta)(\psi + \beta + \eta) \quad (3.1)$$

The inequality (3.1) gives the necessary and sufficient condition for the endemic equilibrium state of the model to be globally asymptotically stable. The interpretation is that, for the endemic equilibrium state to be globally asymptotically stable, the product of total contraction and total breakdown of the Susceptible class given by  $\frac{k\mu[(1-c)P + \varphi + \pi]}{(\beta + k)}$  must be greater than the total removal rate from both the Latent and the Infectious classes given by  $(q + \mu + \beta)(\psi + \beta + \eta)$ . In order to control TB, we must ensure that  $R_0 < 1$ . ( that is ,the Endemic equilibrium state is never stable.)

#### 4. CONCLUSION

In this study, we modeled the role vaccination and treatment play in the dynamics of Tuberculosis. The result of the stability analysis of the endemic equilibrium state shows that it is possible to effectively control or even completely eradicate tuberculosis in any population if effort is made to ensure that the product of total contraction and total breakdown of the Susceptible class given by  $\frac{k\mu[(1-c)P+\varphi+\pi]}{(\beta+k)}$  must be greater than the total removal rate from both the Latent and the Infectious classes given by  $(q + \mu + \beta)(\psi + \beta + \eta)$ . That is,

$$\frac{k\mu[(1-c)P+\varphi+\pi]}{(\beta+k)} > (q + \mu + \beta)(\psi + \beta + \eta)$$

This can be achieved through more effort at immunizing newborn babies through vaccination and through more effort at the treatment of both the latently infected and the actively infected individuals.

#### 5. RECOMMENDATIONS

The incidence of tuberculosis can greatly be minimized or possibly be eradicated in any population if effort is made to ensure that the endemic equilibrium of this model is never stable. That is  $R_0 < 1$ . This can be achieved if the following recommendations are considered.

1. There should be more enlightenment campaign on the dangers of TB and on its symptoms
2. More effort should be made to encourage people to voluntarily go for TB tests by discouraging stigmatization of people infected by the disease.
3. TB tests and treatment should continue to be free-of-charge to enable poor people assess them.
4. People should be educated on the mode of transmission of the disease and on home-care strategies for people infected by the disease.
5. The conditions that promote rapid spread of TB should be discouraged. Such conditions include: overcrowded accommodation, high level of illiteracy, lack or inadequate medical facilities, the tradition of giving birth to many children.
6. There should be provision of more trained personnel and more TB laboratory microscopy services.
7. People who are on treatment for TB should be educated on the need to complete their treatment course. This will help reduce the incidence of drug resistance currently on the increase.
8. People should be educated on the positive relationship between Tuberculosis and HIV/AIDS so that people who are living with HIV or who have AIDS could quickly go for TB tests.
9. All National and International agencies in charge of TB control should be better funded to enable them to continue to provide vaccines, test kits, and drugs for TB prevention, detection and treatment.
10. Assistance of more international organizations should be sort to join those currently at the forefront of the fight for the control of TB.
11. There should be increase in the number of TB care detection. This can be achieved by providing

more test kits and by encouraging people to go for TB tests.

#### REFERENCES.

- [1]. P.V. Acharya and D.S Goldman (1970). "Chemical composition of the cell all of the H37Ra strain of Mycobacterium tuberculosis." (<http://www.ncbi.nlm.nih.gov/pubmed/4988039>).
- [2]. T. Atkins, (2008). "Modeling Transmission Dynamics of Tuberculosis, Including various latent periods". A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the Department of Mathematics in the college of sciences at the University of Central Florida, Orlando Florida USA.
- [3]. S. M. Blower ,A.R McLean ,T.C Porco ;P.M Small;P.C Hopewell ;M.A Sanchez and A.R Moss (1995). "The Intrinsic transmission dynamics of tuberculosis epidemics": Nature Medicine (8): 815 – 821.
- [4]. S.M.Blower ,P.M. Small and P.C.Hopewell (1996). "Control strategies for Tuberculosis epidemics: new models for old problems." Science, 273 (5274): 497 – 500.
- [5]. P.JBrennan and H. Nikaido. (1995). "The envelope of mycobacteria". Annu. Rev. Biochem. 64 : 29 – 63. (<http://www.ncbi.nlm.nih.gov/pubmed/7574484>).
- [6]. Centers for Disease Control and Prevention (CDC) (2000). "Emergence of mycobacterium tuberculosis with extensive resistance to second line drugs – worldwide, 2000 - 2004" (<http://www.ncbi.nlm.nih.gov/pubmed/16557213>).
- [7]. C.Coliijn., T.Cohen and M. Murray (2006). "Mathematical models of Tuberculosis: Accomplishments and Future Challenges". (<http://www.Medscape.com>).
- [8]. J.A.Diekmann, , P.Heesterbeek and Metz J.A.J (1990). "On the definition and the computation of the basic reproduction ratio,  $R_0$  in models for infectious diseases in heterogeneous populations." Mathematical Biosciences, 28: 363 – 382.
- [9]. A.I.Enagi, and M.O.Ibrahim, (2011). "A mathematical Model of Effect of Bacillus Calmette-Guérin Vaccine and Isoniazid Preventive Therapy in Controlling the Spread of Tuberculosis in Nigeria." Journal of Modern Mathematics and Statistics 5(1): 25 – 29.
- [10]. Z.Feng,;W. Huang and C. Castillo-Chavez, (2001). "On the role of variable latent periods in mathematical models for tuberculosis." Journal of Dynamics and Differential Equations, 13(2) : 425 – 452.

- [11]. D.Migliore : N.P.V. Acharya P. Jolles (1966).  
“Characterization of large quantities of glutamic acid in the walls of human virulent strains of mycobacteria”.  
(<http://www.ncbi.nlm.nih.gov/pubmed/4958543>).
- [12]. E.Okoyere, (2007). “Deterministic Compartmental models for HIV and TB. African Institute for Mathematical Sciences”.  
<http://resources.aim.ac.za/archive/2006/eric.pdf>.
- [13]. G.G.Sanga, (2008). “Modeling the role of Diagnosis and Treatment on Tuberculosis (TB) Dynamics.” A thesis submitted in partial fulfillment of a post graduate diploma at African Institute for mathematical Sciences (AIMS).
- [14]. A.Ssematimba ;J.Y.T.Mugisha, and L.S Luboobi, (2005). “Mathematical models for the dynamics of tuberculosis in density dependent populations: The case of internally displaced peoples’ camps (IDPCS) in Uganda.” Journal of Mathematics and Statistics 1(3): 217 – 224.
- [15]. United State Embassy in Nigeria (USEN) (2012). “Nigeria. Tuberculosis Fact Sheet”  
(<http://nigeria.usembassy.gov> )
- [16]. World Health Organization. (2007) “Tuberculosis”  
<http://who.int/mediacentre/factsheets/fs104/en/index.html>.Retreived 12 November 2009.