

A Mathematical Modelling on Tuberculosis Dynamics Incorporating Treatment

(A Case Study of General Hospital Potiskum, Yobe State)

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Abstract

Original Research Article

Tuberculosis was declared an emergency in Nigeria and strategies for the control of tuberculosis in Nigeria were adopted to reduce its prevalence to a level at which the disease will no longer constitute public health problems. In this work, we presented a deterministic (SEIR susceptible, expose/latently infected, infectious and recovered) model incorporating the method of control adopted by national tuberculosis and leprosy control program. We established the disease free and the endemic equilibrium states and carried out the stability analysis of the disease. Free and the equilibrium state. We also carried out numerical simulation of the model to have an insight into the dynamics of the model. We found out that the disease free equilibrium state is stable. The numerical results showed that it would be very difficult to completely eradicate tuberculosis from Nigeria using the method adopted by national tuberculosis and leprosy control program.

Keywords: Differential Equation, Mathematical Modeling, SEIR, susceptible, expose/latently infected, infectious and recovered Tuberculosis (TB).

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INTRODUCTION

Tuberculosis (TB) is a contagious bacterial infection caused by mycobacterium tuberculosis. It usually affects the lungs (pulmonary tuberculosis). It can also affect the central nervous system, the lymphatic system, the brain, spine and the kidneys. The only peoples who have pulmonary TB infectious are the one third of the world population is currently infected with the TB bacillus and new infection are occurring at the rate of one per second. Tuberculosis was among the top ten causes of death worldwide in 2015 when 10.4 million people become ill from TB of which 1.8 million people died from TB including 400.000 with HIV + TB. The disease is airborne and so it's primary transmitted through the respiratory route. When people, who are infected with the disease cough, sneeze spit or talks, the propel TB germs in mucus droplets, known as bacilli, go into the air. A previously uninfected person needs only a small number of these germs to be infected. Cohen et al. [1] One infected individual enters a period of latency. During which he exhibits no symptoms of the disease and not infectious to others. Such a person is said to

have a latent those infections. The latent period can be extremely available length of time. A great majority of those infected (90%) may live with the disease as long as possible without it degenerating or progressing into active TB. Mathematical models provide results such as thresholds, basic reproduction numbers, contact numbers and replacement numbers. This result can help health workers understand and predict the spread of an epidemic and evaluate potential effectiveness of the different control measure to be used. Tuberculosis usually attacks the lungs but can also attack other parts of the body like the kidney, spine, brain, bones, joints etc. The classic symptoms of TB of the lungs are a chronic cough which may result in blood – tinged sputum, fever, nigh, sweats, loss of appetite, weight loss and fatigue. Infection of other organs causes a wide range of symptoms, pneumonia, and lung collapse and enlarge lymph nodes may also occur. Two forms of tuberculosis that become life threatening are:

- Military TB, which means the bacteria have spread throughout the lungs and into the bloodstream.

- 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 programmed and vaccination, usually with bacillus calmette – Guerin (BCG) vaccine given to infants. The directly observed treatment short (DOTS) course is the internally 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 in effected and hinders the entry of drugs. The two antibiotics most commonly used are rifampicin and iconized. However , instead of the short course of antibiotics typically used to cure other bacteria in effected, TB requires much longer period of treatment (around 6 to 24 months) to entirely eliminate mycobacterium from the blood,(center for disease control and prevention). TB has remain a global problem despite many decades of study, the wide spread availability of vaccines, an arsenal of antimicrobial drugs as well as a highly visible world health organization 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 major cause deaths. One third of the world’s population people, carry the TB bacteria, more than nine million of these become sick each year with active TB that can be spread to others. TB poses significant challenges developing economics as primarily effects people during their most productive years. More than 90% of new TB cases and death occur in developing countries.

Nigeria is a country with population of about 140,000,000 people, has an estimated incidence of 311 cases per 100,000 populations. The estimate incidence for SM+ cases is 137 per 100,000 population and estimated prevalence of MDR-TB among new TB cases is 1.9% TB burden is further compounded by high national HIV prevalence of 4.4% (national tuberculosis and leprosy control programme, Abuja, 2016. WHO [2], national tuberculosis and leprosy program was established to provide f framework for the control of TB and leprosy in Nigeria. This was followed by the official launching in 2016 and adoption of dots strategy for the control of TB in the Nigeria in 2017. In 2016 TB was declared an emergency in Nigeria and the stop TB strategy for the control of TB in Nigeria was adopted to reduce the prevalence of tuberculosis to a level at which the disease will no longer constitute public health problems in the country,

BACKGROUND OF THE STUDY

On March 24, 1882 Dr. Robert Koch announced the discovery of Mycobacterium tuberculosis, the bacteria that cause tuberculosis TB. During this time, TB killed one out of every seven people living in the United States and Europe. Dr. Koch’s discovery was the most important steps taken toward the control and elimination of this deadly diseases. The National Tuberculosis & Leprosy Control Program (NTLCP) was established in 1989 and officially launched in February 1991. Its mandate was to coordinate TB and Leprosy control activities in all states in Nigeria in order to significantly reduce the public burden of the two diseases. The NTLCP, which is part of the Ministry of Health, controls most of the funding for work on TB in Nigeria. The Ministry of Health has made considerable progress with the drafting of the National Strategic Plan for Tuberculosis Control which aims to provide Universal Access to Prevention, Diagnosis and Treatment by 2020 in line with its commitments to the World Health Organization (WHO). However, there are several difficulties affecting the success of this plan, such as inadequate budget provisions, access to hard to reach areas, unreported TB cases and inadequate human resources technical capacity. In 1982 a century after Dr Koch’s announcement, the first world TB day was sponsored by the world health organization (WHO) and the international union against tuberculosis and lung diseases (IUATLD). The event was intended to educate the public about the devastating health and economic consequences of TB, its effect on developing countries, and its continued tragic impact on global health.

STATEMENT OF THE PROBLEM

Despite numerous management and control strategies of tuberculosis currently in place, tuberculosis continues to cause great health effect worldwide [3]. A number of studies over the past two decades have shown that tuberculosis contributes too may illness including cardiovascular disease, cancer and endocrine disease such as tuberculosis Colin and Jane [4]. The mathematical model for tuberculosis transmission will be formulated.

AIM AND OBJECTIVES

This project work is aim at formulating mathematical modelling on tuberculosis dynamics incorporating treatment. The following objectives are to be achieved:

- Formulated and analysis a mathematical model on the tuberculosis dynamic and treatment of the disease using SEIR model.
- To determine the stability analyses of the equilibrium points by using system of equations simultaneously.
- To obtain the basic reproduction number and determine equilibrium point by using Jacobian matrix.

SIGNIFICANCE OF THE STUDY

The significances of the study include the following

- The model will help to understand the dynamic and treatment TB.
- The study will also act as a base for further research on the tuberculosis dynamic and treatment and other related diseases.
- The study intends to contribute on strategies of addressing TB and how to reduce it among of TB infected individual.
- The study will create awareness and inform people about the effect of TB dynamic and treatment.

SCOPE AND LIMITATION

This work centered on formulating a mathematical model for tuberculosis dynamics and its analysis by obtaining the equilibrium solution state and stability of such state. This study limited to only tuberculosis disease, however, it can be extended to other epidemic with little modification.

RESEARCH QUESTIONS

- Does mathematical modelling provide a unique approach to gain basic knowledge in tuberculosis dynamic?
- What are the primary ways of contacting with a tuberculosis?
- Does tuberculosis attack the lungs only?
- What are the ways of getting result in mathematical modelling

LITERATURE REVIEW

MATHEMATICAL MODELS OF TUBERCULOSIS

A review on earlier works on the tuberculosis provides the prospective of the proposed study. Many mathematical models have been developed to address tuberculosis transmission dynamic and control.

A. I. Enagi [5], considers a deterministic compartmental model of tuberculosis control strategy adopted by national tuberculosis and leprosy control program. He established the disease free and the endemic equilibrium state and carried out the stability analysis of the disease free and the endemic equilibrium state. He also carried out numerical simulations of the model to have an insight into the dynamics of the model. He found out that the disease free equilibrium state is stable. The numerical simulation showed that it will be very difficult to complete eradicate tuberculosis from Nigeria using this method adopted by national tuberculosis and leprosy control program.

Mugisha *et al.*, [6], formulated mathematical models for the dynamics of tuberculosis in density population required to minimize and therefore eradicate tuberculosis. Both numerical and qualitative analyses were done and the effect of various in the area size and recruitment rates was investigated. Analysis showed

that there existence disease free – equilibrium point provided the characteristics area is greater than the probability of survival from latent stage to infectious stage and the number of latent infectious produced but a typical characteristics area per individual should be at least 0.25 square kilometer in order to minimize tuberculosis incidence. His work suggested that characteristic area can as well be looked at as environmental stressor that can lead to tuberculosis.

Jama [7], the tuberculosis (TB) mortality rate has declined by 37% worldwide since 2000, but the disease still remains 1 of the top 10 causes of death, according to the most recent Global TB Report 2017, released by the World Health Organization. Greater political commitment is needed to address the burden of the disease and meet the goal of ending the TB epidemic by 2030. Tuberculosis was the leading cause of death from a single infectious agent in 2016, ranking above HIV/AIDS. Worldwide, 10.4 million new TB infections were estimated in 2016—10% of which occurred in people with HIV—and about 1.7 million people died of the disease. More than 600 000 new cases of TB with resistance to the most effective first-line drug, rifampicin, were reported, including 490 000 multidrug-resistant TB infections.

Barron Lerner [8], in his paper, revisiting the tuberculosis research of Thomas holnee (2009–2015), examined the association between stress and tuberculosis. He urged that disease may lead to disease immune function and this thus to clinical disease. His study suggested that persons who had experienced stressful situation, such as divorce, death of spouse, or loss of a job were more likely to develop tuberculosis and less likely to recover from it. The scholars in the study also devised a numerical scale (social readjustment rating scale) that qualified stress events with control groups. They also emphasized the need to understand each patient history and view his/her tuberculosis infection as the culmination of a life of emotional hardship. However their study has been criticized by Theorell [9], for its inability to consider the fact that everybody respond differently to stressful situations.

Engel [10], also commented that holmes approach was causal may be bidirectional or even cyclic, thus their works fail to produce statistical convincing studies. On this point the researcher agree with Engels view regarding the gap in Holmes work.

WHO [3], tuberculosis (TB), is still a major cause of death and suffering worldwide. Its control is a global public health issue and therefore needs to be conceived and carried out along with the basic principles of equity, human right to health and social protection. As marginalized people are always greatly exposed to health problems and often face difficulties in accessing care, social and economic determinants of ill

health must be appropriately addressed together with adequate implementation of the specific interventions available today to combat those diseases that disproportionately affect the poorest. The MDGs, in short, called upon all governments to join forces to combat poverty and its consequences, including health conditions like TB, in order to foster development of societies and nations. In this context, community engagement played a pivotal role in reaching the most vulnerable groups, assessing their specific needs and promoting active mobilization of society. Authors like [18;19;20;24] had made more contribution in health regarding epidemiology and public health respectively.

Tracy *et al.*, [11], present a Meta – analysis of the literature on stress and immunity in humans. The result showed substantial evidence for a relation between stress and decrease in functional immune measures (proliferative response tomatoes and natural killer cell activity). The way neuroendocrine mechanism sand health practice might alternate stress has been discussed, evidence for the relations between stress and both functional and enumerative immune measures have been presented. The authors further stated that, stressor duration is important for immune outcomes, and interpersonal events are related to alterations incident immune parameters than social events. However, their work lacked a qualitative and numerical analysis which this study has covered.

According to Koriko and Yusuf [12], in their model “mathematical model to simulate tuberculosis disease population dynamics” TB disease population dynamics was presented their model was compartmentalized as appropriate and the resulting model equation were solved numerically white different instance of disease transmission were presented and addition, the graphical profile of the various sub – population with time were presented and equilibrium of the system were established and analyzed for stability . Using the SIR model, simulated results showed that the population dynamics depends more on the number of actively infected people in population at the initial time and also on the disease incidence transmission rate at giving time. Mostly importantly, they showed that the disease – free equilibrium is stable while the endemic equilibrium may not be stable depending on the model the model parameters. This model is importance to this study, however, the researchers intend to use a model which is the SEIR model, to determine the effect of stress on the transmission dynamics of tuberculosis.

Inyama [13], consider the role of vaccination of new born babies against tuberculosis and treatment of both latently and actively infected individual in controlling the spread of tuberculosis which was mathematically modeled based on the standard SEIR model. The disease – free equilibrium state of the model was established and its stability of the disease – free the routh – Hurwitz theorem. The result of their analysis of

the stability of the disease – free equilibrium state shows that tuberculosis can totally be eradicate if effort is made to ensure that the sum of the rate of recovery of the latent class, the rate at which latently effected Individuals becomes actively infected and the rate of natural death, must have a lower bound.

WHO [14], Tuberculosis research, what is needed new diagnostic tools are particularly a waited in order to improve the diagnosis of disease and latent infection, the rapid detection of drug-resistance, and for use in the pediatric population. Several new diagnostics or diagnostic methods have been endorsed by the WHO since 2017 and many others are under investigation. Whole-genome sequencing is being studied in an attempt to identify relevant mutations that could predict drug resistance: this approach may be potentially useful together with routine diagnostic tests in order to build up individualized therapeutic schemes in severe cases of MDR-TB and extensively drug-resistant TB (XDR-TB). 21 A number of new molecules are currently in the early phases of the development pipeline although it will probably take about a decade to put them into the market, provided that their efficacy and safety will be demonstrated. 22, 23 After many years of financially limited research, two new drugs for MDR-TB (bedaquiline and delamanid) were approved between 2015 and 2016. These two drugs may save lives although they will probably yield a little impact of the global epidemiology of TB for which shorter and more effective regimens for both drug-susceptible disease and latent infection are necessary. 23-25 Vaccines may eventually be the most effective response to TB but the development of effective vaccines is considerably hindered by the complex biology of mycobacteria, whose nature is still partially undefined. 26, 27 BCG, which was first introduced in 1921, is the only currently available vaccine, mostly unsuitable for preventing infection and TB disease and therefore with limited use in specific contexts and for specific target populations (such as children in the first 2-3 years of life where it prevents wide dissemination of bacilli). Authors like [21;22;23] had made a comprehensive gap regarding public health sectors respectively.

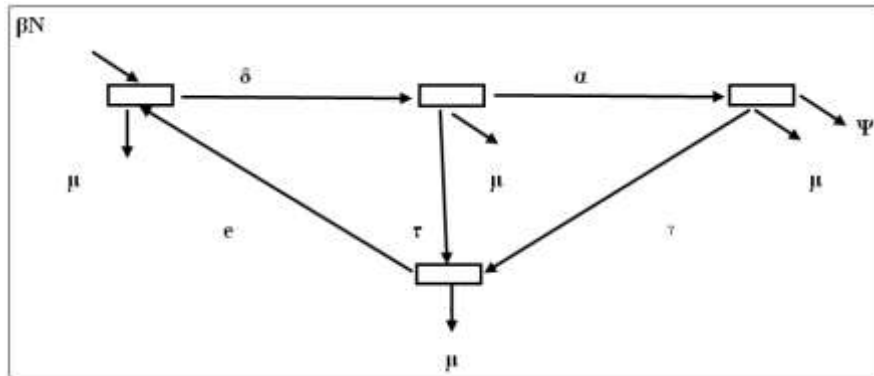
Caroline *et al.*, [15], used two mathematical models to explore the role of the contact structure of the population , and found out that in declining epidemics, localized outbreaks may occurs as a result of contact heterogeneity even in the absence of host or strain variability. They discussed the implications of their finding for tuberculosis control in low incidence setting. Results incidence that clustering of disease can emerge during declining TB epidemics without the explicit inclusion of host or strain divergence, in susceptibility and fitness. Thus some local outbreaks can be expected to occur even in the absence of this sources of individual heterogeneity, as a result of the contact structure of the population. Furthermore, their findings suggest that non – random mixing has a substantial

effect on clustering of the disease in low incidence areas, hence concluding that's non - random mixing in a population leads to the emergency of local disease clusters in declining TB epidemics which occurs even when the initial condition are spatially homogeneous and individual heterogeneity is excluded. Their work is relevant to this study in understanding the contact structure of TB dynamics although this study specially models the effect of stress on the transmission dynamics of tuberculosis.

MATERIALS AND METHODS

MODEL FORMULATION

In this study we formulated a deterministic, compartment model to investigate the transmission dynamics between infected and susceptible individual in a population the progression of tuberculosis within the total population can be simplified using four different equations representing four different groups of people namely, susceptible S(t), Latently infected L(t), infectious I(t), and Recovered R(t), individuals. Model Diagram.



To showing deterministic presentation of the model above

The susceptible population change due to the coming in of new susceptible into the population where the model parameters are at a constant rate βN (through birth or immigration), Natural death rate μ , Tuberculosis contraction rate α , Rate of breakdown of latent TB into infectious TB δ , successful cure of infectious TB patients γ and Death cause as a result of chronic TB Infection at the rate ψ , Successful cure of infectious latent τ and e is rate which recovered

individuals return to susceptible status due to loss immunity. Our study we assumed what there is homogeneous mixing of the population where all people are equally likely to be infected by infectious individuals in case of contact, we assumed equal natural death rate μ for each compartment. The model is represented by the following system of ordinary differential equation in developing the model which are as follow:

$$\frac{dS}{dt} = \beta N - \frac{\alpha SI}{N} + eR - \mu S \text{ ----- (3.1)}$$

$$\frac{dL}{dt} = \frac{\alpha SI}{N} - (\delta + \tau + \mu)L \text{ ----- (3.2)}$$

$$\frac{dI}{dt} = \delta L - (\gamma + \mu + \psi)I \text{ ----- (3.3)}$$

$$\frac{dR}{dt} = \gamma I + \tau L - (e + \mu)R \text{ ----- (3.4)}$$

Let

$$K_1 = \delta + \tau + \mu$$

$$K_2 = \gamma + \mu + \psi$$

$$K_3 = e + \mu$$

Taking (3.1)

$$\frac{dS}{dt} = \beta N - \frac{\alpha SI}{N} + eR - \mu S$$

$$\frac{dL}{dt} = \frac{\alpha SI}{N} - K_1 L \text{ ----- (3.5)}$$

$$\frac{dI}{dt} = \delta L - K_2 I \text{ ----- (3.6)}$$

$$\frac{dR}{dt} = \gamma I + \tau L - K_3 R \text{ ----- (3.7)}$$

EXISTENCE OF DISEASE FREE - EQUILIBRIUM (DFE) STATE

The disease free equilibrium is a steady state solution of the tuberculosis dynamic model with all

infected population equal to zero. The stability of the disease free equilibrium state is extremely important because it help us to investigate the long term behavior

of the system. It can determine whether or the bacterial are capable of invading a population. Now we recall the four equations (3.1) to (3.4).

$$\begin{aligned} \frac{dS}{dt} &= \beta N - \frac{\alpha SI}{N} + eR - \mu S \\ \frac{dL}{dt} &= \frac{\alpha SI}{N} - k_1 L \\ \frac{dI}{dt} &= \delta L - k_2 I \\ \frac{dR}{dt} &= \gamma I + \tau L - k_3 R \end{aligned}$$

At equilibrium, the rate of change of variable is zero i.e

$$\begin{aligned} \frac{dS}{dt} = \frac{dL}{dt} = \frac{dI}{dt} = \frac{dR}{dt} &= 0 \\ (S, L, I, R) &= (W, X, Y, Z) \end{aligned}$$

Then the system of equations becomes,

From equations (3.1) to (3.4)

$$\beta N - \frac{\alpha WY}{N} + eZ - \mu W = 0 \text{ ----- (3.8)}$$

$$\frac{\alpha WY}{N} - K_1 X = 0 \text{ ----- (3.9)}$$

$$\delta X - K_2 Y = 0 \text{ ----- (3.10)}$$

$$\gamma Y + \tau x - K_3 Z = 0 \text{ ----- (3.11)}$$

FROM equation (3.10)),we now solve this system of equations simultaneously to obtain:

$$\begin{aligned} \delta x - k_2 Y &= 0 \\ \delta &= k_2 Y \text{ ----- (3.12)} \end{aligned}$$

$$X = \frac{k_2 Y}{\delta} \text{ ----- (3.13)}$$

Substitute equation (3.13) in to equation (3.9)

$$\frac{\alpha WY}{N} - \frac{K_1 K_2 Y}{\delta} = 0 \text{ ----- (3.14)}$$

$$Y \left(\frac{\alpha W}{N} - \frac{K_1 K_2}{\delta} \right) = 0$$

Either $Y = 0$ or $\frac{\alpha W}{N} - \frac{K_1 K_2}{\delta} = 0$

Substitute $X = 0$ into equation (3.13)

$$Y = 0$$

Substitute $Y = X = 0$ in to equation (3.11)

$$W = 0$$

subtitute $Y = X = W = 0$ into equation (3.8)

$$\beta N - \mu W = 0 \text{ ----- (3.15)}$$

$$\rightarrow W = \frac{\beta N}{\mu} \text{ ----- (3.16)}$$

$$E = \begin{pmatrix} W \\ X \\ Y \\ Z \end{pmatrix} = \begin{pmatrix} \frac{\beta N}{\mu} \\ 0 \\ 0 \\ 0 \end{pmatrix} \text{ ----- (3.17)}$$

As the disease free equilibrium state.

BASIC REPRODUCTION NUMBER

According to Diekmann *et al.*, [10] and Murray [17], the basic reproduction number is denoted by R_0 is the expected number of secondary cases produced, in a completely susceptible population by a typical infected individual. it is one of the most useful threshold parameters, which characterized mathematical

problems concerning infectious diseases if $R_0 < 1$, this implies that, on average an infected individual produces less than one new infected individual during the infectious period and the infectious can be wiped out conversely, if $R > 1$, then each infected individual produced can be wiped out new infection, and the disease is spread in the population.

$$F_i = \left[\frac{df_i}{dx_j} \right] \text{ and } v_i = \left[\frac{dvi}{dx_j} \right] \text{----- (3.18)}$$

$$F = \begin{bmatrix} \frac{df_1}{dL} & \frac{df_1}{dl} \\ \frac{df_2}{dL} & \frac{df_2}{dl} \end{bmatrix} \text{ V} = \begin{bmatrix} \frac{dv_1}{dL} & \frac{dv_1}{dl} \\ \frac{dv_2}{dL} & \frac{dv_2}{dl} \end{bmatrix} \text{----- (3.19)}$$

Now we recall that the system of equations in this model at equilibrium state is

$$\frac{dX}{dt} = \frac{\alpha W Y}{N} - K_1 X$$

$$\begin{aligned} \frac{dY}{dt} &= \delta X - K_2 Y \\ \frac{dW}{dt} &= \beta N - \frac{\alpha W Y}{N} + eZ - \mu W \\ \frac{dZ}{dt} &= \gamma Y + \tau X - K_3 Z \end{aligned}$$

From (3.18) and (3.19)

$$F_i = \begin{bmatrix} \frac{\alpha W Y}{N} \\ 0 \end{bmatrix}$$

$$V_i = \begin{bmatrix} K_1 X \\ K_2 Y - \delta X \end{bmatrix}$$

$$F = \begin{bmatrix} 0 & \frac{\alpha W}{N} \\ 0 & 0 \end{bmatrix} \text{ \& V} = \begin{bmatrix} K_1 & 0 \\ \delta & K_2 \end{bmatrix} \text{----- (3.20)}$$

Taking the inverse of (3.20)

$$V^{-1} = \frac{1}{K_1 K_2} \begin{bmatrix} K_1 & 0 \\ \delta & K_2 \end{bmatrix} \text{----- (3.21)}$$

$$V^{-1} = \begin{bmatrix} \frac{k_1}{k_1 k_2} & 0 \\ \frac{\delta}{k_1 k_2} & \frac{k_2}{k_1 k_2} \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{k_1}{k_1 k_2} & 0 \\ \frac{\delta}{k_1 k_2} & \frac{k_2}{k_1 k_2} \end{bmatrix} = \begin{bmatrix} \frac{1}{k_2} & 0 \\ \frac{\delta}{k_1 k_2} & \frac{1}{k_1} \end{bmatrix}$$

$$FV^{-1} \begin{bmatrix} 0 & \frac{\alpha W}{N} \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{k_2} & 0 \\ \frac{\delta}{k_1 k_2} & \frac{1}{k_1} \end{bmatrix} = \begin{bmatrix} \frac{\alpha \delta W}{N k_1 k_2} & \frac{\alpha W}{N k_2} \\ 0 & 0 \end{bmatrix} \text{----- (3.22)}$$

$(FV^{-1}\lambda I) = 0$

$$\begin{bmatrix} \frac{\alpha \delta W}{N k_1 k_2} & \frac{\alpha W}{N k_2} \\ 0 & 0 \end{bmatrix} - \lambda \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} = 0 \text{----- (3.23)}$$

$$\begin{bmatrix} \frac{\alpha \delta W}{N k_1 k_2} & \frac{\alpha W}{N k_1} \\ 0 & 0 \end{bmatrix} - \begin{bmatrix} \lambda & 0 \\ 0 & \lambda \end{bmatrix} = 0 \text{----- (3.24)}$$

$$= \begin{bmatrix} \frac{\alpha \delta W}{N k_1 k_2} - \lambda & \frac{\alpha W}{N k_1} \\ 0 & -\lambda \end{bmatrix} = 0 \text{----- (3.25)}$$

$$(-\lambda)\left(\frac{\alpha\delta W}{Nk_1 k_2}\right) = 0 \text{-----} (3.26)$$

Either $\lambda_1 = 0$ or $\frac{\alpha\delta W}{Nk_1 k_2} - \lambda = 0$
 $\rightarrow \lambda_2 = \frac{\alpha\delta W}{Nk_1 k_2}$

$$R_0 = \lambda_2 = \frac{\alpha\delta W}{Nk_1 k_2} \text{-----} (3.27)$$

Where,

$$\begin{aligned} k_1 &= \delta + \tau + \mu \\ k_2 &= \gamma + \mu + \psi \\ R_0 &= \frac{\alpha\delta W}{N(\delta + \tau + \mu)(\gamma + \mu + \psi)} \text{-----} (3.28) \end{aligned}$$

LOCAL STABILITY OF DISEASE FREE EQUILIBRIUM

We now investigate the stability of the disease free equilibrium state. To do this we examine the

behavior of the model population near the equilibrium state. The characteristic equation, now recall that the system of equation in this model at equilibrium state is

$$\begin{aligned} \beta N - \frac{\alpha W Y}{N} + eZ - \mu W &= 0 \\ \frac{\alpha W Y}{N} - K_1 X &= 0 \\ \delta X - K_2 Y &= 0 \\ \gamma Y + \tau X - K_3 Z &= 0 \end{aligned}$$

The Jacobian matrix of this system of equations is given by:

$$J(E) = \begin{bmatrix} \frac{-\alpha Y}{N} - \mu & 0 & -\frac{\alpha W}{N} e \\ \frac{\alpha Y}{N} & -k_1 & \frac{\alpha W}{N} 0 \\ 0 & \delta & -k_2 \\ 0 & \tau\gamma - k_3 & -k_3 \end{bmatrix}$$

The characteristic equation is obtained from Jacobian determinant with the Eigen values λ $\det [J - \lambda I] = 0$ becomes

$$\begin{bmatrix} \frac{-\alpha Y}{N} - \mu & 0 & -\frac{\alpha W}{N} e \\ \frac{\alpha Y}{N} & -k_1 & \frac{\alpha W}{N} 0 \\ 0 & \delta & -k_2 \\ 0 & \tau\gamma - k_3 & -k_3 \end{bmatrix} - \lambda \begin{bmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} = 0$$

$$\begin{bmatrix} \frac{-\alpha Y}{N} - \mu & 0 & -\frac{\alpha W}{N} e \\ \frac{\alpha Y}{N} & -k_1 & \frac{\alpha W}{N} 0 \\ 0 & \delta & -k_2 \\ 0 & \tau\gamma - k_3 & -k_3 \end{bmatrix} - \begin{bmatrix} \lambda & 0 & 0 \\ 0 & \lambda & 0 \\ 0 & 0 & \lambda \end{bmatrix} = 0$$

$$\begin{bmatrix} -\mu - \lambda & 0 & -\frac{\alpha W}{N} e \\ 0 & -k_1 - \lambda & \frac{\alpha W}{N} 0 \\ 0 & \delta & -k_2 - \lambda \\ 0 & \tau & \gamma - k_3 - k_3 - \lambda \end{bmatrix} = 0$$

$$-\mu - \lambda \begin{bmatrix} -k_1 - \lambda & \frac{\alpha W}{N} 0 \\ \delta & -k_2 - \lambda \\ \tau & \gamma - k_3 - k_3 - \lambda \end{bmatrix} + \left(\frac{\alpha W}{N}\right) \begin{bmatrix} 0 & -k_1 - \lambda & 0 \\ 0 & \delta & 0 \\ 0 & \tau & -k_3 - \lambda \end{bmatrix} - e \begin{bmatrix} 0 & -k_1 - \lambda & \frac{\alpha W}{N} \\ 0 & \delta & -k_2 - \lambda \\ 0 & \tau & \gamma - k_3 \end{bmatrix} = 0$$

$$\begin{aligned}
 &(-\mu - \lambda)(-k_1 - \lambda) \begin{bmatrix} -k_2 - \lambda & 0 \\ \gamma - k_3 & -k_3 - \lambda \end{bmatrix} + (-\mu - \lambda) \left(\frac{-\alpha W}{N} \right) \begin{bmatrix} \delta & 0 \\ \tau & k_3 - \lambda \end{bmatrix} \\
 &+ (k_1 + \lambda) \left(\frac{-\alpha W}{N} \right) \begin{bmatrix} 0 & 0 \\ 0 & -k_3 - \lambda \end{bmatrix} - (k_1 + \lambda) e \begin{bmatrix} 0 & -k_2 - \lambda \\ 0 & \gamma - k_3 \end{bmatrix} - e \left(\frac{-\alpha W}{N} \right) \begin{bmatrix} 0 & \delta \\ 0 & \tau \end{bmatrix} = 0 \\
 &= (-\mu - \lambda)(-k_1 - \lambda)(-k_2 - \lambda)(-k_3 - \lambda) + (-\mu - \lambda) \left(\frac{-\alpha W}{N} \right) (\delta - k_3 - \lambda) = 0 \\
 &(-\mu - \lambda)(-k_3 - \lambda) \left[k_1 k_2 + k_1 \lambda + k_2 \lambda + \lambda^2 - \frac{\alpha \delta W}{N} \right] = 0 \\
 &\text{Either } -\mu - \lambda = 0 \\
 &-k_3 - \lambda = 0
 \end{aligned}$$

OR

$$\begin{aligned}
 &\lambda^2 + (k_1 + k_2)\lambda + k_1 k_2 - \frac{\alpha \delta W}{N} = 0 \\
 &\rightarrow \lambda_1 = -\mu \\
 &\lambda_2 = -k_3
 \end{aligned}$$

This is quadratic equation

$$\lambda^2 + (k_1 + k_2)\lambda + k_1 k_2 - \frac{\alpha \delta W}{N} = 0$$

Using the general quadratic equation

Where:

$$\begin{aligned}
 &a = 1 \\
 &b = k_1 + k_2 \\
 &c = k_1 k_2 - \frac{\alpha \delta W}{N}
 \end{aligned}$$

Substitute the values into the general quadratic equation.

$$\lambda = \frac{-b \pm \sqrt{b^2 - 4ac}}{2a}$$

$$\begin{aligned}
 \lambda &= \frac{-(k_1 + k_2) \pm \sqrt{(k_1 + k_2)^2 - 4(k_1 k_2 - \frac{\alpha \delta W}{N})}}{2} \\
 &= \frac{-(k_1 + k_2) \pm \sqrt{(k_1 + k_2)^2 - 4k_1 k_2 \left(1 - \frac{\alpha \delta W}{N k_1 k_2}\right)}}{2}
 \end{aligned}$$

Where $\frac{\alpha \delta W}{N k_1 k_2} = R_0$

$$\begin{aligned}
 &\lambda_3 = \frac{-(k_1 + k_2) \pm \sqrt{(k_1 + k_2)^2 - 4k_1 k_2 (1 - R_0)}}{2} \\
 &\lambda_4 = \frac{-(k_1 + k_2) \pm \sqrt{(k_1 + k_2)^2 - 4k_1 k_2 (1 - R_0)}}{2} < 0 \\
 &\text{if } \sqrt{(k_1 + k_2)^2 - 4k_1 k_2 (1 - R_0)} < (k_1 + k_2)
 \end{aligned}$$

Taking the square of both sides

$$\begin{aligned}
 &(k_1 + k_2)^2 - 4k_1 k_2 (1 - R_0) < (k_1 + k_2)^2 \\
 &-4k_1 k_2 (1 - R_0) < 0 \\
 &1 - R_0 < 0 \\
 &1 > R_0 \\
 &R_0 < 1
 \end{aligned}$$

The Jacobean stability technique requires that all Eigen values be negative for stability to hold. The above shows $\lambda_1, \lambda_2, \lambda_3$ are negative while λ_4 is negative

if $R_0 < 1$. Therefore the disease free equilibrium (DFE) is locally asymptotically stable if $R_0 < 1$.

ENDEMIC EQUILIBRIUM STATE

We now state where the disease cannot be totally eradicated but remain in the population. For the

disease to persist in the population, the susceptible class, the latently infected, the infectious class and the recovered class must not be zero at equilibrium state.

From equation (3.14)

$$\frac{\alpha W}{N} - \frac{k_1 k_2}{\delta} = 0$$

Multiply $\frac{N}{\alpha}$ to both sides

$$\frac{N}{\alpha} \cdot \frac{\alpha W}{N} - \frac{k_1 k_2}{\delta} \cdot \frac{N}{\alpha} = 0$$

$$W - \frac{N k_1 k_2}{\alpha \delta} = 0$$

$$W = \frac{N k_1 k_2}{\alpha \delta} \text{----- (3.29)}$$

From equations (3.10)

$$\delta X - K_2 Y = 0$$

dividing both sides by δ

$$\frac{\delta X}{\delta} = \frac{K_2 Y}{\delta}$$

$$X = \frac{K_2 Y}{\delta} \text{----- (3.30)}$$

Substitute $X = \frac{K_2 Y}{\delta}$ into (3.11)

$$\gamma Y + \frac{\tau K_2 Y}{\delta} - K_3 Z = 0$$

$$Z = \frac{1}{K_3} (\gamma Y + \frac{\tau K_2 Y}{\delta}) \text{----- (3.31)}$$

Substitute equation (3.31) in to (3.8)

$$BN - \frac{\alpha W Y}{N} + \frac{e}{k_3} (\gamma Y + \frac{\tau K_2 Y}{\delta}) - \mu W = 0$$

$$\frac{-\alpha W Y}{N} + \frac{e}{k_3} (\gamma Y + \frac{\tau K_2 Y}{\delta}) = -\beta N + \mu W \text{----- (3.32)}$$

$$Y \left(\frac{e}{k_3} + \left(\gamma + \frac{\tau K_2}{\delta} \right) - \frac{\alpha W}{N} \right) = \mu W + \beta N$$

$$Y \left(\frac{\gamma e + \tau K_2 - \alpha W}{k_3 \delta N} \right) = \mu W + \beta N \text{----- (3.33)}$$

Dividing through by the co-efficient of Y

$$Y = \frac{\mu W + \beta N}{\frac{\gamma e + \tau K_2 - \alpha W}{k_3 \delta N}} \text{----- (3.34)}$$

$$Y = \frac{K_3 \delta N (\mu W + \beta N)}{\gamma e + \tau K_2 - \alpha W} \text{----- (3.35)}$$

Now the values of W, X, Y, Z, became this.

$$\begin{pmatrix} W \\ X \\ Y \\ Z \end{pmatrix} = \begin{bmatrix} \frac{N K_1 K_2}{\alpha \delta} \\ \frac{K_2 Y}{\delta} \\ \frac{K_3 \delta N (\mu W + \beta N)}{\gamma e + \tau K_2 - \alpha W} \\ \frac{1}{k_3} (\gamma Y + \frac{\tau K_2 Y}{\delta}) \end{bmatrix} \text{----- (3.36)}$$

As the endemic equilibrium state.

RESULT AND DISCUSSION

VARIABLES AND PARAMETER VALUES

The numerical simulation of the model equation (3.1) to (3.4) using defined parameters are

presented in the table 1.0 and table 1.1 shows the values of some parameters collected from general hospital potiskum, yobe state on five July, 2019. We will vary the key parameters to investigate the impact of

vaccination on the transmission dynamics of tuberculosis using pictorial representation (Graphs obtained from the tables).

Table-1.0: To Shows Values of Parameters Are Presented

Parameters	Values	Source
βN : At constant rate (Through birth or immigration).	0.045	Enagi A. I. (2013) [5]
α : Tuberculosis contraction rate.	0.02	Enagi A. I. (2013) [5]
δ : Rate of breakdown of latent TB into infectious TB.	0.01	Enagi A. I. (2013) [5]
μ : Natural death rate.	0.014	Enagi A. I. (2013) [5]
γ : Successful cure of infection TB patients.	0.23	Enagi A. I. (2013) [5]
e : Is rate which recovered individuals return to the susceptible statue due to loss immunity	0.021	Enagi A. I. (2013) [5]
τ : Successful cure infectious latent.	0.03	
Ψ : Death caused as a result of chronic TB infection at the rate.	0.002	Enagi A. I. (2013) [5]

Table-1.1: To Shows the Susceptible, Latently Infected, Infectious, Recovered People and Time Period

Time/period	0	1	2	3	4	5	6	7	8	9
Susceptible	0	100	200	300	400	500	600	700	800	900
Latently infected	150	140	130	120	110	100	90	80	70	60
Infectious	100	80	63	53	43	33	23	15	7	3
Recovered	0	125	180	200	210	220	225	225	215	200

- T = time period.
- S = susceptible people.
- L = latently infected persons.
- I = infectious persons.
- R = recovered people.

Table-1.2: Shows the Table of Susceptible Against Time

T	0	1	2	3	4	5	6	7	8	9
S	0	100	200	300	400	500	600	700	800	900

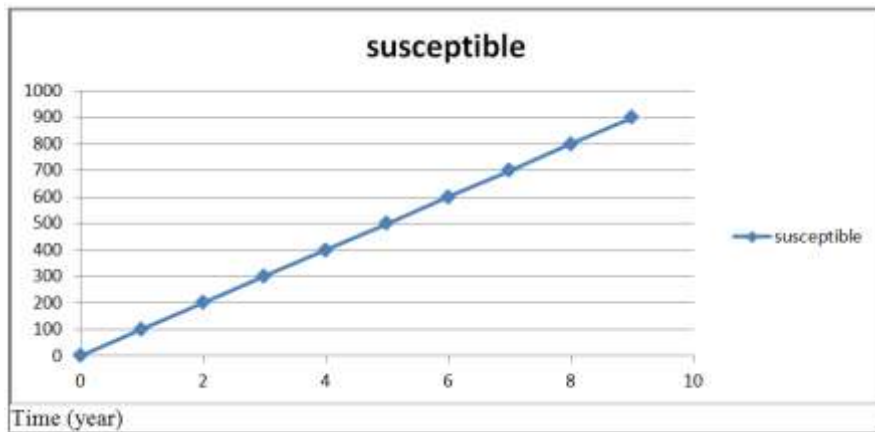


Fig-1.0: Shows the Graph of Susceptible Against Time

However, the table 1.2 and the above figure 1.0 the susceptible population against time with infection rate, shows that the susceptible population increases with time due to the fact that treatment does not confer permanent recovery and recovered individuals may likely be re-infected. Also shows the effect of varying infection rate on the population of susceptible. The figure shows the little change in the number of susceptible over time.

From the model 3.1, we now solve for the susceptible individuals for the period of ten years to get another results.

From equation (3.1)

$$\frac{dS}{dt} = \beta N - \frac{\alpha SI}{N} + eR - \mu S$$

By Separation of Variables

$$dS = \left(\beta N - \frac{\alpha SI}{N} + eR - \mu S \right) dt$$

Now we integrate both sides

$$\int dS = \int \left(\beta N - \frac{\alpha SI}{N} + eR - \mu S \right) dt$$

$$S(t) = \left(\beta N - \frac{\alpha SI}{N} + eR - \mu S \right) \dots\dots\dots (4.1)$$

When t = 0

$$S(t) = \left(\beta N - \frac{\alpha SI}{N} + eR - \mu S \right) t$$

$$S(0) = \left(0.045 - \frac{0.02 \times 0 \times 100}{900} + 0.021 \times 0 - 0.014 \times 0 \right) 0$$

$$= (0.045 - 0 + 0 - 0) 0$$

$$= 0$$

When t=1

$$S(1) = \left(0.045 - \frac{0.02 \times 100 \times 80}{900} + 0.021 \times 125 - 0.014 \times 100 \right) 1$$

$$= 0.045 - \frac{160}{900} + 2.625 - 1.4$$

$$= 0.045 - 0.177 + 2.625 - 1.4$$

$$= -0.132 + 1.225$$

$$= 1.093$$

When t = 2

$$S(2) = \left(0.045 - \frac{0.02 \times 200 \times 63}{900} + 0.021 \times 180 - 0.014 \times 200 \right) 2$$

$$= \left(0.045 - \frac{252}{900} + 3.78 - 2.8 \right) 2$$

$$= (-0.238 + 0.98) 2$$

$$= 0.742 \times 2$$

$$= 1.484$$

When t = 3

$$S(3) = \left(0.045 - \frac{0.02 \times 300 \times 53}{900} + 0.021 \times 200 - 0.014 \times 300 \right) 3$$

$$= (0.045 - 0.35 + 4.2 - 4.2) 3$$

$$= (-0.305 + 0) 3$$

$$= -0.305 \times 3$$

$$= -0.915$$

When t = 4

$$S(4) = \left(0.045 - \frac{0.02 \times 400 \times 43}{900} + 0.021 \times 210 - 0.014 \times 400 \right) 4$$

$$= (0.045 - 0.38 + 4.41 - 5.6) 4$$

$$= (-0.335 - 1.19) 4$$

$$= -1.525 \times 4$$

$$= -6.1$$

when t = 5

$$S(5) = \left(0.045 - \frac{0.02 \times 500 \times 33}{900} + 0.021 \times 220 - 0.014 \times 500 \right) 5$$

$$= (0.045 - 0.36 + 4.62 - 7) 5$$

$$= (-0.315 - 2.38) 5$$

$$= -2.695 \times 5$$

$$= -13.475$$

when t = 6

$$S(6) = \left(0.045 - \frac{0.02 \times 600 \times 23}{900} + 0.021 \times 225 - 0.014 \times 600 \right) 6$$

$$= (0.045 - 0.31 + 4.725 - 8.4) 6$$

$$= (-0.265 - 3.675) 6$$

$$= -3.94 \times 6$$

$$= -23.64$$

When $t=7$

$$\begin{aligned}
 S(7) &= \left(0.045 - \frac{0.02 \times 700 \times 15}{900} + 0.021 \times 225 - 0.014 \times 700\right) 7 \\
 &= (0.045 - 0.23 + 4.725 - 9.8)7 \\
 &= (-0.185 - 5.075)7 \\
 &= -5.26 \times 7 \\
 &= -36.82
 \end{aligned}$$

when $t=8$

$$\begin{aligned}
 S(8) &= \left(0.045 - \frac{0.02 \times 800 \times 7}{900} + 0.021 \times 215 - 0.014 \times 800\right) 8 \\
 &= (0.045 - 0.12 + 4.515 - 11.2)8 \\
 &= (-0.075 - 6.685)8 \\
 &= -6.76 \times 8 \\
 &= -54.08
 \end{aligned}$$

when $t=9$

$$\begin{aligned}
 S(9) &= \left(0.045 - \frac{0.02 \times 900 \times 3}{900} + 0.021 \times 200 - 0.014 \times 900\right) 9 \\
 &= (0.045 - 0.06 + 4.2 - 12.6)9 \\
 &= (-0.015 - 8.4)9 \\
 &= -8.42 \times 9 \\
 &= -75.78
 \end{aligned}$$

\approx - 76 people susceptible in the nine days.

Based on the values we got from the above solution, we can see that at the initial years of treatment that is from (0, 1, 2) is positive values to shows that the treatment of the susceptible individuals is not stable,

and also after two years that is (3, 4,...) is negative values is also to shows that the susceptible individuals are responding to treatment at a stable state. (Although there are chances of reinfection).

Table-1.3: Shows the Table of Latently Infected Against Time

T	0	1	2	3	4	5	6	7	8	9
L	150	140	130	120	110	100	90	80	70	60

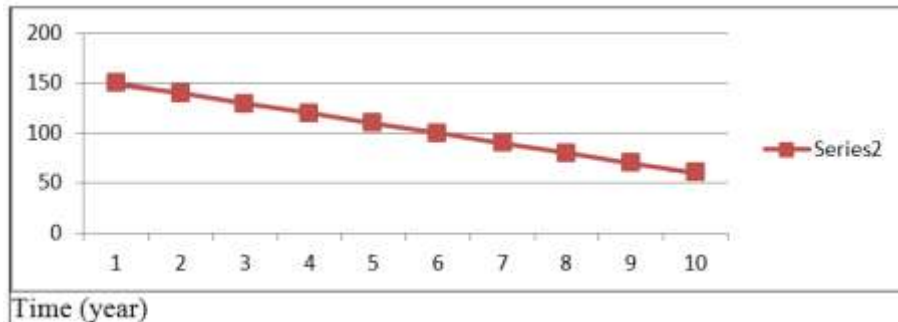


Fig-1.1: Shows the Graph of Latently Infected Against Time

From table 1.3, based on figure 1.1, The population of latently infected against time with infection rate to shows that the population of latently infected decreases with time due to progression to infected compartment. Then shows the effect of varying

infection rate on the population of the latently infected due to treatment.

From the model 3.2, we now solve for the latently infected individuals for the period of ten years to get the results.

From equation (3.2)

$$\frac{dL}{dt} = \frac{\alpha SI}{N} - (\delta + \tau + \mu)L$$

$$dL = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L\right) dt$$

We now integrate both sides

$$\int dL = \int \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right) dt$$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t \text{-----} (4.2)$$

When $t = 0$
 $= \left(\frac{0.02 \times 0 \times 100}{900} - (0.01 + 0.03 + 0.014)150 \right) 0$
 $= 0 - 0.054 \times 0$
 $= 0$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 1$
 $= \left(\frac{0.02 \times 100 \times 80}{900} - (0.01 + 0.03 + 0.014)140 \right) 1$
 $= 0.17 - 0.054 \times 140$
 $= 16.24$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 2$
 $= \left(\frac{0.02 \times 200 \times 63}{900} - (0.01 + 0.03 + 0.014)130 \right) 2$
 $= 0.28 - 0.054 \times 260$
 $= 58.76$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 3$
 $= \left(\frac{0.02 \times 300 \times 53}{900} - (0.01 + 0.03 + 0.014)120 \right) 3$
 $= 0.35 - 0.054 \times 360$
 $= 117.3$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 4$
 $= \left(\frac{0.02 \times 400 \times 43}{900} - (0.01 + 0.03 + 0.014)110 \right) 4$
 $= 0.38 - 0.054 \times 440$
 $= 143.4$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 5$
 $= \left(\frac{0.02 \times 500 \times 33}{900} - (0.01 + 0.03 + 0.014)100 \right) 5$
 $= 0.37 - 0.054 \times 500$
 $= 158$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 6$
 $= \left(\frac{0.02 \times 600 \times 23}{900} - (0.01 + 0.03 + 0.014)90 \right) 6$
 $= 0.31 - 0.054 \times 540$
 $= 138.24$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 7$
 $= \left(\frac{0.02 \times 700 \times 15}{900} - (0.01 + 0.03 + 0.014)80 \right) 7$
 $= 0.23 - 0.054 \times 560$
 $= 98.56$

$$L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right)t$$

When $t = 8$
 $= \left(\frac{0.02 \times 800 \times 7}{900} - (0.01 + 0.03 + 0.014)70 \right) 8$
 $= 0.12 - 0.054 \times 560$
 $= 36.96$
 $L(t) = \left(\frac{\alpha SI}{N} - (\delta + \tau + \mu)L \right) t$

When $t = 9$
 $= \left(\frac{0.02 \times 900 \times 13}{900} - (0.01 + 0.03 + 0.014)60 \right) 9$
 $= 0.06 - 0.054 \times 540$
 $= 3.24$
 $= 3$ people latently infected in the nine days.

The results we obtained from the latently infected has a unique character which increases from the years (0 - 5), but later starts to decrease with the time due to treatment

Table-1.4: Shows the Table of Infectious Against Time

T	0	1	2	3	4	5	6	7	8	9
I	100	80	63	53	43	33	23	15	7	3

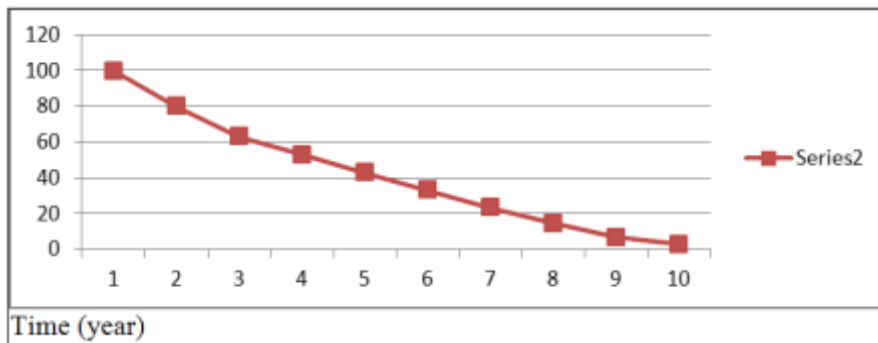


Fig-1.2: The Graph of Infectious Against Time

Hence from figure 1.2, graph infectious to undergoing treatment against time with infection rate, that shows the population of infected individual's decreases due to treatment and progression to recovered compartment. And also shows the effect of varying treatment rate on the population of infected, the figure show that infected population decreases toward zero

because of effective reproduction number being less than 1 and since infected individuals progress in the class of those that undergoing treatment

From the model 3.3, we now solve for the infectious individuals for the period of ten years to get the results.

From equation (3.3)

$$\frac{dI}{dt} = \delta L - (\gamma + \mu + \Psi)I$$

$$dI = (\delta L - (\gamma + \mu + \Psi)I)dt$$

$$\int dI = \int (\delta L - (\gamma + \mu + \Psi)I) dt$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t \text{ ----- (4.3)}$$

At $t = 0$

$$I(0) = (0.01 \times 150 - (0.23 + 0.014 + 0.002)100)0$$

$$= 0.01 \times 150 - (0.23 + 0.014 + 0.002) 0$$

$$= 1.5 - 0.246 \times 0$$

$$= 1.254 \times 0$$

$$= 0$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=1

$$\begin{aligned} I(1) &= (0.01 \times 140 - (0.23 + 0.014 + 0.002)80)1 \\ &= 0.01 \times 140 - (0.23 + 0.014 + 0.002)80 \\ &= 1.4 - 0.246 \times 80 \\ &= 1.154 \times 80 \\ &= 92.32 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=2

$$\begin{aligned} I(2) &= (0.01 \times 130 - (0.23 + 0.014 + 0.002)63)2 \\ &= 0.01 \times 130 - (0.23 + 0.014 + 0.002)63 \\ &= 1.3 - 0.246 \times 126 \\ &= 1.054 \times 126 \\ &= 132.80 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=3

$$\begin{aligned} I(3) &= (0.01 \times 120 - (0.23 + 0.014 + 0.002)53)3 \\ &= 0.01 \times 120 - (0.23 + 0.014 + 0.002)53 \\ &= 1.2 - 0.246 \times 189 \\ &= 0.954 \times 189 \\ &= 180.30 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=4

$$\begin{aligned} I(4) &= (0.01 \times 110 - (0.23 + 0.014 + 0.002)43)4 \\ &= 0.01 \times 110 - (0.23 + 0.014 + 0.002)43 \\ &= 1.1 - 0.246 \times 172 \\ &= 0.854 \times 172 \\ &= 146.88 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=5

$$\begin{aligned} I(5) &= (0.01 \times 100 - (0.23 + 0.014 + 0.002)33)5 \\ &= 0.01 \times 100 - (0.23 + 0.014 + 0.002)33 \\ &= 1 - 0.246 \times 165 \\ &= 0.754 \times 165 \\ &= 124.41 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=6

$$\begin{aligned} I(6) &= (0.01 \times 90 - (0.23 + 0.014 + 0.002)23)6 \\ &= 0.01 \times 90 - (0.23 + 0.014 + 0.002)23 \\ &= 0.9 - 0.246 \times 138 \\ &= 0.654 \times 138 \\ &= 90.25 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=7

$$\begin{aligned} I(7) &= (0.01 \times 80 - (0.23 + 0.014 + 0.002)15)7 \\ &= 0.01 \times 80 - (0.23 + 0.014 + 0.002)15 \\ &= 0.8 - 0.246 \times 105 \\ &= 0.554 \times 105 \\ &= 58.17 \end{aligned}$$

$$I(t) = (\delta L - (\gamma + \mu + \Psi)I)t$$

At t=8

$$I(8) = (0.01 \times 70 - (0.23 + 0.014 + 0.002)7)8$$

$$\begin{aligned}
 &= 0.01 \times 70 - (0.23 + 0.014 + 0.002)7)8 \\
 &= 0.7 - 0.246 \times 56 \\
 &= 0.454 \times 56 \\
 &= 25.42
 \end{aligned}$$

$$\begin{aligned}
 I(t) &= (\delta L - (\gamma + \mu + \Psi)I)t \\
 \text{At } t=9 \\
 I(9) &= (0.01 \times 60 - (0.23 + 0.014 + 0.002)3)9 \\
 &= 0.01 \times 60 - (0.23 + 0.014 + 0.002)3)9 \\
 &= 0.6 - 0.246 \times 27 \\
 &= 0.354 \times 27 \\
 &= 9.55 \\
 &\approx 10 \text{ people infected in the nine days.}
 \end{aligned}$$

From the above model we the values. We obtain to shows the effect of varying treatment rate on the population infected and their progression to recovered compartment.

Table-1.5: Shows the Table Of Recovered Against Time

T	0	1	2	3	4	5	6	7	8	9
R	0	125	180	200	210	220	225	225	215	200

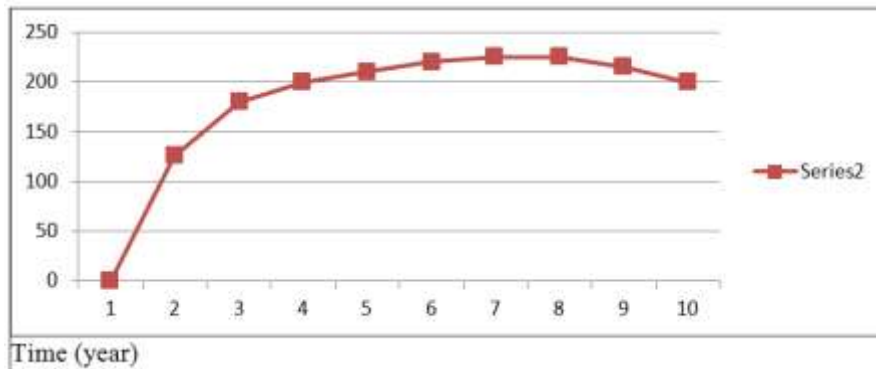


Fig-1.3: The Graph of Recovered Against Time

However, from table 1.5, based on Figure 1.3 graph of recovered individuals against time with infection rate, to shows an increase in the population of recovered individuals after a while and then a decrease due to progression to the susceptible compartment. And also shows the effect of varying treatment rate on the population of infected individuals. The population of infected individuals increase for a while and then

decrease after some time since the effective reproduction number is less than 1 and since individuals undergoing treatment progress later to susceptible class.

From the model 3.4, we now solve for the recovered individuals for the period of ten years to get the results.

From equation (3.4)

$$\begin{aligned}
 \frac{dR}{dt} &= \gamma I + \tau L - (e + \mu)R \\
 dR &= (\gamma I + \tau L - (e + \mu)R)dt
 \end{aligned}$$

Now we integrate both sides

$$\int dR = \int (\gamma I + \tau L - (e + \mu)R)dt$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t \text{ ----- (4.4)}$$

$$\begin{aligned}
 \text{When } t = 0 \\
 R(0) &= (0.23 \times 100 + 0.03 \times 150 - (0.021 + 0.014)0)0 \\
 &= (23 + 4.5 - 0.035)0 \\
 &= 0 \\
 R(t) &= (\gamma I + \tau L - (e + \mu)R)t \\
 \text{when } t = 1
 \end{aligned}$$

$$\begin{aligned}
 R(1) &= (0.23 \times 80 + 0.03 \times 140 - (0.021 + 0.014)125)1 \\
 &= (18.4+4.2 - 0.035)125 \\
 &= 22.565 \times 125 \\
 &= 2.820
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 2$

$$\begin{aligned}
 R(2) &= (0.23 \times 63 + 0.03 \times 130 - (0.021 + 0.014)180)2 \\
 &= (14.49+3.9 - 0.035)360 \\
 &= 18.355 \times 360 \\
 &= 6.607
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 3$

$$\begin{aligned}
 R(3) &= (0.23 \times 53 + 0.03 \times 120 - (0.021 + 0.014)200)3 \\
 &= (12.19+3.6 - 0.035)600 \\
 &= 15.755 \times 600 \\
 &= 9.453
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 4$

$$\begin{aligned}
 R(4) &= (0.23 \times 43 + 0.03 \times 110 - (0.021 + 0.014)210)4 \\
 &= (9.89+3.3 - 0.035)840 \\
 &= 13.155 \times 840 \\
 &= 11.050
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 5$

$$\begin{aligned}
 R(5) &= (0.23 \times 33 + 0.03 \times 100 - (0.021 + 0.014)220)5 \\
 &= (7.59+3 - 0.035)1100 \\
 &= 10.555 \times 1100 \\
 &= 11.610
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 6$

$$\begin{aligned}
 R(6) &= (0.23 \times 23 + 0.03 \times 90 - (0.021 + 0.014)225)6 \\
 &= (5.29+2.7 - 0.035)1350 \\
 &= 7.955 \times 1350 \\
 &= 10.739
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 7$

$$\begin{aligned}
 R(7) &= (0.23 \times 15 + 0.03 \times 80 - (0.021 + 0.014)225)7 \\
 &= (3.45+2.4 - 0.035)1575 \\
 &= 5.815 \times 1575 \\
 &= 9.158
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 8$

$$\begin{aligned}
 R(8) &= (0.23 \times 7 + 0.03 \times 70 - (0.021 + 0.014)215)8 \\
 &= (1.61+2.1 - 0.035)1720 \\
 &= 3.675 \times 1720 \\
 &= 6.321
 \end{aligned}$$

$$R(t) = (\gamma I + \tau L - (e + \mu)R)t$$

When $t = 9$

$$\begin{aligned} R(9) &= (0.23 \times 3 + 0.03 \times 60 - (0.021 + 0.014)200)9 \\ &= (0.69 + 1.8 - 0.035)1800 \\ &= 2.455 \times 1800 \\ &= 4.419 \\ &= 4 \text{ people recovered in the nine days.} \end{aligned}$$

According to the values obtained in this model we can see that the population of infected individuals increase for a while and then later decrease after some time by undergoing treatment.

SUMMARY

In this study, we modeled the effect of varying infection rate and treatment on the transmission dynamics of tuberculosis (TB). The disease free equilibrium state and the endemic equilibrium state of the model were obtained, the basic reproduction number, R_0 was derived and the analysis showed that TB can effectively be controlled or even be eradicated if effort is made to ensure that latently infected individuals are detected and treated and infected individuals appropriately treated.

CONCLUSION

The existence of the disease free equilibrium state implies that there is possibility of complete total eradication of tuberculosis from Nigeria. The negativity of all the eigenvalues arising from the stability analysis carried out in chapter three shows that there will be no return to the tuberculosis endemic state after eradication of tuberculosis from Nigeria. The existence of the endemic equilibrium state in chapter three signifies the possibility of Nigeria remaining a tuberculosis endemic nation.

RECOMMENDATION

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 if $R_0 < 1$, 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.

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