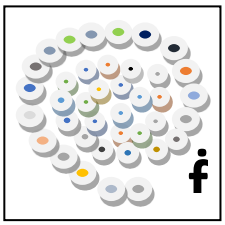

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A deterministic Tuberculosis epidemic infection model incorporating public interventions

F. Sulayman*

Abstract

Tuberculosis (TB) is one of the devastating human ailments. To better understand the dynamics of TB disease, a deterministic compartmental model is designed to describe the dynamics of TB by incorporating public interventions such as vaccination, educational counselling, and treatment. The dynamics of the model are thoroughly investigated by utilizing the theory of the dynamical framework. The effective reproduction number R0 is derived using strict mathematical derivations. The theoretical outcome indicates that the disease-free equilibrium (DFE) is locally asymptotically stable if $R_C < 1$ and the system is uniformly persistent when $R_C > 1$. The qualitative analysis performed on the free model shows the existence of the occurrence of transcritical bifurcation at the threshold quantity of $R_0 = 1$. The numerical simulations analysis of the model demonstrated that an extreme emphasis on tuberculosis public interventions appeared to essentially bring about a decrease in TB epidemic in the community.

Keywords: Tuberculosis; stability; effective reproduction number; transcritical bifurcation; public interventions.

1 Introduction

Tuberculosis (TB) is a communicable disease that affects the lungs. Some studies have suggested that up to a third of the world human population has contracted the disease [9, 66]. Tuberculosis is caused by a family of bacteria called Mycobacterium Tuberculosis [66]. This airborne infectious disease is a public health challenge worldwide: in USA[28] and European nations [1, 8]. TB epidemic occurs globally and remains a major cause of morbidity and mortality [43, 66]. TB is transmitted through droplet infection and droplet nuclei [37]. TB ranks above HIV/AIDS in 2018, with 1 million cases and 1.5 million deaths worldwide [66]. TB disease remains a serious pandemic around the world [41].

TB is currently treated for 6-9 months with a blend anti-microbial treatment for 6–9 months, that causes severe side effects [66, 47]. The TB control procedure of the World Health Organization is at present pointed toward fortifying disease prevention [62], and active work is in progress far and wide to create improved TB vaccines, some of which have entered the clinical research stage Nadolinskaia *et al.* [47]. The Bacille Calmette-Guérin (BCG) vaccine is one of the world's most secure and most affordable vaccines, yet many clinical trials have assessed its adequacy in curtailing the epidemic anywhere from 0 to 84% [57, 26, 12]. Since 1921, BCG has been used for vaccination which has contributed significantly to the prevention of TB Nadolinskaia *et al.* [47]. Valle *et al.* [18] has stated that the predominance of any epidemic was strongly subject to individual's behaviour in a population. It was reported that behavioural change plays a very important role in the spread of ailments. The health education on TB epidemics assumes a significant function in TB management and prevention , and can improve the health information and familiarity with population about TB infection Xiang *et al.* [67].

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*Department of Mathematical Sciences, Ibrahim Badamasi Babangida University, Lapai, Niger State, Nigeria; E-mail: s.fatima@ibbu.edu.ng

present pointed toward fortifying disease prevention [62], and active work is in progress far and wide to create improved TB vaccines, some of which have entered the clinical research stage Nadolinskaia *et al.* [47]. The Bacille Calmette-Guérin (BCG) vaccine is one of the world's most secure and most affordable vaccines, yet many clinical trials have assessed its adequacy in curtailing the epidemic anywhere from 0 to 84% [57, 26, 12]. Since 1921, BCG has been used for vaccination which has contributed significantly to the prevention of TB Nadolinskaia *et al.* [47]. Valle *et al.* [18] has stated that the predominance of any epidemic was strongly subject to individual's behaviour in a population. It was reported that behavioural change plays a very important role in the spread of ailments. The health education on TB epidemics assumes a significant function in TB management and prevention , and can improve the health information and familiarity with population about TB infection Xiang *et al.* [67].

Deterministic models comprising ordinary differential equations (ODEs) were used to explain and monitor TB transmission. Several scholars have used these models to help them understand the processes involved in the dynamics of TB infection. Feng et al. [22] proposed a deterministic model for TB with exogenous re-infection and submitted that an infectious individual can infect another individual with each contact per unit of time. Mlay et al. [42] studied tuberculosis disease transmission with vaccination and treatment as control variables. Huo and Zou [29] formulated a tuberculosis model with two kinds of treatment; that is, treatment at home and treatment in the hospital. Agusto [5] presented a compartmental model with isolation and lost to follow-up for the transmission dynamics of three strains of mycobacterium tuberculosis that include drug sensitive, mult drug resistant (MDR) and extensively drug resistant (XDR) TB strains. Okuonghe and Ikhimwin [52] developed a mathematical model that refined [53] by classifying the latently infected population by their level of awareness and further expanded the number of key factors that positively affect the detection rate of tuberculosis. [50] used a deterministic mathematical model for the transmission dynamics of TB, with vaccination and screening the population, for the purpose of identifying those in need of immediate treatment. Xiang *et al.* [67] examined a TB model with health education and treatment during the latent period of TB. Nagar et al. [48] carried out a research on the dynamics of tuberculosis model with infected population and vaccination. Soyoung et al. [36] proposed and analyzed a compartmental nonlinear deterministic mathematical model for tuberculosis infection, based on Philippine data, by incorporating intervention strategies for mitigating the spread of infection. Aldila et al [2] developed a new model of the dynamics of tuberculosis, with the intervention of vaccination, in an age-structured susceptible compartment. They divided the population in to ten (10) compartments and classified the population according to children and adults. Rahmawati et al. [56] investigated the effectiveness of chemotherapy paradoxical reaction in TB infection. Sharomi et al. [60] formulated a deterministic mathematical model of exogeneous re-infection does not always cause backward bifurcation. Ali et al [3], in 2019, investigated a nonlinear deterministic compartmental model of HIV/AIDS-TB co-infection. They incorporated the awareness of median coverage on the control and prevention of HIV/AIDS and TB within a population of varying size. Omame et al [55] developed and analyzed a deterministic compartmental transmission dynamics model of two-sex, co-infection model for human papillomavirus (HPV) and tuberculosis (TB) in a community. Other existing studies have investigated the influences of vaccination, media awareness, and optimal strategy, on the prevalence of tuberculosis (see [15, 35]).

Inspired by the above research findings, our model improves on previous studies, and contributes to existing literature, as it incorporates compartments for vaccination and educational counselling that the previous studies did not consider. Our aim is to better understand the interplay between these public interventions and TB epidemic.

This article is presented as follows: a mathematical model of TB is considered in Section 2. In Section 3, we focus on the existence of steady state and the stability of both disease free (DFE) and endemic equilibrium points (EEP). The results and discussions of the numerical simulation analysis are reported in Section 4. Finally, the conclusion of the study are given in Section 5.

2 Methods

This section discusses the formulation of TB model governing a nonlinear ODE system to ascertain $S_\iota, V_\iota, P_\iota, E_\iota, I_\iota, R_\iota$ type system, which is the modification of existing literature. A qualitative analysis of the basic properties of the nonlinear ODE version of the model, and an estimation of the model parameters are performed. In addition, numerical simulations are also considered.

2.1 Model formulation

The total population represented by N_ι, is subdivided into six (6) compartments, i.e Susceptible (S_ι), Educated Susceptible (P_ι), Vaccinated (V_ι), Exposed (those people who are infected but the symptom of the disease are not yet visible) (E_ι), Infectious (I_ι), and Recovered (R_ι).
In this model, it is assumed that the number of susceptible population is generated via recruitment and new birth that are not yet vaccinated against tuberculosis infection, at a rate $(1-\rho)\Lambda$ and the arrival of vaccinated individuals that have lost the vaccine efficacy at a rate θ. The susceptible individuals acquired infection following effective contact with infectious individuals at a rate

$$\frac{\beta S_\iota I_\iota}{N_\iota}$$

where β, is the effective contact rate by I_ι, and N_ι is the total population. It is assumed that the exposed individuals do not transmit infection; that is, only infected individuals with symptoms of the disease are capable of transmitting the disease to susceptible individuals [59]. The population of susceptible individuals also decreases as a result of natural death rate and effective communication μ and ψ. Therefore, the rate of change of the susceptible population is given by

$$\frac{dS_\iota}{dt} = (1-\rho)\Lambda - \frac{\beta S_\iota I_\iota}{N_\iota} + \theta V_\iota - (\Psi+\mu)S_\iota$$

Vaccination is given at birth as a preventive measure in children but the efficacy of these vaccines wanes with time. Vaccination individuals are generated from new birth and immigration of successfully vaccinated against infection of tuberculosis disease at a rate $\rho\Lambda$. The potency of vaccines wane when the efficacy period elapses at the rate θ, and it diminishes as a result of natural death at a rate, μ. . Hence, we have

$$\frac{dV_\iota}{dt} = \rho\Lambda - (\theta+\mu)V_\iota.$$

The population of susceptible educated class grows as a result of effective communication at a rate Ψ and decreases due to infection at a reduced rate of

$$\frac{\eta\beta P_\iota I_\iota}{N_\iota}.$$

It enters into exposed class and also diminishes as a result of natural death at a rate and thus, we have

$$\frac{dP_\iota}{dt} = \Psi S_\iota - \frac{\eta\beta P_\iota I_\iota}{N_\iota} - \mu P_\iota.$$

The population of exposed class grows by force of infection

$$\frac{\beta S_\iota I_\iota}{N_\iota} + \frac{\eta\beta P_\iota I_\iota}{N_\iota} + \frac{\sigma\beta R_\iota I_\iota}{N_\iota}$$

and decreases as a result of death rate, due to natural cause, and progression at the rate κ and μ respectively so that

$$\frac{dE_\iota}{dt} = \frac{\beta S_\iota I_\iota}{N_\iota} + \frac{\eta\beta P_\iota I_\iota}{N_\iota} + \frac{\sigma\beta R_\iota I_\iota}{N_\iota} - (\kappa+\mu)E_\iota.$$

The population of infectious individuals grows at a rate κ, and decreases as a result of treatment, death due to natural means, and disease induced death rates, at the rates τ, μ, δ respectively, this gives

$$\frac{dI_\iota}{dt} = \kappa E_\iota - (\mu + \tau + \delta) I_\iota$$

And finally, the population of recovered individuals grows as a result of treatment of infectious individuals at a rate τ and decreases at the loss of temporary immunity at a rate $\frac{\sigma\beta R_\iota I_\iota}{N_\iota}$, so that we have

$$\frac{dR_\iota}{dt} = \tau I_\iota - \frac{\sigma\beta R_\iota I_\iota}{N_\iota} - \mu R_\iota.$$

The following assumptions are considered in developing the model:

- The number of birth rate is equal to death rate.
- A proportion of the total newborns are vaccinated against tuberculosis infection.
- The educational counseling (P_ι) to infected (I_ι) causes the spread of tuberculosis to be very low, as a result of effective communication on the causes and spread of tuberculosis infection.
- Death rate, due to natural cause at a rate μ, took place in all compartments.
- There is temporary immunity of recovered people; therefore, the recovered class go back to exposed class immediately.
- There is temporary conferred on vaccinated individuals which wanes after some time at a certain rate θ.

Therefore, based on the above description and assumptions, the model of tuberculosis with public interventions lead to the schematic diagram Fig. 1 below, and the parameters indicated in the diagram are explained in Table 1.

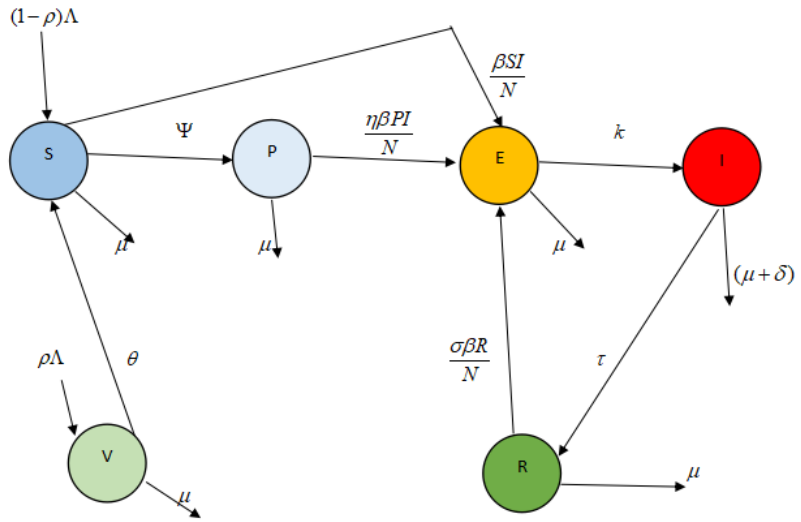

Figure 1: Flow chart of the TB model, Eq. (1).

Table 1: Description of the variable and parameters for the TB model, Eq. (1)

State Variables	Explanation
S_ι	Susceptible individuals
V_ι	Vaccinated individuals
P_ι	Educated Susceptible individuals
E_ι	Exposed individuals
I_ι	Infected individuals
R_ι	Recovered individuals
N_ι	Human population size
Parameters	
Λ	Inflow of recruitment rate into susceptible class
μ	Per capital natural death rate of humans
β	Transmission rate
σ	Reinfection parameter
κ	Progression from E_ι to I_ι
τ	Treatment rate
δ	TB induced mortality rate
Ψ	Information dissemination
η	Reduction in infection by educated susceptible individuals
ρ	Effective immunization

2.2 Tuberculosis model with public interventions

The following SVPEIRE is described by the ordinary differential equation given by

$$\begin{aligned}
\frac{dS_\iota}{dt} &= (1-\rho)\Lambda - \frac{\beta I_\iota S_\iota}{N_\iota} + \theta V_\iota - (\Psi+\mu)S_\iota \\
\frac{dV_\iota}{dt} &= \rho\Lambda - (\theta+\mu)V_\iota \\
\frac{dP_\iota}{dt} &= \Psi S_\iota - \frac{\eta\beta I_\iota P_\iota}{N_\iota} - \mu P_\iota \\
\frac{dE_\iota}{dt} &= \frac{\beta S_\iota I_\iota}{N_\iota} + \frac{\eta\beta I_\iota P_\iota}{N_\iota} + \frac{\sigma\beta I_\iota R_\iota}{N_\iota} - (\kappa+\mu)\,E_\iota \\
\frac{dI_\iota}{dt} &= \kappa E_\iota - (\mu+\tau+\delta)\,I_\iota \\
\frac{dR_\iota}{dt} &= \tau I_\iota - \frac{\sigma\beta I_\iota R_\iota}{N_\iota} - \mu R_\iota
\end{aligned} \tag{1}$$

with the initial conditions given by Eq. (2)

$$N_\iota = S_\iota + V_\iota + P_\iota + E_\iota + I_\iota + R_\iota \tag{2}$$

satisfies the equation

$$\frac{dN_\iota}{dt} = \Lambda - \mu N_\iota - \delta I_\iota(t) \tag{3}$$

derived by the summation of model system (1)
where

$$\lambda = \frac{\beta I_\iota}{N_\iota} \tag{4}$$

where equation (4) is represented by force of infection so that the model system (1) can be re-write as

$$\begin{aligned}
\frac{dS_\iota}{dt} &= (1-\rho)\Lambda - \lambda S_\iota + \theta V - (\Psi+\mu)S_\iota \\
\frac{dV_\iota}{dt} &= \rho\Lambda - (\theta+\mu)V_\iota \\
\frac{dP_\iota}{dt} &= \Psi S - \eta\lambda P_\iota - \mu P_\iota \\
\frac{dE_\iota}{dt} &= \lambda S_\iota + \eta\lambda P_\iota + \sigma\lambda R_\iota - (\kappa+\mu)\, E_\iota \\
\frac{dI_\iota}{dt} &= \kappa E_\iota - (\mu+\delta+\gamma)\, I_\iota \\
\frac{dR_\iota}{dt} &= \gamma I_\iota - \sigma\lambda R_\iota - \mu R_\iota
\end{aligned} \tag{5}$$

2.3 Model analysis

In this subsection, the qualitative analysis of the TB model, Eq. (5), is carried out.

2.3.1 Positivity of solutions

For the tuberculosis infection model system (5) to be epidemiological realistic, it is necessary to prove that all the state variable are positive all the time.

Lemma 2.1: Let initial data be $\{(S_\iota, V_\iota, P_\iota, E_\iota, I_\iota, R_\iota) \geq 0\} \in \Phi$. Then, the solution set $\{S_\iota(t), V_\iota(t), P_\iota(t), E_\iota(t), I_\iota(t), R_\iota(t)\}$ of the model system (5) is non-negative for all $t > 0$.

Proof. As mention in Obasi and Mbah, [51] from the non-linear system of model system (5), we take the first equation

$$\frac{dS_\iota}{dt} = \Lambda - (\lambda + \Psi + \mu)$$

that is

$$\frac{dS_\iota}{dt} \geq -(\lambda+\Psi+\mu)S. \tag{6}$$

$$\Rightarrow \frac{dS_\iota}{S_\iota} \geq (\lambda+\Psi+\mu)dt. \tag{7}$$

integrating (7) by separation of variables gives

$$\int \frac{dS_\iota}{S_\iota} \geq \int (\lambda+\Psi+\mu)dt. \tag{8}$$

therefore,

$$S_\iota(t) \geq S_{(0)} e^{-(\lambda+\Psi+\mu)t} \geq 0.$$

Similarly, it can also been shown that $E_\iota(t) > 0$, $I_\iota(t) > 0$, $R_\iota(t) > 0$, for all t>0.

2.3.2 Invariant region

Lemman 2.2: The solution of the tuberculosis model system (1) are enclosed in the region Φ subset $\in R_+^6$, given by

$$\Phi = \left\{(S_\iota, V_\iota, P_\iota, E_\iota, I_\iota, R_\iota) \in R_+^6, N_\iota \leq \frac{\Lambda}{\mu}\right\}$$

for the initial conditions (2) in Φ.

Proof. The change in the total population is given by

$$\frac{dN_\iota}{dt} = \Lambda - \mu N_\iota - \delta I_\iota(t) \tag{9}$$

which gives

$$\frac{dN_\iota}{dt} \leq \Lambda - \mu N_\iota \tag{10}$$

The solution of equation (10) is given by

$$N_\iota(t) \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N_{\iota 0}\right) e^{-\mu t} \tag{11}$$

where $N_{\iota 0} = N_\iota(0)$
Using Birkhoff Rota's [10] we note that if $N_{\iota 0} < \frac{\Lambda}{\mu}$, $N_\iota \to \frac{\Lambda}{\mu}$ asymptotically as t $\to \infty$ in equation (11) the total population size $N_\iota \to \frac{\Lambda}{\mu}$ which means that $0 \leq N_\iota \leq \frac{\Lambda}{\mu}$. Therefore, all the feasible solution in the model converges in the region Φ [27].

3 Analysis of disease-free equilibrium (DFE), T_0, and basic reproduction number R_0

The disease-free equilibrium (DFE) state, T_0, is a steady state solution where there is no infection in the community. Disease class can be described as the infected human population. Taking the first three equation of (1) with $E_\iota = I_\iota = R_\iota = 0$ into consideration, we arrive at:

$$T_0 = (S_\iota, V_\iota, P_\iota, E_\iota, I_\iota, R_\iota) = \left(\frac{(\theta + (1-\rho)\mu)\Lambda}{(\theta+\mu)(\psi+\mu)}, \frac{\rho\Lambda}{(\theta+\mu)}, \frac{\psi(\theta+(1-\rho)\mu)\Lambda}{\mu(\theta+\mu)(\psi+\mu)}, 0, 0, 0\right) \tag{12}$$

3.1 Basic reproduction number (R_0)

Diekmann et al [17] and [11] define basic reproduction number, R_0 as the effective number of secondary infections caused by a primary infected individual during his entire period of infectiousness. To obtain the basic reproduction number, we used next generation matrix method by [63], where F is the matrix of the new infection terms and V the matrix of the transition terms. Observing that, the matrices F and V are established from the coefficients of E and I, in the fourth and the fifth equations of system (5), the model equations are re-written, starting with newly infective classes, as

$$\frac{dE_\iota}{dt} = \lambda S_\iota + \sigma\lambda R_\iota + \eta\lambda P_\iota - (\kappa+\mu)\, E_\iota, \tag{13}$$

$$\frac{dI_\iota}{dt} = \kappa E_\iota - (\mu + \delta + \gamma)\, I_\iota \tag{14}$$

F and V are evaluated respectively at the disease free equilibrium T_0 which are the Jacobian matrices of F and V to give

$$F = \begin{bmatrix} 0 & c_3 \\ 0 & 0 \end{bmatrix} \tag{15}$$

where

$$c_3 = \frac{\beta\mu(\theta+(1-\rho)\mu)}{(\psi+\mu)(\theta+\mu)} + \frac{\eta\psi\beta(\theta+(1-\rho)\mu)}{(\psi+\mu)(\theta+\mu)}$$

$$V = \begin{bmatrix} (\kappa+\mu) & 0 \\ -\kappa & (\mu+\tau+\delta) \end{bmatrix} \tag{16}$$

from V, we obtained V^{-1} which gives

$$V^{-1} = \begin{bmatrix} \frac{1}{(\kappa+\mu)} & 0 \\ \frac{\kappa}{(\kappa+\mu)(\mu+\tau+\delta)} & \frac{1}{(\mu+\tau+\delta)} \end{bmatrix} \tag{17}$$

. Basic reproduction number is the spectral radius of FV^{-1} [17, 63] and it must be considered when analyzing any epidemiological model [63]. Thus, the effective reproduction number for this epidemiological system is:

$$R_C = \frac{\kappa c_3}{(\kappa+\mu)(\mu+\delta+\gamma)} = \frac{\kappa\beta(\mu+\psi\eta)(\theta+(1-\rho)\mu)}{(\kappa+\mu)(\psi+\mu)(\theta+\mu)(\mu+\tau+\delta)} \tag{18}$$

3.2 Proving the local stability of disease free equilibrium point T_0.

Lemman 2.3: The disease free equilibrium state, T_0 of the model system (5) is stable when $R_C < 1$ and unstable if $R_C > 1$.

Proof. Consider the model system (5)

$$f_1 = \frac{dS_\iota}{dt}, f_2 = \frac{dV_\iota}{dt}, f_3 = \frac{dP_\iota}{dt}, f_4 = \frac{dE_\iota}{dt}, f_5 = \frac{dI_\iota}{dt}, f_6 = \frac{dR_\iota}{dt}, \tag{19}$$

then, the Jacobian of the system (5) will be given by

$$J(T_0) = \begin{pmatrix} \frac{\partial f_1}{\partial S_\iota} & \frac{\partial f_1}{\partial V_\iota} & \frac{\partial f_1}{\partial P_\iota} & \frac{\partial f_1}{\partial E_\iota} & \frac{\partial f_1}{\partial I_\iota} & \frac{\partial f_1}{\partial R_\iota} \\ \frac{\partial f_2}{\partial S_\iota} & \frac{\partial f_2}{\partial V_\iota} & \frac{\partial f_2}{\partial P_\iota} & \frac{\partial f_2}{\partial E_\iota} & \frac{\partial f_2}{\partial I_\iota} & \frac{\partial f_2}{\partial R_\iota} \\ \frac{\partial f_3}{\partial S_\iota} & \frac{\partial f_3}{\partial V_\iota} & \frac{\partial f_3}{\partial P_\iota} & \frac{\partial f_3}{\partial E_\iota} & \frac{\partial f_3}{\partial I_\iota} & \frac{\partial f_3}{\partial R_\iota} \\ \frac{\partial f_4}{\partial S_\iota} & \frac{\partial f_4}{\partial V_\iota} & \frac{\partial f_4}{\partial P_\iota} & \frac{\partial f_4}{\partial E_\iota} & \frac{\partial f_4}{\partial I_\iota} & \frac{\partial f_4}{\partial R_\iota} \\ \frac{\partial f_5}{\partial S_\iota} & \frac{\partial f_5}{\partial V_\iota} & \frac{\partial f_5}{\partial P_\iota} & \frac{\partial f_5}{\partial E_\iota} & \frac{\partial f_5}{\partial I_\iota} & \frac{\partial f_5}{\partial R_\iota} \\ \frac{\partial f_6}{\partial S_\iota} & \frac{\partial f_6}{\partial V_\iota} & \frac{\partial f_6}{\partial P_\iota} & \frac{\partial f_6}{\partial E_\iota} & \frac{\partial f_6}{\partial I_\iota} & \frac{\partial f_6}{\partial R_\iota} \end{pmatrix}, \tag{20}$$

at equilibrium point (T_0), the Jacobian becomes

$$J(T_0) = \begin{pmatrix} -(\Psi+\mu) & \theta & 0 & 0 & -c_4 & 0 \\ 0 & -(\theta+\mu) & 0 & 0 & 0 & 0 \\ \psi & 0 & -\mu & 0 & -\eta\Psi c_4 & 0 \\ 0 & 0 & 0 & -(\kappa+\mu) & (\mu+\eta\Psi)c_4 & 0 \\ 0 & 0 & 0 & \kappa & -(\mu+\tau+\delta) & 0 \\ 0 & 0 & 0 & 0 & \tau & -\mu \end{pmatrix} \tag{21}$$

where

$$c_4 = \frac{\beta(\theta+(1-\rho)\mu)}{(\Psi+\mu)(\theta+\mu)}$$

Clearly, four eigenvalues of (21) are $\lambda_1 = -\mu$ $\lambda_2 = -\mu$ $\lambda_3 = -(\theta+\mu)$ and $\lambda_4 = -(\Psi+\mu)$ while the remaining two eigenvalues are obtained from the 2 by 2 matrix

$$A = \begin{pmatrix} -(\kappa+\mu) & (\mu+\eta\Psi)c_4 \\ \kappa & -(\mu+\tau+\delta) \end{pmatrix}$$

Then, the eigenvalues of A are real and negative, if the Routh- Hurwitz condition is satisfied. By applying Routh-Hurwitz condition
(i) Tr(A)<0 (ii) Det (A) >0
Now,

$$\begin{aligned} Tr(A) &= -(\kappa+\mu) - (\mu+\tau+\delta) \\ &= -(\kappa+2\mu+\tau+\delta) < 0 \end{aligned}$$

$$\begin{aligned} Det(A) &= (\kappa+\mu)(\mu+\tau+\delta) - \kappa(\mu+\eta\Psi)c_4 \\ &= (\kappa+\mu)(\mu+\tau+\delta)\left[1 - \frac{\kappa(\mu+\eta\Psi)c_4}{(\kappa+\mu)(\mu+\tau+\delta)}\right] \end{aligned}$$

$$= (\kappa+\mu)(\mu+\tau+\delta)\,(1-R_C)$$

$= (\kappa+\mu)(\mu+\tau+\delta)\,(1-R_C) > 0$ if $R_C < 1$. Following theorem 2 of van den Driesche and Watmough [63], we conclude that the T_0 point is locally asymptotically stable. Hence the theorem.

Note: From the theorem above, we see that the disease-free equilibrium T_0 losses its stability when the R_C increases to a value greater than 1. Therefore, we may reason that at R_C, the system (5) goes through a bifurcation around its T_0, which are examined in section (3.4)

3.3 Global stability of the disease free equilibrium

Lemman 2.4: For the model system (5), the disease-free equilibrium T_0 is globally asymptotically stable if $R_C \leq 1$ and unstable when $R_C \geq 1$.

Proof. Motivated by Garba and Gumel [23], we use the method of Lyapunov function to establish the global asymptotic stability of TFE T_o as follow:
We construct a Lyapunov function defined as

$$L(E_\iota, I_\iota) = \frac{\beta(\mu+\Psi\eta)(\theta+(1-\rho)\mu)}{\kappa(\Psi+\mu)(\theta+\mu)(\mu+\tau+\delta)}E_\iota + \frac{\beta(\mu+\Psi\eta)(\theta+(1-\rho)\mu)}{\kappa(\Psi+\mu)(\theta+\mu)(\mu+\tau+\delta)}I_\iota \tag{22}$$

By differentiating (22)

$$c_5(\kappa+\mu)(\mu+\tau+\delta)\left[\frac{\kappa\beta}{(\kappa+\mu)(\mu+\tau+\delta)N_\iota}(S_\iota+\eta P_\iota+\sigma R_\iota)-1\right]I_\iota$$

where

$$c_5 = \frac{\beta(\mu+\Psi\eta)(\theta+(1-\rho)\mu)}{(\Psi+\mu)(\theta+\mu)(\mu+\tau+\delta)}$$

$$c_5(\kappa+\mu)(\mu+\tau+\delta)\left[\frac{\kappa\beta}{(\kappa+\mu)(\mu+\tau+\delta)N_\iota}(S_\iota+\eta P_\iota+\sigma R_\iota)-1\right]I_\iota$$

At DFE, $T_0 \rightarrow$

$$S_\iota = \frac{(\theta+(1-\rho)\mu)\Lambda)}{(\theta+\mu)(\Psi+\mu)}$$

$$P_\iota = \frac{\Psi(\theta+(1-\rho)\mu)\Lambda)}{\mu(\theta+\mu)(\Psi+\mu)}$$

$E_\iota = I_\iota = R_\iota = 0$

$$L^1 = c_5(\kappa+\mu)(\mu+\tau+\delta)\left[\frac{\kappa\beta(S_\iota+\eta P_\iota)}{(\kappa+\mu)(\mu+\tau+\delta)S_\iota+V_\iota+P_\iota}-1\right]I_\iota$$

$$S_\iota+\eta P_\iota = \frac{(\theta+(1-\rho)\mu)\Lambda)}{(\theta+\mu)(\Psi+\mu)} + \frac{\eta\Psi(\theta+(1-\rho)\mu)\Lambda)}{\mu(\theta+\mu)(\Psi+\mu)} \tag{23}$$

$$\frac{\mu(\theta+(1-\rho)\mu)\Lambda)+\eta\Psi(\theta+(1-\rho)\mu)\Lambda}{\mu(\theta+\mu)(\psi+\mu)} \tag{24}$$

therefore

$$L^1 = c_5(\kappa+\mu)(\mu+\tau+\delta)\left[\frac{\kappa\beta(\mu+\eta\Psi)(\theta+(1-\rho)\mu)\Lambda}{(\kappa+\mu)(\Psi+\mu)(\theta+\mu)(\mu+\tau+\delta)}-1\right]I_\iota$$

$$L^1 = c_5(\kappa+\mu)(\mu+\tau+\delta)\,(R_C-1)\,I_\iota \leq 0.$$

3.4 Endemic equilibria and bifurcation of the model

The endemic equilibrium of the model system can be obtained by setting the right hand side of system model (1) to zero. However, following the approaches of [50, 58, 30, 52], endemic equilibrium point can be derived without any intervention, in order to understand the dynamical behavior of system by setting $V_\iota = P_\iota = \theta = \psi = \omega = \rho = 0$ in system (5). (This is appropriate in some of those communities where vaccination, knowledge, and understanding, are not reachable or not sufficient to cater for those affected). The basic-free model is obtained as follows:

$$\begin{aligned}
\frac{dS_\iota}{dt} &= \Lambda - \lambda S_\iota - \mu S_\iota \\
\frac{dE_\iota}{dt} &= \lambda S_\iota + \sigma\lambda R_\iota - (\kappa + \mu)\, E_\iota \\
\frac{dI_\iota}{dt} &= \kappa E_\iota - (\mu + \delta + \gamma)\, I_\iota \\
\frac{dR_\iota}{dt} &= \gamma I_\iota - \sigma\lambda R_\iota - \mu R_\iota.
\end{aligned} \tag{25}$$

Where the associated basic reproduction number of the free basic model (25) can also be obtained by following the same procedure of the model with intervention (5), represented by R_0, which is given by

$$R_0 = \frac{\beta\kappa}{(\kappa + \mu)(\mu + \delta + \tau)}. \tag{26}$$

From equation (25), the endemic equilibrium is obtained by

$$S_\iota = \frac{\Lambda}{\lambda + \mu}, E_\iota = \frac{\Lambda(\mu + \delta + \gamma)(\sigma\lambda + \mu)\lambda}{(\lambda + \mu)(g_0\lambda + g_1)}, I_\iota = \frac{\kappa\Lambda(\sigma\lambda + \mu)\lambda}{(\lambda + \mu)(g_0\lambda + g_1)}, R_\iota = \frac{\kappa\sigma\Lambda\lambda}{(\lambda + \mu)(g_0\lambda + g_1)}.$$

Where

$$\begin{aligned}
\lambda &= \frac{\beta I_\iota}{N_\iota} \\
g_0 &= (\kappa + \mu)(\mu + \delta) + \mu\tau \\
g_1 &= \mu(\kappa + \mu)(\mu + \tau + \delta)
\end{aligned} \tag{27}$$

It is therefore easy to get

$$\frac{I_\iota}{N_\iota} = \frac{\kappa\sigma\lambda^2 + \kappa\mu\lambda}{(\sigma(\mu + \delta + \gamma) + \kappa\sigma)\lambda^2 + (g_0 + \mu(\mu + \delta + \gamma) + \kappa(\mu + \gamma)\lambda + g_1} \tag{28}$$

using equation (28) in (27) yields

$$\lambda = \frac{\beta\kappa\sigma\lambda^2 + \beta\kappa\mu\lambda}{g_2\lambda^2 + g_3\lambda + g_1} \tag{29}$$

$$\begin{aligned}
&\Rightarrow \lambda(g_2\lambda^2 + g_3\lambda + G_1) = \beta\kappa(\sigma\lambda + \mu)\lambda \\
&\Rightarrow (g_2\lambda^2 + g_3\lambda + g_1) - \beta\kappa(\sigma\lambda + \mu) = 0 \\
&\Rightarrow g_2\lambda^2 + (g_3 - \beta\kappa\sigma)\lambda + g_1 - \beta\kappa\mu = 0 \\
&G_2\lambda^2 + (g_3 - \beta\kappa\sigma)\lambda + \mu(\kappa + \mu)(\mu + \delta + \gamma) - \beta\kappa\mu = 0 \\
&G_2\lambda^2 + (g_3 - \beta\kappa\sigma)\lambda + \mu(\kappa + \mu)(\mu + \delta + \gamma)\left(1 - \frac{\beta\kappa}{(\kappa + \mu)(\mu + \delta + \gamma)}\right) = 0
\end{aligned}$$

$$g_2\lambda^2 + (g_3 - \beta\kappa\sigma)\lambda + \mu(\kappa + \mu)(\mu + \delta + \gamma)(1 - R_0) = 0, \tag{30}$$

where

$$\begin{aligned}
g_2 &= \sigma(\mu + \delta + \gamma) + \kappa\sigma \\
g_3 &= g_0 + \mu(\mu + \delta + \gamma) + \kappa(\mu + \sigma).
\end{aligned}$$

Theorem 3.1. *The system (25) experiences a transcritical bifurcation across its disease-free equilibrium T_0, when $R_0 = 1$.*

Proof. It has been observed from model system (1) when $R_0 < 1$. Only the disease-free equilibrium occurs between the two equilibria, and locally asymptotically stable wherever $R_0 > 1$ 1 is the threshold condition of the endemic equilibrium point for both the presence and the asymptotic stability criteria, since the disease-free equilibrium T_0 reduces to unstable nature at the $R_0 > 1$ threshold. We may, therefore, conclude that both feasibility and stability changes occur at $R_0 = 1$. Following the papers: Jana et al. [31], Guken- heimer and Holmes [25], Kar and Jana [34], Mondal et al. [44], and Kar and Mondal [32], among others, it is concluded that the system undergoes a transcritical bifurcation at $R_0 = 1$. In Fig. 2, we represent the transcritical bifurcation phenomenon graphically.

4 Numerical simulation and discussion

In this section, TB model (1) is solved numerically with parameter values displayed in Table 2, in order to have insight into its quantitative characteristics of model system (1) and (25) in Matlab. The ode45 solver, which depends on the Runge-Kutta technique was utilize. As appeared in Table 2, the parameter values utilized depended on the existing literatures. The numerical simulations are carried out by varying some of the main parameters in the model to demonstrate different dynamical routine features. The impacts of changing a portion of the main key parameter on the exposed and infected individuals are introduced and potential preventive measures characterised by these variations will be discussed. In the description of the various figure, the values of the parameters that are different in relation to those recorded in Table 2 are shown.

Table 2: Parameters values for the TB model, Eq. (1)

Parameters	Baseline values	Ranges	References
Λ	10	5-10	[32, 65]
ρ	0.75 yr^{-1}	0-1	[6, 21]
θ	0.067 yr^{-1}	0.067-1	[49, 4]
Ψ	5	0 - 40	[52, 53]
η	0.4 yr^{-1}	0-1	[53]
μ	0.02 yr^{-1}	0.0143-0.04	[22]
δ	0.12 yr^{-1}	0-0.15	[45, 33]
β	1.67yr^{-1}	1.5-3.5	[54, 32]
σ	0.5 yr^{-1}	0.25-1	[24, 16]
κ	0.02yr^{-1}	0-0.005	[13, 32]
τ	2 ind^{-1},yr^{-1}	1.5-3.5	[61, 65, 32]

Fig. 2 shows the bifurcation diagram of the basic free model of Eq. (25) as the basic reproduction number (R_0) is varied. A critical value occurs, that is, BP, which corresponds to transcritical bifurcation. From an epidemiological point of view, R_0 is one of the most significant records of epidemic models. This quantity gives the basis for deciding if the infectious disease can spread into an entire community or not [17, 11, 38, 19, 20]. In general, there are several branches of steady states: (i) the upper branch corresponding to stable endemic equilibria, EEP; (ii) the middle branch, representing unstable EEP; and (iii) the lower branch corresponds disease free equilibria, DFE, which can be stable or unstable depending on the magnitudes of R_0. As in our theoretical analysis section, the R_0 quantity can be calculated using equation (26). The emergence of alternate stable states is observed, with both endemic and disease-free equilibria being stable. The convergence to any of these stable states relies upon the initial abundance of individuals in the population, when $R_0 > 1$, only EEP is stable, and this

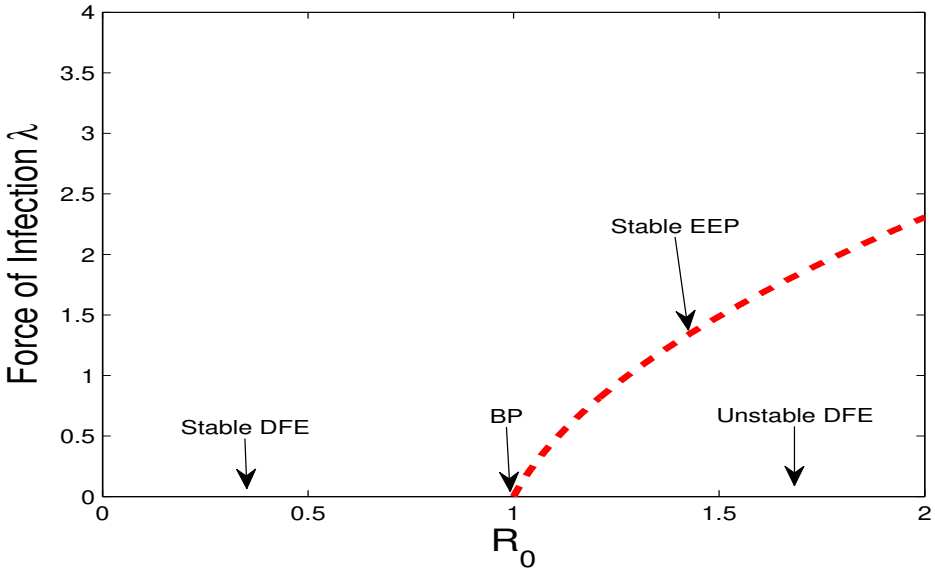

Figure 2: Bifurcation diagram of the basic free model of eq. (25) demonstrating force of infection λ as R_0 varies with threshold BP represents transcritical bifurcation.

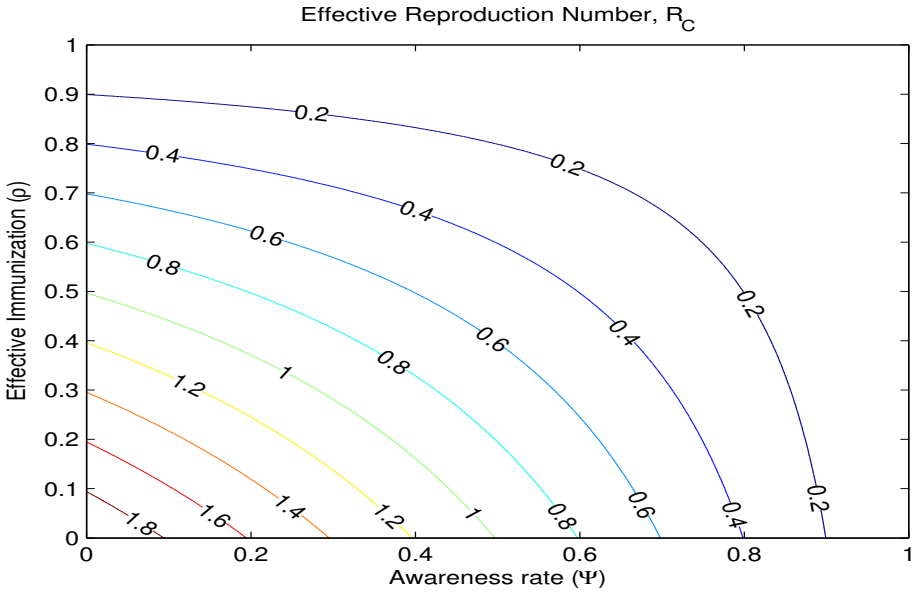

Figure 3: Simulation of the system model (5) showing a contour plot of R_C as a function of information dissemination (awareness rate Ψ and effective immunization ρ.

situation leads to an outbreak of TB disease. Also noticed is that as the basic reproduction number decreases and lies below LP point, i.e., $R_0 < LP$, DFE is stable in this case. Consequently, this situation leads to elimination of the disease.

Fig. 3 depicts the simulation of the system model (1) showing a contour plot of R_C as a function of information dissemination (awareness rate) Ψ and effective immunization ρ. The Fig. 3 indicated that the parameters Ψ and ρ have positive impacts on the effective reproduction number, R_C. Therefore, improving on information dissemination Ψ and effective immunization ρ will bring the value of R_C below one and bring the burden of the TB epidemics very low.

The effects of varying information dissemination i.e., awareness rate (Ψ) between 0 and 5 and decreasing transmission rate (β) from 6.55 to 2, n the exposed and infected classes, at the final time

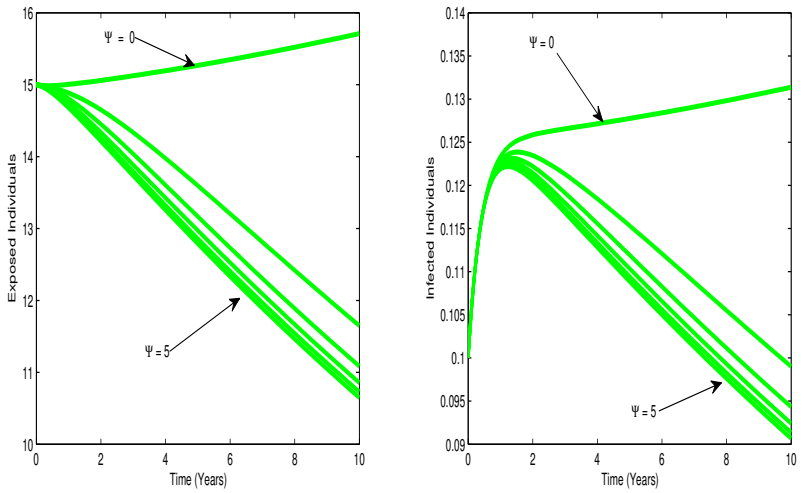

Figure 4: Simulations of system (5) showing the number of exposed and infected individuals. Here, the effect of information dissemination on the susceptible individuals (Ψ) is varied from 0 to 5 with β = 6.55, with 10 years of simulation.

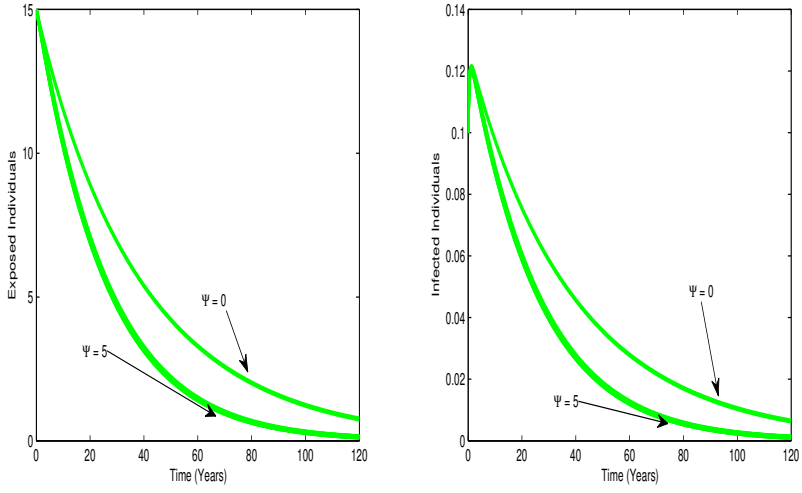

Figure 5: Simulations of system (5) showing the number of exposed and infected individuals. Here, effect of information dissemination on the susceptible individuals (Ψ) is varied from 0 to 5, with β = 2, and 120 years of simulation.

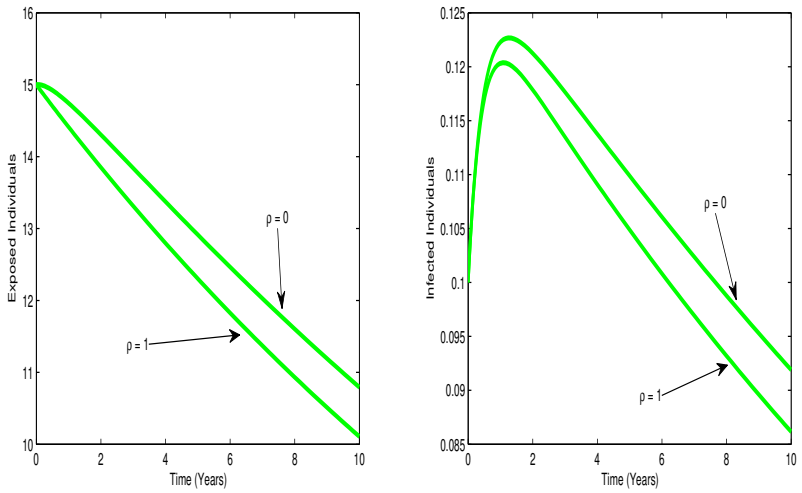

Figure 6: Simulations of system (5) showing the number of exposed and infected individuals. Here, the effect of effective immunization rate on the susceptible individuals (ρ) is varied from 0 to 1, with the fixed parameters in Table 1, and 10 years of simulation.

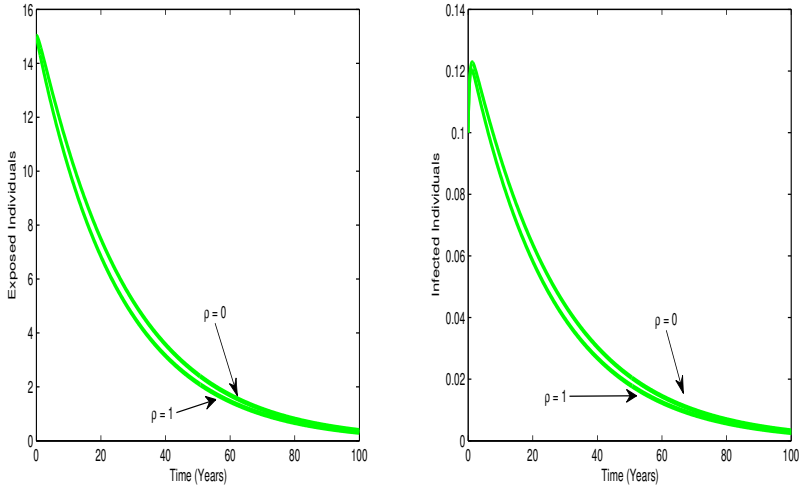

Figure 7: Simulations of system (5) showing the number of exposed and infected individuals. Here, the effect of effective immunization rate on the susceptible individuals (ρ) is varied from 0 to 1, and with the fixed parameters in Table 1, and 100 years of simulation.

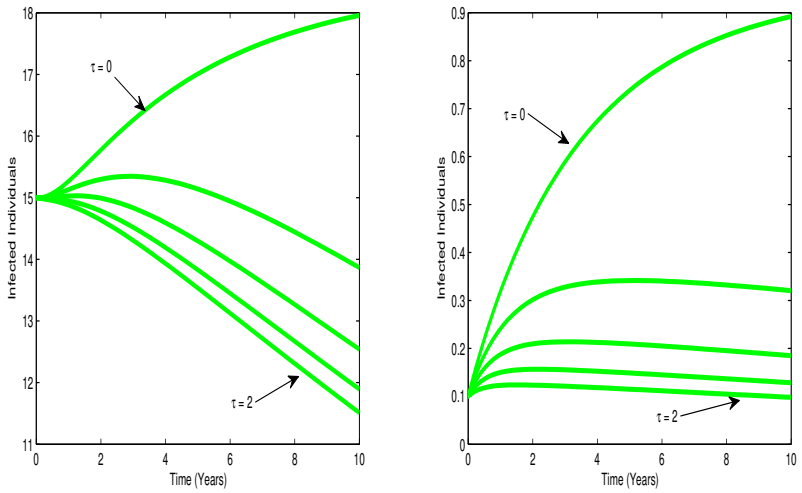

Figure 8: Simulations of system (5) showing the number of exposed and infected individuals. Here, the effect of treatment on the infected individuals (τ) is varied from 0 to 2, with the fixed parameters in Table 1, and 10 years of simulation.

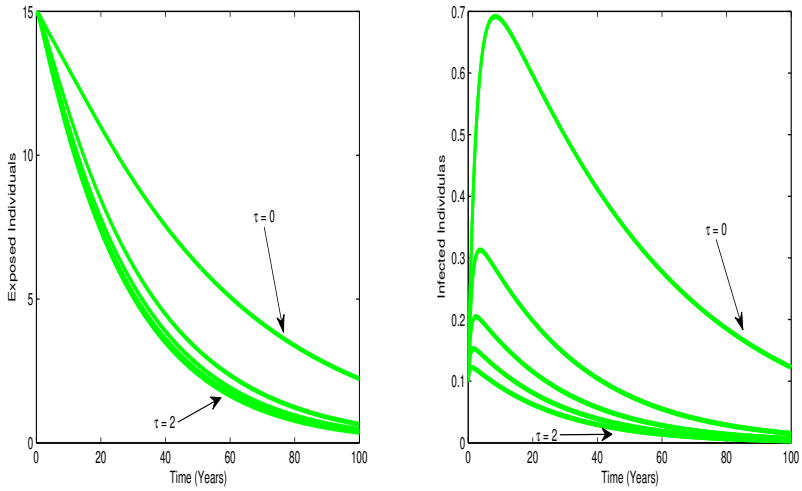

Figure 9: Simulations of system (5) showing the number of exposed and infected individuals. Here, the effect of treatment on the infected individuals (τ) is varied from 0 to 2, with the fixed parameters in Table 1, and 100 years of simulation.

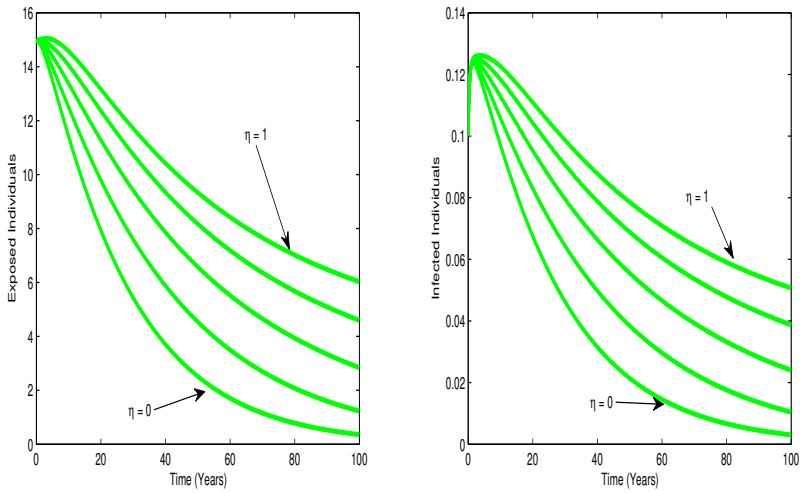

Figure 10: Simulations of system (5) showing the number of infected individuals. Here, the decreased risk of TB infection, due to information dissemination to the susceptible individual, (η), is varied from 0 to 1, with $\Psi = 1$, and 100 years of simulation.

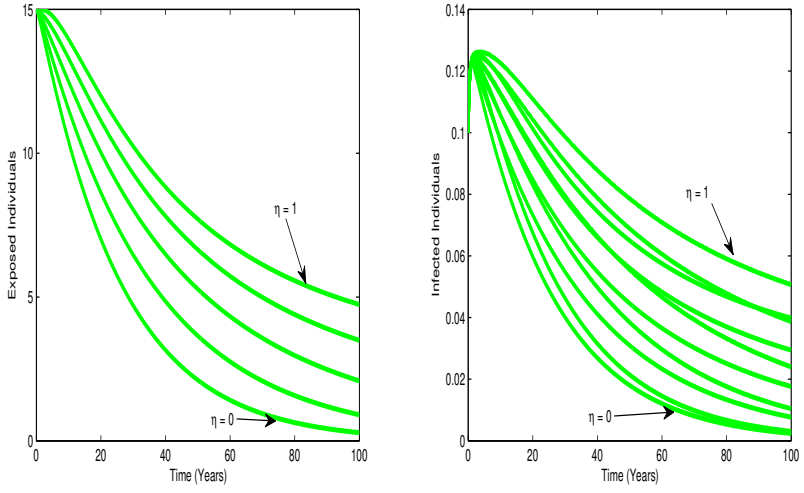

Figure 11: Simulations of system (5) showing the number of infected individuals. Here, the decreased risk of TB infection, due to information dissemination to the susceptible individual, (η), is varied from 0 to 1, with $\Psi = 5$, and 100 years of simulation.

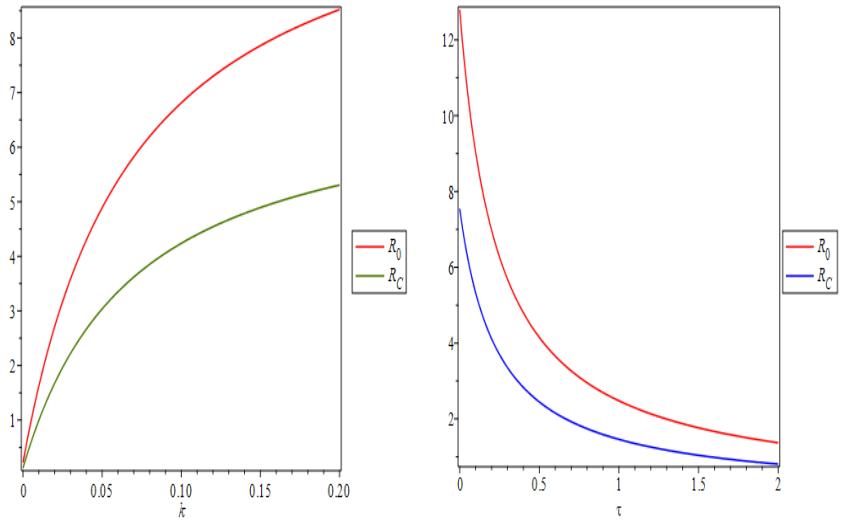

Figure 12: Simulations of system (5) and (25), showing the relationship between basic reproduction numbers and effective reproduction numbers, as κ and τ changes

of 120 years are showed in Figs. 4 and 5, respectively. By implication, improving the information dissemination rate (Ψ), i.e., the rate at which an individual is aware about how TB spreads, and the ability to detect the signs and symptoms of TB, will bring about the reduction of TB transmission rate (β) in the population. The work of Cui et al. [14] and Li and Dong [39] stated that the more prophylactic awareness the population has, the lower will be the risk of spread of TB infection. As such, we clearly observed that increasing information dissemination, as well as decreasing transmission rate, yield a positive impact in the dynamics of both exposed and infected individuals as shown in Figs.5.

Figs. 6 and 7 illustrate the impact of varying immunization rate (ρ) between 0 and 1 on the exposed and infected classes at the final time of 10 years and 100 years respectively. Obviously, as expected, an increase in the value of ρ, from 0 to 1 greatly reduced the number of individuals both the exposed and the infected, but at various rates.

Figs. 8 and 9 show the influences of treatment (τ) between 0 and 2 on the exposed and infected classes at the final time of 10 years and 100 years respectively. Clearly, as anticipated, an increase in the value of τ, from 0 to 2, yields productive positive impacts in the dynamics of TB infection with a reduction of both exposed and infected individuals, yet at different rates.

Figs. 10 and 11 demonstrate when we vary the decreased risk of TB infection, due to information dissemination among susceptible individuals (η), between 0 and 1, at the final time of 10 years and 100 years respectively. Clearly, the reduction of the value of η from 1 to 0, as predicted, reduces the peak value of both exposed and infected classes, but at different rates, based on the estimations of the awareness thresholds for the susceptible groups.

Figs. 12(right) show the comparison between basic reproduction number (R_0) and effective reproduction number (R_C) as progression rate κ varied. We have seen that both R_0 and R_C increase with increasing progression rate κ. Very clearly, R_0 is higher than R_C. That is, as the progression rate increases, both R_0 and R_C increase, and the TB infection spread more in the community. Successful screening programmes that guarantee early discovery and appropriate treatment of exposed individuals, to avoid progression into actively infected groups should be set up.

Figs. 12(left) illustrates the relationship between basic reproduction number (R_0) and effective reproduction number (R_C) as treatment rate τ varied. We observed that both R_0 and R_C decrease with increasing treatment rate of TB individuals undergoing treatment. In this situation, we clearly noted that R_C decreases drastically more than R_0. However, as the population of TB infected individuals decreases, due to treatment, the exposed and infected with TB reduces, which prompts a decreases in both R_0 and R_C. If R_0 and R_C go below one, eventually, the TB burden will fade out from the popu-

lation.

Remarks: Consider the differences in the time window for the various figures, some with 10, 100, or 120 years of simulation. The diagrams with a 10-year time window were utilized to examine the trend of the dynamics of the TB infection for the first few years of simulations or infection case. We see that the outcomes could, without doubt, be drowned, if the graphs have longer time windows. On the other hand, figures with 100 or long term time windows were used to explore the presence of equilibria. Conversely, figures with a time window of 100 or 120 years have been used to examine the nature of equilibria, depending on the values of the parameters used. Additionally, it is very important to mention that the dynamics of tuberculosis are slow, and that TB epidemics grow over many decades [7].

5 Conclusion

A new and plausible mathematical model of TB dynamics has been formulated and mathematically analyzed. The system model (1) extends what has been developed and researched by including vaccination and educational counselling. The mode (1) has DFE that is locally asymptotically stable whenever the effective reproduction number is less than unity. Additionally, it has been indicated that the model with no control measures will go through transcritical bifurcation at the threshold quantity $R_0 = 1$. Transcritical bifurcations is also considered in other biological systems such as [32, 34, 44, 40, 46].

The numerical simulations of the TB model (1), using relevant parameters set out in Table 1, show that the total number of both exposed and infected individuals is significantly reduced as we increase information dissemination (Ψ), immunization rate (ρ), and treatment of actively infected individuals (τ). In addition, if a very large fraction of the susceptible population is promptly educated at the rate Ψ, and the transmission rate is low, it will help to decrease the predominance of TB in the population. One of the more important findings from this analysis is that the numerical results for model (1) demonstrate that TB infection can be eradicated in the long run.

In sum, this study has demonstrated that the possibility of adequately limiting the transmission of tuberculosis infection in a community is extremely bright. Preventive measures, using control techniques, should focus on awareness campaigns and vaccination. The enlightenment services should include helping the general public to speedily recognise the basic signs of TB, such as chronic cough lasting at least 2 weeks, and to provide early immunization to the population.

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