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STABILITY ANALYSIS OF DYNAMICS OF TUBERCULOSIS MODEL IN ASSESSING THE EFFECT OF PUBLIC HEALTH EDUCATION CAMPAIGN.

Ezieke N.[1], Nwasuka S.C.[2], Nwachukwu I.E.[3] and Ajike E.E.[4]

[1]Department of Industrial Mathematics, Ebonyi State University Abakaliki, Ebonyi State Nigeria
[2]Department of Mathematics and Computer Science, Clifford University, Owerrinta, Abia State
[3]Department of Mathematics and Computer Science, Clifford University, Owerrinta, Abia State
[4]Department of Physics, Clifford University, Owerrinta, Abia State

Abstract

In this paper, mathematical model for tuberculosis disease dynamics is presented. The basic mathematical properties of solution of the model are examined; the effect of public health education campaign was assessed which was found the most effective intervention for minimizing the transmission of TB in a population. Finally, the graphical profile of some of the solution of the model is presented and discussed.

Keywords: Tuberculosis, Global Stability, Public Health Education Campaign.

1 Introduction

Tuberculosis (TB) has existed for millennia and remains a major global health problem. It causes ill-health in millions of people each year and in 2015 was one of the top 10causes of death worldwide, ranking above HIV/AIDS as one of the leading causes of death from an infectious disease [1].TB is an infectious disease caused by the bacillus Mycobacterium tuberculosis. It typically affects the lungs (pulmonary TB) but can affect other sites as well (extra pulmonary TB) [2].The disease is spread in the air when people who are sick with pulmonary TB expel bacteria, for example by coughing. Overall, a relatively small proportion of people infected with M. tuberculosis will develop TB disease. However, the probability of developing TB is much higher among people infected with HIV. TB is also more common among men than women, and affects mainly adults in the most economically productive age groups. TB is treated through the use of effective drug. Effective drug treatments were first developed in the 1940s. The most effective first-line anti-TB drug, rifampicin, became available in the 1960s. People with latent TB infection have TB bacteria in their bodies, but they are not sick because the bacteria are not active. People with latent TB infection do not have symptoms, and they cannot spread TB bacteria to others. However, if TB bacteria become active in the body and multiply, the person will go from having latent TB infection to being sick with active TB disease. For this reason, people with latent TB infection are often prescribed treatment to prevent them from developing TB disease. Treatment of latent TB infection is essential for controlling and eliminating TB. Because there are less bacteria in a person with latent TB infection, treatment is much easier. Four regimens are approved for the treatment of latent TB infection. The medications used to treat latent TB infection include: The currently recommended treatment for new cases of drug-susceptible TB is a six-month regimen of four first-line drugs: isoniazid, rifampicin, ethambutol and pyrazinamide. Treatment success rates of 85% or more for new cases are regularly reported to WHO by its Member States. TB treatment saved 49 million lives globally between 2000 and 2015.TB bacteria become active (multiplying in the body) if the immune system can't stop them from growing. When TB bacteria are active, this is called TB disease. TB disease will make a person sick. People with TB disease may spread the bacteria to people with whom they spend many hours.

2. Model Formulation

In our model formulation, the total population size $N(t)$ is divided into six epidemiological classes, VIZ: vaccinated $V(t)$, Susceptible $S(t)$, Exposed $L(t)$, Infectious $I(t)$, Treated $T(t)$ and Recovered $R(t)$. In this model, the vaccinated population increases as a result of the individuals who are recruited by either immigration at the rate Λ and per capital birth rate π. The population however decreases as a result of those babies whose BCG vaccine has expired and due to natural death rate μ.

Table 1: State Variables of the model

Variable	Description
$V(t)$	Number of vaccinated individuals at time, t
$S(t)$	Number of susceptible individuals at time, t
$L(t)$	Number of exposed (latently) individuals at time, t
$I(t)$	Number of infections (active) individuals at time, t
$T(t)$	Number of treated individuals at time, t
$R(t)$	Number of recovered individuals at time, t
$N(t)$	Total Population at time t

Correspondence Author: Ezieke N., Email: eziekenelson@gmail.com, Tel: +2348063474188

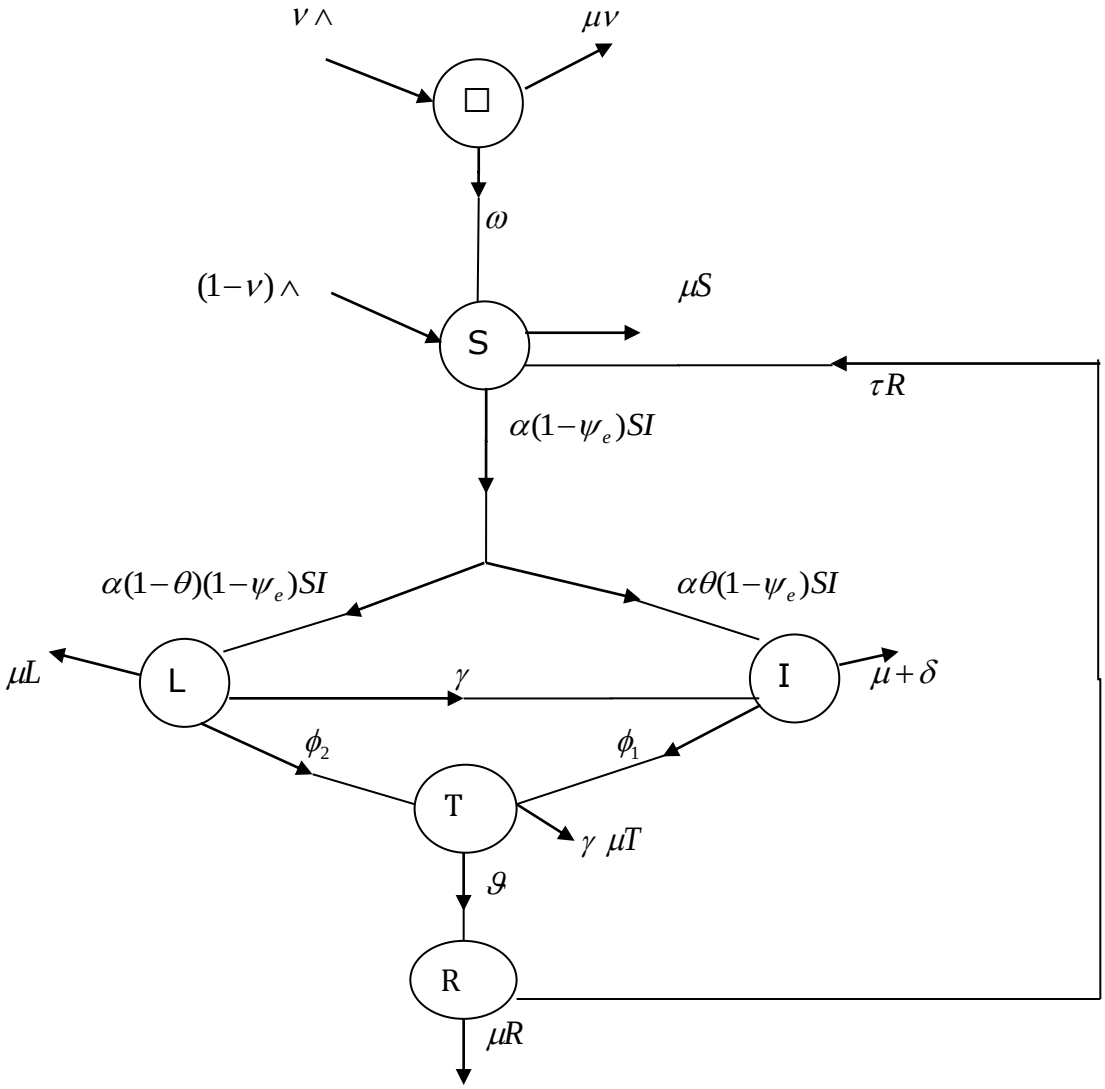

Figure 1: A compartmentalized diagram showing the TB model with public Health Education Campaign

Table 2: Parameters of the model

Parameter	Description
γ	Breakdown rate from the exposed class to the infectious class
α	Infection rate
ν	Vaccination rate
ω	Waning rate of the BCG vaccine
$\wedge$	Recruitment number(due to birth)
$\nu\wedge$	Proportion of vaccinated individuals at birth
$(1-\nu)\wedge$	Proportion of individuals not vaccinated at birth
ψ_e	Public health education campaign
θ	Proportion of individuals acquiring active(infections) TB infection
μ	Natural death rate
δ	Death rate due to TB
ϕ_1	Treatment rate for the infectious individual
ϕ_2	Treatment rate for the latently(exposed) infected individual
ϑ	Recovery rate of the treated individuals
τ	Movement rate of the recovered individual back to the susceptible class

Table 2 : Parameters of the Basic Model with Public Health Education Campaign

Based on our model variables and parameters, assumptions in section 3 and the flow diagram in figure 1, the following non-linear ordinary differential equations were derived.

$$\frac{dV}{dt} = \nu \wedge -(\mu + \omega)\nu \quad (1)$$

$$\frac{dS}{dt} = (1-\wedge) \wedge -\alpha(1-\psi_e)\frac{SI}{N} + \omega V + \tau R - \mu S \quad (2)$$

$$\frac{dL}{dt} = \alpha(1-\theta)(1-\psi_e)\frac{SI}{N} - (\gamma + \mu + \phi_2)L \quad (3)$$

$$\frac{dI}{dt} = \alpha\theta(1-\psi_e)\frac{SI}{N} + \gamma L - (\mu + \delta + \phi_1)I \quad (4)$$

$$\frac{dT}{dt} = \phi_2 L + \phi_1 I - \mu T - \vartheta T \quad (5)$$

$$\frac{dR}{dt} = \vartheta T - (\mu + \tau)R \quad (6)$$

where

$$N = V + S + L + I + T + R \quad (7)$$

Summing (1)-(6) yields

$$\frac{dN}{dt} = \wedge - \mu N - \delta I \quad (8)$$

2.1 MODEL ANALYSIS

The model (1) – (6) is analyzed qualitatively to give insights into its dynamical features that give better understanding of the impacts of vaccination, treated and public health education campaign on the transmission dynamics of TB. First, we have the following important theorems on nonlinear systems of differential equations.

2.2Theorem

Given $f : R^n \to R^n$ is differentiable at X_0, then the partial derivatives $\partial f_i / \partial x_j,\ i, j = 1, \ldots, n,$ all exist at X_0 and for all $x \varepsilon R^n$

$$Df(x_0)x = \sum \frac{\partial f(x_0)}{\partial xj} xj$$

Thus, if *f* is a differentiable function, the derivative Df is given by the $n \times n$ Jacobian matrix.

$$Df = \left[\frac{\partial fi}{\partial xj}\right]$$

2.3 Definition: equilibrium x^* of the system $\dot{x} = f(x)$ is called hyperbolic if all eigen-values of the Jacobian $Df(x^*)$ have non-zero real part.

2.4 Invariant Region

This region will be obtained by considering the following theorem

Theorem 1:The solutions of the system (4) are feasible for all t >0 if theyenter the invariant region Ω.

Proof 1:Let $\Omega = (V + S + L + I + T + R)$ be any solution of the system (4)with non-negativeinitial conditions. From equation (8), in absence of the disease (TB), δ = 0 and equation (8) becomes

$$\frac{dN}{dt} = \wedge - \mu N - \delta I \quad (9)$$

$$\wedge - \mu N \quad (10)$$

$$N \le \frac{\wedge}{\mu} \quad (11)$$

Integrating on both sides we get;

$N \le \frac{\wedge}{\mu} + c$ where c is a constant of integration

Using the initial conditions; when t = 0, N(0) = N_0

$$N_0 - \frac{\wedge}{\mu} \le c$$

$$N \le \frac{\wedge}{\mu} + \left(N_0 - \frac{\wedge}{\mu}\right)$$

Applying Birkhof and Rota's theorem on differential inequality [3], we obtain $0 \le N \le \frac{\wedge}{\mu}$ as $t \to \infty$ the total population approaches

$k = \frac{\wedge}{\mu}$ as $t \to \infty$ which is commonly termed

as the carrying capacity. Therefore, the feasible solutions set of the model (1-6)enters the region

$$\left\{\Omega=(V,S,L,I,T,R)\mu-\delta I\right]V\geq 0,S\geq 0,L\geq 0,I\geq 0,T\geq 0,R\geq 0,N\leq\frac{\wedge}{\mu}\right\}.$$

Thus in this region our model is biologically feasible.

2.5 Analysis of the Basic Model

In this section, (1-6) is qualitatively analyzed to investigate the disease free equilibrium state.

3 Existence of Disease Free Equilibrium Point (DFE),E^0

Let $E(V^0,S^0,L^0,I^0,T^0,R^0)$ be the equilibrium points of the model system (1) -(6).

$$\frac{dV}{dt}=\frac{dS}{dt}=\frac{dE}{dt}=\frac{dI}{dt}=\frac{dT}{dt}=\frac{dR}{dt}=0$$

In case of no disease, L=I=T=R=0 the sum of susceptible and Vaccinated populations is equal to total population.

Hence, the system 1-6 is reduced to

$$\nu=\frac{\nu\wedge}{(\mu+\omega)} \tag{12}$$

and

$$\therefore S=\frac{(1-\nu)\wedge+\nu\omega\wedge}{\mu(\mu+\omega)} \tag{13}$$

The DFE state is thus given by

$$(V^0,S^0,L^0,I^0,T^0,R^0)=\left(\frac{\nu\wedge}{\mu+\omega},\frac{(1-\nu)\wedge+\nu\omega\wedge}{\mu(\mu+\omega)},0,0,0,0\right) \tag{14}$$

(9) Shows the state in which there is no TB infection and is known as the disease-free equilibrium point.

4. The effective Reproduction Number, (R_E)

The effective reproduction number, of the normalized model system (1-6) with vaccination, Treatment and Public health education campaign is:

$$\lambda\left(\lambda-\frac{\left\{\alpha\theta(\gamma+\mu+\phi_2)\left[1-\psi_e(1-\nu)+\nu\omega\right]+\left[\alpha\gamma(1-\theta)(1-\psi_e)(1-\nu)+\nu\omega\right]\right\}}{(\mu+\omega)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)}\right)=0 \tag{15}$$

$$\lambda_1\ \frac{\left\{\alpha\theta(\gamma+\mu+\phi_2)\left[1-\psi_e(1-\nu)+\nu\omega\right]+\left[\alpha\gamma(1-\theta)(1-\psi_e)(1-\nu)+\nu\omega\right]\right\}}{(\mu+\omega)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)} \tag{16}$$

and. $\lambda_2=0$

Clearly, λ_1 is the dominant Eigen-value and therefore becomes the effective reproduction number (RE) of the model (1) - (6).

$$\therefore RE=\frac{\left\{\alpha\theta(\gamma+\mu+\phi_2)\left[1-\psi_e(1-\nu)+\nu\omega\right]+\left[\alpha\gamma(1-\theta)(1-\psi_e)(1-\nu)+\nu\omega\right]\right\}}{(\mu+\omega)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)} \tag{17}$$

Where:

$\frac{1}{\gamma+\mu+\phi_2}$ The duration of latency

$\frac{\gamma}{(\gamma+\mu+\phi_2)}$ The proportion of individuals from the stage that becomes infectious

$\frac{1}{\mu+\delta+\phi_1}$ The effective infectious period

$\frac{\tau}{(\mu+\delta+\phi_1)}$ The number of susceptible infected by one infectious individual during the infectious period.

The details for the computation of the basic reproduction number and the comparison between the effective reproduction numbers with individual or combination ofdifferent interventions areshown in[4]

Local Stability of the Disease-free Equilibrium State (DFE),E_0

In order to obtain conditions for the local stability of the disease-free equilibrium state, we re-write equations (1)-(6) as follows:

$$f1=\vee\wedge-(\mu+\omega)\vee \tag{18}$$

$$f2 = (1-\upsilon)\wedge - \alpha(1-\psi e)\frac{SI}{N} + \omega\upsilon + \tau R - \mu S \tag{19}$$

$$f3 = \alpha(1-\theta)(1-\psi_e)\frac{SI}{N} - (\gamma + \mu + \phi_2)L \tag{20}$$

$$f4 = \alpha\theta(1-\psi_e)\frac{SI}{N} + \gamma L - (\mu + \delta + \phi_1)I \tag{21}$$

$$f5 = \phi_2 L + \phi_1 I - \mu T - \vartheta T \tag{22}$$

$$f6 = \vartheta T - (\mu + \tau)R \tag{23}$$

We now obtain the partial derivatives of $f_1, f_2, f_3, f_4, f_5 and f_6$ with respect to $V, S, L, I, T and R$ as follows:

$$\frac{\partial f_1}{\partial V} = -\mu - \omega,\ \frac{\partial f_1}{\partial S} = 0, \qquad \frac{\partial f_1}{\partial L} = 0,\ \frac{\partial f_1}{\partial I} = 0, \qquad \frac{\partial f_1}{\partial T} = 0 \text{ and } \frac{\partial f_1}{\partial R} = 0 \tag{24}$$

$$\frac{\partial f_2}{\partial V} = \omega,\ \frac{\partial f_2}{\partial S} = -\frac{\alpha(1-\psi_e)I\mu}{\wedge} - \mu,\ \frac{\partial f_2}{\partial L} = 0,\ \frac{\partial f_2}{\partial I} = -\frac{\alpha(1-\psi_e)S\mu}{\wedge},\ \frac{\partial f_2}{\partial T} = 0,\ \frac{\partial f_2}{\partial R} = \tau \tag{25}$$

$$\frac{\partial f_3}{\partial V} = 0,\ \frac{\partial f_3}{\partial S} = \frac{\alpha(1-\psi_e)(1-\theta)I\mu}{\wedge},\ \frac{\partial f_3}{\partial L} = (\gamma + \mu + \phi_2),\ \frac{\partial f_3}{\partial I} = \frac{\alpha(1-\psi_e)(1-\theta)S\mu}{\wedge},\ \frac{\partial f_3}{\partial T} = 0,\ \frac{\partial f_3}{\partial R} = 0 \tag{26}$$

$$\frac{\partial f_4}{\partial V} = 0,\ \frac{\partial f_4}{\partial S} = \frac{\alpha\theta(1-\psi_e)I\mu}{\wedge},\ \frac{\partial f_4}{\partial L} = \gamma,\ \frac{\partial f_4}{\partial I} = \frac{\alpha\theta(1-\psi_e)S\mu}{\wedge} + \gamma + \mu + \phi_1,\ \frac{\partial f_4}{\partial T} = 0,\ \frac{\partial f_4}{\partial R} = 0 \tag{27}$$

$$\frac{\partial f_5}{\partial V} = 0,\ \frac{\partial f_5}{\partial S} = 0,\ \frac{\partial f_5}{\partial L} = \phi_2,\ \frac{\partial f_5}{\partial I} = \phi_1,\ \frac{\partial f_5}{\partial T} = -\mu - \vartheta,\ \frac{\partial f_5}{\partial R} = 0 \tag{28}$$

$$\frac{\partial f_6}{\partial V} = 0,\ \frac{\partial f_6}{\partial S} = 0,\ \frac{\partial f_6}{\partial L} = 0,\ \frac{\partial f_6}{\partial I} = 0,\ \frac{\partial f_6}{\partial T} = \vartheta,\ \frac{\partial f_6}{\partial R} = -\mu - \tau \tag{29}$$

We substitute the above partial derivatives into the Jacobian matrix below

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial L} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial V} & \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial L} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_{21}}{\partial R} \\ \frac{\partial f_3}{\partial V} & \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial L} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial V} & \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial L} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial V} & \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial L} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial R} \\ \frac{\partial f_6}{\partial V} & \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial L} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial T} & \frac{\partial f_6}{\partial R} \end{pmatrix} \tag{30}$$

That is,

$$J = \begin{pmatrix} -\mu-\omega & 0 & 0 & 0 & 0 & 0 \\ \omega & \frac{-\alpha(1-\psi_e)I\mu}{\wedge} & 0 & \frac{-\alpha(1-\psi_e)S\mu}{\wedge} & 0 & \tau \\ 0 & \frac{\alpha(1-\psi_e)(1-\theta)I\mu}{\wedge} & (\gamma+\mu+\phi_2) & \frac{\alpha(1-\psi_e)(1-\theta)S\mu}{\wedge} & 0 & 0 \\ 0 & \frac{\alpha\theta(1-\psi_e)I\mu}{\wedge} & \gamma & \frac{\alpha\theta(1-\psi_e)S\mu}{\wedge} + \gamma + \mu + \phi_1 & 0 & 0 \\ 0 & 0 & \phi_2 & \phi_1 & -\mu-\vartheta & 0 \\ 0 & 0 & 0 & 0 & \vartheta & -\mu-\tau \end{pmatrix} \tag{31}$$

Substituting 30 into 31 yields

$$J(E_0) = \begin{pmatrix} -\mu-\omega & 0 & 0 & 0 & 0 & 0 \\ \omega & -\mu & 0 & \frac{-\alpha(1-\psi_e)\left[(1-\upsilon)\wedge + \upsilon\omega\wedge\right]}{(\mu+\omega)\wedge} & 0 & \tau \\ 0 & 0 & \gamma+\mu+\phi_2 & \frac{\alpha(1-\psi_e)(1-\theta)\left[(1-\upsilon)\wedge + \upsilon\omega\wedge\right]}{(\mu+\omega)\wedge} & 0 & 0 \\ 0 & 0 & \gamma & \frac{\alpha\theta(1-\psi_e)\left[(1-\upsilon)\wedge + \upsilon\omega\wedge\right]}{(\mu+\omega)\wedge} + \gamma + \mu + \phi_1 & 0 & 0 \\ 0 & 0 & \phi_2 & \phi_1 & -\mu-\vartheta & 0 \\ 0 & 0 & 0 & 0 & \vartheta & -\mu-\tau \end{pmatrix} \tag{32}$$

We next compute $|J(E_0)-\lambda I|=0$ as follows:

$$\begin{vmatrix} -\lambda-(\mu+\omega) & 0 & 0 & 0 & 0 & 0 \\ \omega & -\lambda-\mu & 0 & \frac{-\alpha(1-\psi_e)[(1-\upsilon)\wedge+\upsilon\omega\wedge]}{(\mu+\omega)\wedge} & 0 & \tau \\ 0 & 0 & -\lambda+\gamma+\mu+\phi_2 & \frac{\alpha(1-\psi_e)(1-\theta)[(1-\upsilon)\wedge+\upsilon\omega\wedge]}{(\mu+\omega)\wedge} & 0 & 0 \\ 0 & 0 & \gamma & -\lambda+\frac{\alpha\theta(1-\psi_e)[(1-\upsilon)\wedge+\upsilon\omega\wedge]}{(\mu+\omega)\wedge}+\gamma+\mu+\phi_1 & 0 & 0 \\ 0 & 0 & \phi_2 & \phi_1 & -\lambda-\mu-\vartheta & 0 \\ 0 & 0 & 0 & 0 & \vartheta & -\lambda-\mu-\tau \end{vmatrix}=0 \quad (33)$$

From 33, we obtain

$$-(\lambda+\mu+\omega)(\lambda+\mu)(\lambda+\mu+\tau)\left[-\lambda^2+(\gamma+\mu+k_3+\phi_2)\lambda+\gamma k_2-\gamma k_3-k_3\mu-k_3\phi_2\right]=0 \quad (34)$$

Where

$$k_1=\frac{-\alpha(1-\psi_e)[(1-\upsilon)\wedge+\upsilon\omega\wedge]}{(\mu+\omega)\wedge}$$

$$k_2=\frac{\alpha(1-\psi_e)(1-\theta)[(1-\upsilon)\wedge+\upsilon\omega\wedge]}{(\mu+\omega)\wedge}$$

$$k_3=-\lambda+\frac{\alpha\theta(1-\psi_e)[(1-\upsilon)\wedge+\upsilon\omega\wedge]}{(\mu+\omega)\wedge}+\gamma+\mu+\phi_1$$

From 34

$$\lambda_1=-(\mu+\omega),\ \lambda_2=-\mu,\ \lambda_3=-(\mu+\vartheta),\ \lambda_4=-(\mu+\tau)$$

We obtain $\lambda_5 and \lambda_6$ from the quadratic equation below:

$$\lambda^2-(\gamma+\mu+k_3+\phi_2)\lambda+\gamma k_3+k_3\mu+k_3\phi_2-\gamma k_2=0 \quad (35)$$

from 35 *we obtain* λ_5 *and* λ_6 *as* $\lambda_1,\lambda_2,\lambda_3,\lambda_4$

$$\lambda_5,\lambda_6=\frac{1}{2}\gamma+\frac{1}{2}\mu+\frac{1}{2}k_3+\frac{1}{2}\phi_2\pm\frac{1}{2}\sqrt{\gamma 2+2\gamma\mu+4\gamma k_2-2\gamma k_3+2\gamma\phi_2+\mu^2-2\mu k_3+2\mu\phi_2+{k_3}^2-2k_3\phi_2+{\phi_2}^2}$$

Clearly are all less than zero.

It is not clear whether $\lambda_5 and \lambda_6$ are less or greater than zero. We therefore conclude that, the disease- free equilibrium state is locally asymptotically stable if $\lambda_5 and \lambda_6$

are less than zero and unstable if otherwise.

This implies that the determinant of our variation matrix, is positive if and only if $R_E<1$ .Since, the trace of our matrix $J(E_0)$ is less than zero and its determinant is positive when $R_E<1$ then, model system (1-6) is locally asymptotically stable at disease free equilibrium, E_0.

GLOBAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM E_0

Analyzing the global stability of the disease-free equilibrium point we use [4] approach

We write model system 1-6 in the form

$$\begin{cases} \frac{dz_s}{dt}=x(z_s-z_{DFE,S})+x_1z_i \\ \frac{dz_i}{dt}=x_2z_i \end{cases} \quad (36)$$

Where z_s is the vector representing the non-transmitting compartments and z_i is the vector representing the transmitting components.

The DFE is globally asymptotically stable if A has real negative eigenvalues and x_2 is a Metzler matrix.

From system (1-6) we have

$$z_i=(1,i),\quad z_s=(v,s,t,r)$$

$$z_s-z_{DFEs}=\begin{bmatrix} v & - & \frac{v\wedge}{\mu+\omega} \\ s-1 & + & \frac{v\wedge}{\mu+\omega} \\ & \tau & \\ & \gamma & \end{bmatrix} \quad (37)$$

We check if the non-transmitting compartments have real negative eigenvalues and that x_2 is a Metzler matrix.

From (1-6) equation for non-transmitting compartments are

$$x=\begin{bmatrix} -A & 0 & 0 & 0 \\ \omega & \dfrac{-\alpha(1-\psi_e)I\mu}{\wedge} & -\mu & \tau \\ 0 & 0 & -A_1 & 0 \\ 0 & 0 & \vartheta & A_2 \end{bmatrix} \tag{38}$$

Where $A=-\mu-\omega$

$A_1=-\mu-\vartheta$

$A_2=-\mu-\tau$

$$x_1=\begin{bmatrix} 0 & 0 \\ 0 & \dfrac{-\alpha(1-\psi_e)s\mu}{\wedge} \\ \phi_2 & \phi_1 \\ 0 & 0 \end{bmatrix} \tag{39}$$

$$x_2=\begin{bmatrix} \gamma+\mu+\phi_2 & \dfrac{\alpha(1-\psi_e)(1-\theta)s\mu}{\wedge} \\ \gamma & \dfrac{\alpha\theta(1-\psi_e)s\mu}{\wedge}+\delta+\mu+\phi_1 \end{bmatrix} \tag{40}$$

Our direct computation shows that, the eigenvalues of x are real and negative.

This implies that the system $\frac{dz_s}{dt}=x(z_s-z_{DFEs})+x_1z_i$ is globally asymptotically stable at DFE.

More so, since $0\le i_1<1$ we have, $(1-i_1)>0$ and this implies x_2 a Metzler matrix.

Thus, the DFE is globally asymptotically stable.

Theorem (2): The disease –free equilibrium point isglobally asymptotically stable in Ω if $R_E<1$ and unstable if $R_E>1$.

ENDEMIC EQUILIBRIUM OF THE MODEL (EE)

The endemic equilibrium can be obtained when (V,S,L,I,T,R) $\neq 0$. Let the endemic equilibrium of our model system (1-6) be denoted by $EE^*\left(V^*,S^*,L^*,I^*,T^*,R^*\right)$.We wish to derive the endemic equilibrium for $EE^*\left(V^*,S^*,L^*,I^*,T^*,R^*\right)$.

Let $\lambda=\gamma(\theta I+\alpha)$ be force of infection.

$$S^*=\frac{(1-\vee)\wedge}{\left[\alpha(1-\psi_e)\dfrac{I}{N}\lambda^*+(\omega\vee+\tau R+\mu)\right]} \tag{41}$$

$$L^*=\left[\frac{\alpha(1-\theta)(1-\psi_e)\dfrac{SI}{N}}{\left[(\alpha(1-\psi_e)\lambda^*\dfrac{I}{N}+(\omega\vee+\tau R+\mu)(\gamma+\mu+\phi_2)\right]}\right] \tag{42}$$

$$I^*=\alpha\theta(1-\psi_e)\frac{SI}{N}+\frac{\gamma\alpha(1-\theta)(1-\psi_e)\dfrac{SI}{N}}{\left(\alpha(1-\psi_e)\lambda^*\dfrac{I}{N}+(\omega\vee+\tau R+\mu)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)\right)} \tag{43}$$

$$R^*=\frac{\vartheta T}{(\mu+\tau)} \tag{44}$$

Substituting L^* *and* I^* in the equation for the force of infection: $\lambda=\gamma(\theta I+\alpha)$

$$\lambda=\gamma(\theta\alpha\theta(1-\psi_e)\frac{SI}{N}+\frac{\gamma\alpha(1-\theta)(1-\psi_e)\dfrac{SI}{N}}{(\alpha(1-\psi_e)\lambda^*\dfrac{I}{N}+\omega\vee+\tau R+\mu)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)} \tag{45}$$

$$\frac{\lambda\gamma(\theta\alpha\theta(1-\psi_e)\dfrac{SI}{N}+\gamma\alpha(1-\theta)(1-\psi_e)\dfrac{SI}{N}}{(\alpha(1-\psi_e)\lambda^*\dfrac{I}{N})+(\omega\vee+\tau R+\mu)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)} \tag{46}$$

Where $C=\left(\alpha(1-\psi_e)\lambda^*\frac{I}{N}\right)$

$C_1=(\omega\nu+\tau R+\mu)$

$C_2=(\gamma+\mu+\phi_2)$

$C_3=(\mu+\delta+\phi_1)$

And $K_1=\dfrac{\left(\theta\alpha\theta(1-\psi_e)\frac{SI}{N}+\gamma\alpha(1-\theta)(1-\psi_e)\frac{SI}{N}\right)}{\left(\alpha(1-\psi_e)\lambda^*\frac{I}{N}+(\omega\nu+\tau R+\mu)(\gamma+\mu+\phi_2)(\mu+\delta+\phi_1)\right)}$ (47)

Therefore: $C\lambda+(C_1C+C_2-\gamma C_3)\lambda+(C_2C-C_3C)\lambda=0$

Expressing this as a polynomial

$\lambda(X+Y\lambda+Z)=0$

$where\ X=C_1, Y=(C_1C+C_2)-\gamma C_3\ and\ Z=C_2C-C_3C$

$\lambda=0$ (48)

Which corresponds to the disease free equilibrium early discussed and $\lambda(X+Y\lambda+Z)=0$ which corresponds to the existence of two endemic equilibrium points.

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4.1Simulation and Discussion

Table 3: Values for population-dependent parameters of the model(1-6)

S/NO	Variable/Parameter	Value	Source
1	S	82,104,841	CIA [5]
2	V	37,120,961	CIA [5]
3	L	52,136,956	CIA [5]
4	I	5,792,995	CIA [5]
5	N	177,155,754	CIA [5]
6	T	0.0189 yr^{-1}	CIA [5]
7	Λ	3,348,245	CIA [5]

Table 4:Values for population-independent parameters of the model

S/NO	Parameter	Value	Source
1	α	0.0000621 yr^{-1}	[6]
2	ω	0.067 yr^{-1}	[7]
3	γ	0.5 yr^{-1}	[8]
4	θ	0.1 yr^{-1}	Estimated
5	δ	0.00292 yr^{-1}	[9]
6	τ	1.25 yr^{-1}	Estimated
7	ϕ_1	0.7 yr^{-1}	Estimated
8	ϕ_2	0.8 yr^{-1}	Estimated
9	$\psi[e]$	0-1 yr^{-1}	Estimated

We present a summary of the results of our analytical solution in Figures 1below.

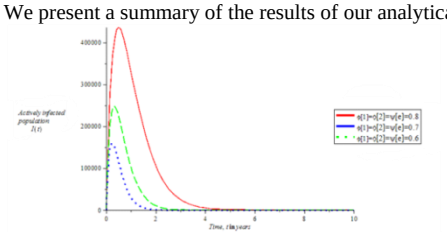

Figure 1: Actively infected population against time.

Figure 1shows the relationship between the actively infected population against time for the cases where the treatment rates and the public health education campaign are varied. The above figure shows a decline in the number of the actively infected individuals. This agrees with reality in the sense that when the treatment rates are effective and high, individuals infected with TB get quick recovery. Similarly, when the public health awareness campaign (advert on radio, TV and print media) on TB is also high, many persons will become more careful in interacting with anyone suspected to have TB infection.

5. Conclusion

In this paper, a mathematical model dynamics for TB incorporating treatment, vaccination and public health education campaign as control measures is presented as a system of ordinary differential equations. The disease free equilibrium is shown to be locally asymptotically stable if λ_5 *and* $\lambda_6 \angle 0$ and unstable if otherwise. The result of our analysis shows that the rate of spread of TB will be less when people infected with TB is reduced. The results obtained from dynamical system analysis and simulations of the models shows that incorporating of treatment and public health education campaign actually reduces the rate of actively infected individuals. The analysis and numerical results also suggest that incorporating treatment, vaccination and public health education campaign simultaneously reduces the rate of actively infected individuals better than when only one is introduced.

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