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Mathematical Model for Mycobacterium Tuberculosis

Jafaar Olasunkanmi LAWAL[1*], Abraham Baba ZHIRI[2], Faruk MURITALA[1], Risqot Garba IBRAHIM[1], Alpha Peter LUKONDE[3]

[1] *Department of Mathematics and Statistics, Kwara State University, Malete, NIGERIA*
[2] *Department of Mathematics, Federal University of Technology, Minna, NIGERIA*
[3] *Department of Mathematics, Karadeniz Technical University, Trabzon, TÜRKİYE*

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ABSTRACT

To demonstrate the dynamics of the Mycobacterium tuberculosis disease population, a mathematical model was presented. The model has five compartments, and the resulting equations were resolved. While multiple cases of illness transmission were simulated using the compartmental model of infectious disease spread for a structured population model, the fundamental reproduction number was found using the next-generation matrix. Based on the results of the simulations, the system's disease-free and endemic equilibrium was created by presenting, analyzing, and graphing the various subpopulations across time. The Homotopy Perturbation Method (HPM) analytical technique was then used to resolve the model.

1. Introduction

Mycobacterium tuberculosis complex organisms are the cause of the airborne infectious disease known as tuberculosis (TB). *Mycobacterium tuberculosis* can cause disease in practically every organ of the body despite being largely a pulmonary pathogen. *Mycobacterium tuberculosis* infection can progress from host containment, in which the bacteria are isolated within granulomas (latent tuberculosis infection), to a contagious state, in which the patient will exhibit symptoms such as coughing up blood, having a fever, sweating excessively at night, and losing weight [1]. Only pulmonary TB that is active can spread. Drug-resistant tuberculosis is a major concern in many contexts, and it continues to be a major source of morbidity and mortality in many low- and middle-income nations [15]. As a lungs illness, tuberculosis is largely transmitted through an active infection of this vital organ. Respiratory droplets that contain the tubercle bacillus are used to spread tuberculosis. These are exhaled by people who have active tuberculosis, and contacts then breathe them in. The physical barriers found in the upper respiratory system will prevent the majority of droplets from entering the body, but those that are smaller than 12 m can get past these barriers and reach the lower respiratory tract and lungs [11]. The fight between the host and infection starts when the germs come into contact with immune system cells. Alveolar macrophages consume inhaled droplets containing minuscule amounts of germs [12]. These macrophages carry the pathogen to draining lymph nodes while harboring it. A little granulomatous lesion forms and houses the germs. Ninety percent of all affected people can attest to this. They won't get sick straight away. However, because the bacteria are still there, there is a chance that a disease could eventually manifest [14]. Illness will immediately follow primary infection in those with weakened immune systems. This is true, for instance, of HIV-positive people, for whom the chance of developing the condition within a year is significantly raised depending on the degree of immunodeficiency. People who successfully contain the infection for years are nonetheless at danger, and an epidemic could happen later when the immune system is compromised. Due to reactivation, an outbreak can happen. Although less probable, disease-causing reinfection is not completely precluded [10].

Understanding the clinical behavior of this pathogen in its sole native host is crucial in order to comprehend the molecular pathogenesis of *Mycobacterium tuberculosis* infection. The settings to which *Mycobacterium tuberculosis* must adapt are exclusively determined by its

[*] *Department of Mathematics and Statistics, Kwara State University, Malete, NIGERIA*
E-mail address: jafaarlawal@gmail.com

natural history within its host because the pathogen has no known reservoir outside of humans. Aerosolized droplets harboring the contagious *Mycobacterium tuberculosis* are virtually completely responsible for the transmission of the disease tuberculosis [13]. These droplets are produced by a person who has Mycobacterium tuberculosis and are inhaled by someone who is not sick. There are four outcomes that could occur upon inhalation of *Mycobacterium tuberculosis*: immediate eradication of the organism. Persistent infection Active disease first appears (primary disease) and reappears years later (reactivation disease). Reactivation illness happens in 510 of people with latent infection who have no underlying medical conditions. Patients with HIV are significantly more likely to have reactivation. The interaction of elements attributed to the organism and the host determines these consequences [2].

2. Material and Method

2.1 Computation of Disease Equilibriums

A five compartment mathematical model was designed for *Mycobacterium tuberculosis* which includes the S-Susceptible, E-Exposed, I-Infected, V-Vaccinated and R-Recovered de- pending on the stage each individual falls on the compartment chain, which is given as follows:

$$\frac{dS}{dt} = \omega + \pi S - (\mu + \lambda I)S \quad (1)$$

$$\frac{dE}{dt} = \lambda SI - (\mu + \beta + \alpha)E \quad (2)$$

$$\frac{dI}{dt} = \beta E - (\mu + \gamma + \delta)I \quad (3)$$

$$\frac{dV}{dt} = \alpha E + \gamma I - (\mu + \Omega)V \quad (4)$$

$$\frac{dR}{dt} = \Omega V - \pi S - \mu R \quad (5)$$

In the formulation of the above mathematical model some important assumptions were properly considered:

1. Recruitment occurs only in the susceptible through birth.
2. Individual can move to expose in an unsafe environment.
3. Exposed individual can be infected through direct or indirect contact.
4. Exposed individual can be vaccinated without being infected.
5. Vaccinated individual can recover after a certain period.
6. Recovered individual can move back to susceptible and start the chain all over if proper action is not taken.
7. Birth rate and death rate are not equal.
8. All parameters are non-negative.
9. Death is liable to the entire compartment.

Equilibrium states of mycobacterium tuberculosis model: At Disease Free Equilibrium (DFE):

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

$$\omega + \pi S - (\mu + \lambda I)S = 0 \quad (6)$$

$$\lambda SI - (\mu + \beta + \alpha)E = 0 \quad (7)$$

$$\beta E - (\mu + \gamma + \delta)I = 0 \quad (8)$$

$$\Omega V - \pi S - \mu R = 0 \quad (9)$$

$$\alpha E + \gamma I - (\mu + \Omega)V = 0 \quad (10)$$

Disease free equilibrium [DFE] state is a state with the absence of disease such that I = 0 then solving equations [6] - [10] the disease-free equilibrium is given as:

$$\{S_0, E_0, I_0, R_0, V_0\} = \left\{\frac{\omega}{\{\mu - \pi\}}, \mathbf{0}, \mathbf{0}, \mathbf{0}, \mathbf{0}, \right\} \quad (11)$$

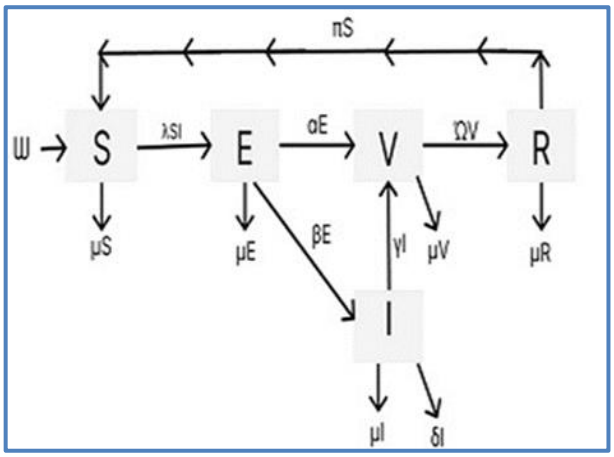

Diagram 1. Schematic diagram of the model

Table 1. Parameter descriptions/interpretation.

Model Parameters	Description/Interpretation
ω	Recruitment into the uninfected Class S
λ	Susceptible Movement into the exposed Class E
α	Exposed Movement into the Vaccinated Class V
Ω	Recovered Movement into the Recovered Class R
γ	Infected Movement into the Vaccinated Class V
β	Contact rate of exposed moving to Infected Class I
μ	Natural Death
δ	Death due to Infection from Class I
π	May return to the Susceptible Class S

Endemic Equilibrium State: This is an equilibrium state where infection is totally assumed to be present such that $\boldsymbol{I \neq 0}$ therefore it is given as:

$$\boldsymbol{S = \frac{\omega}{\mu + \lambda I - \pi}} \quad (12)$$

$$\boldsymbol{E = \frac{\lambda S I}{\mu + \beta + \alpha}} \quad (13)$$

$$\boldsymbol{I = \frac{\beta E}{\mu + e + \delta}} \quad (14)$$

$$\boldsymbol{V = \frac{\alpha \lambda S I(\mu + e + \delta) + e\beta E(\mu + \beta + \alpha)}{(\mu + e + \delta)(\mu + \beta + \alpha)(\mu + \Omega)}} \quad (15)$$

$$\boldsymbol{S = \frac{\Omega V - \pi S}{\mu}} \quad (16)$$

Using equations (1) – (5) to show the Existence and Uniqueness of solution of the model under consideration, we proceed as follows :

$$\frac{dS}{dt} = \omega + \pi S - (\mu + \lambda I)S \quad (17)$$

$$\frac{dS}{dt} \geq -(\mu + \lambda I)S \quad (18)$$

Such that, $S_0 > 0$

$$S \geq e^{\{\mu + \lambda I - \pi\}t} \bullet S_0 \quad (19)$$

$$\frac{dE}{dt} = \lambda S I - (\mu + \beta + \alpha)E \quad (20)$$

$$\frac{dE}{dt} \geq -(\mu + \beta + \alpha)E \quad (21)$$

Such that, $E_0 > 0$

$$E \geq e^{\{\mu + \beta + \alpha\}t} \bullet E_0 \quad (22)$$

$$\frac{dI}{dt} = \beta E - (\mu + e + \delta)I \quad (23)$$

$$\frac{dI}{dt} \geq -(\mu + e + \delta)I \quad (24)$$

Such that, $I_0 > 0$

$$I \geq e^{\{\mu + e + \delta\}t} \bullet I_0 \quad (25)$$

$$\frac{dV}{dt} = \alpha E + eI - (\mu + \Omega)V \quad (26)$$

$$\frac{dV}{dt} \geq -(\mu + \Omega)V \quad (27)$$

Such that, $V_0 > 0$

$$V \geq e^{-\{\mu + \Omega\}t} \bullet V_0 \quad (28)$$

$$\frac{dR}{dt} = \Omega V - \pi S - \mu R \quad (29)$$

$$\frac{dR}{dt} \geq -\mu R \quad (30)$$

Such that, $R_0 > 0$

$$R \geq e^{-\mu t} \bullet R_0 \quad (31)$$

For uniqueness:

$$\boldsymbol{F_1 = \omega + \pi S - (\mu + \lambda I)S} \quad (32)$$

$$\boldsymbol{F_2 = \lambda S I - (\mu + \beta + \alpha)E} \quad (33)$$

$$F_3 = \beta E - (\mu + e + \delta) \quad (34)$$

$$\boldsymbol{F_4 = \Omega V - \pi S - \mu R} \quad (35)$$

$$\boldsymbol{F_5 = \alpha E + eI - (\mu + \Omega)V} \quad (36)$$

Table 2. Parameter values

Parameters	Values
$\boldsymbol{\omega}$	8000
$\boldsymbol{\lambda}$	0.00008
$\boldsymbol{\alpha}$	0.50
$\boldsymbol{\mu}$	0.01
$\boldsymbol{\Omega}$	0.35
$\boldsymbol{\gamma}$	0.070
$\boldsymbol{\delta}$	0.30
$\boldsymbol{\beta}$	0.1
$\boldsymbol{\pi}$	0.010
$\boldsymbol{S_0}$	11000
$\boldsymbol{E_0}$	3500
$\boldsymbol{I_0}$	500
$\boldsymbol{V_0}$	375
$\boldsymbol{R_o}$	300

Then differentiating (32) - (36) we obtained the following equations:

$|\frac{\partial F1}{\partial S}| = |\pi - (\mu + \lambda I)| < \infty, |\frac{\partial F1}{\partial E}| = 0 < \infty, |\frac{\partial F1}{\partial I}| = |\lambda S| < \infty, |\frac{\partial F1}{\partial R}| = 0 < \infty, |\frac{\partial F1}{\partial V}| = 0 < \infty$

$|\frac{\partial F2}{\partial S}| = |\lambda I| < \infty, |\frac{\partial F2}{\partial E}| = |-(\mu + \beta + \alpha)| < \infty, |\frac{\partial F2}{\partial I}| = |\lambda S| < \infty, |\frac{\partial F2}{\partial R}| = 0 < \infty, |\frac{\partial F2}{\partial V}| = 0 < \infty.$

$|\frac{\partial F3}{\partial S}| 0 < \infty, |\frac{\partial F3}{\partial E}| = |\beta| < \infty, |\frac{\partial F3}{\partial I}| = |-(\mu + e + \delta)| < \infty, |\frac{\partial F3}{\partial R}| = 0 < \infty, |\frac{\partial F3}{\partial V}| = 0 < \infty.$

$|\frac{\partial F4}{\partial S}| |\pi| < \infty, |\frac{\partial F4}{\partial E}| = 0 < \infty, |\frac{\partial F4}{\partial I}| = 0 < \infty, |\frac{\partial F4}{\partial R}| = |-\mu| < \infty, |\frac{\partial F4}{\partial V}| = |\Omega| < \infty.$

$|\frac{\partial F4}{\partial S}| 0 < \infty, |\frac{\partial F4}{\partial E}| = |\alpha| < \infty, |\frac{\partial F4}{\partial I}| = |e| < \infty, |\frac{\partial F4}{\partial R}| = 0 < \infty, |\frac{\partial F4}{\partial V}| = |-\{\mu + \Omega\}| < \infty$

It has been clearly shown that all partial derivatives of the whole system of equations [1] -[5] exist and it is also non-negative therefore it is finite, bounded and has a unique solution.

2.2 Computation of Reproduction Number

We then calculate the reproduction number R_0: From equations (2) and (3)

$$\frac{dE}{dt} = \lambda SI - (\mu + \beta + \alpha)E$$

$$\frac{dI}{dt} = \beta E - (\mu + e + \delta)\mathrm{I}$$

Where,

$$R_0 = \rho\,(FV^{-1}) \quad (37)$$

$$\mathrm{F} = \begin{pmatrix} \mathbf{0} & \boldsymbol{\lambda S_0} \\ \mathbf{0} & \mathbf{0} \end{pmatrix} \quad (38)$$

and

$$\mathrm{V_i} = \boldsymbol{V_i^-} - \boldsymbol{V_i^+} \quad (39)$$

$$\mathbf{V_i} = \begin{pmatrix} (\mu + \boldsymbol{\beta} + \boldsymbol{\alpha}) & \mathbf{0} \\ -\boldsymbol{\beta} & (\mu + \boldsymbol{e} + \boldsymbol{\delta}) \end{pmatrix} \quad (40)$$

Where,

$\mathrm{J_1} = (\mu + \boldsymbol{\beta} + \boldsymbol{\alpha})$

$\mathrm{J_2} = (\mu + \boldsymbol{e} + \boldsymbol{\delta})$

$$\mathrm{V^{-1}} = \begin{pmatrix} \frac{\mathbf{1}}{J_1} & \frac{\beta}{J_1 J_2} \\ \mathbf{0} & \frac{\mathbf{1}}{J_2} \end{pmatrix} \quad (41)$$

$$\mathrm{F} = \begin{pmatrix} \mathbf{0} & \frac{\boldsymbol{\lambda S_0}}{J_2} \\ \mathbf{0} & \mathbf{0} \end{pmatrix} \quad \frac{\boldsymbol{\lambda\omega}}{\{\mu - \boldsymbol{\pi}\}\{\mu + \boldsymbol{e} + \boldsymbol{\delta}\}} \quad (42)$$

$$\boldsymbol{R_0} = \frac{\boldsymbol{\lambda\omega}}{\{\mu - \boldsymbol{\pi}\}\{\mu + \boldsymbol{e} + \boldsymbol{\delta}\}} \quad (43)$$

2.3 Analytical Solution of the Governing Model Equation Via Homotopy Perturbation Method (HPM)

The Homotopy Perturbation Method (HPM) was first discovered by Ji-Haun (2000). The Homo- topy Perturbation Method (HPM), which provides analytical approximate solution, is applied to various linear and non-linear equations. The homotopy perturbation method (HPM) is a series expansion method used in the solution of nonlinear partial differential equations.

Solution of the Model Equations

Using the following initial conditions (44) on the governing model equations (1 –5)

$$S(0) = S_0, E(0) = E_0, I(0) = I_0, V(0) = V_0, R(0) = R_0 \quad (44)$$

We then let,

$$\begin{aligned} S &= a_0 + pa_1 + p^2a_2 + \\ E &= b_0 + pb_1 + p^2b_2 + \\ I &= c_0 + pc_1 + p^2c_2 + \\ V &= d_0 + pd_1 + p^2d_2 + \\ R &= e_0 + pe_1 + p^2e_2 + \end{aligned} \quad (45)$$

Applying HPM on (1) - (5) using (48) we obtain the following equations;

$$\left.\begin{aligned} \boldsymbol{P^0}&: \boldsymbol{a_0' = 0} \\ \boldsymbol{P'}&: \boldsymbol{a_1' + \lambda a_0 c_0 + \mu a_0 - \pi a_0 - \mathbb{W} = 0} \\ \boldsymbol{P^2}&: \boldsymbol{a_2' + \lambda a_1 c_0 + \lambda a_0 c_1 + \mu a_1 - \pi a_1 = 0} \end{aligned}\right\} \quad (46)$$

$$\left.\begin{aligned} \boldsymbol{P^0}&: \boldsymbol{b_0' = 0} \\ \boldsymbol{P'}&: \boldsymbol{b_1' + (\mu + \beta + \alpha)a_0 - \lambda a_0 c_0 = 0} \\ \boldsymbol{P^2}&: \boldsymbol{b_2' + (\mu + \beta + \alpha)b_1 - \lambda a_0 c_1 + \lambda a_1 c_0 = 0} \end{aligned}\right\} \quad (47)$$

$$\left.\begin{aligned} \boldsymbol{P^0}&: \boldsymbol{c_0' = 0} \\ \boldsymbol{P'}&: \boldsymbol{c_1' + (\mu + \gamma + \delta)c_0 - \beta b_0 = 0} \\ \boldsymbol{P^2}&: \boldsymbol{c_2' + (\mu + \gamma + \delta)c_1 - \beta b_1 = 0} \end{aligned}\right\} \quad (48)$$

$$\left.\begin{array}{l} \boldsymbol{P^0}: \boldsymbol{d_0' = 0} \\ \boldsymbol{P'}: \boldsymbol{d_1'} + (\mu + \Omega)\boldsymbol{d_0} - \boldsymbol{\alpha b_0} - \boldsymbol{\gamma c_0} = \boldsymbol{0} \\ \boldsymbol{P^2}: \boldsymbol{d_2'} + (\mu + \Omega)\boldsymbol{d_1} - \boldsymbol{\alpha b_1} - \boldsymbol{\gamma c_1} = \boldsymbol{0} \end{array}\right\} \quad (49)$$

$$\left.\begin{array}{l} \boldsymbol{P^0}: \boldsymbol{e_0' = 0} \\ \boldsymbol{P'}: \boldsymbol{e_1'} + \boldsymbol{\pi a_0} - \boldsymbol{\mu e_0} - \Omega \boldsymbol{d_0} = \boldsymbol{0} \\ \boldsymbol{P^2}: \boldsymbol{e_2'} + \boldsymbol{\pi a_0} - \boldsymbol{\mu e_1} - \Omega \boldsymbol{d_1} = \boldsymbol{0} \end{array}\right\} \quad (50)$$

Solving (46) - (50) by direct integration method for P^0 using (44) we obtain the following

$$\left.\begin{array}{l} \boldsymbol{a_0 = S_0} \\ \boldsymbol{b_0 = E_0} \\ \boldsymbol{c_0 = I_0} \\ \boldsymbol{d_0 = V_0} \\ \boldsymbol{e_0 = R_0} \end{array}\right\} \quad (51)$$

Where S_0, E_0, I_0, V_0 and R_0 are all constants initial conditions.

Substituting (51) into (46) - (50) and solve by direct integration method for P^1 , we obtain the following equations.

$$\left.\begin{array}{l} a_1 = (\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0)t \\ b_1 = (\lambda S_0 I_0 - (\mu + \beta + \alpha)E_0 \\ c_1 = (\beta E_0 - (\mu + \delta + \gamma)I_0)\mathrm{t} \\ d_1 = (\gamma I_0 + \alpha E_0 - (\mu + \Omega)V_0)t \\ e_1 = (\Omega V_0 - \mu R_0 - \pi S_0)t \end{array}\right\} \quad (52)$$

Similarly, Substituting (51) and (52) into (46) - (50) and solve by direct integration for P^2, we obtain the following equations.

$$a_2 = \begin{bmatrix} \pi(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0) - \mu(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0) \\ (\lambda(\beta E_0 - (\mu + \beta + \alpha)I_0)S_0 - \lambda I_0(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0) \end{bmatrix} \frac{t^2}{2} \quad (53)$$

$$b_2 = \begin{bmatrix} \pi(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0)I_0 + \lambda S_0(\beta E_0 - (\mu + \delta + \gamma)I_0 \\ -(\mu + \beta + \alpha)(\lambda S_0 I_0 - (\mu + \beta + \delta)E_0 \end{bmatrix} \frac{t^2}{2} \quad (54)$$

$$c_2 = (\beta(\lambda S_0 I_0 - (\mu + \beta + \alpha)E_0) - (\mu + \delta + \gamma)(\beta E_0 - (\mu + \delta + \gamma)I_0)) \frac{t^2}{2} \quad (55)$$

$$d_2 = \begin{bmatrix} \gamma(\beta E_o - (\mu + \delta + \gamma)I_o + \alpha(\lambda S_o I_o - (\mu + \beta + \alpha)E_o \\ -(\mu + \Omega)(\gamma I_o + \alpha E_o - (\mu + \Omega)V_0 \end{bmatrix} \frac{t^2}{2} \quad (56)$$

$$e_2 = \begin{bmatrix} \Omega(\gamma I_0 + \alpha E_0 - (\mu + \Omega)V_0 - \mu(\Omega V_0 - \mu R_0 - \pi S_0) \\ -\pi(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0) \end{bmatrix} \frac{t^2}{2} \quad (57)$$

But, from (45) we have,

$$S = a_0 + pa_1 + p^2 a_2 + \cdots \quad \textbf{(58)}$$

$$E = b_0 + pb_1 + p^2 b_2 + \cdots$$

$$I = c_0 + pc_1 + p^2 c_2 + \cdots$$

$$V = d_0 + pd_1 + p^2 d_2 + \cdot\cdot$$

$$R = e_0 + pe_1 + p^2 e_2 + \cdots$$

then, we let,

$$\left.\begin{array}{l} \lim_{p\to 1} S(t) = \lim_{p\to 1}(a_0 + pa_1 + p^2 a_2 + \cdots) = a_0 + a_1 + a_2 + \cdots \\ \lim_{p\to 1} E(t) = \lim_{p\to 1}(b_0 + pb_1 + p^2 b_2 + \cdots) = b_0 + b_1 + b_2 + \cdots \\ \lim_{p\to 1} I(t) = \lim_{p\to 1}(c_0 + pc_1 + p^2 c_2 + \cdots) = c_0 + b_1 + c_2 + \cdots \\ \lim_{p\to 1} V(t) = \lim_{p\to 1}(d_0 + pd_1 + p^2 d_2 + \cdots) = d_0 + d_1 + d_2 + \cdots \\ \lim_{p\to 1} R(t) = \lim_{p\to 1}(e_0 + pe_1 + p^2 e_2 + \cdots) = e_0 + e_1 + e_2 + \cdots \end{array}\right\} \quad (59)$$

This implies that,

$$S(t)= \lim_{p\to 1} S(t) = \lim_{p\to 1}(a_0 + pa_1 + p^2a_2 + \cdots) = a_0 + a_1 + a_2 + \quad (60)$$

$$S(t) = S_0 + (\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0)t + \pi\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0) - \mu(\omega + \pi S_0 \; \mu S_0 \lambda S_0 I_0) - \lambda(\beta E_0 - (\mu + \delta + \gamma)I_0)S_0 - \lambda I_0(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0)\Big]\frac{t^2}{2}$$

$$E(t)= lim_{p\to 1} E(t) = lim_{p\to 1}(b_0 + pb_1 + p^2b_2 + \cdots) = b_0 + b_1 + b_2 + \cdot\cdot$$

$$E_0 + (\lambda S_0 I_0 - (\mu + \beta + \alpha)E_0)t) \; \lambda\,(\omega + \pi S_0 - \mu S_0 - \lambda S_0 I_0)I_0 + \lambda S_0(\beta E_0 - (\mu + \delta + \gamma)I_0 - (\mu + \beta + \alpha)\lambda S_0 I_0 - (\mu + \beta + \delta)E_0\Big]\frac{t^2}{2} \quad (61)$$

$$I(t) = \lim_{p\to 1} I(t) = \lim_{p\to 1}(c_0 + pc_1 + p^2c_2 + \cdots) = c_0 + b_1 + c_2 +$$

$$I(t) = I_0 + (\beta E_0 - (\mu + \omega + \lambda)I_0)t)\Big[\beta(\lambda S_0 I_0 - (\mu + \beta + \alpha)E_0 - (\mu + \delta + \gamma)\beta E_0 - (\mu + \delta + \gamma)I_0\Big] \quad (62)$$

$$V(t) = lim_{p\to 1} V(t) = lim_{p\to 1}(d_0 + pd_1 + p^2d_2 + \cdots) = d_0 + b_1 + d_2 +$$

$$V(t) = V_0 + (\gamma I_0 + \alpha E_0 - (\mu + \Omega)V_0)t + \gamma(\beta E_0 - (\mu + \delta + \gamma)I_0 + \alpha(\lambda S_0 I_0 - (\mu + \beta + \alpha)E_0 - (\mu + \Omega)(\gamma I_0 + \alpha E_0 - (\mu + \Omega)V_0\Big]\frac{t2}{2} \quad (63)$$

$$R(t) = lim_{p\to 1} R(t) = lim_{p\to 1}(e_0 + pe_1 + p^2e_2 + \cdots) = e_0 + e_1 + e_2 + \cdots$$

$$R(t) = R_0 + (\Omega V_0 - \mu R_0 - \pi S_0)t \quad \Omega(\gamma I_0 + \alpha E_0 - (\mu + \Omega)V_0) - \mu(\Omega V_0 - \mu R_0 - \pi S_0) \; \pi(\omega + \pi S - \mu S - \lambda S\, I)\Big]\frac{t^2}{2} \quad (64)$$

3. Results and Discussion

Figure 1 depicts the graph of Infected class against time for different values of infectious rate β and it was observed that the, Infected class gets more populated as infectious rate increases.

Figure 2 shows the graph of Infected class against time for different values of natural death rate μ and it was observed that the, population of the infected class reduces as natural death increases.

Figure 3 shows the graph of vaccinated class against time for different values of natural death rate μ and it was observed that the, vaccinated class reduces in population as natural death rate increases.

Figure 4 depicts the graph of vaccinated class against time for different values of recovery rate Ω and it was observed that the, vaccinated class reduces in population as recovery rate increases.

Figure 5 displays the graph of recovered class against time for different values of recovery rate Ω and it was observed that the, recovered class increased in population as recovery rate increases.

Figure 6 shows the graph of recovered class against time for different values of transmission rate back to susceptible class π and it was observed that the, population of recovered class reduces as transmission rate back to susceptible class increases.

Figure 7 depicts the graph of exposed class against time for different values of contact rate λ and it was observed that the, exposed class increased in population as contact rate increases.

Figure 8 displays the graph of susceptible class against time for different values of natural death rate μ and it was observed that the, susceptible class decreased in population as natural death rate increases.

Figure 9 shows the graph of susceptible class against time for different values of contact rate λ and it was observed that the, susceptible class decreased in population as contact rate increases.

Figure 10 displays the graph of susceptible class against time for different values of recruitment rate ω and it was observed that the, susceptible class increased in population as recruitment rate increases.

Even if we have a high rate of recovery, Figure 6 shows that the population will still be susceptible to Mycobacterium tuberculosis. It confirms that people are not immune to the disease because they can still return and become vulnerable to it through the transmission parameter, which lowers the population after recovery.

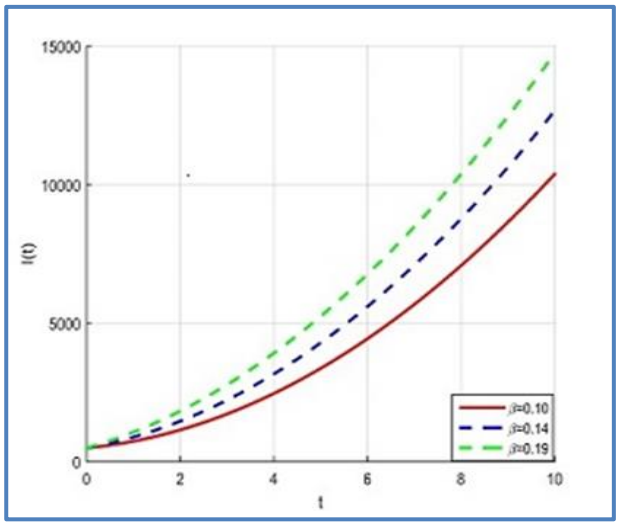

Fig 1. Graph of Infected class against time for different values of β.

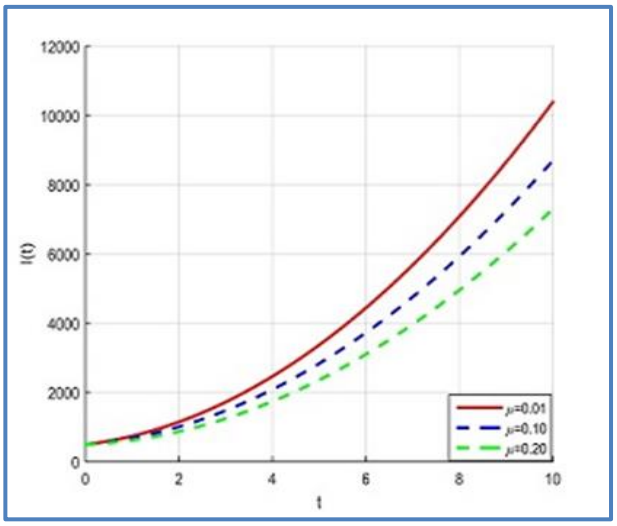

Fig 2. Graph of Infected class against time for different values of μ.

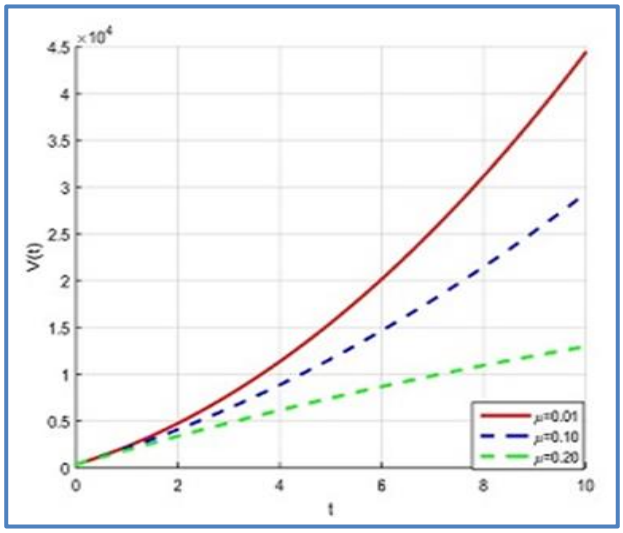

Fig 3. Graph of Vaccinated class against time for different values of μ.

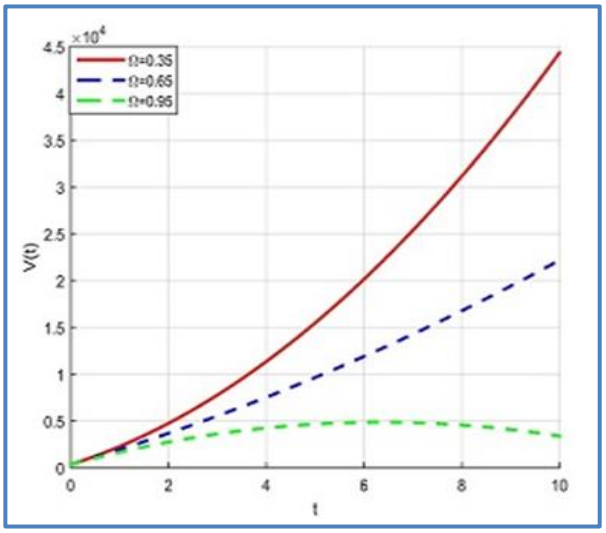

Fig 4. Graph of Vaccinated class against time for different values of Ω.

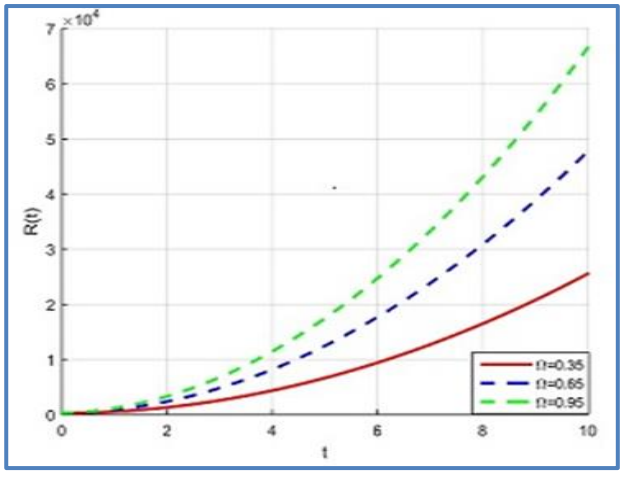

Fig 5. Graph of Recovered class against time for different values of Ω.

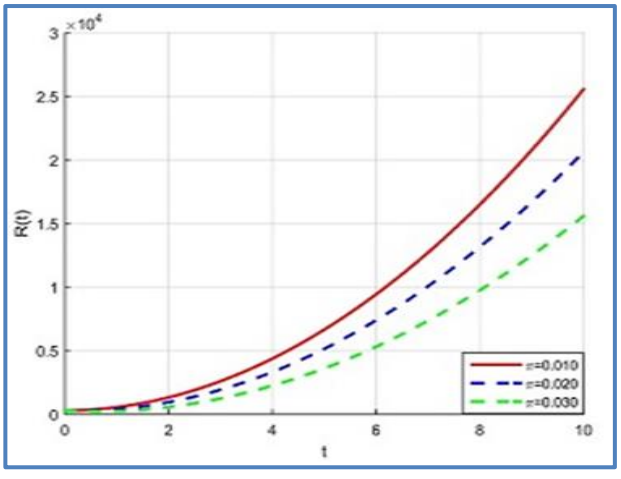

Fig 6. Graph of Recovered class against time for different values of π.

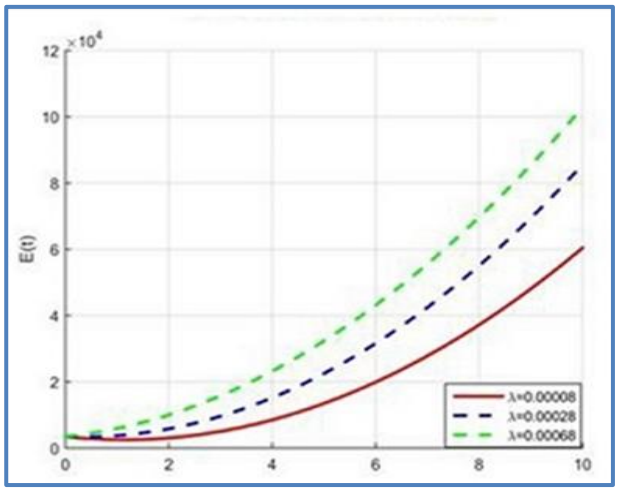

Fig 7. Graph of Exposed class against time for different values of λ.

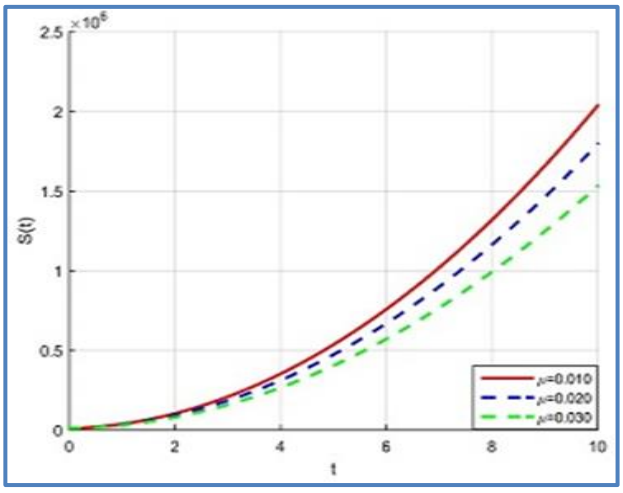

Fig 8. Graph of Susceptible class against time for different values of μ.

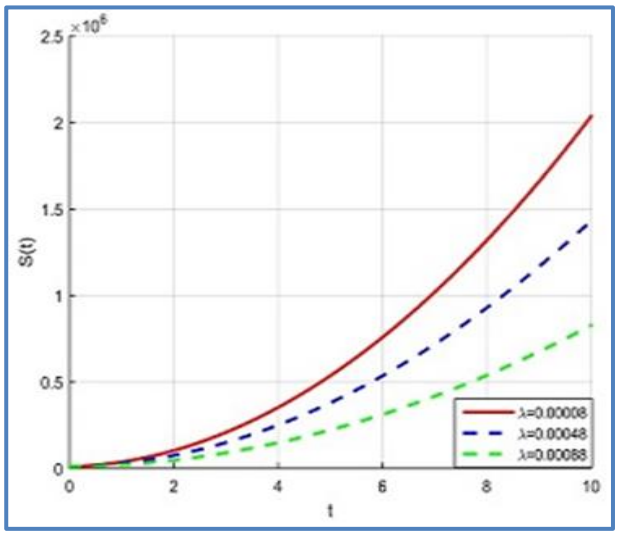

Fig 9. Graph of Susceptible class against time for different values of λ

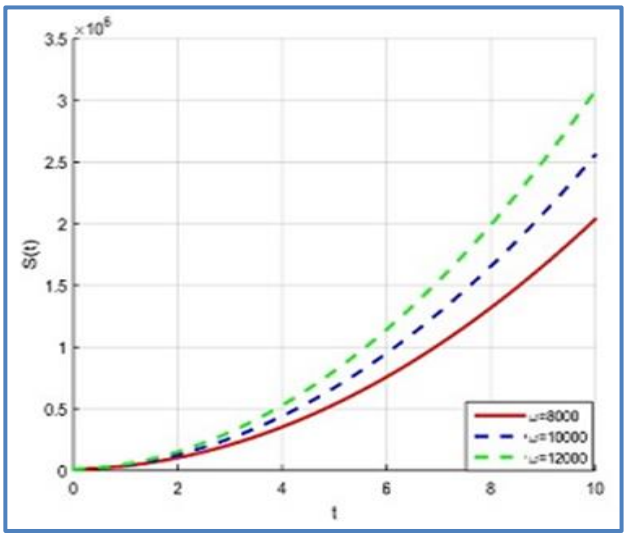

Fig 10. Graph of Susceptible class against time for different values of ω.

According to Figure 1, there will be an exponential growth in the number of infected people if more people are exposed to the disease through the infectious rate and become infectious, which will cause the disease to spread quickly throughout the population. Figure 4 demonstrates how a sharp decline in the vaccina- tion class resulted from more people recovering from the illness. Figure 7 also shows that the disease is spread throughout the community due to an increase in contact rates, which lowers the population that is vulnerable in figure 9. But in general from Figure 5, we saw that, more persons are overcoming the disease, as evidenced by the Mycobacterium tuberculosis recovery count.

4. Conclusion

Mathematical model with five compartments have shown and properly analyze how mycobac- terium tuberculosis can spread across human being. The SEIR model was extended to SEIVR by assuming a vaccinated class (V-Class) which clearly show in the result that if individuals in Exposed class (E-Class) skips the V-Class then the individual goes to the infected class (I- Class) directly because of how contagious mycobaterium tuberculosis is. The Mycobacterium tuberculosis vaccination is long term process although it effectiveness is seen through the rate at which Ω moves from V-Class to R-Class. So an alternative solution was created using

the model by reducing the contact rate (β) which flows between the E-Class and I-Class in other to lower the rate of I-Class and Death due to disease Infection as shown in the figures 1 -11. Consequently, when the Recovery Rate is high, the Infected Population experiences a significant decrease, while the Recovered Population exhibits exponential growth. As a result, the disease diminishes, and the Susceptible Population grows. In conclusion, it can be inferred that the disease has a propensity to fade away over time.

Declaration

Author Contribution: Conceive- J.O.L.; A.B.Z.; R.G.I.; Design- J.O.L., A.B.Z.; Supervision- A.B.Z; F.M.; R.G.I.; A.P.L.; Experimental Performance, Data Collection and/or Processing- J.O.L.; A.B.Z.; F.M; A.P.L.; Analysis and/or Interpretation- A.B.Z.; A.P.L; Literature Review- J.O.L.; A.B.Z.; F.M.; R.G.I; Writers- J.O.L.; A.B.Z.; R.G.I.;Critical Reviews– A.B.Z; F.M.; A.P.L.

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Orcid-ID

Jafaar Olasunkanmi Lawal https://orcid.org/0000-0001-6127-8274
Abraham Baba Zhiri https://orcid.org/0000-0003-2213-6796
Faruk Muritala https://orcid.org/0000-0002-5857-9874
Risqot Garba Ibrahim https://orcid.org/0000-0002-0458-1653
Alpha Peter Lukonde https://orcid.org/0000-0001-6813-4505

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