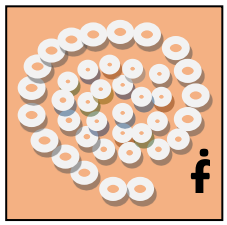

International Journal of Mathematical Analysis and Modelling

(Formerly Journal of the Nigerian Society for Mathematical Biology)

Volume 8 Issue 2, 2025

ISSN (Print): 2682 - 5694
ISSN (Online): 2682 – 5708
www.tnsmb.org

Mathematical model for dynamics of Tuberculosis epidemiology with optimal control strategies

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Abstract

Tuberculosis (TB) is caused by bacteria (Mycobacterium tuberculosis) and it most often affects the lungs. TB is spread through the air when people with lung TB cough, sneeze or spit. A person needs to inhale only a few germs to become infected. To curtail the disease a mathematical model strictly on the Susceptible - Vaccinated - Latent - Active - Chronic - Treatment-Recovery (SVLACTR) compartments for tuberculosis is formulated. We assume the recruitment factor to be Vertical. Already existing tuberculosis data was used in verifying the model to comprehend the transmission of TB dynamics as it will help in warding off, regulating, eliminating the disease, and enhance the well being of lives. We show that the Global Dynamics are completely figured out by basic infection rate R_0^{TB}, that if $R_0^{TB} < 1$ the disease free equilibrium is locally and globally stable. But if $R_0^{TB} > 1$, and endemic equilibrium exist and is uncertain. Sensitivity analysis was performed on the model parameters in R_0^{TB} to determine the effect of each of the parameter value on the TB propagation. Four control strategies, vaccination, reduce contact rate, treatment and sensitization are introduced. By combining these strategies, we can effectively control the spread of TB, improve lives, and ultimately work towards eradicating the disease from the population. This study contributes to the existing literature by highlighting the importance of a multi-faceted approach to TB control and eradication. The model system was solved using the classical Runge-Kutta of order four, forward in 60 years and the algorithm were implemented using MATLAB and MAPLE

Keywords and phrases: tuberculosis; mycobacterium tuberculosis; mathematical model; control strategies

1 Introduction

Tuberculosis (TB) is one of the major public health threats, competing with the human immunodeficiency virus (HIV) as the cause of death due to infectious diseases worldwide. Although a declining trend in TB incidence, prevalence and mortality has been observed over the last decade, elimination of the disease at global level is still out of reach, and massive resource investment is still required ([1]), we shall consider some research and build on it to have a way to curtail the spread of TB.

According to World Health Organization [2] TB is an infection caused by Mycobacterium Tuberculosis. Practically each and every part of human body with some exception can be affected with TB. Lungs, skin, bones, brain, eyes, intestine, liver, kidneys, spine can all be affected with TB infection. As per organ involvement clinical presentation will vary. Lung TB will have cough fever weight loss, bone TB will present with pain, intestinal TB will have abdominal pain, weight loss, eye TB will present with redness of eyes.

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Common cause for TB development is malnutrition and immunological compromises. Most of us harbor dormant TB germs in our body which gets activated whenever we have immunological compromises. Such immunological compromises can happen if we don't eat on time or don't sleep on time, it can also occur if we have long fasting gaps, if we work at odd hours or we are under stress. Tuberculosis spreads easily where people gather in crowds or where people live in crowded conditions. People with HIV/AIDS and other people with weakened immune systems have a higher risk of catching tuberculosis than people with typical immune systems. Drugs called antibiotics can treat tuberculosis, However, some forms of the bacteria do not respond well to treatments.

According to Center for Disease Control (CDC) and Prevention says Tuberculosis (TB) is the ninth leading cause of death worldwide and the first leading cause from a single infectious agent. TB is also the leading killer of HIV-positive people. In 2016, 10.4 million people fell ill with TB. Quarter of those people were from Africa (2.5 million people). In the same year, 1.7 million died from TB globally with 417,000 deaths (over 25 %) from the African region. Between 2000 and 2014, 10 million lives were saved in the African region through TB diagnosis and treatment. Ending the TB epidemic by 2030 is among the health targets of the Sustainable Development Goals (SDGs). The 2013 Abuja Declaration also sets the targets of ending TB in Africa by 2030 [3]. TB is an airborne Disease. It spreads between human through inhaling the air infected with TB germs through cough, sneeze or spit from an infected person. Common symptoms of active lung TB are cough with sputum and blood at times, chest pains, weakness, weight loss, fever and night sweats.

TB has effective treatment and can be cured. Majority of the cases can be cured when treatment is done properly. It is treated with a standard 6 months course of four antimicrobial drugs under the supervision of a health worker or a trained person. Multidrug-resistant tuberculosis (MDR-TB) is a form of TB caused by bacteria that does not respond to isoniazid and rifampicin, the two most powerful, first-line anti-TB drugs. MDR-TB is treatable and curable by using second-line drugs. However, second-line treatment options are limited and require extensive chemotherapy (up to 2 years of treatment) with medicines that are expensive and toxic. Extensively drug-resistant TB (XDR-TB) is a more serious form of MDR-TB caused by bacteria that do not respond to the most effective second-line anti-TB drugs, often leaving patients without any further treatment options. In August 14, 2019, the FDA approved a new drug for extensively drug – resistant TB. There is a slightly effective vaccine for TB. It provides mild protection against the infection transmission. Stopping the transmission between adults is the first effective step of prevention. This is done by identifying patients with active TB to cure them. Another aspect is identifying people with latent TB and prevent the development of the active infectious TB. Tuberculosis infection control through preventing the transmission in different settings such as hospitals is crucial step for prevention. The pasteurization of milk also helps to prevent humans from getting bovine TB.

Better understanding of the prevalence of drug resistance against tuberculosis is one of the key elements in the control of TB. Drug resistance, in combination with other factors, results in increased morbidity and mortality due to tuberculosis. Drug-resistant strains of TB is rapidly emerging worldwide [4]. The WHO reported alarming rise of not only multidrug-resistant (MDR) TB but also of extreme drug-resistant TB (XDR TB) globally. Both treatment and management of such cases are well beyond the capacity of any developing country. Globally, there were about 0.5 million cases of MDR TB [5]. The death rate in MDR cases is high (50–60%) and is often associated with a short span of disease (4–16 weeks) [6]. Several factors have been identified for the development of MDR cases. These include non-adherence to therapy, lack of direct observed treatment, limited or interrupted drug supplies, poor quality of drugs, widespread availability of anti-TB drugs without prescription, poor medical management, and poorly-managed national control programmes [7].

The diagnosis of TB among children is difficult. Moreover, young children cannot produce sputum. Estimates indicate that children constitute about 10% of all new cases in high-burden areas [8]. Clinical signs and symptoms and scoring system have been used for the diagnosis of TB among children [9]. Various diagnostic techniques have been used for improving the diagnosis among children. These include culture, serodiagnosis, and nucleic acid amplification [10]. In certain countries, the Bacille Calmette-

Guérin (BCG) vaccine is given to babies or small children to prevent TB. The vaccine prevents TB outside of the lungs but not in the lungs.

2 Methodology/Model formulation

2.1 Description of the model

We propose Seven compartmental **SVLACTR** model. Also, we considered the diminishing/ reducing effect of TB vaccine as well as four interventions (Awareness, Vaccination, treatment and Reduction of contact rate) for the optimal control strategy. These will help to have a better understanding of the epidemiology of TB. In using the **SVLACTR** model we divide the host population into seven compartments of individuals: The susceptible compartment *(S)*, these are individuals whom are not yet infected but are at risk of becoming infected of TB, the vaccinated compartment *(V)* is referred to the group of individuals who have been vaccinated by TB. The latent class *(L)*, these are group of individuals who have been infected by TB but are not showing symptoms. The active class *(A)*, is the TB class of individuals who are infected and at the same time showing symptoms. The Chronic compartment *(C)*, is the group of individuals who have been infected by TB for an extended period of time and may continue to experience symptoms even after the acute phase has passed. The treatment compartment *(T)*, is the class of population who have been treated and are free from TB. The recovery compartment The (**R**) compartment comprises of individuals that have recovered from the disease as a result of anti-viral treatment, spontaneous clearance and vaccinated individuals. Thus *N(t)* the total population, is the sum of the entire compartments in the model written as *N(t) = S(t) + V(t) + L(t) + A(t) + C(t) + T(t) + R(t).* The population will grow through newborns at a rate θ, proportion of newborns who are not immunized at a rate λ. The parameter ν shows the proportion of children who are non-immunized born to infected mothers. Following a effective public awareness campaign, we assume that newborns will receive successful immunization at a rate of $\theta\lambda$ or unsuccessfully at rate $\theta(1-\lambda)$, the population $\theta\lambda\nu C$ is assumed to enter into the chronic compartment and the rest, $\theta\lambda(1-\nu C)$ Stays in the group of individuals who are prone to infection where ξ and ω are the disease transmission rates relative to susceptible individuals in S(t) and L(t) compartments respectively. β is the transfer rate to the active (infection) compartment while Ω and Γ are rate of infectious individuals moving to treatment and chronic compartment respectively, and Δ is the transfer rate from chronic to treatment compartment. Υ and Φ are the the rate at which individuals recover from a disease, either through their own immune system (spontaneous recovery) or through treatment with antiviral drugs respectively. τ is the vaccination rate and ρ decline in TB vaccine effectiveness over time. μ and κ are the natural mortality rate and the TB induced death rate respectively.

2.1.1 Model Assumptions

The assumptions made in using the model are:

1. Individuals are not born with immunity to a particular disease, and even if they receive a vaccine, the protection it offers can wear off over time. - Studies have shown that vaccine-induced immunity can decline over time due to various factors, such as: - Decreasing antibody titers - Loss of immune memory cells - Changes in immune response with age

2. The individuals in the population interact and mix evenly, with no specific groups or structures forming.

3. The population is in a state of equilibrium, where the number of new individuals entering the population (through birth) is equal to the number of individuals leaving the population (through natural death) i.e Birth rate is equal to natural death rate

4. The newborn carriers is overall lower than the sum of carriers that have died and the population is shifting from carrier state to a stable immune state

5. We assume that the individuals moving from the active compartment to the chronic compartment is lower than the sum of the individuals moving to treatment and isolated compartments respectively. Also, the rate of individuals moving out of the chronic compartment is greater than the rate at which individuals moves in. It is equally worthy to note that we assume those receiving treatment and being isolated can die as a result of natural death but not by TB. The figure below represents the flow diagram of the **SVLACTR** model of Tuberculosis with vital dynamics.

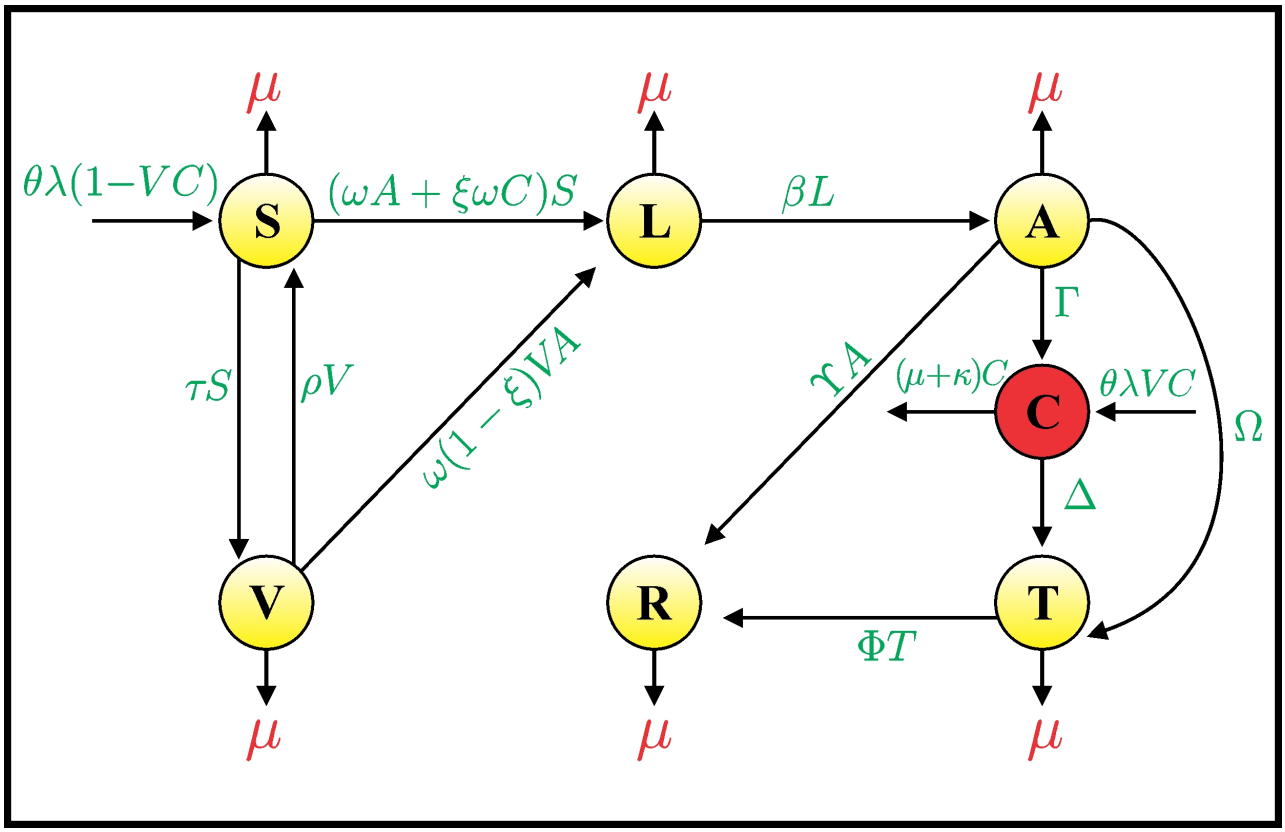

Figure 1: Flow diagram for transmission dynamics of onchocerciasis model.

2.1.2 Model equation

Considering the descriptions, assumptions and the model diagram the following model equations are obtained.

$$\frac{dS}{dt} = \theta\lambda(1 - VC(t)) + \rho V(t) - (\tau + \mu + \omega A(t) + \xi\omega C(t))S(t) \tag{2.1}$$

$$\frac{dV}{dt} = \tau S(t) - (\mu + \rho + \omega(1 - \xi)A(t))V(t) \tag{2.2}$$

$$\frac{dL}{dt} = (\omega A(t) + \xi\omega C(t))S(t) + \omega(1 - \xi)V(t)A(t) - (\mu + \beta)L(t) \tag{2.3}$$

$$\frac{dA}{dt} = \beta L(t) - (\Upsilon + \mu + \Gamma + \Omega)A(t) \tag{2.4}$$

$$\frac{dC}{dt} = \theta\lambda V(t)C(t) + \Gamma A(t) - (\Delta + \mu + \kappa)C(t) \tag{2.5}$$

$$\frac{dT}{dt} = \Omega A(t) + \Delta C(t) - (\mu + \phi)T(t) \tag{2.6}$$

$$\frac{dR}{dt} = \Upsilon A(t) + \Phi T(t) - \mu R(t) \tag{2.7}$$

Combining the equations above give the system of Tuberculosis dynamics equations below.

$$\begin{cases} \frac{dS}{dt} &= \theta\lambda(1-VC(t)) + \rho V(t) - (\tau+\mu+\omega A(t)+\xi\omega C(t))S(t) \\ \frac{dV}{dt} &= \tau S(t) - (\mu+\rho+\omega(1-\xi)A(t))V(t) \\ \frac{dL}{dt} &= (\omega A(t)+\xi\omega C(t))S(t) + \omega(1-\xi)V(t)A(t) - (\mu+\beta)L(t) \\ \frac{dA}{dt} &= \beta L(t) - (\Upsilon+\mu+\Gamma+\Omega)A(t) \\ \frac{dC}{dt} &= \theta\lambda V(t)C(t) + \Gamma A(t) - (\Delta+\mu+\kappa)C(t) \\ \frac{dT}{dt} &= \Omega A(t) + \Delta C(t) - (\mu+\phi)T(t) \\ \frac{dR}{dt} &= \Upsilon A(t) + \Phi T(t) - \mu R(t) \end{cases} \tag{2.8}$$

With the initial conditions S(0) = $S_0 > 0$, V(0) = $V_0 > 0$, L(0) = $L_0 \geq 0$, A(0) = $A_0 \geq 0$, C(0) = $C_0 \geq 0$, T(0) = $T_0 \geq 0$, R(0) = $R_0 \geq 0$.

Table 2.1: Description of the model variables

Variables	Description
$S(t)$	Susceptible Class
$V(t)$	Vaccinated Class
$L(t)$	Latent Class
$A(t)$	Acute Class
$C(t)$	Chronic Class
$T(t)$	Treatment Class
$R(t)$	Recovery Class

Table 2.2: Description of Parameter Values

Parameter	Values	Source
θ	5	[11]
ρ	0.67	[12]
τ	0.5	[12]
μ	0.0101	[?]
ω	0.6501	[12]
ξ	0.53521	[13]
Υ	0.5	Assumed
Φ	0.02	[14]
Ω	0.1	[12]
Δ	5	Assumed
ϕ	0.042553	[13]
Γ	0.5	Assumed
κ	0.027855	[13]
β	0.39818	[15]

3 Analysis of the model

3.1 Permanence of a Model

A model is considered permanent if it possesses the ability to sustain itself and persist over time, without succumbing to extinction or dissipation. In the context of system (3.8), permanence plays a crucial role in determining the stability of the endemic equilibrium. If system (3.8) exhibits weak permanence, the endemic equilibrium may be vulnerable to perturbations, leading to instability or even non-existence. On the other hand, if system (3.8) demonstrates strong permanence, the endemic equilibrium will exhibit resilience and stability, withstanding perturbations and maintaining its stability over time. This distinction highlights the importance of permanence in understanding the long-term behavior of the system and the fate of the endemic equilibrium.

We assumed that system (3.8) is a strong permanence system if there are constants $P_1, p_1 > 0$ such that for each positive solution of (S(t), V(t), L(t), A(t), C(t), T(t), R(t)) of system (3.8) with initial conditions S(0) = $S_0 > 0$, V(0) = $V_0 > 0$, L(0) = $L_0 \geq 0$, A(0) = $A_0 \geq 0$, C(0) = $C_0 \geq 0$, T(0) = $T_0 \geq 0$, R(0) = $R_0 \geq 0$. satisfies

$P_1 \geqslant \lim_{t\to\infty} SupS(t) \geqslant \lim_{t\to\infty} InfS(t) \geqslant p_1$
$P_1 \geqslant \lim_{t\to\infty} SupV(t) \geqslant \lim_{t\to\infty} InfV(t) \geqslant p_1$
$P_1 \geqslant \lim_{t\to\infty} SupL(t) \geqslant \lim_{t\to\infty} InfL(t) \geqslant p_1$
$P_1 \geqslant \lim_{t\to\infty} SupA(t) \geqslant \lim_{t\to\infty} InfA(t) \geqslant p_1$
$P_1 \geqslant \lim_{t\to\infty} SupC(t) \geqslant \lim_{t\to\infty} InfC(t) \geqslant p_1$
$P_1 \geqslant \lim_{t\to\infty} SupT(t) \geqslant \lim_{t\to\infty} InfT(t) \geqslant p_1$
$P_1 \geqslant \lim_{t\to\infty} SupR(t) \geqslant \lim_{t\to\infty} InfR(t) \geqslant p_1$

3.2 Existence and Uniqueness of the Model:

The validity and authenticity of any mathematical model depends on whether the given system of equations has a solution, and if the solution is unique. We shall use the Lipchitz condition to verify the existence and uniqueness of solution for the system of equations (3.9).
Let the system of equation (3.9) be as follows

$$\begin{cases} F_1 = \theta\lambda(1 - VC(t)) + \rho V(t) - (\tau + \mu + \omega A(t) + \xi\omega C(t))S(t) \\ F_2 = \tau S(t) - (\mu + \rho + \omega(1-\xi)A(t))V(t) \\ F_3 = (\omega A(t) + \xi\omega C(t))S(t) + \omega(1-\xi)V(t)A(t) - (\mu + \beta)L(t) \\ F_4 = \beta L(t) - (\Upsilon + \mu + \Gamma + \Omega)A(t) \\ F_5 = \theta\lambda V(t)C(t) + \Gamma A(t) - (\Delta + \mu + \kappa)C(t) \\ F_6 = \Omega A(t) + \Delta C(t) - (\mu + \phi)T(t) \\ F_7 = \Upsilon A(t) + \Phi T(t) - \mu R(t) \end{cases} \tag{3.1}$$

Theorem 3.1 (Derrick and Groosman) cited in [16]: Let D denote the region $|t-t_0| \leq a$, $||x-x_0|| \leq b$ such that $x = (x_0, x_2, x_3, ..., x_n)$ and And suppose that $f(t,x)$ satisfies the Lipchitz condition, $||f(t,x_1) - f(t,x_2)|| \leq k||x_1 - x_2||$ whenever the pairs $(t,x_1) and (t,x_2)$ belong to D, where k is a positive constant then there is a constant $\delta > 0$ such that a unique continous vector solution $x(t)$ of the system in the interval $t - t_0 \leq \delta$. It is important to note that the condition is satisfied by the requirement that $\frac{\partial F_i}{\partial x_j}$ where $i, j = 1, 2, 3, ...$ be continuous and bounded in D Considering the model equations (3.9), we are interested in the region $0 \leq \alpha \leq R$. We look for the bounded solution in the region and whose partial derivatives satisfy $f \leq \alpha \leq 0$. where α and δ are positive constants.
Let D denote the region $0 \leq \alpha \leq R$, then equation (3.9) have a unique solution. We show that $\frac{\partial F_i}{\partial x_j}$ where $i, j = 1, 2, 3, 4, 5, 6, 7$ Are continuous and bounded in D

Theorem 3.2. [17] Let $\varrho(0)$ = (S(0), V(0), L(0), A(0), C(0), T(0), R(0)) ϵ R_+^7 be the initial condition for model (3.9). Hence, the set of solutions { S(0), V(0), L(0), A(0), C(0), T(0), R(0)} is non

negative for all t > 0 and N $\leqslant \frac{\theta\lambda}{\mu}$.

Proof: Let t_1 = sup $\{t > 0 : S(t_0) > 0, L(t_0) > 0, I(t_0) > 0, C(t_0) > 0, T(t_0) > 0, E(t_0) > 0, R(t_0) > 0, \forall t_0 \in [0, t]\}$. Since S(0), V(0), L(0), CA(0), C(0), T(0) and R(0) are all non negative then $t_1 > 0$. If $t_1 < \infty$, using the variation of the constant formula of the first equation of model (3.1) at t_1 we have;

$$\frac{dS}{dt} = \theta\lambda(1 - VC(t)) + \rho V(t) - (\tau + \mu + \omega A(t) + \xi\omega C(t))S(t).$$

This is linear and can be written as

$$\frac{dS}{dt} - (\tau + \mu + \omega A(t) + \xi\omega C(t))S(t) = \theta\lambda(1 - VC(t)) + \rho V(t) \tag{3.2}$$

with IF = $e^{(\tau+\mu+\omega A(t)+\xi\omega C(t)))dt}$
multiplying both sides of (3.10) by IF and rearranging we have;

$$\frac{d}{dt}\left[S(t)e^{(\tau+\mu)t+\int_0^t P(y)dy}\right] = \left[\theta\lambda(1 - VC(t)) + \rho V(t)\right] e^{(\tau+\mu)t+\int_0^t P(y)dy}, \tag{3.3}$$

Where $P = [\omega(A(t) + \xi C(t))]$.
Therefore,

$$S(t_1)e^{(\tau+\mu)t+\int_0^t P(y)dy} - S(0) = \int_0^{t_1} \left[\theta\lambda(1 - VC(r)) + \rho V(r)\right] \left[e^{(\tau+\mu)r+\int_0^t P(y)dy}\right] dr \tag{3.4}$$

Hence,

$$\begin{aligned} S(t_1) = S(0)e^{(\tau+\mu)t+\int_0^t P(y)dy} + \left[e^{(-\tau-\mu)t-\int_0^t P(y)dy}\right] \int_0^{t_1} \theta\lambda(1 - VC(r)) \\ +\rho V(r) \left[e^{(\tau+\mu)t+\int_0^t P(y)dy}\right] dr \geqslant 0 \end{aligned} \tag{3.5}$$

We can go further to prove for other state variables following same approach. Hence, we show that the region $\varrho(t)$ is positively-invariant for the formulated Tuberculosis model (3.9) with
$\varrho(t) \geqslant 0 \epsilon R_+^7$
From (4.1) we obtain,

$$\frac{dN}{dt} = \theta\lambda - \mu N - \kappa C(t) \tag{3.6}$$

But κ and the state parameter C are positive in equation (3.1), therefore we have;

$$\frac{dN}{dt} = \theta\lambda - \mu N \tag{3.7}$$

Solving equation (3.7) by integrating factor method with the initial condition N(0) = N_0 we have;

$$N \leqslant \frac{\theta\lambda}{\mu} + (N_0 - \frac{\theta\lambda}{\mu})e^{-\mu t} \tag{3.8}$$

In conclusion, we have $N(t) \to \frac{\theta\lambda}{\mu}$ as $t \to \infty$, This means that $0 < N \leqslant \frac{\theta\lambda}{\mu}$. Furthermore, if $N > \frac{\theta\lambda}{\mu}$, then the solution either enter $\varrho(t)$ in a finite time . So, it can be concluded that $\varrho(t)$ attracts all the solutions in R_+^7 and hence all the properties hold for the system to become positive invariant, unique and boundedness in the region by $\varrho(t)$. Thus, the feasible region of the system is ,

$$\varrho = \{(S, V, L, A, C, T, R) \in R_+^7 : 0 < N \leqslant \frac{\theta\lambda}{\mu}\} \tag{3.9}$$

Disease-Free Equilibrium:
Is referred to as TB-free equilibrium steady state is described as the state at which the population if free from the presence of TB infection, to obtain the TBFE we set the right hand side of model (3.1) to zero(0). i.e

$$\begin{cases} 0 = \theta\lambda(1 - VC(t)) + \rho V(t) - (\tau + \mu + \omega A(t) + \xi\omega C(t))S(t) \\ 0 = \tau S(t) - (\mu + \rho + \omega(1-\xi)A(t))V(t) \\ 0 = (\omega A(t) + \xi\omega C(t))S(t) + \omega(1-\xi)V(t)A(t) - (\mu+\beta)L(t) \\ 0 = \beta L(t) - (\Upsilon + \mu + \Gamma + \Omega)A(t) \\ 0 = \theta\lambda V(t)C(t) + \Gamma A(t) - (\Delta + \mu + \kappa)C(t) \\ 0 = \Omega A(t) + \Delta C(t) - (\mu + \phi)T(t) \\ 0 = \Upsilon A(t) + \Phi T(t) - \mu R(t) \end{cases} \tag{3.10}$$

Solving equation (3.10)for Disease Free Equilibrium (DFE) points and noting that in the absence of TB disease, L=A=C=T=0. We have the disease free equilibrium points denoted by, $D_* = (S^*, V^*, L^*, A^*, C^*, T^*, R^*)$ as:

$$D_* = \left\{ \frac{\theta(\mu+\rho)}{\mu(\mu+\rho+\tau)}, \frac{(\mu+\rho)\theta\lambda}{\mu(\mu+\rho+\tau)}, 0, 0, 0, 0, 0, \right\} \tag{3.11}$$

The above partial derivatives exist, continuous and are bounded, Hence, by theorem 3.1, the model has a unique solutions.

The Basic Reproductive Number R_0^{TB}:
The basic reproductive number of Tuberculosis epidemiological threshold is represented by the symbol $R_0^{TB} = \rho(FV^{-1})$, and ρ denotes the dominant eigenvalue, where F is the term which contains secondary infections and V contains other terms which do not include secondary infections or in other way F and V are the new infection and transmission term respectively. To derive the expression in terms of parameters for the basic reproduction number of the system (3.1), we have

$$F = \begin{bmatrix} (\omega A(t) + \xi\omega C(t))S(t) \\ 0 \\ 0 \end{bmatrix}, V = \begin{bmatrix} (\omega A(t) + \xi\omega C(t))S(t) + \omega(1-\xi)V(t)A(t) - (\mu+\beta)L(t) \\ \beta L(t) - (\Upsilon + \mu + \Gamma + \Omega)A(t) \\ \theta\lambda V(t)C(t) + \Gamma A(t) - (\Delta + \mu + \kappa)C(t) \end{bmatrix}$$

At Tuberculosis DFE, the Jacobian matrices of F and V are evaluated, giving the following;

$$F^* = S^0 \begin{bmatrix} 0 & \omega & \xi\omega \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, V^* = \begin{bmatrix} -(\mu+\beta) & \omega & 0 \\ \beta & -(\Upsilon+\mu+\Gamma+\Omega) & 0 \\ 0 & \Gamma & -(\Delta+\mu+\kappa) \end{bmatrix}$$

Let $P = (\mu + \beta)$,
$Q = (\Upsilon + \mu + \Gamma + \Omega) and$
$R = (\Delta + \mu + \kappa)$ Such that V^* becomes

$$V^* = \begin{bmatrix} -P & \omega & 0 \\ \beta & -Q & 0 \\ 0 & \Gamma & -R \end{bmatrix}$$

$$V^{*-1} = \begin{bmatrix} \frac{-1}{PQ-\beta\omega} & \frac{-\omega}{PQ-\beta\omega} & 0 \\ \frac{-\beta}{PQ-\beta\omega} & \frac{-P}{PQ-\beta\omega} & 0 \\ \frac{\beta\Gamma}{\beta R\omega - PQR} & \frac{P\Gamma}{\beta R\omega - PQR} & \frac{-1}{R} \end{bmatrix} \tag{3.12}$$

$$F^*V^{*-1} = \begin{bmatrix} \frac{(\beta\xi\Gamma\omega+R\beta\omega)S^0}{R\beta\omega-PQR} & \frac{(PQ-\beta\omega)\xi\Gamma P\omega S^0-(\beta R\omega-PQR)P\omega S^0}{(PQ-\beta\omega)(\beta R\omega-PQR)} & \frac{\xi\omega S^0}{R} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \tag{3.13}$$

The largest absolute value (spectral radius) of the eigenvalues of the matrix (3.13) gives the basic reproductive number, which is;
$\mid FV^{-1} - \lambda I \mid$. Where I is Identity Matrix. Perfoming the operation yiels our basic reproduction number R_0^{HBV}

$$R_0^{TB} = \frac{\beta\omega(\xi\Gamma + \Delta + \mu + \kappa)}{[(\Delta + \mu + \kappa)\beta\omega - (\mu + \beta)(\Upsilon + \mu + \Gamma + \Omega)\Delta + \mu + \kappa]} \tag{3.14}$$

Equation (3.14) represents the average number of secondary infections produced by an active and/or chronic individual when introduced into the susceptible population.

Theorem 3.3 [18] If $R_0^{TB} < 1$ then the equilibrium point D_0 is locally asymptotically stable otherwise unstable if $R_0^{TB} > 1$.
Proof: The desired Jacobian matrix J_{D_0} of the model (2.1) at DFE is given by;

$$J_{D_0} = \begin{bmatrix} -\tau-\mu & \rho & 0 & -\omega S^0 & -\xi\omega S^0 & 0 & 0 \\ \tau & -\mu-\rho & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu-\beta & \omega S^0 & \xi\omega S^0 & 0 & 0 \\ 0 & 0 & \beta & -(\Upsilon+\mu+\Gamma+\Omega) & 0 & 0 & 0 \\ 0 & 0 & 0 & \Gamma & -(\Delta+\mu+\kappa) & 0 & 0 \\ 0 & 0 & 0 & \Omega & \Delta & -\mu-\phi & 0 \\ 0 & 0 & 0 & \Upsilon & 0 & \Phi & -\mu \end{bmatrix} \tag{3.15}$$

By expanding the determinant of the characteristic equation $|J(D_0) - \lambda| = 0$, we obtained
$\lambda_1 = -\tau - \mu < 0$,
$\lambda_2 = -\mu - \rho < 0$,
$\lambda_3 = -\mu - \phi < 0$,
$\lambda_4 = -\mu < 0$,
Therefore the remaining three eigenvalues are eigenvalues of

$$J_{D_1} = \begin{bmatrix} -\mu-\beta & \omega S^0 & \xi\omega S^0 \\ \beta & -\Upsilon-\mu-\Gamma-\Omega & 0 \\ 0 & \Gamma & -\Delta-\mu-\kappa \end{bmatrix} \tag{3.16}$$

From the above equation (3.16) let $X = \Upsilon + \mu + \Gamma - + \Omega$ and $Y = \Delta - \mu - \kappa$ so that the characteristic equation become easier to operate.
Again by expanding the determinant of the characteristic equation $|JD_1|$ in equation (3.16) we have the characteristic polynomial to be ;

$$\lambda^3 + P_1\lambda^2 + P_2\lambda + P_3 = 0 \tag{3.17}$$

Where;
$P_1 = \mu + \beta + X + Y$,
$P_2 = \mu X + \mu Y + \beta X + \beta Y + XY - \beta\omega S^0$ and
$P_3 = (\mu + \beta)XY + \omega Y S^0 + \xi\Gamma\beta\omega S^0$

The characteristic polynomial in (3.17) follows the Routh-Hurwiz criteria, where the associated Hurwiz matrix to (3.17) is;

$$\begin{bmatrix} P1 & P_3 & 0 \\ 1 & P_2 & 0 \\ 0 & P_1 & P_3 \end{bmatrix} \tag{3.18}$$

The necessary and sufficient condition for the polynomial to have roots with negative real part are;
(a). $D_1 = P_1 > 0$
(b). $D_2 = P_1P_2 > P_3$,
(c). $D_3 = P_1P_2P_3 - P_3^2 > 0, \Longrightarrow P_3 > 0$
Hence, it will be good enough to conclude that the disease free equilibrium is locally asymptotically stable if $R_0^{TB} > 1$ and the necessary Routh-Hurwiz conditions are satisfied otherwise its unstable.

Endemic Equilibrium Point: In Epidemiology, the term endemic equilibrium point refers to the point at which the number of cases of a disease in a population remains constant over time. The endemic equilibrium point can be influenced by a variety of factors, including the transmission rate of the disease, the effectiveness of prevention and treatment measures, and the natural immunity of the population. In some cases, the endemic equilibrium point may be zero, meaning that the disease is completely eliminated from the population. The system (3.1) be at equilibrium to a steady-state where the tuberculosis disease exists in the population. This is denoted by $D_0^{**} = (S^{**}, V^{**}, L^{**}, A^{**}, C^{**}, T^{**}, R^{**})$.
Equating the left side of system (4.1) to zero we have,

$$\begin{cases} 0 = \theta\lambda(1 - VC(t)) + \rho V(t) - (\tau + \mu + \omega A(t) + \xi\omega C(t))S(t) \\ 0 = \tau S(t) - (\mu + \rho + \omega(1-\xi)A(t))V(t) \\ 0 = (\omega A(t) + \xi\omega C(t))S(t) + \omega(1-\xi)V(t)A(t) - (\mu + \beta)L(t) \\ 0 = \beta L(t) - (\Upsilon + \mu + \Gamma + \Omega)A(t) \\ 0 = \theta\lambda V(t)C(t) + \Gamma A(t) - (\Delta + \mu + \kappa)C(t) \\ 0 = \Omega A(t) + \Delta C(t) - (\mu + \phi)T(t) \\ 0 = \Upsilon A(t) + \Phi T(t) - \mu R(t) \end{cases} \tag{3.19}$$

Demonstrating equation (3.19) making the other state variables in terms of A^{**} we arrived at;

$$\begin{cases} S^{**} = \frac{\theta\lambda[\mu+\rho+\omega(1-\xi)A^{**}]}{\tau\Gamma A^{**}} \\ V^{**} = \frac{\theta\lambda}{\Gamma A^{**}} \\ L^{**} = \frac{(\Upsilon+\mu+\Gamma+\Omega)A^{**}}{\beta} \\ T^{**} = \frac{[\Omega(\Delta+\mu+\kappa)-\theta\lambda+\Delta\Gamma]A^{**}}{(\Delta+\mu+\kappa)-\theta\lambda} \\ C^{**} = \frac{\Gamma A^{**}}{(\Delta+\mu+\kappa)-\theta\lambda} \\ R^{**} = \frac{\Upsilon\mu[(\Delta+\mu+\kappa)-\theta\lambda]A^{**}+\phi[\Delta+\mu+\kappa)-\theta\lambda+\Delta\Gamma]A^{**}}{[\Delta+\mu+\kappa)-\theta\lambda]A^{**}} \end{cases} \tag{3.20}$$

Equation (3.20) is the endemic equilibrium state for all the state variables/parameter.

3.3 Optimal Control Analysis

When a tuberculosis (TB) outbreak occurs, it is crucial for health authorities to implement effective interventions to control the spread of the disease. However, selecting the most effective control measures can be a daunting task. To overcome this challenge, we will utilize the insights gained from the sensitivity analysis conducted earlier. By applying the results of the sensitivity analysis, we can identify the most effective control strategies and measures that, when adopted, will minimize the impact of

TB on the population. This will enable health authorities to make informed decisions and implement targeted interventions to mitigate the outbreak and protect public health.
To combat the spread of TB, we utilize four control variables in the TB model (3.8). As previously discussed, vaccination is the initial intervention strategy employed to prevent new infections and halt the multiplication of infected cells. By administering vaccines, we aim to significantly reduce the number of new cases and thereby contain the outbreak. This will lower the un-vaccinated rate of individuals going into the susceptible population and it is denoted by $\Lambda_1(t)$. The second intervention $\Lambda_2(t)$, is to reduce the chances of contacts with infectious and chronic individuals. This will lower the successful contact rates Ω and Γ relative to infectious and chronic individuals respectively. The third control variable we considered is to encourage or maximize the treatment and isolation of TB infected patients by stepping up their rates and this intervention is denoted by $\Lambda_3(t)$. Lastly, the fourth control variable $\Lambda_4(t)$ is TB awareness. Where the control variables $\Lambda_1(t)$, $\Lambda_2(t)$, $\Lambda_3(t)$, and $\Lambda_4(t)$ are functions of time, making the control system time-dependent.

$$\begin{cases} \frac{dS}{dt} &= \theta\lambda(1-VC)+\rho V-(\tau+\mu+\omega A+\xi\omega C)S-(1-\Lambda_1)S-(1-\Lambda_2)S-(1-\Lambda_4)S \\ \frac{dV}{dt} &= \tau S-(\mu+\rho+\omega(1-\xi)AV-(1-\Lambda_1)V-(1-\Lambda_4)V \\ \frac{dL}{dt} &= (\omega A+\xi\omega C)S+\omega(1-\xi)VA-(\mu+\beta)-(1-\Lambda_1)L \\ \frac{dA}{dt} &= \beta L-(\Upsilon+\mu+\Gamma+\Omega)A-(1-\Lambda_2)A-(1-\Lambda_3)A \\ \frac{dC}{dt} &= \theta\lambda VC+\Gamma A-(\Delta+\mu+\kappa)C-(1-\Lambda_2)C-(1-\Lambda_3)C-(1-\Lambda_4)C \\ \frac{dT}{dt} &= \Omega A+\Delta C-(\mu+\phi)T-(1-\Lambda_3)T-(1-\Lambda_4)T \\ \frac{dR}{dt} &= \Upsilon A+\Phi T-\mu R+(1-\Lambda_1)S+(1-\Lambda_2)S+(1-\Lambda_4)S+(1-\Lambda_1)V+(1-\Lambda_4)V \end{cases} \tag{3.21}$$

$$(1-\Lambda_1)L+(1-\Lambda_2)A+(1-\Lambda_3)A+(1-\Lambda_2)C+(1-\Lambda_3)C+(1-\gamma_4)C$$

With the initial conditions S(0) = $S_0 > 0$, V(0) = $V_0 > 0$, L(0) = $L_0 \geq 0$, A(0) = $A_0 \geq 0$, C(0) = $C_0 \geq 0$, T(0) = $T_0 \geq 0$, R(0) = $R_0 \geq 0$.

$$J(\Lambda_1(t),\Lambda_2(t),\Lambda_3(t),\Lambda_4(t)) = \int_0^{T_f} \{\psi_1 S(t)+\psi_2 V(t)+\psi_3 L(t)+\psi_4 A(t)+\psi_5 C(t)$$

$$+\psi_6 T(t)+\psi_7 R(t)+\frac{1}{2}(\psi_8\Lambda_1^2(t)+\psi_9\Lambda_2^2(t)+\psi_{10}\Lambda_3^2(t)+\psi_{11}\Lambda_4^2(t))\}dx \tag{3.22}$$

The weight constants $(\psi_1,\psi_2,\psi_3,\psi_4,...,\psi_{11})$ and the final time (T_f) are used to optimize the control system, where: $\psi_1,\psi_2,\psi_3,\psi_4,...,\psi_{11}$ represent the relative importance of each control variable $(\Lambda_1,\Lambda_1,\Lambda_1,\Lambda_1)$ in the objective function, T_f represents the final time point of the control interval
The goal is to find the optimal controls that minimizes or maximizes the objective function, subject to the dynamics of the system and the constraints imposed by the weight constants and final time.

$$J(\Lambda_1^*(t),\Lambda_2^*(t),\Lambda_3^*(t),\Lambda_4^*(t)) = minJ\{(\Lambda_1(t),\Lambda_2(t),\Lambda_3(t),\Lambda_4(t))\}, \tag{3.23}$$

where $\Lambda_1(t),\Lambda_2(t),\Lambda_3(t),\Lambda_4(t) \in \Lambda$ and
$\Lambda = \{(\Lambda_1(t),\Lambda_2(t),\Lambda_3(t),\Lambda_4(t)) : [0,1],\Lambda_1(t),\Lambda_2(t),\Lambda_3(t),\Lambda_4(t)\}$ is Measurable with respect to the Lebesgue measure

3.2.1 Existence of a Feasible Control Solution

In this section, we present proof for the existence of the control variables $\Lambda_1(t),\Lambda_2(t),\Lambda_3(t),\Lambda_4(t) \in \Lambda$. We have shown that the model (3.8) is bounded and non-negative, this finding enables us to establish the existence of an optimal control strategy over a finite time, and by definition, the control variables form a convex set (i.e after taking the eigenvalues of the Hessian Matrix of 4.32 we found out that all other parameters are zero except that of the control parameters).
The optimized system is well-defined and can be utilized to validate the optimal control system described by equation (4.32). Additionally, the objective function's integrand,
$\{\psi_1 S(t)+\psi_2 V(t)+\psi_3 L(t)+\psi_4 A(t)+\psi_5 C(t)+\psi_6 T(t)+\psi_7 R(t)+\frac{1}{2}(\psi_8\Lambda_1^2(t)+\psi_9\Lambda_2^2(t)+\psi_{10}\Lambda_3^2(t)+\psi_{11}\Lambda_4^2(t),\}$ is convex on the control set Λ. Hence the control system exist.

3.2.2 3.3.2 Hamiltonian Optimal System

For optimal control problems (4.32) and (4.34), the Lagrangian and Hamiltonian are utilized. The Pontryagin maximum principle provides the necessary conditions that are satisfied by the optimal solution. The optimal control problem can be represented in the Lagrangian form as follows; L= $\{S(t), V(t), L(t), A(t), C(t), T(t), R(t)\Lambda_1(t), \Lambda_2(t), \Lambda_3(t), \Lambda_4(t)\}$
= $\psi_1 S(t) + \psi_2 V(t) + \psi_3 L(t) + \psi_4 A(t) + \psi_5 C(t) + \psi_6 T(t) + \psi_7 R(t) + \frac{1}{2}(\psi_8 \Lambda_1^2(t) + \psi_9 \Lambda_2^2(t) + \psi_{10}\Lambda_3^2(t) + \psi_{11}\Lambda_4^2(t))$

In our quest to minimize the Lagrangian, we employ the Pontryagin maximum principle, a fundamental tool in optimal control theory. By applying this principle, we successfully derive the Hamiltonian (H) function, which plays a crucial role in determining the optimal control strategy. The Hamiltonian function, denoted by H, is a crucial construct that encapsulates the dynamics of the system and the performance criterion. By analyzing the Hamiltonian, we can determine the optimal control policy that minimizes the Lagrangian, thereby achieving the desired optimal outcome. The Hamiltonian function is a vital intermediate step in the optimization process, and its derivation marks a significant milestone in our pursuit of the optimal control solution. L= $\{S(t), V(t), L(t), A(t), C(t), T(t), R(t)\Lambda_1(t), \Lambda_2(t), \Lambda_3(t), \Lambda_4(t)\}$

$$+\ell_1\frac{dS}{dt} + \ell_2\frac{dV}{dt} + \ell_3\frac{dL}{dt} + \ell_4\frac{dA}{dt} + \ell_5\frac{dC}{dt} + \ell_6\frac{dT}{dt} + \ell_7\frac{dR}{dt} \tag{3.24}$$

Where $\ell_1, \ell_2, \ell_3, \ell_4, \ell_5, \ell_6, \ell_7$ and $\Lambda_1(t), \Lambda_2(t), \Lambda_3(t), \Lambda_4(t), \Lambda_5(t), \Lambda_6(t), \Lambda_7(t)$ are adjoint and control variables respectively.

$$\begin{cases} \frac{\ell_1}{dt} = -\{\psi_1 - \ell_1(\tau + \omega A + \xi\omega C + \mu - \Lambda_1 - \Lambda_2 + 2) + \ell_2(\omega A + \xi\omega C) + \ell_7(-\Lambda_1 - \Lambda_2 + 2)\} \\ \frac{\ell_2}{dt} = -\{\psi_2 - \ell_2(\rho + \omega - \Lambda_1 + 1) + \ell_3(\xi A + \xi) + \ell_7(1 - \Lambda_1)\} \\ \frac{\ell_3}{dt} = -\{\psi_3 - \ell_3(\omega - \xi + \Lambda_2 + \Lambda_3 + 2) + \ell_4\mu + \ell_5\beta + \ell_6(1 - \Lambda_3)\} \\ \frac{\ell_4}{dt} = -\{\psi_4 - \ell_1(\beta - (\Upsilon + \mu + \Gamma + \Omega) - \ell_7(-\Lambda_3 - \Lambda_4 + 2))\} \\ \frac{\ell_5}{dt} = -\{\psi_5 - \ell_6(\theta + \lambda - \Lambda_3 + 1) + \ell_7(\Gamma - \Lambda_4 + 1)\} \\ \frac{\ell_6}{dt} = -\{\psi_6 - \ell_6(\mu + \phi - \Lambda_4 + 1) + \ell_7(\Omega - \Lambda_4 + 1)\} \\ \frac{\ell_7}{dt} = -\{\psi_7 - \ell_1\rho + \ell_7(\Upsilon)\} \end{cases} \tag{3.25}$$

Applying the conditions $\ell_x(T_f) = 0$ where $x = 1, 2, 3, 4, 5, 6, 7$. We can therefore write the control variables in form of:

$$\begin{aligned} \Lambda_1(t) &= min\left\{1, max\left(0, \frac{-\ell_1 S - \ell_2 V + \ell_7(S + V)}{\psi_8}\right)\right\} \\ \Lambda_2(t) &= min\left\{1, max\left(0, \frac{-\ell_3 S - \ell_2 A - \ell_3 C + \ell_7(S + A + C)}{\psi_9}\right)\right\} \\ \Lambda_3(t) &= min\left\{1, max\left(0, \frac{-\ell_3 A - \ell_4 C + \ell_7(A + C)}{\psi_{10}}\right)\right\} \\ \Lambda_2(t) &= min\left\{1, max\left(0, \frac{-\ell_1 S - \ell_4 C - \ell_5 T - \ell_5 L + \ell_7(S + C + L + T)}{\psi_{11}}\right)\right\} \end{aligned} \tag{3.26}$$

4 Numerical simulations/sensitivity analysis

4.1 Sensitivity Analysis

In this section, we analyze how sensitive the parameters in the R_0^{TB} are to changes in the spread of TB. This will help us identify which parameters have the most significant impact on the spread of the

disease. Sensitivity analysis is carried out on each parameters, this is used in checking and identifying parameters that are liable to impacting the reproductive number [19]. To measure the sensitivity of a parameter (ϱ), we use the formula:

$$\vartheta_{\varrho}^{R_0} = \left(\frac{\partial R_0}{\partial \varrho}\right) \cdot \left(\frac{\varrho}{R_0}\right)$$

Using this formula, we calculate the sensitivity index for each parameter in R_0^{TB} as follows:

Table 3.1: Sensitivity Indices of the Parameters in R_0^{TB}

Parameter	Values	Source
μ	0.0101	+
ω	0.6501	-
ξ	0.53521	-
Υ	0.5	+
Ω	0.1	-
Δ	5	+
Γ	0.5	-
κ	0.027855	+
β	0.39818	+

The sensitivity indices of the parameters in equation (3.22) play a crucial role in understanding the dynamics of TB spread in the population. By analyzing the values in Table 4.1, we observe that certain parameters $\mu, \Upsilon, \Delta, \kappa, \beta$ have a positive sensitivity index, which means they are directly linked to the spread of TB. When these parameters increase or decrease, the spread of TB also increases or decreases, consequently affecting the burden of TB in the population. In other words, if these parameters increase, the spread of TB will also increase, leading to a higher burden of TB in the population, and vice versa.

On the other hand, certain parameters $\omega, \xi, \Omega, \Gamma$ have a negative sensitivity index, indicating an inverse relationship with the spread of TB. When these parameters increase, the spread of TB decreases, leading to a lower burden of TB in the population. Conversely, when these parameters decrease, the spread of TB increases, resulting in a higher burden of TB in the population.
This analysis provides valuable insights into the dynamics of TB spread and the impact of various parameters on the burden of TB in the population. By understanding these relationships, we can develop strategies to control the spread of TB and mitigate the burden of TB in the population.

4.2 Numerical Simulation

In this section, we will use numerical methods to solve the models (4.1) specifically, we will employ the classical 4^{th} order Runge-Kutta using MATLAB to find the solutions to the dynamic equations with initial conditions for all the state variables in the model equation (4.1). This will allow us to visualize and analyze the behavior of the models over time. After solving the model equations we go ahead to plot graphs of each of the state variable after considering the control measures.

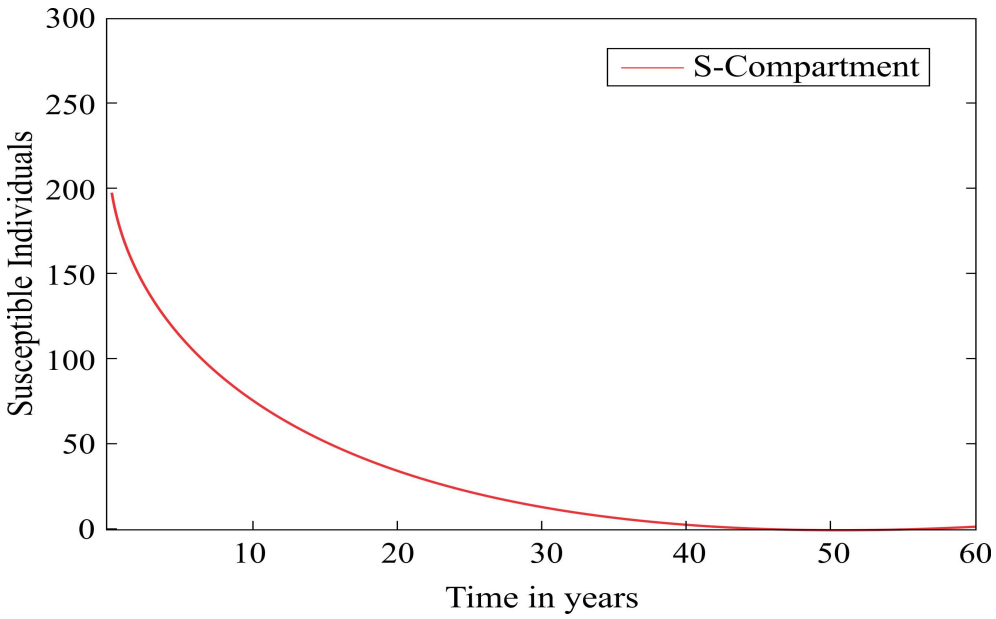

Figure 4.1: Graph of Susceptible Individuals

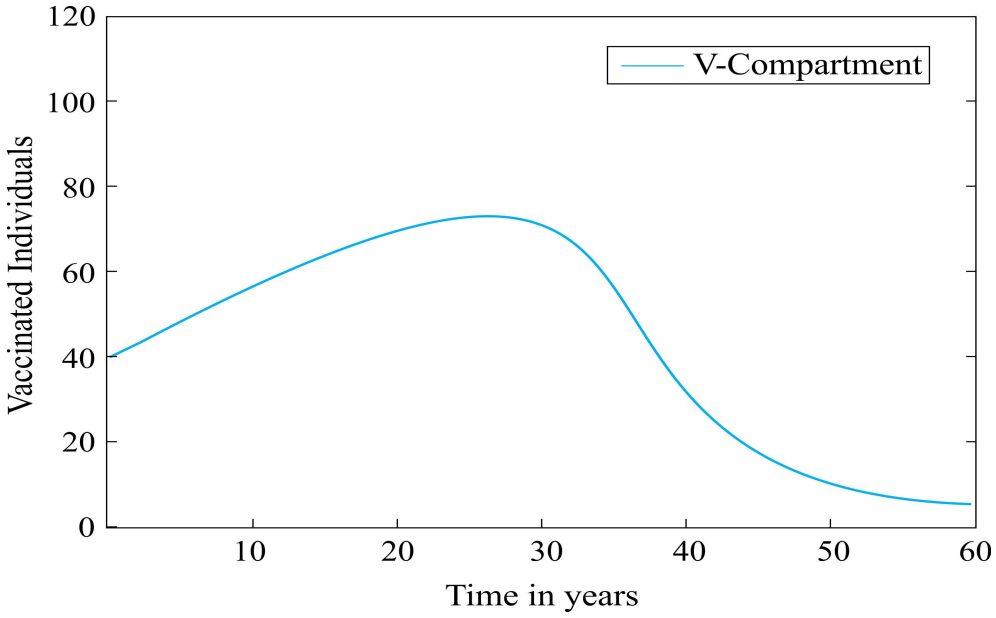

Figure 4.2: Graph of Vaccinated Individuals

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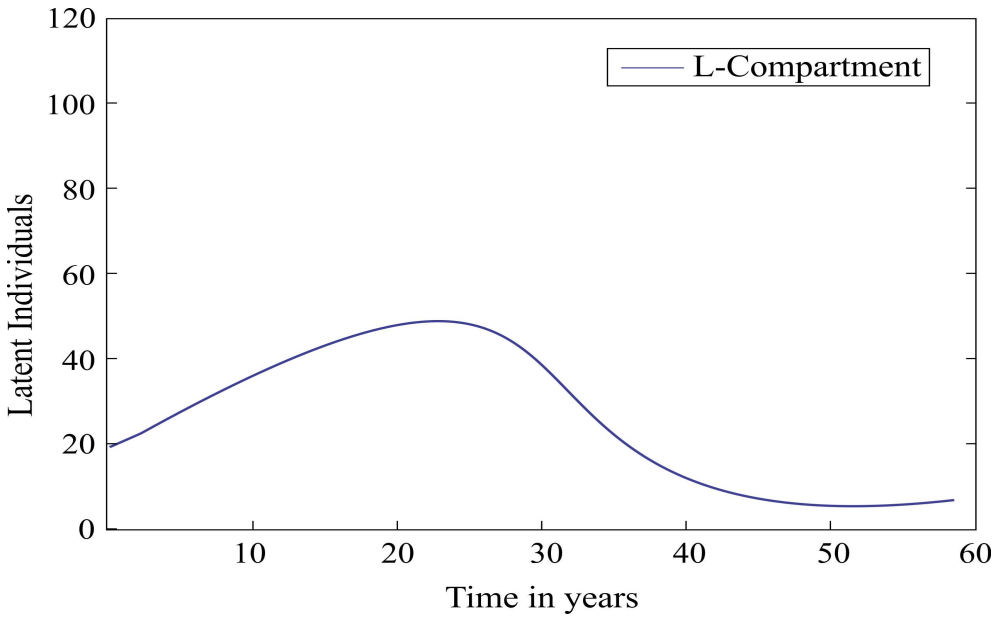

Figure 4.3: Graph of Latent Individuals

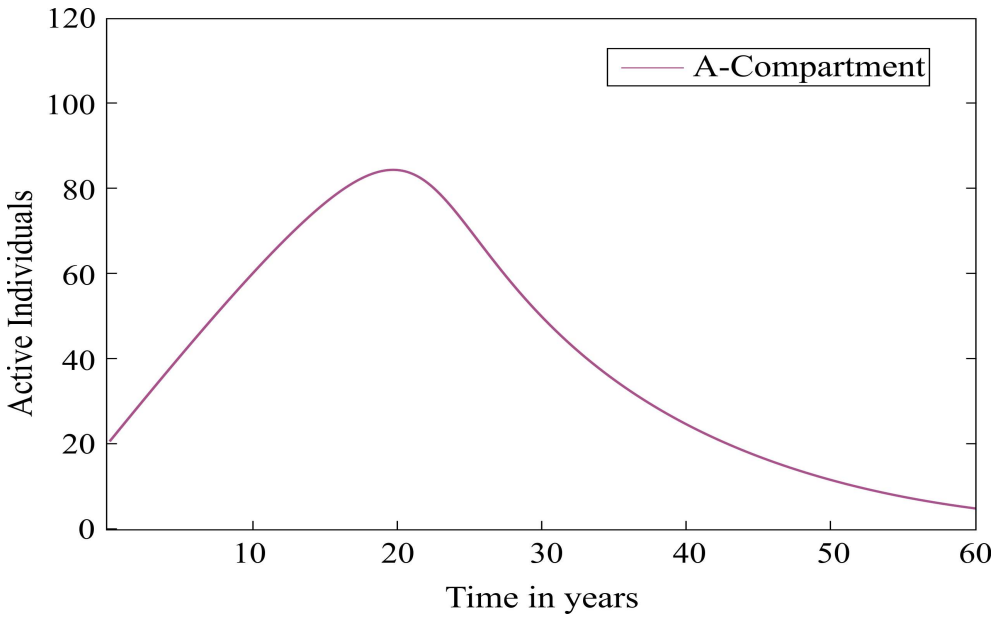

Figure 4.4: Graph of Vaccinated Individuals

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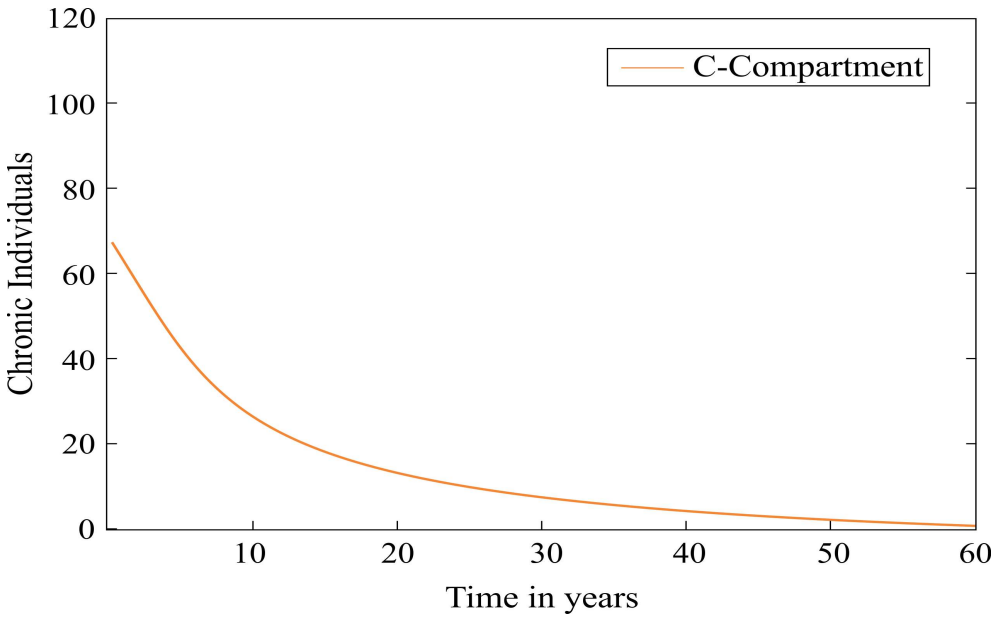

Figure 4.5: Graph of Chronic Individuals

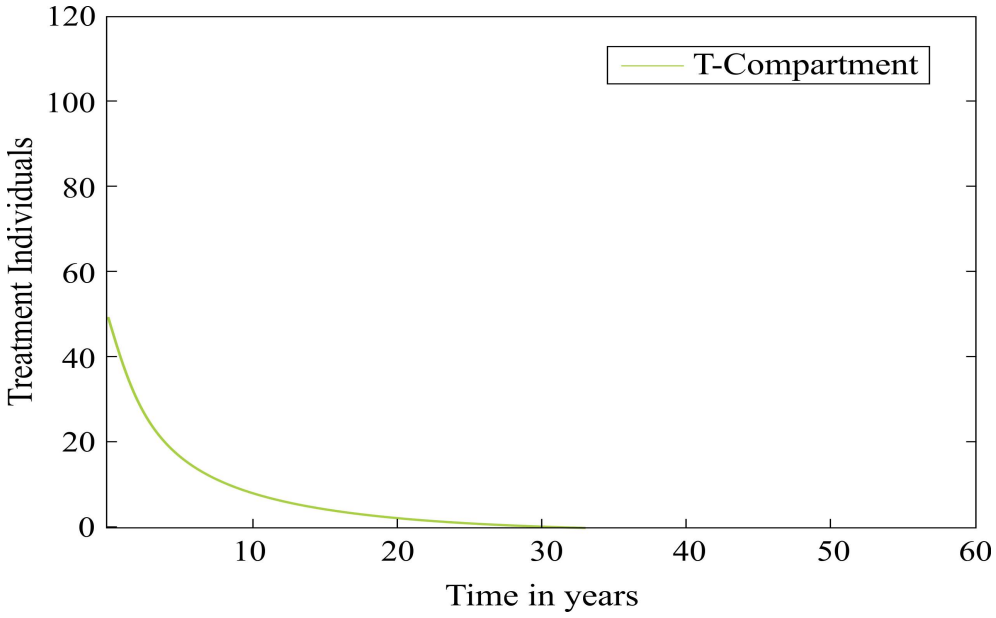

Figure 4.6: Graph of Individuals that undergo treatment

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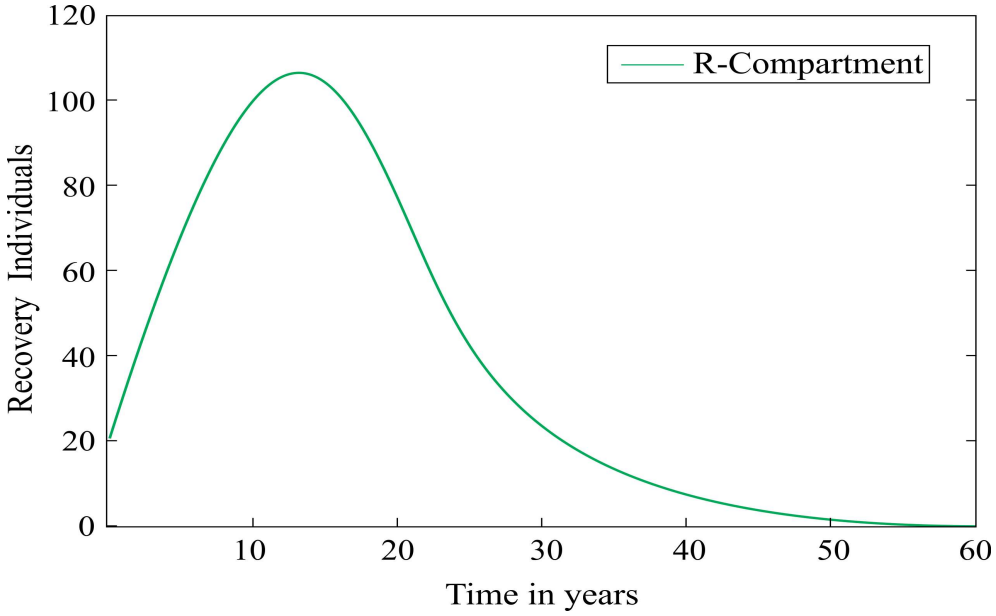

Figure 4.7: Graph of Recovered Individuals

5 Discussions and Conclusion

In these work we split the discussions based on model analysis.

5.1 Model formulation

This research has achieved a significant milestone by expanding a mathematical model of Tuberculosis (TB) to include two crucial aspects: the gradual loss of TB vaccine immunity over time (waning parameter) and an additional category for chronic cases. Our findings reveal that individuals who have recovered from TB eventually lose their immunity as the vaccine's effectiveness wears off, making them susceptible to the disease once again. This phenomenon contributes to the decline in the number of individuals in the recovered class, as illustrated in Figure 4.7. Moreover, our model shows a notable decrease in the number of individuals in the latent (Figure 4.3), active (Figure 4.4), and chronic (Figure 4.5) stages, which can be attributed to the inclusion of treatment and exclusion compartments in our model. These compartments effectively capture the impact of treatment and isolation on the spread of TB, leading to a reduction in the number of cases across all stages.

5.2 Existence and uniqueness

We demonstrate that the reliability and accuracy of a mathematical model hinge on the existence and uniqueness of a solution to the underlying system of equations. To establish the validity of our model, we employ the Lipchitz condition as a criterion to verify the existence and uniqueness of a solution, ensuring that the system has a unique solution that accurately represents the behavior of the real-world phenomenon being modeled, we then concluded that $\varrho(t)$ attracts all the solutions in R^7_+ and hence all the properties hold for the system to become positive invariant, unique and boundedness in the region by $\varrho(t)$.

5.3 Discussion based on DFE and EE

Our model analysis reveals that there are two equilibrium states: a disease-free equilibrium and an endemic equilibrium, which coexist in a stable manner, as confirmed by bifurcation analysis. This bistability indicates that the disease can persist in the population at either a low or high level, depending on the balance between the rates of transmission and recovery.

5.4 Sensitivity analysis

Our sensitivity analysis identifies the key parameters that drive the epidemiology of TB, as shown in Table 4.1. Specifically, parameters with a positive index have a direct relationship with the basic reproductive number, amplifying the spread of the disease, while parameters with a negative index have an inverse relationship, reducing the spread of the disease. This insight into the relative importance of each parameter can inform strategies to control and mitigate the impact of TB in the population.

5.5 Optimal control

Our analysis reveals that a multi-faceted approach is crucial to effectively combat the spread of Tuberculosis (TB). We incorporate four control variables into the TB model (3.8) to strategically combat the disease. Firstly, vaccination is employed as a preventive measure to hinder new infections and impede the spread of infected cells. By administering vaccines, we aim to substantially reduce the number of new cases, thereby curbing the outbreak. This intervention reduces the number of unvaccinated individuals entering the susceptible population, denoted by $\Lambda_1(t)$. The second intervention $\Lambda_2(t)$, is to reduce the chances of contacts with infectious and chronic individuals. This will lower the successful contact rates relative to infectious and chronic individuals. The third control variable we considered is to encourage or maximize the treatment and isolation of TB infected patients by stepping up their rates and this intervention is denoted by $\Lambda_3(t)$. Lastly, the fourth control variable $\Lambda_4(t)$ is TB awareness. Where the control variables $\Lambda_1(t)$, $\Lambda_2(t)$, $\Lambda_3(t)$, and $\Lambda_4(t)$ are functions of time, making the control system time-dependent.

5.6 Numerical simulation

Our numerical simulations demonstrate that the model is effective in curbing the spread of TB, and appreciate the control strategies in complementing the model. Specifically, awareness campaigns, vaccination programs, for individuals prone to the disease are crucial in containing the spread of TB. If these strategies are implemented, our simulations shows that TB can be brought under control within a shorter timeframe considered in our model, as evident in Figures 4.3, 4.4, and 4.5. This emphasizes the importance of integrating mathematical modeling with public health interventions to effectively manage and eliminate TB in the population.
Ultimately, it is the government's duty to ensure that its citizens have access to adequate medical care and policies during an epidemic outbreak. While implementing comprehensive control strategies may be resource-intensive and potentially unrealistic, we recommend a pragmatic approach that prioritizes vaccination at birth, combined treatment and exclusion, and regular public awareness campaigns. By focusing on these key interventions, the government can effectively manage the spread of the disease, even with limited resources. This balanced approach will enable the government to fulfill its responsibility to protect its citizens' health and well-being during public health crises.

5. Conclusion

The SVLACTR model, with its sensitivity analysis, has provided valuable insights into the dynamics of Tuberculosis (TB), serving as a crucial tool in controlling its spread. Our findings emphasize the importance of prioritizing TB awareness and maximizing vaccination at birth to reach the critical vaccination threshold. Additionally, applying social distancing measures with contacted individuals help to minimize contact rates. In cases where vaccination fails and infection occurs, prompt anti-viral treatment and isolation are essential. By combining these strategies, we can effectively control the spread of TB, improve lives, and ultimately work towards eradicating the disease from the population. This integrated approach will play a vital role in mitigating the impact of TB and promoting public health.

APPENDIX

Proof of theorem 3.1

For F_1

$$|\frac{\partial F_1}{\partial S}| = |\xi\omega C| < \infty \qquad |\frac{\partial F_1}{\partial V}| = |-\theta\lambda C + \rho| < \infty$$

$$|\frac{\partial F_1}{\partial L}| = 0 < \infty \qquad |\frac{\partial F_1}{\partial A}| = |\omega S| < \infty$$

$$|\frac{\partial F_1}{\partial C}| = |-\theta\lambda V - \xi\omega S| < \infty \qquad |\frac{\partial F_1}{\partial T}| = 0 < \infty$$

$$|\frac{\partial F_1}{\partial R}| = 0 < \infty$$

For F_2

$$|\frac{\partial F_2}{\partial S}| = |\tau| < \infty \qquad |\frac{\partial F_2}{\partial V}| = |-(\mu+\rho+\omega(1-\xi)A)| < \infty$$

$$|\frac{\partial F_2}{\partial L}| = 0 < \infty \qquad |\frac{\partial F_2}{\partial A}| = |\omega(1-\xi)V| < \infty$$

$$|\frac{\partial F_2}{\partial C}| = |0 < \infty \qquad |\frac{\partial F_3}{\partial T}| = 0 < \infty$$

$$|\frac{\partial F_2}{\partial R}| = 0 < \infty$$

For F_3

$$|\frac{\partial F_3}{\partial S}| = |\omega A + \xi\omega C| < \infty \qquad |\frac{\partial F_3}{\partial V}| = |\omega(1-\xi)A| < \infty$$

$$|\frac{\partial F_3}{\partial L}| = |-\mu-\beta| < \infty \qquad |\frac{\partial F_3}{\partial A}| = |\omega+\omega(1-\xi)V| < \infty$$

$$|\frac{\partial F_3}{\partial C}| = |\xi\omega S| < \infty \qquad |\frac{\partial F_3}{\partial T}| = 0 < \infty$$

$$|\frac{\partial F_3}{\partial R}| = 0 < \infty$$

For F_4

$$|\frac{\partial F_4}{\partial S}| = 0 < \infty \qquad |\frac{\partial F_4}{\partial V}| = 0 < \infty$$

$$|\frac{\partial F_4}{\partial L}| = |\beta| < \infty \qquad |\frac{\partial F_4}{\partial A}| = |-\Upsilon + \mu - \Gamma - \Omega| < \infty$$

$$|\frac{\partial F_4}{\partial C}| = 0 < \infty \qquad |\frac{\partial F_4}{\partial T}| = 0 < \infty$$

$$|\frac{\partial F_4}{\partial R}| = 0 < \infty$$

For F_5

$$\left|\frac{\partial F_5}{\partial S}\right| = 0 < \infty \qquad \left|\frac{\partial F_5}{\partial V}\right| = |\theta\lambda C| < \infty$$

$$\left|\frac{\partial F_5}{\partial L}\right| = 0 < \infty \qquad \left|\frac{\partial F_5}{\partial A}\right| = |\theta\lambda V - (\Delta + \mu + \kappa)| < \infty$$

$$\left|\frac{\partial F_5}{\partial C}\right| = 0 < \infty \qquad \left|\frac{\partial F_5}{\partial T}\right| = 0 < \infty$$

$$\left|\frac{\partial F_5}{\partial R}\right| = 0 < \infty$$

For F_6

$$\left|\frac{\partial F_6}{\partial S}\right| = 0 < \infty \qquad \left|\frac{\partial F_6}{\partial V}\right| = 0 < \infty$$

$$\left|\frac{\partial F_6}{\partial L}\right| = 0 < \infty \qquad \left|\frac{\partial F_6}{\partial A}\right| = |\Omega| < \infty$$

$$\left|\frac{\partial F_6}{\partial C}\right| = |\Delta| < \infty \qquad \left|\frac{\partial F_6}{\partial T}\right| = |-\mu - \phi| < \infty$$

$$\left|\frac{\partial F_6}{\partial R}\right| = 0 < \infty$$

For F_7

$$\left|\frac{\partial F_7}{\partial S}\right| = 0 < \infty \qquad \left|\frac{\partial F_7}{\partial V}\right| = 0 < \infty$$

$$\left|\frac{\partial F_7}{\partial L}\right| = 0 < \infty \qquad \left|\frac{\partial F_7}{\partial A}\right| = |\Upsilon| < \infty$$

$$\left|\frac{\partial F_7}{\partial C}\right| = 0 < \infty \qquad \left|\frac{\partial F_7}{\partial T}\right| = |\Phi| < \infty$$

$$\left|\frac{\partial F_7}{\partial R}\right| = |-\mu| < \infty$$

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