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A Mathematical Approach for Assessing the Effect of Treatment Modalities on Tuberculosis Dynamics

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Abstract

In this paper, we examined the mechanism of Mycobacterium tuberculosis disease. Airborne tuberculosis is a disease that is putting the world's human beings at risk. This model carried out six-dimensional compartments represent the susceptible individual, latently exposed individual, individual infected at home, individual infected in at public place, individual infected at the hospital and the rate of recovered class. In this model, we described all compartments of the transmissible illness of Mycobacterium tuberculosis (MT) and how its spread in general population communities. The genus Mycobacterium is believed to have evolved more than 150 million years ago; to lower the prevalence of infectious cases in the community, our theoretical framework proposed a strategy for preventing and controlling tuberculosis infections. In our model tuberculosis is created that involves three types of medication:, individual treatment at home, individual treatment at a general ayurvedic health care provider and individual treatment at a hospital. We find out the basic reproduction number R_0. The disease-free population is globally asymptotically stable if $R_0 < 1$ and the equilibrium between endemics is globally asymptotically stable if $R_0 > 1$. Our model demonstrates the significant detrimental effect of home treatment and individual treatment at the hospital and Individual people who have been infected in public get cured by taking medication. The Lyapunov function is used to derive the TB disease in the community is globally asymptotically stable. We used a Jacobian matrix to examine local stability and diagonal stability. By using the Routh-Hurwitz criteria, we analysed the local stability of cubic polynomials. We derived sustainable and non-negatively feasible solutions. We used random values in MATLAB to simulate the result of the model.

Key words: Mathematical model, Disease free equilibrium point, feasible region, boundedness, Lyapunov function, Global stability, Routh-Hurwitz Stability Criterion.

Introduction

A comprehensive overview of tuberculosis comprises everything from the disease's beginnings to the invention of medications and therapeutic strategies aimed at decreasing and limiting its consequences. The names "White Plague," "phthisis" and "consumption" have all been applied to tuberculosis over history. Most experts agree that earlier, more primitive species within the same genus, Mycobacterium are the source of the infectious agent, Mycobacterium tuberculosis diseases. 2014 witnessed the reconstruction of a tuberculosis genome from remains found in southern Peru, and the outcomes of an exciting new DNA study demonstrated that the disease may have originated less than 6,000 years ago in humans. The first recorded case of tuberculosis occurred approximately 9,000 years ago, although researchers hypothesized that humans first contracted the disease across Africa approximately 5,000 years ago [1].

Human beings were infected with tuberculosis (TB). across commercial paths. Additionally, it propagates to African domesticated creatures like goats and dairy cattle. It is thought that the disease was contracted by sea lions that were genetically modified on the beaches of Africa and travelled throughout the Atlantic to the continent of South America. The very first individuals who contracted the virus would have been hunters. Investigation exploring the origins and evolution of the complex of Mycobacterium tuberculosis has revealed that an infectious agent specific to humans experienced a population bottleneck and was probably the most recent common ancestral species of the complex. According to the examination of mycobacterial interspersed repetitive units, the period of bottleneck can be estimated to be approximately 40,000 years ago. This time frame coincides with the era after Homo sapiens left Africa [2-3]. The Mycobacterium bovis lineage was also dated by this study of mycobacterial interspersed repetitive components that indicated it had begun spreading approximately 6,000 years ago, suggesting it could have something to do with early husbandry and domestication of animals. The bacteria can be found in Neolithic human remains. Additionally, a controversial discovery has raised the possibility that a 500,000-year-old Homo erectus fossil contains a record of tuberculosis lesions [4-8]. The World Health Organization (WHO) proclaimed a global tuberculosis epidemic in 1993 and pointed out that "poorly managed tuberculosis programs have the potential to make TB deadly. Almost three-quarters of new instances of the disease occur in twenty-two countries and the WHO has focused special attention and assistance on these countries. Guidelines for preventing the spread of tuberculosis in healthcare facilities in settings with limited resources have just been published by the World Health Organization. The regulations focus on low-cost ventilation methods (such as removing curtains or establishing appropriate exterior areas to accommodate people receiving healthcare or accompanying sick individuals) [9-11]. The emphasis is on primary district health care resources that do not have sufficient resources for implementing more expensive interventions like particular breathing apparatuses and negative-pressure separateness rooms, which should be considered only for referral infrastructure. Only in laboratory environments and at facilities that provide preventative measures for latent infection is tuberculin testing of the skin advised. While these recommendations are intended for nations with limited resources, they may also be relevant in certain situations within this nation. For instance, when the weather cooperates, individuals with alleged tuberculosis can wait outside in open spaces until public transport and medical attention can be organized in overcrowded inadequately funded homeless shelters. The overwhelming majority of adult individuals who contract tuberculosis are in their most productive years of employment. All different ages, though are vulnerable [12-16].

Regions with middle and low incomes are responsible for over 80 percent of cases and mortality. Worldwide, tuberculosis (TB) is a worldwide health concern. WHO's Southeast Asian Region was responsible for 46% of the total number of new cases of TB in 2022, with the African Region following in second with 23% and the Western Pacific Islands region with 18%. The 30 nations with the highest TB burden accounted for about 87% of all new cases of TB, with Bangladesh, China, the Democratic Republic of the Congo, India, Indonesia, Nigeria, Pakistan and the Philippines encompassing over two-thirds of the global total. The WHO End TB Strategy target of zero is far from reality, with approximately 50 percent of TB patients and their households facing catastrophic expenses 20% of total income from the household) for all costs (direct medical expenditures, non-medical subjects' expenditures, and indirect costs like employment losses). The team in question an increased risk of illness exists in those with immune systems that are weakened such as those with HIV, diabetes, malnutrition, or using tobacco products. 2.2 million new cases of tuberculosis (TB) worldwide in 2022 were linked to nutritional deficiency 0.89 million to infection with HIV 0.73 million to using alcohol disorders, 0.70 million to smoking, and 0.37 million to hypertension [17-22].

During the early 1900, tuberculosis was one of the most serious medical issues the UK was dealing with. There was a royal authority established in 1901. The task assigned to the commission of inquiry was to investigate possible human-animal tuberculosis connections. This study sought to ascertain whether tuberculosis in humans and animals was the same disease and whether infections in humans and animals were possible. Later, in 1919, the Commission adopted the UK Medical Research Council as its new name. Albert Schatz, Elizabeth Bugie and Selman Waksman identified the type of bacteria Streptomyces griseus in 1944, which is the source of streptomycin. Streptomycin was the first antibiotic to demonstrate efficacy against M. tuberculosis. The majority of people concur that this discovery marked the start of the tuberculosis epidemic in the modern era. Streptomycin was used in combination with para-amino salicylic acid, which was found in 1946, to stop the development of drug resistance to various formulations, thereby improving patient outcomes. A few years later, in 1952, a medication known as the first oral mycobactericidal medication was created, marking the beginning of the real revolution. When rifampin was introduced in the 1970 It accelerated the healing process and significantly decreased tuberculosis cases until the 1980 [23-33].

In the United States, there were about 8,916 cases of tuberculosis in 2019. To eradicate tuberculosis (TB) in the United States of America, it is imperative to uphold and enhance current prevention and treatment goals while stepping up efforts to detect and treat latent tuberculosis (LTBI) in those most at risk. The current U.S. population translates that into about 330 cases annually, falling short of the 1 case per million individual TB eradication criteria. Tuberculosis (TB) affects people worldwide and is a significant issue in our nation. Due to the disease's lack of borders, TB affects people in the US. TB can strike anyone at any time [28-30]. The We Are TB Patients Group and the National TB Controllers Society collaborated in concert with the CDC to showcase the stories of those who have been afflicted with the illness, as well as the work of experts in TB prevention and control [34-35].

Methodology

In this paper, we explored the investigation of the TB disease model. The analysis of tuberculosis can be classified into six stages Susceptible, exposed, infected at home, infected at public place, infected at hospital and the recovered cases. In this model we determined by the basic reproduction number R_0, If $R_0 < 1$ then the disease-free equilibrium is globally asymptotically stable, if $R_o > 1$ the endemic equilibrium is globally asymptotically stable. People are more infected in public when they travelled from hospital to home; they get re-infected again in public places. In our research, we established the presumption that there's a uniform combining of the general population and we found out disease free equilibrium point, the structure of ordinary differential equations applied to build a model of this frame. Sustainable domain Ω is delineated as the feasible region and the positively invariant system is examined. The Lyapunov function is used to define the equivalent method that is typically used to define the stability of intricate epidemiologic compartmental model. The boundedness condition is used to demonstrate the region of the disease model and we apply the exponential expansion condition to explore the negative state. We used the global stability condition to examine the infected region. The non-negative initial condition is used to find out the positive result of the TB reduced case. The Jacobian matrix is used to examine the local stability of the disease rate.

Model of the parameter

S : The number of suspectable individuals

E : Latent exposed individuals

I_1 : Symptomatic infectious patients receiving home treatment at beginning stage

I_2 : individual treatment at general ayurvedic health care provider

I_3 : Symptomatic infectious patients requiring treatment at hospital

R : The number of recovered individuals

α : Recruitment rate of TB Population

β : Rate of susceptible people to transferable to exposed stage

μ : Natural death rate of TB Population

γ : Exposed people get Individual infected at home by nature

π : Exposed class of individual get infection at public place

τ : Exposed class of individual get re-infection at hospital

ω : Rate of progression to I_1 class from I_2 class

ξ : Rate of progression to I_2 class from I_3 class.

θ : Rate of progression to I_3 class from I_2 class

σ : Rate of progression to I_2 class from I_1 class

δ : Rate of progression to I_2 class from R class

Λ : Rate of progression to I_3 class from R class

Model of the diagram

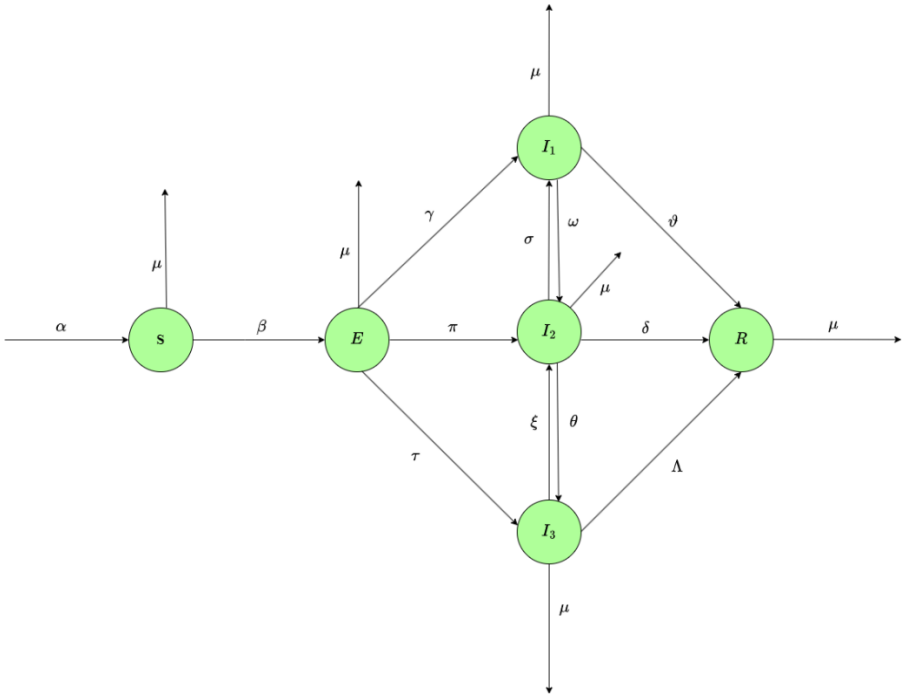


Fig.1.Flowchart of the TB

Model of the equation

There are six groups within the total size of the population of $N(t)$,which is composed of those who are susceptible S, latent exposed individuals E, symptomatic infectious patients receiving home treatment I_1, individual treatment at general ayurvedic health care provider I_2, symptomatic infectious patients requiring hospital treatment I_3 and patients who recovered (those who developed calcified points in their lungs) R. whereas

$N(t) = S(t) + E(t) + I_1(t) + I_2(t) + I_3$ (t)+ R (t)

The following transmission diagram (see Fig. 1) demonstrates the structure of the model.

$\frac{ds}{dt} = \alpha - \beta S - \mu S$

$$\frac{dE}{dt} = \beta S - \mu E - \gamma E - \pi E - \tau E$$

$$\frac{dI_1}{dt} = \gamma E + \omega I_2 - \sigma I_1 - \mu I_1 - \vartheta I_1 \quad (1)$$

$$\frac{dI_2}{dt} = \pi E + \sigma I_1 + \theta I_3 - \delta I_2 - \xi I_2 - \omega I_2 - \mu I_2$$

$$\frac{dI_3}{dt} = \tau E + \xi I_2 - \mu I_3 - \theta I_3 - \Lambda I_3$$

$$\frac{dR}{dt} = \vartheta I_1 + \delta I_2 + \Lambda I_3 - \mu R$$

Since R only appears in the fifth equation, it should be noted that every equation involving R has been separated from the others. As a result, we only need to think about (1)'s component.

$$\frac{ds}{dt} = \alpha - \beta S - \mu S$$

$$\frac{dE}{dt} = \beta S - \mu E - \gamma E - \pi E - \gamma E$$

$$\frac{dI_1}{dt} = \gamma E + \omega I_2 - \sigma I_1 - \mu I_1 - \vartheta I_1 \quad (2)$$

$$\frac{dI_2}{dt} = \pi E + \sigma I_1 + \theta I_3 - \delta I_2 - \xi I_2 - \omega I_2 - \mu I_2$$

$$\frac{dI_3}{dt} = \lambda E + \xi I_2 - \mu I_3 - \theta I_3 - \Lambda I_3$$

It is assumed that every parameter that exists is a constant. The percentage of retained information is denoted by α. For interaction with the I_1 and I_2 classes, the transmitted rates are ω, correspondingly. The natural death rate μ and the rate of progression from detected latent infection with tuberculosis (TB) to I_1 class γ *and* σ are given. The rate at which latent TB becomes identified and progresses to I_2 class is symbolized by σ. The progression rate from I_2 class is represented as ξ , ω, π and the progression rate from I_3 class represented as θ, τ. by ω. The rate at which active tuberculosis is successfully treated in the I_1 class. ϑ, δ and Λ indicates the disease controlled and recovered case.

Subject to the initial condition

$$S > 0, E > 0, I_1 > 0, I_2 > 0, I_3 > 0\ , R > 0 \quad (3)$$

Disease Free Equilibrium Point (DFE)

$$\frac{ds}{dt} = \alpha - \beta S - \mu S$$

$$E = I_1 = I_2 = I_3 = R = 0$$

$$\alpha - (\beta + \mu)S$$

$$S = \frac{\alpha}{\beta + \mu}$$

DFE = $\left(\frac{\alpha}{\beta+\mu}\right.$, 0 ,0,0,0,0) (3)

Endemic equilibrium point

The epidemiological point of equilibrium of the $S, E, I_1, I_2, I_3 and\ R$ of TB model disease can be calculated through the use of the substitution strategy to get solutions from the equations (1).

The endemic equilibria are given by

$$S^* = 0$$

$$E^* = \beta\left(\frac{\frac{\alpha}{\beta+\mu}}{\pi+\mu+\gamma+\tau}\right)$$

$$I_1{}^* = \frac{\gamma\left[\frac{\beta S}{\pi+\mu+\gamma+\tau}\right]+\omega I_2}{\sigma+\mu+\vartheta} \tag{4}$$

$$I_2{}^* = \frac{\pi\left[\frac{\beta S}{\pi+\mu+\gamma+\tau}\right]+\sigma\left[\frac{\gamma E+\omega I_2}{\sigma+\mu+\vartheta}\right]+\theta I_3}{\delta+\xi+\omega+\mu}$$

$$I_3{}^* = \frac{\lambda\left[\frac{\beta S}{\pi+\mu+\gamma+\tau}\right]}{\xi+\mu+\theta+\Lambda}$$

$$R^* = \frac{\vartheta\left[\frac{\gamma E+\omega I_2}{\sigma+\mu+\vartheta}\right]+\delta\left[\frac{\pi E+\sigma I_1+\theta I_3}{\delta+\xi+\omega+\mu}\right]+\frac{\omega[\lambda E]}{\xi+\mu+\theta+\Lambda}}{\mu}$$

Reproduction number

The next-generation matrix method will be used to determine R_0 after we have distinguished the classes in our model. One of the infectious virus classes is tuberculosis. The TB reproduction number R_0 will therefore be established.

I are the infected cases, we find out Reproduction number R_0.

Let $X = (\mathrm{S}, \mathrm{E}, I_1, I_2, I_3, \mathrm{R})$
F be the sign of TB increasing case
V be the sign of TB outgoing case

$$F = \begin{pmatrix} \pi E + \sigma I_1 + \theta I_3 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \qquad V = \begin{pmatrix} (\delta + \xi + \omega + \mu) I_2 \\ (\beta + \mu) S \\ (\mu + \gamma + \pi + \tau) E \\ (\sigma + \mu + \vartheta) I_1 \\ (\mu + \theta + \Lambda) I_3 \\ (\Lambda + \mu) R \end{pmatrix} \tag{5}$$

$$F = \pi E + \sigma I_1 + \theta I_3$$

$$V = \delta + \xi + \omega + \mu$$

$$FV^{-1} = \frac{\pi E + \sigma I_1 + \theta I_3}{\delta + \xi + \omega + \mu}$$

$$R_0 = \frac{\pi E + \sigma I_1 + \theta I_3}{\delta + \xi + \omega + \mu} \tag{6}$$

The fundamental reproduction number R_0 is obtained from equation (5). Therefore, in the disease-free case, the equilibrium points of the SE$I_1 I_2 I_3 R$ model is asymptotically stable.

Lemma.1. The sustainable domain Ω is delineated as follows:

$$\Omega = \{(\mathrm{S}, \mathrm{E}, I_1, I_2, I_3, R) \in R_+^6 : S + E + I_1 + I_2 + I_3 + R \leq \frac{\alpha}{\mu}\}$$

The system (1) is positively stable with initial condition $S(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, I_3(0) \geq, R(0) \geq 0$.

Proof. By summing the system (1) equations, we obtain:

$$\frac{dN}{dt} = \alpha - \mu N \tag{6}$$

The equation is as follows: $0 \leq N(t) \leq \frac{\alpha}{\mu} + N(0)e^{-\mu t}$, where $N(0)$ represents the starting number of individuals. The region is thus $N(t) \leq \frac{\alpha}{\mu}$ as $t \to \infty$.

$$\Omega = \{(\mathrm{S}, \mathrm{E}, I_1, I_2, I_3, R\ ) \in R_+^6 : S + E + I_1 + I_2 + I_3 + R \leq \frac{\alpha}{\mu}\} \tag{7}$$

is a set that is positively invariant for system (1). The changing effects of system (2) on the region Ω will be examined throughout this paper.

Existence and uniqueness of the solution

An independent system of first-order equations nonlinear ordinary differential equations describes the system (1). The matrix structure shown below can be used to rewrite it:

$$\dot{X}\ (t) = F(X)t, \qquad \text{were, } X(t) = \begin{pmatrix} S \\ E \\ I_1 \\ I_2 \\ I_3 \\ R \end{pmatrix} = \begin{pmatrix} x_1 \\ x_2 \\ x_3 \\ x_4 \\ x_5 \\ x_6 \end{pmatrix} \tag{8}$$

F is the function C^∞ on R_+^6 described by

$$F(X(t) = \begin{pmatrix} f_1(x_1, \ldots\ldots, x_6) \\ f_2(x_1, \ldots\ldots, x_6) \\ f_3(x_1, \ldots\ldots, x_6) \\ f_4(x_1, \ldots\ldots, x_6) \\ f_5(x_1, \ldots\ldots, x_6) \\ f_6(x_1, \ldots\ldots, x_6) \end{pmatrix} \tag{9}$$

$$F(X(t) = \begin{pmatrix} \alpha - ax_1 \\ \beta x_1 - bx_2 \\ \gamma x_2 + \omega x_4 - cx_3 \\ \pi x_2 + \sigma x_3 - dx_4 \\ \tau x_2 - ex_5 \\ \vartheta x_2 + \delta x_4 + \Lambda x_5 + \mu x_6 \end{pmatrix} \tag{10}$$

Here $a = \alpha - ax_1\ , b = \mu + \gamma + \pi + \tau\ ,\ c = \sigma + \mu + \vartheta, d = \vartheta + \delta + \xi + \omega + \mu$ (11)

and $e = \mu + \theta + \Lambda - \xi$

Furthermore, given the initial condition $(t_0 x_0) R \times R_+^6$ and the fact that $X(t) = (x_1(t), x_2(t), x_3(t), x_4(t), x_5(t), x_6(t))$ and that F is a class of c_1, we are able to deduce the existence and uniqueness of the maximum solution to the Cauchy problem related to the differential equation (1) locally Lipschitzian on R_+^6.

It follows that this solution is also of class C^∞ since F belongs to the same class.

Positivity of the solution
Adding up each of system (1)'s equations gives us

$N = M\alpha - \beta S - \mu S + \beta S - \mu E - \gamma E - \pi E - \tau E + \gamma E + \omega I_2 - \sigma I_1 - \mu I_1 - \vartheta I_1$
$+ \pi E + \sigma I_1 + \theta I_3 - \delta I_2 - \xi I_2 - \omega I_2 - \mu I_2 + \tau E + \xi I_2 - \mu I_3 - \Lambda I_3$

$N = \alpha - \mu N$ simplifying the derived representation of $\dot{N} = \alpha - \mu N$ with $N = (S + E + I_1 + I_2 + I_3 + R)$
Assuming that there is no infectious disease throughout the population as a whole, $N = S$ holds true. $E = I_1 = I_2 = I_3 = R = 0$ is inferred by this

By putting $\dot{N} = 0$, $\alpha - \mu N = 0$, as we have. We obtain

$N = \frac{\alpha}{\mu}$
According to the result (2), it is expected that the spread of tuberculosis will naturally lower N (that is, $N > \frac{\alpha}{\mu}$) in the population when there is no disease. Given the hypothetical system (1), its feasible region.

$\Omega = \{(S, E, I_1, I_2, I_3, R) \in R_+^6, 0 \le N \le \frac{\alpha}{\mu} + \in\}$ (12)

We obtain the following results where $\in$ is a constant with a positive value with respect to the simulation system (1) that represents the TB dynamics in the population.

Theorem.1
Globally asymptotically stable in Ω^*. is the endemic equilibrium state $E^*(S^*, E^*, I_1^*, I_2^*, I_3^*, R^*)$ if R_0>1.
The equivalent method that is typically used to define the stability of intricate epidemiologic compartmental models that is, by establishing the Lyapunov function "L" as follows has been applied here [19, 20, 21, and 23].

$L : \{(S, E, I_1, I_2, I_3, R) \in \Omega^*\} \to R$

where $\Omega^* = \{(S, E, I_1, I_2, I_3, R) \in R_+^6 : S(t), E(t), I_1(t), I_2(t), I_3(t), R(t) > 0\}$

$L = W_1\left\{S - S^* In\left(\frac{S}{S^*}\right)\right\} + W_2\left\{E - E^* In\left(\frac{E}{E^*}\right)\right\} + W_3\left\{I_1 - I_1^* In\left(\frac{I_1}{I_1^*}\right)\right\} + W_4\left\{I_2 - I_2^* In\left(\frac{I_2}{I_2^*}\right)\right\} + W_5\left\{I_3 - I_3^* In\left(\frac{I_3}{I_3^*}\right)\right\} + W_6\{R - R^* In\left(\frac{R}{R^*}\right)\}$ (13)

In this case, the non-negative constants in Ω are W_1, W_2, W_3, W_4, W_5, and W_6. Now, if we take the Lyapunov function L 's time derivative of the function, we obtain that

$\frac{dL}{dt} = W_1\left(\frac{S - S^*}{S}\right)\frac{dS}{dt} + W_2\left(\frac{E - E^*}{S}\right)\frac{dE}{dt} + W_3\left(\frac{I_1 - I_1^*}{I_1}\right)\frac{dI_1}{dt} + W_4\left(\frac{I_2 - I_2^*}{I_2}\right)\frac{dI_2}{dt} + W_5\left(\frac{I_3 - I_3^*}{I_3}\right)\frac{dI_3}{dt} + W_6\left(\frac{R - R^*}{R}\right)\frac{dR}{dt}$

$\frac{dL}{dt} = W_1\left(\frac{S - S^*}{S}\right)\{\alpha - \beta S - \mu S\} + W_2\left(\frac{E - E^*}{S}\right)\{\beta S - \mu E - \gamma E - \pi E - \tau E\} + W_3\left(\frac{I_1 - I_1^*}{I_1}\right)\{\gamma E + \omega I_2 - \sigma I_1 - \mu I_1 - \vartheta I_1\} + W_4\left(\frac{I_2 - I_2^*}{I_2}\right)\{\pi E + \sigma I_1 + \theta I_3 - \delta I_2 - \xi I_2 - \omega I_2 - \mu I_2\} + W_5\left(\frac{I_3 - I_3^*}{I_3}\right)\{\tau E + \xi I_2 - \mu I_3 - \Lambda I_3\} + W_6\left(\frac{R - R^*}{R}\right)\{\vartheta I_1 + \delta I_2 + \Lambda I_3 - \mu I_3\}$
(14)

At an endemic equilibrium point we have that

$\frac{dL}{dt} = W_1\left(\frac{S - S^*}{S}\right)\left[\frac{\beta + \mu}{S} - \frac{\beta + \mu}{S}\right] + W_2\left(\frac{E - E^*}{S}\right)\left[\frac{\mu + \gamma + \pi + \tau}{E} - \frac{\mu + \gamma + \pi + \tau}{E}\right] + W_3\left(\frac{I_1 - I_1^*}{I_1}\right)[(\frac{\sigma + \mu + \vartheta}{I_1} - \frac{\sigma + \mu + \vartheta}{I_1})]$

$$+ W_4\left(\frac{I_2-I_2^*}{I_2}\right)\left[\frac{(\sigma+\mu+\vartheta)}{I_2}-\frac{(\sigma+\mu+\vartheta)}{I_2}\right]+W_5\left(\frac{I_3-I_3^*}{I_3}\right)\left[\frac{(\mu+\Lambda)}{I_3}-\frac{(\mu+\Lambda)}{I_3}\right]+W_6\left(\frac{R-R^*}{R}\right)\left[\left(\frac{\mu}{R}-\frac{\mu}{R}\right)\right] \quad (15)$$

$\frac{dL}{dt} = W_1\left(\frac{S-S^*}{S}\right)^2 + M(\mathrm{S},\mathrm{E},I_1,I_2,I_3,R)$ ~According to the method that was used [21, 22, 23], indicates that the function $M(\mathrm{S},\mathrm{E},I_1,I_2,I_3,R)$ is non-positive. Particularly, $M \leq 0$for each $\mathrm{S},\mathrm{E},I_1,I_2,I_3,R > 0$, significance that $\frac{dL}{dt} \leq 0$ and $\frac{dL}{dt} = 0$.

When $\mathrm{S} = S^*, \mathrm{S} = S^*, \mathrm{E} = E^*, I_1 = {I_1}^*, I_2 = {I_2}^*, I_3 = {I_3}^*, R = R^*$

Therefore, the predominant equilibrium point, or singleton $\mathbb{E}^*$, is the largest compact persistent set in the domain for which$\frac{dL}{dt} = 0$. Thus, assuming $R_0 > 1$, we can deduce that $\mathbb{E}^*$ is asymptotically stable worldwide in Ω^* by applying the LaSalle invariance hypothesis [24, 25].

Theorem.2

When combined with the initial condition (3) the resulting solution set of the proposed model (1), which is $\{\mathrm{S}(\mathrm{t}), \mathrm{E}(\mathrm{t}), I_1(t), I_2(t), I_3(t), R(t)\}$, is non-negative for $t > 0$.

Proof

The study's recommendation (32) states that we analyse the first equation while taking equation (1)'s non-linear system into consideration.

$$\frac{ds}{dt} = \alpha - \beta S - \mu S \quad (16)$$

Which means that

$$\frac{ds}{dt} \geq -(\beta - \mu)S \quad (17)$$

We obtain $\mathrm{S}(\mathrm{t}) \geq S(0)e^{-(\beta-\mu)t}$ through integrating the equation (17) and applying the exponential expansion condition. This indicates that $\mathrm{S}(\mathrm{t}) \geq 0$..

Theorem.3

Given any non-negative initial condition, the resultant solution$(\mathrm{S}(\mathrm{t}), \mathrm{E}(\mathrm{t}), I_1(t), I_2(t), I_3(t), R(t)) \in R_+^6$ of the system (1) is optimistic at any time $t \geq 0$.

Proof

$$\frac{dS}{dt} \mid S = 0 \ , \ = \ \alpha \geq 0$$

$$\frac{dE}{dt} \mid E = 0 \ , \ = \ \beta S \ \geq 0$$

$$\frac{dI_1}{dt} \mid I_1 = 0 \ , = \ \gamma E + \omega I_2 \geq 0 \quad (18)$$

$$\frac{dI_2}{dt} \mid I_2 = 0 \ , = \ \pi E + \sigma I_1 + \theta I_3 \geq 0$$

$$\frac{dI_3}{dt} \mid I_3 = 0 \ , = \ \tau E + \xi I_2 \geq 0$$

$$\frac{dR}{dt} \mid R = 0 \ , \ = \vartheta I_1 + \delta I_2 + \Lambda I_3 \geq 0$$

As desired

Local stability

Two well-known theorems regarding local stability are presented in this section. Once more, take into consideration the system (1) as an expression of $U; V; W; X; Y; Z$ as follows.

$$U = \alpha - \beta S - \mu S$$
$$V = \beta S - \mu E - \gamma E - \pi E - \tau E$$
$$W = \gamma E + \omega I_2 - \sigma I_1 - \mu I_1 - \vartheta I_1$$
$$X = \pi E + \sigma I_1 + \theta I_3 - \delta I_2 - \xi I_2 - \omega I_2 - \mu I_2 \quad (19)$$
$$Y = \tau E + \xi I_2 - \mu I_3 - \theta I_3 - \Lambda I_3$$
$$Z = \vartheta I_1 + \delta I_2 + \Lambda I_3 - \mu I_3$$

The following are the partial derivates of the function with respect to state variables, such as

$$\frac{\partial U}{\partial S} = -(\beta + \mu) ; \frac{\partial U}{\partial E} = 0 ; \frac{\partial U}{\partial I_1} = 0 ; \frac{\partial U}{\partial I_2} = 0 ; \frac{\partial U}{\partial I_3} = 0 ; \frac{\partial U}{\partial R} = 0$$
$$\frac{\partial V}{\partial E} = -(\mu + \gamma + \pi + \tau) ; \frac{\partial V}{\partial S} = \beta ; \frac{\partial V}{\partial I_1} = 0 ; \frac{\partial V}{\partial I_2} = 0 ; \frac{\partial V}{\partial I_3} = 0 ; \frac{\partial V}{\partial R} = 0$$
$$\frac{\partial W}{\partial I_1} = -(\sigma + \mu I_1 + \vartheta I_1) ; \frac{\partial W}{\partial S} = 0 ; \frac{\partial W}{\partial E} = \gamma ; \frac{\partial W}{\partial I_2} = \omega ; \frac{\partial W}{\partial I_3} = 0 ; \frac{\partial W}{\partial R} = 0$$
$$\frac{\partial X}{\partial I_2} = -(\delta I_2 + \xi I_2 + \omega I_2 + \mu) ; \frac{\partial X}{\partial S} = 0 ; \frac{\partial X}{\partial E} = \pi ; \frac{\partial X}{\partial I_1} = 0 ; \frac{\partial X}{\partial I_3} = \theta ; \frac{\partial X}{\partial R} = 0 \quad (20)$$
$$\frac{\partial Y}{\partial I_3} = -(\theta + \mu + \Lambda); \frac{\partial Y}{\partial S} = 0 ; \frac{\partial Y}{\partial E} = \tau ; \frac{\partial Y}{\partial I_1} = 0 ; \frac{\partial Y}{\partial I_2} = \xi ; \frac{\partial Y}{\partial R} = 0$$
$$\frac{\partial Z}{\partial R} = -\mu ; \frac{\partial Z}{\partial S} = 0 ; \frac{\partial Z}{\partial E} = 0 ; \frac{\partial Z}{\partial I_1} = 0 ; \frac{\partial Z}{\partial I_2} = 0 ; \frac{\partial Z}{\partial I_3} = 0$$

Therefore, the model's Jacobian matrix has the following structure:

$$J = \begin{bmatrix} -(\beta+\mu) & 0 & 0 & 0 & 0 & 0 \\ \beta & -(\mu+\gamma+\pi+\tau) & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\sigma+\mu I_1+\vartheta I_1) & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\delta I_2+\xi I_2+\omega I_2+\mu) & \theta & 0 \\ 0 & \tau & 0 & \xi & -(\theta+\mu+\Lambda) & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu \end{bmatrix} \quad (21)$$

Lemma.2

The matrix J is Volterra-Lyapunov stable.

$$A = J = \begin{bmatrix} -(\beta+\mu)+\lambda & 0 & 0 & 0 & 0 & 0 \\ \beta & -(\mu+\gamma+\pi+\tau)+\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\sigma+\mu I_1+\vartheta I_1)+\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\delta I_2+\xi I_2+\omega I_2+\mu)+\lambda & \theta & 0 \\ 0 & \tau & 0 & \xi & -(\theta+\mu+\Lambda)+\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu+\lambda \end{bmatrix} \tag{22}$$

$$D = -J = \begin{bmatrix} (\beta+\mu)+\lambda & 0 & 0 & 0 & 0 & 0 \\ \beta & (\mu+\gamma+\pi+\tau)+\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & (\sigma+\mu I_1+\vartheta I_1)+\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & (\delta I_2+\xi I_2+\omega I_2+\mu)+\lambda & \theta & 0 \\ 0 & \tau & 0 & \xi & (\theta+\mu+\Lambda)+\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu+\lambda \end{bmatrix} \tag{23}$$

$$D_1 = -J = \begin{bmatrix} (\beta+\mu)+\lambda & 0 & 0 & 0 & 0 \\ \beta & (\mu+\gamma+\pi+\tau)+\lambda & 0 & 0 & 0 \\ 0 & 0 & (\sigma+\mu I_1+\vartheta I_1)+\lambda & 0 & 0 \\ 0 & 0 & 0 & (\delta I_2+\xi I_2+\omega I_2+\mu)+\lambda & \theta \\ 0 & 0 & 0 & \xi & (\theta+\mu+\Lambda)+\lambda \end{bmatrix} \tag{24}$$

Clearly $-A_{11} = (\beta+\mu)+\lambda > 0$ Let us consider $D_1 = -J$ be a 5×5 matrices obtained by deleting last row and column in equation (23)

$$\widetilde{D} = D_2 = -J = \begin{bmatrix} (\beta+\mu)+\lambda & 0 & 0 & 0 \\ \beta & (\mu+\gamma+\pi+\tau)+\lambda & 0 & 0 \\ 0 & 0 & (\sigma+\mu I_1+\vartheta I_1)+\lambda & 0 \\ 0 & 0 & 0 & (\delta I_2+\xi I_2+\omega I_2+\mu)+\lambda \end{bmatrix} \tag{25}$$

Clearly $-A_{11} = (\beta+\mu)+\lambda > 0$ Let us consider $D_2 = -J$ be a 4×4 matrices obtained by deleting last row and column in equation (24).

Lemma.3

The matrix $\widetilde{D}$ is diagonal stable.

Proof

Step.1: $D_{44} > 0$

Step.2: We shall prove the matrix D is diagonal stable.

$$\widetilde{D} = J = \begin{bmatrix} (\beta+\mu)+\lambda & 0 & 0 \\ \beta & (\mu+\gamma+\pi+\tau)+\lambda & 0 \\ 0 & 0 & (\sigma+\mu I_1+\vartheta I_1)+\lambda \end{bmatrix} \tag{26}$$

Clearly, $\widetilde{D}_{11} > 0, \widetilde{D}_{12} > 0, \widetilde{D}_{13} > 0$

It remains to show that det $(D_3) > 0$

$$\det(\widetilde{D}) = (\beta+\mu)+\lambda)(\mu+\gamma+\pi+\tau)+\lambda)\,(\sigma+\mu I_1+\vartheta I_1)+\lambda)(\delta I_2+\xi I_2+\omega I_2+\mu)+\lambda) \quad (27)$$

Therefore, $\widetilde{D}$ is diagonal stable

We now show that $\widetilde{D}^{-1}$ is diagonally stable.

$$\widetilde{D}^{-1} = J = \begin{bmatrix} \frac{1}{(\beta+\mu)+\lambda} & 0 & 0 \\ \frac{(\sigma+\mu I_1+\vartheta I_1)+\lambda}{((\beta+\mu)+\lambda)((\mu+\gamma+\pi+\tau)+\lambda)} & \frac{1}{(\mu+\gamma+\pi+\tau)+\lambda} & 0 \\ 0 & 0 & \frac{1}{(\sigma+\mu I_1+\vartheta I_1)+\lambda} \end{bmatrix} \quad (28)$$

$\widetilde{D}^{-1}{}_{11} > 0, \widetilde{D}^{-1}{}_{12} > 0, \widetilde{D}^{-1}{}_{13} > 0$

Therefor $\widetilde{D}^{-1}$ is diagonal stable.

(Routh-Hurwitz Stability Criterion)

Theorem 4

The EE of the TB model (25) is locally asymptotically stable if $R_o > 1$.

Proof

Proof. From the linearization, we obtain the following characteristic equation for EE

$$a_o\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \quad (29)$$

$a_0 = 1$

$a_1 = (\beta+\mu)+(\mu+\gamma+\pi+\tau)+R_0$

$a_2 = (\beta+\mu)+(\mu+\gamma+\pi+\tau)+R_0[(\beta+\mu)+(\mu+\gamma+\pi+\tau)]$ (30)

$a_3 = (\beta+\mu)+(\mu+\gamma+\pi+\tau)(R_0)$

By using the Routh-Hurwitz criteria, we can analyse the local stability of EE. The cubic polynomial in equation (29) has the negative real parts if all the coefficients in equation (30) are positive and det $(H_i) > 0, \forall_i =$ = 1;2; 3. So, we define three matrices H as follows,

$$H_1 = [a_1]$$

$$H_2 = \begin{bmatrix} a_1 & 0 \\ 0 & a_2 \end{bmatrix}$$

$$H_3 = \begin{bmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{bmatrix}$$

where a_1 , a_2 , a_3 are coefficient that written in equation (29). Then, we know that det$(H_1) = a_1 > 0$ and det $(H_2) = a_1a_1 > 0$ because a_1 , $a_1 > 0$. Meanwhile, det (H_3) is positive if $R_o > 1$. Hence, the EE of model (30) is locally asymptotically stable if $R_o > 1$.

Numerical simulation

The numerical co-disease model simulations that we ran aided in demonstrating the results of the qualitative examination. The mathematical representation of the co-disease simulation was done using variables and parameters. To illustrate our assumptions, we took some of the values for parameters as given. The simulation's numerical results

have been implemented using MATLAB for programming the parameters and the compartment characteristics. Using MATLAB, numerical estimations of the major outcomes of the theoretical TB disease models have been carried out. One of the primary objectives of our investigation is to find out how prescription medications affect people who have tuberculosis

.

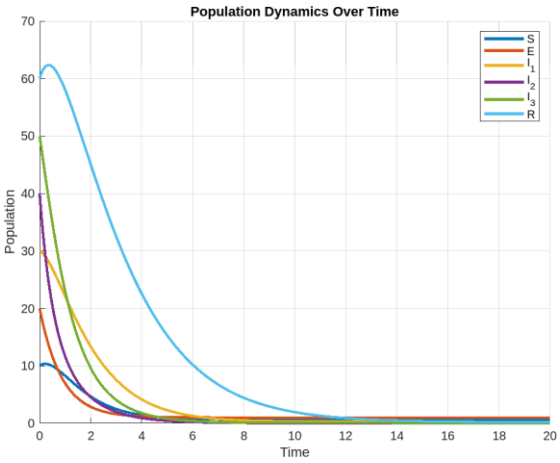


Fig.2 Population Dynamics Over Time $\alpha = 1.172$

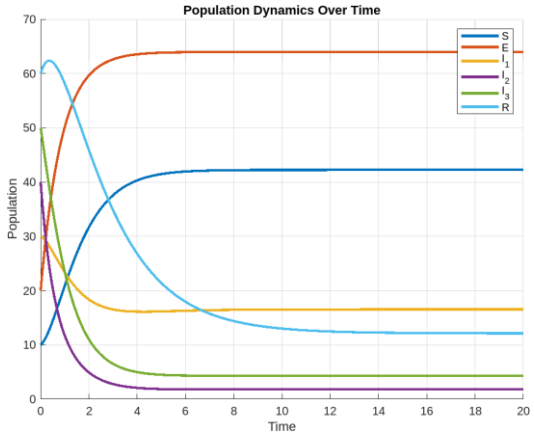
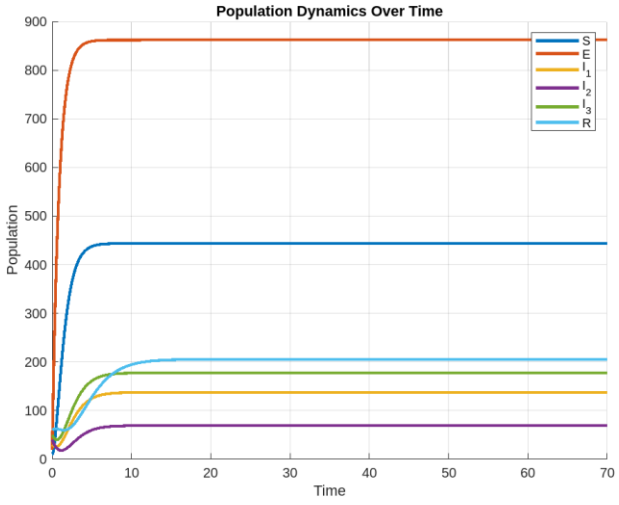


Fig.2 Population Dynamics Over Time $\alpha = 74.203$



α =Fig.3 Population Dynamics Over Time $\alpha = 1000$

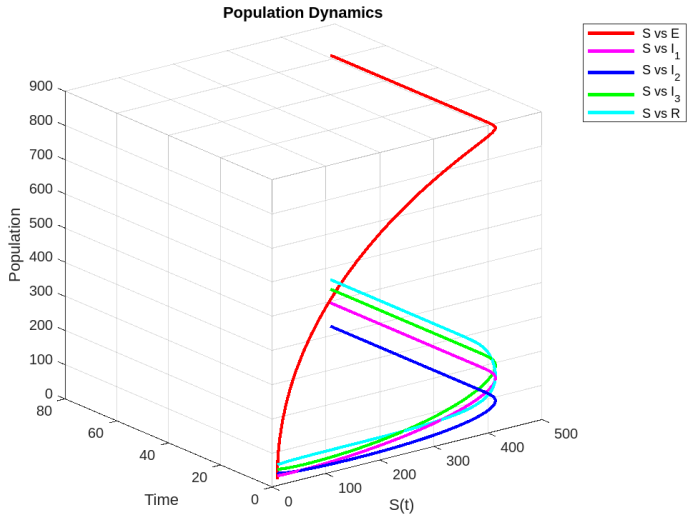


Fig.4 TB Prevalence across Temporal horizons

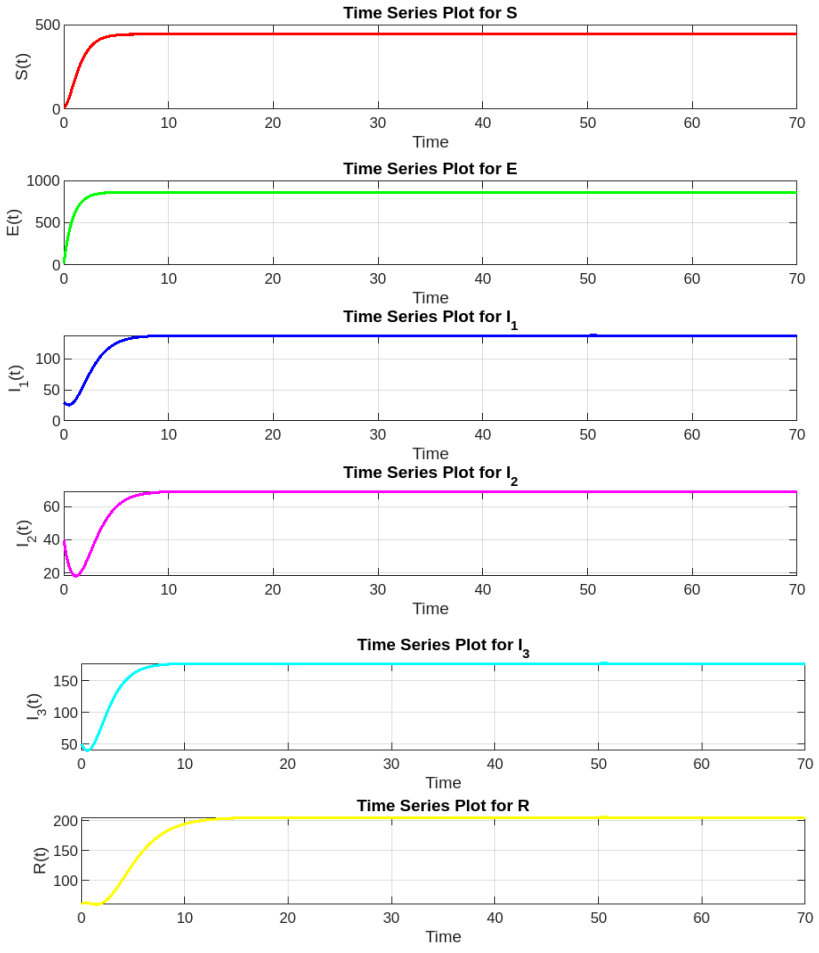


Fig.5 Temporal exploration: Time series representation of all stages

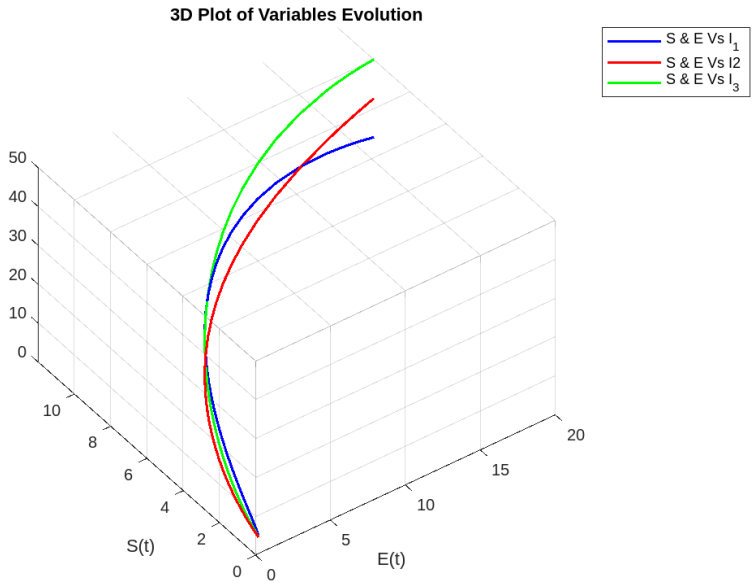


Fig.6 Prevalence across temporal vertical zone

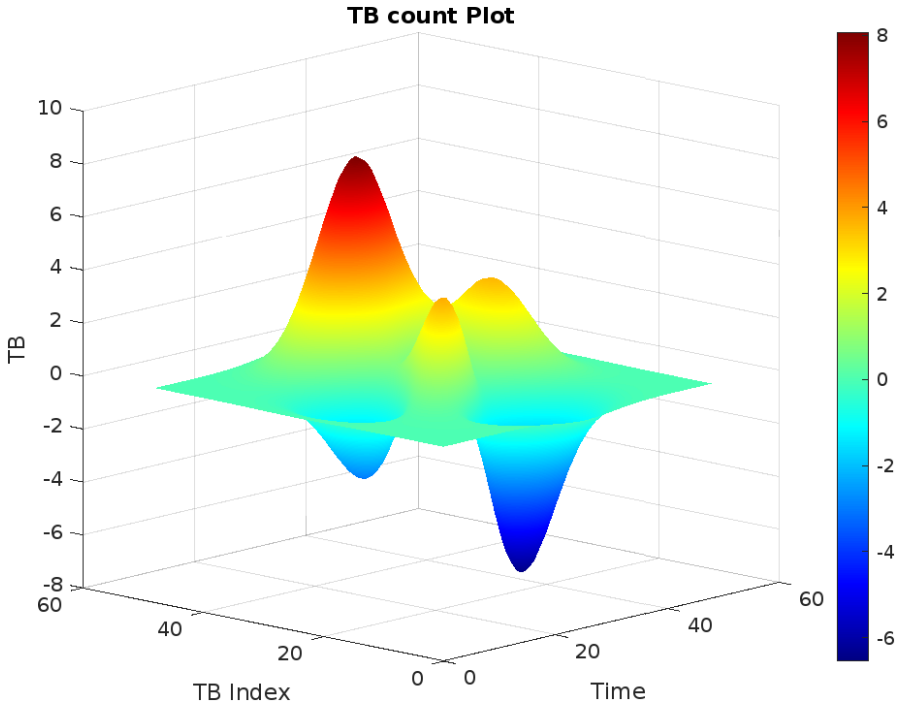


Fig.7 TB incidence graph: Tracking the numbers

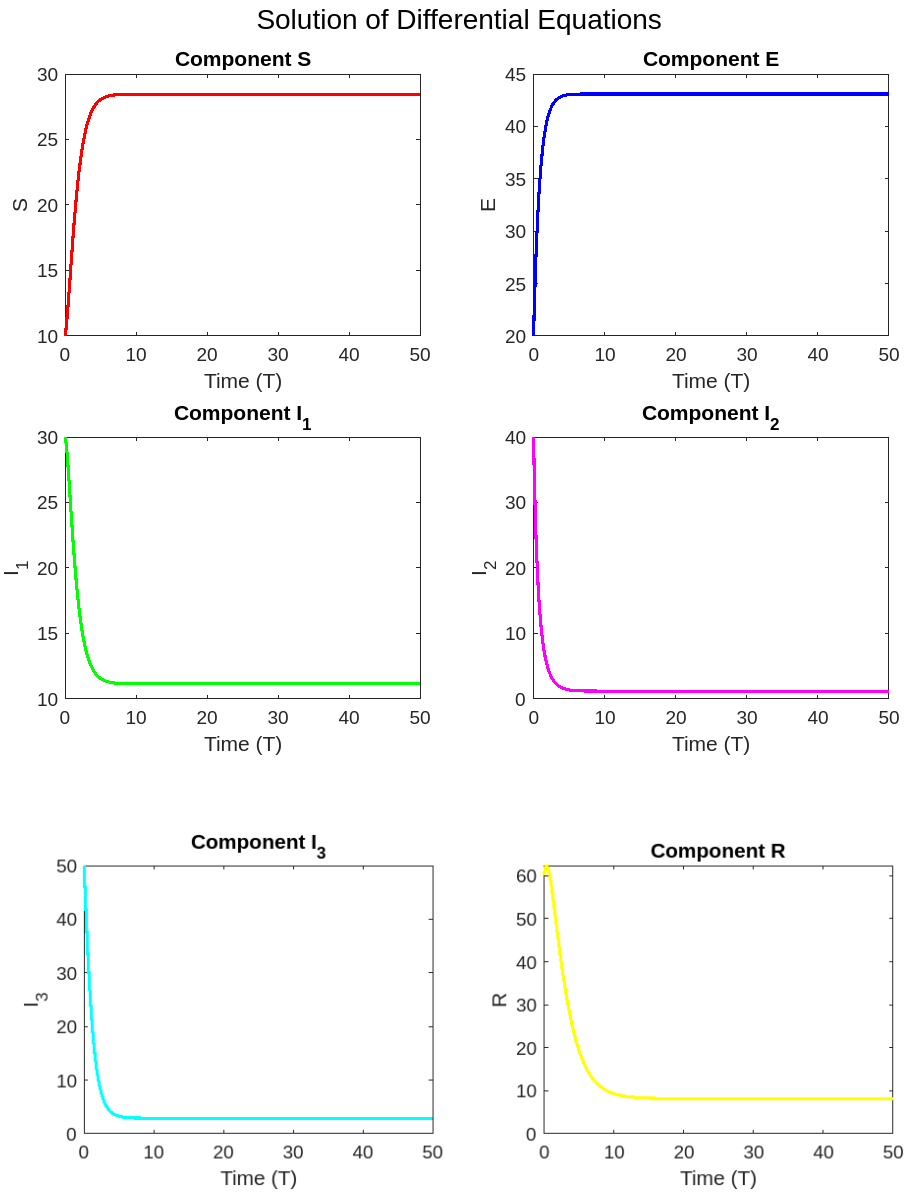


Fig.8 Visualizing the dynamics: Differential equation solutions

In Fig.1 We used random values in MATLAB to find out the disease-free equilibrium is globally asymptotically stable when $\alpha = 1.172;\ \ \vartheta = 0.24;\ \sigma = 0.02;\ \theta = 0.4;\ \ \gamma = 0.30;\ \omega = 0.50;\ \pi = 0.2;\ \beta = 0.7;\ \mu = 0.46;\ \Lambda = 0.49;\ \tau = 0.10\ ;\ \xi = 0.9;\ \ \delta = 0.4$

In Fig.2 We used random values in MATLAB the disease-free equilibrium is globally asymptotically stable when $\alpha = 74.203\ ;\ \ \vartheta = 0.24;\ \sigma = 0.02;\ \theta = 0.4;\ \ \gamma = 0.30;\ \omega = 0.50;\ \pi = 0.2;\ \beta = 0.7;\ \mu = 0.46;\ \Lambda = 0.49;\ \tau = 0.10;\ \xi = 0.9;\ \ \delta = 0.4$

In Fig.3-4 We used random data in MATLAB to find out the disease of TB is globally asymptotically stable when $\alpha = 1000$; $\vartheta = 0.24$; $\sigma = 0.02$; $\theta = 0.4$; $\gamma = 0.30$; $\omega = 0.50$; $\pi = 0.2$; $\beta = 0.7$; $\mu = 0.46$; $\Lambda = 0.49$; $\tau = 0.40$; $\xi = 0.9$; $\delta = 0.4$

In Fig.5-6 We used random data in MATLAB to find out the infection of TB is globally asymptotically stable when $\alpha = 1.172$; $\vartheta = 0.24$; $\sigma = 0.02$; $\theta = 0.4$; $\gamma = 0.30$; $\omega = 0.50$; $\pi = 0.2$; $\beta = 0.7$; $\mu = 0.46$; $\Lambda = 0.49$; $\tau = 0.10$; $\xi = 0.9$; $\delta = 0.4$

In Fig. Figures 7-8 show that the TB count plot and visualizing the dynamics of the TB people iv varies compartments by using Differential equation solutions based on the parameter values. $\alpha = 50$; $\vartheta = 0.24$; $\sigma = 0.02$; $\theta = 0.4$; $\gamma = 0.30$; $\omega = 0.50$; $\pi = 0.2$; $\beta = 0.7$; $\mu = 0.46$; $\Lambda = 0.49$; $\tau = 0.10$; $\xi = 0.9$; $\delta = 0.4$

Conclusion

In this paper, we analysed the TB disease model in six six-compartment stages, this model has found positivity of the presence of tuberculosis. A tuberculosis model was analysing to show the dynamics of the TB disease, we found out the basic reproduction number R_0, If $R_0 < 1$, then the disease-free equilibrium is globally asymptotically stable. At any time (t) the tuberculosis disease exists in the region, we used the Lyapunov stability function to control the disease rate, the TB population is globally asymptotically stable. We used a Jacobian matrix to examine the local stability and diagonal stability. We derived sustainable and non-negatively feasible solutions. By using the Routh-Hurwitz criteria, we analysed the local stability of endemic equilibrium. We used MATLAB to simulate the result of the model. Numerical simulation was analysed and the symptomatic infectious patients requiring hospital treatment, symptomatic infectious patients receiving home treatment and Individuals infected at receiving treatment public places, these three compartments were cured and recovered by proper medication.

The process of analysing and assessing each stage of the patient's case study is included in the interpretation of suggested controls. This helps to rectify the earlier stages of the disease's evaluation and supports the corrective actions of the disease-controlled system. To preserve control because maintaining control of the disease is must be the main goal, it is a dynamic function. Depending on the patient's condition we can virtually control the tuberculosis disease after some medical case studies. In the future, we want to focus on developing drug-resistant and chemotherapy procedures in order to manage tuberculosis in the general population.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Data Availability Statement:

The data used to support the findings of this study are included within the article.

Conflicts of Interest:

The writers declare that no conflicts of interest exist.

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