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Stability analysis of a dynamical model of tuberculosis with incomplete treatment

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Abstract

A simple deterministic epidemic model for tuberculosis is addressed in this article. The impact of effective contact rate, treatment rate, and incomplete treatment versus efficient treatment is investigated. We also analyze the asymptotic behavior, spread, and possible eradication of the TB infection. It is observed that the disease transmission dynamics is characterized by the basic reproduction ratio $\Re_0$; if $\Re_0 < 1$, there is only a disease-free equilibrium which is both locally and globally asymptotically stable. Moreover, for $\Re_0 > 1$, a unique positive endemic equilibrium exists which is globally asymptotically stable. The global stability of the equilibria is shown via Lyapunov function. It is also obtained that incomplete treatment of TB causes increase in disease infection while efficient treatment results in a reduction in TB. Finally, for the estimated parameters, some numerical simulations are performed to verify the analytical results. These numerical results indicate that decrease in the effective contact rate λ and increase in the treatment rate γ play a significant role in the TB infection control.

Keywords: Tuberculosis; Deterministic model; Basic reproduction ratio; Local stability; Global stability; Lyapunov function

1 Introduction

Tuberculosis (TB) is an infectious disease caused by mycobacterium tuberculosis, which most frequently affects the lungs (pulmonary TB). However, in rare conditions, brain, kidney, skin, spinal cord, and central nervous system are also affected. It is an ancient disease whose existence evidences are found in the relics of ancient Egypt, China, and India [1]. TB is a communicable disease and can be spread through air by active pulmonary TB patients that propel the TB bacteria into the atmosphere by spit, cough, or sneezing. An individual needs to breathe in few TB bacteria to become infected. Approximately 10 percent of infected individuals develop active TB disease, while the remaining 90 percent remain in their latent stage. Latently infected individuals do not transmit TB infection as they are asymptomatic. Moreover, individuals with immune compromised diseases (HIV and diabetic patients) are at higher risk of TB infection. The symptoms and signs of active TB include: coughing that lasts for about two weeks, fever, chest pain, weight loss, fatigue, night sweats. TB is a curable infectious disease using drug therapy [2]; however, inappropriate or incomplete treatments can cause a severe resistant form of TB. Although BCG

vaccine is available to control TB, its effectiveness to prevent the disease is still controversial, with a reported efficacy rate of 50 percent [3].

In spite of significant work done in TB control and treatment, still one-third of population across the world is latently infected, producing a source for active TB in the future. Globally, in 2018 about 10 million TB new cases were reported, causing more than 1.4 million deaths. Besides this, approximately 1.2 million TB patients died with HIV-negative and 0.25 million people died with HIV-positive in 2018. Most of the deaths were reported in middle and low income countries like India, Nigeria, Indonesia, Pakistan, Philippines, and South Africa. Southeast Asia contributed approximately 44 percent incidence of TB worldwide [4].

Mathematical modeling has a great role in predicting the transmission dynamics and construction of control strategies for a disease. These models also enhance our knowledge about the basic transmission dynamics of the diseases. Distinct models for TB transmission dynamics have been developed [5–9] either to provide strategies for TB spread/control or to evaluate its rampaged effect. In 1962 the first model for TB transmission was formulated by Waaler and Anderson[5]. Okuonghae [9] presented a model to study the impact of heterogeneity in transmission of TB. Yang et al.[10] studied TB transmission models in which they investigated incomplete treatment and studied the role of slow and fast transmission on the TB infection. Zhang and Feng [11] proposed a TB model to study its spread in a host population incorporating incomplete treatment and isolation. Trauera et al. [12] proposed a model investigating the transmission of TB infection in badly endemic areas of Asia-Pacific. They studied the impact of partial and temporary vaccine efficacy. Liu and Zhang [13] developed a TB model to study the role of treatment and vaccination on TB transmission. Their results showed that TB can be controlled if treatment rate or vaccine rate or its efficacy maintain a specific value of threshold.

Keeping the above-mentioned literature and discussion in mind, in our present work we are going to develop a TB transmission model in order to study its dynamics. On the basis of this model the role of efficient treatment and incomplete treatment will be studied. Further, to show the stability of equilibrium states, the Lyapunov–LaSalle method will be used as in [14]. In this connection, the total host population is separated into five disjoint groups, namely susceptible $S(t)$, exposed stage $E(t)$, active TB stage $I(t)$, which is also termed infectious, treated population $T(t)$, and recovered population $R(t)$. The parameter π shows the recruitment rate which occurs in the susceptible class only. Susceptible individuals acquire the TB infection from infectious individuals as a result of close contact with each other at a rate $\lambda S(t)(I(t) + \beta T(t))$, where λ is the effective contact rate, while $\beta(0 \leq \beta < 1)$ shows reduction in infectiousness depending on the stage of treatment. The natural death rate is η, while the disease-related death rates are δ_1 and δ_2 respectively for infectious $I(t)$ and treated $T(t)$ classes with $\delta_1 > \delta_2$. α expresses the transfer rate from class $E(t)$ to class $I(t)$. Parameter γ shows the treatment rate for infectious class. The individuals leave the treated class at a rate θ, a fraction $p\theta T$ of which enter the recovered class due to efficient treatment and $(1 - p)\theta T$ reenter the exposed class due to incomplete or inappropriate treatment. The parameter p $(0 < p \leq 1)$ reflects the part of efficient treatment. It is assumed that at the exposed stage individuals do not cause infection and the treated individuals can cause infection i.e. can transmit the TB infection. The aforementioned assumptions about the transmission model and the transfer process among distinct classes

lead to the following system of autonomous differential equations:

$$\begin{cases} \frac{dS}{dt} = \pi - \lambda S(I + \beta T) - \eta S, \\ \frac{dE}{dt} = \lambda S(I + \beta T) - (\eta + \alpha)E + (1 - p)\theta T, \\ \frac{dI}{dt} = \alpha E - (\eta + \delta_1 + \gamma)I, \\ \frac{dT}{dt} = \gamma I - (\eta + \delta_2 + \theta)T, \\ \frac{dR}{dt} = p\theta T - \eta R. \end{cases} \tag{1}$$

To write model (1) in a simple form, we use the following substitutions:

$$h_1 = \eta + \alpha, \qquad h_2 = \eta + \delta_1 + \gamma, \qquad h_3 = \eta + \delta_2 + \theta.$$

Thus, model (1) will take the form

$$\begin{cases} \frac{dS}{dt} = \pi - \lambda S(I + \beta T) - \eta S, \\ \frac{dE}{dt} = \lambda S(I + \beta T) - h_1 E + (1 - p)\theta T, \\ \frac{dI}{dt} = \alpha E - h_2 I, \\ \frac{dT}{dt} = \gamma I - h_3 T, \\ \frac{dR}{dt} = p\theta T - \eta R. \end{cases} \tag{2}$$

Initial conditions for the proposed model are $S_0 > 0$, $E_0 \geq 0$, $I_0 \geq 0$, $T_0 \geq 0$, $R_0 \geq 0$ at $t = 0$. For the future study of the topic, we suggest to the readers that they consider the model for the fractional order case. Besides, the existence, stability, and numerical simulations may be obtained with the help of recent development in the subject as given in [15–22].

The current work is organized as follows. Section 2 is for discussion of some fundamental properties of the model. The basic reproduction ratio and stability analysis of the disease-free equilibrium are discussed in Sect. 3. Section 4 is devoted to highlighting the existence, uniqueness, and stability of positive endemic equilibrium. Section 5 is for numerical simulations. Conclusion and discussion of the findings are highlighted in Sect. 6.

2 Positivity and boundedness

Since the proposed model in the present research work shows human population, it is essential to disclose that all the state variables are nonnegative. For this purpose, we shall state and prove the result below.

Theorem 2.1 *The solution of system* (2) *with given initial conditions will always remain nonnegative for* $t > 0$.

Proof Suppose $t_s = \sup\{t > 0 : S_0 > 0, E_0 > 0, I_0 > 0, T_0 > 0, R_0 > 0\}$. The first equation of system (2) is recalled here as follows:

$$\frac{dS}{dt} = \pi - \lambda S(I + \beta T) - \eta S.$$

Letting $\zeta(t) = \lambda(I + \beta T)$ yields

$$\frac{dS}{dt} = \pi - \big(\zeta(t) + \eta\big)S,$$

which implies that

$$d\left(S(t)\exp\left\{\eta t+\int_0^t \zeta(e)\,de\right\}\right)=\pi\exp\left\{\eta t+\int_0^t \zeta(e)\,de\right\}dt,$$

hence

$$S(t_s)\exp\left\{\eta t_s+\int_0^{t_s} \zeta(e)\,de\right\}-S(0)=\int_0^{t_s}\pi\left[\exp\left\{\eta t+\int_0^t \zeta(e)\,de\right\}\right]dt.$$

Thus, we have

$$\begin{aligned}S(t_s)&=S(0)\exp\left\{-\eta t_s-\int_0^{t_s} \zeta(e)\,de\right\}\\&\quad+\exp\left\{-\eta t_s-\int_0^{t_s} \zeta(e)\,de\right\}\times\int_0^{t_s}\pi\left[\exp\left\{\eta t+\int_0^t \zeta(e)\,de\right\}\right]dt>0.\end{aligned}$$

In the same manner it can be disclosed that other variables are also nonnegative for $t>0$. Hence the required result is obtained. □

Next, for the proposed TB model, we consider a biologically feasible domain $\Psi\subset\mathbb{R}^5_+$, where $\Psi=\{(S(t),E(t),I(t),T(t),R(t))\epsilon\mathbb{R}^5_+:0\le N(t)\le\frac{\pi}{\eta}\}$.

Lemma 2.2 *The closed set* Ψ *is positively invariant in* $\mathbb{R}^5_+$.

Proof From system (2), we have

$$\frac{dN}{dt}=\pi-\eta N-\delta_1 I-\delta_2 T,$$

which implies

$$\frac{dN}{dt}\le\pi-\eta N.$$

It is obvious that $\frac{dN}{dt}\le 0$, provided $N(t)\ge\frac{\pi}{\eta}$, this gives

$$N(t)\le\left(N(0)-\frac{\pi}{\eta}\right)e^{-\eta t}+\frac{\pi}{\eta}.$$

Clearly, $N(t)\le\frac{\pi}{\eta}$ if $N(0)\le\frac{\pi}{\eta}$. Moreover, for $N(0)>\frac{\pi}{\eta}$, then the solutions either enter Ψ in finite time or $N(t)$ asymptotically approaches $\frac{\pi}{\eta}$ as $t\to\infty$. Hence the required result is obtained. □

Thus, it is meaningful to study the proposed TB model in feasible region Ψ. For the upcoming results, we suggest the readers some recent related results for the stabilities and numerical techniques given in [23–29].

3 Stability of disease-free equilibrium (DFE)

The DFE of our proposed TB transmission model can be obtained by taking the right-hand sides of the model equal to zero, and we get the following result:

$$\xi = (S_0, 0, 0, 0, 0),$$

where $S_0 = \frac{\pi}{\eta}$.

Now, to examine the qualitative behavior of model (2), it is necessary to determine a threshold quantity, called basic reproduction ratio ($\Re_0$). For this purpose, the next generation operator method [30] is used. For computation of $\Re_0$, the corresponding matrices are as follows:

$$\mathfrak{F} = \begin{pmatrix} 0 & \lambda S_0 & \lambda\beta S_0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$\mathfrak{V} = \begin{pmatrix} h_1 & 0 & -(1-p)\theta \\ -\alpha & h_2 & 0 \\ 0 & -\gamma & h_3 \end{pmatrix}.$$

Using $\Re_0 = \rho(\mathfrak{F}\mathfrak{V}^{-1})$, where $\rho(A)$ is the spectral radius of matrix A, so $\Re_0$ is obtained as follows:

$$\Re_0 = \frac{\alpha\lambda S_0(h_3 + \beta\gamma)}{h_1h_2h_3 - (1-p)\theta\alpha\gamma}.$$

$\Re_0$ shows the average number of newly generated TB infection by a single actively TB patient.

In the case when $p = 1$, there is no inappropriate or incomplete treatment (100 percent efficient treatment). If we replace $\Re_0$ by $\Re_{01}$ in this case, then

$$\Re_{01} = \frac{\alpha\lambda S_0(h_3 + \beta\gamma)}{h_1h_2h_3}.$$

Obviously, $\Re_{01} < \Re_0$, thus incomplete treatment (efficient treatment) causes increase (decrease) in the TB infection.

Next we use Theorem 2 in [30] to present the result below.

Theorem 3.1 *The DFE* (ξ) *of our proposed model is locally asymptotically stable* (*LAS*) *if* $\Re_0 < 1$, *but for* $\Re_0 > 1$ *it is unstable.*

Proof The system of equations of model (2) at equilibrium state is

$$\begin{cases} \pi - \lambda S(I + \beta T) - \eta S = 0, \\ \lambda S(I + \beta T) - h_1 E + (1-p)\theta T = 0, \\ \alpha E - h_2 I = 0, \\ \gamma I - h_3 T = 0, \\ p\theta T - \eta R = 0. \end{cases} \tag{3}$$

The Jacobian matrix of the above system at DFE state is

$$J = \begin{pmatrix} -\eta & 0 & -\lambda S_0 & -\lambda\beta S_0 & 0 \\ 0 & -h_1 & \lambda S_0 & \lambda\beta S_0 & 0 \\ 0 & \alpha & -h_2 & 0 & 0 \\ 0 & 0 & \gamma & -h_3 & 0 \\ 0 & 0 & 0 & p\theta & -\eta \end{pmatrix}.$$

A characteristic equation of the above matrix with eigenvalues μ is

$$\det(J - \mu I) = \det \begin{pmatrix} -(\eta+\mu) & 0 & -\lambda S_0 & -\lambda\beta S_0 & 0 \\ 0 & -(h_1+\mu) & \lambda S_0 & \lambda\beta S_0 & 0 \\ 0 & \alpha & -(h_2+\mu) & 0 & 0 \\ 0 & 0 & \gamma & -(h_3+\mu) & 0 \\ 0 & 0 & 0 & p\theta & -(\eta+\mu) \end{pmatrix} = 0.$$

After performing some simplification, we get

$$\det(J - \mu I) = (\eta+\mu)^2 \det \begin{pmatrix} -(h_1+\mu) & \lambda S_0 & \lambda\beta S_0 \\ \alpha & -(h_2+\mu) & 0 \\ 0 & \gamma & -(h_3+\mu) \end{pmatrix} = 0.$$

Thus

$$(\eta+\mu)^2 = 0 \quad \Rightarrow \quad \mu_1 = \mu_2 = -\eta,$$

or

$$\det \begin{pmatrix} -(h_1+\mu) & \lambda S_0 & \lambda\beta S_0 \\ \alpha & -(h_2+\mu) & 0 \\ 0 & \gamma & -(h_3+\mu) \end{pmatrix} = 0. \tag{4}$$

To determine the nature of the eigenvalues in (4), we use the Routh–Hurwitz criteria. To do this, we obtain the characteristic equation of (4)

$$\mu^3 + a_1\mu^2 + a_2\mu + a_3 = 0, \tag{5}$$

where

$$a_1 = h_1 + h_2 + h_3,$$

$$a_2 = h_1h_2 + h_2h_3 + h_1h_3 - \alpha\lambda S_0,$$

$$a_3 = h_1h_2h_3 - \alpha\gamma(1-p)\theta - \alpha\lambda(h_3 + \gamma\beta)S_0.$$

Clearly, $a_1 > 0$ and $a_3 = (h_1h_2h_3 - \alpha\gamma(1-p)\theta)(1 - \Re_0) > 0$ as $\Re_0 < 1$, also it is obvious that $a_1a_2 > a_3$.

Therefore the Routh–Hurwitz criterion is satisfied. Hence we have obtained that either all the roots of the characteristic equation are negative or have negative real part. Thus the required result is obtained. □

The biological implication of the above theorem is that TB can be completely controlled when $\Re_0 < 1$. It is indispensable to disclose that the DFE is globally asymptotically stable (GAS) in order to guarantee that the eradication of TB is independent of the initial size of population. Thus, we obtain the following result.

Theorem 3.2 *The DFE of model* (2) *is GAS in* Ψ *if* $\Re_0 < 1$.

Proof Assume that

$$\Omega = A_1E + A_2I + A_3T$$

is a Lyapunov function, where $A_1 = \alpha h_3$, $A_2 = h_1h_3$ and $A_3 = \alpha(\lambda\beta S + (1-p)\theta)$.

The derivative of the above Lyapunov function gives

$$\dot{\Omega} = A_1\dot{E} + A_2\dot{I} + A_3\dot{T}. \tag{6}$$

Using the values of $\dot{E}$, $\dot{I}$, $\dot{T}$ in (6), we obtain

$$\dot{\Omega} = A_1\big[\lambda S(I + \beta T) - h_1E + (1-p)\theta T\big] + A_2[\alpha E - h_2I] + A_3[\gamma I - h_3T].$$

Now, taking into account the values of A_1, A_2, and A_3, we get

$$\begin{aligned}
\dot{\Omega} &= (A_1\lambda S - A_2h_2 + A_3\gamma)I \\
&\leq (A_1\lambda S_0 - A_2h_2 + A_3\gamma)I \\
&\leq \big(h_1h_2h_3 - \alpha\gamma\big(\lambda\beta S_0 + (1-p)\theta\big)\big)\left(\frac{\lambda\alpha(h_3 + \beta\gamma)S_0}{h_1h_2h_3 - (1-p)\theta\alpha\gamma} - 1\right) \\
&= \big(h_1h_2h_3 - \alpha\gamma\big(\lambda\beta S_0 + (1-p)\theta\big)\big)(\Re_0 - 1).
\end{aligned}$$

Thus $\dot{\Omega} < 0$ if $\Re_0 < 1$. Therefore the singleton set ξ is the largest compact invariant set in Ψ. Thus, by LaSalle's invariance principle [16], the DFE is GAS in Ψ. □

The above statement indicates that a population can get rid of the TB infection if and only if $\Re_0 < 1$.

4 Endemic equilibrium (EE)

4.1 Existence

Let $\Xi = (S^*, E^*, I^*, T^*, R^*)$ be the EE of model (2). We obtain the expressions for EE of proposed model (2) as given below:

$$\begin{cases} S^* = \frac{\pi}{xI^*+\eta}, \\ E^* = \frac{h_2 I^*}{\alpha}, \\ T^* = \frac{\gamma I^*}{h_3}, \\ R^* = \frac{p\theta\gamma}{\eta h_3} I^*, \\ I^* = \frac{\eta}{x}(\Re_0 - 1), \end{cases} \tag{7}$$

where $x = \frac{\lambda(h_3+\beta\gamma)}{h_3}$.

It is obvious that a unique positive EE exists if $\Re_0 > 1$. The above results are summarized in the lemma given below.

Lemma 4.1 *The proposed TB model* (2) *has a unique positive EE provided* $\Re_0 > 1$.

4.2 Global stability (GS)

Here, we present an important result regarding the GS of the EE. This result is presented in the form of a theorem stated and proved below.

Theorem 4.2 *Let* $\Re_0 > 1$. *Then the EE* $\Xi = (S^*, E^*, I^*, T^*, R^*)$ *of system* (2) *is GAS.*

Proof To show that the EE is GAS, a Lyapunov function as given in [14, 31, 32] is considered here as follows:

$$\begin{aligned} \Pi = {} & \left(S - S^* - S^* \ln\left(\frac{S}{S^*}\right)\right) + \left(E - E^* - E^* \ln\left(\frac{E}{E^*}\right)\right) \\ & + A\left(I - I^* - I^* \ln\left(\frac{I}{I^*}\right)\right) \\ & + B\left(T - T^* - T^* \ln\left(\frac{T}{T^*}\right)\right), \end{aligned}$$

here positive constants A and B are to be evaluated later. Further, the EE $\Xi = (S^*, E^*, I^*, T^*, R^*)$ satisfies

$$\begin{cases} \pi = \lambda S^*(I^* + \beta T^*) + \eta S^*, \\ h_1 E^* = \lambda S^*(I^* + \beta T^*) + (1-p)\theta T^*, \\ h_2 I^* = \alpha E^*, \\ h_3 T^* = \gamma I^*. \end{cases} \tag{8}$$

On differentiating Π, we get

$$\dot{\Pi} = \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{E^*}{E}\right)\dot{E} + A\left(1 - \frac{I^*}{I}\right)\dot{I} + B\left(1 - \frac{T^*}{T}\right)\dot{T}.$$

Placing the values of $\dot{S}$, $\dot{E}$, $\dot{I}$, and $\dot{T}$, we get

$$\begin{aligned}\dot{\Pi} &= \big(1 - S^*/S\big)\big(\lambda S^*\big(I^* + \beta T^*\big) + \eta S^* - \lambda S(I + \beta T) - \eta S\big)\\ &\quad + \big(1 - E^*/E\big)\big(\lambda(I + \beta T) - h_1 E + (1-p)\theta T\big)\\ &\quad + A\big(1 - I^*/I\big)(\alpha E - h_2 I) + B\big(1 - T^*/T\big)(\gamma I - h_3 T).\end{aligned}$$

After simplification, we have

$$\begin{cases}\dot{\Pi} = -\frac{\eta}{S}(S - S^*)^2 + \lambda S^* I^*(2 - \frac{S^*}{S} - \frac{S}{S^*}\frac{I}{I^*}\frac{E^*}{E})\\ \quad + \lambda\beta S^* T^*(2 - \frac{S^*}{S} - \frac{S}{S^*}\frac{T}{T^*}\frac{E^*}{E}) + I(\lambda S^* - Ah_2 + B\gamma)\\ \quad + T(\lambda\beta S^* + (1-p)\theta - Bh_3) + E(A\alpha - h_1) + (1-p)\theta T^*(1 - \frac{T}{T^*}\frac{E^*}{E})\\ \quad + A\alpha E^*(1 - \frac{E}{E^*}\frac{I^*}{I}) + B\gamma I^*(1 - \frac{I}{I^*}\frac{T^*}{T}).\end{cases} \tag{9}$$

We choose positive constants A and B such that

$$\begin{cases}\lambda S^* - Ah_2 + B\gamma = 0,\\ \lambda\beta S^* + (1-p)\theta - Bh_3 = 0,\\ A\alpha - h_1 = 0.\end{cases} \tag{10}$$

The solution of the above equations gives

$$\begin{cases}A = \frac{h_1}{\alpha},\\ B = \frac{\beta h_1 h_3 + \alpha(1-p)\theta}{\alpha(h_3 + \beta\gamma)}.\end{cases}$$

Now, using the above results in (9), we get

$$\begin{cases}\dot{\Pi} = -\frac{\eta}{S}(S - S^*)^2 + \lambda S^* I^*(2 - \frac{S^*}{S} - \frac{S}{S^*}\frac{I}{I^*}\frac{E^*}{E})\\ \quad + \lambda\beta S^* T^*(2 - \frac{S^*}{S} - \frac{S}{S^*}\frac{T}{T^*}\frac{E^*}{E}) + (1-p)\theta T^*(1 - \frac{T}{T^*}\frac{E^*}{E})\\ \quad + A\alpha E^*(1 - \frac{E}{E^*}\frac{I^*}{I}) + B\gamma I^*(1 - \frac{I}{I^*}\frac{T^*}{T}).\end{cases} \tag{11}$$

Making use of the second equation in (8) and multiplying E^* to the third equation in (10) yields

$$\begin{aligned}&h_1 E^* = \lambda S^*\big(I^* + \beta T^*\big) + (1-p)\theta T^*,\\ &h_1 E^* = A\alpha E^*.\end{aligned}$$

Hence, on comparison it follows that

$$-\lambda S^*\big(I^* + \beta T^*\big) - (1-p)\theta T^* + A\alpha E^* = 0.$$

Now, consider a function $g_1(X)$ where $X = (m_1, m_2, m_3, m_4)$ and $m_1 = \frac{S}{S^*}$, $m_2 = \frac{E}{E^*}$, $m_3 = \frac{I}{I^*}$, $m_4 = \frac{T}{T^*}$. Multiplying $g_1(X)$ with the above equation, we obtain

$$-\lambda S^*\big(I^* + \beta T^*\big)g_1(X) - (1-p)\theta T^* g_1(X) + A\alpha E^* g_1(X) = 0. \tag{12}$$

Multiplying B to the fourth equation in (8) and T^* to the second equation in (10) yields

$$\lambda\beta S^* T^* + (1-p)\theta T^* = Bh_3 T^*,$$
$$B\gamma I^* = Bh_3 T^*.$$

Comparing the above equations, we get

$$\lambda\beta S^* T^* + (1-p)\theta T^* - B\gamma I^* = 0.$$

Also, consider a function $g_2(X)$, where $X = (m_1, m_2, m_3, m_4)$ and $m_1 = \frac{S}{S^*}$, $m_2 = \frac{E}{E^*}$, $m_3 = \frac{I}{I^*}$, $m_4 = \frac{T}{T^*}$. Multiplying $g_2(X)$ with the above equation, we obtain

$$\lambda\beta S^* T^* g_2(X) + (1-p)\theta T^* g_2(X) - B\gamma I^* g_2(X) = 0. \tag{13}$$

On plugging equation (12) and equation (13) into equation (11), we get

$$\begin{cases} \dot{\Pi} = -\frac{\eta}{S}(S-S^*)^2 + \lambda S^* I^* (2 - \frac{S^*}{S} - \frac{S}{S^*}\frac{I}{I^*}\frac{E^*}{E} - g_1(X)) \\ \quad + \lambda\beta S^* T^* (2 - \frac{S^*}{S} - \frac{S}{S^*}\frac{T}{T^*}\frac{E^*}{E} - g_1(X) + g_2(X)) \\ \quad + (1-p)\theta T^* (1 - \frac{T}{T^*}\frac{E^*}{E} - g_1(X) + g_2(X)) \\ \quad + A\alpha E^* (1 - \frac{E}{E^*}\frac{I^*}{I} + g_1(X)) + B\gamma I^* (1 - \frac{I}{I^*}\frac{T^*}{T} - g_2(X)). \end{cases} \tag{14}$$

Now, we choose the functions $g_1(X)$ and $g_2(X)$ to get zero coefficients of E^* and I^*. So we have

$$g_1(X) = \frac{E}{E^*}\frac{I^*}{I} - 1,$$
$$g_2(X) = 1 - \frac{I}{I^*}\frac{T^*}{T}.$$

Using these values of $g_1(X)$ and $g_2(X)$ along with making use of variables m_1, m_2, m_3, and m_4 in (14), we obtain

$$\begin{aligned} \dot{\Pi} = {} & -\frac{\eta}{S}\left(S - S^*\right)^2 + \lambda S^* I^* \left(3 - \frac{1}{m_1} - \frac{m_1 m_3}{m_2} - \frac{m_2}{m_3}\right) \\ & + \lambda\beta S^* T^* \left(4 - \frac{1}{m_1} - \frac{m_1 m_4}{m_2} - \frac{m_2}{m_3} - \frac{m_3}{m_4}\right) \\ & + (1-p)\theta T^* \left(3 - \frac{m_4}{m_2} - \frac{m_2}{m_3} - \frac{m_3}{m_4}\right). \end{aligned} \tag{15}$$

As

$$\frac{1}{m_1} \cdot \frac{m_1 m_3}{m_2} \cdot \frac{m_2}{m_3} = 1,$$
$$\frac{1}{m_1} \cdot \frac{m_1 m_4}{m_2} \cdot \frac{m_2}{m_3} \cdot \frac{m_3}{m_4} = 1,$$
$$\frac{m_4}{m_2} \cdot \frac{m_2}{m_3} \cdot \frac{m_3}{m_4} = 1.$$

Since, for real numbers $\nu_1, \nu_2, \nu_3, \ldots, \nu_n \geq 0$, the following inequality holds:

$$\nu_1 + \nu_2 + \nu_3 + \cdots + \nu_n \geq n \sqrt[n]{\nu_1 \cdot \nu_2 \cdot \nu_3 \cdot \cdots \cdot \nu_n}.$$

As a special case, when

$$\nu_1 \cdot \nu_2 \cdot \nu_3 \cdot \cdots \cdot \nu_n = 1,$$

then

$$\nu_1 + \nu_2 + \nu_3 + \cdots + \nu_n \geq n.$$

Using the above inequality, we have

$$\begin{aligned}
&3 - \frac{1}{m_1} - \frac{m_1 m_3}{m_2} - \frac{m_2}{m_3} \leq 0 \\
&4 - \frac{1}{m_1} - \frac{m_1 m_4}{m_2} - \frac{m_2}{m_3} - \frac{m_3}{m_4} \leq 0 \\
&3 - \frac{m_4}{m_2} - \frac{m_2}{m_3} - \frac{m_3}{m_4} \leq 0.
\end{aligned}$$

Thus from (15) we get

$$\dot{\Pi} \leq 0.$$

Therefore LaSalle's invariance principle [33] ensures that EE is GAS provided $\Re_0 > 1$. Hence the required result is obtained. □

5 Numerical simulation

Here, we are going to demonstrate the behavior of the proposed TB infection model through numerical simulation. System (2) is further used to determine the impact of some intervention strategies to control the TB infection from spread. The iterative scheme RK-4 is utilized to solve the system, while the simulation is conducted through the MATLAB software. The parameter values for simulations are either from Ronoh et al. [34] or reasonably chosen estimates. To do this, consider the parametric values as follows:

$$\begin{aligned}
&\pi = 0.2, \quad \lambda = 0.7, \quad \alpha = 0.25, \quad \eta = 0.1, \quad \delta_1 = 0.15, \\
&\delta_2 = 0.05, \quad \gamma = 0.2, \quad \theta = 0.1000, \quad \beta = 0.1, \quad p = 0.9,
\end{aligned}$$

and the calculated value of $\Re_0$ is

$$\Re_0 = 2.4950 > 1.$$

The initial conditions are as follows:

$$S(0) = 7, \quad E(0) = 2, \quad I(0) = 1, \quad T(0) = 0, \quad R(0) = 0,$$

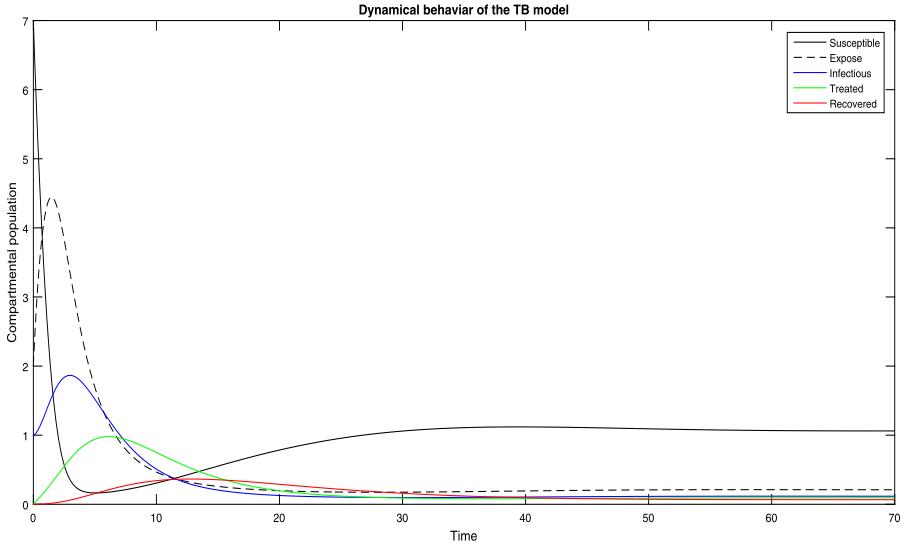

Figure 1 The plot shows the dynamics of all classes when $\Re_0 = 2.4950 > 1$

at the initial level not treated or recovered population is considered. Using the above data, the resulting graph is presented in Fig. 1. From this figure, it can be seen that the trajectories of the solutions of model (2) converge to the EE point, this indicates that the disease persists in the host population if $\Re_0 > 1$, which provides justification to our statement that EE is GAS if $\Re_0 > 1$.

Moreover, to assess the role of effective contact rate and treatment rate on TB transmission, we decrease the contact rate λ and increase the treatment rate γ. In this case, the parameter values are as follows:

$$\pi = 0.2, \quad \lambda = 0.35, \quad \alpha = 0.25, \quad \eta = 0.1, \quad \delta_1 = 0.15,$$
$$\delta_2 = 0.05, \quad \gamma = 0.5, \quad \theta = 0.1000, \quad \beta = 0.1, \quad p = 0.9,$$

and the calculated value of $\Re_0$ is

$$\Re_0 = 0.8484 < 1.$$

Using the above data with the same initial conditions, we present the resulting graph in Fig. 2. From this figure, it can be seen that the trajectories of the solutions of model (2) converge to the DFE point. This indicates that the disease will die out in the host population if $\Re_0 < 1$, which provides justification to our statement that DFE is GAS if $\Re_0 < 1$.

6 Conclusions

This paper is focused on the analysis of a mathematical model to assess the TB transmission in a host population. The threshold quantity $\Re_0$ is obtained using the next-generation method. It is shown that there are two possible equilibria of the model, one is DFE which exists and is locally and globally asymptotically stable if $\Re_0 < 1$, in this case the TB disease dies out. The other is EE which exists and is GAS if $\Re_0 > 1$, in this case TB becomes endemic. Moreover, it is investigated that incomplete treatment causes increase in the TB

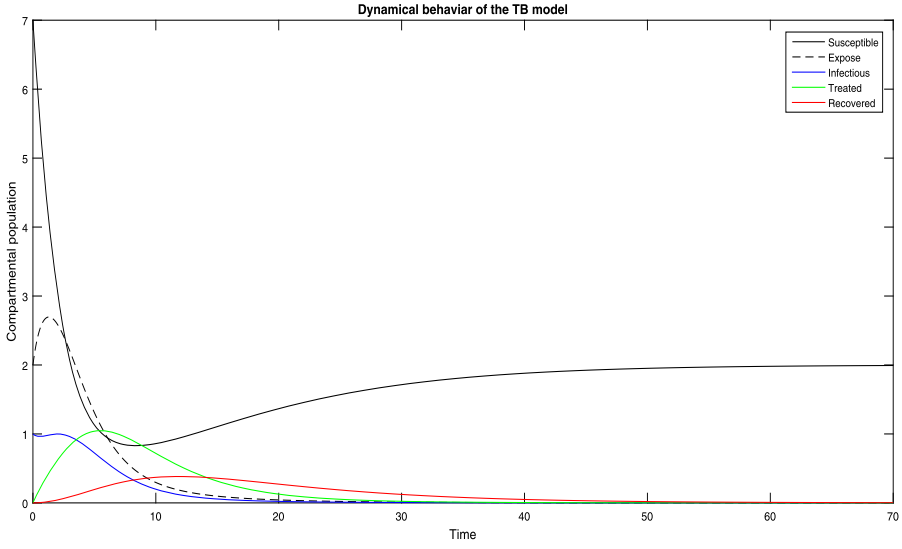

Figure 2 The plot shows the dynamics of all classes when $\Re_0 = 0.8484 < 1$

infection. The numerical results also show that decrease in the effective contact rate and increase in treatment coverage can minimize the spread of the TB infection.

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Authors' contributions
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