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A novel tuberculosis model incorporating a Caputo fractional derivative and treatment effect via the homotopy perturbation method

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Abstract

Background Tuberculosis (TB), caused by Mycobacterium tuberculosis, is a contagious infectious disease that primarily targets the lungs but can also impact other critical systems such as the bones, joints, and neurological system. Despite significant efforts to combat TB, it remains a major global health concern. To address this challenge, this study aims to explore and evaluate various tuberculosis control approaches using a mathematical modeling framework.

Results The study utilized a novel SEITR mathematical model to investigate the impact of treatment on physical limitations in tuberculosis. The model underwent qualitative analysis to validate key aspects, including positivity, existence, uniqueness, and boundedness. Disease-free and endemic equilibria were identified, and both local and global stability of the model was thoroughly examined using the derived reproduction number. To estimate the impact of each parameter on each compartment, sensitivity analysis was conducted, and numerical simulations were performed using Maple 18 software with the homotopy perturbation method. The obtained results are promising and highlight the potential of the proposed interventions to significantly reduce tuberculosis virus prevalence. The findings emphasize the significance of fractional-order analysis in understanding the effectiveness of treatment strategies for mitigating tuberculosis prevalence. The study suggests that the time fractional dynamics of TB treatment correspond to the treatment's efficacy, as the conceptual results showed that non-local interactions between the disease and the treatment may lead to more accurate ways of eradicating tuberculosis in real-world scenarios. These insights contribute to a better understanding of effective treatment strategies and their potential impact on tuberculosis control and public health.

Conclusions In conclusion, scientists, researchers, and healthcare personnel are urged to take action and utilize the discoveries from this research to facilitate the eradication of the hazardous tuberculosis bacteria.

Keywords Tuberculosis, Lyapunov, Treatment, Caputo derivative, Homotopy perturbation method, Global stability

Background

Tuberculosis (TB) is a highly contagious disease caused by the Mycobacterium tuberculosis bacteria, primarily affecting densely populated areas and posing a significant public health threat. It spreads through airborne transmission when infected individuals cough or have close contact with others. Common symptoms include a persistent cough, chest pain, weight loss, and a fever. Diagnosis involves a comprehensive assessment of medical history and various examinations, such as X-rays and tests. TB is particularly prevalent in low- and middle-income countries with limited access to healthcare, impacting millions of people worldwide. An active TB patient can infect 5 to 15 susceptible individuals in their environment (WHO 2021).

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To combat TB, several preventive measures have been proposed, including treatment interventions (Luju and Yan 2014; Ramli et al. 2019; Clark et al. 2019). (Luju and Yan 2014) incorporated treatment interruptions and latent periods into their approach, while (Ramli et al. 2019) emphasized the importance of multiple drug administration's within specific timeframes. Clark et al. (2019) conducted sensitivity analyses to identify effective strategies for treatment and prevention during epidemics. (Ullah et al. 2020a, b) employed the modified Adams–Bashforth technique to calculate numerical solutions for a fractional tuberculosis model, highlighting the significance of the proposed derivative in understanding TB dynamics and control. (Fatmawati et al. 2020) focused on investigating TB infection dynamics in children and adults using a novel fractional model and fractional calculus, particularly the Caputo and Atangana–Baleanu derivatives. Their results indicated a significant reduction in TB infection among individuals already affected by the disease when the fractional order was decreased. (Ullah et al. 2020a, b) explored optimal TB control techniques involving vaccines and treatment, emphasizing the importance of community-based TB control and showcasing the impact of disease control through simulations. Furthermore, (Ullah et al. 2018) proposed a fractional-order model for simulating TB dynamics in Khyber Pakhtunkhwa, Pakistan, utilizing the Caputo derivative to demonstrate its superiority over classical models.

Recent research has focused on investigating the age-specific aspects of TB infection, with newborn vaccination being suggested as a preventive measure (Tilahun et al. 2020; Mengistu et al. 2020). The urgency to develop a more effective and longer-lasting TB vaccine has heightened due to the emergence of drug-resistant strains. However, surpassing the efficacy and durability of the current BCG vaccine has proven to be a significant challenge (Schrager et al. 2020). To address this challenge, researchers have developed the TB Vaccine Development Pathway web tool (Roordink et al. 2021), which serves as a valuable resource for guiding vaccine development from discovery to deployment, ultimately aiming to eradicate TB.

Various preventive strategies,including therapy interventions, infant immunization, and innovative vaccine development according to (Zhang et al. 2023; Kelemu Mengistu and Witbooi 2019), have been proposed. It is crucial to employ well-designed trials and models to plan for success and ensure the timely distribution of vaccines to those most in need. Different numerical methods, such as the homotopy perturbation method (Kolawole et al. 2023; Olayiwola et al. 2023) and the Laplace Adomian decomposition method (Yunus et al. 2023), have been employed to control various diseases, including COVID-19 and Lassa fever. Fractional calculus has also been utilized to develop mathematical models for epidemic diseases, including TB. The Caputo fractional derivative operator has been employed to describe TB transmission dynamics, taking into account memory behavior (Danane et al. 2020). Additionally, Zhang et al. (2021) delve further into the application of the Caputo operator for TB transmission and prevention, highlighting the crucial role of appropriate treatment in minimizing TB transmission and prevalence. Ahmad et al. (2021) consider a mathematical model for TB that incorporates the Caputo and Fabrizio derivatives with an exponential kernel. Furthermore, (Zafar et al. 2022) examine a newly discovered fractional operator (FO) with a ML non-singular kernel for different fractional orders, utilizing the fractional Caputo and Atangana–Baleanu–Caputo predictor and corrector approaches. Fractional-order derivatives in the context of epidemic disorders have been extensively studied in previous research (Liu et al. 2023; Farman et al. 2023; Nazir et al. 2020; Okyere and Ackora-Prah 2023; Ahmed et al. 2021; Hajaj and Odibat 2023) using operators such as Atangana–Baleanu–Caputo, Caputo–Fabrizio, and Caputo fractional derivatives. Several fractional-order TB models have investigated the impact of quarantine, hospitalization, and treatment, while (Ullah et al. 2023) took a unique deterministic epidemic approach, examining the influence of treatment adherence and awareness on TB dynamics.

Despite the wealth of research on fractional-order models for tuberculosis, the specific effects of treatment rate and effective contact rate on the disease have not been thoroughly explored. This research seeks to fill this gap and contribute valuable insights into how these factors influence tuberculosis dynamics. By analyzing the influence of treatment rate on the fractional order and the impact of the effective contact rate on tuberculosis transmission, this study aims to enhance our understanding of disease control strategies and pave the way for more effective and targeted interventions to combat tuberculosis. To achieve this, we modified the model presented by (Kereyu and Demie 2021) to suit the study of tuberculosis dynamics in Nigeria, specifically considering the incorporation of the treatment class, leading to a significant improvement in disease cure.

The paper's structure includes a detailed description of the model formulation and description in "Model formulation and description" section, the model analysis in "Model analysis" section, an exploration of the method in "Methods" section, the presentation of

results in "Results" section, and a conclusion in "Discussion" section.

Model formulation and description

A five-compartmental model of tuberculosis is described, consisting of susceptible ($S(t)$), exposed $E(t)$, infected $I(t)$, treatment $T(t)$, and recovered $R(t)$ compartments. Susceptible individuals are those who are vulnerable to contracting the disease if they come into contact with someone who is infected. Exposed individuals $E(t)$ are the group of individuals who have come into contact with an infectious agent but have not yet developed symptoms of the disease. The infected class comprises confirmed cases of people who have developed the disease and are prone to transmitting it to others. Individuals undergoing treatment for any illness are at risk of developing tuberculosis, while those who have recovered are denoted as R(t) and receiving treatment are denoted as T(t). The parameter Λ shows the recruitment rate which occurs in the susceptible class only, and susceptible individuals have the tuberculosis through contact with the infected individuals at the rate $\frac{\beta}{N}S(t)I(t)$ where β is the effective contact rate. δ represents the death due to TB, while η also represents the treatment rate of infected class. τ indicates the progression rate of exposed to infected class, and σ represents natural cure rate. The re-infection rate is φ, and the individual leaves the treatment class at the rate θ. Based on the details mentioned earlier, a nonlinear SEITR model for tuberculosis is formulated, and this model is illustrated through the schematic diagram in Fig. 1.

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \mu S(t) - \frac{\beta}{N}S(t)I(t) \\ \frac{dE}{dt} &= \frac{\beta}{N}S(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) \\ \frac{dI}{dt} &= \tau E(t) - (\mu + \eta + \delta)I(t) \\ \frac{dT}{dt} &= \eta I(t) - (\mu + \theta)T(t) \\ \frac{dR}{dt} &= \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t) \end{aligned} \tag{1}$$
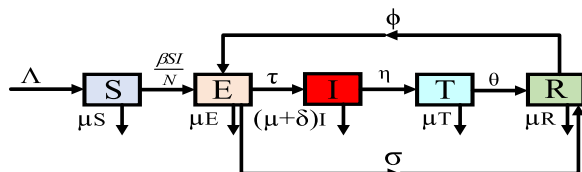

Fig. 1 Schematic diagram of SEITR tuberculosis model

Equation (1) can be rewritten as

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \mu S(t) - KS(t)I(t) \\ \frac{dE}{dt} &= KS(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) \\ \frac{dI}{dt} &= \tau E(t) - (\mu + \eta + \delta)I(t) \\ \frac{dT}{dt} &= \eta I(t) - (\mu + \theta)T(t) \\ \frac{dR}{dt} &= \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t) \end{aligned} \tag{2}$$

where $K = \frac{\beta}{N}$.

With the initial conditions:

$$S(0) = \kappa_1, E(0) = \kappa_2, I(0) = \kappa_3, T(0) = \kappa_4, R(0) = \kappa_5. \tag{3}$$

Table 1 shows the description of variables and parameters.

Preliminaries of fractional calculus

In this section of the paper, some preliminaries definitions are discussed based on fractional calculus.

Definition 1 A fractional order can be represented by $\kappa \in (0, 1]$ using the Riemann–Liouville method of the function $g \in L^{-1}([0, T], R)$ s

Table 1 Description of variables and parameters

	Description 2
Variable	
$S(t)$	Susceptible state
$E(t)$	Exposed state
$I(t)$	Infected state
$T(t)$	Treated state
$R(t)$	Recovered state
Parameters	
Λ	Recruitment rate of susceptible
β	Effective contact rate
σ	Natural cure rate
η	Treatment rate of infected class
μ	Natural death
δ	Death due to TB at infected class
φ	Reinfection rate
θ	Rate at which individual leaves the treated class

$I_0^\kappa f(t) = \frac{1}{\Gamma(\kappa)} \int_0^t (t-x)^{\kappa-1} g(x)\mathrm{d}x$, and the following properties are satisfied:

  i. $I^\kappa I^\alpha \zeta(t) = I^{\kappa+\alpha}\xi(t)$,

  ii. $I^\kappa I^\alpha \zeta(t) = I^\alpha I^\kappa \xi(t)$,

  iii. $I^\kappa t^\beta = \frac{\Gamma(\beta+1)}{\Gamma(\kappa+\beta+1)} t^{\kappa+\beta}$.

Definition 2 The Caputo derivative of a in the interval $[0,T]$ can be written as ${}_0^c D^\kappa g(t) = \frac{1}{\Gamma(m-\kappa)} \int_0^t (t-x)^{m-\kappa-1} g^{(m)}(x)\mathrm{d}x$, where $m = |\kappa| + 1$ and $|\kappa|$ represents the integer part of κ in [0,1].

Model analysis

In this section, we shall conduct the qualitative analysis of the mathematical model, and we shall do this with an integer order $\alpha = 1$ to fully study the properties of the mathematical model and show its potential for real-life applications.

Positivity and boundedness

This section focuses on investigating the positivity and boundedness of solutions to the model in Eq. (2) with the initial condition described in Eq. (3).

Theorem 1 *For $t \geq 0$, the system of Eq. (2) and the starting condition (3) have a positive solution.*

Proof Assume that Eq. (2) has a solution $S(t)$, $E(t)$, $I(t)$, $T(t)$, and $R(t)$ with the initial condition (3). So, we have

$$\frac{\mathrm{d}^\alpha S(t)}{\mathrm{d}t^\alpha} = \Lambda \geq 0, \quad \frac{\mathrm{d}^\alpha E(t)}{\mathrm{d}t^\alpha} = \varphi R + \mathrm{KSI} \geq 0, \quad \frac{\mathrm{d}^\alpha I(t)}{\mathrm{d}t^\alpha}$$

$$= \tau E \geq 0, \quad \frac{\mathrm{d}^\alpha T(t)}{\mathrm{d}t^\alpha} = \eta I \geq 0, \quad \frac{\mathrm{d}^\alpha R(t)}{\mathrm{d}t^\alpha}$$

$$= \theta T + \sigma E \geq 0.$$

Also, for boundedness, let $N(t) = S(t) + E(t) + I(t) + T(t) + R(t)$ which gives

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \mu N(t) - \delta I(t) \leq \Lambda - \mu N(t).$$

Therefore, solution is positive for all $t \geq 0$ and hence bounded.

Dynamic behaviors of the model

The dynamic behavior of the model was investigated by obtaining the disease-free and endemic equilibrium of the tuberculosis model.

Disease-free equilibrium

A disease-free equilibrium (DFE) is a population that is entirely free of infectious diseases. Thus, when there is no infection $I = 0$, the equilibrium points result in: $E_1 = (S^0, E^0, I^0, T^0, R^0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$.

Endemic equilibrium points

Endemic equilibrium is a steady state of an infectious disease in a population in which the prevalence of the disease remains stable over time. Hence, $E \neq I \neq 0$ and the following thresholds are obtained:

$$S^* = \frac{N((\mu+\rho+\tau)(\mu+\eta+\delta)(\mu+\theta)(\mu+\varphi)) - \varphi\rho(\mu+\eta+\delta)(\mu+\theta) - \eta\tau\varphi\theta}{\tau(\mu+\theta)(\mu+\varphi)\beta},$$

$$E^* = \frac{-(N(\mu+\eta+\delta)(\mu+\rho+\tau)(\mu+\theta)(\mu+\varphi)\mu - (\mu+\eta+\delta)(\mu+\theta)N\mu\varphi\sigma - \eta N\tau\varphi\theta(\mu+\eta+\delta) - (\mu+\theta)(\mu+\varphi)(\mu+\eta+\delta)\tau\Lambda\beta)}{\tau\beta(\mu+\eta+\delta)(\mu+\rho+\tau)(\mu+\eta+\delta)(\mu+\theta)(\mu+\varphi) - (\mu+\eta+\delta)(\mu+\theta)\sigma\varphi\tau\beta - \eta\tau^2\beta\varphi\theta)}$$

$$I^* = \frac{(\mu+\eta+\delta)N\mu\varphi - (\mu+\varphi)(\mu+\rho+\tau)(\mu+\eta+\delta)N\mu(\mu+\theta) + \Lambda\beta\tau(\mu+\varphi)(\mu+\theta) + N\eta\mu\varphi\tau\theta}{\tau\beta(\mu+\eta+\delta)(\mu+\rho+\tau)(\mu+\eta+\delta)(\mu+\theta)(\mu+\varphi) - (\mu+\eta+\delta)(\mu+\theta)\sigma\varphi\tau\beta - \eta\tau^2\beta\varphi\theta)} \qquad (4)$$

$$T^* = \frac{-\left(-(\mu+\eta+\delta)N\mu\varphi\sigma\eta + (\mu+\varphi)(\mu+\theta)(\mu+\rho+\tau)(\mu+\eta+\delta)N\mu\eta - \Lambda\beta\tau\eta(\mu+\varphi)(\mu+\theta) - N\eta^2\mu\varphi\tau\theta\right)}{\beta(\mu+\eta+\delta)(\mu+\rho+\tau)(\mu+\theta)^2(\mu+\varphi) - (\mu+\theta)^2\sigma\varphi\beta - \eta\tau\beta\varphi\theta(\mu+\theta))}$$

$$R^* = \frac{-(-(\mu+\varphi)(\mu+\theta)\Lambda\beta\tau + N(\mu+\rho+\tau)(\mu+\eta+\delta)(\mu+\varphi)(\mu+\theta)\mu - N\mu\varphi\sigma(\mu+\eta+\delta)(\mu+\varphi) - N\eta\mu\varphi\tau\theta)((\mu+\eta+\delta)(\mu+\varphi)\sigma + \eta\tau\theta)}{(((\mu+\eta+\delta)(\mu+\rho+\tau)(\mu+\theta)(\mu+\varphi)) - (\varphi\sigma(\mu+\rho+\tau)(\mu+\theta)) - \eta\tau\varphi\theta(\mu+\eta+\delta))((\mu+\theta)(\mu+\varphi)\tau\beta)}.$$

Basic reproduction number

The reproductive number of the SEITR model associated with the reproductive power of the disease is defined by $R_0 = \rho(G)$ where ρ is the spectral radius of the next-generation matrix $G = FV^{-1}$.

So the R_0 of the model Eq. (2) at diseases-free equilibrium is obtained as:

$$F = \begin{bmatrix} \frac{\beta S^0}{N} \\ 0 \\ 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} (\mu + \tau + \sigma)E \\ -\tau E + (\mu + \eta + \delta)I \\ -\eta I + (\mu + \theta)T \end{bmatrix}. \quad (5)$$

$$\text{Given that } F = \begin{bmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} \\ \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} \end{bmatrix} = \begin{bmatrix} 0 & \frac{\beta S^0}{N} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (6)$$

$$\text{and } V = \begin{bmatrix} \frac{\partial v_1}{\partial E} & \frac{\partial v_1}{\partial I} & \frac{\partial v_1}{\partial T} \\ \frac{\partial v_2}{\partial E} & \frac{\partial v_2}{\partial I} & \frac{\partial v_2}{\partial T} \\ \frac{\partial v_3}{\partial E} & \frac{\partial v_3}{\partial I} & \frac{\partial v_3}{\partial T} \end{bmatrix} = \begin{bmatrix} (\mu + \tau + \sigma) & 0 & 0 \\ -\tau & (\mu + \eta + \delta) & 0 \\ -\eta & (\mu + \theta) & 0 \end{bmatrix}. \quad (7)$$

So, the next-generation matrix is resulted in

$$G = FV^{-1} = \begin{bmatrix} \frac{\beta \Lambda \tau}{N(\mu+\sigma+\tau)(\eta+\mu+\delta)} & \frac{\beta \Lambda}{(\eta+\mu+\delta)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad (8)$$

Therefore, $R_0 = \frac{\beta \Lambda \tau}{N(\mu+\sigma+\tau)(\eta+\mu+\delta)}$ which represent the dominant eigenvalue.

Local stability for disease-free equilibrium

At the equilibrium state, the equations of the model (2) can be written as follows

$$\begin{cases} \Lambda - \mu S(t) - KS(t)I(t) = 0, \\ KS(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) = 0, \\ \tau E(t) - (\mu + \eta + \delta)I(t) = 0, \\ \eta I(t) - (\mu + \theta)T(t) = 0, \\ \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t) = 0, \end{cases} \quad (9)$$

where $K = \frac{\beta}{N}$.

At the disease-free equilibrium point, the Jacobian matrix of Eq. (9) is obtained as

$$J = \begin{pmatrix} -\mu & 0 & \frac{-\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & -\mu - \rho - \tau & \frac{\beta\Lambda}{N\mu} & 0 & \varphi \\ 0 & \tau & -\delta - \mu - \eta & 0 & 0 \\ 0 & 0 & \eta & -\mu - \theta & 0 \\ 0 & \sigma & 0 & 0 & -\mu - \varphi \end{pmatrix}. \quad (10)$$

Also, the characteristics equation can be obtained using $\det(J(E_1) - \lambda I)$ to obtain the eigenvalues λ.

$$\det \begin{pmatrix} -\mu - \lambda & 0 & \frac{-\beta\Lambda}{N\mu} & 0 & 0 \\ 0 & (-\mu - \rho - \tau) - \lambda & \frac{\beta\Lambda}{N\mu} & 0 & \varphi \\ 0 & \tau & (-\delta - \mu - \eta) - \lambda & 0 & 0 \\ 0 & 0 & \eta & (-\mu - \theta) - \lambda & 0 \\ 0 & \sigma & 0 & 0 & (-\mu - \varphi) - \lambda \end{pmatrix} = 0. \quad (11)$$

The characteristics equation derived from Eq. (11) is

$$(\lambda + \mu)\left(\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0\right) = 0.$$

Using the Routh–Hurwitz criterion stability (Gantmacher 1959) of order four, $a_3 a_2 a_1 > a_1^2 + a_3^2 a_0$ where $a_i > 0$.

$$a_3 = 4\mu + \eta + \varphi + \rho + \delta + \tau + \theta, \quad (12)$$

$$\begin{aligned} a_2 =& 6\mu^2 + (3(\eta + \varphi + \delta + \rho + \tau + \theta)\mu \\ &+ (\eta + \varphi + \rho + \delta + \tau)\theta \\ &+ (\eta + \varphi + \delta - 1)\tau + (\varphi + \sigma)(\eta + \delta)), \end{aligned} \quad (13)$$

$$\begin{aligned} a_1 =& 4\mu^3 + (3(\eta + \varphi + \delta + \rho + \tau + \theta)\mu^2 \\ &+ (2(\eta + \varphi + \rho + \delta + \tau)\theta + 2(\eta + \varphi + \delta - 1)\tau \\ &+ 2(\varphi + \sigma)(\eta + \delta))\mu + ((\eta + \varphi + \delta - 1)\tau \\ &+ (\varphi + \sigma)(\eta + \delta))\theta + \varphi\tau(\eta + \delta - 1)), \end{aligned} \quad (14)$$

$$\begin{aligned} a_0 =& \mu^4 + (\eta + \varphi + \delta + \rho + \tau + \theta)\mu^3 \\ &+ ((\eta + \varphi + \rho + \delta + \tau)\theta \\ &+ (\eta + \varphi + \delta - 1)\tau + (\varphi + \sigma)(\eta + \delta))\mu^2 \\ &+ ((\eta + \varphi + \delta - 1)\tau + (\varphi + \sigma)(\eta + \delta))\theta \\ &+ \varphi\tau(\eta + \delta - 1))\mu + \varphi\tau\theta(\delta - 1). \end{aligned} \quad (15)$$

From the above equations, it can be observed that $a_3 > 0, a_2 > 0, a_1 > 0$ and $a_0 > 0$.

So, the Routh–Hurwitz criterion is satisfied.

Hence, the disease-free equilibrium of the system is asymptotically stable.

Global stability of endemic equilibrium

Theorem 2 *If $R_0 > 1$, the endemic equilibrium point of the model Eq.* (2) *is globally asymptotically stable.*

where P and Q can be extracted from Eq. (20) and then give the formula as $\frac{dV}{dt} = P - Q$ which is then written as:

$$P = \frac{(S - S^*)}{S}[\Lambda - \mu] + \frac{(E - E^*)}{E}\left[\begin{array}{c} K(S - S^*)(I - I^*) \\ + \varphi(R - R^*) \end{array}\right] + \frac{(I - I^*)}{I}\left[\tau(E - E^*)\right] + \frac{(T - T^*)}{T}\left[\eta(I - I^*)\right] + \frac{(R - R^*)}{R}\left[\begin{array}{c} \theta(T - T^*) \\ + \sigma(E - E^*) \end{array}\right] \quad (21)$$

Proof The global stability is constructed using Lyapunov function as follows:

$$\left\{\begin{array}{l} V(S^*, E^*, I^*, T^*, R^*) = \left(S - S^* - S^* \ln\frac{S}{S^*}\right) + \left(E - E^* - E^* \ln\frac{E}{E^*}\right) + \left(I - I^* - I^* \ln\frac{I}{I^*}\right) \\ + \left(T - T^* - T^* \ln\frac{T}{T^*}\right) + \left(R - R^* - R^* \ln\frac{R}{R^*}\right) \end{array}\right\}. \quad (16)$$

Obtaining the derivative of Lyapunov of the solution of Eq. (2) results in:

$$\frac{dV}{dt} = \left(\frac{S - S^*}{S}\right)\frac{dS}{dt} + \left(\frac{E - E^*}{E}\right)\frac{dE}{dt} + \left(\frac{I - I^*}{I}\right)\frac{dI}{dt} + \left(\frac{T - T^*}{T}\right)\frac{dT}{dt} + \left(\frac{R - R^*}{R}\right)\frac{dR}{dt}, \quad (17)$$

where we have

$$\left\{\begin{array}{l} \frac{dS}{dt} = \Lambda - \mu S(t) - KS(t)I(t), \\ \frac{dE}{dt} = KS(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) \\ \frac{dI}{dt} = \tau E(t) - (\mu + \eta + \delta)I(t), \\ \frac{dT}{dt} = \eta I(t) - (\mu + \theta)T(t), \\ \frac{dR}{dt} = \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t). \end{array}\right. \quad (18)$$

Inserting (18) into (17), we arrive at

and

$$Q = \frac{(S - S^*)^2}{S}K(I - I^*) + \frac{(E - E^*)^2}{E}$$
$$(\mu + \tau + \sigma) + \frac{(I - I^*)^2}{I}(\mu + \eta + \delta) \quad (22)$$
$$+ \frac{(T - T^*)^2}{T}(\mu + \theta) + \frac{(R - R^*)^2}{R}(\mu + \varphi)$$

Therefore, if $P < Q$, the $\frac{dV}{dt}$ will be negative, also if $\frac{dV}{dt} = 0$ if and only if $S(t) = S^*, E(t) = E^*, I(t) = I^*$, $T(t) = T^*, R(t) = R^*$. Hence, the largest invariant set in $\left\{(S(t), E(t), I(t), T(t), R(t)) : \frac{dV}{dt} = 0\right\}$ is the single point set E^*. Using the LaSalle's invariance principle adopted by (LaSalle 1976), the endemic equilibrium is globally asymptotically stable if $P < Q$.

$$\frac{dV}{dt} = \left\{\begin{array}{l} \left(\frac{S - S^*}{S}\right)[\Lambda - \mu S - KSI] + \left(\frac{E - E^*}{E}\right)[KSI - (\mu + \tau + \sigma)E + \varphi R] \\ + \left(\frac{I - I^*}{I}\right)[\tau E - (\mu + \eta + \delta)I] + \left(\frac{T - T^*}{T}\right)[\eta I - (\mu + \theta)T] + \left(\frac{R - R^*}{R}\right)[\theta T + \sigma E - (\mu + \varphi)R] \end{array}\right\}. \quad (19)$$

Then, we obtain

$$\frac{dV}{dt} = \left\{\begin{array}{l} \frac{(S - S^*)}{S}\Lambda - \mu\frac{(S - S^*)}{S} - K\frac{(S - S^*)^2}{S}(I - I^*) + K(S - S^*)(I - I^*)\frac{(E - E^*)}{E} - (\mu + \tau + \sigma)\frac{(E - E^*)^2}{E} \\ + \varphi\frac{(E - E^*)}{E}(R - R^*) + \tau(E - E^*)\frac{(I - I^*)}{I} - (\mu + \eta + \delta)\frac{(I - I^*)^2}{I} + \eta(I - I^*)\frac{(T - T^*)}{T} - (\mu + \theta)\frac{(T - T^*)^2}{T} \\ + \theta(T - T^*)\frac{(R - R^*)}{R} + \sigma(E - E^*)\frac{(R - R^*)}{R} - (\mu + \varphi)\frac{(R - R^*)^2}{R}, \end{array}\right\}, \quad (20)$$

Methods

Homotopy perturbation method

Our study focuses on utilizing the homotopy perturbation method as a numerical approach to solve the mathematical model. The homotopy perturbation method is presented below, and we explore its effectiveness in accomplishing this task.

Firstly, consider the classical order procedure for solving coupled system of differential equation

$$\Theta(\upsilon) = m(t), \quad t \in \Phi. \tag{23}$$

Subject to the boundary condition

$$\Omega(\upsilon, \upsilon_n) = 0 \quad t \in \Pi \tag{24}$$

where Θ indicates the operator of differential, Ω represents the boundary condition operator, $m(t)$ denotes the analytic function, and υ_n is the normal vector derivative. The function $\Theta(\upsilon)$ is divided into two which are:

$$\Theta(\upsilon) = L_H(\upsilon) + N_H(\upsilon), \tag{25}$$

$L_H(\upsilon)$ and $N_H(\upsilon)$ denotes the linear and nonlinear operator in the equation.

So, (25) implies

$$L_H(\upsilon) + N_H(\upsilon) = m(t), \quad t \in \Phi. \tag{26}$$

The homotopy for Eq. (26) can be obtained as

$$G(g, p) = (1 - p)\,[L_H(g) - L_H(\upsilon_0)] + p\,[\Theta(g) - m(t)] = 0. \tag{27}$$

where p is an encoding parameter that can change from [0, 1] by a deformation process. Equation (27) is reduced further to yield:

$$G(g,p) = L_H(g) - L_H(\upsilon_0) + p[L_H(\upsilon_0)] + p[N_H(\upsilon_0) - m(t)] = 0, \tag{28}$$

as $p \to 0$, Equation (28) gives:

$$G(g, 0) = L_H(g) - L_H(\upsilon_0) = 0 \tag{29}$$

And when $p \to 1$,

$$G(g, 1) = \Theta(g) - m(t) = 0. \tag{30}$$

Equation (30) can be written in the form of power series and gives

$$g(t) = g_0(t) + pg_1(t) + p^2 g_2(t) + \cdots p^n g_n(t). \tag{31}$$

As a result of combining (31) and (28), and comparing coefficients of equal powers of p,

we have the solution:

$$g(t) = \lim_{p \to 1} g_n(t) = g_0(t) + g_1(t) + g_2(t) + g_3(t) + g_4(t) + \cdots \tag{32}$$

The application of He's homotopy perturbation method (He 1999) is subsequently demonstrated for a system of ordinary differential equations of fractional order using the following algorithm:

The fractional-order derivative of Eq. (2) can be written as:

$$\left.\begin{array}{l} \frac{^C\mathrm{d}^{\alpha_1} S(t)}{\mathrm{d}t^{\alpha_1}} = \Lambda - \mu S(t) - KS(t)I(t) \\[2mm] \frac{^C\mathrm{d}^{\alpha_2} E(t)}{\mathrm{d}t^{\alpha_2}} = KS(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) \\[2mm] \frac{^C\mathrm{d}^{\alpha_3} I(t)}{\mathrm{d}t^{\alpha_3}} = \tau E(t) - (\mu + \eta + \delta)I(t) \\[2mm] \frac{^C\mathrm{d}^{\alpha_4} T(t)}{\mathrm{d}t^{\alpha_4}} = \eta I(t) - (\mu + \theta)T(t) \\[2mm] \frac{^C\mathrm{d}^{\alpha_5} R(t)}{\mathrm{d}t^{\alpha_5}} = \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t) \end{array}\right\}. \tag{33}$$

Using the HPM method as described in Olayiwola et al. (2023) and Abdulaziz et al. (2008), the theory of HPM can be applied to Eq. (33). Consider a system of ordinary differential equations of Caputo fractional order α defined as:

$$\frac{^C\mathrm{d}S_i^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} = f(t, S_1, S_2, S_3, \ldots S_n) \quad i \in N, \tag{34}$$

Constructing a homotopy for Eq. (34) is written as follows:

$$(1 - p)\frac{^C\mathrm{d}S_i^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} - p\left(\frac{^C\mathrm{d}S_i^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} - f(t, S_1, S_2, S_3, \ldots S_n)\right) = 0, \quad i \in N. \tag{35}$$

Simplifying Eq. (35) results in

$$\frac{^c\mathrm{d}S_i^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} = p\big(f(t, S_1, S_2, S_3, \ldots S_n)\big), \tag{36}$$

where $p \in (0, 1)$ is an embedding parameter. At $p = 0$, Eq. (36) becomes linear such that the following equation is obtained:

$$\frac{^c\mathrm{d}S_i^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} = 0. \tag{37}$$

At $p = 1$, the original equation in (37) is obtained. Let's assume a solution series embedding the parameter p for (36) such that

$$S_i(t) = s_{i0} + ps_{i1} + p^2 s_{i2} + p^3 s_{i3}, \ldots p^n s_{in}, \tag{38}$$

Substituting Eq. (38) into Eq. (36) and comparing the coefficients of equal powers of p, the following series of equations is obtained:

$$p^{0:} : \frac{{}^c\mathrm{d}S_{i0}^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} = 0$$

$$p^1 : \frac{{}^c\mathrm{d}S_{i2}^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} = f_{i1}(t, s_{10}, s_{20}, s_{30}, \dots s_{n0}),$$

$$p^2 : \frac{{}^c\mathrm{d}S_{i3}^{\alpha_1}(t)}{\mathrm{d}t^{\alpha_1}} = f_{i2}(t, s_{10}, s_{20}, s_{30}, \dots s_{n0}, s_{11}, s_{21}, s_{31}, \dots s_{n1}),$$

$$(39)$$

and so on. Also, these systems of equation in (39) can be solved by applying the Riemann–Liouville fractional integral operator I^{α_1} to obtain the values of $s_{i1}(t), s_{i2}(t), s_{i3}(t) \dots$. Thus, the solution of (34) is obtained as:

$$S_{iN}(t) = \sum_{k=0}^{N-1} s_{ik}(t). \tag{40}$$

Numerical experiment

This section detailed how the homotopy perturbation technique was used to run the numerical simulation that produced the approximate solution to the SEITR epidemic model.

The SEITR epidemic model's Caputo time fractional-order derivative in (2) is provided as:

$$
\begin{aligned}
{}^C D_t^{\alpha_1} S(t) &= \Lambda - \mu S(t) - KS(t)I(t) \\
{}^C D_t^{\alpha_2} E(t) &= KS(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) \\
{}^C D_t^{\alpha_3} I(t) &= \tau E(t) - (\mu + \eta + \delta)I(t), \\
{}^C D_t^{\alpha_2} T(t) &= \eta I(t) - (\mu + \theta)T(t), \\
{}^C D_t^{\alpha_2} R(t) &= \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t),
\end{aligned}
\tag{41}
$$

where $K = \frac{\beta}{N}$ and ${}^C D_t^{\alpha_i} = \frac{{}^C \mathrm{d}^{\alpha_i}}{\mathrm{d}t^{\alpha_i}}$ for $i = 1, 2, 3, 4, 5$. Constructing a homotopy for (39),

The approximate solution of (41) can be assumed as:

$$
\begin{aligned}
S(t) &= s_0(t) + p\, s_1(t) + p^2\, s_2(t) + \cdots p^n\, s_n(t), \\
E(t) &= e_0(t) + p\, e_1(t) + p^2\, e_2(t) + \cdots p^n\, e_n(t), \\
I(t) &= i_0(t) + p\, i_1(t) + p^2\, i_2(t) + \cdots p^n\, i_n(t), \\
T(t) &= v_0(t) + p\, v_1(t) + p^2\, v_2(t) + \cdots p^n\, v_n(t), \\
R(t) &= r_0(t) + p\, r_1(t) + p^2\, r_2(t) + \cdots p^n\, r_n(t),
\end{aligned}
\tag{43}
$$

substituting (43) for (42) and trying to compare coefficients with identical p powers

$$p^0 : \overset{\bullet}{s_0}(t) = 0,\ \overset{\bullet}{e_0}(t) = 0,\ \overset{\bullet}{i_0}(t) = 0,\ \overset{\bullet}{v_0}(t) = 0,\ \overset{\bullet}{r_0}(t) = 0, \tag{44}$$

Solving Eq. (42) yields

$$s_0(t) = s_0,\ e_0(t) = e_0,\ i_0(t) = i_0,\ v_0(t) = v_0,\ r_0(t) = r_0. \tag{45}$$

Similarly comparing the coefficients of p^1,

$$
\begin{aligned}
\frac{\mathrm{d}^{\alpha_1} S_1(t)}{\mathrm{d}t^{\alpha_1}} &= \Lambda - \mu s_0(t) - Ks_0(t)i_0(t), \\
\frac{\mathrm{d}^{\alpha_2} E_1(t)}{\mathrm{d}t^{\alpha_2}} &= Ks_0(t)i_0(t) - (\mu + \tau + \sigma)e_0(t) + \varphi\, r_0(t), \\
\frac{\mathrm{d}^{\alpha_3} I_1(t)}{\mathrm{d}t^{\alpha_3}} &= \tau\, e_0(t) - (\mu + \eta + \delta)i_0(t), \\
\frac{\mathrm{d}^{\alpha_4} T_1(t)}{\mathrm{d}t^{\alpha_4}} &= \eta i_0(t) - (\mu + \theta)v_0(t), \\
\frac{\mathrm{d}^{\alpha_5} R_1(t)}{\mathrm{d}t^{\alpha_5}} &= \theta v_0(t) + \sigma\, e_0(t) - (\mu + \varphi)r_0(t).
\end{aligned}
\tag{46}
$$

Applying the property of Riemann–Liouville integral (Liouville 1832) in Definition 1, and using solution (45), results in the system of equations,

$$
\begin{aligned}
(1-p)\frac{\mathrm{d}^{\alpha_1} S(t)}{\mathrm{d}t^{\alpha_1}} &= p\left(\frac{\mathrm{d}^{\alpha_1} S(t)}{\mathrm{d}t^{\alpha_1}} + \Lambda - \mu S(t) - KS(t)I(t) \right), \\
(1-p)\frac{\mathrm{d}^{\alpha_2} E(t)}{\mathrm{d}t^{\alpha_2}} &= p\left(\frac{\mathrm{d}^{\alpha_2} E(t)}{\mathrm{d}t^{\alpha_2}} + KS(t)I(t) - (\mu + \tau + \sigma)E(t) + \varphi R(t) \right), \\
(1-p)\frac{\mathrm{d}^{\alpha_3} I(t)}{\mathrm{d}t^{\alpha_3}} &= p\left(\frac{\mathrm{d}^{\alpha_3} I(t)}{\mathrm{d}t^{\alpha_3}} + \tau E(t) - (\mu + \eta + \delta)I(t) \right), \\
(1-p)\frac{\mathrm{d}^{\alpha_4} T(t)}{\mathrm{d}t^{\alpha_4}} &= p\left(\frac{\mathrm{d}^{\alpha_4} T(t)}{\mathrm{d}t^{\alpha_4}} + \eta I(t) - (\mu + \theta)T(t) \right), \\
(1-p)\frac{\mathrm{d}^{\alpha_5} R(t)}{\mathrm{d}t^{\alpha_5}} &= p\left(\frac{\mathrm{d}^{\alpha_5} R(t)}{\mathrm{d}t^{\alpha_5}} + \theta T(t) + \sigma E(t) - (\mu + \varphi)R(t) \right).
\end{aligned}
\tag{42}
$$

$$S_1(t) = (\Lambda - Ks_0i_0 - \mu s_0)\frac{t^{\alpha_1}}{\Gamma(\alpha_1 + 1)}.$$

$$E_1(t) = (Ks_0i_0 - \varphi r_0 + (\sigma + \tau + \mu)e_0)\frac{t^{\alpha_2}}{\Gamma(\alpha_2 + 1)}.$$

$$I_1(t) = (-(\delta + \eta + \mu)i_0 + \tau e_0)\frac{t^{\alpha_3}}{\Gamma(\alpha_3 + 1)}.$$

$$Q_1(t) = (\eta i_0 - (\mu + \theta)v_0)\frac{t^{\alpha_4}}{\Gamma(\alpha_4 + 1)}.$$

$$R_1(t) = (\sigma e_0 + \theta v_0 - (\varphi + \mu)r_0)\frac{t^{\alpha_5}}{\Gamma(\alpha_5 + 1)}$$

$$(47)$$

The coefficients of p^2 equally yield:

$$\frac{d^{\alpha_1}S_2(t)}{dt^{\alpha_1}} = -\mu s_1(t) - Ks_0(t)i_1(t) - Ks_1(t)i_0(t),$$

$$\frac{d^{\alpha_2}E_2(t)}{dt^{\alpha_2}} = Ks_0(t)i_1(t) + Ks_1(t)i_0(t) - (\mu + \tau + \sigma)e_1(t) + \varphi r_1(t),$$

$$\frac{d^{\alpha_3}I_2(t)}{dt^{\alpha_3}} = \tau e_1(t) - (\mu + \eta + \delta)i_1(t),$$

$$\frac{d^{\alpha_4}T_2(t)}{dt^{\alpha_4}} = \eta i_1(t) - (\mu + \theta)v_1(t),$$

$$\frac{d^{\alpha_5}R_2(t)}{dt^{\alpha_5}} = \theta v_1(t) + \sigma e_1(t) - (\mu + \varphi)r_1(t).$$

$$(48)$$

By solving these equations, the second approximation is achieved as follows: $s_2(t) = Ks_0((\sigma + \eta + \mu)i_0 - \tau e_0)\frac{t^{\alpha_3+\alpha_1}}{\Gamma(\alpha_3+\alpha_1+1)} + Ki_0(Ks_0i_0 + \mu s_0 - \Lambda)\frac{t^{2\alpha_1}}{\Gamma(2\alpha_1+1)} + \mu(Ks_0i_0 + \mu s_0 - \Lambda)\frac{t^{2\alpha_1}}{\Gamma(2\alpha_1+1)}$

$$e_2(t) = -Ks_0((\sigma + \eta + \mu)i_0 - \tau e_0)\frac{t^{\alpha_3+\alpha_1}}{\Gamma(\alpha_3 + \alpha_1 + 1)}$$

$$- Ki_0(Ks_0i_0 + \mu s_0 - \Lambda)\frac{t^{\alpha_1+\alpha_2}}{\Gamma(\alpha_1 + \alpha_2 + 1)}$$

$$- \varphi((\mu + \varphi)r_0 - \sigma e_0 - \theta v_0))\frac{t^{\alpha_5+\alpha_2}}{\Gamma(\alpha_5 + \alpha_2 + 1)}$$

$$+ \sigma(-Ks_0i_0 + (\tau + \mu + \sigma)e_0 - \varphi r_0)\frac{t^{2\alpha_2}}{\Gamma(2\alpha_2 + 1)}$$

$$+ \tau(-Ks_0i_0 + (\tau + \mu + \sigma)e_0 - \varphi r_0)\frac{t^{2\alpha_2}}{\Gamma(2\alpha_2 + 1)}$$

$$+ \mu(-Ks_0i_0 + (\tau + \mu + \sigma)e_0 - \varphi r_0)\frac{t^{2\alpha_2}}{\Gamma(2\alpha_2 + 1)}$$

$$i_2(t) = -\tau(-Ks_0i_0 + (\tau + \mu + \sigma)e_0 - \varphi r_0)$$

$$\frac{t^{\alpha_2+\alpha_3}}{\Gamma(\alpha_2 + \alpha_3 + 1)} + \sigma(\sigma i_0 + (\tau + \mu)i_0 - \tau i_0)$$

$$\frac{t^{2\alpha_3}}{\Gamma(2\alpha_3 + 1)} + \eta(\sigma i_0 + (\tau + \mu)i_0 - \tau i_0)\frac{t^{2\alpha_3}}{\Gamma(2\alpha_3 + 1)}$$

$$+ \mu(\sigma i_0 + (\tau + \mu)i_0 - \tau i_0)\frac{t^{2\alpha_3}}{\Gamma(2\alpha_3 + 1)}$$

$$T_2(t) = \mu(-\eta i_0 + (\theta + \mu)v_0)\frac{t^{2\alpha_4}}{\Gamma(2\alpha_4 + 1)}$$

$$- \theta(-\eta i_0 + (\theta + \mu)v_0)\frac{t^{\alpha_2+\alpha_3}}{\Gamma(\alpha_2 + \alpha_3 + 1)}$$

$$- \eta((\sigma i + \eta + \mu)i_0 - \tau e_0)\frac{t^{\alpha_3+\alpha_4}}{\Gamma(\alpha_3 + \alpha_4 + 1)}$$

$$r_2(t) = \mu(\mu r_0 + \varphi r_0 - (\theta - \sigma)v_0)\frac{t^{2\alpha_5}}{\Gamma(2\alpha_5 + 1)} + \varphi(\mu r_0$$

$$+ \varphi r_0 - (\theta - \sigma)v_0)\frac{t^{2\alpha_5}}{\Gamma(2\alpha_5 + 1)}$$

$$- \theta(-\eta i_0 + (\theta + \mu)v_0)\frac{t^{\alpha_4+\alpha_5}}{\Gamma(\alpha_4 + \alpha_5 + 1)}$$

$$- \sigma(-Ks_0i_0 + (\mu + \tau + \sigma)e_0 - \varphi r_0)\frac{t^{\alpha_5+\alpha_2}}{\Gamma(\alpha_5 + \alpha_2 + 1)}$$

where $\alpha_1 = \alpha_2 = \alpha_3 = \alpha_4 = \alpha_5 = \alpha$.

In the same way, subsequent approximations $s_k(t), e_k(t), i_k(t), T_k(t), r_k(x, t), k = 3, 4, \ldots n$ can be computed using MATHEMATICA software.

Results

Numerical simulations and analysis of $\Re_0$.

### Experiment I

Sensitivity analysis on R_0

We analyze the effect of some model parameter on R_0 using the formula presented below and parameter in Table 2

**Table 2** Parameters of the model and their respective values

Parameter	Value	Unit	References
Λ	5.42	yr^{-1}	Schrager et al. (2020)
β	0.0375	yr^{-1}	Ullah et al. (2020a)
σ	0.25	yr^{-1}	Syahrini et al. (2017)
μ	0.018	yr^{-1}	Schrager et al. (2020)
δ	0.19	yr^{-1}	Schrager et al. (2020)
φ	0.0013	yr^{-1}	Tilahun et al. (2020)
θ	0.01	yr^{-1}	Ullah et al. (2023)
τ	0.0019	yr^{-1}	Schrager et al. (2020)
N	206,000,000	yr^{-1}	Ullah et al. (2018)
η	$0 < \eta < 1.0$	yr^{-1}	Estimated
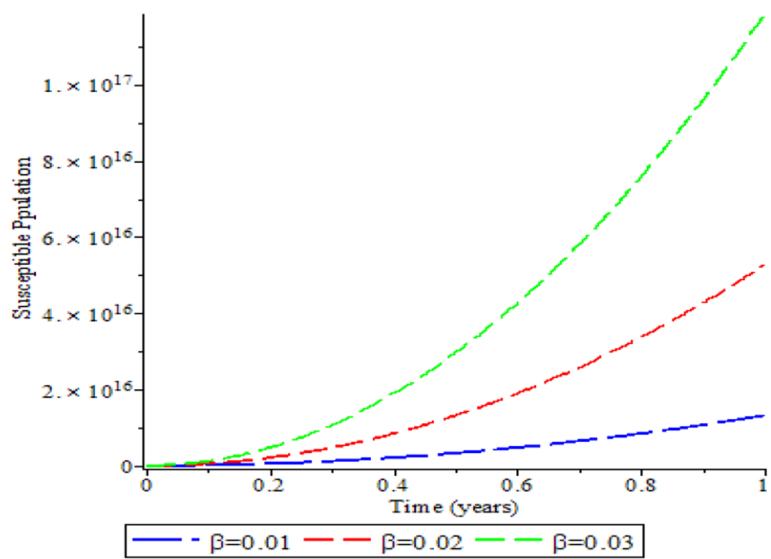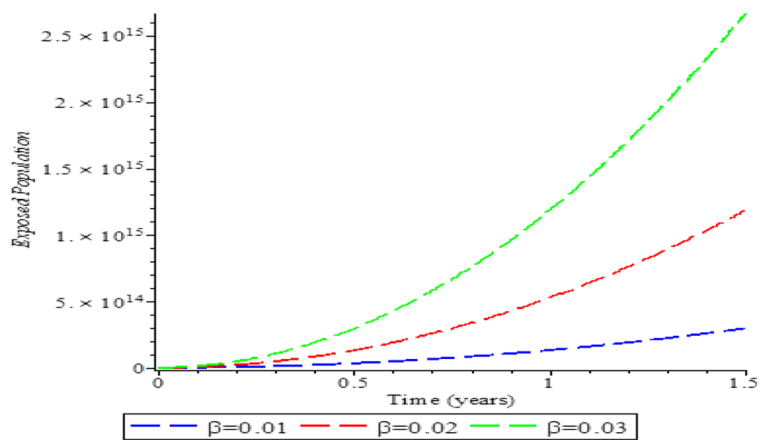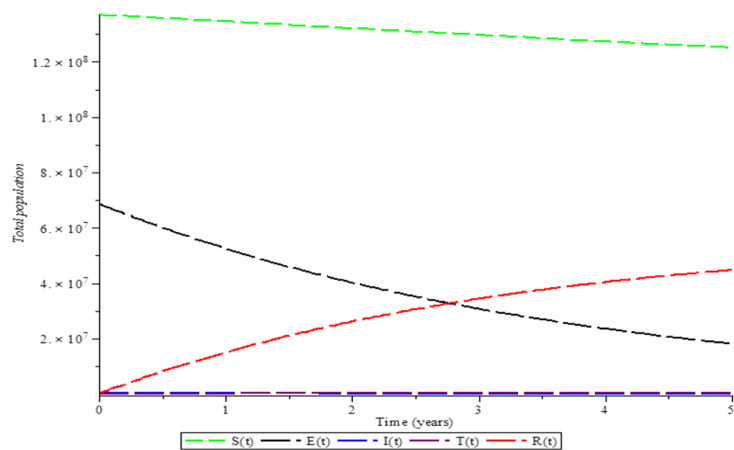
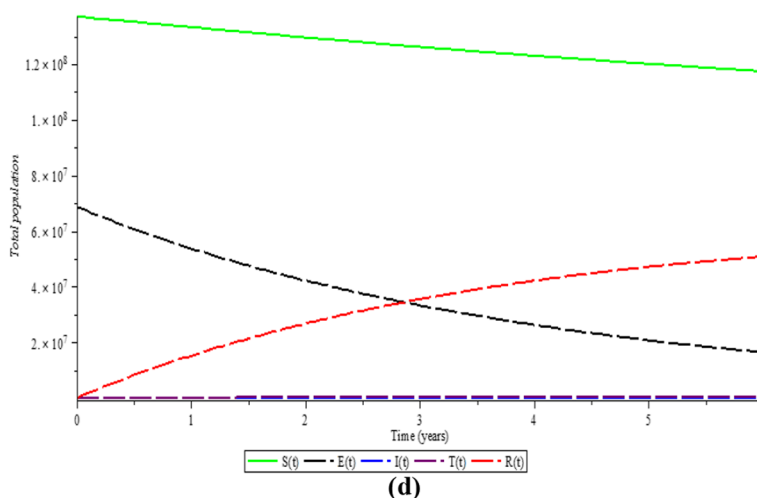**Fig. 2** **a**–**d** Display the behavior of the all population at various β levels

Fig. 2 continued

$$S_F^{R_0} = \frac{\partial R_0}{\partial F} \cdot \frac{F}{\Re_0} \text{ where } F = (\beta, \sigma, \eta, \mu, \delta, \tau). \qquad (49)$$

The sensitivity of parameters η changed in the infected group. This suggests that raising the treatment rate η leads to a fall in the proportion of infected persons seeking treatment, which decreases the R_0. To reduce the virus transmission, we must lower the rate at which exposed individuals become infectious, denoted by σ. Additionally, a decrease in sigma also negatively impacts the basic reproduction number R_0. Hence, the rate should be decreased. Lastly, prioritizing treatment plays a vital role in reducing TB virus transmission effectively.

Experiment II
Numerical result analysis
To eradicate tuberculosis infection, we present the numerical results graphically by applying the homotopy perturbation method to the tuberculosis model. Additionally, we present and assess the result of the effective contact rate (β) for the susceptible, exposed, infected, treated, and recovered groups in Fig. 2.

Discussion
In this research, two experiments were conducted to investigate the dynamic behavior of the tuberculosis model. Experiment II is presented in Figs. 3 and 4, illustrating the impact of the treatment rate η on the susceptible, exposed, infected, treated, and recovered population growth. The graphs revealed a decrease in the susceptible, exposed, and infected populations as the fractional order increased. Interestingly, the treated class showed an increase with higher treatment values, while the recovered class indicated a rise in population, suggesting that many individuals would recover from the disease. Notably, a higher treatment rate resulted in a decrease in the population.

Furthermore, the study analyzed the effect of the contact rate, which revealed that a higher rate led to fewer susceptible individuals becoming infected. Experiment I provided sensitivity index results in Table 3, and Fig. 5 displays the graph of each parameter affecting the basic reproductive number R_0. Through this analysis, the study identified key parameters significantly influencing the growth of the basic reproductive number. The results and graph demonstrated that increasing the treatment rate η correlated with a decline in the proportion of infected individuals seeking treatment, consequently reducing the reproductive number. Additionally, the rate at which exposed individuals become infectious τ positively impacts R_0, highlighting the need to reduce the value of τ to control virus spread. On the other hand, σ had a negative effect on the reproductive number, indicating that this rate should be decreased as well.

Stability analysis for both local and global equilibrium points was conducted. The disease-free equilibrium was found to be locally asymptotically stable when R_0 using the Routh–Hurwitz criterion for stability, implying that the tuberculosis disease would be eradicated from the population in this case. Furthermore, the endemic equilibrium was confirmed to be globally stable using the Lyapunov function, and no other equilibrium points were identified.

In conclusion, the study highlights that efficient treatment is a crucial control measure for eradicating tuberculosis. By understanding the impact of various parameters on the disease dynamics, we can develop more effective strategies to combat tuberculosis and minimize its spread in the population.
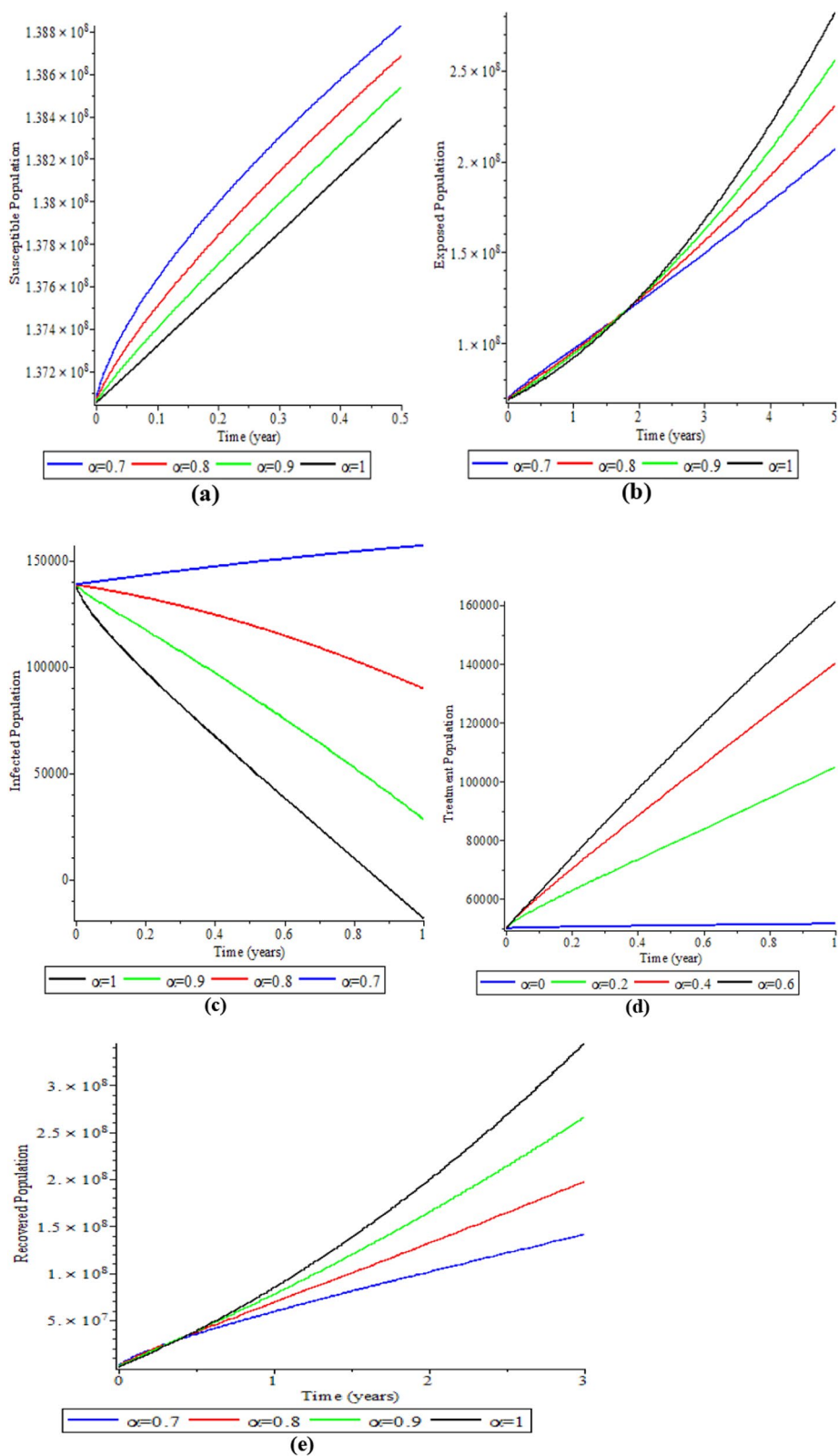
Fig. 3 **a**–**e** The dynamic effect of tuberculosis model with control and without control $\eta = 0, 0.3, 0.6, 0.9$ at different values of order a
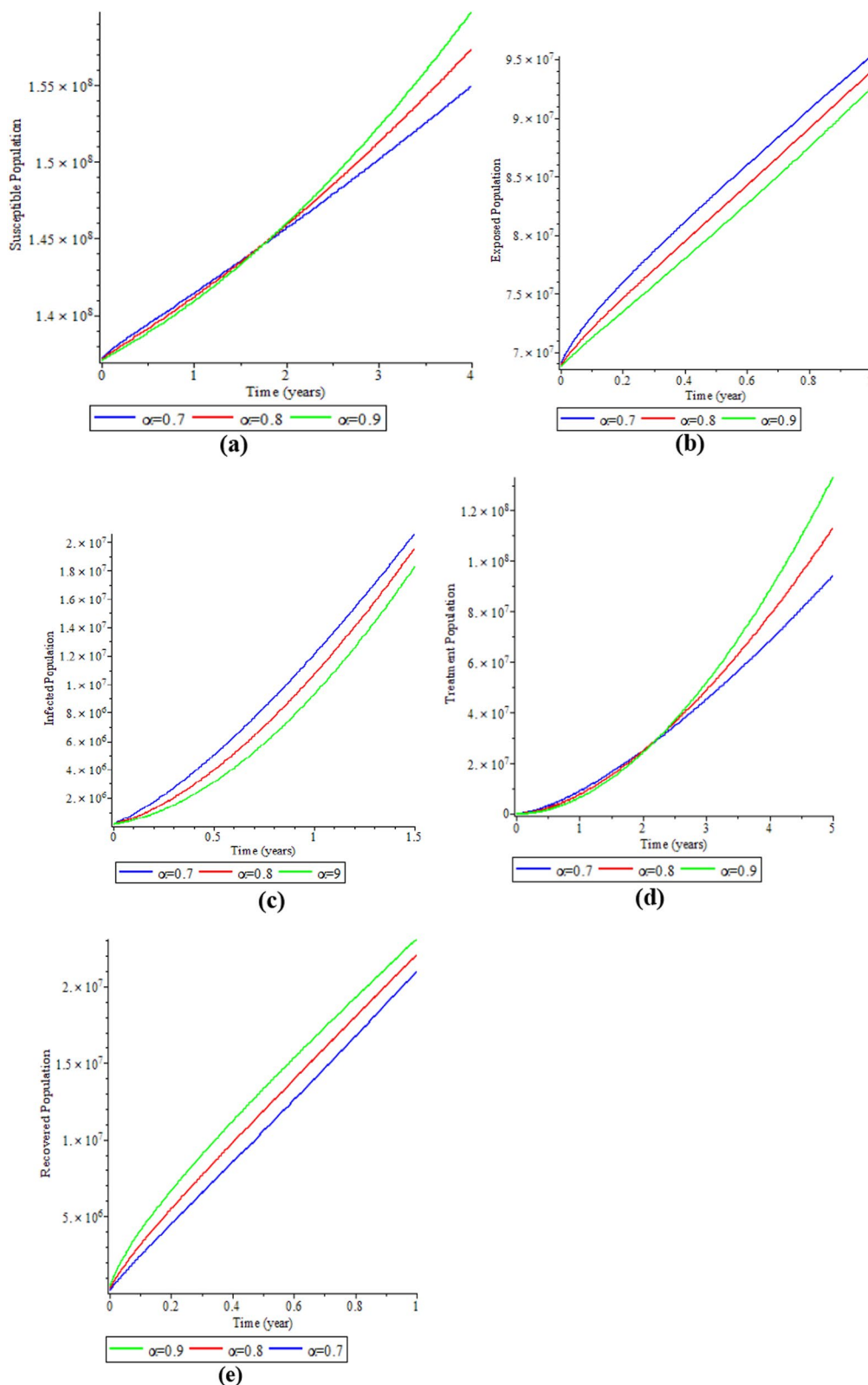
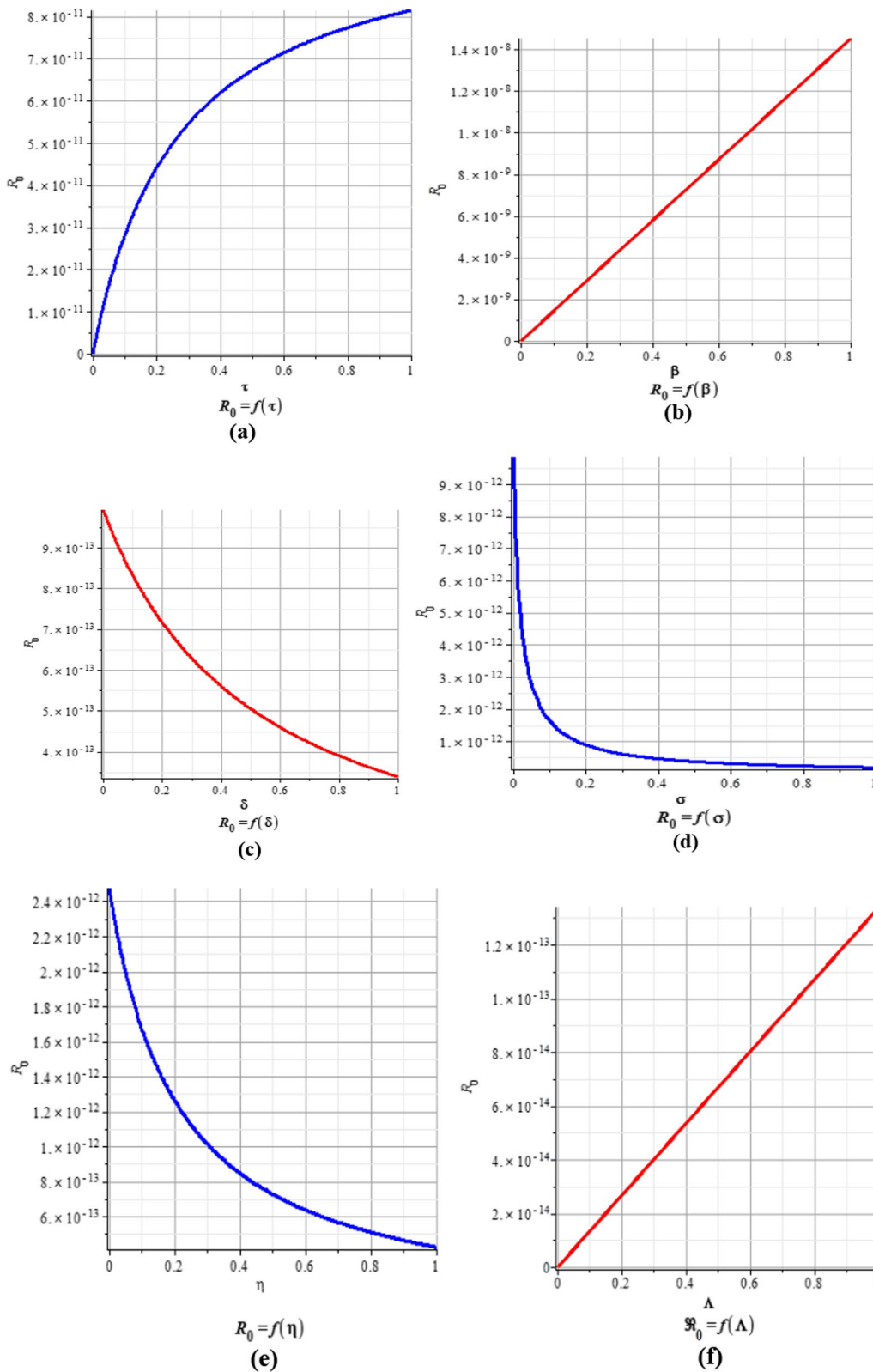**Fig. 4 a–e** The dynamic behavior of a fractional TB model with control $\eta = 10$ for various α values

Fig. 5 **a**–**f** Display the impact of each parameter on R_0

Table 3 Sensitivity indices of each parameter on R_0

Parameters	Value
β	1
σ	-0.926268988
η	-0.706214689
μ	-1.092115097
δ	-0.268361582
τ	0.9929603563

Conclusions

This study introduces a mathematical model of tuberculosis (TB) along with a control measure to assess the effectiveness of interventions. The focus lies on a fractional-order Caputo model for TB, enabling a measurement of control methods' impact. In our analysis, we conducted disease-free and endemic equilibrium, positivity and boundedness assessments of the solution, as well as local and global stability evaluations. Additionally, we explored the basic reproduction number and performed sensitivity analysis. The results consistently highlighted the critical role of early and effective treatment in curbing the spread of this serious disease. To accurately estimate the time fractional derivative, a reliable numerical method based on the homotopy perturbation method was developed. The numerical findings were visually depicted through graphs, providing valuable insights. In conclusion, our study emphasizes the importance of seeking timely diagnosis and treatment for individuals affected by diseases to prevent further transmission within society. The control measure proposed in the model underscores the significance of proactive measures in managing and controlling tuberculosis.

Abbreviations
HPM   Homotopy perturbation method
TB   Tuberculosis
R_0 Reproductive number
DFE   Disease-free equilibrium
EE   Endemic equilibrium
SEITR   Susceptible–exposed–infected–treatment–recovered

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