



Research article

A dynamical approach for a fractional-order SEIL epidemic model embedding time delay

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Abstract: In this paper, we suggest a fractional-order susceptible, exposed, infectious, and latent (SEIL) epidemic model with discrete time delays that provide for biological factors and memory effects in the spread of disease. Caputo derivatives and fixed-point theory are implemented, and we identify the existence and uniqueness of solutions, as well as the basic reproduction number for both endemic and disease-free equilibria. Local stability is analyzed through characteristic equations and linearization, while numerical simulations confirm theoretical results and illustrate the influence of fractional order, delays, and parameters. The findings show that fractional-delay models provide a more flexible and effective framework for studying disease dynamics and control.

Keywords: time-delay system; Caputo fractional derivatives; stability analysis; basic reproduction number; optimal control; Pontryagin maximum principle

Mathematics Subject Classification: 34A08, 34A12, 34D20, 49J15

1. Introduction

Tubercle bacillus, the germ that causes the disease of tuberculosis (TB), continues to pose a serious threat to human health, accounting for 2–3 million deaths globally in a year. Each structure inside the body, including the brain, intestines, kidneys, bones, endometrium, and more, may develop tuberculosis. Tubercle bacillus infection is the main contributory factor of tuberculosis infections, which account for quite a few of all deaths [9]. Although it is now within the most substantial, prevalent, and fatal infectious diseases in the world, tuberculosis represents one of the very first health problems that were actually recognized. Patients with tuberculosis can live healthy lives for months without showing any signs of the disease. The immune system of the patient tackles the Tubercle bacillus bacteria in the middle of this time to try and prevent the respiratory infection from spreading [39].

In this work, we introduce a general fractional-order system, and we include control theory related to the dynamic initialization response in [16]. Hariharan and Shangeranesh [14] studied a specialized fractional-order mathematical model to determine the relationship between obesity and cancer and developed a treatment strategy. A generalized status of an integer order differential equation is called a fractional order differential equation (FODE). It can be useful in many fields, including pure and applied research, where it can be utilized as a physical model for various processes [3, 30]. In [34], this paper implemented the Caputo derivatives to develop a fractional-order susceptible, vaccinated, exposed, infectious, and recovered (SVEIR) model for African swine fever virus in order to investigate this type of behavior. Fractional derivatives of Riemann–Liouville were introduced in [1, 13]. A preventive model of prey-predator infection in both animal populations that focuses on stability analysis and the most effective control approach for managing the spread of infectious diseases was examined in [15].

Many biological and technological fields have demonstrated various applications for fractional derivatives and integrals of this Riemann–Liouville kind. These consist of research on complex phenomena such as vibration, diffusion, controllability, the Stokes problem, thermoelasticity, and bioengineering [4, 8, 22]. Time delays and fractional derivatives are two elements of delay fractional differential equations (DFDEs). Determining infectious periods in several kinds of epidemic models has been effectively modeled using delay differential equations. Even though some epidemic models utilize fractional-order systems without any kind of time delay, almost all delay systems of equations include integer-order derivatives. There are a lot of outstanding inquiries concerning the theory of the several impulsive delay differential equations that have been studied in [22, 27].

The stability of a multiple DFDE system was examined by Deng et al. in [11]. In [36], scrutinized susceptible, infectious, and susceptible (SIS) models with a time delay in the infectious class and an acceptable range of sizes for each individual. Furthermore, in constant population size models of the SIS type with delays, asymptotically stable endemic equilibria have been identified. The phenomenon of a non-linear system of delay differential equations involved in the stability analysis of equilibrium points was discussed by Ruan in [31]. Yang and Xu [38] constructed a TB model by taking into account both new and recurring infections. Paul et al. [27] investigated both the delayed and the stability of the disease-free and endemic equilibrium points of the non-delayed model. Kar and Mondal [23] mentioned additional recurrent infections, domestic growth, and infection again within the treated in a basic model of tuberculosis. Kim and Jung [19] presented the optimum control methods with

minimal implementation costs by reducing the number of infected and highly dangerous latent cases of tuberculosis.

Bashier [7] developed an optimal control model by applying delay differential equations based on the SIR epidemic time delay model. The fractional delay model has been examined by several researchers using analytical and numerical methods. As a result, the primary goal of the researchers is to develop an analytical or numerical solution to DFDEs. Therefore, a variety of methods have been used, including the Adams–Bashforth–Moulton algorithm [5], new iterative method [6], Chebyshev pseudospectral method [35], Legendre–Gauss collocation method [29], operational matrix based on poly-Bernoulli polynomials [28], and Runge Kutta-type method [32]. Although accurate solutions are a fundamental reference, as demonstrated by the range of approaches and their evaluations of comparisons, the development and improvement of numerical techniques are generally still necessary for solving DFDEs with high accuracy and effectiveness.

Novelty of the work: The authors choose the fractional-order susceptible, exposed, infectious, and latent (SEIL) model with discrete delay, because it offers an additional and biologically accurate framework for researching the interactions between infectious diseases. This model takes memory and hereditary characteristics into consideration, which becomes acceptable by the use of Caputo fractional derivatives. In comparison to standard integer-order systems, the model's accuracy is increased by including the time delay, which takes into consideration the incubation and latency periods that are prevalent in many epidemics. The model provides more flexibility in understanding complicated epidemic behaviours by identifying many stages of disease transmission through the dividing of the population into susceptible, exposed, infectious, and latent compartments.

In this research, in order to better simulate biological processes and memory in disease transmission, we develop and examine a fractional-order SEIL epidemic model with time delays. We use fixed-point theorems to demonstrate the existence and uniqueness of solutions using Caputo derivatives of order. Pontryagin's maximum principle for fractional systems with delay is used to frame an optimal control problem and determine the basic reproduction number, disease-free, and endemic equilibria. Linearization and characteristic equations are used to analyze stability, and numerical simulations justify the theoretical findings. The results demonstrate that fractional-order models with delays present a dynamic and valuable framework for analyzing and controlling the development of infectious diseases.

The structure of the paper is as follows: In Section 2, the proposed model is formulated. In Section 3, the boundedness, existence, and uniqueness of the fractional delay order problem are illustrated. In Section 4, the equilibria and reproduction number part are established. In Section 5, the local stability of the equilibrium points is obtained. In Section 6, the optimum controllability for the system of equations with control terms is determined. In Section 7, the numerical analysis of the model is examined, and the mathematical results of the fractional derivative are presented.

2. Model formulation

In this section, we establish a model of fractional-order infection with tuberculosis over an infected population. The proposed model consists of four components, namely $S(t)$, $E(t)$, $I(t)$, and $L(t)$.

$$\begin{cases} {}^c D_t^\alpha S(t) = \Lambda - \mu S(t) - \beta S(t)I(t), \\ {}^c D_t^\alpha E(t) = \beta S(t)I(t) - (\mu + \vartheta + \varphi)E(t) + p(t)\nu I(t) + \psi L(t), \\ {}^c D_t^\alpha I(t) = \varphi E(t) - (\nu + \mu)I(t), \\ {}^c D_t^\alpha L(t) = (1 - p(t))\nu I(t) + \vartheta E(t) - (\mu + \psi)L(t). \end{cases} \quad (2.1)$$

Here, $0 < \alpha \leq 1$, where, $N = S + E + I + L$. ${}^c D_t^\alpha$ represents the Caputo fractional derivative of order α . According to the suggested methodology, people are categorized as susceptible (S), exposed (or high-risk latent) (E), infectious (I) from active TB, and low-risk (L) from permanently latent TB infection.

Symbol	Description
Λ	Recruitment rate through birth or immigration into the susceptible class
μ	Natural death rate from the susceptible class
β	Infection rate due to contact with infectious individuals
φ	Progression rate from exposed (E) to infectious (I) class
ϑ	Rates at which individuals in People do not move from class E to class I.
ν	Rates at which individuals in E are treated and move to treated class (L)
ψ	Relapse rate from treated class (L) back to exposed class (E)
$p(t)$	Fraction of infectious people who delay their treatment
$1 - p(t)$	Rate of success in treatment for active TB

A well-known occurrence in fractional calculus is the theory of the time DFDEs. The given conventional time delay differential equation system can therefore be generalized into the time DFDEs system as follows. The time delay ω is identical to the tuberculosis latent period. The following is the outline of our calculated fractional-order model of tuberculosis disease transmission with time delay.

$$\begin{cases} {}^c D_t^\alpha S(t) = \Lambda - \mu S(t) - \beta S(t)I(t), \\ {}^c D_t^\alpha E(t) = \beta S(t)I(t) - (\mu + \vartheta + \varphi)E(t) + p(t)\nu I(t - \omega) + \psi L(t), \\ {}^c D_t^\alpha I(t) = \varphi E(t) - \nu I(t - \omega) - \mu I(t), \\ {}^c D_t^\alpha L(t) = \nu I(t - \omega) - p(t)\nu I(t - \omega) + \vartheta E(t) - (\mu + \psi)L(t), \end{cases} \quad (2.2)$$

subject to initial conditions, $S(0) = S_0$, $E(0) = E_0$, $I(x) = I_0$, and $L(0) = L_0$, where $x \in [-\omega, 0]$.

All state variables $S(t)$, $E(t)$, $I(t)$, and $L(t)$ are assumed to be non-negative for all $t \geq 0$. The delay term $I(t - \omega)$ is incorporated to represent delays in diagnosis and treatment, which are significant in tuberculosis dynamics and biologically meaningful only for $\omega > 0$. The initial conditions are prescribed as continuous functions on $[-\omega, 0]$, ensuring the well-posedness of the system in the space of continuous functions. Furthermore, each term in the model preserves dimensional consistency. The recruitment, transmission, progression, and removal terms are constructed to ensure that the flow between compartments is balanced and biologically interpretable. The infection term $\beta S(t)I(t)$ is assumed to be instantaneous, reflecting immediate transmission upon contact, as commonly adopted in epidemic models.

The latent (incubation) period is already captured through the compartmental progression $S \rightarrow E \rightarrow I$, where $E(t)$ represents the exposed class. Hence, introducing an additional delay in the infection term may lead to redundancy.

3. Well-posedness

The system (2.2) now becomes

$$\begin{aligned} {}^c D_t^\alpha S(t) &= F_1[t, S(t), I(t)], \\ {}^c D_t^\alpha E(t) &= F_2[t, E(t), S(t), I(t), L(t), I(t - \omega)], \\ {}^c D_t^\alpha I(t) &= F_3[t, I(t), E(t), I(t - \omega)], \\ {}^c D_t^\alpha L(t) &= F_4[t, L(t), E(t), I(t - \omega)], \end{aligned}$$

along with the initial conditions considered as

$$S(0) = \phi_1, E(0) = \phi_2, I(x) = \phi_3, L(0) = \phi_4. \quad (3.1)$$

The fractional time delay equation is an attribute that we examine. We describe the analysis for $I(t)$, and it will be similar for the other equations in system (2.2):

$${}^c D_t^\alpha I(t) = F_3[t, I(t), E(t), I(t - \omega)], t \in [0, T], 0 < \alpha \leq 1, \quad (3.2)$$

with the initial conditions,

$$I(t) = \phi_3, \quad t \in [-\omega, 0], \quad (3.3)$$

where $I \in \mathbb{R}^n, T > 0$, and $F_3 : [0, T] \times \mathbb{R}^n \times \mathbb{R}^n$ into \mathbb{R}^n is continuous.

Lemma 3.1. *According to Cong and Tuan [10], if a function $\phi \in C([-\omega, T]; \mathbb{R}^n)$ is a solution of the delay integral equation, then it is a solution of the initial value problem (IVP) (3.2) and (3.3) on the interval $[-\omega, T]$:*

$$I(t) = I(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \eta)^{\alpha-1} F_3(\eta, I(\eta), I(\eta - \omega)) d\eta, \forall t \in [0, T], \quad (3.4)$$

with the initial condition,

$$I(t) = \phi_3, \quad t \in [-\omega, 0]. \quad (3.5)$$

Theorem 3.1. *Assume that $F_3 : [0, T] \times \mathbb{R}^n \times \mathbb{R}^n$ into \mathbb{R}^n is continuous and in agreement with the non-delay variable's Lipschitz condition as follows. There is a continuous function $L : [0, T] \times \mathbb{R}^n \rightarrow \mathbb{R}_{\geq 0}^+$ that is non-negative:*

$$\|F_3(t, I, Z) - F_3(t, I_1, Z)\| \leq L(t, Z) \|I - I_1\|, \forall t \in [0, T], I, Z, I_1 \in \mathbb{R}^n. \quad (3.6)$$

Further, a particular distributed solution ϕ exists for the IVP (3.2) and (3.3) on the interval $[-\omega, T]$.

Proof. The IVP (3.2)–(3.3) is identical to (3.2) with (3.4), as per Lemma (3.1). Initially, we consider for a time such that $0 < T \leq \omega$, then (3.4) indicates

$$I(t) = I(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \eta)^{\alpha-1} F_3(\eta, I(\eta), I(\eta - \omega)) d\eta, \forall t \in [0, T].$$

On the interval $[0, T]$, there is only one solution to this integral equation, according to Cong and Tuan [10]. We obtain the result by m_ω^* and establish,

$$\phi_T(t, m_3) = \begin{cases} m_3, & t \in [-\omega, 0], \\ m_\omega^*(t), & t \in [0, T]. \end{cases} \quad (3.7)$$

Hence, the unique solution to problems (3.4)–(3.5) on $[-\omega, T]$ is $\phi_T(t, \phi_3)$. So, in a different case where $T > \omega$, we organize the interval $[0, T]$ into $[0, \omega] \cup \dots \cup [(k_0 - 1)\omega, k_0\omega] \cup [k_0\omega, T]$, where $k_0 \in \mathbb{N}$ and $0 \geq k_0\omega < \omega$. Following the same views previously, we are able to identify a unique solution of the IVP (3.4)–(3.5), which is established by ϕ_ω on the interval $[-\omega, \omega]$. We employ induction to demonstrate the occurrence of the interval's unique solution $[-\omega, k_0\omega]$. Assuming that there is a unique solution to problems (3.4)–(3.5) on the interval $[-\omega, k_\omega]$ for any $1 \leq k < k_0$, we indicate the solution by $\phi_{k\omega}(m_3)$. We define an operator $G_{(k+1)\omega, m_3} : C([k\omega, (k+1)\omega]; \mathbb{R}^n) \rightarrow C([k\omega, (k+1)\omega]; \mathbb{R}^n)$ on $[k\omega, (k+1)\omega]$ as follows:

$$\begin{aligned} (G_{(k+1)\omega, m_3} m)(t) &= I(0) + \frac{1}{\Gamma(\alpha)} \int_0^{k\omega} (t - \eta)^{\alpha-1} F_3(\eta, \phi_{k\omega}(\eta, m_3), \phi_{k\omega}(\eta - \omega, m_3)) d\eta \\ &+ \frac{1}{\Gamma(\alpha)} \int_{k\omega}^t (t - \eta)^{\alpha-1} F_3(\eta, m(\eta), \phi_{k\omega}(\eta - \omega, m_3)) d\eta, \quad \forall t \in [k\omega, (k+1)\omega], \end{aligned}$$

Assume that β_k is a positive constant such that $\beta_k > 2 \max_{t \in [k\omega, (k+1)\omega]} L(t, \phi_{k\omega}(t - \omega, m_3))$. We define an innovative approach d_{β_m} on the space $C([k\omega, (k+1)\omega]; \mathbb{R}^n)$.

$$d_{\beta_k}(m, m_1) = \sup_{t \in [k\omega, (k+1)\omega]} \frac{\|m(t) - m_1(t)\|}{H_\alpha(\beta_k t^\alpha)}, \quad \forall m, m_1 \in C([k\omega, (k+1)\omega]; \mathbb{R}^n),$$

where the Mittag–Leffler function is expressed as $H_\alpha : \mathbb{R} \rightarrow \mathbb{R}$. As soon as it is equipped with the metric d_{β_k} , the space $C([k\omega, (k+1)\omega]; \mathbb{R}^n)$ is complete. We illustrate that on $(C([k\omega, (k+1)\omega]; \mathbb{R}^n), d_{\beta_k})$, the operator $G_{(k+1)\omega, m_3}$ is contractive. In particular, for any $t \in (k\omega, (k+1)\omega)$ and any $m, m_1 \in C([k\omega, (k+1)\omega]; \mathbb{R}^n)$, we have

$$\begin{aligned} \|(G_{(k+1)\omega, m_3} m)(t) - (G_{(k+1)\omega, m_3} m_1)(t)\| &\leq \frac{\max_{t \in [k\omega, (k+1)\omega]} L(t, \phi_{k\omega}(t - \eta, m_3))}{\Gamma(\alpha)} \int_{k\omega}^t (t - \eta)^{\alpha-1} \|m(\eta) - m_1(\eta)\| d\eta, \\ &\leq \frac{\max_{t \in [k\omega, (k+1)\omega]} L(t, \phi_{k\omega}(t - \omega, m_3))}{\Gamma(\alpha)} \int_{k\omega}^t (t - \eta)^{\alpha-1} H_\alpha(\beta_k \eta^\alpha) \frac{\|m(\eta) - m_1(\eta)\|}{H_\alpha(\beta_k \eta^\alpha)} d\eta. \end{aligned} \quad (3.8)$$

This implies that,

$$\begin{aligned} \frac{\|(G_{k\omega, m_3} m)(t) - (G_{k\omega, m_3} m_1)(t)\|}{H_\alpha(\beta_k t^\alpha)} &\leq \frac{\max_{t \in [k\omega, (k+1)\omega]} L(t, \phi_{k\omega}(t - \omega, m_3))}{H_\alpha(\beta_k t^\alpha)} d_{\beta_k}(m, m_1) \frac{1}{\Gamma(\alpha)} \int_{k\omega}^t (t - \eta)^{\alpha-1} H_\alpha(\beta_k \eta^\alpha) d\eta, \\ &\leq \frac{\max_{t \in [k\omega, (k+1)\omega]} L(t, \phi_{k\omega}(t - \omega, m_3))}{H_\alpha(\beta_k t^\alpha)} d_{\beta_k}(m, m_1) \frac{1}{\Gamma(\alpha)} \int_0^t (t - \eta)^{\alpha-1} H_\alpha(\beta_k \eta^\alpha) d\eta, \end{aligned} \quad (3.9)$$

$$\begin{aligned} &\leq \frac{\max_{\mathbf{t} \in [k\omega, (k+1)\omega]} L(\mathbf{t}, \phi_{k\omega}(\mathbf{t} - \omega, \mathbf{m}_3))}{H_\alpha(\beta_k \mathbf{t}^\alpha)} d_{\beta_k}(\mathbf{m}, \mathbf{m}_1) I_0^\alpha \left({}^c D_{\mathbf{t}}^\alpha \left(\frac{H_\alpha(\beta_k \eta^\alpha)}{\beta_k} \right) \right), \\ &\leq \frac{\max_{\mathbf{t} \in [k\omega, (k+1)\omega]} L(\mathbf{t}, \phi_{k\omega}(\mathbf{t} - \omega, \mathbf{m}_3))}{\beta_k} d_{\beta_k}(\mathbf{m}, \mathbf{m}_1). \end{aligned}$$

For all $\mathbf{t} \in [k\omega, (k+1)\omega]$,

$$\begin{aligned} d_{\beta_k}(G_{(k+1)\omega, \mathbf{m}_3} \mathbf{m}, G_{(k+1)\omega, \mathbf{m}_3} \mathbf{m}_1) &\leq \frac{\max_{\mathbf{t} \in [k\omega, (k+1)\omega]} L(\mathbf{t}, \phi_{k\omega}(\mathbf{t} - \omega, \mathbf{m}_3))}{\beta_k} d_{\beta_k}(\mathbf{m}, \mathbf{m}_1), \\ &\leq \frac{1}{2} d_{\beta_k}(\mathbf{m}, \mathbf{m}_1), \end{aligned} \quad (3.10)$$

for all $\mathbf{m}, \mathbf{m}_1 \in C([k\omega, (k+1)\omega]; \mathbb{R}^n)$. According to a structure of the Banach fixed-point theorem, each fixed point is distinct $\mathbf{m}_{(k+1)\omega}^* \mathcal{G}_{(k+1)\omega, \mathbf{m}_3}$ in $C([k\omega, (k+1)\omega]; \mathbb{R}^n)$. Put

$$\phi_{(k+1)\omega}(\mathbf{t}, \mathbf{m}_3) = \begin{cases} \phi_{k\omega}(\mathbf{t}, \mathbf{m}_3), \forall \mathbf{t} \in [-\omega, k\omega], \\ \mathbf{m}_{(k+1)\omega}^*(\mathbf{t}), \forall \mathbf{t} \in [k\omega, (k+1)\omega]. \end{cases} \quad (3.11)$$

In that case, the unique solution of the problem (3.4)–(3.5) is $\phi_{(m+1)\omega}(\mathbf{t}, \mathbf{m}_3)$ on $[-\omega, (k+1)\omega]$. Subsequently, we obtain a controller $\mathcal{G}_{\mathbf{m}_3} : C([k_0\omega, T]; \mathbb{R}^n) \rightarrow C([k_0\omega, T]; \mathbb{R}^n)$ on the interval $[k_0\omega, T]$ by

$$\begin{aligned} (\mathcal{G}_{\mathbf{m}_3})(\mathbf{t}) &= \mathbf{I}(0) + \frac{1}{\Gamma(\alpha)} \int_0^{k_0\omega} (\mathbf{t} - \eta)^{\alpha-1} F_3(\eta, \phi_{k_0\omega}(\eta, \mathbf{m}_3), \phi_{k_0\omega}(\eta - \omega, \mathbf{m}_3)) d\eta \\ &\quad + \frac{1}{\Gamma(\alpha)} \int_{k_0\omega}^{\mathbf{t}} (\mathbf{t} - \eta)^{\alpha-1} F_3(\eta, \mathbf{m}(\eta), \phi_{k_0\omega}(\eta - \omega, \mathbf{m}_3)) d\eta, \forall \mathbf{t} \in [k_0\omega, T]. \end{aligned}$$

Since β_{k_0} is a positive constant, let $\beta_{k_0} > 2 \max_{\mathbf{t} \in [k_0\omega, T]} L(\mathbf{t}, \phi_{k_0\omega}(\mathbf{t} - \omega, \mathbf{m}_3))$. An innovative approach $d_{\beta_{k_0}}$ is defined for the space $C([k_0\omega, T]; \mathbb{R}^n)$:

$$d_{\beta_{k_0}}(\mathbf{m}, \mathbf{m}_1) := \sup_{\mathbf{t} \in [k_0\omega, T]} \frac{\|\mathbf{m}(\mathbf{t}) - \mathbf{m}_1(\mathbf{t})\|}{H_\alpha(\beta_{k_0} \mathbf{t}^\alpha)}.$$

We have proven the controller \mathbf{m}_3 contains a particular fixed point by restating the previous arguments. Identify a function,

$$\phi_T(\mathbf{t}, \mathbf{m}_3) = \begin{cases} \phi_{k_0\omega}(\mathbf{t}, \mathbf{m}_3), \forall \mathbf{t} \in [-\omega, k_0\omega], \\ \mathbf{m}^*(\mathbf{t}), \forall \mathbf{t} \in [k_0\omega, T]. \end{cases}$$

Clearly, ϕ_T is the only solution to problem (3.4)–(3.5) on the interval $[-\omega, T]$. \square

We will now examine a response to system (2.2)'s boundedness and non-negativeness. Define the biologically meaningful set

$$\Omega = \left((S, E, I, L) \in \mathbb{R}_+^4 : S + E + I + L \leq \frac{\Lambda}{\mu} \right).$$

Here, $S(t)$ remains non-negative if $S(0) \geq 0$. Although the equations include non-negative elements, This remains non-negative for $E(t)$, $I(t)$, and $L(t)$, as long as the initial conditions and delayed terms $I(t - \omega)$ are also non-negative.

Let us consider $N(t) = S(t) + E(t) + I(t) + L(t)$. By taking the α^{th} fractional derivative, we get

$${}^c D_t^\alpha N(t) = {}^c D_t^\alpha S(t) + {}^c D_t^\alpha E(t) + {}^c D_t^\alpha I(t) + {}^c D_t^\alpha L(t).$$

Through the implementation of the model (2.2),

$${}^c D_t^\alpha N(t) = \Lambda - \mu N. \quad (3.12)$$

The Laplace transform is executed to determine the equation, and thus we obtain

$$N(t) = \left(N(0) - \frac{\Lambda}{\mu} \right).$$

Thus,

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}. \quad (3.13)$$

Using a fractional version of the Gronwall inequality, the solutions are ultimately bounded by $\frac{\Lambda}{\mu}$. Thus, S , E , I , and L are all bounded, and the system (2.2) is positive invariant.

4. Equilibrium points and basic reproduction number

This section determines the system's equilibrium point first, and then employs the disease-free equilibrium (DFE) point to get the basic reproduction number for the proposed model.

The model (2.2) that has been suggested includes two non-negative equilibrium points: $E = I = L = 0$ in DFE and $E \neq 0$, $I \neq 0$, and $L \neq 0$ in endemic equilibrium (EE) [18]. The equilibrium points are obtained as

$$E_1 = \left[\frac{\Lambda}{\mu}, 0, 0, 0 \right], \quad (4.1)$$

and

$$E_2 = \left\{ \frac{\Lambda}{\mu + \beta I^*}, \frac{(v + \mu)}{\varphi} I^*, I^*, \frac{(1 - p)v\varphi + (v + \mu)\vartheta}{\varphi(\mu + \psi)} I^* \right\}. \quad (4.2)$$

Here,

$$I^* = \frac{\Lambda\phi(\mu + \psi)}{\mu(\mu^2 + \phi\psi + \mu(v + \phi + \psi + \varphi) + v(\phi - p\phi + \psi + \varphi))} - \frac{\mu}{\beta},$$

where E_1 refers to the DFE point and E_2 refers to the EE point.

Although the system involves a discrete time delay ω , the delay appears only in the transition (progression) terms and does not directly affect the new infection terms. Therefore, the basic reproduction number R_0 can still be derived using the standard next-generation matrix [33,36] approach by considering the infection subsystem at the DFE.

Let \mathcal{F} and \mathcal{V} denote the matrices of new infection terms and transition terms, respectively. Then, the basic reproduction number is given by

$$R_0 = r(\mathcal{F}\mathcal{V}^{-1}),$$

where $r(\cdot)$ denotes the spectral radius.

Since the delay does not appear in \mathcal{F} , it does not influence the value of R_0 , but only affects the transient dynamics and stability behavior of the system. Utilizing the affected populations (E, I, and L), the Jacobian is as follows:

$$J = \begin{bmatrix} -(\mu + \vartheta + \varphi) & \beta S + p\nu & \psi \\ \varphi & -(\nu + \mu) & 0 \\ \vartheta & (1 - p)\nu & -(\mu + \psi) \end{bmatrix}.$$

From the above J, consider F and V as the primary infection and out-flow population as mentioned below:

$$F = \begin{bmatrix} 0 & \beta S & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} (\mu + \vartheta + \varphi) & -p\nu & -\psi \\ -\varphi & (\nu + \mu) & 0 \\ -\vartheta & -(1 - p)\nu & (\mu + \psi) \end{bmatrix}.$$

We can obtain the next generation matrix FV^{-1} as given below:

$$FV^{-1} = \begin{bmatrix} \frac{\beta S \varphi (\mu + \psi)}{K} & \frac{\beta S (\mu + \varphi) (\mu + \psi) + \mu \vartheta}{K} & \frac{\beta S \varphi \psi}{K} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

Here, $K = \mu^2(\mu + \varphi + \vartheta + \psi + \nu) + \mu\nu((1 - p)\varphi + \psi + \vartheta) + \mu\psi\varphi$. The basic reproduction number is the spectrum radius of the matrix described above, and is written as

$$R_0 = r(FV^{-1}) = \frac{\beta S^* \varphi (\mu + \psi)}{K}.$$

At the DFE, where $S^* = \frac{\Lambda}{\mu}$,

$$R_0 = \frac{\beta S^* \varphi (\mu + \psi)}{K}.$$

This is the basic reproduction number R_0 , and this affects whether an infection has the ability to spread through the population.

5. Stability analysis

The Jacobian equation matrix (quantization matrix) of model (2.2) is analyzed for the DFE and EE points' stability analysis at each equilibrium point. We then solve its characteristic equation and determine the eigenvalues of the matrix [18].

$$\begin{cases} {}^c D_t^\alpha S(t) = \Lambda - \mu S(t) - \beta S(t)I(t), \\ {}^c D_t^\alpha E(t) = \beta S(t)I(t) - (\mu + \vartheta + \varphi)E(t) + p(t)vI(t - \omega) + \psi L(t), \\ {}^c D_t^\alpha I(t) = \varphi E(t) - vI(t - \omega) - \mu I(t), \\ {}^c D_t^\alpha L(t) = vI(t - \omega) - p(t)vI(t - \omega) + \vartheta E(t) - (\mu + \psi)L(t). \end{cases} \quad (5.1)$$

For $\omega = 0$, the system reduces to an ordinary differential equation, and the local stability is determined using the Routh–Hurwitz criteria. For $\omega > 0$, the characteristic equation becomes transcendental. We analyze the possibility of purely imaginary roots $\lambda = ik$ to determine stability switching and the existence of Hopf bifurcation. Assuming the linearized system (2.2), the Jacobian matrix is obtained as follows:

$$J(S, E, I, L) = \begin{bmatrix} -(\mu + \beta I) & 0 & -\beta S & 0 \\ \beta I & -(\mu + \vartheta + \varphi) & \beta S + pve^{-\lambda\omega} & \psi \\ 0 & \varphi & -ve^{-\lambda\omega} - \mu & 0 \\ 0 & \vartheta & (1 - p)ve^{-\lambda\omega} & -(\mu + \psi) \end{bmatrix}.$$

Based on the characteristic equation provided as

$$|J(S, E, I, L) - \lambda I| = \begin{vmatrix} -(\mu + \beta I) - \lambda & 0 & -\beta S & 0 \\ \beta I & -(\mu + \vartheta + \varphi) - \lambda & \beta S + pve^{-\lambda\omega} & \psi \\ 0 & \varphi & (-ve^{-\lambda\omega} - \mu) - \lambda & 0 \\ 0 & \vartheta & (1 - p)ve^{-\lambda\omega} & -(\mu + \psi) - \lambda \end{vmatrix} = 0,$$

this results in,

$$\lambda^4 + M_1\lambda^3 + M_2\lambda^2 + M_3\lambda + M_4 + (N_1\lambda^3 + N_2\lambda^2 + N_3\lambda)e^{-\lambda\omega} = 0, \quad (5.2)$$

where

$$\begin{aligned} M_1 &= 4\mu + \vartheta + \beta I + \psi + \varphi, \\ M_2 &= 6\mu^2 + 3\mu(\vartheta + \varphi + \psi + \beta I) + \psi(\vartheta + \varphi) + \beta I(\psi + \vartheta + \varphi), \\ M_3 &= 4\mu^3 + 3\mu^2(\psi + \beta I + \vartheta + \varphi) + 2\mu\psi(\vartheta + \varphi + \beta I) + \vartheta\beta I(2\mu + \psi) + \varphi\beta I(2\mu + \psi), \\ M_4 &= \mu^4 + \mu^3(\varphi + \psi + \beta I + \vartheta) + \mu^2\psi(\vartheta + \varphi + \beta I) + \mu^2\beta I(\varphi + \vartheta) + \beta I\psi\mu(\vartheta + \varphi), \\ N_1 &= v, \\ N_2 &= v(\beta I + \varphi + \vartheta + \psi + \mu), \\ N_3 &= \mu^2v(1 + \mu) + \mu^2v(\varphi + \psi + \beta I) + \mu\psi v(\varphi + \vartheta + \beta I) + \mu v\beta I(1 + \varphi + \vartheta) + v\psi\beta I(\varphi + \vartheta). \end{aligned}$$

It is commonly recognized that once all of the roots of an equilibrium point's characteristic equation contain negative real components, the equilibrium point is asymptotically stable.

Theorem 5.1. *The DFE of the delayed model (2.2) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix of model (2.2) is

$$J(S, E, I, L) = \begin{bmatrix} -(\mu + \beta I) & 0 & -\beta S & 0 \\ \beta I & -(\mu + \vartheta + \varphi) & \beta S + pve^{-\lambda\omega} & \psi \\ 0 & \varphi & -ve^{-\lambda\omega} - \mu & 0 \\ 0 & \vartheta & (1 - p)ve^{-\lambda\omega} & -(\mu + \psi) \end{bmatrix}.$$

For the DFE point E_1 of (4.1),

$$|J(E_1) - \lambda I| = \begin{bmatrix} -\mu & 0 & -\beta(\frac{\Delta}{\mu}) & 0 \\ 0 & -(\mu + \vartheta + \varphi) & \beta(\frac{\Delta}{\mu}) + pve^{-\lambda\omega} & \psi \\ 0 & \varphi & -ve^{-\lambda\omega} - \mu & 0 \\ 0 & \vartheta & (1 - p)ve^{-\lambda\omega} & -(\mu + \psi) \end{bmatrix}.$$

The eigenvalues of the Jacobian matrix J are the solutions of the characteristic equation.

$$|J(E_1) - \lambda I| = 0.$$

From the above, we have $-\mu$ as one of the eigenvalues and other eigenvalues as the roots of the following characteristic equation:

$$\lambda^3 + K_1\lambda^2 + K_2\lambda + K_3 + (L_1\lambda^2 + L_2\lambda + L_3)e^{-\lambda\omega} = 0. \quad (5.3)$$

Without resolving (5.3), we can determine the sufficient and necessary requirements for stability using the Routh–Hurwitz stability criteria [2]. All the terms are positive, and the term $K_1K_2 - K_3$ is also positive if $R_0 < 1$. Further, for the delay $\omega \geq 0$, we use $\lambda = i\kappa$, and by the theorem from Section (3) of [17], we conclude that all of $J(E_1)$'s eigenvalues are negative according to the Routh–Hurwitz criteria, indicating that E_1 is locally asymptotically stable. \square

Theorem 5.2. *The EE of the delayed model (2.2) is locally asymptotically stable if $R_0 > 1$ and unstable if $R_0 < 1$.*

Proof. We will look at the characteristic equation given in (2.2) and evaluated at E_2 , which provides

$$\lambda^4 + M_1\lambda^3 + M_2\lambda^2 + M_3\lambda + M_4 + (N_1\lambda^3 + N_2\lambda^2 + N_3\lambda)e^{-\lambda\omega} = 0. \quad (5.4)$$

Then for $\omega = 0$, (5.4) reduces to

$$\lambda^4 + M_1\lambda^3 + M_2\lambda^2 + M_3\lambda + M_4 + N_1\lambda^3 + N_2\lambda^2 + N_3\lambda = 0,$$

where $M_1, M_2, M_3, M_4, N_1, N_2$, and N_3 are all positive and stable in accordance to the Routh–Hurwitz criterion [37]. The fundamental part of each of the roots of (5.4) is negative. Similarly, when $\omega > 0$, let $\lambda = i\omega$, where $\omega > 0$ is speculated to be the origin of (5.4). By applying the same methodology used for the DFE, we can determine that E_2 is asymptotically stable for $R_0 > 1$, since the roots of the characteristic equation have no positive real part. \square

6. Optimal controllability

This section investigates an optimum control problem with time delay that provides the TB epidemic mitigations, and it has been included in TB mathematical model. The optimal control for the dynamics of COVID-19 with cost effectiveness techniques was obtained in [12].

$$\begin{cases} {}^c D_t^\alpha S(t) &= \Lambda - \mu S(t) - (1 - v_1(t))\beta S(t)I(t), \\ {}^c D_t^\alpha E(t) &= (1 - v_1(t))\beta S(t)I(t) - (\mu + (1 + v_2(t))\vartheta + \varphi)E(t) + (1 - v_3(t))p(t)vI(t - \omega) + \psi L(t), \\ {}^c D_t^\alpha I(t) &= \varphi E(t) - vI(t - \omega) - \mu I(t), \\ {}^c D_t^\alpha L(t) &= (1 - (1 - v_3(t))p(t))vI(t - \omega) + (1 + v_2(t))\vartheta E(t) - (\mu + \psi)L(t), \end{cases} \quad (6.1)$$

Subject to initial conditions, $S(0) = S_0$, $E(0) = E_0$, $I(x) = I_0$, and $L(0) = L_0$.

The distancing control $v_1(t)$ indicates an effort to prevent individuals who are susceptible from spreading. Health-related activities, education, isolation of infected individuals, and prevention of infectious individuals are all related to $v_1(t)$. The case-finding control approach involves the identification of particularly exposed individuals and the treatment of latent tuberculosis, $v_2(t)$. The chance of developing the TB disease is significantly reduced by treatment for tuberculosis. A tuberculosis control campaign places a great deal of importance on case-finding because certain groups have an extremely high risk of contracting the disease once infected. In the case study holding $v_3(t)$, the efforts needed to finish treating infectious patients are discussed, including strategies used to keep drug intake consistent until a lasting cure is obtained. In order to take into consideration the non-linear societal costs associated with implementing control measures, the controls are seen as quadratic functions. In an optimal control issue, this quadratic control is a frequent form of an objective functional [20, 26]. The numbers of exposed and low-risk latent individuals E and L as well as the number of infected individuals I are directly and indirectly impacted by the control $v_3(t)$ in our model.

Our objective in this portion is to minimize the expense of control measures while reducing the number of individuals exposed. To put it mathematically, for a definite terminal time t_f , this is attained by solving the fractional optimum control issue below in order to minimize the objective functional denoted by

$$J(v_1, v_2, v_3) = \int_0^{t_f} \left(C_1 E(t) + C_2 I(t) + \frac{1}{2} D_1 v_1^2(t) + \frac{1}{2} D_2 v_2^2(t) + \frac{1}{2} D_3 v_3^2(t) \right) dt. \quad (6.2)$$

Assuming that the relative intervention costs are nonlinear, we utilize a quadratic form for the controls. The objective functional's size and significance determine the equilibrium factors that the coefficients D_1, D_2 , and D_3 contribute. The fundamental objectives are to find a similar set of state variables $X^* = S^*, E^*, I^*, L^*$ across the defined time period $[0, t_f]$, as well as the optimal controls (v_1^*, v_2^*, v_3^*) under the limits of the dynamic control system which minimizes the objective functional (6.2) as follows:

$$J(v_1^*, v_2^*, v_3^*) = \min_{(v_1, v_2, v_3) \in \Omega} J(v_1, v_2, v_3), \quad (6.3)$$

where, $\Omega = \{v_1(t), v_2(t), v_3(t) | 0 \leq v_i(t) \leq v_{imax} < 1, i = 1, 2, 3, \text{ and } t \in [0, t_f]\}$ is a control set.

6.1. Characterization of an optimal control

We implement the Pontryagin maximum principle [21, 25] to deduce the necessary conditions for this optimum control. Since, according to the system (6.1), there exists an optimal control for minimizing function (6.3).

Theorem 6.1. *Given an optimal control pair (v_1^*, v_2^*, v_3^*) and co-state variables S^*, E^*, I^*, L^* that minimize the objective functional $J(X, v_1, v_2, v_3)$, there exists adjoint variables $\lambda(\mathbf{t}) := (\lambda_1(\mathbf{t}), \lambda_2(\mathbf{t}), \lambda_3(\mathbf{t}), \lambda_4(\mathbf{t})) \in \mathbb{R}^4$ that satisfy the following adjoint equations:*

$$\begin{cases} {}^c D_{\mathbf{t}}^{\alpha} \lambda_1 &= \lambda_1 \beta I + \lambda_1 \mu - \lambda_1 \beta I v_1 - \lambda_2 \beta I + \lambda_2 \beta I v_1, \\ {}^c D_{\mathbf{t}}^{\alpha} \lambda_2 &= C_1 + \lambda_2 \mu - \lambda_3 \varphi + \lambda_2 v_2 \vartheta + \lambda_2 \vartheta + \lambda_2 \varphi - \lambda_4 \vartheta v_2 - \lambda_4 \vartheta, \\ {}^c D_{\mathbf{t}}^{\alpha} \lambda_3 &= C_2 + \lambda_2 \beta S v_1 - \lambda_2 \beta S + \lambda_1 \beta S - \lambda_1 \beta S v_1 + \lambda_3 \mu \\ &\quad + \chi_{[0, T-\omega]} v(p(v_3 - 1)\lambda_2(\mathbf{t} + \omega) + \lambda_3(\mathbf{t} + \omega) - (1 - p + p v_3)\lambda_4(\mathbf{t} + \omega)), \\ {}^c D_{\mathbf{t}}^{\alpha} \lambda_4 &= \lambda_4 \mu - \lambda_2 \psi + \lambda_4 \psi, \end{cases} \quad (6.4)$$

with transversality conditions

$$\lambda_i(\mathbf{t}_f) = 0, \quad i = 1, 2, 3, 4. \quad (6.5)$$

The continuous optimal control function is piecewise characterized as

$$\begin{aligned} v_1^*(\mathbf{t}) &= \min \left\{ \max \left\{ 0, \frac{(\lambda_2 - \lambda_1) \beta S^* I^*}{D_1} \right\}, 1 \right\}, \\ v_2^*(\mathbf{t}) &= \min \left\{ \max \left\{ 0, \frac{(\lambda_2 - \lambda_4) \vartheta E^*}{D_2} \right\}, 1 \right\}, \\ v_3^*(\mathbf{t}) &= \min \left\{ \max \left\{ 0, \frac{(\lambda_2 - \lambda_4) p v I^*(\mathbf{t} - \omega)}{D_3} \right\}, 1 \right\}. \end{aligned} \quad (6.6)$$

Proof. Define the specified Hamiltonian condition in this manner:

$$\begin{aligned} H(S, E, I, L, v_1, v_2, v_3, \lambda) &= C_1 E(\mathbf{t}) + C_2 I(\mathbf{t}) + \frac{D_1}{2} v_1^2 + \frac{D_2}{2} v_2^2 + \frac{D_3}{2} v_3^2 \\ &\quad + \lambda_1 [\Lambda - \mu S(\mathbf{t}) - (1 - v_1) \beta S I(\mathbf{t})] \\ &\quad + \lambda_2 [\beta S I(\mathbf{t})(1 - v_1) - (\mu + (1 + v_2) \vartheta + \varphi) E(\mathbf{t}) \\ &\quad + (1 - v_3) p(\mathbf{t}) v I(\mathbf{t} - \omega) + \psi L(\mathbf{t})] \\ &\quad + \lambda_3 [\varphi E(\mathbf{t}) - v I(\mathbf{t} - \omega) - \mu I(\mathbf{t})] \\ &\quad + \lambda_4 [(1 - (1 - v_3) p(\mathbf{t})) v I(\mathbf{t} - \omega) + (1 + v_2) \vartheta E(\mathbf{t}) - (\mu + \psi) L(\mathbf{t})]. \end{aligned}$$

We define the characteristic function as follows:

$$\chi_{[\mathbf{t}_i, \mathbf{t}_g]}(\mathbf{t}) = \begin{cases} 1, & \mathbf{t} \in [\mathbf{t}_i, \mathbf{t}_g], \\ 0, & \text{otherwise.} \end{cases}$$

According to the Pontryagin maximum principle, the adjoint variables are derived $\lambda = (\lambda_1, \lambda_2, \lambda_3, \lambda_4) \in \mathbb{R}^4$ and satisfy the following canonical equations:

$${}^c D_{\mathbf{t}}^{\alpha} \lambda_1 = -\frac{\partial H}{\partial S} S(\mathbf{t}), \quad {}^c D_{\mathbf{t}}^{\alpha} \lambda_2 = -\frac{\partial H}{\partial E} E(\mathbf{t}),$$

$${}^c D_t^\alpha \lambda_3 = -\frac{\partial H}{\partial I(t)} - \chi_{[0, T-\omega]}(t) \frac{\partial H}{\partial I(t-\omega)} \Big|_{t+\omega}, \quad {}^c D_t^\alpha \lambda_4 = -\frac{\partial H}{\partial L} L(t). \quad (6.7)$$

We apply (6.5) and modify the previously identified inequalities by adding the corresponding variable derivatives, and the adjoint equations provide more understanding (6.4). We obtain the following from deriving the optimality condition:

$$\frac{\partial H}{\partial v_1} = 0, \quad \text{at } v_1 = v_1^*, \quad \frac{\partial H}{\partial v_2} = 0, \quad \text{at } v_2 = v_2^*, \quad \frac{\partial H}{\partial v_3} = 0, \quad \text{at } v_3 = v_3^*.$$

Thus, we have

$$v_1^*(t) = \frac{(\lambda_2 - \lambda_1)\beta S^* I^*}{D_1}, \quad v_2^*(t) = \frac{(\lambda_2 - \lambda_4)\vartheta E^*}{D_2}, \quad v_3^*(t) = \frac{(\lambda_2 - \lambda_4)p\nu I^*(t-\omega)}{D_3}. \quad (6.8)$$

According to the corresponding optimal controls in equation (6.6), if the values of (v_1^*, v_2^*, v_3^*) are greater than 1, then we consider them as 1. Otherwise, we treat the values as 0 if the values are negative. By applying the optimality system, the minimum Hamiltonian H^* is obtained by replacing v_1^*, v_2^* , and v_3^* into (6.1) and (6.2) as follows:

$$\left\{ \begin{array}{l} {}^c D_t^\alpha S^*(t) = \Lambda - \mu S^* - (1 - v_1(t))\beta S^*(t)I^*(t), \\ {}^c D_t^\alpha E^*(t) = (1 - v_1(t))\beta S^*(t)I^*(t) - (\mu + (1 + v_2(t))\vartheta + \varphi)E^*(t) \\ \quad + (1 - v_3(t))p(t)\nu I^*(t-\omega) + \psi L^*(t), \\ {}^c D_t^\alpha I^*(t) = \varphi E^*(t) - \nu I^*(t-\omega) - \mu I^*(t), \\ {}^c D_t^\alpha L^*(t) = (1 - (1 - v_3(t))p(t))\nu I^*(t-\omega) + (1 + v_2(t))\vartheta E^*(t) - (\mu + \psi)L^*(t). \end{array} \right.$$

The initial conditions corresponding to the optimal system variables are described in (3.1). The adjoint system is described by (6.4) with transversality conditions described by $\lambda_i(t_f) = 0$, where $i = 1, 2, 3, 4$ and v_1^*, v_2^* , and v_3^* as the optimal controls are confirmed by (6.6). This complete the proof. \square

7. Numerical simulations

In this section, we present the numerical simulations obtained using the Runge–Kutta method [24] adapted for a system of DFDEs. The model parameters used are listed in Table 1. The compartments S, E, I, and L represent the susceptible, exposed, infected, and latent populations, respectively. All simulations are carried out for a fractional-order system with $\alpha = 0.9$, which effectively captures the memory-dependent characteristics inherent in disease transmission processes.

Table 1. The values of the model parameters defined in (2.2).

Parameters	Λ	β_1	β_2	p	μ	ζ	φ	δ	θ	ϵ
Values	0.8	0.2	0.25	0.1	0.1	0.1	0.2	0.1	0.1	0.1

From Figure 1(a), the dynamics of the susceptible population are illustrated for two different values of the delay parameter, namely $\tau = 0$ (no delay) and $\tau = 6$. The plots clearly demonstrate the impact of incorporating a delay: While both cases exhibit a decline in the susceptible population over time,

the introduction of a delay ($\tau = 6$) significantly alters the trajectory. In particular, the delayed system shows a more gradual decline, indicating a slower depletion of the susceptible class. This suggests that the inclusion of a biologically realistic incubation or response time influences the overall system behavior. Moreover, the application of control strategies in the delayed case leads to a more pronounced stabilization of the susceptible population.

Similarly, Figure 1(b) shows the dynamics of the exposed population under both delay and non-delay scenarios. For the non-delay case, the difference between controlled and uncontrolled scenarios is relatively modest. However, when the delay parameter is set to $\tau = 6$, the effects of control become more evident, with the exposed population exhibiting a delayed and moderated growth. This implies that the introduction of delay not only reflects real-world transmission lags but also enhances the effectiveness of intervention strategies over time.

A comparable trend is observed in the infected population, as depicted in Figure 1(c). Without delay, the control measures have limited impact in flattening the infection curve. In contrast, the delayed model results in a significant reduction in the peak of infections, illustrating how delay and fractional dynamics jointly contribute to capturing the latency, transmission lag, and eventual recovery more realistically. These dynamics are further supported by the trends seen in the latent population, shown in Figure 1(d), which follow a similar path under the influence of both delay and control.

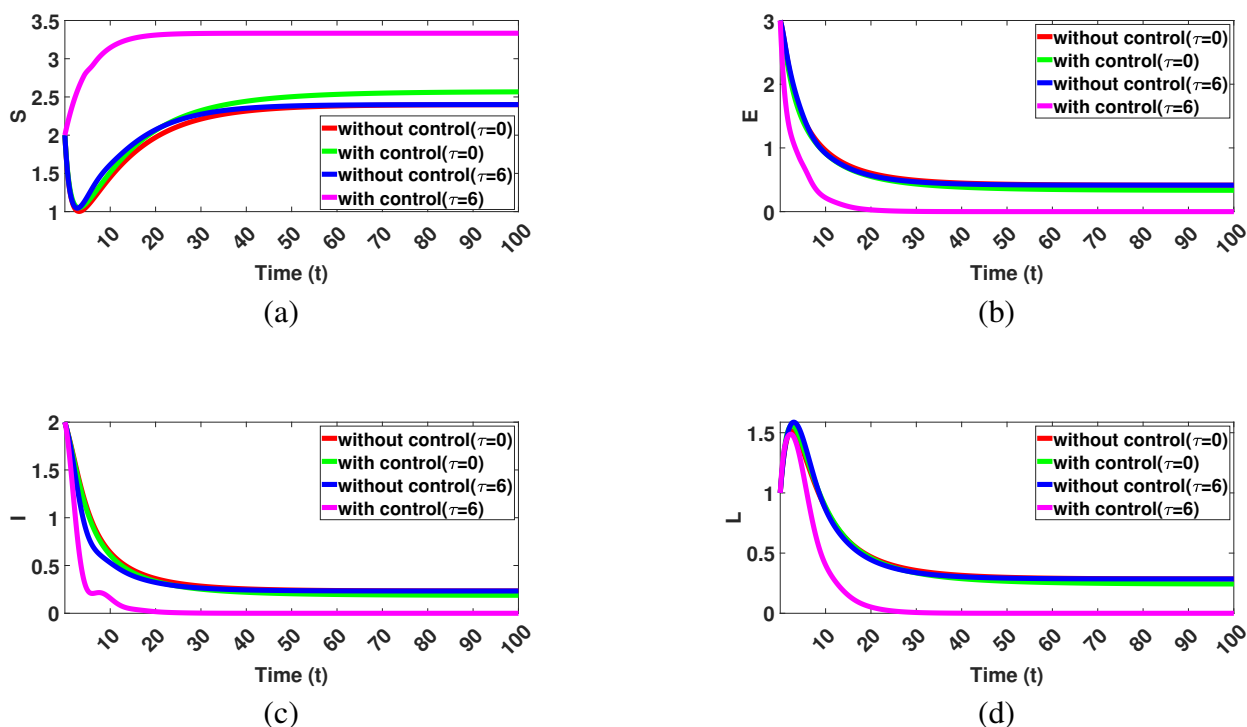


Figure 1. Plots (a)–(d) represent the solution of the population $S(t)$, $E(t)$, $I(t)$, and $L(t)$ in the model (2.2) with the effect of control variable, for different values of delay parameter τ .

The effectiveness of control strategies is systematically depicted in Figure 2, where the control variables v_1 , v_2 , and v_3 are shown in the respective subplots. Each control targets a specific aspect of the disease dynamics: v_1 aims to reduce transmission, v_2 enhances recovery, and v_3 minimizes the latency period. The application of these controls, particularly in the presence of delay, significantly

suppresses the peaks in the exposed and infected compartments and helps maintain a more stable disease trajectory. The figures indicate that the controls have a stronger and more sustained effect when a biologically significant delay is incorporated into the model. In the absence of delay, the control variables show limited efficacy, further emphasizing the importance of delay in representing the realistic progression of the disease.

In summary, the numerical simulations confirm the crucial role of fractional-order delay differential equations in modeling infectious disease dynamics. The presence of a delay not only enhances the biological realism of the model but also improves the efficacy of control interventions. These findings underscore the importance of incorporating both fractional dynamics and time delays in the design of effective public health strategies aimed at controlling infectious disease outbreaks.

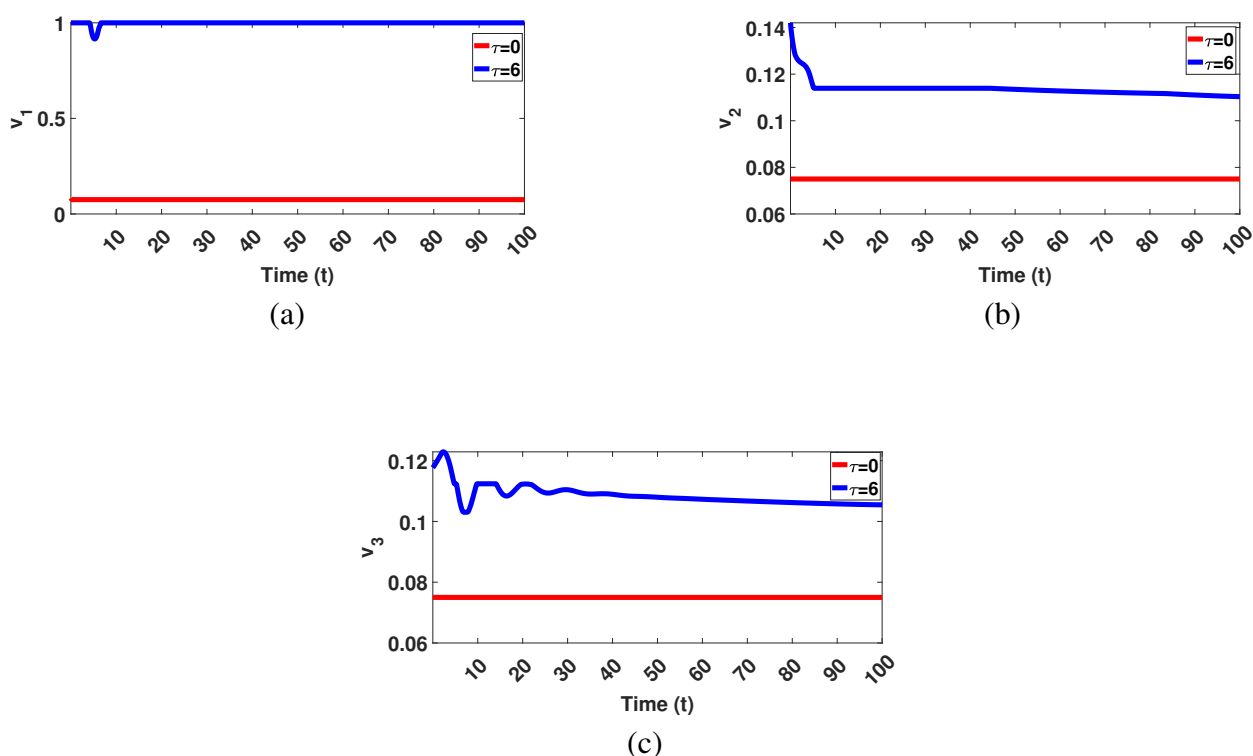


Figure 2. Plots (a)–(c) represent the values of control variable v_1 , v_2 , and v_3 over time for the fractional order $\alpha = 0.9$ with different delay parameters.

8. Conclusions

In this study, we investigated the fractional-order type of the delayed SEIL epidemiological model that identifies this epidemiological situation. The fractional derivative Caputo scenario was studied, which is appropriate for problems with the initial conditions. We proved that the suggested system has two equilibrium points. The stability conditions were investigated through the reproduction number together with the DFE and EE points. This specific fractional-order delay model's solution was obtained using a recently developed numerical method. The effectiveness of the theoretical conclusions and the reliability of the numerical methodology have been proven using numerical simulations.

Author contributions

Suganya Dhandapani: Writing–Original draft, Methodology, Conceptualization, Resources, Formal analysis; Bhuvaneswari Venkatasubramaniam: Writing–Review and Editing, Conceptualization, Supervision, Resources; Ibraheem M. Alsulami: Methodology, Investigation, Software, Resources, Formal analysis, Funding Acquisition, Project Administration, Review and Editing; Amer Alsulami: Resources, Investigation, Validation, Writing–Review and Editing; Hariharan Soundararajan: Conceptualization, Writing–Review and Editing, Visualization, Resources, Formal analysis; Shangerganesh Lingeshwaran: Writing–Review and Editing, Validation, Supervision. The authors jointly contributed to the research and manuscript preparation.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare no conflicts of interest.

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