



Research article

Investigation of chaos control in a fractional-order model of lysogenic bacteria under phage therapy

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Abstract: This paper investigates a fractional-order model of lysogenic bacteria and its chaos control, with a focus on phage therapy. A mathematical model is formulated to describe bacterial dynamics, and chaos control techniques are applied to stabilize the system. The Caputo fractional operator is used to construct a fractional-order framework that incorporates memory effects and enables reliable numerical approximation. The proposed model is analyzed with respect to positivity, boundedness, and the existence and uniqueness of solutions. The Lipschitz condition and fixed-point theory are utilized to establish the uniqueness of solutions. The local and global stability of the disease-free and endemic equilibrium points are examined through Lyapunov stability theory. Chaos control conditions are derived to investigate the influence of the model's parameters on the emergence and suppression of chaotic behavior. Numerical simulations based on the Caputo fractional operator illustrate the dynamics of bacterial populations under phage therapy. Furthermore, the effects of the model's parameters and fractional-order values are analyzed using a two-step Lagrange polynomial numerical scheme, demonstrating the effectiveness of the proposed approach in controlling infected bacterial populations.

Keywords: lysogenic bacteria; phage therapy; stochastic model; chaos control; Caputo fractional derivative

Mathematics Subject Classification: 92Bxx, 92Fxx

1. Introduction

The relationship between mathematics and biology has continuously stimulated the development of new approaches, methods, and concepts for the modeling of biological phenomena. Mathematicians are encouraged to explore the applicability of mathematical techniques to biological systems [1], while biologists increasingly recognize mathematical modeling as a valuable complement to experimental and empirical investigations. Cremer et al. [2] used a deterministic mathematical model to investigate evolutionary and population dynamics involving interactions between non-mutant and mutant populations in a constant environment. Their study adopted a simplified framework based on a constant population size and a well-mixed population without a spatial structure.

Bacterial populations continue to attract considerable attention from mathematicians because their growth, replication, adaptation, and survival mechanisms can be effectively represented through mathematical formulations [3]. Under suitable assumptions, deterministic mathematical models have been developed in both continuous- and discrete-time frameworks to analyze a variety of population behaviors, including the dynamics of single bacterial strains, the coexistence of multiple phenotypically distinct mutants, and interactions among heterogeneous populations. Such population models are frequently represented by nonlinear differential equations and have contributed significantly to understanding bacterial population dynamics [4].

Although mathematics and biology are often regarded as distinct disciplines, they share a strong connection through the development and analysis of mathematical models. Biological systems are frequently represented through mathematical abstractions that facilitate the understanding of complex interactions and dynamic behaviors. Even simple population growth models illustrate the interdisciplinary relationship between mathematics and biology and demonstrate how mathematical approaches can provide valuable insights into biological processes [5].

Mathematical models have become indispensable tools for understanding the dynamics of biological systems and infectious processes. Differential-equation-based models provide a systematic framework for describing interactions among different biological populations and for evaluating the effects of intervention strategies. Such models have been extensively applied in population dynamics, epidemiology, and biological systems to investigate the stability, persistence, and long-term behavior of complex processes [6].

Fractional calculus extends the classical concepts of differentiation and integration to non-integer and complex orders [7]. Fractional derivatives and integrals have attracted significant attention because of their ability to describe memory and hereditary effects in complex systems. Among the available fractional operators, the Caputo derivative is particularly useful because it allows the incorporation of standard initial and boundary conditions that possess clear physical interpretations. Recent advances in fractional calculus and its applications have been extensively discussed in the literature [8, 9].

Recent studies have further demonstrated the effectiveness of fractional calculus in describing memory-dependent and nonlocal dynamic phenomena. For instance, Meng et al. [10] investigated the collective dynamics of fractional oscillators with fluctuating frequencies in Erdős–Rényi random networks, while Lin and Wang [11] analyzed the influence of time delays on collective resonant behaviors in coupled fractional oscillators with mass fluctuations. Moreover, Meng et al. [12] examined the tempered anomalous dynamics, synchronization, and stability properties of globally coupled harmonic oscillators in fluctuating potential fields. These studies highlight the capability of

fractional-order operators to capture complex memory effects and collective behaviors that are difficult to represent using classical integer-order models.

Fractional-order models are often more flexible and realistic than their integer-order counterparts because the fractional derivative introduces an additional degree of freedom that can capture memory-dependent dynamics. These models have been successfully applied to describe the behavior of a wide range of biological, physical, and engineering systems. One of the most important characteristics of fractional-order models is their ability to represent global and hereditary effects that are not adequately captured by classical integer-order formulations [13].

By using the Caputo operator, fractional-order models can be solved numerically, enabling the investigation of systems' behavior under varying parameter values and initial conditions. Numerical simulations have also been used to analyze the controllability and observability properties of dynamic systems and to determine their equilibrium points [14].

Exploring Caputo fractional derivatives in disease modeling presents a promising direction for future research. This approach has the potential to provide more precise and detailed insights into disease dynamics, thereby contributing to improved disease prevention and control strategies. As fractional calculus and epidemiology continue to evolve, innovative methodologies and applications are expected to emerge [15, 16].

Recent developments in generalized fractional calculus have further demonstrated the effectiveness of fractional operators in modeling memory-dependent and non-local dynamic systems. Fractional-order formulations have been successfully used to investigate collective dynamics in Erdős Rényi random networks, time-delay-induced resonant behaviors in coupled fractional oscillators, and tempered anomalous dynamics in globally coupled oscillator systems [10–12]. Fractional-order modeling has also been applied to ecological systems through predator-prey interactions with prey refuge effects, where fractional operators were shown to capture memory-dependent population dynamics more accurately than classical integer-order formulations. In addition, the synchronization properties of discrete-time fractional-order quaternion-valued uncertain neural networks have been investigated, demonstrating the capability of fractional calculus to describe complex dynamic behaviors in nonlinear systems and control applications [17, 18]. These studies collectively highlight the ability of fractional operators to capture hereditary effects, long-range interactions, and historical dependencies that cannot be adequately represented using the classical integer-order model. Motivated by these advantages, the present study adopts the Caputo fractional derivative to describe the memory-dependent dynamics of lysogenic bacterial populations under phage therapy.

The stability conditions under which a proposed controller can suppress chaos globally in a fractional-order chaotic system have been extensively investigated. Numerical simulations further demonstrate the effectiveness of such control strategies. Depending on the selected parameter values, a hyperchaotic or oscillatory system may be transformed into a periodic or quasiperiodic system [19].

Fractional-order calculus has gained considerable attention in both mathematics and engineering because of its effectiveness in describing nonlinear dynamic systems. Although numerous fractional-order chaotic systems have been proposed, many studies focus primarily on autonomous systems or non-autonomous systems with sinusoidal perturbations, while comparatively less attention has been devoted to their control and stabilization [20].

The present work applies the Caputo fractional derivative to a fractional-order model of lysogenic bacterial dynamics under phage therapy. The proposed framework incorporates memory effects

through the Caputo operator and provides a mathematical description of the interactions among susceptible, lysogenic, infected, resistant, and phage populations. The existence and uniqueness of solutions are established using fixed-point theory, while stability and chaos control analyses are performed to investigate the qualitative behavior of the system. Numerical simulations are conducted to examine the influence of the model's parameters and fractional-order dynamics on bacterial populations. The findings provide useful insights into the management and control of bacterial infections through phage-based therapeutic strategies.

- Section 1 presents the introduction and background of the study.
- Section 2 formulates the proposed stochastic lysogenic bacterial model and its fractional-order extension.
- Section 3 provides the mathematical analysis of the developed model, including its positivity, boundedness, the invariant regions, existence and uniqueness of the solutions, an equilibrium analysis, and a numerical formulation.
- Sections 4, 5, and 6 present the stability analysis, chaos control analysis, and computational analysis based on the Caputo fractional operator, respectively.
- Section 7 presents the numerical simulations and discusses the obtained results.
- Section 8 concludes the study and outlines potential directions for future research.

Preliminaries

In this section, several fundamental concepts and definitions from fractional calculus are presented, which will be used throughout the subsequent analysis.

Definition 1. Let $E \in L^1([0, T], \mathbb{R})$ be a continuous function. The Riemann-Liouville fractional integral of order $\varsigma \in (0, 1)$ is defined by

$${}^{RL}_0 I_t^\varsigma E(t) = \frac{1}{\Gamma(\varsigma)} \int_0^t (t - \phi)^{\varsigma-1} E(\phi) d\phi. \quad (1.1)$$

where $\Gamma(\cdot)$ denotes the gamma function.

Definition 2. Let $E \in L^1([0, T], \mathbb{R})$ be a sufficiently smooth function. The Caputo fractional derivative of order ς ($n - 1 < \varsigma < n$, $n \in \mathbb{N}$) is defined as

$${}_0^C D_t^\varsigma E(t) = \frac{1}{\Gamma(n - \varsigma)} \int_0^t (t - \phi)^{n-\varsigma-1} \frac{d^n}{d\phi^n} E(\phi) d\phi. \quad (1.2)$$

For the particular case $0 < \varsigma < 1$ ($n = 1$), the Caputo derivative reduces to

$${}_0^C D_t^\varsigma E(t) = \frac{1}{\Gamma(1 - \varsigma)} \int_0^t (t - \phi)^{-\varsigma} E'(\phi) d\phi. \quad (1.3)$$

2. Mathematical model and formulation

Mathematical modeling plays a vital role in understanding both epidemic and non-epidemic biological phenomena. In the present study, a novel stochastic mathematical model is developed

to investigate the dynamics of lysogenic bacteria under phage therapy and to evaluate strategies for controlling infected bacterial populations. The proposed model divides the total bacterial population into five biologically meaningful compartments according to their infection status and interaction with bacteriophages.

The state variables of the model are defined as follows:

- $S(t)$: Susceptible (non-lysogenic) bacteria that are vulnerable to infection;
- $L(t)$: Lysogenic bacteria carrying integrated phage genetic material without immediate lytic activity;
- $I(t)$: Infected bacteria undergoing the lytic phase, where active phage replication takes place;
- $R(t)$: Resistant bacteria that have developed resistance to conventional antibacterial treatment and may contribute to the persistence of infection;
- $P(t)$: Therapeutic bacteriophage population used to suppress bacterial growth and control infection.

The biological interpretation of these compartments reflects the progression of bacterial populations under phage therapy. Susceptible bacteria may become lysogenic following an interaction with phages; lysogenic bacteria can transition to the infected class; infected bacteria may develop resistance; and resistant bacteria interact with therapeutic phages that contribute to infection control. The phage population acts as a biological treatment agent that regulates bacterial growth and reduces the spread of infection.

To account for environmental variability and uncertainty in bacterial growth, infection transmission, resistance development, and phage–bacteria interactions, the proposed framework is referred to as a stochastic model. The stochastic characteristics represent random fluctuations that may arise from biological heterogeneity, environmental disturbances, and variability in treatment responses. The corresponding fractional-order formulation further incorporates memory and hereditary effects that influence the long-term evolution of the bacterial population.

The interactions among these compartments represent the principal biological processes involved in lysogenic infection, lytic activation, resistance development, and phage-mediated bacterial control. Susceptible bacteria may become lysogenic following exposure to bacteriophages, while lysogenic bacteria can subsequently enter the lytic phase and become actively infected. Infected bacteria may evolve into resistant strains that are more difficult to eliminate using conventional antibacterial treatments. Phage therapy is incorporated through the bacteriophage compartment, which acts as a biological control agent by reducing bacterial burden and suppressing the proliferation of resistant bacteria.

The model's parameters and their biological interpretations are described as follows. The parameter α denotes the recruitment or production rate of susceptible bacteria. The parameter β represents the transmission coefficient governing interactions between susceptible and lysogenic bacteria. The parameter γ denotes the therapeutic contribution of the bacteriophage population to bacterial suppression. The parameter μ represents the natural mortality rate of bacterial populations. The parameter γ_1 describes the interaction rate associated with resistant bacterial dynamics. The parameter β_1 denotes the rate of progression from the lysogenic state to the lytic infected state. The parameter β_2 represents the rate at which infected bacteria develop resistance, while β_3 denotes the phage-induced elimination rate of resistant bacteria.

Figure 1 illustrates the biological transitions among the five compartments of the proposed SLIRP model. The flow diagram depicts the movement of bacteria from the susceptible class to the lysogenic and infected classes, the emergence of resistant bacteria, and the regulatory role of therapeutic bacteriophages in controlling bacterial populations under phage therapy.

Given the biological assumptions, the temporal evolution of each compartment is governed by bacterial recruitment, infection transmission, lysogenic progression, resistance development, natural mortality, and phage-mediated control mechanisms. Consequently, the proposed SLIRP model is formulated as follows.

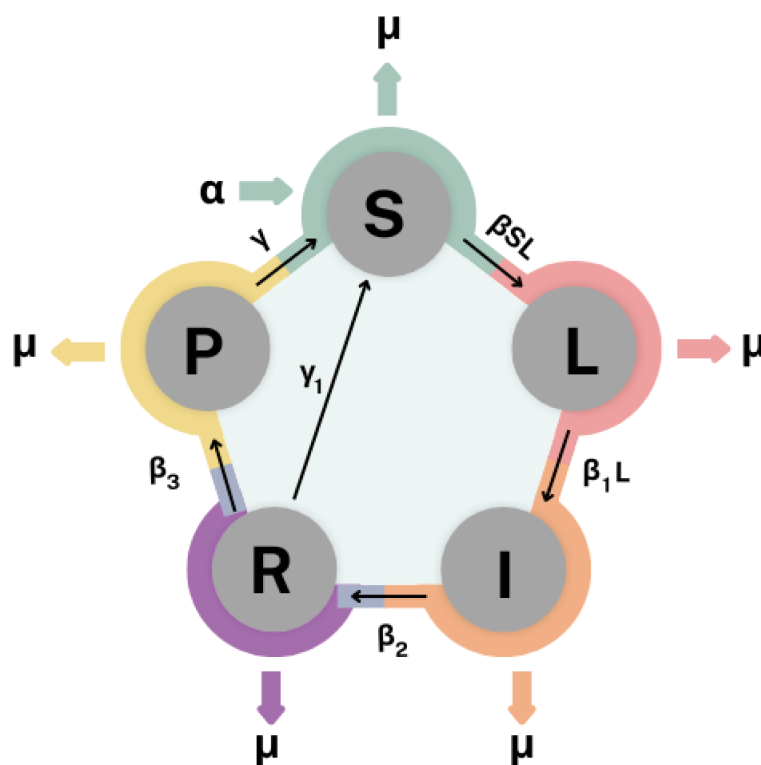


Figure 1. Flow diagram of the SLIRP model.

The compartmental transitions illustrated in Figure 1 lead to the following system of differential equations governing the dynamics of the SLIRP model:

$$\begin{aligned}
 \frac{dS}{dt} &= \alpha - \beta S L + \gamma P - \mu S + \gamma_1 R, \\
 \frac{dL}{dt} &= \beta S L - \mu L - \beta_1 L, \\
 \frac{dI}{dt} &= \beta_1 L - (\beta_2 + \mu) I, \\
 \frac{dR}{dt} &= \beta_2 I - (\beta_3 + \mu + \gamma_1) R, \\
 \frac{dP}{dt} &= \beta_3 R - (\gamma + \mu) P.
 \end{aligned} \tag{2.1}$$

The corresponding initial conditions are given by

$$\begin{aligned}
S(0) &= S^0 \geq 0, \\
L(0) &= L^0 \geq 0, \\
I(0) &= I^0 \geq 0, \\
R(0) &= R^0 \geq 0, \\
P(0) &= P^0 \geq 0,
\end{aligned} \tag{2.2}$$

where S^0 , L^0 , I^0 , R^0 , and P^0 denote the initial population densities of susceptible, lysogenic, infected, resistant bacteria, and therapeutic bacteriophages, respectively. Since these quantities represent biological populations, all initial conditions are assumed to be non-negative.

Fractional-order SLIRP model

Fractional-order systems generalize classical integer-order systems by introducing derivatives of non-integer order. Such models have been widely used to describe biological systems that exhibit memory and hereditary effects, which cannot be adequately represented by conventional integer-order differential equations. Motivated by these advantages, the proposed SLIRP model is extended to a fractional-order framework using the Caputo derivative. The successful application of fractional-order models in ecological systems, predator-prey interactions, and synchronization of complex neural networks further supports their suitability for describing biological systems with memory and hereditary effects [17, 18].

In biological systems, bacterial growth, lysogenic infection, resistance development, and phage-host interactions may depend not only on the current state of the system but also on its previous states. These memory-dependent effects arise from environmental influences, delayed biological responses, and historical interactions among bacterial populations. Fractional calculus provides an effective mathematical tool for incorporating such hereditary characteristics into the model's dynamics.

Although the present study is motivated by uncertainties associated with biological processes, the governing equations do not explicitly contain stochastic diffusion terms such as Wiener processes or Gaussian white noise. Instead, randomness is reflected indirectly through uncertainties in the biological parameters, environmental fluctuations, and variations in initial population sizes. The mathematical analysis developed in this work therefore focuses on the deterministic fractional-order representation of the SLIRP system while retaining the ability of fractional operators to capture memory-dependent dynamics.

The Caputo fractional derivative was selected in preference to other fractional operators because of its suitability for biological and epidemiological applications. Unlike the Riemann–Liouville derivative, the Caputo derivative allows the use of classical initial conditions expressed in terms of integer-order derivatives, which possess direct biological interpretations and can be measured experimentally. This property facilitates the formulation of physically meaningful initial value problems and simplifies the analysis of existence, uniqueness, and stability of solutions. Furthermore, the Caputo operator effectively captures memory and hereditary effects that are characteristic of bacterial growth, lysogenic infection, resistance development, and phage-host interactions. Compared with more recent operators such as the Caputo-Fabrizio and Atangana-Baleanu derivatives, the Caputo derivative has a well-established mathematical framework and has been extensively applied in biological and epidemiological modeling, making it particularly suitable for the present study.

For analytical consistency, the same fractional order $\varsigma \in (0, 1]$ is assigned to all state variables, $S(t)$, $L(t)$, $I(t)$, $R(t)$, and $P(t)$. This assumption implies that all compartments exhibit the same degree of memory and hereditary effects throughout the evolution of the system. The adoption of a common fractional order enables the development of a unified analytical framework for establishing the positivity, boundedness, existence, uniqueness, stability, and numerical properties of the proposed model. The investigation of multi-order fractional systems, where different compartments possess different memory characteristics, is beyond the scope of the present work and remains a direction for future research.

Accordingly, the fractional-order SLIRP model is formulated as follows:

$$\begin{aligned} {}^C D_t^\varsigma S(t) &= \alpha - \beta S L + \gamma P - \mu S + \gamma_1 R, \\ {}^C D_t^\varsigma L(t) &= \beta S L - (\mu + \beta_1) L, \\ {}^C D_t^\varsigma I(t) &= \beta_1 L - (\beta_2 + \mu) I, \\ {}^C D_t^\varsigma R(t) &= \beta_2 I - (\beta_3 + \mu + \gamma_1) R, \\ {}^C D_t^\varsigma P(t) &= \beta_3 R - (\gamma + \mu) P. \end{aligned} \quad (2.3)$$

The associated initial conditions are

$$\begin{aligned} S(0) = S^0 \geq 0, \quad L(0) = L^0 \geq 0, \quad I(0) = I^0 \geq 0, \\ R(0) = R^0 \geq 0, \quad P(0) = P^0 \geq 0. \end{aligned} \quad (2.4)$$

Throughout this study, all the model's parameters are assumed to be positive constants.

3. Analysis of the proposed model

3.1. Non-negativity and boundedness of solutions

The biological validity of the proposed fractional-order SLIRP model requires that all state variables remain non-negative for all future times whenever non-negative initial conditions are prescribed. In addition, the solutions must remain bounded to ensure that the model describes realistic bacterial population dynamics. This subsection establishes the positivity and boundedness of the system solutions.

Similarly, for the lysogenic compartment, we have

$$\begin{aligned} {}^C D_t^\varsigma L(t) &= \beta S(t)L(t) - (\mu + \beta_1)L(t) \\ &\geq -(\mu + \beta_1)L(t). \end{aligned} \quad (3.1)$$

Let

$$k_2 = \mu + \beta_1.$$

Considering the comparison equation

$${}^C D_t^\varsigma y(t) = -k_2 y(t), \quad y(0) = L^0,$$

we obtain

$$L(t) \geq L^0 E_{\varsigma}(-(\mu + \beta_1)t^{\varsigma}). \quad (3.2)$$

For the infected compartment, we have

$$\begin{aligned} {}^C D_t^{\varsigma} I(t) &= \beta_1 L(t) - (\beta_2 + \mu)I(t) \\ &\geq -(\beta_2 + \mu)I(t). \end{aligned} \quad (3.3)$$

Let

$$k_3 = \beta_2 + \mu.$$

Then

$$I(t) \geq I^0 E_{\varsigma}(-(\beta_2 + \mu)t^{\varsigma}). \quad (3.4)$$

For the resistant compartment, we have

$$\begin{aligned} {}^C D_t^{\varsigma} R(t) &= \beta_2 I(t) - (\beta_3 + \mu + \gamma_1)R(t) \\ &\geq -(\beta_3 + \mu + \gamma_1)R(t). \end{aligned} \quad (3.5)$$

Let

$$k_4 = \beta_3 + \mu + \gamma_1.$$

Hence,

$$R(t) \geq R^0 E_{\varsigma}(-(\beta_3 + \mu + \gamma_1)t^{\varsigma}). \quad (3.6)$$

For the phage compartment, we have

$$\begin{aligned} {}^C D_t^{\varsigma} P(t) &= \beta_3 R(t) - (\gamma + \mu)P(t) \\ &\geq -(\gamma + \mu)P(t). \end{aligned} \quad (3.7)$$

Let

$$k_5 = \gamma + \mu.$$

Therefore,

$$P(t) \geq P^0 E_{\varsigma}(-(\gamma + \mu)t^{\varsigma}). \quad (3.8)$$

Consequently,

$$\begin{aligned} S(t) &\geq S^0 E_{\varsigma}(-(\beta \|L\|_{\infty} + \mu)t^{\varsigma}), \\ L(t) &\geq L^0 E_{\varsigma}(-(\mu + \beta_1)t^{\varsigma}), \\ I(t) &\geq I^0 E_{\varsigma}(-(\beta_2 + \mu)t^{\varsigma}), \\ R(t) &\geq R^0 E_{\varsigma}(-(\beta_3 + \mu + \gamma_1)t^{\varsigma}), \\ P(t) &\geq P^0 E_{\varsigma}(-(\gamma + \mu)t^{\varsigma}). \end{aligned} \quad (3.9)$$

Since the Mittag–Leffler function satisfies

$$E_{\varsigma}(-at^{\varsigma}) > 0, \quad a > 0,$$

for all $t \geq 0$ and $0 < \varsigma \leq 1$, all state variables remain non-negative whenever the initial conditions are non-negative. Therefore, the proposed fractional-order SLIRP model preserves positivity and yields biologically meaningful solutions for all $t \geq 0$.

3.2. Positively invariant region

An invariant region is a subset of the state space such that any trajectory originating within the region remains there for all future times. Establishing a positively invariant region guarantees that the state variables of the proposed model remain biologically meaningful and non-negative throughout the evolution of the system.

Theorem 1. The solutions of System (2) with non-negative initial conditions remain in the positive orthant

$$\mathbb{R}_+^5 = \{(S, L, I, R, P) \in \mathbb{R}^5 : S, L, I, R, P \geq 0\},$$

for all $t \geq 0$.

Proof.

To verify the positive invariance of the feasible region, we evaluate the vector field on each boundary plane of the state variables. Thus, we have

$$\begin{aligned} {}^C D_t^{\varsigma} S(t) \Big|_{S=0} &= \alpha + \gamma P + \gamma_1 R \geq 0, \\ {}^C D_t^{\varsigma} L(t) \Big|_{L=0} &= \beta S \geq 0, \\ {}^C D_t^{\varsigma} I(t) \Big|_{I=0} &= \beta_1 L \geq 0, \\ {}^C D_t^{\varsigma} R(t) \Big|_{R=0} &= \beta_2 I \geq 0, \\ {}^C D_t^{\varsigma} P(t) \Big|_{P=0} &= \beta_3 R \geq 0. \end{aligned} \tag{3.10}$$

The inequalities in (3.10) show that the vector field points inward on each boundary of the positive orthant. Consequently, trajectories cannot cross the coordinate planes into the negative region.

Therefore, any solution that starts with non-negative initial conditions remains non-negative for all $t \geq 0$. Combined with the existence and uniqueness results established subsequently, the proposed fractional-order SLIRP model is mathematically well-posed and biologically feasible. $\square$

3.3. Existence and uniqueness analysis

In this subsection, the existence and uniqueness of solutions for the proposed fractional-order SLIRP model are established using fixed-point theory. Specifically, Schauder's fixed-point theorem is used to prove the existence of solutions, while Banach's contraction principle is used to establish uniqueness. These results ensure that the model is mathematically well-posed and that its solutions can be approximated reliably through numerical methods. Furthermore, the existence of a unique solution guarantees that the dynamic behavior of the system is completely determined by the prescribed initial

conditions. For $0 < \varsigma \leq 1$, System (2) is reformulated in the Caputo fractional-order framework, which enables the incorporation of memory and hereditary effects into the bacterial dynamics.

Let $\lambda(t) := (S(t), L(t), I(t), R(t), P(t))^T$ denote the state vector, and define

$$\begin{aligned}\zeta_1(t, \lambda(t)) &= \alpha - \beta S(t)L(t) + \gamma P(t) - \mu S(t) + \gamma_1 R(t), \\ \zeta_2(t, \lambda(t)) &= \beta S(t)L(t) - \mu L(t) - \beta_1 L(t), \\ \zeta_3(t, \lambda(t)) &= \beta_1 L(t) - (\beta_2 + \mu)I(t), \\ \zeta_4(t, \lambda(t)) &= \beta_2 I(t) - (\beta_3 + \mu + \gamma_1)R(t), \\ \zeta_5(t, \lambda(t)) &= \beta_3 R(t) - (\gamma + \mu)P(t).\end{aligned}\tag{3.11}$$

Using the initial conditions and the fractional integral operator, the system can be expressed in integral form as

$$\begin{aligned}S(t) &= S^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \zeta_1(\phi, \lambda(\phi)) d\phi, \\ L(t) &= L^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \zeta_2(\phi, \lambda(\phi)) d\phi, \\ I(t) &= I^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \zeta_3(\phi, \lambda(\phi)) d\phi, \\ R(t) &= R^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \zeta_4(\phi, \lambda(\phi)) d\phi, \\ P(t) &= P^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \zeta_5(\phi, \lambda(\phi)) d\phi.\end{aligned}\tag{3.12}$$

Let

$$\lambda(t) := \begin{pmatrix} S(t) \\ L(t) \\ I(t) \\ R(t) \\ P(t) \end{pmatrix}, \quad \lambda^0 := \lambda(0) = \begin{pmatrix} S^0 \\ L^0 \\ I^0 \\ R^0 \\ P^0 \end{pmatrix}, \quad \rho(\phi, \lambda(\phi)) := \begin{pmatrix} \zeta_1(\phi, \lambda(\phi)) \\ \zeta_2(\phi, \lambda(\phi)) \\ \zeta_3(\phi, \lambda(\phi)) \\ \zeta_4(\phi, \lambda(\phi)) \\ \zeta_5(\phi, \lambda(\phi)) \end{pmatrix}.\tag{3.13}$$

Using the vector quantities defined in (3.13), the componentwise system (3.12) can be written in the compact form

$$\lambda(t) = \lambda^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \rho(\phi, \lambda(\phi)) d\phi.\tag{3.14}$$

Let us consider the Banach space $\mathfrak{J}[0, \kappa] = \varpi$ equipped with the norm.

$$\|S, L, I, R, P\| = \max_{t \in [0, \kappa]} [S, L, I, R, P]$$

Define a mapping $\mathfrak{N} : \hbar \rightarrow \hbar$ by

$$\mathfrak{N}\lambda(t) = \lambda^0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t-\phi)^{\varsigma-1} \rho(\phi, \lambda(\phi)) d\phi$$

On a nonlinear function, we can apply the hypothesis

F_1 : There exists a constant $\Psi_o, \Psi_o > 0$, thus

$$|\rho(t, \lambda(t))| \leq \Psi_o |\lambda(t)| + \Psi_o$$

F_2 : There exists a constant $L_o > 0$ such that, for all $\lambda, \lambda_1 \in \tilde{h}$,

$$|\rho(t, \bar{\lambda}) - \rho(t, \bar{\lambda}_1)| \leq L_o |\bar{\lambda} - \bar{\lambda}_1|$$

Theorem 2: If the assumption F_1 holds, then System (2) has at least one solution.

Proof: To prove the boundedness of $\aleph$, consider $\diamond = \lambda \in \phi | \psi \geq \|\lambda\|$, where

$$\psi \geq \max_{t \in [0, \kappa]} \frac{\lambda_0 + \frac{\psi_o \kappa^\varsigma}{\Gamma(\varsigma+1)}}{1 - \frac{\psi_o \kappa^\varsigma}{\Gamma(\varsigma+1)}} \quad (3.15)$$

is a closed and convex subset of $\tilde{h}$. After that,

$$\begin{aligned} \|\aleph \lambda\| &= \max_{t \in [0, \kappa]} \left| \lambda_0 + \frac{1}{\Gamma(\varsigma)} \int_0^t (t - \phi)^{\varsigma-1} \rho(\phi, \lambda(\phi)) d\phi \right| \\ &\leq |\lambda_0| + \frac{1}{\Gamma(\varsigma)} \int_0^t (t - \phi)^{\varsigma-1} |\rho(\phi, \lambda(\phi))| d\phi \\ &\leq |\lambda_0| + \frac{1}{\Gamma(\varsigma)} \int_0^t (t - \phi)^{\varsigma-1} [\psi_o |\lambda(t)| + \psi_o] d\phi \\ &\leq |\lambda_0| + [\psi_o \|\lambda(t)\| + \psi_o] \frac{\kappa^\varsigma}{\Gamma(\varsigma+1)} \leq \psi \end{aligned} \quad (3.16)$$

Since $\lambda \in \emptyset \implies \aleph(\emptyset) \subseteq \emptyset$, it demonstrates that $\aleph$ is bounded. Let $t_1 < t_2 \in [0, \kappa]$, in order to demonstrate the complete continuity of $\aleph$, let

$$\begin{aligned} \|\aleph \lambda(t_2) - \aleph \lambda(t_1)\| &= \left| \frac{1}{\Gamma(\varsigma)} \int_0^{t_2} (t_2 - \phi)^{\varsigma-1} \rho(\phi, \lambda(\phi)) d\phi \right. \\ &\quad \left. - \frac{1}{\Gamma(\varsigma)} \int_0^{t_1} (t_1 - \phi)^{\varsigma-1} \rho(\phi, \lambda(\phi)) d\phi \right| \\ &\leq [\psi_o \|\lambda(t)\| + \psi_o] \frac{t_2^\varsigma - t_1^\varsigma}{\Gamma(\varsigma+1)} \end{aligned} \quad (3.17)$$

This shows that $\|\aleph \lambda(t_2) - \aleph \lambda(t_1)\| \rightarrow 0$ as $t_2 \rightarrow t_1$. This implies the continuity of $\aleph$. Consequently, by Schauder's fixed point theorem, System (2) has at least one solution. Moreover, Banach's fixed point theorem guarantees the uniqueness of this solution.

Theorem 3: The System (2) has a unique solution if condition $F2$ is satisfied.

Proof: Let $\bar{\lambda}, \bar{\lambda}_1 \in \hbar$. Take

$$\begin{aligned} \|\aleph \bar{\lambda} - \aleph \bar{\lambda}_1\| &= \max_{t \in [0, \kappa]} \left| \frac{1}{\Gamma(\varsigma)} \int_0^t (t_2 - \phi)^{\varsigma-1} \rho(\phi, \bar{\lambda}) d\phi \right. \\ &\quad \left. - \frac{1}{\Gamma(\varsigma)} \int_0^t (t_1 - \phi)^{\varsigma-1} \rho(\phi, \bar{\lambda}_1) d\phi \right| \\ &\leq \frac{T^\varsigma}{\Gamma(\varsigma + 1)} L_o |\bar{\lambda} - \bar{\lambda}_1|. \end{aligned} \quad (3.18)$$

Thus, $\aleph$ is a contraction. By applying the Banach fixed-point theorem, we conclude that System (2) has a unique solution.

3.4. Numerical analysis of fractional equations via the Caputo operator

Theorem 4: Supposing $(\wp, |\cdot|)$ be a Banach space and $\mathfrak{I} : \wp \rightarrow \wp$ be a map satisfying:

$$\|\mathfrak{I}_o - \mathfrak{I}_\varrho\| \leq \varpi \|X - \mathfrak{I}_o\| + \pi \|o - \varrho\|$$

For all $o, \varrho \in \wp$, where $0 \leq \varpi, 0 \leq \pi < 1$. Then $\mathfrak{I}$ is Picard $\mathfrak{I}$ -stable.

Theorem 5: Consider $\mathfrak{I}$ a self-map defined by the following expression:

$$\begin{aligned} \mathfrak{I}[S_i(t)] &= S_{i+1} = S_i(0) + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \alpha - \beta S_i L_i + \gamma P_i - \mu S_i + \gamma_1 R_i \} \right], \\ \mathfrak{I}[L_i(t)] &= L_{i+1} = L_i(0) + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta S_i L_i - \mu L_i - \beta_1 L_i \} \right], \\ \mathfrak{I}[I_i(t)] &= I_{i+1} = I_i(0) + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_1 L_i - (\beta_2 + \mu) I_i \} \right], \\ \mathfrak{I}[R_i(t)] &= R_{i+1} = R_i(0) + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_2 I_i - (\beta_3 + \mu + \gamma_1) R_i \} \right], \\ \mathfrak{I}[P_i(t)] &= P_{i+1} = P_i(0) + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_3 R_i - (\gamma + \mu) P_i \} \right] \end{aligned} \quad (3.19)$$

Provided that the iteration is $\mathfrak{I}$ -stable in

$$\begin{aligned} (1 - \beta(k_1 + k_2)\mathfrak{F}_1(\varphi) - \gamma\mathfrak{F}_2(\varphi) - \mu\mathfrak{F}_3(\varphi) - \gamma_1\mathfrak{F}_4(\varphi)) &< 1 \\ (1 + \beta(k_1 + k_2)\mathfrak{F}_1(\varphi) - \mu + \beta)\mathfrak{F}_3(\varphi) &< 1 \\ (1 + \beta_1\mathfrak{F}_4(\varphi) - (\beta_2 + \mu)\mathfrak{F}_6(\varphi)) &< 1 \end{aligned} \quad (3.20)$$

$$(1 + \beta_2 \mathfrak{L}_7(\varphi) - (\beta_3 + \mu + \gamma_1) \mathfrak{L}_9(\varphi)) < 1$$

$$(1 + \beta_3 \mathfrak{L}_{10}(\varphi) - (\mu + \gamma) \mathfrak{L}_{12}(\varphi)) < 1$$

Proof

$$\begin{aligned}
 \mathfrak{I}[S_\ell(t) - S_i(t)] &= S_\ell(t) - S_i(t) \\
 &\quad + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \alpha - \beta S_\ell L_\ell + \gamma P_\ell - \mu S_\ell + \gamma_1 R_\ell \} \right] \\
 &\quad - \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \alpha - \beta S_i L_i + \gamma P_i - \mu S_i + \gamma_1 R_i \} \right], \\
 \mathfrak{I}[L_\ell(t) - L_i(t)] &= L_\ell(t) - L_i(t) \\
 &\quad + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta S_\ell L_\ell - \mu L_\ell - \beta_1 L_\ell \} \right] \\
 &\quad - \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta S_i L_i - \mu L_i - \beta_1 L_i \} \right], \\
 \mathfrak{I}[I_\ell(t) - I_i(t)] &= I_\ell(t) - I_i(t) \\
 &\quad + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_1 L_\ell - (\beta_2 + \mu) I_\ell \} \right] \\
 &\quad - \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_1 L_i - (\beta_2 + \mu) I_i \} \right], \\
 \mathfrak{I}[R_\ell(t) - R_i(t)] &= R_\ell(t) - R_i(t) \\
 &\quad + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_2 I_\ell - (\beta_3 + \mu + \gamma_1) R_\ell \} \right] \\
 &\quad - \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_2 I_i - (\beta_3 + \mu + \gamma_1) R_i \} \right], \\
 \mathfrak{I}[P_\ell(t) - P_i(t)] &= P_\ell(t) - P_i(t) \\
 &\quad + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_3 R_\ell - (\gamma + \mu) P_\ell \} \right] \\
 &\quad - \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \{ \beta_3 R_i - (\gamma + \mu) P_i \} \right]
 \end{aligned} \tag{3.21}$$

By taking the norm of both sides of Eq. (3.21), we get

$$\begin{aligned}
\|\mathfrak{I}[S_\ell(t) - S_i(t)]\| &= \left\| S_\ell(t) - S_i(t) + \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\alpha - \beta S_\ell L_\ell + \gamma P_\ell - \mu S_\ell + \gamma_1 R_\ell\} \right] \right. \\
&\quad \left. - \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\alpha - \beta S_i L_i + \gamma P_i - \mu S_i + \gamma_1 R_i\} \right] \right\| \\
\|\mathfrak{I}[L_\ell(t) - L_i(t)]\| &= \left\| L_\ell(t) - L_i(t) + \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{S_\ell L_\ell - \mu L_\ell - \beta_1 L_\ell\} \right] \right. \\
&\quad \left. - \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{S_i L_i - \mu L_i - \beta_1 L_i\} \right] \right\| \\
\|\mathfrak{I}[I_\ell(t) - I_i(t)]\| &= \left\| I_\ell(t) - I_i(t) + \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\beta_1 L_\ell - (\beta_2 + \mu) I_\ell\} \right] \right. \\
&\quad \left. - \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\beta_1 L_i - (\beta_2 + \mu) I_i\} \right] \right\| \\
\|\mathfrak{I}[R_\ell(t) - R_i(t)]\| &= \left\| R_\ell(t) - R_i(t) + \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\beta_2 I_\ell - (\beta_3 + \mu + \gamma_1) R_\ell\} \right] \right. \\
&\quad \left. - \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\beta_2 I_i - (\beta_3 + \mu + \gamma_1) R_i\} \right] \right\| \\
\|\mathfrak{I}[P_\ell(t) - P_i(t)]\| &= \left\| P_\ell(t) - P_i(t) + \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\beta_3 R_\ell - (\gamma + \mu) P_\ell\} \right] \right. \\
&\quad \left. - \mathcal{L}^{-1} \left[\frac{1}{\chi_s} \mathcal{L}\{\beta_3 R_i - (\gamma + \mu) P_i\} \right] \right\|
\end{aligned} \tag{3.22}$$

By applying the triangle inequality to Eq. (3.22) and simplifying, we obtain

$$\begin{aligned}
\|\mathfrak{I}[S_\ell(t) - S_i(t)]\| &\leq \|S_\ell(t) - S_i(t)\| + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \left\{ \|\beta S_\ell(L_\ell - L_i)\| \right. \right. \\
&\quad + \|\beta L_\ell(S_\ell - S_i)\| + \|\gamma(P_\ell - P_i)\| \\
&\quad \left. \left. + \|\mu(S_\ell - S_i)\| + \|\gamma_1(R_\ell - R_i)\| \right\} \right] \\
\|\mathfrak{I}[L_\ell(t) - L_i(t)]\| &\leq \|L_\ell(t) - L_i(t)\| + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \left\{ \|\beta(S_\ell - S_i)\| \cdot \|L_\ell\| \right. \right. \\
&\quad \left. \left. + \beta\|S_\ell\| \cdot \|L_\ell - L_i\| - \mu\|L_\ell - L_i\| - \beta_1\|L_\ell - L_i\| \right\} \right] \\
\|\mathfrak{I}[I_\ell(t) - I_i(t)]\| &\leq \|I_\ell(t) - I_i(t)\| + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \left\{ \|\beta_1(L_\ell - L_i)\| \right. \right. \\
&\quad \left. \left. + \|(\beta_2 + \mu)(I_\ell - I_i)\| \right\} \right] \\
\|\mathfrak{I}[R_\ell(t) - R_i(t)]\| &\leq \|R_\ell(t) - R_i(t)\| + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \left\{ \|\beta_2(I_\ell - I_i)\| \right. \right. \\
&\quad \left. \left. + \|(\beta_3 + \mu + \gamma_1)(R_\ell - R_i)\| \right\} \right] \\
\|\mathfrak{I}[P_\ell(t) - P_i(t)]\| &\leq \|P_\ell(t) - P_i(t)\| + \mathcal{L}^{-1} \left[\frac{1}{\chi_\varsigma} \mathcal{L} \left\{ \|\beta_3(R_\ell - R_i)\| \right. \right. \\
&\quad \left. \left. + \|(\gamma + \mu)(P_\ell - P_i)\| \right\} \right]
\end{aligned} \tag{3.23}$$

Considering the relative impact of both solutions, we choose

$$\begin{aligned}
\|S_\ell(t) - S_i(t)\| &\cong \|L_\ell(t) - L_i(t)\| \\
&\cong \|I_\ell(t) - I_i(t)\| \\
&\cong \|R_\ell(t) - R_i(t)\| \\
&\cong \|P_\ell(t) - P_i(t)\|
\end{aligned} \tag{3.24}$$

Moreover, the convergent sequences $S_\ell, L_\ell, I_\ell, R_\ell, P_\ell$ are bounded. We have five different positive constants, M_1, M_2, M_3, M_4, M_5 , for all t such that

$$\|S_\ell\| < M_1, \|L_\ell\| < M_2, \|I_\ell\| < M_3, \|R_\ell\| < M_4, \|P_\ell\| < M_5.$$

Further, considering the equations above, we get

$$\begin{aligned}
 \|\mathfrak{I}[S_\ell(t) - S_i(t)]\| &\leq \left[1 - \beta(k_1 + k_2)\mathfrak{F}_1(\varphi) - \gamma\mathfrak{F}_2(\varphi) - \mu\mathfrak{F}_3(\varphi) - \gamma_1\mathfrak{F}_4(\varphi)\right] \cdot \|S_\ell - S_i\| \\
 \|\mathfrak{I}[L_\ell(t) - L_i(t)]\| &\leq \left[1 + \beta(k_1 + k_2)\mathfrak{F}_1(\varphi) - \mu + \beta\right]\mathfrak{F}_3(\varphi) \cdot \|L_\ell - L_i\| \\
 \|\mathfrak{I}[I_\ell(t) - I_i(t)]\| &\leq \left[1 + \beta_1\mathfrak{F}_4(\varphi) - (\beta_2 + \mu)\mathfrak{F}_6(\varphi)\right] \cdot \|I_\ell - I_i\| \\
 \|\mathfrak{I}[R_\ell(t) - R_i(t)]\| &\leq \left[1 + \beta_2\mathfrak{F}_7(\varphi) - (\beta_3 + \mu + \gamma_1)\mathfrak{F}_9(\varphi)\right] \cdot \|R_\ell - R_i\| \\
 \|\mathfrak{I}[P_\ell(t) - P_i(t)]\| &\leq \left[1 + \beta_3\mathfrak{F}_{10}(\varphi) - (\mu + \gamma)\mathfrak{F}_{12}(\varphi)\right] \cdot \|P_\ell - P_i\|
 \end{aligned} \tag{3.25}$$

As a consequence of this, $\mathfrak{I}$ has a fixed point. Considering the equations listed above, we can assume the following:

$$\mathfrak{I} = \begin{cases} (1 - \beta(k_1 + k_2)\mathfrak{F}_1(\varphi) - \gamma\mathfrak{F}_2(\varphi) - \mu\mathfrak{F}_3(\varphi) - \gamma_1\mathfrak{F}_4(\varphi) \\ (1 + \beta(k_1 + k_2)\mathfrak{F}_1(\varphi) - \mu + \beta)\mathfrak{F}_3(\varphi) \\ (1 + \beta_1\mathfrak{F}_4(\varphi) - (\beta_2 + \mu)\mathfrak{F}_6(\varphi)) \\ (1 + \beta_2\mathfrak{F}_7(\varphi) - (\beta_3 + \mu + \gamma_1)\mathfrak{F}_9(\varphi)) \\ (1 + \beta_3\mathfrak{F}_{10}(\varphi) - (\mu + \gamma)\mathfrak{F}_{12}(\varphi)) \end{cases} \tag{3.26}$$

3.5. Equilibrium point analysis

In-depth analysis of the equilibrium points is presented in this section. To examine these equilibrium points, by solving System (2), if $S^* = \frac{\alpha}{\mu}$ then we have $L^* = 0$, $I^* = 0$, $R^* = 0$, and $P^* = 0$, which are equilibrium points that are disease-free

$$E_0 = \left(\frac{\alpha}{\mu}, 0, 0, 0, 0\right)$$

The endemic equilibrium points will be

$$E^* = (S^*, L^*, I^*, R^*, P^*)$$

After substituting the parameter values, we obtain $S^* = 9.28571$, $L^* = 0.0939411$, $I^* = 0.0867148$, $R^* = 0.433574$, and $P^* = 0.100056$. E^* is deemed to be absolutely stable if the system's equilibrium point remains asymptotically stable for all possible delays. Conversely, it is considered to be conditionally stable if the equilibrium point is asymptotically stable only for certain ranges of delays but not universally.

4. Stability analysis

Equilibrium point stability, characterized by Lyapunov functions, is fundamental in systems and control theory. This method enables an analysis of asymptotic stability for both linear and nonlinear systems without requiring explicit solutions to the differential equation. The asymptotic stability of a scalar positive definite function with a negative definite time derivative is well established. In this context, we present a key lemma for analyzing the global stability of the developed system.

The following inequality, widely used in Lyapunov-based stability analyses of fractional-order dynamic systems with Caputo derivatives, is adopted from the fractional Lyapunov stability framework presented in [21].

Lemma 4.1. Suppose that for any $t \geq t_0$, $E(t) \in \mathbb{R}^+$ is a continuous function. Then

$${}^c D_t^\varsigma \left(E - E^* - E^* \ln \frac{E}{E^*} \right) \leq \left(1 - \frac{E^*}{E} \right) {}^c D_t^\varsigma E(t), \quad (4.1)$$

where $E^* \in \mathbb{R}^+$ and $0 < \varsigma < 1$.

Theorem 6: The disease-free equilibrium C_0 is globally asymptotically stable if the reproductive number is less than 1

Proof:

Define the Lyapunov function as follows:

$$E = (S - S_0 - S_0 \log \frac{S}{S_0}) + L + I + R + P \quad (4.2)$$

From the lemma, we get-

$${}^c D_t^\varsigma E \leq (1 - \frac{S_0}{S}) {}^c D_t^\varsigma S + {}^c D_t^\varsigma L + {}^c D_t^\varsigma I + {}^c D_t^\varsigma R + {}^c D_t^\varsigma P$$

Substituting the derivative values from (2), we have

$$\begin{aligned} {}^c D_t^\varsigma E &\leq (1 - \frac{S_0}{S})(\alpha - \beta S L + \gamma P - \mu S + \gamma_1 R) + (\beta S L - \mu L - \beta_1 L) \\ &\quad + (\beta_1 L - (\beta_2 + \mu)I) + (\beta_2 I - (\beta_3 + \mu + \gamma_1)R) + (\beta_3 R - (\gamma + \mu)P) \end{aligned}$$

Replacing $S = S - S_0, L = L - L_0, I = I - I_0, R = R - R_0, P = P - P_0$, we can have the following,

$$\begin{aligned} {}^c D_t^\varsigma E &\leq \left(1 - \frac{S_0}{S} \right) (\alpha - \beta(S - S_0)(L - L_0) + \gamma(P - P_0) \\ &\quad - \mu(S - S_0) + \gamma_1(R - R_0)) \\ &\quad + (\beta(S - S_0)(L - L_0) - \mu(L - L_0) - \beta_1(L - L_0)) \\ &\quad + (\beta_1(L - L_0) - (\beta_2 + \mu)(I - I_0)) \\ &\quad + (\beta_2(I - I_0) - (\beta_3 + \mu + \gamma_1)(R - R_0)) \\ &\quad + (\beta_3(R - R_0) - (\gamma + \mu)(P - P_0)) \end{aligned} \quad (4.3)$$

As is evident, for

$$R_0 < 1 \quad \implies {}^c D_t^\varsigma E \leq 0$$

and ${}^c D_t^\varsigma E = 0$ only when,

$$S = S - S_0, \quad L = L - L_0, \quad I = I - I_0 \quad R = R - R_0 \quad P = P - P_0$$

It is concluded that the disease-free equilibrium state C_0 is globally asymptotically stable.

Considering the specific Lyapunov function defined earlier, $\{S, L, I, R, P\}$, $E < 0$ is the endemic equilibrium point C^* .

Theorem 7: The endemic equilibrium points C^* is globally asymptotically stable if the reproductive number $R_0 > 1$

Proof: We introduce a Volterra-type Lyapunov function given by:

$$\begin{aligned} E_Y = & Y_1 \left(S - S^* - S^* \log \frac{S}{S^*} \right) + Y_2 \left(L - L^* - L^* \log \frac{L}{L^*} \right) \\ & + Y_3 \left(I - I^* - I^* \log \frac{I}{I^*} \right) + Y_4 \left(R - R^* - R^* \log \frac{R}{R^*} \right) \\ & + Y_5 \left(P - P^* - P^* \log \frac{P}{P^*} \right) \end{aligned} \quad (4.4)$$

where $Y_i = 1, 2, 3, 4, 5$ are positive constants. After substituting Eq (4.4) into the main system, apply Lemma (4.1),

$$\begin{aligned} {}^c D_t^\varsigma E \leq & Y_1 \left(\frac{S - S^*}{S} \right) {}^c D_t^\varsigma S + Y_2 \left(\frac{L - L^*}{L} \right) {}^c D_t^\varsigma L + Y_3 \left(\frac{I - I^*}{I} \right) {}^c D_t^\varsigma I \\ & + Y_4 \left(\frac{R - R^*}{R} \right) {}^c D_t^\varsigma R + Y_5 \left(\frac{P - P^*}{P} \right) {}^c D_t^\varsigma P. \end{aligned} \quad (4.5)$$

In Eq (4.5), the values of derivative can be written as

$$\begin{aligned}
{}^c D_t^s E \leq & Y_1 \cdot \frac{S - S^*}{S} \cdot \alpha - Y_1 \cdot \frac{S - S^*}{S} \cdot \beta S L + Y_1 \cdot \frac{S - S^*}{S} \cdot \gamma P \\
& - Y_1 \cdot \frac{S - S^*}{S} \cdot \mu S + Y_1 \cdot \frac{S - S^*}{S} \cdot \gamma_1 R \\
& + Y_2 \cdot \frac{L - L^*}{L} \cdot \beta S L - Y_2 \cdot \frac{L - L^*}{L} \cdot \mu L - Y_2 \cdot \frac{L - L^*}{L} \cdot \beta_1 L \\
& + Y_3 \cdot \frac{I - I^*}{I} \cdot \beta_1 L - Y_3 \cdot \frac{I - I^*}{I} \cdot (\beta_2 + \mu) I \\
& + Y_4 \cdot \frac{R - R^*}{R} \cdot \beta_2 I - Y_4 \cdot \frac{R - R^*}{R} \cdot (\beta_3 + \mu + \gamma_1) R \\
& + Y_5 \cdot \frac{P - P^*}{P} \cdot \beta_3 R - Y_5 \cdot \frac{P - P^*}{P} \cdot (\gamma + \mu) P
\end{aligned} \tag{4.6}$$

By putting, $S = S - S^*$, $L = L - L^*$, $I = I - I^*$, $R = R - R^*$, $P = P - P^*$, we can write as

$$\begin{aligned}
{}^c D_t^s E \leq & Y_1 \cdot \frac{S - S^*}{S} \cdot [\alpha - \beta(S - S^*)(L - L^*) + \gamma(P - P^*) \\
& - \mu(S - S^*) + \gamma_1(R - R^*)] \\
& + Y_2 \cdot \frac{L - L^*}{L} \cdot [\beta(S - S^*)(L - L^*) - \mu(L - L^*) - \beta_1(L - L^*)] \\
& + Y_3 \cdot \frac{I - I^*}{I} \cdot [\beta_1(L - L^*) - (\beta_2 + \mu)(I - I^*)] \\
& + Y_4 \cdot \frac{R - R^*}{R} \cdot [\beta_2(I - I^*) - (\beta_3 + \mu + \gamma_1)(R - R^*)] \\
& + Y_5 \cdot \frac{P - P^*}{P} \cdot [\beta_3(R - R^*) - (\gamma + \mu)(P - P^*)]
\end{aligned} \tag{4.7}$$

Let $Y_1 = Y_2 = Y_3 = Y_4 = Y_5 = 1$. We can organize the above as follows:

$$\begin{aligned}
 {}^c D_t^\zeta E \leq & \alpha - \alpha \cdot \frac{S^\star}{S} - \beta \cdot \frac{(S - S^\star)^2}{S} \cdot L + \beta \cdot \frac{(S - S^\star)^2}{S} \cdot L^\star \\
 & + \gamma P - \gamma P^\star - \gamma P \cdot \frac{S^\star}{S} + \gamma P^\star \cdot \frac{S^\star}{S} \\
 & - \mu \cdot \frac{(S - S^\star)^2}{S} \\
 & + \gamma_1 R - \gamma_1 R^\star - \gamma_1 R \cdot \frac{S^\star}{S} + \gamma_1 R^\star \cdot \frac{S^\star}{S} \\
 & + \beta \cdot \frac{(L - L^\star)^2}{L} \cdot S - \beta \cdot \frac{(L - L^\star)^2}{L} \cdot S^\star - (\mu + \beta_1) \cdot \frac{(L - L^\star)^2}{L} \\
 & + \beta_1 L - \beta_1 L \cdot \frac{I^\star}{I} - \beta_1 L^\star + \beta_1 L^\star \cdot \frac{I^\star}{I} \\
 & - (\beta_2 + \mu) \cdot \frac{(I - I^\star)^2}{I} \\
 & + I\beta_2 - \beta_2 I^\star - \beta_2 I \cdot \frac{R^\star}{R} + \beta_2 I^\star \cdot \frac{R^\star}{R} \\
 & - (\beta_3 + \mu + \gamma_1) \cdot \frac{(R - R^\star)^2}{R} \\
 & + \beta_3 R - \beta_3 R^\star - \beta_3 R \cdot \frac{P^\star}{P} + \beta_3 R^\star \cdot \frac{P^\star}{P} \\
 & - (\gamma + \mu) \cdot \frac{(P - P^\star)^2}{P}
 \end{aligned} \tag{4.8}$$

We can write

$${}^c D_t^\zeta E \leq F_1 - F_2, \tag{4.9}$$

where,

$$\begin{aligned}
 F_1 = & \alpha + \beta \cdot \frac{(S - S^\star)^2}{S} \cdot L^\star + \gamma P + \gamma P^\star \cdot \frac{S^\star}{S} \\
 & + \gamma_1 R + \gamma_1 R^\star \cdot \frac{S^\star}{S} + \beta \cdot \frac{(L - L^\star)^2}{L} \cdot S \\
 & + \beta_1 L + \beta_1 L^\star \cdot \frac{I^\star}{I} + I\beta_2 + I^\star \cdot \frac{R^\star}{R} \\
 & + \beta_3 R + \beta_3 R^\star \cdot \frac{P^\star}{P}
 \end{aligned} \tag{4.10}$$

$$\begin{aligned}
F_2 = & -\alpha \cdot \frac{S^*}{S} - \beta \cdot \frac{(S - S^*)^2}{S} \cdot L - \gamma P^* - \gamma P \cdot \frac{S^*}{S} \\
& - \mu \cdot \frac{(S - S^*)^2}{S} - \gamma_1 R^* - \gamma_1 R \cdot \frac{S^*}{S} \\
& - \beta \cdot \frac{(L - L^*)^2}{L} \cdot S^* - (\mu + \beta_1) \cdot \frac{(L - L^*)^2}{L} \\
& - \beta_1 L^* \cdot \frac{I^*}{I} - \beta_1 L^* \\
& - (\beta_2 + \mu) \cdot \frac{(I - I^*)^2}{I} - \beta_2 I^* - \beta_2 I \cdot \frac{R^*}{R} \\
& - (\beta_3 + \mu + \gamma_1) \cdot \frac{(R - R^*)^2}{R} - \beta_3 R^* - \beta_3 R \cdot \frac{P^*}{P} \\
& - (\gamma + \mu) \cdot \frac{(P - P^*)^2}{P}
\end{aligned} \tag{4.11}$$

We examine that if

$$F_1 < F_2 \quad \implies^c D_t^s E < 0,$$

when $S = S^*, L = L^*, I = I^*, R = R^*, P = P^*$, then

$$F_1 - F_2 = 0 \quad \implies^c D_t^s E = 0$$

from the above result we can see that $C^* = S^*, L^*, I^*, R^*, P^*$ is the most extensive compact invariant singleton set within.

$$\{(S^*, L^*, I^*, R^*, P^*) \in \Theta :^c D_t^s E = 0\}.$$

In the invariant region, the endemic equilibrium point C^* is globally asymptotically stable if $F_1 < F_2$.

5. Chaos control

The equilibrium points of the System (2) are stabilized through the application of the linear feedback regulation method (17) to a fractional-order system with a controlled design.

$$\begin{aligned}
{}^c D_t^s S(t) &= \alpha - \beta S L + \gamma P - \mu S + \gamma_1 R(S - \dot{S}) \\
{}^c D_t^s L(t) &= \beta S L - \mu L - \beta_1 L(L - \dot{L}) \\
L {}^c D_t^s I(t) &= \beta_1(L) - (\beta_2 + \mu)I(I - \dot{I}) \\
{}^c D_t^s R(t) &= \beta_2 I - (\beta_3 + \mu + \gamma_1)R(R - \dot{R}) \\
{}^c D_t^s P(t) &= \beta_3 R - (\gamma + \mu)P(P - \dot{P})
\end{aligned} \tag{5.1}$$

Let S^* , L^* , I^* , R^* , and P^* denote the components of the equilibrium point of System (2). The Jacobian matrix associated with the system is then given by

$$J = \begin{pmatrix} -\beta L - \mu & -\beta S & 0 & \gamma_1 & \gamma \\ \beta L & \beta S - \mu - \beta_1 & 0 & 0 & 0 \\ 0 & \beta_1 & -\beta_2 - \mu & 0 & 0 \\ 0 & 0 & \beta_2 & -\beta_3 - \mu - \gamma_1 & 0 \\ 0 & 0 & 0 & \beta_3 & -(\gamma + \mu) \end{pmatrix}.$$

Choosing $\alpha = 0.5$, $\beta = 0.07$, $\gamma = 0.08$, $\mu = 0.05$, $\gamma_1 = 0.04$, $\beta_1 = 0.6$, $\beta_2 = 0.6$, $\beta_3 = 0.03$, the characteristic polynomial according to the equilibrium point is as follows:

$$C(\psi) = \psi^5 - 0.9\psi^4 - 0.1756\psi^3 - 0.00789\psi^2 + 0.00044525\psi + 0.00002535 \quad (5.2)$$

For Eq (5.2), the characteristic roots are

$$\begin{aligned} \psi_1 &= -0.13, & \psi_2 &= -0.05, \\ \psi_3 &= 0.05, & \psi_4 &= -0.65, & \psi_5 &= -0.12. \end{aligned} \quad (5.3)$$

Based on these eigenvalues, the stability of the equilibrium point can be assessed.

The results presented in Figures 2 and 3 demonstrate the existence of complex nonlinear dynamics in the proposed SLIRP model. The oscillatory trajectories observed in the susceptible, lysogenic, infected, resistant, and phage populations indicate chaotic behavior arising from strong interactions among the bacterial compartments. The phase plane portraits shown in Figure 3a and 3b also reveal the nonlinear coupling between the susceptible and lysogenic populations and between the resistant and infected populations, confirming the complexity of the system's dynamics.

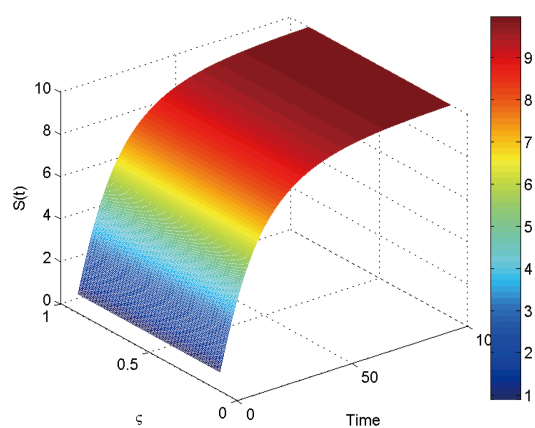
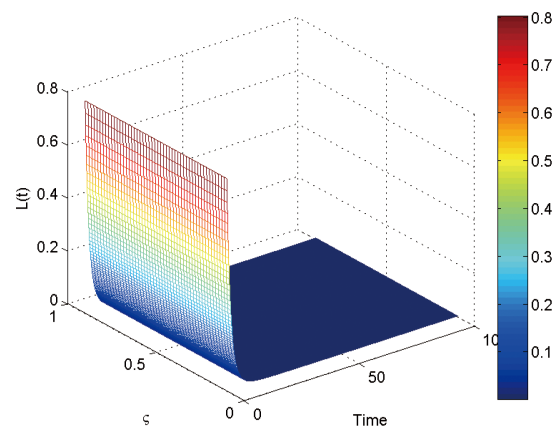
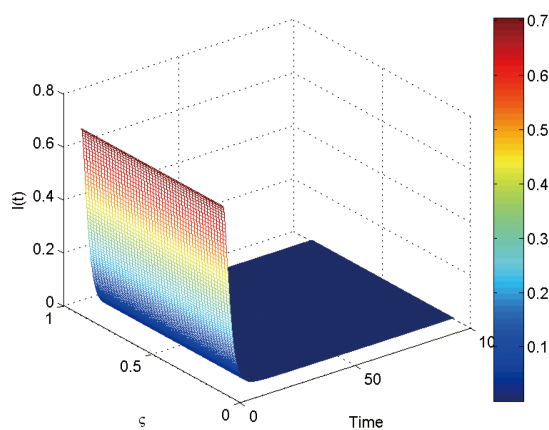
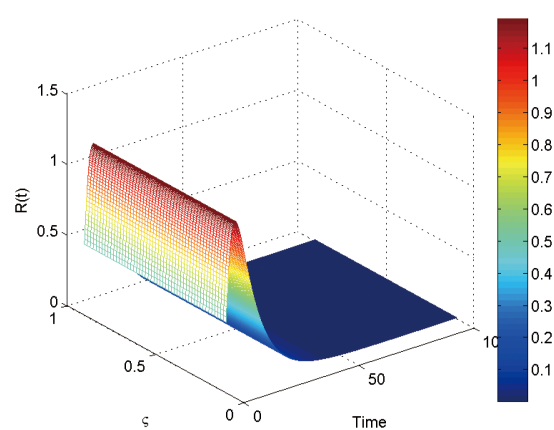
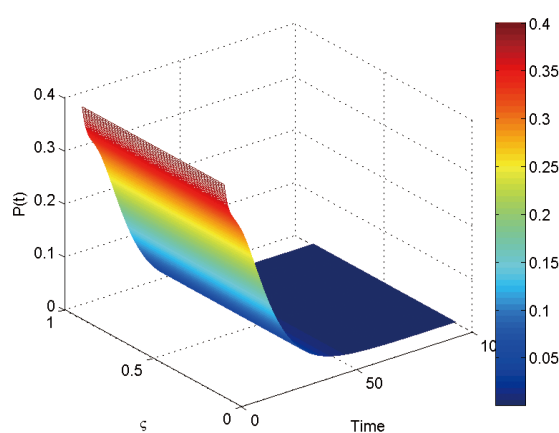
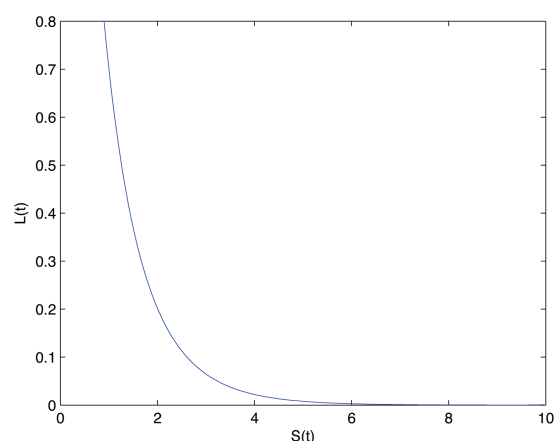
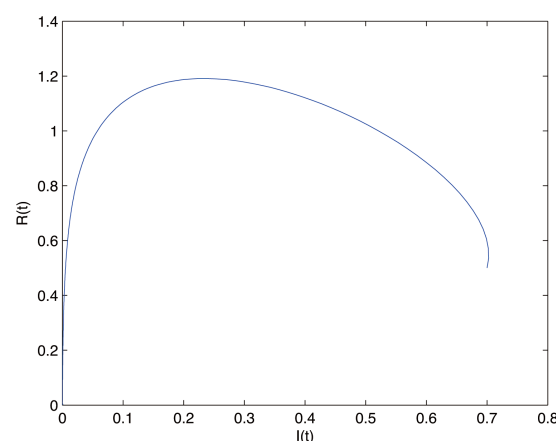
(a) Susceptible bacteria $S(t)$.(b) Lysogenic bacteria $L(t)$.(c) Infected bacteria $I(t)$.(d) Resistant bacteria $R(t)$.(e) Phage population $P(t)$.

Figure 2. Chaotic temporal dynamics of the susceptible, lysogenic, infected, resistant, and phage populations in the fractional-order SLIRP model.



(a) Phase portrait of the susceptible and lysogenic populations (S, L).



(b) Phase portrait of the resistant and infected populations (R, I).

Figure 3. Phase plane analysis of the fractional-order SLIRP model illustrates the nonlinear interactions between the susceptible and lysogenic populations and between the resistant and infected populations. The closed trajectories and irregular patterns indicate complex dynamic behavior and strong coupling among the model's compartments.

6. Computational analysis with the fractional operator

The Caputo derivative, when used with a power-law kernel, can effectively model processes exhibiting power-law behavior. By substituting the time derivative with the Caputo derivative and using Newton polynomial interpolation, we incorporate power-law effects from fractional calculus. We present a numerical approach for solving System (2) by using a Newton polynomial basis and applying the power-law kernel with fractional integration. This results in

$$\begin{aligned}
 S(t_{\epsilon+1}) &= S(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \int_{t_m}^{t_{m+1}} S_1(t, S) \phi^{1-\varphi} (t_{\epsilon+1} - \phi)^{\varsigma+1} d\phi \\
 L(t_{\epsilon+1}) &= L(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \int_{t_m}^{t_{m+1}} L_1(t, L) \phi^{1-\varphi} (t_{\epsilon+1} - \phi)^{\varsigma+1} d\phi \\
 I(t_{\epsilon+1}) &= I(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \int_{t_m}^{t_{m+1}} I_1(t, I) \phi^{1-\varphi} (t_{\epsilon+1} - \phi)^{\varsigma+1} d\phi \\
 R(t_{\epsilon+1}) &= R(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \int_{t_m}^{t_{m+1}} R_1(t, R) \phi^{1-\varphi} (t_{\epsilon+1} - \phi)^{\varsigma+1} d\phi \\
 P(t_{\epsilon+1}) &= P(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \int_{t_m}^{t_{m+1}} P_1(t, P) \phi^{1-\varphi} (t_{\epsilon+1} - \phi)^{\varsigma+1} d\phi
 \end{aligned} \tag{6.1}$$

Recall the Newton polynomial:

$$\begin{aligned}
 U(t, S) \simeq & U(t_{\epsilon-2}, S_{\epsilon-2}) + \frac{1}{\Delta t} [U(t_{\epsilon-1}, S_{\epsilon-1}) - U(t_{\epsilon-2}, S_{\epsilon-2})] \cdot (\phi - t_{\epsilon-2}) \\
 & + \frac{1}{2\Delta t^2} [U(t_{\epsilon}, S_{\epsilon}) - 2U(t_{\epsilon-1}, S_{\epsilon-1}) + U(t_{\epsilon-2}, S_{\epsilon-2})] \cdot (\phi - t_{\epsilon-1})
 \end{aligned} \quad (6.2)$$

Substituting Eq. (6.2) into Eq. (6.1), we obtain:

$$\begin{aligned}
 S(t_{\epsilon+1}) = & S(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} S_1(t_{m-2}, S^{m-2}) t_{m-2}^{1-\varphi} \int_{t_m}^{t_{m+1}} (t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
 & + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{\Delta t} \left[t_{m-1}^{1-\varphi} S_1(t_{m-1}, S^{m-1}) - t_{m-2}^{1-\varphi} S_1(t_{m-2}, S^{m-2}) \right] \\
 & \times \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
 & + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{2\Delta t^2} [t_m^{1-\varphi} S_1(t_m, S^m) - 2t_{m-1}^{1-\varphi} S_1(t_{m-1}, S^{m-1}) \\
 & + t_{m-2}^{1-\varphi} S_1(t_{m-2}, S^{m-2})] \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(\phi - t_{m-1})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi
 \end{aligned} \quad (6.3a)$$

$$\begin{aligned}
 L(t_{\epsilon+1}) = & L(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} L_1(t_{m-2}, L^{m-2}) t_{m-2}^{1-\varphi} \int_{t_m}^{t_{m+1}} (t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
 & + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{\Delta t} \left[t_{m-1}^{1-\varphi} L_1(t_{m-1}, L^{m-1}) - t_{m-2}^{1-\varphi} L_1(t_{m-2}, L^{m-2}) \right] \\
 & \times \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
 & + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{2\Delta t^2} [t_m^{1-\varphi} L_1(t_m, L^m) - 2t_{m-1}^{1-\varphi} L_1(t_{m-1}, L^{m-1}) \\
 & + t_{m-2}^{1-\varphi} L_1(t_{m-2}, L^{m-2})] \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(\phi - t_{m-1})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi
 \end{aligned} \quad (6.3b)$$

$$\begin{aligned}
 I(t_{\epsilon+1}) = & I(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} I_1(t_{m-2}, I^{m-2}) t_{m-2}^{1-\varphi} \int_{t_m}^{t_{m+1}} (t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
 & + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{\Delta t} \left[t_{m-1}^{1-\varphi} I_1(t_{m-1}, I^{m-1}) - t_{m-2}^{1-\varphi} I_1(t_{m-2}, I^{m-2}) \right] \\
 & \times \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi
 \end{aligned}$$

$$\begin{aligned}
& + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{2\Delta t^2} [t_m^{1-\varphi} I_1(t_m, I^m) - 2t_{m-1}^{1-\varphi} I_1(t_{m-1}, I^{m-1}) \\
& + t_{m-2}^{1-\varphi} I_1(t_{m-2}, I^{m-2})] \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(\phi - t_{m-1})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi
\end{aligned} \quad (6.3c)$$

$$\begin{aligned}
R(t_{\epsilon+1}) &= R(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} R_1(t_{m-2}, R^{m-2}) t_{m-2}^{1-\varphi} \int_{t_m}^{t_{m+1}} (t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
& + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{\Delta t} [t_{m-1}^{1-\varphi} R_1(t_{m-1}, R^{m-1}) - t_{m-2}^{1-\varphi} R_1(t_{m-2}, R^{m-2})] \\
& \times \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
& + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{2\Delta t^2} [t_m^{1-\varphi} R_1(t_m, R^m) - 2t_{m-1}^{1-\varphi} R_1(t_{m-1}, R^{m-1}) \\
& + t_{m-2}^{1-\varphi} R_1(t_{m-2}, R^{m-2})] \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(\phi - t_{m-1})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi
\end{aligned} \quad (6.3d)$$

$$\begin{aligned}
P(t_{\epsilon+1}) &= P(0) + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} P_1(t_{m-2}, P^{m-2}) t_{m-2}^{1-\varphi} \int_{t_m}^{t_{m+1}} (t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
& + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{\Delta t} [t_{m-1}^{1-\varphi} P_1(t_{m-1}, P^{m-1}) - t_{m-2}^{1-\varphi} P_1(t_{m-2}, P^{m-2})] \\
& \times \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
& + \frac{1}{\Gamma(\varsigma)} \sum_{m=2}^{\epsilon} \frac{1}{2\Delta t^2} [t_m^{1-\varphi} P_1(t_m, P^m) - 2t_{m-1}^{1-\varphi} P_1(t_{m-1}, P^{m-1}) \\
& + t_{m-2}^{1-\varphi} P_1(t_{m-2}, P^{m-2})] \int_{t_m}^{t_{m+1}} (\phi - t_{m-2})(\phi - t_{m-1})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi
\end{aligned} \quad (6.3e)$$

By calculating the integral values in the equations above, we get

$$\int_{t_m}^{t_{m+1}} (t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi = \frac{(\Delta t)^{\varsigma}}{\varsigma} [(\epsilon - m + 1)^{\varsigma} - (\epsilon - m)^{\varsigma}] \quad (6.4)$$

$$\begin{aligned}
\int_{t_m}^{t_{m+1}} (\phi - t_m - 2)(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi &= \frac{(\Delta t)^{\varsigma+1}}{\varsigma(\varsigma + 1)} \Big[\\
& (\epsilon - m + 1)^{\varsigma}(\epsilon - m + 3 + 2\varsigma) \\
& - (\epsilon - m)^{\varsigma}(\epsilon - m + 3 + 3\varsigma) \Big]
\end{aligned} \quad (6.5)$$

$$\begin{aligned}
& \int_{t_m}^{t_{m+1}} (\phi - t_m - 2)(\phi - t_{m-1})(t_{\epsilon+1} - \phi)^{\varsigma-1} d\phi \\
&= \frac{(\Delta t)^{\varsigma+2}}{\varsigma(\varsigma+1)(\varsigma+2)} [(\epsilon - m + 1)^{\varsigma}(2(\epsilon - m)^2 + (3\varsigma + 10)(\epsilon - m) \\
&\quad + 2\varsigma^2 + 9\varsigma + 12) - (\epsilon - m)^{\varsigma}(2(\epsilon - m)^2 + (5\varsigma + 10)(\epsilon - m) \\
&\quad + 6\varsigma^2 + 18\varsigma + 12)]
\end{aligned} \tag{6.6}$$

Therefore, we obtain

$$\begin{aligned}
S(t_{\epsilon+1}) &= S(0) + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+1)} \sum_{m=2}^{\epsilon} S_1(t_{m-2}, S^{m-2}) t_{m-2}^{1-\varphi} Q_1 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+2)} \sum_{m=2}^{\epsilon} (t_{m-1}^{1-\varphi} S_1(t_{m-1}, S^{m-1}) - t_{m-2}^{1-\varphi} S_1(t_{m-2}, S^{m-2})) Q_2 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+3)} \sum_{m=2}^{\epsilon} \left[t_m^{1-\varphi} S_1(t_m, S^m) - 2t_{m-1}^{1-\varphi} S_1(t_{m-1}, S^{m-1}) \right. \\
&\quad \left. + t_{m-2}^{1-\varphi} S_1(t_{m-2}, S^{m-2}) \right] Q_3
\end{aligned} \tag{6.7}$$

$$\begin{aligned}
L(t_{\epsilon+1}) &= L(0) + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+1)} \sum_{m=2}^{\epsilon} L_1(t_{m-2}, L^{m-2}) t_{m-2}^{1-\varphi} Q_1 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+2)} \sum_{m=2}^{\epsilon} (t_{m-1}^{1-\varphi} L_1(t_{m-1}, L^{m-1}) - t_{m-2}^{1-\varphi} L_1(t_{m-2}, L^{m-2})) Q_2 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+3)} \sum_{m=2}^{\epsilon} \left[t_m^{1-\varphi} L_1(t_m, L^m) - 2t_{m-1}^{1-\varphi} L_1(t_{m-1}, L^{m-1}) \right. \\
&\quad \left. + t_{m-2}^{1-\varphi} L_1(t_{m-2}, L^{m-2}) \right] Q_3
\end{aligned} \tag{6.8}$$

$$\begin{aligned}
I(t_{\epsilon+1}) &= I(0) + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+1)} \sum_{m=2}^{\epsilon} I_1(t_{m-2}, I^{m-2}) t_{m-2}^{1-\varphi} Q_1 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+2)} \sum_{m=2}^{\epsilon} (t_{m-1}^{1-\varphi} I_1(t_{m-1}, I^{m-1}) - t_{m-2}^{1-\varphi} I_1(t_{m-2}, I^{m-2})) Q_2 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+3)} \sum_{m=2}^{\epsilon} \left[t_m^{1-\varphi} I_1(t_m, I^m) - 2t_{m-1}^{1-\varphi} I_1(t_{m-1}, I^{m-1}) \right. \\
&\quad \left. + t_{m-2}^{1-\varphi} I_1(t_{m-2}, I^{m-2}) \right] Q_3
\end{aligned} \tag{6.9}$$

$$\begin{aligned}
R(t_{\epsilon+1}) &= R(0) + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+1)} \sum_{m=2}^{\epsilon} R_1(t_{m-2}, R^{m-2}) t_{m-2}^{1-\varphi} Q_1 \\
&\quad + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma+2)} \sum_{m=2}^{\epsilon} (t_{m-1}^{1-\varphi} R_1(t_{m-1}, R^{m-1}) - t_{m-2}^{1-\varphi} R_1(t_{m-2}, R^{m-2})) Q_2
\end{aligned}$$

$$+ \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma + 3)} \sum_{m=2}^{\epsilon} \left[t_m^{1-\varphi} R_1(t_m, R^m) - 2t_{m-1}^{1-\varphi} R_1(t_{m-1}, R^{m-1}) \right. \\ \left. + t_{m-2}^{1-\varphi} R_1(t_{m-2}, R^{m-2}) \right] Q_3 \quad (6.10)$$

$$P(t_{\epsilon+1}) = P(0) + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma + 1)} \sum_{m=2}^{\epsilon} P_1(t_{m-2}, P^{m-2}) t_{m-2}^{1-\varphi} Q_1 \\ + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma + 2)} \sum_{m=2}^{\epsilon} \left(t_{m-1}^{1-\varphi} P_1(t_{m-1}, P^{m-1}) - t_{m-2}^{1-\varphi} P_1(t_{m-2}, P^{m-2}) \right) Q_2 \\ + \frac{(\Delta t)^{\varsigma}}{\Gamma(\varsigma + 3)} \sum_{m=2}^{\epsilon} \left[t_m^{1-\varphi} P_1(t_m, P^m) - 2t_{m-1}^{1-\varphi} P_1(t_{m-1}, P^{m-1}) \right. \\ \left. + t_{m-2}^{1-\varphi} P_1(t_{m-2}, P^{m-2}) \right] Q_3 \quad (6.11)$$

Where,

$$Q_1 = (\epsilon - m + 1)^{\varsigma} - (\epsilon - m)^{\varsigma} \quad (6.12)$$

$$Q_2 = (\epsilon - m + 1)^{\varsigma}(\epsilon - m + 3 + 2\varsigma) \\ - (\epsilon - m)^{\varsigma}(\epsilon - m + 3 + 3\varsigma) \quad (6.13)$$

$$Q_3 = (\epsilon - m + 1)^{\varsigma} \left[2(\epsilon - m)^2 + (3\varsigma + 10)(\epsilon - m) \right. \\ \left. + 2\varsigma^2 + 9\varsigma + 12 \right] \\ - (\epsilon - m)^{\varsigma} \left[2(\epsilon - m)^2 + (5\varsigma + 10)(\epsilon - m) \right. \\ \left. + 6\varsigma^2 + 18\varsigma + 12 \right] \quad (6.14)$$

7. Numerical results and discussion

The numerical simulations are performed to investigate the influence of fractional-order dynamics on the evolution of susceptible, lysogenic, infected, resistant, and phage populations under the proposed SLIRP framework. The initial conditions are selected as follows:

$$[S(0)=0.70, \quad L(0)=0.55, \quad I(0)=0.43, \quad R(0)=0.68, \quad P(0)=0.70.]$$

The obtained results demonstrate the impact of memory effects introduced through the Caputo fractional derivative. Different values of the fractional-order parameter are considered to evaluate how the system approaches equilibrium under varying degrees of memory. In general, lower fractional orders produce slower transient dynamics due to stronger memory effects, whereas values approaching unity recover behavior similar to the classical integer-order model. The simulations also provide insight into the effectiveness of phage therapy in suppressing infected and resistant bacterial populations while maintaining overall system stability.

A graphical representation of the developed model, obtained using the proposed numerical scheme and MATLAB implementation, is illustrated in Figures 4–9. Figure 4 provides a three-dimensional

representation of the phage population as a function of time and fractional order, while Figures 5–9 illustrate the temporal evolution of the susceptible, lysogenic, infected, resistant, and phage populations, respectively.

Figure 4 presents a three-dimensional representation of the phage population $P(t)$ as a function of time and the fractional order ξ . The surface illustrates how changes in the fractional order affect the transient behavior of the phage population. Lower fractional orders produce slower transient dynamics, reflecting the stronger memory effects introduced by the fractional-order formulation.

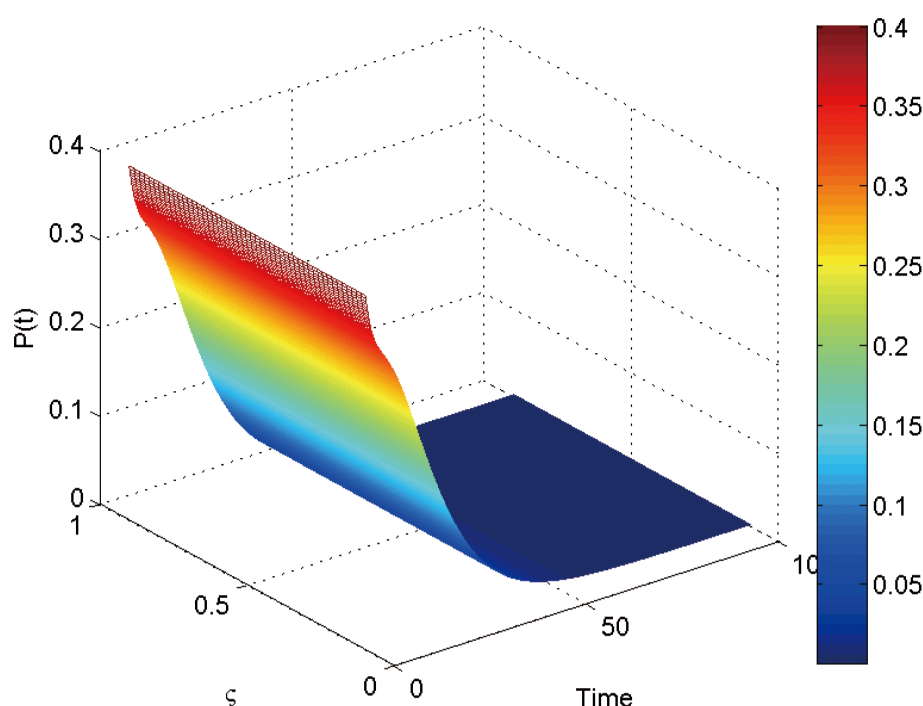


Figure 4. Three-dimensional evolution of the phage population $P(t)$ with respect to time and the fractional order ξ using the Caputo fractional-order model.

Figure 5 illustrates the dynamics of the susceptible bacterial population. The trajectories indicate that the susceptible compartment gradually approaches a stable state over time. The variation observed for different fractional orders highlights the influence of memory effects on bacterial growth. Lower fractional orders slow the convergence process, indicating that historical states continue to affect the present dynamics. Biologically, this behavior reflects the persistence of bacterial interactions within the environment.

Figure 6 presents the evolution of the lysogenic bacterial population. An overall decline in lysogenic bacteria is observed as the system progresses toward equilibrium. This behavior suggests that phage-mediated regulation reduces the persistence of dormant infections and limits the transition toward active bacterial propagation. The fractional-order parameter significantly affects the rate of decline, demonstrating the importance of memory effects in lysogenic dynamics.

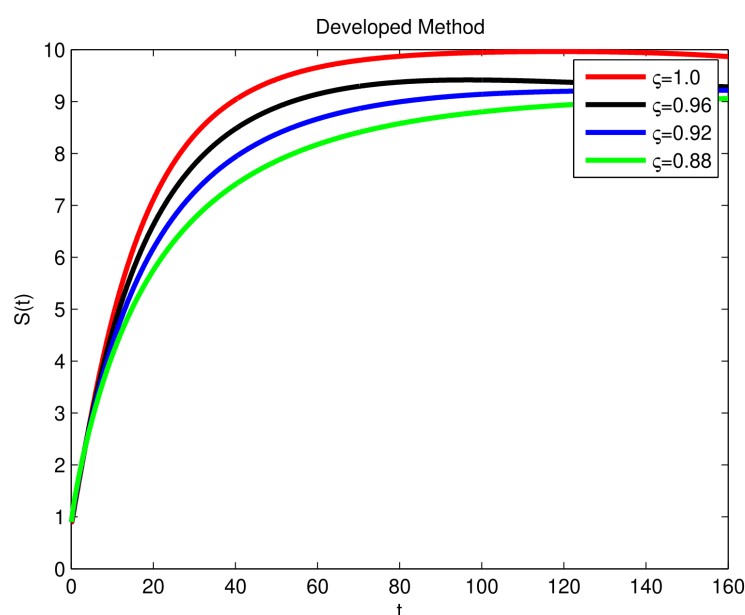


Figure 5. Evolution of the susceptible bacterial population $S(t)$ for different values of the fractional order ξ .

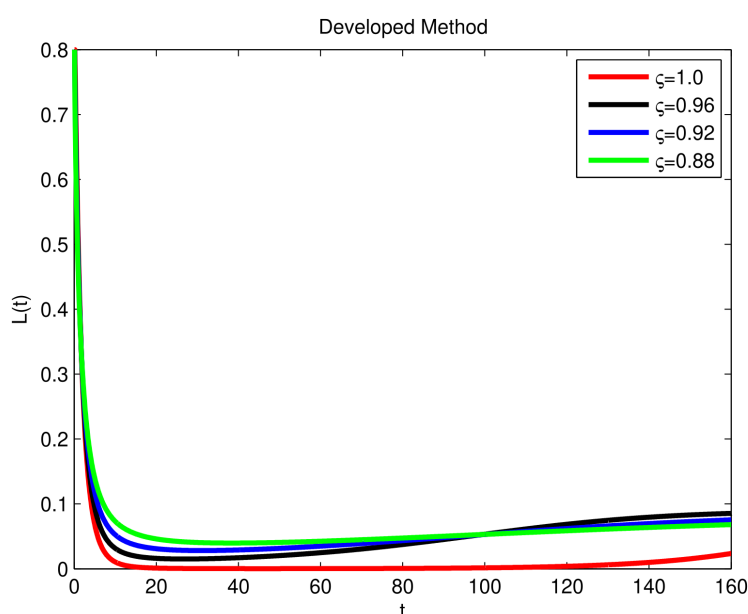


Figure 6. Evolution of the lysogenic bacterial population $L(t)$ for different values of the fractional order ξ .

Figure 7 depicts the behavior of infected bacteria. The infected compartment decreases with time and eventually approaches a stable level, indicating the effectiveness of phage activity and natural removal mechanisms. The results suggest that stronger memory effects lead to smoother transitions and reduced oscillatory behavior. From a biological perspective, the reduction in infected bacteria represents successful suppression of bacterial infection within the population.

Figure 8 shows the dynamics of resistant bacteria. The resistant compartment exhibits a decreasing trend, demonstrating that phage therapy contributes to limiting the growth of bacterial strains that are difficult to eliminate through conventional treatment. The observed behavior confirms the potential of the proposed model to capture resistance control mechanisms and evaluate their long-term impact.

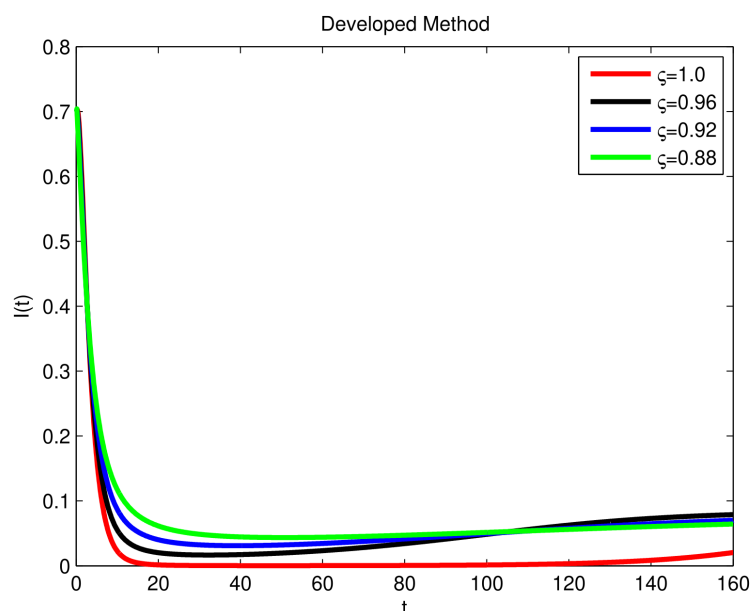


Figure 7. Evolution of the infected bacterial population $I(t)$ for different values of the fractional order ξ .

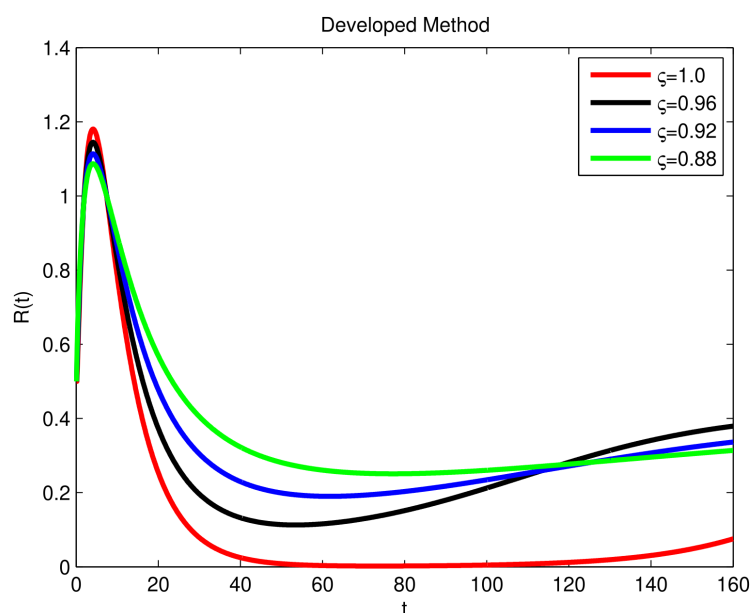


Figure 8. Evolution of the resistant bacterial population $R(t)$ for different values of the fractional order ξ .

Figure 9 illustrates the temporal evolution of the phage population. The phage concentration

initially responds to the presence of infected and resistant bacteria and subsequently approaches a stable state. This behavior confirms the regulatory role of therapeutic phages in controlling bacterial growth. The results indicate that phage therapy acts as an effective biological control mechanism capable of stabilizing the overall system dynamics.

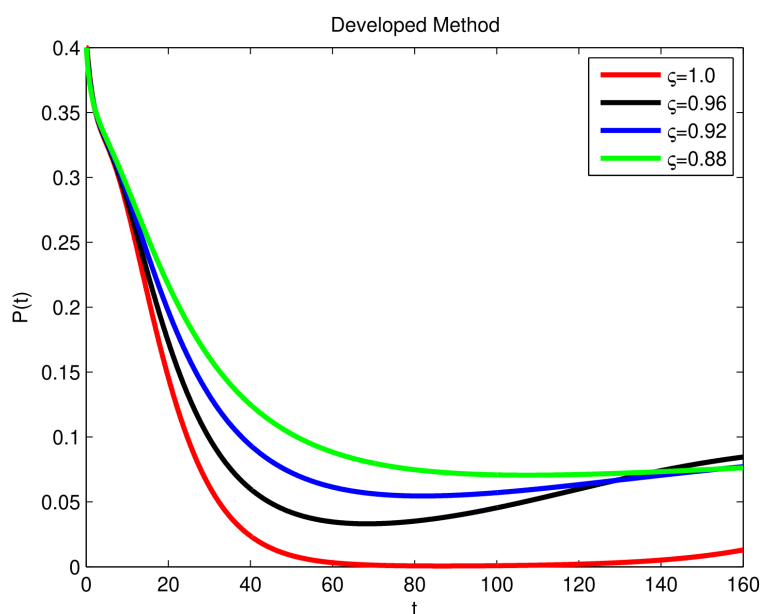


Figure 9. Evolution of the phage population $P(t)$ for different values of the fractional order ξ .

Overall, the numerical results confirm the theoretical findings obtained in the stability and existence analyses. The fractional-order formulation provides additional flexibility for describing memory-dependent bacterial processes and produces smoother dynamic behavior than the corresponding integer-order model. Furthermore, the simulations demonstrate that phage therapy plays a significant role in reducing infected and resistant bacterial populations while maintaining system's stability. These findings support the applicability of fractional-order models for understanding and controlling complex bacterial transmission dynamics.

8. Conclusions

This study investigated the dynamics of lysogenic bacterial populations under phage therapy using a fractional-order SLIRP mathematical model. The classical compartmental model was extended through the Caputo fractional derivative to incorporate memory and hereditary effects that are commonly observed in biological systems. The proposed model was analyzed mathematically with respect to its positivity, boundedness, and positively invariant regions, and the existence and uniqueness of solutions.

The equilibrium structure of the system was examined, and both local and global stability analyses were performed using Lyapunov-based techniques. In addition, chaos-control strategies were investigated to suppress irregular oscillatory behavior and improve the stability of the bacterial population dynamics. The obtained theoretical results demonstrate that the proposed control

mechanism can effectively regulate the system and reduce undesirable fluctuations associated with bacterial growth and infection progression.

Numerical simulations were conducted using the Caputo fractional operator and a two-step Lagrange polynomial approximation scheme. The use of the Caputo fractional derivative provided a mathematically tractable framework for incorporating memory effects while preserving biologically meaningful initial conditions, thereby improving the realism of the proposed bacterial population model. The computational results confirmed the theoretical findings and illustrated the influence of the model's parameters and fractional-order values on the dynamics of the susceptible, lysogenic, infected, resistant, and phage compartments. The simulations further revealed that fractional-order dynamics provide additional flexibility in describing biological processes with memory effects when compared with the classical integer-order models.

Overall, the proposed fractional-order SLIRP framework provides a useful mathematical tool for studying lysogenic bacterial dynamics and evaluating the potential impact of phage-based therapeutic interventions. Future research may consider incorporating explicit stochastic perturbations, time delays, variable-order fractional operators, and more complex phage–host interactions to further improve the realism of the model.

Author contributions

Lal Khan: conceptualization, methodology, software, validation, writing the original draft. Kokab Khan: methodology, formal analysis, investigation, software, validation, visualization, writing original draft, writing review and editing. Hsien-Tsung Chang: supervision, project administration, funding acquisition, resources, writing – review and editing. All authors have read and approved the final version of the manuscript.

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 that they have no conflict of interest.

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