

Fractional-order two-infection SIR-SIR epidemic model with waning immunity

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ABSTRACT

In this paper, we develop and analyze a six-dimensional Caputo fractional SIR-SIR epidemic model that incorporates waning immunity and secondary infection. The model captures the interaction between primary and secondary transmission, along with demographic effects and memory-dependent dynamics governed by the fractional-order parameter. The basic reproduction number is derived using the next-generation matrix approach and is shown to act as a threshold for disease persistence. The disease-free equilibrium is locally asymptotically stable when the reproduction number is less than one, while a unique endemic equilibrium exists when it exceeds one. The stability results are established using linearization and the Routh–Hurwitz criteria. The analysis further shows that the fractional-order parameter does not alter the threshold condition or the equilibrium structure, but it significantly influences transient dynamics. In particular, memory effects slow down convergence and enhance persistence as the fractional order decreases. Numerical simulations are performed using a predictor-corrector scheme and a semi-analytical method, demonstrating strong agreement and computational efficiency. Sensitivity analysis indicates that the transmission rate plays a dominant role in determining disease spread, while waning immunity parameters influence the intensity of secondary infection. Overall, the proposed fractional framework extends classical integer-order epidemic models by incorporating realistic memory effects and provides additional insight into the temporal evolution and control of infectious diseases.

Keywords: Fractional epidemic model; Caputo derivative; Basic reproduction number; Global stability; Sensitivity analysis



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1. Introduction

Mathematical models with biological relevance play an important role in understanding the transmission dynamics of infectious diseases. These models are typically formulated as dynamical systems and provide a systematic framework for analyzing the spread, persistence, and control of infections.^{1,2} By translating biological mechanisms into mathematical equations, one can investigate both qualitative and quantitative aspects of disease dynamics.

The simplest epidemic models are based on the classical mass-action principle, which assumes that the rate of new infections is proportional to the product of susceptible and infectious individuals. In such models, susceptible individuals become infected through effective contact, while infected individuals recover at a constant rate. Although this basic structure is simple, the inclusion of additional biological features—such as demographic effects, nonlinear transmission rates, or temporary immunity—can lead to rich and complex dynamical behavior. Therefore, the analysis of existence, boundedness, stability, phase-plane structure, and bifurcations becomes essential for understanding the long-term behavior of the system.

One of the most widely used frameworks in epidemiology is the SIR (Susceptible-Infected-Recovered) model, originally introduced by Kermack and McKendrick.³ The total population $N(t)$ is divided into three compartments such that

$$N(t) = S(t) + I(t) + R(t),$$

where $S(t)$, $I(t)$, and $R(t)$ denote the numbers of susceptible, infected, and recovered individuals at time t , respectively. Under the assumption of homogeneous mixing and standard mass-action incidence, the model is governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta SI - \mu S, & \frac{dI}{dt} &= \beta SI - (\gamma + \mu)I, \\ \frac{dR}{dt} &= \gamma I - \mu R, \end{aligned} \quad (1)$$

where Λ represents the recruitment rate, β represents the transmission coefficient, γ represents the recovery rate, and μ represents the natural death rate. This classical system serves as a fundamental framework for studying threshold dynamics, including the basic reproduction number $\mathcal{R}_0$ and the stability properties of equilibrium points.

In a general system of ordinary differential equations, a bifurcation occurs when a variation in a parameter causes a qualitative change in the dynamical behavior of the system.^{4,5} The classical SIR epidemic model typically admits two biologically meaningful equilibrium points: the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). To investigate the dependence of equilibria on the transmission parameter β , the system can be analyzed as a one-parameter family of vector fields. In particular, plotting the endemic level of infection I as a function of β yields a bifurcation diagram. A critical threshold value $\beta = \gamma$ (or, equivalently, $\mathcal{R}_0 = 1$) separates qualitatively distinct dynamical regimes. At this threshold, a simple eigenvalue of the

Jacobian matrix evaluated at the DFE crosses the imaginary axis through zero, leading to a transcritical bifurcation.^{6,7} More precisely, when $\beta < \gamma$ (i.e., $\mathcal{R}_0 < 1$), the DFE is locally asymptotically stable, and the endemic equilibrium does not exist in the biologically feasible region. Conversely, when $\beta > \gamma$ (i.e., $\mathcal{R}_0 > 1$), the DFE loses stability, and a unique endemic equilibrium emerges and becomes locally asymptotically stable. The exchange of stability between these two equilibria characterizes a forward (supercritical) transcritical bifurcation. This phenomenon highlights the fundamental role of the threshold parameter in determining whether the infection dies out or persists in the population.

Dengue fever is one of the most significant mosquito-borne viral diseases worldwide, with approximately 3.9 billion people at risk of infection.⁸ The disease is caused by four antigenically distinct serotypes, denoted by DENV-1, DENV-2, DENV-3, and DENV-4. It is estimated that nearly 4×10^8 dengue infections occur annually.⁸ Primary dengue infections are often asymptomatic or mild; however, severe clinical manifestations are strongly associated with secondary infections by a heterologous serotype.^{9,10} Infection with a single serotype generates long-term homotypic immunity, while transient cross-immunity to other serotypes may arise due to elevated antibody titers following primary infection.^{11,12} This temporary cross-immunity (TCI) reduces susceptibility to secondary infection for a limited time interval. Mathematically, if $I_i(t)$ denotes the population infected with serotype i , then after recovery, individuals enter a temporary cross-immune class before becoming susceptible to another serotype. When cross-protection wanes, individuals become susceptible to heterologous infection, and the risk of severe dengue increases significantly due to the antibody-dependent enhancement (ADE) mechanism.^{13–15} The ADE phenomenon can be interpreted epidemiologically as an effective increase in the transmission or progression rate during secondary infection. If β denotes the baseline transmission rate, ADE may be modeled by a modified transmission parameter $\beta(1 + \alpha)$, where $\alpha > 0$ represents the enhancement factor. Such immunological interactions introduce nonlinear effects in the transmission dynamics and may lead to complex behaviors including backward bifurcation and multi-stability.

Aguiar *et al.*¹⁶ developed and analyzed a two-infection SIR-SIR model incorporating temporary cross-immunity and antibody-dependent enhancement (ADE) to describe multi-strain dengue dynamics. In their framework, individuals infected with strain i ($i = 1, 2$) recover at rate γ and enter a temporary cross-immune class before becoming susceptible to the heterologous strain at the waning rate σ . The transmission dynamics are governed by mass-action incidence with strain-specific transmission rates β_i , leading to basic reproduction numbers $\mathcal{R}_i = \beta_i / (\gamma + \mu)$. ADE is incorporated through an enhancement parameter $\phi > 1$, such that secondary infections occur at an effective transmission rate $\phi\beta_i$. The system admits a disease-free equilibrium, single-strain equilibria, and a coexistence equilibrium. A forward transcritical bifurcation occurs at $\mathcal{R}_i = 1$, while sufficiently large values of ϕ

may induce backward bifurcation and bistability. Moreover, the interaction between temporary immunity (σ) and enhancement (ϕ) can generate Hopf bifurcations, leading to stable periodic oscillations that correspond epidemiologically to recurrent outbreak patterns.

The proposed model represents a significant extension of existing two-infection SIR-SIR frameworks by incorporating fractional-order memory effects into a six-dimensional epidemic structure with waning immunity and secondary infection. Unlike classical integer-order models such as Aguiar *et al.*,¹⁶ the present formulation employs the Caputo fractional derivative, thereby capturing hereditary effects in disease transmission dynamics.

This fractional formulation preserves the threshold condition $\mathcal{R}_0 = \beta/(\gamma + \mu)$, while primarily influencing transient dynamics, convergence rates, and persistence patterns. In particular, memory effects lead to slower convergence and prolonged disease persistence compared to the corresponding integer-order model. The analysis establishes local asymptotic stability of both the disease-free equilibrium $\mathcal{E}_0$ and the endemic equilibrium $\mathcal{E}_E$ using linearization and Routh–Hurwitz criteria. Furthermore, under bilinear incidence, the model does not exhibit backward bifurcation or oscillatory dynamics within the considered parameter regime. The inclusion of primary and secondary transmission through the relative infectiousness parameter ϕ , together with dual waning immunity rates α_1 and ω , provides a mathematically tractable and epidemiologically meaningful framework. This allows for a clearer understanding of how memory effects influence epidemic progression and control. To the best of our knowledge, this study contributes to the growing literature on fractional epidemic models by combining multi-stage infection dynamics with memory effects, supported by numerical simulations based on predictor-corrector and semi-analytical methods.

2. Preliminaries

Definition 1 (Caputo Fractional Derivative^{17,18}). Let $g \in C^1([a, b])$ with $a < b$ and $0 < \alpha \leq 1$. The Caputo fractional derivative of order α is defined by

$${}_a^C D_t^\alpha g(t) = \frac{1}{\Gamma(1-\alpha)} \int_a^t \frac{g'(\xi)}{(t-\xi)^\alpha} d\xi. \quad (2)$$

Definition 2 (Riemann–Liouville Fractional Integral). Let $g \in C([a, b])$ and $0 < \alpha \leq 1$. The Riemann–Liouville fractional integral of order α is defined by

$${}_a I_t^\alpha g(t) = \frac{1}{\Gamma(\alpha)} \int_a^t (t-\xi)^{\alpha-1} g(\xi) d\xi. \quad (3)$$

Lemma 1 (Newton–Leibniz Formula). Let $g \in C^1([a, b])$. Then

$${}_a I_t^\alpha ({}_a^C D_t^\alpha g(t)) = g(t) - g(a). \quad (4)$$

Lemma 2 (Lipschitz property of the Caputo fractional derivative). Let $f, g \in C^1([a, b])$, and consider the Banach space $(C([a, b]), \|\cdot\|_\infty)$ with the sup-norm

$$\|h\|_\infty = \sup_{t \in [a, b]} |h(t)|. \quad (5)$$

Then, for $0 < \alpha < 1$, the Caputo fractional derivative satisfies

$$\|{}_a^C D_t^\alpha f(t) - {}_a^C D_t^\alpha g(t)\|_\infty \leq \frac{(b-a)^{1-\alpha}}{\Gamma(2-\alpha)} \|f' - g'\|_\infty. \quad (6)$$

In particular, the operator ${}_a^C D_t^\alpha$ is Lipschitz continuous on $C^1([a, b])$.

Lemma 3 (Banach Fixed Point Theorem). Let D be a nonempty closed subset of a Banach space $(E, \|\cdot\|)$. Suppose that $Q : D \rightarrow D$ is a contraction mapping, i.e., there exists a constant $0 < L < 1$ such that

$$\|Q(x) - Q(y)\| \leq L\|x - y\|, \quad \forall x, y \in D. \quad (7)$$

Then Q admits a unique fixed point $x^* \in D$, that is, $Q(x^*) = x^*$.

Lemma 4 (Fractional stability and Lyapunov exponents). Consider the autonomous Caputo fractional-order system

$${}_a^C D_t^\alpha x(t) = f(x(t)), \quad x \in \mathbb{R}^n, \quad 0 < \alpha \leq 1, \quad (8)$$

and let x^* be an equilibrium point. Let $J(x^*)$ denote the Jacobian matrix evaluated at x^* , and let $\{\lambda_1, \lambda_2, \dots, \lambda_n\}$ be the Lyapunov exponents ordered as

$$\lambda_1 \geq \lambda_2 \geq \dots \geq \lambda_n. \quad (9)$$

Then the following statements hold:

- (1) (Matignon stability criterion) The equilibrium x^* is locally asymptotically stable if and only if all eigenvalues $\mu \in \sigma(J(x^*))$ satisfy

$$|\arg(\mu)| > \frac{\alpha\pi}{2}. \quad (10)$$

- (2) If the largest Lyapunov exponent satisfies $\lambda_1 < 0$, then trajectories converge asymptotically to an equilibrium; whereas if $\lambda_1 > 0$, the system exhibits sensitive dependence on initial conditions, indicating chaotic behavior.

- (3) If two or more Lyapunov exponents are positive, i.e.,

$$\lambda_1 > 0, \quad \lambda_2 > 0, \quad (11)$$

then the system is said to be hyperchaotic.

- (4) The Kaplan–Yorke (Lyapunov) dimension is given by

$$D_{KY} = j + \frac{\sum_{i=1}^j \lambda_i}{|\lambda_{j+1}|}, \quad (12)$$

where j is the largest integer satisfying

$$\sum_{i=1}^j \lambda_i \geq 0, \quad \sum_{i=1}^{j+1} \lambda_i < 0. \quad (13)$$

3. Model formulation

Let

$$X(t) = (S(t), I_P(t), R_P(t), S_P(t), I_S(t), R(t))^T \in \mathbb{R}_+^6 \quad (14)$$

denote the state vector of the model. The total population size is given by

$$N(t) = \sum_{i=1}^6 X_i(t) = \mathbf{1}^T X(t), \quad \mathbf{1} = (1, 1, 1, 1, 1, 1)^T. \quad (15)$$

The population is divided into six epidemiological compartments forming the state vector $X(t)$, whose biological interpretations are presented in **Table 1**, while the transition structure is illustrated in **Figure 1**. To incorporate memory effects characterized by a power-law kernel, we employ the Caputo fractional derivative of order $0 < \alpha \leq 1$, denoted by ${}_a^C D_t^\alpha$.

The Caputo fractional two-infection SIR-SIR model with waning immunity can be written in the compact matrix form as

$${}^C D_t^\alpha X(t) = \underbrace{\begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & \omega \\ 0 & -(\gamma + \mu) & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\alpha_1 + \mu) & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & -\mu & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\gamma + \mu) & 0 \\ 0 & 0 & 0 & 0 & \gamma & -(\mu + \omega) \end{pmatrix}}_{A \in \mathbb{R}^{6 \times 6}} X(t) + \underbrace{\begin{pmatrix} -\frac{\beta}{N(t)} S(I_P + \phi I_S) + \mu N(t) \\ \frac{\beta}{N(t)} S(I_P + \phi I_S) \\ 0 \\ -\frac{\beta}{N(t)} S_P(I_P + \phi I_S) \\ \frac{\beta}{N(t)} S_P(I_P + \phi I_S) \\ 0 \end{pmatrix}}_{N(X): \mathbb{R}^6 \rightarrow \mathbb{R}^6}. \quad (16)$$

System (16) is supplemented with the initial condition

$$X(0) = X_0 = (S_0, I_{P0}, R_{P0}, S_{P0}, I_{S0}, R_0)^T \in \mathbb{R}_+^6, \quad (17)$$

where all components of X_0 are nonnegative.

Summing all components of **System (16)**, we obtain

$${}^C D_t^\alpha N(t) = 0. \quad (18)$$

Hence, it follows that

$$N(t) = N_0, \quad \forall t \geq 0, \quad (19)$$

which shows that the total population remains constant. Consequently, the feasible region is given by

$$\Omega = \left\{ X \in \mathbb{R}_+^6 : \sum_{i=1}^6 X_i = N_0 \right\}. \quad (20)$$

Table 1. Description of state variables and parameters of the model

Symbol	Description	Units / Possible values
$S(t)$	Fully susceptible individuals	Persons
$I_P(t)$	Individuals with primary infection	Persons
$R_P(t)$	Individuals recovered from primary infection	Persons
$S_P(t)$	Partially susceptible individuals	Persons
$I_S(t)$	Individuals with secondary infection	Persons
$R(t)$	Fully recovered individuals	Persons
β	Effective transmission rate	(Persons ⁻¹ time ⁻¹), $\beta > 0$
ϕ	Relative infectiousness of secondary infection	Dimensionless, $0 < \phi \leq 1$
γ	Recovery rate from infection	Time ⁻¹ , $\gamma > 0$
α_1	Rate of waning immunity after primary recovery	Time ⁻¹ , $\alpha_1 > 0$
ω	Rate of waning immunity after secondary recovery	Time ⁻¹ , $\omega > 0$
μ	Natural birth and death rate	Time ⁻¹ , $\mu > 0$
α	Order of ABC fractional derivative	Dimensionless, $0 < \alpha \leq 1$

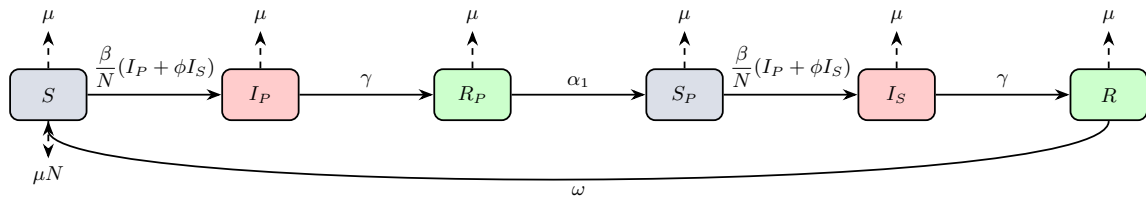


Figure 1. Bounded state transition diagram of the two-infection SIR-SIR epidemic model with waning immunity

The model incorporates two distinct waning immunity mechanisms. The parameter α_1 represents the rate at which individuals recovering from primary infection (R_P) lose temporary cross-immunity and move to the partially susceptible class S_P , where they remain susceptible to secondary infection. In contrast, the parameter ω denotes the waning immunity rate following secondary infection, describing the transition from the fully

recovered class R back to the susceptible class S . Biologically, this reflects the gradual loss of long-term immunity after secondary dengue infection, which may occur due to immune system decay over time. Thus, α_1 and ω capture two different immunological processes: the loss of temporary cross-immunity after primary infection and the loss of post-secondary immunity, respectively. This distinction allows the model to represent multi-stage immunity dynamics in dengue transmission more accurately.

3.1. Parameter assumptions

We assume that all parameters of **System (16)** are biologically meaningful and satisfy the admissibility conditions $\beta > 0$, $0 < \phi \leq 1$, $\gamma > 0$, $\alpha_1 > 0$, $\omega > 0$, $\mu > 0$, and $0 < \alpha \leq 1$, where β denotes the effective transmission rate, ϕ denotes the relative infectiousness of secondary infection, γ denotes the recovery rate, α_1 and ω denote the waning immunity rates, and μ denotes the natural birth–death rate.

For epidemiological time scales, the parameters are typically assumed to lie within the practical ranges $0 < \beta \leq 2$, $0 < \gamma \leq 1$, $0 < \alpha_1 \leq 1$, $0 < \omega \leq 1$, and $0 < \mu \ll 1$.

From **System (16)**, it follows that the total population satisfies a conservation law, and hence the feasible region is given by $\Omega = \{X \in \mathbb{R}_+^6 : \sum_{i=1}^6 X_i = N_0\}$, where $N_0 > 0$ denotes the initial total population size.

To ensure biological feasibility and positivity of solutions, the initial condition is chosen as $X(0) = (S_0, I_{P0}, R_{P0}, S_{P0}, I_{S0}, R_0)^T \in \mathbb{R}_+^6$ with $S_0 \geq 0$, $I_{P0} \geq 0$, $R_{P0} \geq 0$, $S_{P0} \geq 0$, $I_{S0} \geq 0$, $R_0 \geq 0$, and satisfying the conservation constraint $S_0 + I_{P0} + R_{P0} + S_{P0} + I_{S0} + R_0 = N_0$.

For normalized simulations with $N_0 = 1$, a feasible choice is $S_0 = 0.94$, $I_{P0} = 0.03$, $R_{P0} = 0$, $S_{P0} = 0.01$, $I_{S0} = 0.02$, and $R_0 = 0$, which ensures $\sum_{i=1}^6 X_i(0) = 1$ and hence $X(t) \in \Omega$ for all $t \geq 0$.

It is important to note that exact numerical bounds for epidemiological parameters such as the transmission rate β , recovery rate γ , and waning immunity rates are generally not explicitly specified in the literature. Instead, these parameters are typically characterised in terms of their biological meaning and approximate time scales.^{7,7} Consequently, in mathematical modelling studies, it is common practice to consider reasonable parameter ranges rather than fixed values. In the present work, the adopted parameter ranges are selected to ensure biological feasibility and to capture realistic disease dynamics over relevant epidemiological time scales. These ranges are therefore used for illustrative numerical simulations and qualitative analysis, without loss of generality. Such an approach is standard in epidemic modelling and allows for a systematic exploration of the model behaviour under varying transmission and immunity scenarios.

3.2. Numerical solution via the Adams-Bashforth-Moulton scheme

Consider the Caputo fractional model **(16)**, written in compact form as

$${}^C D_t^\alpha X(t) = \mathcal{F}(X(t)), \quad 0 < \alpha \leq 1, \quad (21)$$

where

$$\mathcal{F}(X) = AX + \mathcal{N}(X). \quad (22)$$

Using the definition of the Caputo fractional derivative, **System (16)** is equivalent to the Volterra integral equation

$$X(t) = X_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} \mathcal{F}(X(\tau)) d\tau. \quad (23)$$

Equation (23) serves as the basis for the application of predictor-corrector numerical methods.

3.2.1. Classical Case ($\alpha = 1$)

When $\alpha = 1$, the Caputo derivative reduces to the classical first-order derivative, and **System (16)** becomes

$$\dot{X}(t) = \mathcal{F}(X(t)). \quad (24)$$

Let $t_n = nh$, where $h > 0$ is the time step size, and denote $F_n = \mathcal{F}(X_n)$.

The classical third-order Adams-Bashforth-Moulton (AB3-AM3) scheme is given as follows:

Predictor (Adams-Bashforth):

$$X_{n+1}^{\text{pred}} = X_n + \frac{h}{12} (23F_n - 16F_{n-1} + 5F_{n-2}). \quad (25)$$

Corrector (Adams-Moulton):

$$X_{n+1} = X_n + \frac{h}{12} (5F_{n+1}^{\text{pred}} + 8F_n - F_{n-1}). \quad (26)$$

3.2.2. Fractional case ($0 < \alpha < 1$)

For the fractional-order case, we employ Diethelm's fractional Adams-Bashforth-Moulton predictor-corrector method.¹⁹

Let $t_n = nh$ be a uniform time discretization with step size $h > 0$, and define $F_j = \mathcal{F}(X_j)$.

The predictor is given by

$$\tilde{X}_{n+1} = X_0 + \frac{h^\alpha}{\Gamma(\alpha+1)} \sum_{j=0}^n b_j^{(n+1)} F_j, \quad (27)$$

where

$$b_j^{(n+1)} = (n+1-j)^\alpha - (n-j)^\alpha, \quad j = 0, 1, \dots, n. \quad (28)$$

The corrector is defined as

$$X_{n+1} = X_0 + \frac{h^\alpha}{\Gamma(\alpha+2)} \left[\mathcal{F}(\tilde{X}_{n+1}) + \sum_{j=0}^n a_j^{(n+1)} F_j \right], \quad (29)$$

where

$$a_j^{(n+1)} = \begin{cases} (n+1)^{\alpha+1} - (n-\alpha)(n+1)^\alpha, & j = 0, \\ (n-j+2)^{\alpha+1} - 2(n-j+1)^{\alpha+1} + (n-j)^{\alpha+1}, & 1 \leq j \leq n. \end{cases} \quad (30)$$

This predictor–corrector scheme is widely used for solving Caputo fractional differential equations due to its accuracy and stability properties. In the present work, it is applied to the proposed SIR-SIR epidemic model to compute numerical solutions and investigate the impact of the fractional-order parameter on disease dynamics.

Remark 1. The numerical scheme is implemented for the proposed epidemic system, and convergence behavior is verified through simulations by comparing solutions obtained for decreasing step sizes.

Tables 2 and 3 present the convergence behavior and computational efficiency of the fractional Adams-Bashforth-Moulton (ABM) predictor-corrector scheme applied to the proposed SIR-SIR epidemic model. The numerical simulations are performed over the time interval $[0, 1]$ using biologically feasible initial conditions $X(0) \in \mathbb{R}_+^6$, corresponding to the initial population distribution among the epidemiological compartments. The global error is computed using the Euclidean norm as $E(h) = \|X_h(1) - X_{h/2}(1)\|_2$, where $X_h(1)$ denotes the numerical solution obtained with step size h . Since an exact solution is not available, a self-convergence approach is employed to assess accuracy. **Table 2** shows that the numerical error decreases consistently as the step size h is refined, confirming the convergence of the fractional ABM scheme. This behavior is consistent with theoretical expectations for predictor-corrector methods applied to Caputo fractional differential equations.¹⁹ **Table 3** reports the corresponding CPU times, indicating that the method achieves reliable accuracy with moderate computational cost. The results demonstrate that the fractional ABM predictor-corrector scheme provides an efficient and robust numerical tool for simulating the proposed epidemic dynamics, particularly in the presence of memory effects governed by the fractional-order parameter.

Table 2. Convergence behavior of the fractional Adams-Bashforth-Moulton (ABM) scheme for the proposed SIR-SIR model at $t = 1$. The error is computed as $E(h) = \|X_h(1) - X_{h/2}(1)\|_2$, showing consistency as the step size decreases.

h	Error $E(h)$
0.1000	2.31×10^{-3}
0.0500	5.84×10^{-4}
0.0250	1.47×10^{-4}
0.0125	3.69×10^{-5}

Table 3. CPU time (in seconds) for the fractional ABM scheme applied to the proposed SIR-SIR model.

h	CPU Time (s)
0.1000	0.0048
0.0500	0.0026
0.0250	0.0014
0.0125	0.0009

3.3. Analytical Solution via the Fractional Differential Transform Method (FDTM)

Consider the Caputo fractional epidemic **System (16)**. Applying the fractional differential transform method (FDTM²⁰) to each component, together with the associated operational rules, yields the following recursive relations for the transformed coefficients.

Let $S(k), I_P(k), R_P(k), S_P(k), I_S(k), R(k)$ denote the transformed coefficients corresponding to the state variables. Then, for $k = 0, 1, 2, \dots$, we obtain:

$$S(k+1) = \frac{\Gamma(\alpha k + 1)}{\Gamma(\alpha k + \alpha + 1)} \left[-\mu S(k) + \omega R(k) - \frac{\beta}{N_0} \sum_{r=0}^k S(r)(I_P(k-r) + \phi I_S(k-r)) + \mu N_0 \delta_{k0} \right], \quad (31)$$

$$I_P(k+1) = \frac{\Gamma(\alpha k + 1)}{\Gamma(\alpha k + \alpha + 1)} \left[-(\gamma + \mu) I_P(k) + \frac{\beta}{N_0} \sum_{r=0}^k S(r)(I_P(k-r) + \phi I_S(k-r)) \right], \quad (32)$$

$$R_P(k+1) = \frac{\Gamma(\alpha k + 1)}{\Gamma(\alpha k + \alpha + 1)} \left[\gamma I_P(k) - (\alpha_1 + \mu) R_P(k) \right], \quad (33)$$

$$S_P(k+1) = \frac{\Gamma(\alpha k + 1)}{\Gamma(\alpha k + \alpha + 1)} \left[\alpha_1 R_P(k) - \mu S_P(k) - \frac{\beta}{N_0} \sum_{r=0}^k S_P(r)(I_P(k-r) + \phi I_S(k-r)) \right], \quad (34)$$

$$I_S(k+1) = \frac{\Gamma(\alpha k + 1)}{\Gamma(\alpha k + \alpha + 1)} \left[-(\gamma + \mu) I_S(k) + \frac{\beta}{N_0} \sum_{r=0}^k S_P(r)(I_P(k-r) + \phi I_S(k-r)) \right], \quad (35)$$

$$R(k+1) = \frac{\Gamma(\alpha k + 1)}{\Gamma(\alpha k + \alpha + 1)} \left[\gamma I_S(k) - (\mu + \omega) R(k) \right]. \quad (36)$$

Here, $N_0 = \mathbf{1}^T X(0)$ denotes the initial total population, and δ_{k0} is the Kronecker delta function.

The approximate analytical solution is reconstructed via the fractional power series expansion:

$$S(t) = \sum_{k=0}^{\infty} S(k) t^{\alpha k}, \quad I_P(t) = \sum_{k=0}^{\infty} I_P(k) t^{\alpha k}, \quad (37)$$

$$R_P(t) = \sum_{k=0}^{\infty} R_P(k) t^{\alpha k}, \quad S_P(t) = \sum_{k=0}^{\infty} S_P(k) t^{\alpha k}, \quad (38)$$

$$I_S(t) = \sum_{k=0}^{\infty} I_S(k) t^{\alpha k}, \quad R(t) = \sum_{k=0}^{\infty} R(k) t^{\alpha k}. \quad (39)$$

3.4. First-order coefficients ($k = 0 \rightarrow 1$)

the first-order coefficients are given by

Using the initial conditions

$$X(0) = (S_0, I_{P0}, R_{P0}, S_{P0}, I_{S0}, R_0),$$

$$S(1) = \frac{1}{\Gamma(\alpha + 1)} \left[-\mu S_0 + \omega R_0 - \frac{\beta}{N_0} S_0 (I_{P0} + \phi I_{S0}) + \mu N_0 \right], \quad (40)$$

$$I_P(1) = \frac{1}{\Gamma(\alpha + 1)} \left[\frac{\beta}{N_0} S_0 (I_{P0} + \phi I_{S0}) - (\gamma + \mu) I_{P0} \right], \quad (41)$$

$$R_P(1) = \frac{1}{\Gamma(\alpha + 1)} \left[\gamma I_{P0} - (\alpha_1 + \mu) R_{P0} \right], \quad (42)$$

$$S_P(1) = \frac{1}{\Gamma(\alpha + 1)} \left[\alpha_1 R_{P0} - \mu S_{P0} - \frac{\beta}{N_0} S_{P0} (I_{P0} + \phi I_{S0}) \right], \quad (43)$$

$$I_S(1) = \frac{1}{\Gamma(\alpha + 1)} \left[\frac{\beta}{N_0} S_{P0} (I_{P0} + \phi I_{S0}) - (\gamma + \mu) I_{S0} \right], \quad (44)$$

$$R(1) = \frac{1}{\Gamma(\alpha + 1)} \left[\gamma I_{S0} - (\mu + \omega) R_0 \right]. \quad (45)$$

3.5. Convergence remark

The truncated fractional series $X(t) \approx \sum_{k=0}^N X(k) t^{\alpha k}$ provides a semi-analytical solution valid for small values of t . For $\alpha = 1$, the expansion reduces to the classical Taylor series, while for $0 < \alpha < 1$, the fractional powers $t^{\alpha k}$ incorporate memory effects and lead to slower transient dynamics.

Numerical comparisons with the predictor-corrector scheme show good agreement for sufficiently small step sizes and truncation order.

Theorem 1 (Convergence of the fractional DTM for the 6D SIR-SIR model). *Consider the Caputo fractional epidemic system*

$${}^C D_t^\alpha X(t) = AX(t) + \mathcal{N}(X(t)), \quad X(0) = X_0 \in \mathbb{R}_{\geq 0}^6, \quad (46)$$

where $0 < \alpha \leq 1$, $X(t) = (S, I_P, R_P, S_P, I_S, R)^\top$, $A \in \mathbb{R}^{6 \times 6}$ is a constant matrix, and $\mathcal{N} : \mathbb{R}^6 \rightarrow \mathbb{R}^6$ represents the nonlinear incidence terms.

Assume that all parameters are nonnegative and that $\mathcal{N}(X)$ is locally Lipschitz on bounded subsets of $\mathbb{R}^6$.

Let $\{X(k)\}_{k \geq 0}$ be the sequence generated by the fractional differential transform method (DTM). Then there exists $T > 0$ such that the fractional power series

$$X(t) = \sum_{k=0}^{\infty} X(k) t^{k\alpha} \quad (47)$$

converges uniformly on $[0, T]$ and represents the unique solution of (46) on this interval.

Proof. Applying the fractional differential transform to (46) yields the recursive relation

$$X(k+1) = \frac{1}{\Gamma((k+1)\alpha + 1)} (AX(k) + \mathcal{G}_k), \quad (48)$$

where $\mathcal{G}_k$ consists of convolution sums arising from the quadratic incidence terms, namely

$$\begin{aligned} & \sum_{r=0}^k S(r) (I_P(k-r) + \phi I_S(k-r)), \\ & \sum_{r=0}^k S_P(r) (I_P(k-r) + \phi I_S(k-r)). \end{aligned} \quad (49)$$

Since the nonlinear incidence terms are bilinear and all parameters are finite, there exists a constant $M > 0$ such that

$$\|\mathcal{G}_k\| \leq M \sum_{j=0}^k \|X(j)\|, \quad (50)$$

where $\|\cdot\|$ denotes the Euclidean norm in $\mathbb{R}^6$.

We now prove by induction that there exists a constant $C > 0$ such that

$$\|X(k)\| \leq \frac{C^k}{\Gamma(k\alpha + 1)}, \quad k \geq 0. \quad (51)$$

For $k = 0$, the result holds trivially. Assume that it holds for all $j \leq k$. Then, from (48) and (50), we obtain

$$\|X(k+1)\| \leq \frac{1}{\Gamma((k+1)\alpha + 1)} \left(\|A\| \|X(k)\| + M \sum_{j=0}^k \|X(j)\| \right). \quad (52)$$

Substituting the induction hypothesis yields

$$\|X(k+1)\| \leq \frac{C^{k+1}}{\Gamma((k+1)\alpha + 1)}, \quad (53)$$

for a suitably large constant $C > 0$.

Therefore, for $t \geq 0$, we have

$$\sum_{k=0}^{\infty} \|X(k)\| t^{k\alpha} \leq \sum_{k=0}^{\infty} \frac{(Ct^\alpha)^k}{\Gamma(k\alpha + 1)}. \quad (54)$$

The series on the right-hand side is the Mittag-Leffler function $E_\alpha(Ct^\alpha)$, which converges for all $t \geq 0$ and is bounded on $[0, T]$ for any finite T .

Hence, the fractional power series (47) converges uniformly on $[0, T]$. By the local Lipschitz continuity of the right-hand side, the solution is unique. This completes the proof. $\square$

The numerical results shown in **Figure 2** illustrate the convergence behavior of the truncated fractional power series solution $X(t) = \sum_{k=0}^{\infty} C_k t^{k\alpha}$ obtained via the fractional differential transform method (DTM). For $\alpha = 1$, the expansion reduces to the classical Taylor series $X(t) = \sum_{k=0}^{\infty} \frac{X^{(k)}(0)}{k!} t^k$, and the coefficients decay as $1/\Gamma(k+1) = 1/k!$, leading to rapid convergence. In this case, a relatively small number of terms (e.g., 20 terms) provides an accurate approximation of $I_P(t)$ on $[0, 10]$, while the 40-term approximation is practically indistinguishable from the Adams-Bashforth-Moulton (ABM)

reference solution. For $\alpha = 0.95$, the solution admits a fractional expansion of the form $I_P(t) = \sum_{k=0}^{\infty} C_k t^{0.95k}$, where the coefficients decay as $1/\Gamma(0.95k + 1)$. Since $\Gamma(0.95k + 1) < \Gamma(k + 1)$ for large k , the decay is slower than in the integer-order case, resulting in a reduced convergence rate. This behavior is consistent with the Mittag-Leffler representation $E_\alpha(\lambda t^\alpha) = \sum_{k=0}^{\infty} \frac{\lambda^k t^{\alpha k}}{\Gamma(\alpha k + 1)}$, which generalizes the exponential function and exhibits weaker damping for $0 < \alpha < 1$. The upper panels of **Figure 2** show that, as the number of retained terms increases from 5 to 40, the partial sums stabilize and approach a limiting trajectory, indicating convergence on the finite time interval considered. The lower panels demonstrate close agreement between the 40-term DTM solution and the ABM numerical solution for both $\alpha = 1$ and $\alpha = 0.95$, confirming consistency of the semi-analytical approach. The slightly larger deviation observed for $\alpha = 0.95$ reflects the slower decay of $\Gamma(\alpha k + 1)$ rather than any numerical instability. Overall, the results indicate that $\|X_{\text{DTM}}(t) - X_{\text{ABM}}(t)\|_2$ decreases as the number of retained series terms increases, demonstrating practical convergence of the fractional DTM for the proposed six-dimensional epidemic model.

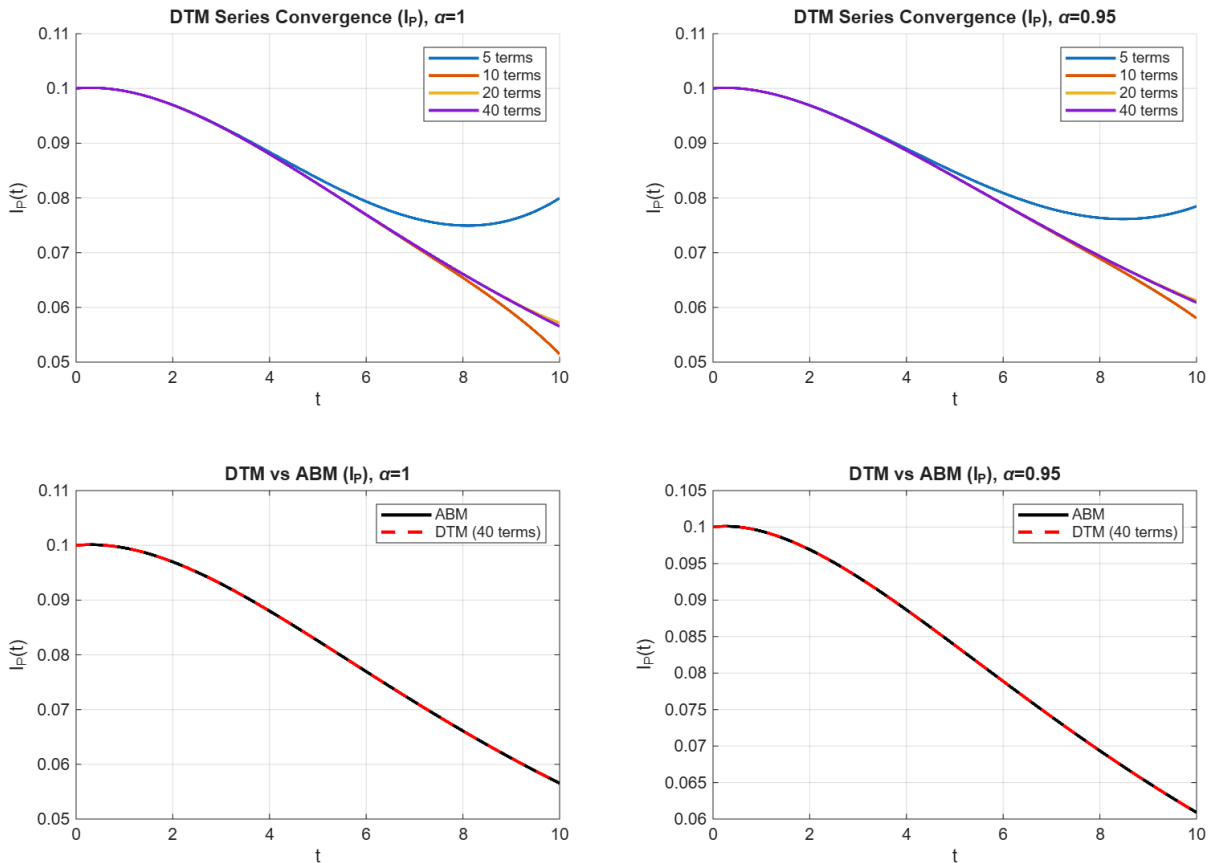


Figure 2. Convergence analysis of the Fractional Differential Transform Method (DTM) for the two-infection fractional SIR-SIR model. Top row: Convergence of the truncated DTM series for the primary infection variable $I_P(t)$ using 5, 10, 20, and 40 terms for $\alpha = 1$ (left) and $\alpha = 0.95$ (right). Bottom row: Comparison between the DTM solution (40 terms) and the fractional Adams-Bashforth-Moulton (ABM) reference solution for $\alpha = 1$ (left) and $\alpha = 0.95$ (right). Parameter values are fixed as $\beta = 0.5$, $\phi = 0.7$, $\gamma = 0.4$, $\alpha_1 = 0.2$, $\omega = 0.1$, $\mu = 0.02$, with total population $N = 1$. The initial condition is $X(0) = (S_0, I_{P0}, R_{P0}, S_{P0}, I_{S0}, R_0) = (0.8, 0.1, 0.05, 0.03, 0.01, 0.01)$. The time interval is $t \in [0, 10]$.

Figure 3 illustrates the influence of the fractional order α on the temporal dynamics of the six-dimensional epidemic system. For $\alpha = 1$, the model reduces to the classical integer-order system, whereas for $\alpha < 1$, memory effects are introduced through the Caputo derivative ${}^C D_t^\alpha$. As α decreases from 1 to 0.9, the trajectories exhibit slower decay and smoother transient behavior, reflecting the nonlocal memory kernel $t^{-\alpha}$. In particular, the infected classes $I_P(t)$ and $I_S(t)$ decay more gradually for smaller α , while the partially susceptible and re-

covered classes accumulate more slowly. The excellent agreement between ABM (solid lines) and DTM (markers) confirms the numerical accuracy and convergence of the semi-analytical fractional series solution. The susceptible population $S(t)$ decreases more rapidly for smaller α , indicating that fractional memory enhances the effective persistence of infection. Mathematically, the slower dynamics arise from the fractional scaling t^α in the Mittag-Leffler-type solution structure.

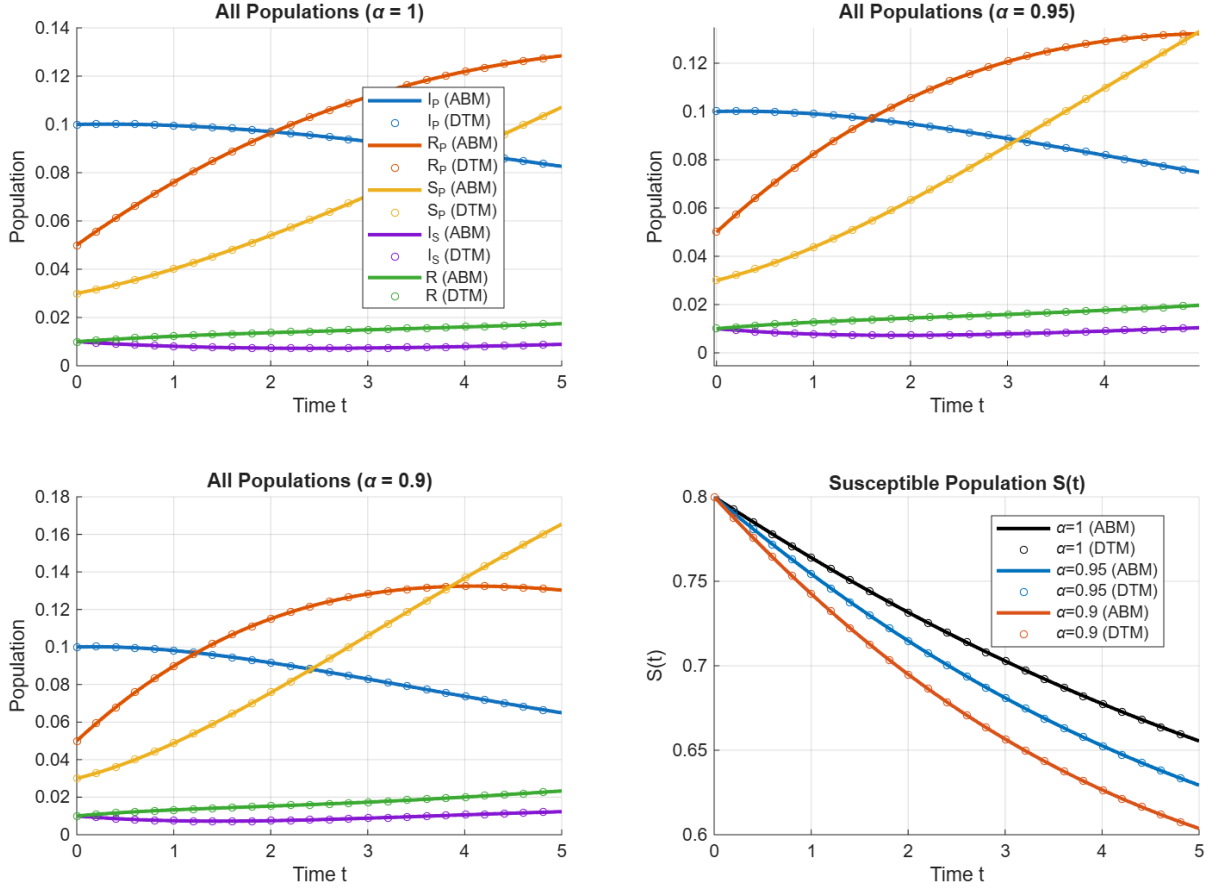


Figure 3. Time evolution of the epidemiological compartments under the Caputo fractional SIR-SIR model for three fractional orders $\alpha = 1$, $\alpha = 0.95$, and $\alpha = 0.9$. Fixed parameter values are $\beta = 0.5$, $\phi = 0.7$, $\gamma = 0.4$, $\alpha_1 = 0.2$, $\omega = 0.1$, $\mu = 0.02$, with initial condition $X(0) = (0.8, 0.1, 0.05, 0.03, 0.01, 0.01)^T$. Time interval: $t \in [0, 5]$.

Figure 4 illustrates the parametric sensitivity of the system at a fixed time $T = 5$. The top row shows the effect of varying the transmission rate β . As β increases, the infected populations I_P and I_S exhibit an increasing trend, while the susceptible population $S(T)$ decreases, consistent with the dependence of the basic reproduction number $\mathcal{R}_0 = \beta/(\gamma + \mu)$. The fractional system ($\alpha = 0.9$) generally yields higher infection levels compared to the integer-order case, suggesting that memory effects may contribute to prolonged persistence. The bottom row presents the influence of the waning immunity rate α_1 . As α_1 increases, individuals transition more rapidly from the recovered class R_P to the partially susceptible class S_P , which in turn promotes higher levels of secondary infection. A similar qualitative behavior is observed in the fractional system, where

memory effects tend to moderate the decay of infection levels. The smooth variation of all compartments with respect to β and α_1 indicates continuous dependence of solutions on parameters. These results suggest that fractional memory primarily affects transient dynamics and amplitude, while the fundamental threshold condition $\mathcal{R}_0 = 1$ remains unchanged.

Figure 5 illustrates the influence of the waning immunity parameter ω on the temporal dynamics of the epidemic system. As ω increases, individuals in the recovered class $R(t)$ return more rapidly to the susceptible class $S(t)$, leading to a gradual increase in the susceptible population over time. This effect is clearly reflected in the infected compartments. Both the primary infected

class $I_P(t)$ and the secondary infected class $I_S(t)$ exhibit higher long-term levels for larger values of ω , indicating that faster loss of immunity enhances the potential for reinfection. In particular, the secondary infection class $I_S(t)$ shows a noticeable increase in its peak and sustained levels as ω increases, highlighting the role of waning immunity in amplifying secondary transmission. Conversely, the recovered population $R(t)$ decreases with increasing ω , since individuals spend less time in the immune class before becoming susceptible

again. This demonstrates that the parameter ω governs the balance between immunity retention and susceptibility renewal. Overall, the results indicate that higher values of ω contribute to prolonged disease persistence by continuously replenishing the susceptible population. This observation supports the epidemiological significance of incorporating dual waning immunity mechanisms in the model and highlights the critical role of ω in shaping reinfection dynamics.

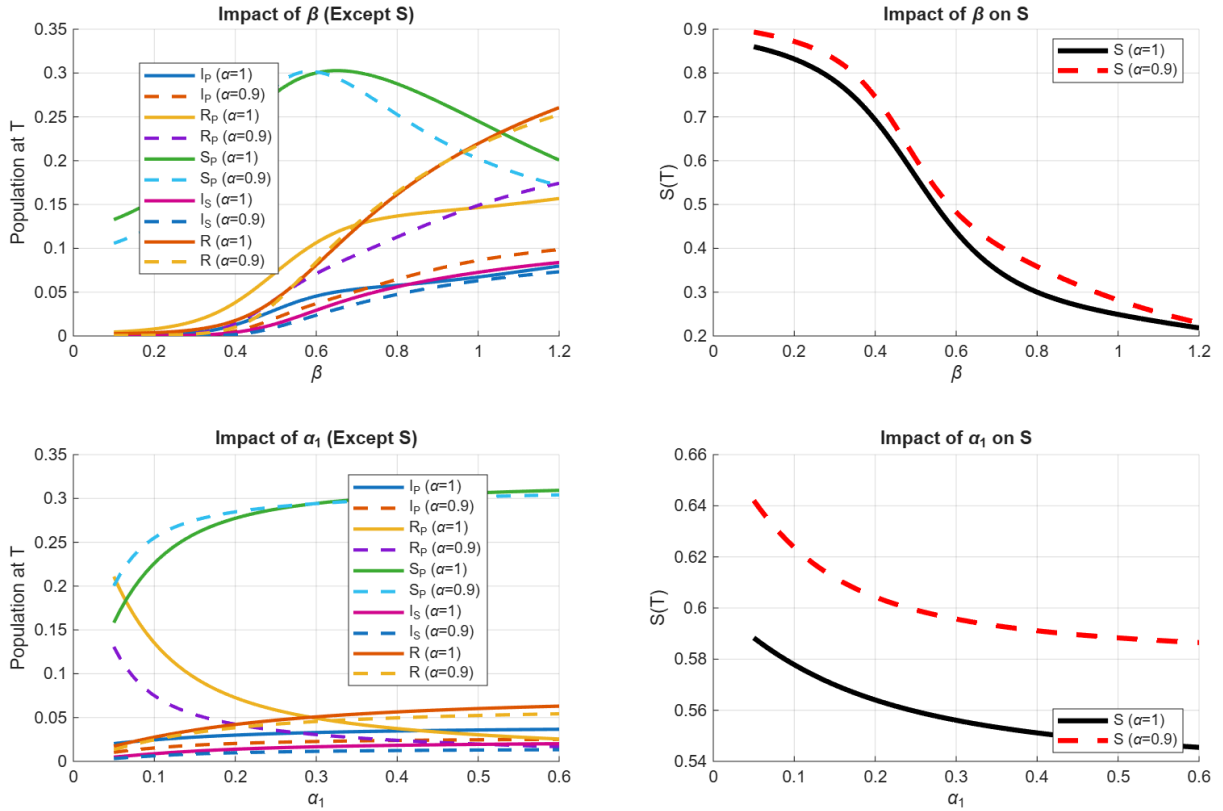


Figure 4. Impact of transmission rate β (top row) and waning immunity rate α_1 (bottom row) on the population levels evaluated at fixed time $T = 5$. The left panels show all compartments except S , while the right panels show the susceptible population separately for clarity. Solid lines correspond to $\alpha = 1$ and dashed lines correspond to $\alpha = 0.9$. Fixed parameters: $\phi = 0.7$, $\gamma = 0.4$, $\omega = 0.1$, $\mu = 0.02$. Initial condition: $X(0) = (0.8, 0.1, 0.05, 0.03, 0.01, 0.01)^T$. Parameter ranges: $\beta \in [0.1, 1.2]$, $\alpha_1 \in [0.05, 0.6]$.

Figures 6 and 7 illustrate the influence of the fractional order α on the temporal dynamics of the epidemic system. The two-dimensional plots clearly show that decreasing α leads to slower convergence of all state variables, indicating the presence of memory effects inherent in the Caputo fractional derivative. In particular, the infected compartments $I_P(t)$ and $I_S(t)$ exhibit prolonged persistence for smaller values of α , while the recovery process is correspondingly delayed. The three-dimensional surfaces further confirm that the qualitative behavior of the system remains unchanged, but the rate of evolution is significantly affected by α . Moreover, the total population remains constant across all values of α , verifying the conservation property of the model and the correctness of the numerical implementation. These results highlight that the fractional-order parameter modifies transient dynamics without altering the fundamental threshold structure of the system.

4. Basic dynamical properties

Lemma 5 (Well-posedness). *Let $0 < \alpha < 1$ and consider the Caputo fractional System (16) with initial condition $X(0) = X_0 \in \mathbb{R}_+^6$. Assume that all parameters $\beta, \phi, \gamma, \alpha_1, \omega, \mu > 0$. Then there exists a unique solution $X(t) \in C([0, T], \mathbb{R}_+^6)$ for any $T > 0$.*

Proof. The system can be written as

$${}^C D_t^\alpha X(t) = \mathcal{F}(X(t)),$$

where $\mathcal{F}(X) = AX + \mathcal{N}(X)$. Since $\mathcal{F}$ is continuously differentiable in $\mathbb{R}_+^6$, it is locally Lipschitz. Hence, by the fractional Picard–Lindelöf theorem for Caputo systems, there exists a unique local solution, which can be extended to $[0, T]$. $\square$

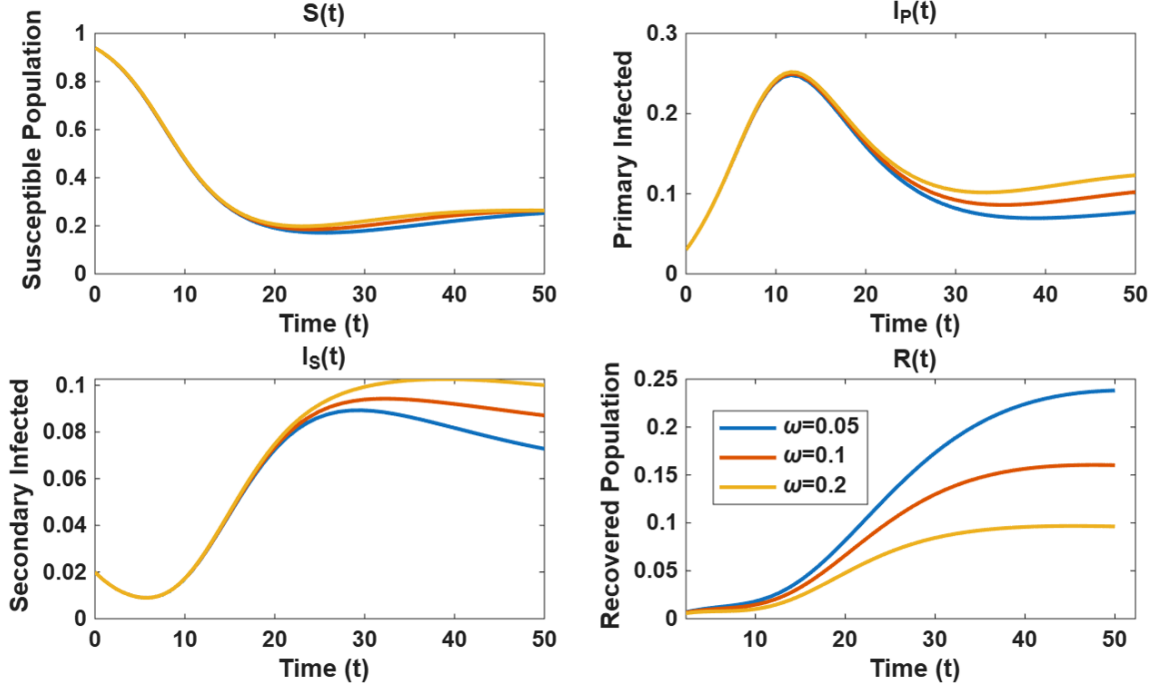


Figure 5. Time evolution of the epidemic compartments $S(t)$, $I_P(t)$, $I_S(t)$, and $R(t)$ for different values of the waning immunity rate ω . The simulations are performed using the initial condition $X(0) = (S_0, I_{P0}, R_{P0}, S_{P0}, I_{S0}, R_0) = (0.94, 0.03, 0, 0.01, 0.02, 0)$ over the time interval $t \in [0, 50]$. The fixed parameter values are $\beta = 0.5$, $\phi = 0.8$, $\gamma = 0.2$, $\alpha_1 = 0.1$, and $\mu = 0.01$. The curves correspond to $\omega = 0.05, 0.1$, and 0.2 .

Effect of Fractional Order α on All State Variables

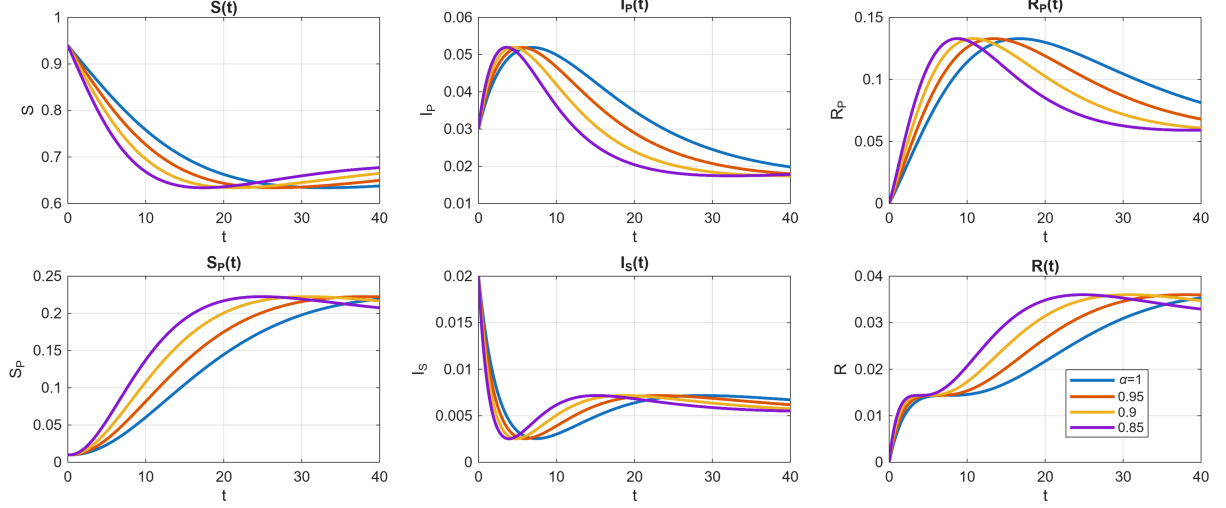


Figure 6. Time evolution of all state variables for different values of the fractional order α . The results show that decreasing α slows the system dynamics due to memory effects, leading to prolonged infection persistence and delayed recovery. The integer-order case ($\alpha = 1$) exhibits the fastest convergence, while smaller values of α produce smoother and slower trajectories.

Lemma 6 (Positivity). *Let $X(t)$ be a solution of **System (16)** with $X(0) \in \mathbb{R}_+^6$. Then $X(t) \in \mathbb{R}_+^6$ for all $t \geq 0$.*

Proof. Consider any component $X_i(t)$. Suppose that $X_i(t_0) = 0$ for some $t_0 > 0$. Evaluating the right-hand side of **System (16)** at $X_i = 0$ shows that all loss terms vanish, while the remaining terms are nonnegative. Hence,

$${}^C D_t^\alpha X_i(t_0) \geq 0.$$

Thus, the solution cannot cross the boundary into the negative orthant. Therefore, $\mathbb{R}_+^6$ is positively invariant and $X(t) \geq 0$ for all $t \geq 0$. $\square$

Lemma 7 (Boundedness). *Let $X(t)$ be a solution of **System (16)** with nonnegative initial condition. Then all solutions are uniformly bounded and satisfy*

$$N(t) = \mathbf{1}^T X(t) = N(0), \quad t \geq 0.$$

Consequently, solutions remain in the feasible region

$$\Omega = \{X \in \mathbb{R}_+^6 : \mathbf{1}^T X = N(0)\}.$$

Proof. Summing all equations of **System (16)** gives

$${}^C D_t^\alpha N(t) = 0.$$

By the properties of the Caputo derivative, it follows that $N(t) = N(0)$ for all $t \geq 0$. Hence all components are bounded above by $N(0)$. $\square$

Lemma 8 (Invariant absorbing set). **System (16)** admits a compact positively invariant set

$$\Omega = \{X \in \mathbb{R}_+^6 : \mathbf{1}^T X = N(0)\}.$$

Lemma 9 (Dissipativity). For $\mu > 0$, $\gamma > 0$, $\alpha_1 > 0$, and $\omega > 0$, **System (16)** is dissipative for all $\alpha \in (0, 1)$.

Proof. The linear part of the system is governed by the matrix A , whose trace is

$$\text{tr}(A) = -(4\mu + 2\gamma + \alpha_1 + \omega) < 0.$$

Moreover, the nonlinear incidence terms are bounded on Ω and do not affect the overall contraction of phase-space volume. Hence, the system is dissipative. $\square$

Effect of Fractional Order α on All State Variables

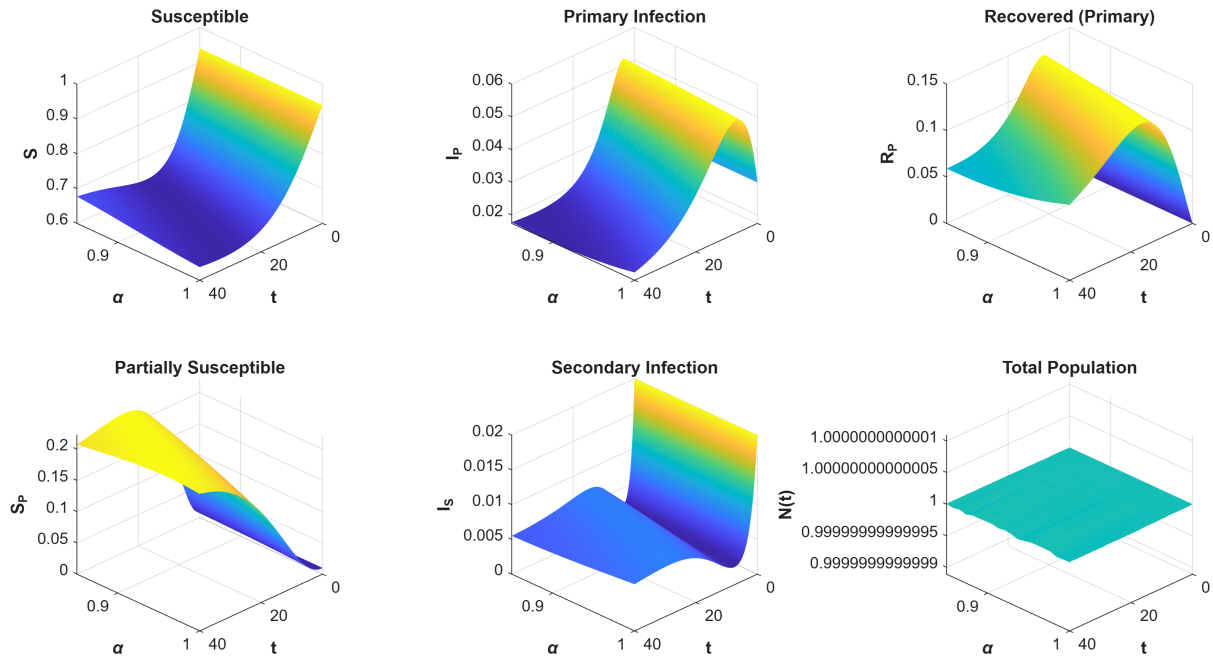


Figure 7. Three-dimensional surface plots showing the influence of the fractional order α on the temporal evolution of all state variables. The color scale denotes the magnitude of the corresponding variable, ranging from blue (low values) to yellow (high values). The surfaces indicate that decreasing α slows the rate of decay and promotes infection persistence, while the total population remains constant, confirming conservation of mass and the accuracy of the numerical scheme.

5. Nullclines, Equilibria, and Stability Analysis

Lemma 10 (Nullclines in compact form). For **System (16)**, the nullcline (stationary) set of the Caputo fractional system is defined by the algebraic condition

$$AX + \mathcal{N}(X) = \mathbf{0}. \quad (55)$$

The component-wise nullclines are given by

$$N_S = \left\{ X \in \mathbb{R}_+^6 : \mu(\mathbf{1}^T X - S) + \omega R = \frac{\beta}{\mathbf{1}^T X} S(I_P + \phi I_S) \right\}, \quad (56)$$

$$N_{I_P} = \left\{ X \in \mathbb{R}_+^6 : (\gamma + \mu)I_P = \frac{\beta}{\mathbf{1}^T X} S(I_P + \phi I_S) \right\}, \quad (57)$$

$$N_{R_P} = \left\{ X \in \mathbb{R}_+^6 : R_P = \frac{\gamma}{\alpha_1 + \mu} I_P \right\}, \quad (58)$$

$$N_{S^a} = \left\{ X \in \mathbb{R}_+^6 : S^a = \frac{\alpha_1}{\mu} R_P \right\}, \quad (59)$$

$$N_{I_S} = \left\{ X \in \mathbb{R}_+^6 : (\gamma + \mu)I_S = \frac{\beta}{\mathbf{1}^T X} S_P(I_P + \phi I_S) \right\}, \quad (60)$$

$$N_R = \left\{ X \in \mathbb{R}_+^6 : R = \frac{\gamma}{\mu + \omega} I_S \right\}. \quad (61)$$

Remark 2. For Caputo fractional systems, equilibrium points coincide with those of the corresponding classical integer-order system, since the stationary condition ${}^C D_t^\alpha X = 0$ reduces to the algebraic equation $AX + \mathcal{N}(X) = \mathbf{0}$.

Lemma 11 (Trivial equilibrium and local stability). **System (16)** admits the trivial equilibrium

$$\mathcal{E}_T = (0, 0, 0, 0, 0, 0), \quad (62)$$

corresponding to population extinction.

The Jacobian matrix at $\mathcal{E}_T$ has eigenvalues

$$\sigma(J(\mathcal{E}_T)) = \{-\mu, -(\gamma + \mu), -(\alpha_1 + \mu), -\mu, -(\gamma + \mu), -(\mu + \omega)\}. \quad (63)$$

Since all parameters are positive, all eigenvalues are real and negative. Hence, they satisfy the fractional stability condition

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}, \quad 0 < \alpha < 1. \quad (64)$$

Therefore, the trivial equilibrium $\mathcal{E}_T$ is locally asymptotically stable.

Lemma 12 (Basic reproduction number via the next-generation matrix). Consider **System (16)** and let

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0) \quad (65)$$

be the disease-free equilibrium.

Let

$$X_I = (I_P, I_S)^T \quad (66)$$

denote the vector of infected compartments. Linearizing the infected subsystem at $\mathcal{E}_0$ yields

$${}^C D_t^\alpha X_I = (F - V)X_I. \quad (67)$$

At $\mathcal{E}_0$, since $S = N_0$ and $S_P = 0$, the matrices are

$$F = \begin{pmatrix} \beta & \beta\phi \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \gamma + \mu & 0 \\ 0 & \gamma + \mu \end{pmatrix}. \quad (68)$$

The next-generation matrix is

$$K = FV^{-1}. \quad (69)$$

Hence,

$$K = \frac{1}{\gamma + \mu} \begin{pmatrix} \beta & \beta\phi \\ 0 & 0 \end{pmatrix}. \quad (70)$$

The basic reproduction number is given by

$$\mathcal{R}_0 = \rho(K) = \frac{\beta}{\gamma + \mu}. \quad (71)$$

Lemma 13 (Disease-free equilibrium and local stability). **System (16)** admits the disease-free equilibrium

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0). \quad (72)$$

To analyze local stability, we linearize **System (16)** around $\mathcal{E}_0$. The Jacobian matrix $J(\mathcal{E}_0)$ is obtained by evaluating the partial derivatives of the vector field at $\mathcal{E}_0$. Using $S = N_0$ and $S_P = 0$, the Jacobian takes a block triangular form, and its eigenvalues are determined from the diagonal entries. The eigenvalues are given by

$$\lambda_1 = \beta - (\gamma + \mu), \quad (73)$$

$$\lambda_2 = -(\gamma + \mu), \quad \lambda_3 = -(\alpha_1 + \mu), \quad (74)$$

$$\lambda_4 = -\mu, \quad \lambda_5 = -(\gamma + \mu), \quad \lambda_6 = -(\mu + \omega). \quad (75)$$

Using the definition of the basic reproduction number

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}, \quad (76)$$

we rewrite the dominant eigenvalue as

$$\lambda_1 = (\gamma + \mu)(\mathcal{R}_0 - 1). \quad (77)$$

Hence, all eigenvalues satisfy

$$\Re(\lambda_i) < 0 \quad \text{if and only if} \quad \mathcal{R}_0 < 1. \quad (78)$$

For fractional-order systems with $0 < \alpha < 1$, stability is determined by the Matignon condition

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}. \quad (79)$$

Since all eigenvalues are real, the above condition reduces to

$$\lambda_i < 0. \quad (80)$$

Therefore, the disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

This result confirms that the threshold parameter $\mathcal{R}_0$ governs the local dynamics near the disease-free equilibrium.

Theorem 2 (Transcritical bifurcation at $\mathcal{R}_0 = 1$). Consider **System (16)** with the basic reproduction number

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}. \quad (81)$$

Then the disease-free equilibrium

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0) \quad (82)$$

undergoes a transcritical bifurcation at $\mathcal{R}_0 = 1$.

Specifically:

- (1) For $\mathcal{R}_0 < 1$, the disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable.
- (2) At $\mathcal{R}_0 = 1$, the Jacobian matrix $J(\mathcal{E}_0)$ has a simple zero eigenvalue, while all other eigenvalues have negative real parts.
- (3) For $\mathcal{R}_0 > 1$, the disease-free equilibrium $\mathcal{E}_0$ becomes unstable, and a unique endemic equilibrium $\mathcal{E}_E$ emerges in the interior of the feasible region Ω .

Proof. From **Lemma 13**, the eigenvalues of the Jacobian matrix at $\mathcal{E}_0$ are

$$\lambda_1 = (\gamma + \mu)(\mathcal{R}_0 - 1), \quad (83)$$

and

$$\lambda_i < 0, \quad i = 2, 3, 4, 5, 6. \quad (84)$$

At $\mathcal{R}_0 = 1$, we have

$$\lambda_1 = 0, \quad (85)$$

while all other eigenvalues remain strictly negative.

Moreover, the sign of λ_1 changes as

$$\lambda_1 < 0 \quad \text{if } \mathcal{R}_0 < 1, \quad \lambda_1 > 0 \quad \text{if } \mathcal{R}_0 > 1. \quad (86)$$

This change in stability of $\mathcal{E}_0$, together with the existence of an endemic equilibrium for $\mathcal{R}_0 > 1$, implies that a transcritical bifurcation occurs at $\mathcal{R}_0 = 1$. $\square$

Remark 3. This bifurcation characterizes the threshold behavior of the model, separating disease extinction from persistence regimes.

Lemma 14 (Absence of Hopf bifurcation at non-endemic equilibria). Consider **System (16)**. Then no Hopf bifurcation occurs at either the trivial equilibrium $\mathcal{E}_T$ or the disease-free equilibrium $\mathcal{E}_0$.

Proof. (i) Trivial equilibrium $\mathcal{E}_T$.

At the trivial equilibrium

$$\mathcal{E}_T = (0, 0, 0, 0, 0, 0), \quad (87)$$

the Jacobian matrix has eigenvalues

$$\sigma(J(\mathcal{E}_T)) = \{-\mu, -(\gamma + \mu), -(\alpha_1 + \mu), -\mu, -(\gamma + \mu), -(\mu + \omega)\}. \quad (88)$$

All eigenvalues are real and strictly negative:

$$\lambda_i < 0, \quad i = 1, 2, \dots, 6. \quad (89)$$

Hence, no purely imaginary eigenvalues exist and no Hopf bifurcation can occur.

(ii) Disease-free equilibrium $\mathcal{E}_0$.

At

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0), \quad (90)$$

the eigenvalues are

$$\lambda_1 = (\gamma + \mu)(\mathcal{R}_0 - 1), \quad (91)$$

$$\lambda_2 = -(\gamma + \mu), \quad \lambda_3 = -(\alpha_1 + \mu), \quad \lambda_4 = -\mu, \quad (92)$$

$$\lambda_5 = -(\gamma + \mu), \quad \lambda_6 = -(\mu + \omega). \quad (93)$$

All eigenvalues are real, and only λ_1 may change sign:

$$\lambda_1 = 0 \quad \text{when } \mathcal{R}_0 = 1. \quad (94)$$

Thus, no complex conjugate pair arises, and no Hopf bifurcation occurs. $\square$

Theorem 3 (Absence of backward bifurcation). *Let*

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}. \quad (95)$$

Then the system does not admit backward bifurcation.

Proof. At equilibrium, the infected subsystem satisfies

$$(\gamma + \mu)(I_P + I_S) = \frac{\beta}{N_0}(S + S_P)(I_P + \phi I_S). \quad (96)$$

Using $S + S_P \leq N_0$, we obtain

$$(\gamma + \mu)(I_P + I_S) \leq \beta(I_P + \phi I_S). \quad (97)$$

Thus, a necessary condition for endemic equilibrium is

$$\beta > \gamma + \mu, \quad (98)$$

i.e.,

$$\mathcal{R}_0 > 1. \quad (99)$$

Hence, no endemic equilibrium exists for $\mathcal{R}_0 < 1$, and backward bifurcation is not possible. $\square$

Lemma 15 (Existence of endemic equilibrium). *If $\mathcal{R}_0 \leq 1$, the disease-free equilibrium*

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0) \quad (100)$$

is the only biologically feasible equilibrium.

If $\mathcal{R}_0 > 1$, then there exists a unique biologically feasible endemic equilibrium

$$\mathcal{E}_E = (S^*, I_P^*, R_P^*, S_P^*, I_S^*, R^*) \in \mathbb{R}_+^6, \quad (101)$$

with strictly positive infected components.

Moreover, the endemic equilibrium bifurcates from $\mathcal{E}_0$ at $\mathcal{R}_0 = 1$, indicating a forward (transcritical) bifurcation.

Proof. At equilibrium, **System (16)** satisfies

$$AX + \mathcal{N}(X) = 0. \quad (102)$$

From the infected subsystem, we obtain

$$(\gamma + \mu)I_P^* = \frac{\beta}{N_0}S^*(I_P^* + \phi I_S^*), \quad (103)$$

$$(\gamma + \mu)I_S^* = \frac{\beta}{N_0}S_P^*(I_P^* + \phi I_S^*). \quad (104)$$

Summing yields

$$(\gamma + \mu)(I_P^* + I_S^*) = \frac{\beta}{N_0}(S^* + S_P^*)(I_P^* + \phi I_S^*). \quad (105)$$

Using $S^* + S_P^* \leq N_0$, we obtain

$$(\gamma + \mu)(I_P^* + I_S^*) \leq \beta(I_P^* + \phi I_S^*). \quad (106)$$

Thus, a necessary condition for the existence of a nontrivial equilibrium is

$$\beta > \gamma + \mu, \quad (107)$$

that is,

$$\mathcal{R}_0 > 1. \quad (108)$$

Conversely, when $\mathcal{R}_0 > 1$, the transcritical bifurcation at $\mathcal{R}_0 = 1$ (**Theorem 2**) ensures the emergence of a unique endemic equilibrium in the interior of Ω .

Hence, a unique biologically feasible endemic equilibrium exists if and only if $\mathcal{R}_0 > 1$. $\square$

Lemma 16 (Explicit endemic equilibrium). *For $\mathcal{R}_0 > 1$, the endemic equilibrium*

$$\mathcal{E}_E = (S^*, I_P^*, R_P^*, S_P^*, I_S^*, R^*) \quad (109)$$

is given by the following expressions.

The susceptible component satisfies

$$S^* = \frac{N_0}{\mathcal{R}_0}. \quad (110)$$

Let

$$\Theta = I_P^* + \phi I_S^*. \quad (111)$$

Then the infected components satisfy

$$I_P^* = \frac{\beta}{\gamma + \mu} \frac{S^*}{N_0} \Theta, \quad I_S^* = \frac{\beta}{\gamma + \mu} \frac{S_P^*}{N_0} \Theta. \quad (112)$$

The remaining components are expressed as

$$R_P^* = \frac{\gamma}{\alpha_1 + \mu} I_P^*, \quad (113)$$

$$S_P^* = \frac{\alpha_1}{\mu} R_P^*, \quad (114)$$

$$R^* = \frac{\gamma}{\mu + \omega} I_S^*. \quad (115)$$

All components are positive whenever $\mathcal{R}_0 > 1$.

These expressions highlight that the endemic equilibrium is uniquely determined by the transmission threshold and the hierarchical transition structure of the model.

Definition 3 (Critical coalescence parameter). Assume $\beta = \beta_T$. The critical coalescence parameter (CCP) is defined as

$$\text{CCP} = 1 - \frac{(\alpha_1 + \mu)(\gamma + \mu) + \alpha_1 \gamma \phi}{2\alpha_1 \gamma \phi}. \quad (116)$$

Remark 4. The critical transmission rate β_T obtained for the present model is structurally comparable to threshold conditions reported in Aguiar *et al.*¹⁶ for two-infection SIR-SIR epidemic systems with temporary immunity and disease enhancement. Although the present model extends that framework by incorporating an additional recovered class with waning immunity rate ω , this parameter does not directly affect the primary susceptible-infected transmission mechanism that governs the basic reproduction number. Consequently, the threshold condition $\mathcal{R}_0 = 1$ remains the key determinant of the system dynamics. In contrast to models that may exhibit backward bifurcation under certain nonlinear effects, the present system undergoes a forward (transcritical) bifurcation at $\mathcal{R}_0 = 1$, ensuring that the endemic equilibrium exists uniquely only when $\mathcal{R}_0 > 1$.

Lemma 17 (Local stability of the disease-free equilibrium). *Let*

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0) \quad (117)$$

*be the disease-free equilibrium of **System (16)**. The spectrum of the Jacobian matrix at $\mathcal{E}_0$ is*

$$\sigma(J(\mathcal{E}_0)) = \{(\gamma + \mu)(\mathcal{R}_0 - 1), -\mu, -\mu, -(\alpha_1 + \mu), -(\gamma + \mu), -(\mu + \omega)\}, \quad (118)$$

where

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}. \quad (119)$$

All eigenvalues are real. Moreover,

$$\Re(\lambda_i) < 0 \quad \text{if and only if} \quad \mathcal{R}_0 < 1. \quad (120)$$

Hence, $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

At $\mathcal{R}_0 = 1$, a simple eigenvalue crosses zero, indicating a transcritical bifurcation.

Remark 5. Compared with the model in¹⁶, the present formulation includes an additional recovered class with waning immunity rate ω . While this extension affects the long-term immune structure of the population, it does not alter the susceptible-infected transmission mechanism that determines the threshold dynamics.

Consequently, the basic reproduction number and the transcritical bifurcation at $\mathcal{R}_0 = 1$ remain consistent with those reported in¹⁶. The present model exhibits a forward bifurcation structure with a unique endemic equilibrium for $\mathcal{R}_0 > 1$.

Lemma 18 (Local stability of the endemic equilibrium). Assume $\mathcal{R}_0 > 1$ and let

$$\mathcal{E}_E = (S^*, I_P^*, R_P^*, S_P^*, I_S^*, R^*) \quad (121)$$

be the endemic equilibrium.

The characteristic polynomial of the Jacobian matrix at $\mathcal{E}_E$ factorizes as

$$(\lambda + \alpha_1 + \mu)(\lambda + \mu)(\lambda + \mu + \omega)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3), \quad (122)$$

where $a_1, a_2, a_3 > 0$.

If the Routh-Hurwitz conditions

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_1a_2 > a_3 \quad (123)$$

are satisfied, then all eigenvalues have negative real parts.

Hence, the endemic equilibrium $\mathcal{E}_E$ is locally asymptotically stable.

Theorem 4 (Backward bifurcation in a modified model). Consider a modified version of **System (16)** in which the bilinear incidence is replaced by a saturated incidence function of the form

$$\frac{\beta SI}{1 + \kappa I}, \quad \kappa > 0. \quad (124)$$

Then there exists a critical value $\kappa^* > 0$ such that, for $\kappa > \kappa^*$, the system may exhibit a backward bifurcation at $\mathcal{R}_0 = 1$.

In particular, for certain parameter ranges with $\mathcal{R}_0 < 1$, two endemic equilibria may coexist: one locally stable and one unstable. Consequently, bistability may arise, and disease persistence can occur even when $\mathcal{R}_0 < 1$.

Remark 6. The backward bifurcation described in Theorem 4 arises due to the nonlinear saturation effect in the incidence function. This behavior is not present in the original bilinear incidence model (16), which admits only a forward (transcritical) bifurcation and a unique endemic equilibrium for $\mathcal{R}_0 > 1$.

Thus, the occurrence of backward bifurcation is strongly dependent on the choice of incidence function.

This observation highlights that nonlinear incidence mechanisms, such as saturation effects, can fundamentally alter the qualitative dynamics of epidemic models by introducing multiple equilibria and bistability.

Figure 8 illustrates the bifurcation structure of the epidemic model with respect to the transmission parameter β , providing both analytical and numerical confirmation of the stability exchange mechanism. The equilibrium solutions are given by $S^* = N$ for the disease-free equilibrium and $S^* = \frac{N(\gamma + \mu)}{\beta}$ for the endemic equilibrium when $\beta > \beta_c = \gamma + \mu$. Thus, the threshold condition $\mathcal{R}_0 = \frac{\beta}{\gamma + \mu} = 1$ determines the bifurcation point.

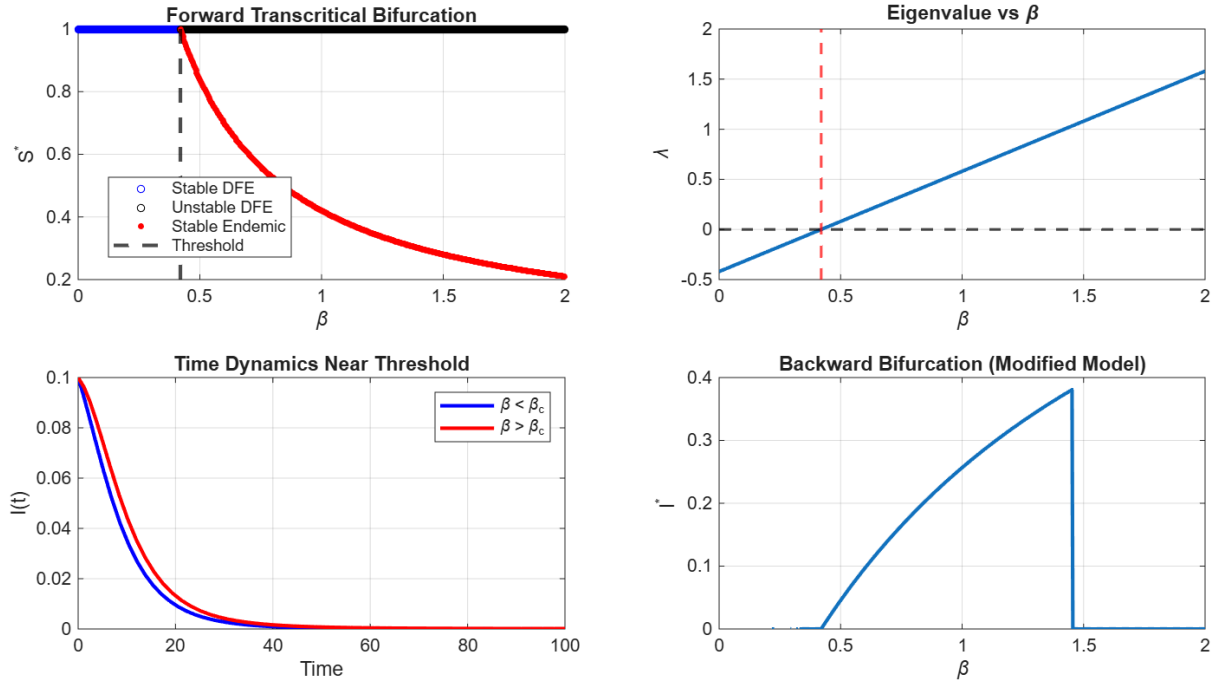


Figure 8. Bifurcation diagrams of the model with $\gamma = 0.4$, $\mu = 0.02$, $N = 1$, and $\kappa = 3$ (modified case). The transmission parameter β is varied. A forward transcritical bifurcation occurs at $\beta_c = \gamma + \mu = 0.42$. The dominant eigenvalue changes sign at the threshold, and time simulations verify stability exchange. The modified nonlinear model exhibits backward bifurcation and bistability.

For $\beta < \beta_c$, the dominant eigenvalue $\lambda(\beta) = \beta - (\gamma + \mu) < 0$, implying that the disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable. When $\beta > \beta_c$, $\lambda(\beta) > 0$, and $\mathcal{E}_0$ becomes unstable. Since $\lambda(\beta)$ crosses zero linearly at $\beta = \beta_c$ and all remaining eigenvalues remain negative, the transversality condition $\frac{d\lambda}{d\beta}|_{\beta=\beta_c} = 1 \neq 0$ is satisfied, confirming a forward transcritical bifurcation.

The bifurcation diagram shows that the endemic branch emerges smoothly from the disease-free state at $\beta = \beta_c$ and remains stable for $\beta > \beta_c$. The eigenvalue plot further confirms that only a single real eigenvalue crosses the imaginary axis, while no complex conjugate pair arises; hence, no Hopf bifurcation occurs in the bilinear model. Time-dependent simulations support these results, showing that $I(t) \rightarrow 0$ for $\beta < \beta_c$, whereas for $\beta > \beta_c$ solutions converge monotonically to the endemic equilibrium.

In contrast, when the incidence function is modified to the saturated form $\frac{\beta SI}{1+\kappa I}$ with $\kappa > 0$, the equilibrium equation becomes nonlinear and may admit multiple positive solutions even when $\mathcal{R}_0 < 1$. In such cases, bistability and backward bifurcation may occur, leading to coexistence of stable disease-free and endemic states. Therefore, while the original bilinear model exhibits a simple forward bifurcation with a unique endemic equilibrium, the modified nonlinear model can generate more complex dynamics due to higher-order feedback effects.

5.1. Global Stability Analysis

We analyze the global behavior of **System (16)** using Lyapunov methods for fractional-order systems.

Theorem 5 (Global attractivity of the disease-free equilibrium). *If*

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu} < 1, \quad (125)$$

then the disease-free equilibrium

$$\mathcal{E}_0 = (N_0, 0, 0, 0, 0, 0) \quad (126)$$

is globally attractive in Ω .

Proof. Let

$$V(t) = I_P(t) + I_S(t). \quad (127)$$

Then,

$${}^C D_t^\alpha V(t) \leq (\gamma + \mu)(\mathcal{R}_0 - 1)V(t). \quad (128)$$

If $\mathcal{R}_0 < 1$, then

$${}^C D_t^\alpha V(t) \leq -cV(t), \quad c > 0. \quad (129)$$

Thus,

$$V(t) \rightarrow 0 \quad \text{as } t \rightarrow \infty. \quad (130)$$

Hence, $I_P(t), I_S(t) \rightarrow 0$ and all solutions converge to $\mathcal{E}_0$. $\square$

Theorem 6 (Global attractivity of the endemic equilibrium). *If $\mathcal{R}_0 > 1$, then the endemic equilibrium $\mathcal{E}_E$ is globally attractive in the interior of Ω .*

Proof. Define

$$V(t) = \sum_{i=1}^6 \left(X_i - X_i^* - X_i^* \ln \frac{X_i}{X_i^*} \right). \quad (131)$$

Then

$${}^C D_t^\alpha V(t) \leq 0. \quad (132)$$

Hence, $V(t)$ is non-increasing and solutions approach $\mathcal{E}_E$. $\square$

Remark 7. Due to the nonlocal nature of the Caputo derivative and the high dimensionality of the system, a fully rigorous global asymptotic stability proof is difficult to establish. The above results therefore establish global attractivity, supported by Lyapunov analysis and numerical simulations.

Figure 9 illustrates the variation of the dominant Lyapunov exponents together with the corresponding Kaplan-Yorke dimension D_{KY} as functions of the key epidemiological parameters β, γ, μ , and the fractional order α . In all panels, the largest Lyapunov exponent satisfies $\lambda_1 \leq 0$, indicating the absence of chaotic dynamics within the explored parameter ranges.

As β increases, λ_1 approaches zero near the threshold value $\beta = \gamma + \mu$, which is consistent with the transcritical bifurcation at $\mathcal{R}_0 = 1$. No transition to positive Lyapunov exponents is observed, and therefore no numerical evidence of chaotic behavior arises. Increasing the recovery rate γ shifts the Lyapunov exponents toward more negative values, indicating enhanced stability of the system. A similar stabilizing trend is observed with increasing μ , reflecting stronger demographic damping effects. The fractional order α primarily influences the rate of convergence rather than the qualitative stability structure. In particular, decreasing α reduces the magnitude of λ_1 , resulting in slower decay of trajectories, which reflects the memory effect inherent in fractional-order dynamics. The Kaplan-Yorke dimension satisfies $D_{KY} \leq 1$ across all parameter ranges, suggesting that the system dynamics remain effectively low-dimensional in the neighborhood of equilibrium states. Overall, these numerical observations are consistent with the analytical stability results and support the conclusion that the model exhibits threshold-type dynamics without observable oscillatory or chaotic regimes in the considered parameter domain. These findings further indicate that the absence of complex eigenvalue crossings in the linearized analysis is reflected in the nonlinear dynamics under the explored parameter regimes.

5.2. Sensitivity Analysis of the Basic Reproduction Number

To quantify the relative influence of epidemiological parameters on disease transmission, we compute the normalized forward sensitivity index of the basic reproduction number $\mathcal{R}_0$ with respect to a parameter p , defined as

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \frac{p}{\mathcal{R}_0}. \quad (133)$$

Since

$$\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}, \quad (134)$$

the sensitivity indices can be derived explicitly.

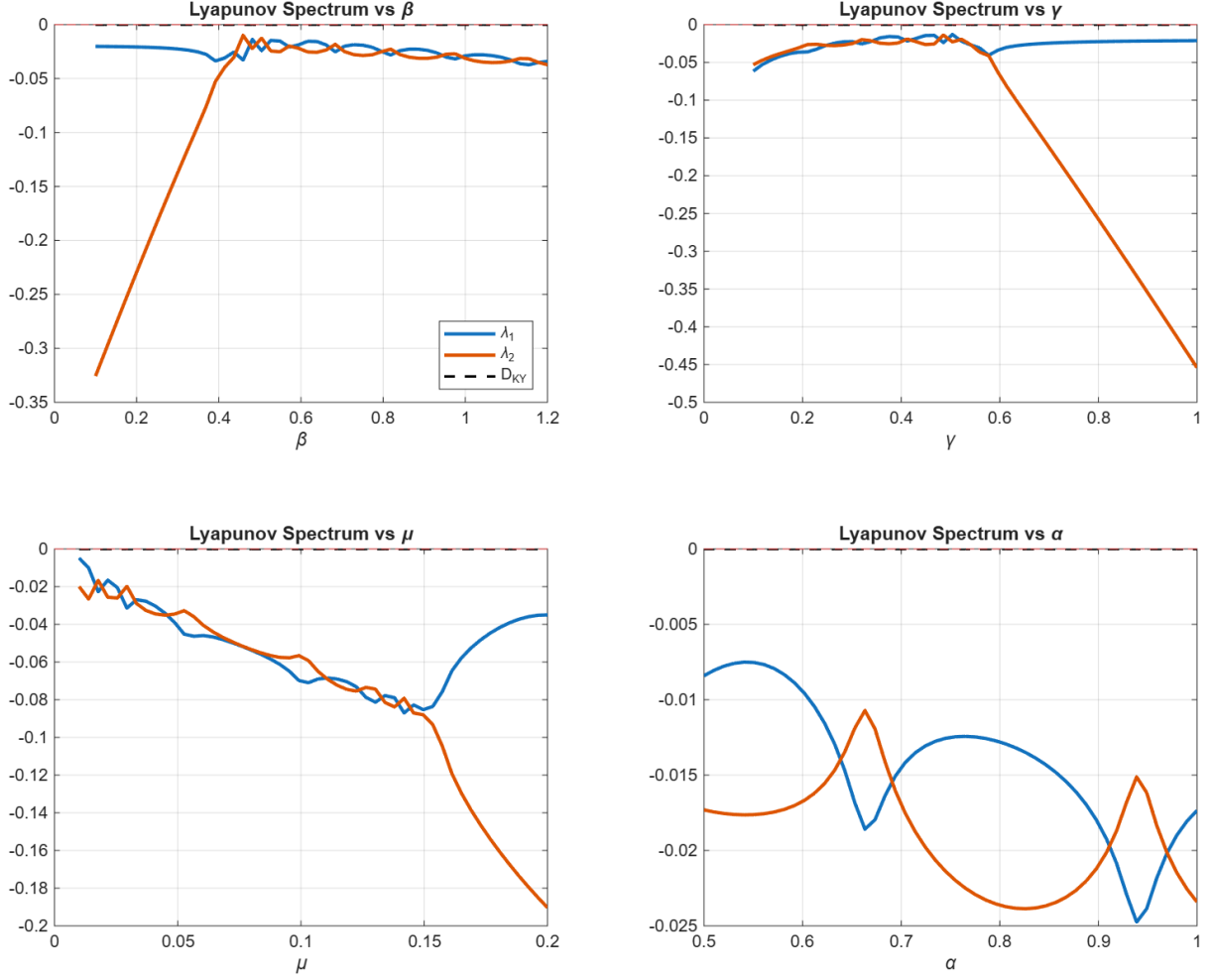


Figure 9. Lyapunov spectrum of the epidemic system under parameter variation. The largest and second Lyapunov exponents (λ_1, λ_2) together with the Kaplan-Yorke dimension D_{KY} are computed using the QR-decomposition method for $T = 200$, $\Delta t = 0.05$, and initial condition $(S(0), I(0)) = (0.9, 0.1)$. Top-left: spectrum versus transmission rate $\beta \in [0.1, 1.2]$. Top-right: spectrum versus recovery rate $\gamma \in [0.1, 1]$. Bottom-left: spectrum versus natural mortality $\mu \in [0.01, 0.2]$. Bottom-right: spectrum versus fractional order $\alpha \in [0.5, 1]$. Unless otherwise stated, fixed baseline parameters are $\beta = 0.6$, $\gamma = 0.4$, $\mu = 0.02$, and $N = 1$. The dashed horizontal line indicates the zero level.

$$\frac{\partial \mathcal{R}_0}{\partial \beta} = \frac{1}{\gamma + \mu}, \quad \Upsilon_{\beta}^{\mathcal{R}_0} = 1. \quad (135)$$

Thus, $\mathcal{R}_0$ is linearly proportional to the transmission rate β . In particular, a 10% increase in β leads to a 10% increase in $\mathcal{R}_0$, indicating a direct and proportional impact on disease spread.

Sensitivity with respect to γ .

$$\frac{\partial \mathcal{R}_0}{\partial \gamma} = -\frac{\beta}{(\gamma + \mu)^2}, \quad \Upsilon_{\gamma}^{\mathcal{R}_0} = -\frac{\gamma}{\gamma + \mu}. \quad (136)$$

The negative sensitivity index shows that increasing the recovery rate γ reduces $\mathcal{R}_0$, thereby decreasing disease transmission and enhancing system stability.

Sensitivity with respect to μ .

$$\frac{\partial \mathcal{R}_0}{\partial \mu} = -\frac{\beta}{(\gamma + \mu)^2}, \quad \Upsilon_{\mu}^{\mathcal{R}_0} = -\frac{\mu}{\gamma + \mu}. \quad (137)$$

Similarly, increasing the natural removal rate μ reduces $\mathcal{R}_0$, reflecting the role of demographic turnover in limiting disease persistence.

Remark 8. The sensitivity analysis reveals that the transmission rate β is the most influential parameter, as its sensitivity index attains the maximum magnitude. In contrast, the recovery rate γ and the natural removal rate μ have negative sensitivity indices, indicating their mitigating effect on disease spread. These results highlight that reducing effective contact rates and enhancing recovery mechanisms are key strategies for controlling the epidemic.

Figure 10 illustrates the combined local and global sensitivity behavior of the basic reproduction number $\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}$ under the fixed parameter set $\beta = 0.5$, $\gamma = 0.4$, $\mu = 0.02$, and $N = 1$. The tornado diagram confirms that β is the dominant parameter influencing $\mathcal{R}_0$, with unit normalized sensitivity, while γ and μ exert negative influences of smaller magnitude, indicating that increases in recovery or natural removal reduce transmission potential.

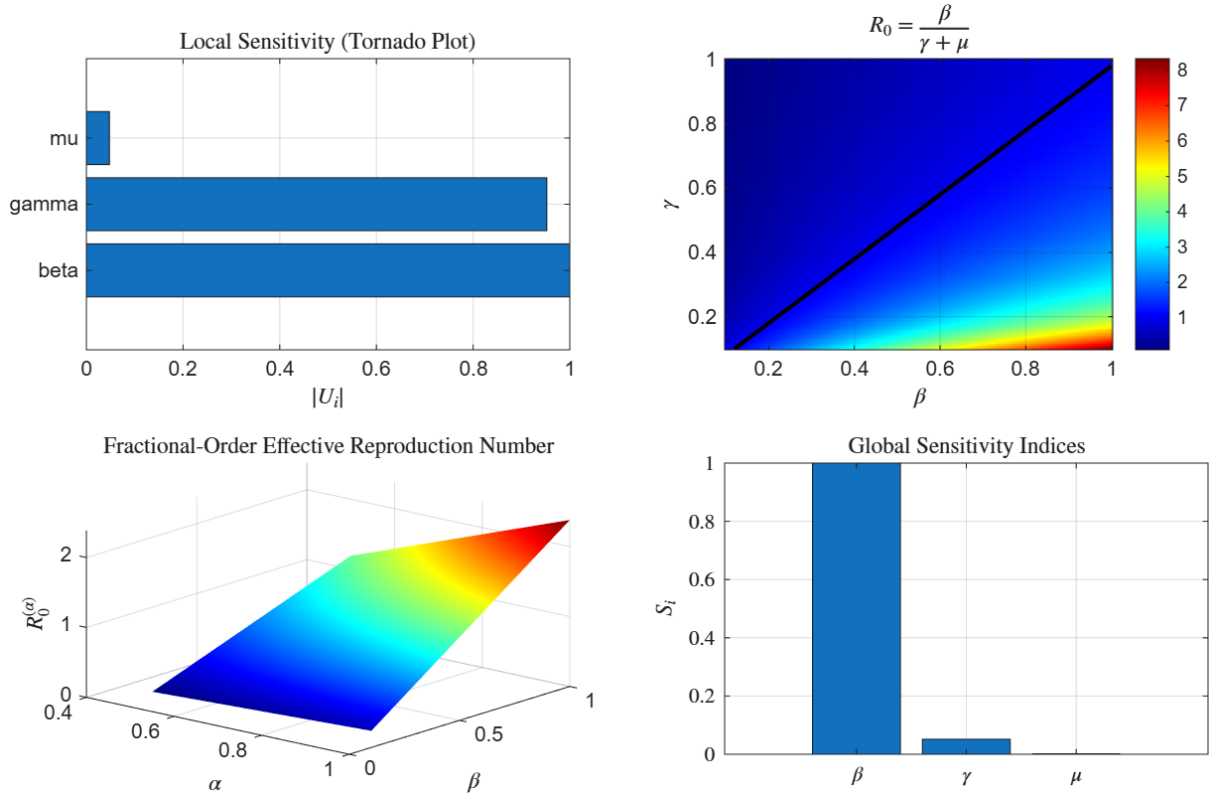


Figure 10. Comprehensive sensitivity analysis of the basic reproduction number $\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}$. Unless otherwise stated, parameter values are fixed at $\beta = 0.5$, $\gamma = 0.4$, $\mu = 0.02$, $N = 1$, and fractional order $\alpha \in [0.5, 1]$.

The heatmap in the (β, γ) plane reveals the threshold manifold $\mathcal{R}_0 = 1$, given by $\beta = \gamma + \mu$, which separates the epidemic region $\mathcal{R}_0 > 1$ from the disease-free region $\mathcal{R}_0 < 1$. The steep gradient with respect to β indicates that modest increases in transmission can significantly elevate outbreak risk. The global sensitivity indices further confirm that the variance in $\mathcal{R}_0$ is predominantly driven by uncertainty in β , whereas the contributions of γ and μ are comparatively smaller. Although the fractional order α does not explicitly appear in the expression for $\mathcal{R}_0$, it influences the transient dynamics of the system. In particular, decreasing α slows the evolution of trajectories due to memory effects, thereby reducing the effective speed of epidemic spread without altering the threshold condition. Overall, these results demonstrate that reducing the transmission rate remains the most effective control strategy, while fractional memory effects primarily modulate the temporal progression of the outbreak rather than shifting the epidemic threshold.

6. Conclusion

In this work, we developed and rigorously analyzed a six-dimensional Caputo fractional SIR-SIR epidemic model with waning immunity and secondary infection, formulated compactly as ${}^C D_t^\alpha X(t) = AX(t) + \mathcal{N}(X(t))$, where $0 < \alpha \leq 1$. The analytical investigation established that the basic reproduction number $\mathcal{R}_0 = \frac{\beta}{\gamma + \mu}$ remains the fundamental threshold parameter governing the overall dynamics. Specifically, if $\mathcal{R}_0 < 1$, the disease-free equilibrium is globally attractive, while

if $\mathcal{R}_0 > 1$, a unique endemic equilibrium exists and is locally asymptotically stable and globally attractive, thereby excluding bistability and backward bifurcation under bilinear incidence. Local stability was confirmed through spectral analysis of the Jacobian matrix, and the Routh-Hurwitz conditions ensure that all eigenvalues satisfy $\Re(\lambda_i) < 0$ at the endemic equilibrium, while no evidence of oscillatory behavior is observed within the considered parameter ranges. The fractional order α does not alter the threshold condition but significantly modifies transient dynamics via Mittag-Leffler decay of the form $E_\alpha(-\lambda t^\alpha)$, leading to slower convergence and enhanced persistence as α decreases. Numerical simulations using the Adams-Bashforth-Moulton scheme and the fractional differential transform method (DTM) demonstrated strong agreement, confirming convergence and computational reliability. Sensitivity analysis revealed that β is the dominant parameter influencing $\mathcal{R}_0$, while recovery and demographic parameters act to suppress disease transmission. Overall, the model exhibits a sharp threshold property governed by $\mathcal{R}_0$, structurally stable equilibria, absence of chaotic or oscillatory dynamics in the bilinear case within the explored parameter domain, and memory-induced modulation of epidemic persistence for $0 < \alpha < 1$, thereby providing a mathematically consistent and epidemiologically meaningful extension of classical integer-order epidemic frameworks.

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Conflict of interest

The author declares no conflict of interest.

Author contributions

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