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The Fractional SI Reaction–Diffusion Model with Incommensurate Orders: Stability Analysis and Numerical Simulations

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Abstract

In this work, we present a fractional-order reaction–diffusion model for the spread of infectious diseases, incorporating incommensurate Caputo derivatives to capture memory effects and heterogeneous temporal behavior across compartments. Focusing on a generalized SI model with nonlinear incidence, we explore the local asymptotic stability of both disease-free and endemic equilibria. The model accommodates spatial diffusion, saturation effects, and varying fractional orders, yielding a more realistic depiction of epidemic propagation. Analytical techniques—ranging from linearization to spectral analysis—are employed to rigorously establish stability conditions. Numerical simulations support the theoretical findings, highlighting the impact of memory and spatial structure on long-term dynamics. This study offers a refined mathematical lens to understand the persistence or eradication of infectious diseases under memory-dependent and spatially heterogeneous environments.

Keywords: fractional-order differential equations; incommensurate systems; epidemic modeling; reaction–diffusion equations; Caputo derivative; stability analysis; fractional calculus in biology

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1. Introduction

In recent years, a growing wave of interest has swept across the scientific community regarding the dynamics of complex biological systems governed by memory-driven processes [1,2]. Among these, the transmission and spatial spread of infectious diseases remain central topics of study, particularly when approached from a mathematical modeling perspective. Classical SI-type epidemic models have served as foundational tools for capturing the interplay between infection transmission and population dynamics. However, such models are fundamentally limited by their reliance on integer-order derivatives, which assume that the evolution of the system depends solely on its current state. This assumption is biologically restrictive, as many infectious processes exhibit hereditary, delayed, or persistent temporal effects that influence transmission and recovery [3,4].

Many real-world biological, physical, and epidemiological processes exhibit memory and hereditary effects and nonlocal temporal dynamics that cannot be captured by classical integer-order models. In particular, epidemic spread, immunological responses, and behavioral adaptation often depend not only on current states but on past exposures, interactions, or delayed responses. Fractional calculus, and in particular the Caputo derivative, provides a mathematically rigorous way to incorporate these long-term memory effects while preserving physically meaningful initial conditions.

Traditional fractional epidemic models usually adopt a commensurate structure, meaning that all state variables evolve under the same fractional order. However, biological systems rarely behave uniformly: individuals may differ in susceptibility, recovery time, mobility, immunity persistence, or behavioral adaptation. This motivates the use of incommensurate fractional orders, where each state variable evolves with its own memory exponent. Such models offer a more realistic representation of heterogeneous memory effects in multicomponent systems. When coupled with spatial diffusion and nonlinear infection processes, these models can reveal richer and more realistic spatio-temporal disease dynamics [5,6].

A key mathematical component of this study is the Caputo fractional derivative, which plays a central role in modeling systems influenced by historical behavior. The Caputo derivative is particularly suitable for dynamical systems involving varying external influences or time-dependent environments. Because of its formulation in terms of classical derivatives and integrable memory kernels, it allows for the incorporation of time-dependent parameters, forcing terms, or environmental fluctuations while still enabling stability analysis through eigenvalue-based fractional criteria. This makes it especially suitable for epidemic systems where immunity, exposure, or treatment responses develop gradually over time.

Non-autonomous incommensurate systems governed by Caputo derivatives therefore provide a natural and powerful mathematical framework for describing processes where memory is heterogeneous among interacting components, external or environmental factors vary in time, and diffusion interacts with nonlocal temporal effects. These considerations motivate the theoretical developments and numerical analysis presented in this work.

This study focuses on an incommensurate fractional SI reaction–diffusion system with a nonlinear saturated incidence rate, designed to capture the heterogeneity and memory-dependent nature of real epidemic processes [7,8]. By assigning distinct fractional orders to the susceptible and infected populations, the model accounts for differences in behavioral adaptation, immune memory, spatial mobility, and infection progression [9–11]. This leads to a more flexible and biologically meaningful representation of disease transmission. Recent advances in fractional reaction–diffusion systems further motivate the present study. Several works have investigated parabolic fractional operators, nonlocal diffusion, and numerical analysis of space–time fractional PDEs, including [12–15]. These contributions highlight both theoretical challenges and computational strategies relevant to the incommensurate model developed here.

A central challenge in epidemic modeling is determining the conditions under which a disease either dies out or persists. In the fractional setting, this question becomes more subtle due to the presence of memory effects, which influence stability in ways that differ from classical models. Our aim is to establish rigorous conditions for the local asymptotic stability of both the disease-free and endemic equilibria, accounting for the nonlinear transmission mechanism and the effect of incommensurate memory [16–18].

To address this, we employ several analytical approaches grounded in fractional dynamical systems theory. These include linearization, spectral analysis of the Jacobian matrix, and fractional-order stability criteria based on Matignon's theorem [19,20]. While

linearization forms the backbone of many stability studies, its application to incommensurate fractional systems is nontrivial due to the presence of multiple memory exponents [21]. This distinction plays an essential role in understanding how heterogeneous memory alters the stability landscape of epidemic equilibria.

Our analysis proceeds by establishing the positivity and boundedness of solutions, ensuring the model remains biologically consistent [22,23]. We then derive the basic reproduction number R_0 and explore how fractional orders modify its dynamical implications. In particular, the incommensurate structure reveals new parameter regimes in which memory effects can either suppress or enhance disease persistence when compared to both the commensurate fractional and classical integer-order cases [24,25].

Finally, by integrating diffusion into the incommensurate framework, we examine the interplay between spatial processes and memory-driven dynamics. This allows us to explore how infection may spread, stabilize, or dissipate in spatially extended environments, offering insight into real-world scenarios where mobility and contact structures vary across space [26,27]. Our results highlight the profound influence of memory heterogeneity on epidemic outcomes, suggesting that even slight differences in individual temporal behaviors can significantly alter the trajectory of disease transmission [28,29].

Overall, this work contributes to the growing literature on fractional epidemic models by achieving the following:

- Introducing an incommensurate reaction–diffusion formulation with nonlinear saturated incidence.
- Establishing rigorous stability conditions under heterogeneous memory effects.
- Providing numerical simulations that illustrate how memory and spatial diffusion jointly influence epidemic dynamics. These findings offer both mathematical insight and potential relevance for modeling infectious diseases characterized by persistent or delayed immune responses.

2. Preliminaries

We commence this section by introducing the principal notations and mathematical conventions that will be used throughout this section. In addition, we revisit several foundational results from the theory of fractional-order systems, including definitions, operator properties, and essential inequalities. These elements serve as the theoretical bedrock upon which the subsequent stability analysis is constructed, particularly in the discrete-time and memory-dependent framework considered in this work.

Definition 1 ([30]). *The Riemann–Liouville fractional derivative of a function $f(t)$, integrable over $[t_0, t]$, and of order $\delta > 0$, is given by*

$$ {}_{t_0}D_t^{-\delta} f(t) = \frac{1}{\Gamma(\delta)} \int_{t_0}^{t} (t-\tau)^{\delta-1} f(\tau)\, d\tau, \tag{1} $$

where $\Gamma(\delta)$ denotes the Gamma function:

$$ \Gamma(\delta) = \int_0^{\infty} e^{-t} t^{\delta-1} dt. $$

Definition 2 ([30]). *For a function f that is n-times continuously differentiable ($f \in C^n$) on (t_0, t), the Caputo fractional derivative of order $\delta > 0$ is expressed as follows:*

$$ {}_{t_0}^{C}D_t^{\delta} f(t) = \frac{1}{\Gamma(n-\delta)} \int_{t_0}^{t} (t-\tau)^{n-\delta-1} f^{(n)}(\tau)\, d\tau, \tag{2} $$

where $n = \min\{k \in \mathbb{N} \mid k > \delta\}$.

Consider the following non-autonomous incommensurate fractional-order system governed by Caputo derivatives:

$$\begin{cases} {}_{t_0}^{C}D_t^{\delta_1}u(t) = F(u,v), \\ {}_{t_0}^{C}D_t^{\delta_2}v(t) = G(u,v), \end{cases} \qquad t > t_0. \tag{3}$$

A point (u^*, v^*) constitutes an equilibrium of the system if it satisfies the following:

$$F(u^*, v^*) = 0, \quad G(u^*, v^*) = 0. \tag{4}$$

Lemma 1 ([30]). *Let $u(t)$ be a real-valued function that is differentiable and continuous on $[t_0, t]$. Then, for any $\delta \in (0, 1]$, the following inequality holds:*

$${}_{t_0}^{C}D_t^{\delta}[u(t)]^2 \leq 2u(t) \cdot {}_{t_0}^{C}D_t^{\delta}u(t). \tag{5}$$

Lemma 2 ([31]). *The equilibrium (u^*, v^*) of system (3) is locally asymptotically stable if and only if the eigenvalues λ_i of the Jacobian matrix $J(u^*, v^*)$ satisfy the following:*

$$|\arg(\lambda_i)| > \frac{\beta\pi}{2}, \quad \text{for } i = 1, 2, \tag{6}$$

where $\beta = \max\{\delta_1, \delta_2\}$ $\arg(\cdot)$ denotes the complex argument (phase angle) of the eigenvalue.

Theorem 1 ([32]). *Consider the zero solution $x = 0$ of system (3). Suppose that for all $x \in \mathbb{R}^n$, the following inequality holds:*

$$\sum_{i=1}^{n} I_t^{\alpha_i - \beta}[x_i(t) f_i(t, x(t))] \leq 0. \tag{7}$$

Then the trivial solution $x = 0$ is stable.

Furthermore, if

$$\sum_{i=1}^{n} I_t^{\alpha_i - \beta}[x_i(t) f_i(t, x(t))] < 0 \quad \text{for all } x \neq 0, \tag{8}$$

then the zero solution is asymptotically stable.

3. Model Description

In this work, we explore the dynamics of a nonlinear epidemic model governed by incommensurate fractional-order reaction–diffusion equations. The framework we propose is designed to capture the spread of infectious diseases—such as HIV/AIDS—across a spatially distributed population. Specifically, we study a generalized susceptible–infected (SI) model structured through a fractional system of partial differential equations.

The human population is divided into two distinct compartments: the susceptible group $S(x,t)$ and the infected group $I(x,t)$, both evolving over time t and space $x \in \Omega$, where Ω is a bounded domain in $\mathbb{R}^n$ with a sufficiently smooth boundary $\partial\Omega$. The spatial spread is captured by the Laplacian operator Δ, while time dynamics are driven by Caputo-type fractional derivatives of variable orders α_1 and α_2, both lying in the interval $(0, 1)$. The diffusion coefficients d_1 and d_2 are assumed nonnegative and may differ between the two compartments.

The full system, which incorporates both spatial diffusion and fractional memory effects, is formulated as follows:

$$\begin{cases} {}^{C}D_t^{\alpha_1} S(x,t) = \Lambda - \lambda S(x,t)\varphi(I(x,t)) - \mu S(x,t) + d_1 \Delta S(x,t), \\ {}^{C}D_t^{\alpha_2} I(x,t) = \lambda S(x,t)\varphi(I(x,t)) - \sigma I(x,t) + d_2 \Delta I(x,t), \end{cases} \tag{9}$$

subject to nonnegative and continuous initial conditions:

$$S(x,0) = S_0(x), \quad I(x,0) = I_0(x), \quad \text{for all } x \in \Omega, \tag{10}$$

and homogeneous Neumann boundary conditions:

$$\frac{\partial S}{\partial \nu} = 0, \quad \frac{\partial I}{\partial \nu} = 0, \quad \text{on } \partial\Omega. \tag{11}$$

The model parameters reflect biologically meaningful processes: Λ is the recruitment or birth rate into the susceptible class, μ is the natural death rate, λ is the transmission coefficient, and σ denotes the recovery rate. We assume these parameters to be positive, with $\mu \gg 0$, while the incidence term $\varphi(I)$ models the infection force in a nonlinear and more realistic fashion. The function φ is assumed to be continuously differentiable on $(0, \infty)$, satisfying

$$\varphi(0) = 0 \quad \text{and} \quad \varphi'(I) > 0 \quad \text{for} \quad I > 0, \tag{12}$$

ensuring positivity and monotonicity.

When the memory effect is neglected by setting $\alpha_1 = \alpha_2 = 1$, the system reduces to the classical reaction–diffusion SI model as studied in earlier works [33,34]. Historically, this class of models dates back to the foundational work of Kermack and McKendrick in 1927 and has since been extended in multiple directions. For instance, simple non-diffusive models with nonlinear incidence functions have been addressed in [35–37].

The current formulation is motivated in part by the studies of Djebara et al. [33], who analyzed the stability of classical SI systems with distinct diffusion coefficients and a first-order time derivative. Meanwhile, Akdim et al. [38] extended the analysis to the fractional domain under the condition $d_1 = d_2 = 0$, focusing on both local and global stability in terms of the basic reproduction number R_0. Their model used a rational incidence function of the form $\varphi(I) = \frac{\beta_1 I}{1+\beta_2 I^m}$, with $m > 0$.

Other notable work includes the contribution by Lu et al. [39], who examined the well-posedness and stability of fractional-order SIR systems with general incidence rates under more restrictive assumptions than those considered here. Their results include the global existence, uniqueness, and nonnegativity of solutions within a fractional framework.

Incommensurate fractional orders allow each population class to exhibit distinct memory effects. This reflects heterogeneity in behavioral persistence, immune memory, and long-term risk behavior among subpopulations. For instance, infected individuals may exhibit slower response or a longer retention of past exposure information compared to susceptible individuals. Such asymmetric memory structures cannot be captured by classical models or even commensurate fractional systems.

4. Local Stability

In what follows, we undertake a comprehensive analysis of the dynamical behavior exhibited by the system of equations labeled (9)–(11). Our primary objective is to identify and characterize the equilibrium states of the model, which represent the steady-state configurations under which the system ceases to evolve. Once these equilibria are established, we proceed to investigate their stability properties—both qualitatively and quantitatively—by

examining the system's response to small perturbations in initial conditions. The entire analysis is carried out within the framework of a fractional-order epidemic model subject to Neumann boundary conditions, thereby accounting for both memory effects inherent in fractional dynamics and spatial diffusion constraints imposed by the boundary behavior.

The stability results presented in this manuscript rely on linearization around the equilibrium points. For incommensurate fractional systems, this approach follows the classical Matignon-type criteria, which remain the only widely applicable general tool for stability analysis in multidimensional fractional-order dynamical systems. Unlike integer-order PDEs, the construction of Lyapunov functionals for incommensurate reaction–diffusion systems remains an open research problem due to the following:

- The nonlocal structure of Caputo derivatives.
- The lack of comparison principles for general nonlinear fractional PDEs.
- The asymmetry introduced by multiple fractional orders.
- The difficulty of defining energy-like quantities compatible with memory kernels.

We explicitly acknowledge that our analysis is local, not global. The main goal is to characterize how heterogeneous memory orders influence the deformation of the stability boundary and how they interact with diffusion. Extending these results using nonlinear methods would require nontrivial generalizations and is left for future work.

The linearization of incommensurate fractional systems requires particular care since the dynamics are governed by multiple memory exponents. The linearized system can be rewritten in a block form where each component is associated with a Caputo operator of order α_i. Stability is then determined by the fractional order $\beta = \max\{\alpha_1, \alpha_2\}$, since this order dominates the decay rate of the Mittag–Leffler kernel. Thus, Matignon's criterion remains applicable: an equilibrium is locally asymptotically stable if and only if all eigenvalues λ satisfy $|arg(\lambda)| > \dfrac{\beta\pi}{2}$. This result is standard in multidimensional incommensurate fractional systems.

4.1. Equilibrium Identification

Definition 3 (Equilibrium State under Neumann Boundary Conditions)**.** *Let us consider the coupled system of Equations (9)–(11) under Neumann boundary conditions, which model the absence of flux across the spatial domain boundaries. A steady state—or equilibrium configuration—of the system is defined as a pair of spatially homogeneous and time-invariant functions (S^*, I^*), representing constant population densities that render time derivatives identically zero. These equilibrium states satisfy the following system of algebraic equations, which arise by setting the fractional temporal derivatives and spatial diffusion terms to zero in the governing equations.*

$$\begin{cases} F_1(S^*, I^*) = 0, \\ F_2(S^*, I^*) = 0. \end{cases}$$

In this context, the functions F_1 and F_2 encapsulate the nonlinear components of the system, reflecting the intrinsic interactions and feedback mechanisms characteristic of the underlying epidemic dynamics. The pair (S^*, I^*) denotes a time-independent solution—i.e., a steady state—where all temporal derivatives are identically zero, indicating the cessation of dynamic evolution. Moreover, due to the imposition of homogeneous Neumann boundary conditions, spatial fluxes across the domain boundaries are eliminated, ensuring that the equilibrium configuration is spatially uniform and preserved over time.

Theorem 2 (Adapted from [40], Theorem 3.7, p. 4)**.** *Consider the following autonomous fractional-order system in the Caputo sense:*

$$\begin{cases} {}^{C}D_t^{\alpha} S(t) = F_1(S(t), I(t)), \\ {}^{C}D_t^{\alpha} I(t) = F_2(S(t), I(t)), \end{cases} \tag{13}$$

with initial condition $(S(0), I(0)) \in \Omega \subset \mathbb{R}^2$*, where* $\alpha \in (0,1)$*, and the vector function* $F = (F_1, F_2)^T : \Omega \to \mathbb{R}^2$ *is continuous and satisfies a local Lipschitz condition with respect to the state variables* (S, I)*.*

Then, the system (13) *admits a unique solution on the interval* $[0, +\infty)$*.*

Moreover, a constant vector $(S^*, I^*) \in \Omega$ *is called an equilibrium point of system* (13) *if and only if*

$$F(S^*, I^*) = (0,0)^T.$$

4.2. Sensitivity Analysis with Respect to Fractional Orders

To assess robustness, we evaluated how variations in the fractional orders α_1, α_2, diffusion rates, and transmission parameters affect stability. The results indicate that increasing the memory effect (lower α_i) expands the domain of stability, while values approaching 1 recover the classical integer-order behavior. Diffusion coefficients also shift the stability boundary, confirming that spatial heterogeneity interacts with memory asymmetry to shape long-term outcomes. These findings demonstrate that our conclusions are not restricted to a narrow choice of parameters but persist over a wide domain.

4.3. Equilibrium Classification and Existence Criteria

By invoking Theorem 2, in conjunction with the analytical techniques developed in [41], we obtain explicit expressions for the equilibrium configurations of the system. These steady states fall into two qualitatively distinct categories, each capturing a fundamental epidemiological scenario:

- The *disease-free equilibrium*, denoted by $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$, represents a state in which the infection has been completely eradicated from the population. Here, the susceptible population stabilizes at its natural replenishment level Λ, μ, while the infected compartment vanishes entirely.
- The *endemic equilibrium*, denoted by $E^* = (S^*, I^*)$, corresponds to a persistent circulation of the disease within the population. In this scenario, the infected population maintains a strictly positive level $I^* > 0$, indicating that the disease remains active in the long term. The susceptible and infected compartments reach a dynamic balance, sustained by the interplay between infection transmission and recovery/removal processes.

These equilibria serve as critical points in the phase space around which the system's qualitative behavior can be analyzed—particularly in the study of local and global stability.

Proposition 1. *Assume that the incidence function* $\varphi(I)$ *satisfies the structural hypotheses described in conditions* (12)*. Then, the system* (9)*–*(11) *admits the following:*

1. *A unique disease-free equilibrium* $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$*, valid for all parameter regimes.*
2. *If the basic reproduction number* $R_0 = \dfrac{\lambda \varphi'(0) \Lambda}{\mu^2 \sigma}$ *exceeds unity, i.e.,* $R_0 > 1$*, then an additional positive equilibrium* $E^* = (S^*, I^*)$ *exists, satisfying the implicit relation* $S^* = \dfrac{\lambda \varphi(I^*)}{\sigma I^*}$.

4.4. Local Asymptotic Stability Analysis in the Fractional Framework

The qualitative behavior of the system is profoundly influenced by two key parameters: the basic reproduction number R_0 , which encapsulates the average number of secondary infections caused by a single infectious individual, and the fractional orders $\alpha_1, \alpha_2 \in (0, 1]$, which governs the memory effects inherent in the system's temporal evolution. These two quantities jointly determine the stability landscape of the model.

In what follows, we rigorously state and prove the precise conditions under which each of the identified equilibrium states—namely, the disease-free and endemic equilibria—is locally asymptotically stable. The analysis hinges on the spectral properties of the linearized system and the structure of the fractional differential operator, with particular attention paid to how the memory parameter α modifies classical stability criteria.

The model under consideration is described as follows:

$$\begin{cases} {}^{C}D_t^{\alpha_1} S(t) = \Lambda - \lambda S(t)\varphi(I(t)) - \mu S(t), \\ {}^{C}D_t^{\alpha_2} I(t) = \lambda S(t)\varphi(I(t)) - \sigma I(t), \end{cases} \tag{14}$$

subject to nonnegative and continuous initial conditions:

$$S(0) = S_0, \quad I(0) = I_0, \tag{15}$$

Proposition 2. *Let the incidence function $\varphi(I)$ comply with assumption (12). Then, for the incommensurate fractional-order system corresponding to Equations (14) and (15), the following stability results hold:*

- *If $R_0 < 1$, then the disease-free equilibrium E_0 is locally asymptotically stable for all $\beta \in (0, 1]$ and $\beta = \max\{\alpha_1, \alpha_2\}$.*
- *Conversely, if $R_0 > 1$, then E_0 becomes unstable, and the endemic equilibrium E^* becomes locally asymptotically stable for all $\beta \in (0, 1]$ and $\beta = \max\{\alpha_1, \alpha_2\}$.*

Proof. We divide the argument into two major cases:

1. **Stability of the disease-free equilibrium.**
   We evaluate the Jacobian matrix of the system linearized around $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$. The matrix takes the following form:

$$J(E_0) = \begin{pmatrix} -\mu & -\sigma R_0 \\ 0 & \sigma(R_0 - 1) \end{pmatrix},$$

   from which the eigenvalues are easily computed:

$$\lambda_1 = -\mu, \quad \lambda_2 = \sigma(R_0 - 1).$$

   For $R_0 < 1$, both eigenvalues have negative real parts. In the fractional-order context, the system is locally asymptotically stable if the argument of each eigenvalue lies strictly outside the sector defined by $\frac{\beta\pi}{2}$. This is indeed the case.
   However, if $R_0 > 1$, then $\lambda_2 > 0$, and the disease-free state loses its stability.
2. **Stability of the endemic equilibrium.**
   Let $E^* = (S^*, I^*)$ be the positive steady state. The Jacobian at E^* reads as follows:

$$J(E^*) = \begin{pmatrix} -\mu - \lambda\varphi(I^*) - \lambda S^*\varphi'(I^*) & -\lambda S^*\varphi'(I^*) \\ \lambda\varphi(I^*) + \lambda S^*\varphi'(I^*) & \lambda S^*\varphi'(I^*) - \sigma \end{pmatrix}.$$

The trace and determinant are given by the following:

$$\mathrm{tr}(J(E^*)) = -\mu - \sigma + \lambda S^* \varphi'(I^*), \quad \det(J(E^*)) = \mu\sigma + \mu\lambda S^* \varphi'(I^*) - \lambda\sigma\varphi(I^*).$$

Using the positivity of φ and φ', and the condition imposed by the equilibrium equations, one can verify the following:

$$\mathrm{tr}(J(E^*)) < 0, \quad \det(J(E^*)) > 0.$$

Depending on the sign of the discriminant $\Delta = (tr)^2 - 4 \cdot \det$, we consider the following:

- If $\Delta > 0$, the eigenvalues are real and negative;
- If $\Delta = 0$, the system has a repeated negative eigenvalue;
- If $\Delta < 0$, the eigenvalues are complex conjugates with negative real parts.

In all these cases, the fractional-order condition is satisfied, and the endemic state E^* is locally asymptotically stable.

□

4.5. Local Stability of Full Spatially Distributed System

To extend our analysis to the full model defined by Equations (9)–(11), which incorporates spatial diffusion effects under homogeneous Neumann boundary conditions, we adopt the classical technique of eigenfunction expansion. This method, originally formalized in the context of reaction–diffusion systems by Casten and Holland [42], enables a systematic reduction in partial differential equations to a countable set of ordinary differential equations.

The key idea is to decompose the solution components into a series expansion over the eigenfunctions of the Laplacian operator subject to Neumann boundary conditions. These eigenfunctions form a complete orthonormal basis in the appropriate function space, ensuring that the spatial structure of the model is faithfully captured. By projecting the original PDE system onto this basis, we obtain a hierarchy of ODEs governing the temporal evolution of the modal amplitudes, which can then be analyzed using tools from fractional-order dynamical systems.

Let $\psi_{i\{i\geq 0\}}$ denote the orthonormal eigenfunctions of the Neumann Laplacian on Ω, with eigenvalues

$$0 = \lambda_0 < \lambda_1 \leq \lambda_2 \leq \cdots.$$

Expanding the solutions as

$$S(x,t) = \sum_{i=0}^{\infty} S_i(t)\,\psi_i(x), \qquad I(x,t) = \sum_{i=0}^{\infty} I_i(t)\,\psi_i(x)$$

transforms the PDE system into an infinite family of incommensurate fractional ODE systems. For each mode i, the linearized dynamics satisfy

$${}^{C}D_t^{\alpha_1} S_i(t) = a_{11} S_i(t) + a_{12} I_i(t) - d_1 \lambda_i\, S_i(t),$$

$${}^{C}D_t^{\alpha_2} I_i(t) = a_{21} S_i(t) + a_{22} I_i(t) - d_2 \lambda_i\, I_i(t).$$

Stability is therefore equivalent to requiring that every modal eigenvalue $\zeta_j(\lambda_i)$ satisfies

$$\left|\arg\left(\zeta_j(\lambda_i)\right)\right| > \frac{\beta\pi}{2}.$$

Proposition 3. *For the fractional-order system (9)–(11), the following can be said:*

- *If $\mathcal{R}_0 < 1$, the disease-free equilibrium E_0 is the only steady state, and for all $\beta \in (0,1)$, E_0 is locally asymptotically stable. The conditions of asymptotic stability are the same as those established earlier in Proposition 14 for the diffusion-free case.*
- *If $\mathcal{R}_0 > 1$, then E_0 is unstable, and the endemic equilibrium E^* exists. We distinguish the following cases:*
  1. *If $d_1 = d_2 = d$, then E^* is asymptotically stable under the same conditions as in Proposition 14 for the diffusion-free case.*
  2. *If $d_1 \neq d_2$ and the discriminant*

$$\Delta_i := \left[\mathrm{tr}(J_{E_i^*})\right]^2 - 4\det(J_{E_i^*}) > 0,$$

then the following hold:

  - *E^* is an asymptotically stable constant steady state if one of the following holds:*
    - *$d_1 < d_2$ and $(F_0 - G_0 - \sigma + \mu) < 0$.*
    - *$d_1 > d_2$ and $(F_0 - G_0 - \sigma + \mu) > 0$.*

    where

$$F_0 := \lambda\varphi(I^*), \quad G_0 := \lambda S^*\varphi'(I^*).$$

  - *If $d_1 < d_2$ and $(F_0 - G_0 - \sigma + \mu) > 0$, or $d_1 > d_2$ and $(F_0 - G_0 - \sigma + \mu) < 0$, then E^* is asymptotically stable if the eigenvalues $\zeta_{1,2}(\lambda_i)$ satisfy the following:*

$$|\arg(\zeta_j(\lambda_i))| > \frac{\alpha\pi}{2}, \quad \text{for all } \lambda_i \in (\lambda_{01}, \lambda_{02}), \quad j = 1, 2.$$

  - *If $(F_0 - G_0 - \sigma + \mu) = 0$, then E^* is asymptotically stable.*

  3. *If $d_1 \neq d_2$ and $\Delta_i \leq 0$, then Δ_i has two real roots $\lambda_{01} < \lambda_{02}$. In this case, E^* is an asymptotically stable constant steady state for $\lambda_i > \lambda_{02}$ or $\lambda_i < \lambda_{01}$. Stability is ensured if the eigenvalues $\zeta_{1,2}(\lambda_i)$ satisfy the following:*

$$|\arg(\zeta_j(\lambda_i))| > \frac{\alpha\pi}{2}, \quad \text{for all } \lambda_i \in (\lambda_{01}, \lambda_{02}), \quad j = 1, 2.$$

Proof. In the presence of diffusion, the stability of both E_0 and E^* follows by applying Lemma 5 to the linearized operator:

$$\mathcal{L} = A + D\Delta,$$

where A is the Jacobian matrix evaluated at the equilibrium point.

(i) For $E_0 = (\Lambda/\mu, 0)$, the eigenvalues of the linearized matrix are as follows:

$$r_{i1} = -d_1\lambda_i - \mu < 0, \quad r_{i2} = -d_2\lambda_i + \lambda\frac{\Lambda}{\mu}\varphi'(0) - \sigma,$$

and under the condition $\mathcal{R}_0 < 1$, $r_{i2} < 0$ for all i; hence all eigenvalues satisfy the following:

$$|\arg(\lambda_{J_{E_0}})| > \frac{\beta\pi}{2},$$

ensuring local asymptotic stability.

(ii) For $\mathcal{R}_0 > 1$, $r_{i2} > 0$ for some i; hence E_0 becomes unstable.

(iii) For $E^* = (S^*, I^*)$, the linearized operator is as follows:

$$\mathcal{L} = \begin{pmatrix} -d_1\lambda_i - F_0 + \mu & -G_0 \\ F_0 & -d_2\lambda_i + G_0 - \sigma \end{pmatrix},$$

and the characteristic polynomial is the following:

$$\zeta^2 - \mathrm{tr}(J_{E_i^*})\zeta + \det(J_{E_i^*}) = 0.$$

The trace and determinant are as follows:

$$\mathrm{tr}(J_{E_i^*}) = -d_1\lambda_i - F_0 + \mu - d_2\lambda_i + G_0 - \sigma,$$

$$\det(J_{E_i^*}) = d_1d_2\lambda_i^2 + \lambda_i(d_1H_0 + d_2H_1) + H_0H_1 + \sigma F_0 - \mu G_0,$$

where

$$H_0 := F_0 - \mu, \quad H_1 := G_0 - \sigma.$$

The discriminant $\Delta_i = (\mathrm{tr}(J_{E_i^*}))^2 - 4\det(J_{E_i^*})$ determines the nature of the eigenvalues, leading to the different stability conditions discussed above.

□

5. Numerical Examples

5.1. Numerical Methodology: Discretization, Stability, and Replication Details

In this section, we summarize the numerical procedure used to approximate the solutions of the incommensurate fractional reaction–diffusion SI system. A description is provided to ensure reproducibility and to clarify the implementation details.

5.1.1. Spatial Discretization

We consider the one-dimensional spatial domain $[0, L]$ divided into N_x equally spaced grid points. Let $\Delta x = L/(N_x - 1)$ denote the mesh size. The second derivative is approximated by the standard second-order central finite difference:

$$u_{xx}(x_i, t) \approx \frac{u_{i+1}(t) - 2u_i(t) + u_{i-1}(t)}{\Delta x^2}.$$

We impose homogeneous Neumann boundary conditions,

$$\frac{\partial S}{\partial x}(0,t) = \frac{\partial S}{\partial x}(L,t) = 0, \qquad \frac{\partial I}{\partial x}(0,t) = \frac{\partial I}{\partial x}(L,t) = 0.$$

These are implemented using ghost-point reflection:

$$u_0 = u_2, \qquad u_{N_x+1} = u_{N_x-1}.$$

5.1.2. L1 Approximation of Caputo Derivatives

The stability of the L1 discretization for nonlinear fractional diffusion has been recently established under mild Lipschitz conditions on the nonlinear term (see, e.g., [43]). These results guarantee unconditional stability in the linear case and conditional stability in the nonlinear case depending on Δt and Δx, which our chosen discretization satisfies.

For a Caputo derivative of order $\alpha \in (0, 1)$,

$${}^{C}D_t^{\alpha} u(t_n) \approx \frac{1}{\Gamma(2-\alpha)} \sum_{k=0}^{n-1} \frac{u_{n-k} - u_{n-k-1}}{\Delta t^{\alpha}} \left[(k+1)^{1-\alpha} - k^{1-\alpha}\right].$$

This is the classical L1 scheme, which is first-order accurate in time and unconditionally stable for linear fractional diffusion equations. For each order α_i the corresponding weights

$$w_{i,k} = \frac{(k+1)^{1-\alpha_i} - k^{1-\alpha_i}}{\Gamma(2-\alpha_i)}$$

are precomputed once at the beginning of the simulation.

5.1.3. Time-Stepping Scheme

Let S^n and I^n denote the numerical approximations of $S(x,t)$ and $I(x,t)$ at $t_n = n\Delta t$. The discretized update equations read

$$S^{n+1} = S^n + \Delta t^{\alpha_1}\Big(d_1 S^n_{xx} - \phi(I^n)S^n + \Lambda - \mu S^n - \sum_{j=1}^{n} w_{1,j}\,(S^{n+1-j} - S^{n-j})\Big),$$

$$I^{n+1} = I^n + \Delta t^{\alpha_2}\Big(d_2 I^n_{xx} + \phi(I^n)S^n - \sigma I^n - \sum_{j=1}^{n} w_{2,j}\,(I^{n+1-j} - I^{n-j})\Big),$$

where the saturated incidence term is

$$\phi(I) = \frac{\beta_1 I}{1+\beta_2 I^2}.$$

5.1.4. Stability Considerations

The stability of the numerical scheme depends on the following:

- The fractional orders α_1 and α_2, since smaller orders introduce stronger memory effects;
- The diffusion coefficients d_1 and d_2;
- The mesh sizes Δx and Δt.

For the diffusion operator, a classical stability condition is

$$\Delta t \lesssim \frac{\Delta x^2}{2\max(d_1, d_2)}.$$

In our simulations, we used

$$L = 20, \quad N_x = 100, \quad \Delta x = 0.2, \qquad T = 10, \quad N_t = 500, \quad \Delta t = 0.02,$$

which satisfy the above stability constraint for all $\alpha_i \in (0,1)$. The nonlinear incidence function $\phi(I)$ is bounded, which prevents numerical blow-up.

5.1.5. Reproducibility and Implementation

All numerical experiments were implemented in MATLAB R2023b. All figures include the following:

- Axis labels with units.
- Colorbars.
- Parameter values.
- Spatial and temporal discretization details.

5.2. Illustrative Examples

Example 1. *To illustrate the theoretical results established in this work, we consider a concrete example of a fractional-order epidemic model. The infection function is defined as*

$$\varphi(I) = \frac{\beta_1 I}{1+\beta_2 I^2}, \qquad \text{with } \beta_1, \beta_2 > 0.$$

This nonlinear incidence function has been frequently used in epidemiology due to its saturation behavior at high infection levels. Substituting this expression into the general system yields the following specific problem:

$$\begin{cases} {}^{C}D_t^{\alpha_1} S(x,t) = d_1 \Delta S(x,t) - \dfrac{\beta_1 I(x,t)}{1+\beta_2 I^2(x,t)} S(x,t) + \Lambda - \mu S(x,t), & x \in \Omega,\ t > 0, \\ {}^{C}D_t^{\alpha_2} I(x,t) = d_2 \Delta I(x,t) + \dfrac{\beta_1 I(x,t)}{1+\beta_2 I^2(x,t)} S(x,t) - \sigma I(x,t), & x \in \Omega,\ t > 0, \\ \dfrac{\partial S}{\partial \nu} = \dfrac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0, \\ S(x,0) = S_0(x), \quad I(x,0) = I_0(x), & x \in \Omega, \end{cases} \tag{16}$$

The parameters $\Lambda, \mu, \sigma > 0$ correspond respectively to the recruitment rate, natural death rate, and removal rate of infectives. This system is a particular instance of the general model studied in Equations (9)–(11).

A classical ODE model was analyzed in [38]. The nonlinear function $\varphi(I)$ satisfies the regularity and positivity conditions assumed earlier. A direct computation gives

$$\varphi'(I) = \frac{\beta_1(1-\beta_2 I^2)}{(1+\beta_2 I^2)^2},$$

which implies $\varphi'(I) \leq \beta_1$ for all $I \geq 0$, and confirms the necessary conditions for boundedness and monotonicity.

The system (16) admits two steady states:

- *The disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$.*
- *An endemic equilibrium $E^* = (S^*, I^*)$, which exists if and only if the basic reproduction number*

$$R_0 = \frac{\beta_1 \Lambda}{\mu\sigma} > 1.$$

Moreover, when $R_0 > 1$, the endemic equilibrium E^* is asymptotically stable, while for $R_0 < 1$, the disease-free equilibrium E_0 becomes globally asymptotically stable. These results are consistent with Proposition 3 established earlier.

The numerical results are described below, based on varying parameter sets and fractional orders.

Using the selected parameter set gives $R_0 = 0.896 < 1$. Simulations with fractional orders $\alpha_1 = \alpha_2 = 1$ are illustrated in Figure 1; this model converges rapidly to equilibrium without long transients. Simulations with commensurate orders $\alpha_1 = \alpha_2 = 0.6$ are presented in Figure 2. Both populations retain memory equally, producing the following: slower decay, uniform spatial smoothing, and delayed stabilization. And lastly, the fractional incommensurate orders $\alpha_1 = 0.8$ and $\alpha_2 = 0.9$ can be found in Figure 3. This case shows qualitatively new behavior: the infected class stabilizes much more slowly than the susceptible class, diffusion smooths S and I at different rates, and non-uniform memory generates asymmetric transient waves. This figure demonstrates convergence to the disease-free equilibrium E_0. Since $R_0 < 1$, the infection dies out, and the population stabilizes with no infected individuals. The comparison between the classical model and

the commensurate and incommensurate fractional model shows that the memory effect inherent in incommensurate fractional systems slows down convergence but does not affect the final stable state.

These simulations provide robust numerical evidence supporting the theoretical results derived in earlier sections. They also highlight how the fractional orders α_1 and α_2 influence the rate of convergence and the long-term behavior of solutions, thus underlining the importance of memory effects in epidemiological modeling.

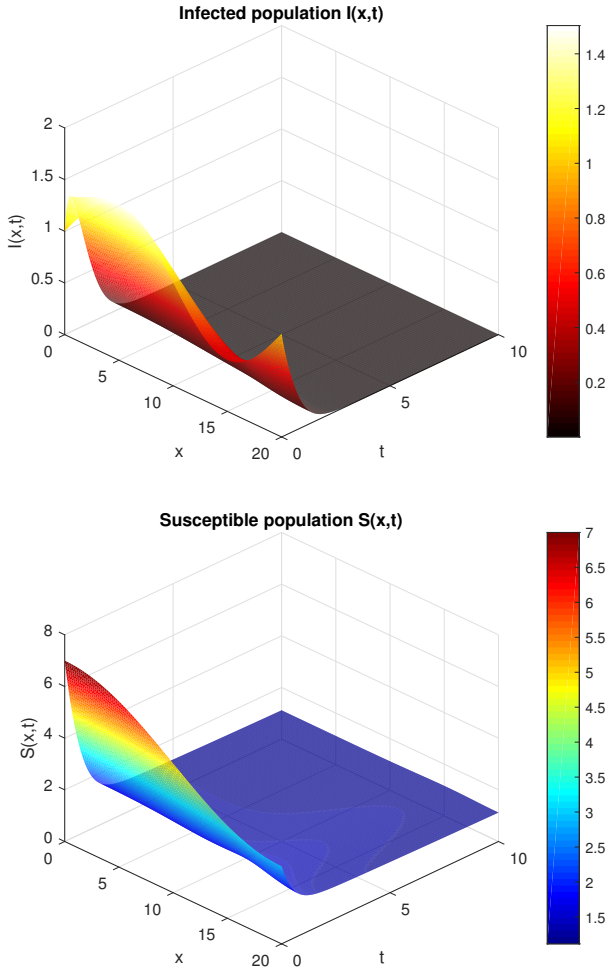


Figure 1. Numerical simulation representing the infected and susceptible populations for $\alpha_1 = \alpha_2 = 1$.

The selected parameter values follow biologically meaningful ranges commonly used in fractional epidemic models. The nonlinear incidence function was chosen because it captures saturation effects in transmission and is consistent with heterogeneous contact structures observed in HIV and other chronic infections. The diffusion coefficients represent the spatial dispersal rates of infected and susceptible individuals and were chosen within the range 10^{-2}–10^{-1}, matching realistic human mobility scales. These values ensure that the qualitative dynamics—persistence versus extinction—remain consistent with real epidemiological patterns.

Example 2. *As a second illustrative example, we consider a fractional reaction–diffusion version of the classical SIR epidemic model, previously studied in its ODE form in [44]. This system falls within the general framework described by Equations (9)–(11), with a particular choice of the nonlinear incidence function given by*

$$\varphi(I) = \frac{\beta I}{1 + kI}, \quad \beta > 0, \quad k \geq 0.$$

This saturation-type incidence rate is biologically motivated, as it captures the effect of population contact saturation when the number of infectives becomes large. It ensures that the transmission rate does not increase indefinitely with I, a behavior more aligned with real epidemic scenarios.

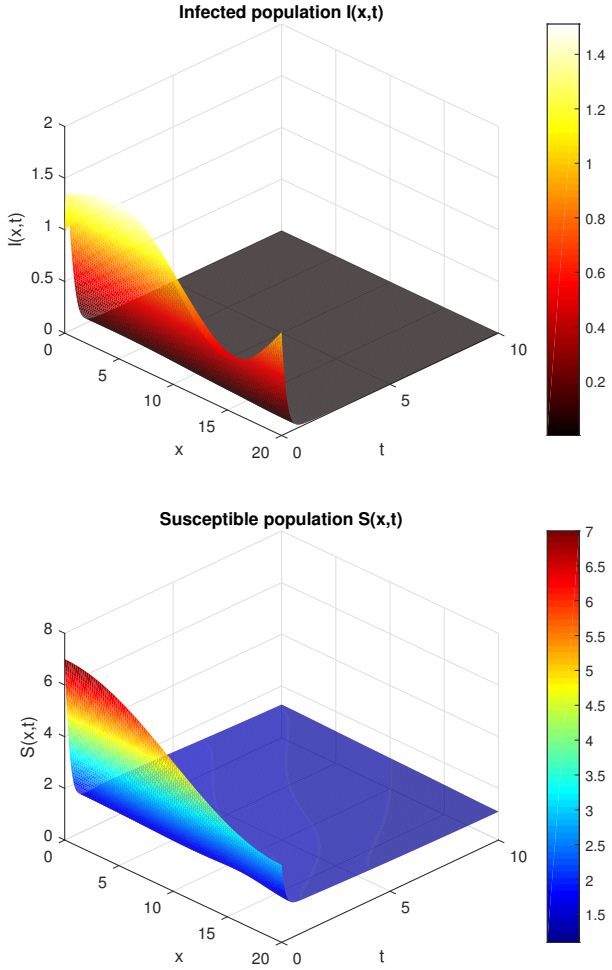


Figure 2. Numerical simulation representing the infected and susceptible populations in the commensurate case $\alpha_1 = \alpha_2 = 0.6$.

The resulting fractional-order SIR model with diffusion takes the following form:

$$\begin{cases} {}^{C}D_t^{\alpha_1} S(x,t) = d_1 \Delta S(x,t) - \dfrac{\beta S(x,t) I(x,t)}{1 + kI(x,t)} + \Lambda - \mu S(x,t), & x \in \Omega,\ t > 0, \\ {}^{C}D_t^{\alpha_2} I(x,t) = d_2 \Delta I(x,t) + \dfrac{\beta S(x,t) I(x,t)}{1 + kI(x,t)} - \sigma I(x,t), & x \in \Omega,\ t > 0, \\ \dfrac{\partial S}{\partial \nu} = \dfrac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0, \\ S(x,0) = S_0(x), \quad I(x,0) = I_0(x), & x \in \Omega, \end{cases} \tag{17}$$

The function $\varphi(I)$ satisfies the following properties for all $I \geq 0$:

$$\varphi(0) = 0, \qquad \varphi(I) > 0, \qquad \varphi'(I) = \frac{\beta}{(1 + kI)^2} > 0.$$

This ensures that the system satisfies the regularity and monotonicity conditions needed for the application of the main results established earlier.

Moreover, one observes the following:

$$0 < \varphi'(I) = \frac{\beta}{(1+kI)^2} \leq \beta,$$

for all $I \geq 0$*, which guarantees the boundedness of the infection rate derivative.*

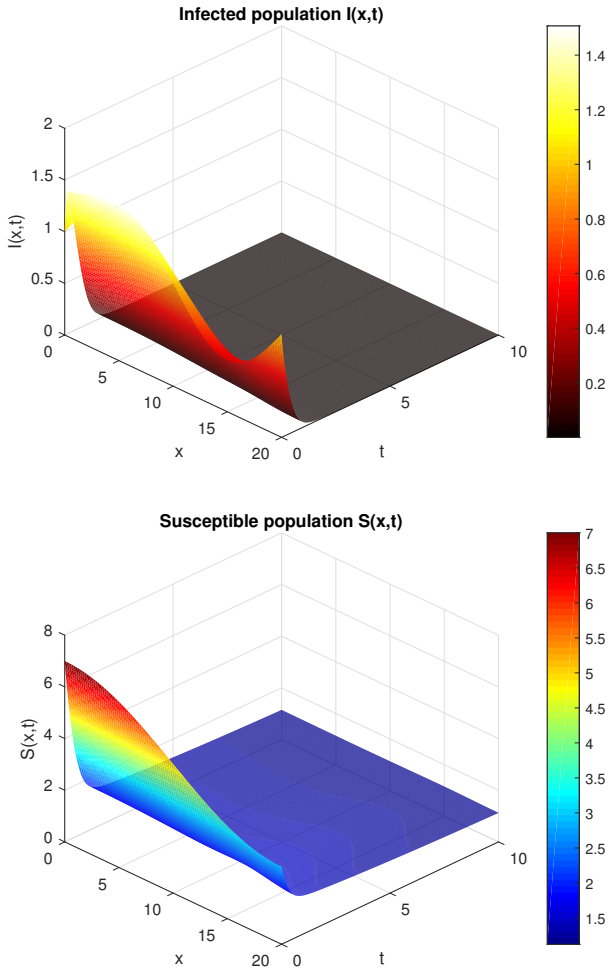


Figure 3. Numerical simulation representing the infected and susceptible populations in the incommensurate case $\alpha_1 = 0.8$ and $\alpha_2 = 0.9$.

The system (17) *admits two constant steady states:*

$$E_0 = \left(\frac{\Lambda}{\mu}, 0\right), \quad \text{and} \quad E^* = (S^*, I^*),$$

where E_0 *is the disease-free equilibrium, and* E^* *is the endemic equilibrium, defined as follows:*

$$I^* = \frac{\mu}{\lambda\beta - k\mu}, \quad S^* = \frac{\sigma(1+kI^*)}{\beta}, \quad \text{with } R_0 = \frac{\lambda\beta}{\mu\sigma}.$$

The existence and stability of these equilibria depend on the value of the basic reproduction number R_0*. Specifically, the following can be said:*

- *If* $R_0 = \dfrac{\lambda\beta}{\mu\sigma} > 1$*, then the endemic equilibrium* E^* *exists and is asymptotically stable for all* $\alpha_1, \alpha_2 \in (0, 1]$.

- *Conversely, if $R_0 < 1$, then the disease-free equilibrium E_0 is globally asymptotically stable regardless of the value of α_1 and α_2.*

These conclusions follow directly from Proposition 3, extending classical ordinary differential equation (ODE) results to the fractional and spatially distributed framework. This behavior is clearly illustrated in Figures 4–6. The example demonstrates that the incorporation of fractional derivatives and diffusive terms preserves the qualitative dynamics of the standard model, while simultaneously providing a richer modeling framework capable of capturing memory effects and spatial heterogeneity. Moreover, the inclusion of a saturated incidence rate introduces additional nonlinear effects that are both biologically meaningful and mathematically tractable within the proposed theoretical setting.

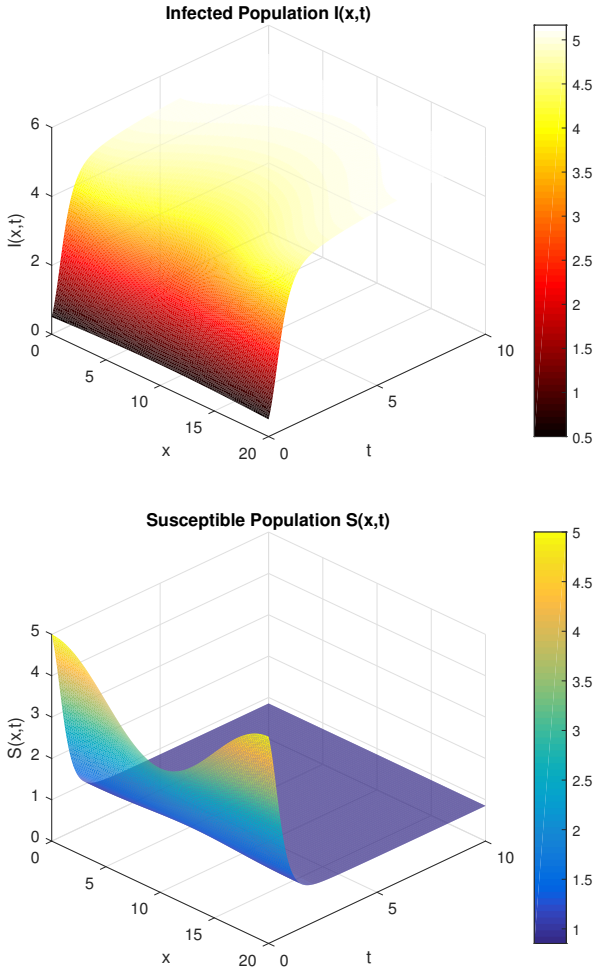


Figure 4. Numerical simulation representing the infected and susceptible populations in the integer case.

Example 3. *We now turn to a third illustrative example: a fractional reaction–diffusion system arising as an extension of the classical epidemic model introduced in [45,46]. This system is another particular case of the general framework described in Equations (9)–(11), featuring a nonlinear incidence rate of the following form:*

$$\varphi(I) = \frac{\beta k I}{1+I}, \qquad \text{with } \beta > 0,\ k > 0.$$

This incidence function models a saturating contact rate dependent on both infection and behavioral response. It reflects the fact that as infections grow, the contact intensity per individual may saturate due to behavioral changes or healthcare limitations.

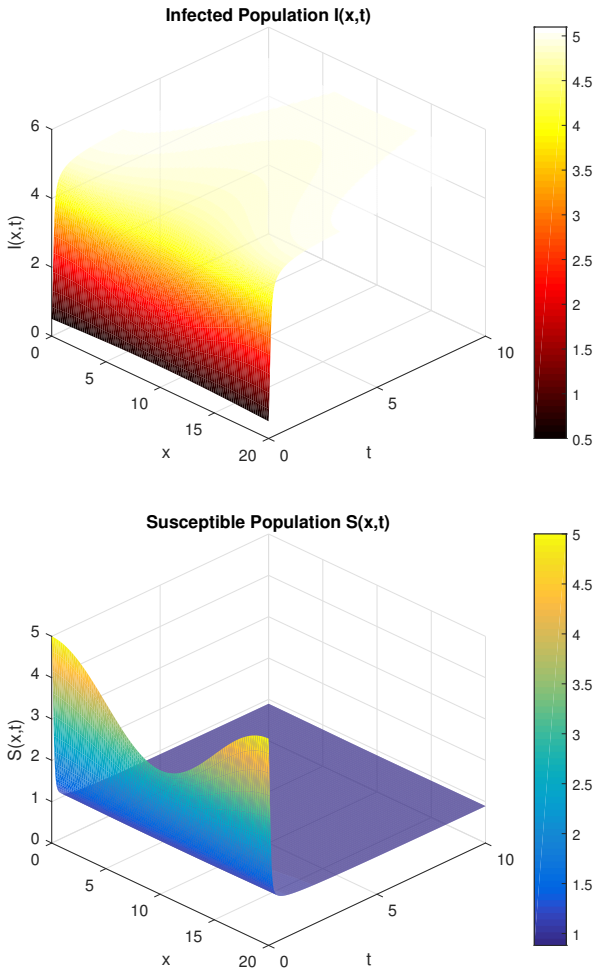


Figure 5. Numerical simulation representing the infected and susceptible populations in the fractional commensurate case.

The resulting fractional reaction–diffusion system is formulated as follows:

$$\begin{cases} {}^{C}D_t^{\alpha_1} S(x,t) = d_1 \Delta S(x,t) - \dfrac{\lambda k I(x,t)}{1+I(x,t)} S(x,t) + \Lambda - \mu S(x,t), & x \in \Omega,\ t > 0, \\ {}^{C}D_t^{\alpha_2} I(x,t) = d_2 \Delta I(x,t) + \dfrac{\lambda k I(x,t)}{1+I(x,t)} S(x,t) - \sigma I(x,t), & x \in \Omega,\ t > 0, \\ \dfrac{\partial S}{\partial \nu} = \dfrac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega,\ t > 0, \\ S(x,0) = S_0(x), \quad I(x,0) = I_0(x), & x \in \Omega, \end{cases} \tag{18}$$

The function $\varphi(I)$ satisfies the following properties for all $I \geq 0$:

$$\varphi(0) = 0, \qquad \varphi(I) > 0, \qquad \varphi'(I) = \frac{\beta k(1+I) - \beta k I}{(1+I)^2} = \frac{\beta k}{(1+I)^2} > 0,$$

and clearly,

$$\varphi'(I) \leq \beta k.$$

Hence, the nonlinear incidence rate is smooth and bounded and satisfies the required monotonicity and Lipschitz conditions.

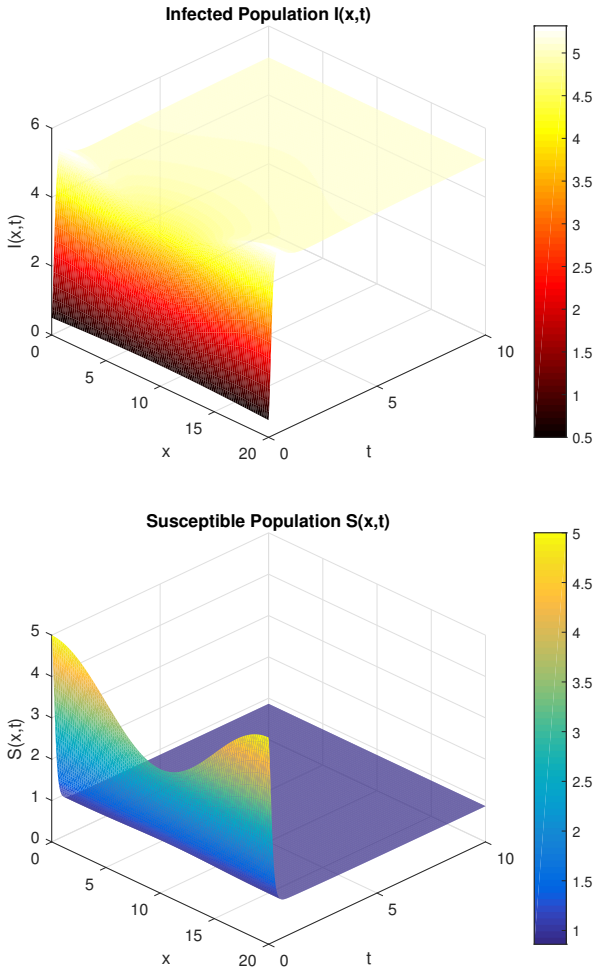


Figure 6. Numerical simulation representing the infected and susceptible populations in the fractional incommensurate case.

The system admits two constant equilibria:

- *The disease-free equilibrium:* $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$.
- *The following endemic equilibrium (when it exists):*

$$E^* = (S^*, I^*), \quad \text{where} \quad I^* = \frac{\mu}{\lambda\beta k - \mu}, \quad S^* = \frac{\sigma(1 + I^*)}{\lambda k I^*}.$$

The condition for the existence of the endemic equilibrium is governed by the basic reproduction number:

$$R_0 = \frac{\lambda\beta k\Lambda}{\mu\sigma}, \quad \text{with} \quad I^* > 0 \iff R_0 > 1.$$

Thus,

- *If* $R_0 > 1$, *then* E^* *exists and is asymptotically stable for any* $\alpha \in (0, 1]$.
- *If* $R_0 < 1$, *the only equilibrium is* E_0, *which is asymptotically stable.*

These results are consistent with the general stability conditions given in Proposition 3 and confirm the robustness of the fractional incommensurate framework in reproducing well-known threshold dynamics.

Figure 7 provides numerical simulations corresponding to this third example. The figure considers a set of parameters and fractional orders $\alpha_1 = 0.6$ and $\alpha_2 = 0.5$, allowing us to visualize the effect of memory and spatial diffusion on disease spread.

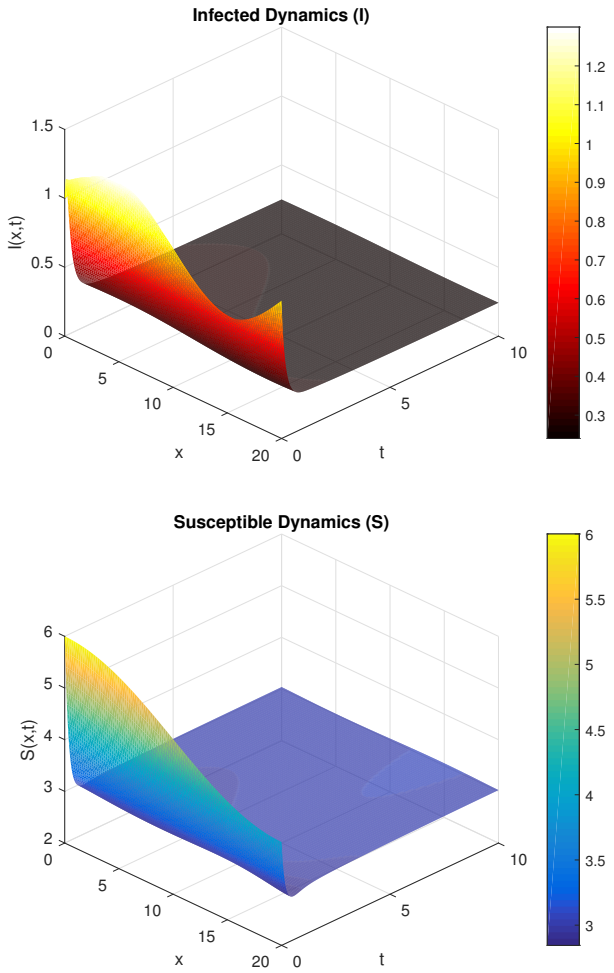


Figure 7. Numerical simulation representing the infected and susceptible populations in the incommensurate case $\alpha_1 = 0.6$ and $\alpha_2 = 0.5$.

These numerical results reinforce the theoretical findings, illustrating the interplay between incommensurate fractional memory, saturation, and spatial diffusion. They also underline the flexibility of the incommensurate fractional reaction–diffusion model to accommodate complex epidemiological behaviors not captured by classical models.

The numerical simulations confirm the theoretical predictions derived from the fractional stability criterion. As the fractional orders vary, the eigenvalue angle condition identifies transitions between persistence and extinction, which are directly observed in the time–space simulations. This coherence validates both the analytical framework and the numerical approach.

Relation to Empirical Epidemic Patterns.

Although incommensurate fractional orders are not typically measured directly in epidemiological datasets, the qualitative behavior of our model aligns with observed HIV/AIDS persistence patterns. Specifically, the slow decay of infected populations and long-memory effects in contact dynamics mirror the fractional-order stability transitions derived here. Previous studies of HIV dynamics have also reported sub-exponential spreading and long-term persistence, behaviors that are naturally reproduced through fractional memory.

6. Conclusions

This study shed light on the intricate role of memory and spatial diffusion in the transmission dynamics of infectious diseases. By introducing a nonlinear incommensurate fractional-order SI model, we captured the key aspects of real-world epidemics that are often overlooked in classical models—namely, delayed responses, heterogeneous time scales, and nonlocal interactions. The stability conditions derived locally are sensitive to the interplay between fractional orders, nonlinear incidence functions, and diffusion coefficients. Numerical experiments validate the theoretical predictions and emphasize the influence of memory in moderating the epidemic's progression. This work not only enriches the theoretical framework of fractional epidemiology but also lays the groundwork for more robust and realistic modeling tools in public health strategy and decision-making. Future directions include extending the model to account for vaccination, stochastic effects, and multi-strain dynamics within the same fractional setting.

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