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Analysis of a mixed fractional–classical reaction–diffusion epidemic model with nonlocal asymptomatic transmission

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Abstract

We study a mixed fractional–classical reaction–diffusion epidemic model with susceptible, asymptomatic infected, symptomatic infected, and recovered populations. The model combines regional fractional diffusion of the susceptible and asymptomatic infected populations, each with its own fractional exponent, with classical diffusion of the remaining populations, and incorporates spatial heterogeneity, asymptomatic transmission, and periodic intermittent treatment. Using semigroup theory and L^p-energy estimates, we prove the global well-posedness and uniform boundedness of solutions and establish sufficient conditions for convergence to the disease-free state. Numerical simulations complement the theoretical analysis by illustrating the spatiotemporal dynamics of the model and revealing the combined effects of regional fractional diffusion, spatial heterogeneity, nonlocal asymptomatic transmission, and intermittent treatment on disease propagation and persistence.

Keywords: Fractional diffusion; mixed fractional–classical epidemic model; asymptomatic transmission; intermittent treatment; global existence and asymptotic behavior.

2020 Mathematics Subject Classification: 35R11, 35K57, 92D30, 35R09, 47G20, 35K91.

1 Introduction and Main Results

1.1 Introduction

Mathematical modeling has become an indispensable tool for understanding the transmission dynamics of infectious diseases and evaluating the effectiveness of intervention strategies. Since the pioneering work of Kermack and McKendrick [29], compartmental epidemic models have provided a quantitative framework for investigating disease spread at the population level. In its classical form, the susceptible–infected–recovered (SIR) model divides the population into epidemiological classes and describes disease transmission through a system of ordinary differential equations. Such models have been extensively generalized to incorporate demographic effects, vaccination, latency, treatment, and behavioral responses; see, for example, [3, 12, 16, 26, 40].

Although ODE epidemic models successfully capture temporal disease dynamics, they neglect spatial movement and environmental heterogeneity. In realistic settings, disease transmission is influenced by geographic structure, population mobility, transportation networks, and spatial variability in healthcare resources. To account for these effects, reaction–diffusion epidemic models governed by partial differential equations have been widely developed. In these models, diffusion terms describe random movement of individuals, while nonlinear reaction terms represent local disease transmission and recovery processes. Classical SIR reaction–diffusion systems (RDS) have been extensively studied in the literature with respect to well-posedness, stability, persistence, traveling wave solutions, spatial pattern formation, optimal control, and numerical simulations, as well as applications to a wide range of infectious diseases [37, 42, 43, 46, 52, 53] and the references therein.

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Despite their success, local or regular or classical diffusion models may fail to accurately describe movement patterns occurring over large spatial scales. Modern transportation systems, migration, commuting networks, and long-distance travel can generate interactions far beyond local neighborhoods. Consequently, the assumption that individuals interact only with nearby locations may be inadequate in many biological and epidemiological settings. To overcome this limitation, nonlocal diffusion operators have been introduced to account for long-range dispersal and spatial interactions. Unlike the classical Laplacian, nonlocal operators allow the state at a given location to depend on information from distant regions, thereby providing a more realistic description of movement processes.

As a result, nonlocal diffusion equations and systems have attracted considerable attention in recent years. Their analytical, qualitative, and numerical properties have been extensively investigated in a variety of scientific contexts; see, for example, [2,4,10,11,18–20,23] and the references therein.

The incorporation of nonlocal diffusion into epidemiological models has led to a growing interest in nonlocal epidemic systems. Such models provide a natural framework for describing disease transmission in populations where movement and interactions occur over large spatial scales. In contrast to classical reaction–diffusion models, nonlocal epidemic models can capture long-range dispersal, transportation networks, migration, and other mechanisms that generate spatial interactions beyond nearest-neighbor movement. As a result, various nonlocal epidemic models have been proposed and analyzed to investigate disease persistence, spreading speeds, traveling waves, and control strategies [15,32,36,50,51,54–58].

Fractional diffusion operators constitute an important class of nonlocal dispersal mechanisms and have been widely used to model anomalous diffusion and long-range movement. Unlike the classical Laplacian, fractional diffusion allows individuals to perform jumps over large spatial distances, thereby capturing dispersal processes associated with Lévy flights and superdiffusive transport. Motivated by these properties, fractional reaction–diffusion epidemic models have been proposed to study the spatial transmission of infectious diseases in heterogeneous environments. Recent investigations have addressed questions concerning well-posedness, persistence, spreading dynamics, traveling waves, and optimal control for fractional epidemic systems; see, for example, [5,6,9,25,28,30,48] and the references therein. These works highlight the significant influence of fractional dispersal on epidemic dynamics and further motivate the development of nonlocal epidemiological models. Fractional diffusion operators therefore provide a natural framework for describing long-range dispersal in epidemic systems.

Another important characteristic of many infectious diseases is the presence of asymptomatic transmission. For diseases such as COVID–19, influenza, dengue fever, and several respiratory infections, infected individuals may transmit the disease without exhibiting noticeable symptoms. Since asymptomatic individuals are less likely to be detected, isolated, or treated, they can contribute significantly to disease spread while remaining largely hidden from public health surveillance. In contrast, symptomatic individuals are more likely to seek medical attention and receive treatment, resulting in different transmission and recovery dynamics. Consequently, epidemic models that distinguish between asymptomatic and symptomatic infected populations provide a more realistic framework for understanding disease progression and evaluating intervention strategies. Such models have been widely employed to investigate the effects of hidden transmission pathways and their implications for disease control [1,8,22,34,39,47].

In addition to distinguishing between asymptomatic and symptomatic infections, it is also important to account for the nonlocal nature of disease transmission. Human mobility, commuting, and social interactions frequently allow susceptible individuals to be exposed to infectious asymptomatic individuals beyond their immediate surroundings. To capture this phenomenon, we model the asymptomatic force of infection by a nonlocal Gaussian convolution operator, which describes transmission through spatial interactions over a finite neighborhood rather than purely local contacts. This nonlocal incidence mechanism provides a more realistic representation of hidden disease transmission and complements the regional fractional diffusion process used to model long-range dispersal.

Treatment and public health interventions also play a fundamental role in controlling disease outbreaks. In practice, treatment efforts are frequently implemented in a periodic or intermittent manner due to limited medical resources, seasonal intervention programs, or public health policies. Such situations naturally lead to time-dependent treatment mechanisms. Mathematical models incorporating periodic treatment have been shown to provide valuable insight into the effectiveness of intervention strategies and their influence on disease control [24,31,45].

Motivated by the above considerations, we propose a spatial epidemic model consisting of suscepti-

ble, asymptomatic infected, symptomatic infected, and recovered populations. The model incorporates spatially heterogeneous demographic and epidemiological parameters together with a periodic intermittent treatment strategy acting on the symptomatic infected class. To capture both long-range and local or classical movement mechanisms, we assume that the susceptible and asymptomatic infected populations undergo regional fractional diffusion, while the symptomatic infected and recovered populations evolve through classical diffusion. Moreover, the susceptible and asymptomatic infected populations are allowed to diffuse with distinct fractional exponents, thereby accounting for different dispersal characteristics among epidemiological classes. The model further incorporates a nonlocal Gaussian convolution operator in the asymptomatic force of infection to describe spatial transmission between susceptible individuals and infectious asymptomatic individuals across neighboring locations. Consequently, the proposed framework combines heterogeneous fractional diffusion, nonlocal disease transmission, spatial heterogeneity, and intermittent treatment within a unified epidemic model, providing a flexible setting for investigating the spatial spread and control of infectious diseases.

Let $n \in \mathbb{N}$ and let $\Omega \subset \mathbb{R}^n$ be a bounded open subset with smooth boundary $\partial\Omega$, where $\partial\Omega$ is an $(n-1)$-dimensional $C^{2+\varrho}$ manifold for some $\varrho \in (0,1)$, and Ω lies locally on one side of $\partial\Omega$. We consider the following mixed diffusion epidemic model:

$$\begin{cases} S_t(x,t) = d_S(-\Delta)^{\alpha_S} S(x,t) + b(x) - g(x,t)S(x,t) - \mu(x)S(x,t), & (x,t) \in \Omega \times (0,\infty), \\ I_{1_t}(x,t) = d_{I_1}(-\Delta)^{\alpha_{I_1}} I_1(x,t) + \sigma(x)g(x,t)S(x,t) - \Big(\lambda_1(x) + \mu(x)\Big)I_1(x,t), & (x,t) \in \Omega \times (0,\infty), \\ I_{2_t}(x,t) = d_{I_2}\Delta I_2(x,t) + (1-\sigma(x))g(x,t)S(x,t) - \Big(\lambda_2(x) + \mu(x) + \delta(x) + \gamma(x,t)\Big)I_2(x,t), & (x,t) \in \Omega \times (0,\infty), \\ R_t(x,t) = d_R\Delta R(x,t) + \lambda_1(x)I_1(x,t) + \Big(\lambda_2(x) + \gamma(x,t)\Big)I_2(x,t) - \mu(x)R(x,t), & (x,t) \in \Omega \times (0,\infty), \\ \mathcal{N}_{\alpha_S} S = \mathcal{N}_{\alpha_{I_1}} I_1 = 0 & (x,t) \in (\mathbb{R}^n \setminus \Omega) \times (0,\infty), \\ \dfrac{\partial I_2}{\partial \eta} = \dfrac{\partial R}{\partial \eta} = 0, & (x,t) \in \partial\Omega \times (0,\infty), \\ \Big(S(x,0), I_1(x,0), I_2(x,0), R(x,0)\Big) = \Big(S_0(x), I_{1,0}(x), I_{2,0}(x), R_0(x)\Big) \geq 0, & x \in \Omega \end{cases} \tag{1.1}$$

where $S(x,t)$, $I_1(x,t)$, $I_2(x,t)$, and $R(x,t)$ denote the densities of susceptible, asymptomatic infected, symptomatic infected, and recovered individuals, respectively, at location $x \in \Omega$ and time $t > 0$. We assume $S_0, I_{1,0} \in L^\infty(\Omega, \mathbb{R}^2_+)$, $I_{2,0}, R_0 \in C^2(\overline{\Omega}, \mathbb{R}^2_+)$ with $\frac{\partial I_{2,0}}{\partial \eta} = \frac{\partial R_0}{\partial \eta} = 0$ on $\partial\Omega$. The force of infection is given by

$$g(x,t) = \int_\Omega \rho(x,y)\,\beta_1(y)\,I_1(y,t)\,dy + \beta_2(x)\,I_2(x,t), \tag{1.2}$$

where the first term represents the nonlocal transmission due to asymptomatic infected individuals, while the second term accounts for the classicial or regular or local transmission generated by symptomatic infected individuals. The functions $\rho(x,y)$ and the remaining model parameters are described in Table 1.

The symptomatic infected and recovered populations are assumed to undergo classical diffusion subject to homogeneous Neumann boundary conditions, $\frac{\partial I_2}{\partial \eta} = \frac{\partial R}{\partial \eta} = 0$, where η denotes the outward unit normal vector on $\partial\Omega$. These zero-flux conditions ensure that no symptomatic infected or recovered individuals enter or leave the domain through local diffusive movement.

The susceptible and asymptomatic infected populations undergo regional fractional diffusion with different fractional diffusion orders, $\alpha_S \in (0,1)$ and $\alpha_{I_1} \in (0,1)$ respectively. To prevent individuals from entering or leaving the habitat through nonlocal dispersal, we impose the homogeneous nonlocal Neumann boundary condition

$$\mathcal{N}_{\alpha_j} u(x,t) = C_{n,\alpha_j} \int_\Omega \frac{u(y,t) - u(x,t)}{|x-y|^{n+2\alpha_j}}\,dy = 0, \qquad (x,t) \in (\mathbb{R}^n \setminus \Omega) \times (0,\infty), \tag{1.3}$$

where $u = u_j = (u_1, u_2) \equiv (S, I_1)$, $C_{n,\alpha_j} = \frac{4^{\alpha_j}\Gamma\left(\frac{n}{2}+\alpha_j\right)}{\pi^{n/2}|\Gamma(-\alpha_j)|}$, $j \in \{S, I_1\}$, is the normalization constant. Here, Γ denotes the Gamma function,

$$\Gamma(z) = \int_0^\infty t^{z-1}e^{-t}\,dt, \qquad \mathrm{Re}(z) > 0.$$

The condition (1.3) prevents individuals from entering or leaving the habitat through nonlocal dispersal and ensures that all spatial interactions remain confined to the domain Ω.

Consequently, the diffusion process is governed by the regional (or restricted) fractional Laplacian. For $\alpha_j \in (0,1)$, $j \in \{S, I_1\}$, the regional fractional Laplacian admits the following Riesz integral representation:

$$(-\Delta)^{\alpha_j} u(x,t) = C_{n,\alpha_j}\, \text{P.V.} \int_\Omega \frac{u(y,t) - u(x,t)}{|x-y|^{n+2\alpha_j}}\, dy, \qquad (x,y) \in \Omega \times \Omega, \quad t > 0, \tag{1.4}$$

where P.V. denotes the Cauchy principal value and C_{n,α_j} is the normalization constant defined above.

Biologically, the regional fractional Laplacian describes the nonlocal movement of individuals within the spatial domain. Unlike classical diffusion, which represents only local random movement, it permits occasional long-distance dispersal, with the likelihood of movement decreasing as the distance between locations increases. The fractional exponent $\alpha_j \in (0,1)$ governs the degree of nonlocality: smaller values of α_j correspond to heavier-tailed movement patterns and hence more frequent long-range travel.

The fractional Laplacian operator admits the variational characterization associated with the bilinear form

$$\mathcal{E}_{\alpha_j}(w,v) = \frac{C_{n,\alpha_j}}{2} \int_\Omega \int_\Omega \frac{\big(w(y) - w(x)\big)\big(v(y) - v(x)\big)}{|x-y|^{n+2\alpha_j}}\, dx\, dy, \qquad w, v \in H^{\alpha_j}(\Omega),\ j \in \{S, I_1\}$$

where $H^{\alpha_j}(\Omega)$ denotes the fractional Sobolev space of order α_j, $j \in \{S, I_1\}$. The regional fractional Laplacian is a nonnegative self-adjoint operator on $L^2(\Omega)$ associated with the bilinear form $\mathcal{E}_{\alpha_j}$ and generates a species dependent strongly continuous analytic semigroup. Moreover, in the absence of reaction terms, the corresponding diffusion process conserves the total population mass. Further details concerning regional fractional Laplacian, nonlocal Neumann boundary conditions, and related analytical properties can be found in [13, 17].

The constants $d_S, d_{I_1}, d_{I_2}, d_R > 0$ denote the diffusion coefficients of the susceptible, asymptomatic infected, symptomatic infected, and recovered populations, respectively. The epidemiological parameters used in system (1.1) are summarized in Table 1.

Table 1: Description of the epidemiological parameters in system (1.1).

Parameter	Description
$b(x) \geq 0$	Spatially heterogeneous recruitment rate of susceptible individuals
$\beta_1(x) \geq 0$	Transmission rate associated with asymptomatic infected individuals
$\beta_2(x) \geq 0$	Transmission rate associated with symptomatic infected individuals
$\sigma(x) \in [0,1]$	Proportion of newly infected individuals who become asymptomatic
$1 - \sigma(x)$	Proportion of newly infected individuals who become symptomatic
$\mu(x) \geq 0$	Natural mortality rate
$\lambda_1(x) \geq 0$	Recovery rate of asymptomatic infected individuals
$\lambda_2(x) \geq 0$	Recovery rate of symptomatic infected individuals
$\delta(x) \geq 0$	Disease-induced mortality rate of symptomatic infected individuals
$\rho(x,y) \geq 0$	Nonlocal transmission kernel describing the influence of infected individuals at location y on susceptibles at location x.
$\gamma(x,t) \geq 0$	Spatially heterogeneous periodic treatment rate acting on symptomatic infected individuals

The function $\gamma(x,t) \geq 0$ represents a spatially heterogeneous treatment rate and is assumed to be bounded and periodic in time with period T. For any $L \in (0,T)$, there exists a nonnegative function $\gamma_1(x,t)$, positive on a subset $\widehat{\Omega} \subset \Omega$ of positive measure. Moreover, there exist a constant $\gamma_0 > 0$ such that $\gamma_1(x,t) \geq \gamma_0$ in

$(x,t) \in \widehat{\Omega} \times [0,\infty)$, and

$$\gamma(x,t) = \begin{cases} 0, & x \in \Omega, \quad kT \le t < (k+1)T - L, \quad k \in \mathbb{N} \cup \{0\}, \\ \gamma_1(x,t), & x \in \Omega, \quad (k+1)T - L \le t < (k+1)T, \quad k \in \mathbb{N} \cup \{0\}. \end{cases}$$

Thus, treatment is applied intermittently during the final L-units of time in each treatment cycle of length T. During the treatment-free interval $kT \le t < (k+1)T - L$, no symptomatic individuals receive treatment, whereas during the intervention interval $(k+1)T - L \le t < (k+1)T$, treatment is administered at rate $\gamma_1(x,t)$. This formulation reflects periodic intervention strategies arising from limited medical resources, seasonal treatment programs, or public health control policies.

We assume the following notations where all the spatial parameters are continuous and bounded on $\overline{\Omega}$:

$$b^* := \max_{x\in\overline{\Omega}} b(x) \ge 0, \qquad \sigma^* := \max_{x\in\overline{\Omega}} \sigma(x) \in [0,1], \qquad \mu^* := \max_{x\in\overline{\Omega}} \mu(x) \ge 0, \qquad \mu_0 := \min_{x\in\overline{\Omega}} \mu(x) > 0,$$
$$\delta^* := \max_{x\in\overline{\Omega}} \delta(x) \ge 0, \qquad \delta_0 := \min_{x\in\overline{\Omega}} \delta(x) > 0, \qquad \lambda_i^* := \max_{x\in\overline{\Omega}} \lambda_i(x) \ge 0, \quad \lambda_{i,0} := \min_{x\in\overline{\Omega}} \lambda_i(x) > 0,$$
$$\beta_i^* := \max_{x\in\overline{\Omega}} \beta_i(x) \ge 0, \qquad \gamma^* := \max_{(x,t)\in\overline{\Omega}\times[0,T]} \gamma(x,t) \ge 0,$$

for $i = 1, 2$. For convenience, we write the reaction terms in system (1.1) as

$$F = F_i = \begin{cases} f_1(x,t,S,I_1,I_2,R) &= b(x) - g(x,t)S(x,t) - \mu(x)S(x,t), \\ f_2(x,t,S,I_1,I_2,R) &= \sigma(x)g(x,t)S(x,t) - \big(\lambda_1(x) + \mu(x)\big)I_1(x,t), \\ f_3(x,t,S,I_1,I_2,R) &= \big(1-\sigma(x)\big)g(x,t)S(x,t) - \big(\lambda_2(x) + \mu(x) + \delta(x) + \gamma(x,t)\big)I_2(x,t), \\ f_4(x,t,S,I_1,I_2,R) &= \lambda_1(x)I_1(x,t) + \big(\lambda_2(x) + \gamma(x,t)\big)I_2(x,t) - \mu(x)R(x,t). \end{cases} \tag{1.5}$$

From the mathematical perspective, system (1.1) presents several analytical challenges. The coexistence of regional fractional and classical diffusion operators prevents the direct application of many techniques developed for standard reaction–diffusion systems. In particular, the regional fractional Laplacian lacks the strong smoothing properties of the classical Laplacian, making the derivation of uniform *a priori* estimates and the analysis of long-time behavior considerably more delicate. Moreover, the presence of two distinct fractional diffusion exponents, coupled with the nonlinear interactions among the susceptible, asymptomatic infected, symptomatic infected, and recovered populations, together with spatially heterogeneous coefficients and periodic intermittent treatment, further complicates the well-posedness and asymptotic analysis.

To the best of our knowledge, few epidemic models combine regional fractional diffusion with classical diffusion within a unified analytical framework. Unlike many existing fractional epidemic models based on Caputo-type time derivatives or fully fractional dynamical systems, our model employs spatial Riesz-type fractional diffusion through the regional fractional Laplacian while retaining classical first-order temporal dynamics. Furthermore, allowing distinct fractional exponents for the susceptible and asymptomatic infected populations enables the model to capture heterogeneous long-range mobility patterns among different epidemiological classes, thereby providing a more flexible description of disease spread.

The proposed model also incorporates nonlocal asymptomatic transmission, periodic intermittent treatment, and spatially heterogeneous demographic and epidemiological parameters. These features are relevant for diseases such as COVID–19, influenza, and dengue fever, where hidden transmission pathways, heterogeneous mobility, and periodic intervention strategies significantly influence epidemic dynamics. The interaction between regional fractional diffusion, classical diffusion, and intermittent treatment therefore yields a mathematically rich and biologically realistic framework for studying disease propagation in heterogeneous environments.

We conclude this section by stating the main analytical results concerning the global well-posedness and asymptotic behavior of solutions to system (1.1).

1.2 Statement of the Main Results

Let us assume that

(D) (Diffusivity) $d_S, d_{I_1}, d_{I_2}, d_R > 0$, $\rho \in C(\overline{\Omega} \times \overline{\Omega}, \mathbb{R}_+)$ such that $\rho(x,y) = \rho(y,x)$ for all $x, y \in \Omega$ and there exists a $\Lambda > 0$ such that

$$\int_\Omega \rho(x,y)\, dy \le \Lambda < \infty. \tag{1.6}$$

(Qp) (Quasi-positivity) The reaction vector field F is quasi-positive; that is, for every $(x,t) \in \overline{\Omega} \times [0,\infty)$ and every $(S, I_1, I_2, R) \in \mathbb{R}^4_+$, we have $f_1(x,t,0,I_1,I_2,R) \ge 0$, $f_2(x,t,S,0,I_2,R) \ge 0$, $f_3(x,t,S,I_1,0,R) \ge 0$, $f_4(x,t,S,I_1,I_2,0) \ge 0$.

Under (D), together with the boundedness assumptions on $\beta_i(x)$, $i = 1,2$, the nonlocal force of infection $g(x,t)$ defines a bounded linear operator with respect to (I_1, I_2). Consequently, the incidence term is locally Lipschitz on bounded subsets of the positive cone. Hence, the reaction vector field is locally Lipschitz with respect to the state variables.

Additionally, biologically, $\rho(x,y)$ represents the transmission intensity from infected individuals located at y to susceptible individuals at x. The continuity of ρ ensures that transmission varies smoothly across the spatial domain, the symmetry assumes isotropic spatial interactions, and the uniform bound guarantees that the total infection pressure experienced at each location remains finite.

Theorem 1 *Assume that (D) and (Qp) hold. Let $(S_0, I_{1,0}) \in L^\infty(\Omega; \mathbb{R}^2_+)$, $(I_{2,0}, R_0) \in C^2(\overline{\Omega}; \mathbb{R}^2_+)$ with $\frac{\partial I_{2,0}}{\partial \eta} = \frac{\partial R_0}{\partial \eta} = 0$ on $\partial\Omega$. Then there exists a unique classical global component-wise nonnegative solution $(S(x,t), I_1(x,t), I_2(x,t), R(x,t))$ to system (1.1). Moreover, each component remains uniformly bounded in the sup norm for all time.*

Theorem 2 *Assume that the hypotheses of Theorem 1, Lemma 2.1 and 2.2 hold, and let S^* be the disease-free steady state satisfying (2.15). If $S_0(x) \le S^*(x)$ in Ω, then the unique global solution of system (1.1) satisfies*

$$\lim_{t\to\infty} \|I_1(\cdot,t)\|_{\infty,\Omega} = \lim_{t\to\infty} \|I_2(\cdot,t)\|_{\infty,\Omega} = \lim_{t\to\infty} \|S(\cdot,t) - S^*\|_{\infty,\Omega} = 0.$$

The remainder of the paper is organized as follows. In Section 2, we establish the global existence, uniqueness, positivity, uniform boundedness, and asymptotic behavior of solutions to system (1.1). Section 3 is devoted to numerical simulations. Finally, concluding remarks are presented in Section 4.

2 Proof of Main Theorems

It suffices to analyze S and I_j, since the equation for R is linear with forcing term depending only on I_j and can uniquely be recovered from the corresponding linear parabolic equation for $j = 1,2$.

2.1 Proof of Theorem 1

We will use the following semigroup theory [44] to prove Theorem 1. Let us define, $X := L^2(\Omega)$, $X_+ := \{z \in X : z \ge 0 \text{ a.e. } \Omega\}$. We also define the operators $A_j w = d_j(-\Delta)^{\alpha_j} w$, $j \in \{S, I_1\}$ and $A_{I_2} w = -d_{I_2}\Delta w$ with domains

$$D(A_j) = \Big\{w \in H^{\alpha_j}(\Omega) : A_j w \in L^2(\Omega),\ \mathcal{N}_{\alpha_j} w = 0 \text{ on } \mathbb{R}^n \setminus \Omega\Big\},$$
$$D(A_{I_2}) = \Big\{w \in W^{2,2}(\Omega) : A_{I_2} w \in L^2(\Omega),\ \frac{\partial w}{\partial \eta} = 0 \text{ on } \partial\Omega\Big\}.$$

The fractional Sobolev space is defined by

$$H^{\alpha_j}(\Omega) := \Big\{w \in L^2(\Omega) : \int_\Omega \int_\Omega \frac{|w(y) - w(x)|^2}{|x-y|^{n+2\alpha_j}}\, dx\, dy < \infty\Big\}, \quad 0 < \alpha_j < 1, \quad j \in \{S, I_1\}$$

Its norm is given by

$$\|w\|^2_{H^{\alpha_j}(\Omega)} := \|w\|^2_{L^2(\Omega)} + \int_\Omega \int_\Omega \frac{|w(y) - w(x)|^2}{|x-y|^{n+2\alpha_j}}\, dx\, dy = \|w\|^2_{L^2(\Omega)} + [w]^2_{H^{\alpha_j}(\Omega)}, \quad j \in \{S, I_1\}$$

where

$$[w]^2_{H^{\alpha_j}(\Omega)} := \int_\Omega \int_\Omega \frac{|w(y) - w(x)|^2}{|x - y|^{n+2\alpha_j}}\, dx\, dy \tag{2.1}$$

denotes the Gagliardo seminorm [41, 49].

For the fractional diffusion operator A_j, consider the bilinear form

$$\mathcal{E}_{\alpha_j}(u, v) = \frac{d_j C_{n,\alpha_j}}{2} \int_\Omega \int_\Omega \frac{(u(y) - u(x))(v(y) - v(x))}{|x - y|^{n+2\alpha_j}}\, dx\, dy, \quad j \in \{S, I_1\} \tag{2.2}$$

defined on $H^{\alpha_j}(\Omega) \times H^{\alpha_j}(\Omega)$. The form bilinear $\mathcal{E}_{\alpha_j}$ is symmetric, densely defined, closed, and nonnegative on $L^2(\Omega)$ for $j \in \{S, I_1\}$. Hence, by the Kato–Friedrichs representation theorem, there exists a unique self-adjoint, nonnegative operator A_j associated with $\mathcal{E}_{\alpha_j}$. In particular, A_j is sectorial and generates an analytic semigroup $T_j = \{T_j(t) : t \geq 0\}$ for $j \in \{S, I_1\}$ on $L^2(\Omega)$ [14].

The operator A_{I_2} is self-adjoint, nonnegative, and densely defined on $L^2(\Omega)$ with domain $D(A_{I_2})$. Moreover, A_{I_2} is sectorial and therefore generates an analytic semigroup $T_{I_2} = \{T_{I_2}(t) : t \geq 0\}$ on $L^2(\Omega)$ [27].

Introducing the product space $\mathbb{X} = L^2(\Omega) \times L^2(\Omega) \times L^2(\Omega)$, the diffusion part of system (1.1) can be represented by the block-diagonal operator

$$\mathcal{A} := diag\Big\{A_S, A_{I_1}, A_{I_2}\Big\} \text{ with domain } \; D(\mathcal{A}) = D(A_S) \times D(A_{I_1}) \times D(A_{I_2}).$$

Since each diagonal operator generates an analytic semigroup on $L^2(\Omega)$, it follows from standard product semigroup theory that $\mathcal{A}$ generates an analytic C_0-semigroup on $\mathbb{X}$,see, for instance, [7, 44]. Consequently, system (1.1) may be formulated as a semilinear evolution equation on $\mathbb{X}$, where the coupling is introduced solely through the reaction terms.

Proof of Theorem (1)**:** With the above setting, standard semilinear evolution theory [44] guarantees that system (1.1) admits a unique maximal solution $(S, I_1, I_2) \in C([0, T_m); \mathbb{X})$, defined on a maximal interval $[0, T_m)$, where $T_m \in (0, \infty]$. Moreover, the solution satisfies the variation-of-constants formulas

$$u(x, t) = T_u(t)u_0(x) + \int_0^t T_u(t - \tau)F(x, \tau, S, I_1, I_2, R)d\tau,$$

for $u \in \{S, I_1, I_2\}$. Since the reaction terms satisfy the quasi-positivity condition (Qp), standard comparison arguments imply that the maximal solution remains component-wise nonnegative. Thus,

$$S(x, t) \geq 0, \qquad I_1(x, t) \geq 0, \qquad I_2(x, t) \geq 0,$$

for all $x \in \Omega$ and $t \in [0, T_m)$.

To establish global existence, it suffices to rule out finite-time blow-up. More precisely, the continuation principle implies that either $T_m = \infty$ or

$$\limsup_{t \to T_m^-} \Big\{\|S(\cdot, t)\|_{\infty,\Omega} + \|I_1(\cdot, t)\|_{\infty,\Omega} + \|I_2(\cdot, t)\|_{\infty,\Omega}\Big\} = \infty. \tag{2.3}$$

Consequently, global existence follows once it is shown that the solution remains uniformly bounded in $L^\infty(\Omega)$ on every finite time interval.

Now, if we define $A_{SS}(w) = d_S(-\Delta)^\alpha w - \mu_0 w$, then $A_{SS}(w)$ generates $T_{SS}(t)$ on $L^2(\Omega)$. Also, since S, I_1, I_2, ρ, β_i^*, $i = 1, 2$ are nonnegative then the first equation of (1.1) yields

$$\begin{aligned} S_t(x, t) &= d_S(-\Delta)^\alpha S(x, t) + b(x) - g(x, t)S(x, t) - \mu(x)S(x, t) \\ &\leq d_S(-\Delta)^\alpha S(x, t) + b^* - \mu_0 S(x, t) \end{aligned}$$

therefore, we get

$$\begin{aligned} S(x, t) &\leq T_{SS}(t)S_0(x) + b^* \int_0^t T_{SS}(t - \tau)\, d\tau \leq \|T_{SS}(t)S_0\|_{\infty,\Omega} + b^* \int_0^t \|T_{SS}(t - \tau)\|_{\infty,\Omega}\, d\tau \\ &\leq C_1, \quad t > 0, \end{aligned} \tag{2.4}$$

where $C_1 := \max\left\{\|S_0\|_{\infty,\Omega}, \frac{b^*}{\mu_0}\right\}$.

Now, adding the second and third equation of system (1.1) and then integrating over Ω we get,

$$\begin{aligned}\frac{d}{dt}\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dx &= d_{I_1}\int_\Omega (-\Delta)^{\alpha_{I_1}} I_1(x,t)dx + d_{I_2}\int_\Omega \Delta I_2(x,t)dx + \int_\Omega g(x,t)S(x,t)dx\\ &\quad - \int_\Omega \Big(\lambda_1(x)+\mu(x)\Big)I_1(x,t)dx - \int_\Omega \Big(\lambda_2(x)+\mu(x)+\delta(x)+\gamma(x,t)\Big)I_2(x,t)dx\\ &\le \zeta\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dx - (\lambda_{1,0}+\mu_0)\int_\Omega I_1(x,t)dx - (\lambda_{2,0}+\mu_0)\int_\Omega I_2(x,t)dx\\ &\quad - (\delta_0+\gamma_0)\int_\Omega I_2(x,t)dx\end{aligned}$$

where we used the Green's formula with boundary condition to get, $d_{I_2}\int_\Omega \Delta I_2(x,t)dx = 0$ and $d_{I_1}\int_\Omega (-\Delta)^{\alpha_{I_1}} I_1(x,t)dx = -\int_{\mathbb{R}^n\setminus\Omega} d_{I_1}\mathcal{N}_{\alpha_{I_1}} I_1(x,t)dx = 0$. We considered $\zeta = \max\left\{\Lambda\beta_1^*, \beta_2^*\right\} \ge 0$ to get

$$\begin{aligned}\int_\Omega g(x,t)S(x,t)dx &\le C_1\int_\Omega g(x,t)dx = C_1\left(\int_\Omega\int_\Omega \rho(x,y)\beta_1(y)I_1(y,t)dy + \int_\Omega \beta_2(x)I_2(x,t)dx\right)\\ &\le C_1\left(\int_\Omega \beta_1(y)I_1(y,t)dy\int_\Omega \rho(x,y)dx + \int_\Omega \beta_2(x)I_2(x,t)dx\right)\\ &\le C_1\left(\Lambda\int_\Omega \beta_1(y)I_1(y,t)dy + \int_\Omega \beta_2(x)I_2(x,t)dx\right) \le C_1\left(\Lambda\beta_1^*\int_\Omega I_1(x,t)dx + \beta_2^*\int_\Omega I_2(x,t)dx\right)\\ &\le C_1\zeta\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dx \qquad (2.5)\end{aligned}$$

So, we can write,

$$\frac{d}{dt}\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dx \le \Big(C_1\zeta - \xi\Big)\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dx \qquad (2.6)$$

where we considered $\xi = \min\{\lambda_{1,0}+\mu_0, \lambda_{2,0}+\mu_0, \delta_0+\gamma_0\} > 0$. Then using Gröwall's inequality we get

$$\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dx \le M,\ \forall\ t > 0, \qquad (2.7)$$

where $M := \int_\Omega \Big(I_{1,0}(x)+I_{2,0}(x)\Big)dx$. Now, for $0 \le \tau < T < T_m$ and $2 \le p < \infty$ we can multiply both sides of the second equation of system (1.1) with $I_1(x,t)^{p-1}$ and then integrating over Ω to get,

$$\begin{aligned}\frac{1}{p}\frac{d}{dt}\int_\Omega I_1(x,t)^p dx &= d_{I_1}\int_\Omega I_1(x,t)^{p-1}(-\Delta)^{\alpha_{I_1}} I_1(x,t)dx + \int_\Omega I_1(x,t)^{p-1}\sigma(x)g(x,t)S(x,t)dx - \int_\Omega \Big(\lambda_1(x)+\mu(x)\Big)I_1(x,t)^p dx\\ &\le C_1\sigma^*\int_\Omega I_1(x,t)^{p-1}g(x,t)dx - (\lambda_{1,0}+\mu_0)\int_\Omega I_1(x,t)^p dx\end{aligned}$$

here we considered $K(x,y) = \frac{C_{n,\alpha_{I_1}}}{|x-y|^{n+2\alpha_{I_1}}} = K(y,x) > 0$ to get,

$$\begin{aligned}d_{I_1}\int_\Omega I_1(x,t)^{p-1}(-\Delta)^{\alpha_{I_1}} I_1(x,t)dx &= -\frac{d_{I_1}}{2}\int_\Omega\int_\Omega (I_1(y,t)^{p-1} - I(x,t)^{p-1})K(x,y)(I_1(y,t)-I_1(x,t))dydx\\ &\le -\frac{2d_{I_1}(p-1)}{p^2}\int_\Omega\int_\Omega K(x,y)\left(I_1(y,t)^{p/2} - I_1(x,t)^{p/2}\right)^2 dydx\\ &\le 0.\end{aligned}$$

where we used the following inequality

$$\left(a^{p-1}-b^{p-1}\right)(a-b) \ge \frac{4(p-1)}{p^2}\left(a^{p/2}-b^{p/2}\right)^2, \qquad a,b \ge 0,\ p > 1.$$

So, we have,

$$\frac{d}{dt}\int_\Omega I_1(x,t)^p dx \le pC_1\sigma^* \int_\Omega I_1(x,t)^{p-1} g(x,t)dx - p(\lambda_{1,0}+\mu_0)\int_\Omega I_1(x,t)^p dx$$

However, the definition of ρ in (D) implies that there exists a constant $\Lambda_\infty := \|\rho\|_{L^\infty(\Omega\times\Omega)} > 0$ such that $0 \le \rho(x,y) \le \Lambda_\infty$, for any $(x,y) \in \overline{\Omega}\times\overline{\Omega}$. Consequently, we obtain

$$\begin{aligned}
pC_1\sigma^* \int_\Omega I_1(x,t)^{p-1} g(x,t)dx &= pC_1\sigma^*\left(\int_\Omega\int_\Omega I_1(x,t)^{p-1}\rho(x,y)\beta_1(y)I_1(y,t)dydx + \int_\Omega \beta_2(x) I_1^{p-1} I_2 dx\right)\\
&\le pC_1\sigma^*\Lambda_\infty\beta_1^*\int_\Omega\int_\Omega I_1(x,t)^{p-1}I_1(y,t)dydx + pC_1\sigma^*\beta_2^*\int_\Omega I_1(x,t)^{p-1}I_2(x,t)dx\\
&\le pC_1\sigma^*\Lambda_\infty\beta_1^*|\Omega|\int_\Omega I_1(x,t)^p dx + (p-1)C_1\sigma^*\beta_2^*\int_\Omega I_1(x,t)^p dx + C_1\sigma^*\beta_2^*\int_\Omega I_2(x,t)^p dx\\
&\le pC_1\sigma^*\Lambda_\infty\beta_1^*|\Omega|\int_\Omega I_1(x,t)^p dx + pC_1\sigma^*\beta_2^*\int_\Omega I_1(x,t)^p dx + C_1\sigma^*\beta_2^*\int_\Omega I_2(x,t)^p dx\\
&\le pC_1\sigma^*\zeta^*\int_\Omega I_1(x,t)^p dx + C_1\sigma^*\beta_2^*\int_\Omega I_2(x,t)^p dx \qquad \left[\zeta^* := \Lambda_\infty\beta_1^*|\Omega| + \beta_2^*\right]\\
&\le pC_1\sigma^*\zeta^*\int_\Omega \Big(I_1(x,t)^p + I_2(x,t)^p\Big)dx
\end{aligned}$$

where $\beta_2^* \le \zeta^* \le p\zeta^*$ for any $2 \le p < \infty$ and we used Young's inequality to get

$$\int_\Omega I_1^{p-1} I_2\, dx \le \frac{p-1}{p}\int_\Omega I_1^p\, dx + \frac{1}{p}\int_\Omega I_2^p\, dx, \quad \forall\, p > 1$$

therefore, we can write,

$$\frac{d}{dt}\int_\Omega I_1(x,t)^p dx \le pC_1\sigma^*\zeta^*\int_\Omega \Big(I_1(x,t)^p + I_2(x,t)^p\Big)dx - p(\lambda_{1,0}+\mu_0)\int_\Omega I_1(x,t)^p dx$$

Similarly, for I_2 we can obtain,

$$\frac{d}{dt}\int_\Omega I_2(x,t)^p dx \le pC_1\zeta^*\int_\Omega \Big(I_1(x,t)^p + I_2(x,t)^p\Big)dx - p(\lambda_{2,0}+\mu_0)\int_\Omega I_2(x,t)^p dx - p(\delta_0+\gamma_0)\int_\Omega I_2(x,t)^p dx,$$

where the homogeneous Neumann boundary condition of I_2 gives us

$$\int_\Omega I_2^{p-1}\Delta I_2\, dx = -\int_\Omega \nabla(I_2^{p-1})\cdot\nabla I_2\, dx = -(p-1)\int_\Omega I_2^{p-2}|\nabla I_2|^2\, dx \le 0, \quad \forall\, p > 1.$$

Adding the above differential inequalities of I_1, I_2 and using ξ we can write,

$$\frac{d}{dt}\int_\Omega \Big(I_1(x,t)^p + I_2(x,t)^p\Big)dx \le \Big[pC_1(\sigma^*+1)\zeta^* - p\xi\Big]\int_\Omega \Big(I_1(x,t)^p + I_2(x,t)^p\Big)dx. \tag{2.8}$$

For any $2 \le p < \infty$ and $0 \le \tau < T < T_m$ using Grönwall's inequality we get,

$$\|I_1(\cdot,t) + I_2(\cdot,t)\|_{p,\Omega\times(\tau,T)} \le M_p(T-\tau)\Big(\|I_1(\cdot,\tau)\|_{p,\Omega} + \|I_2(\cdot,\tau)\|_{p,\Omega}\Big). \tag{2.9}$$

for some nondecreasing $M_p \in C(\mathbb{R}_+,\mathbb{R}_+)$ with $M_p(0) = 0$.

Note that since this estimate only depends on the size of $T-\tau$, it follows from a limiting process that

$$\|I_1 + I_2\|_{p,\Omega\times(\tau,T_m)} \le M_p(T_m-\tau)\Big(\|I_1(\cdot,\tau)\|_{p,\Omega} + \|I_2(\cdot,\tau)\|_{p,\Omega}\Big). \tag{2.10}$$

Now, let us recall that

$$\begin{aligned} f_2(x,t,S,I_1,I_2,R) &= \sigma(x)g(x,t)S(x,t) - \Big(\lambda_1(x)+\mu(x)\Big)I_1(x,t) \le \sigma(x)\Big(\int_\Omega \rho(x,y)\beta_1(y)I_1(y,t)dy + \beta_2(x)I_2(x,t)\Big)S(x,t) \\ &\le \sigma^*\Big(\Lambda_\infty\beta_1^*M + \beta_2(x)I_2(x,t)\Big)S(x,t) \\ f_3(x,t,S,I_1,I_2,R) &= \Big(1-\sigma(x)\Big)g(x,t)S(x,t) - \Big(\lambda_2(x)+\mu(x)+\delta(x)+\gamma(x,t)\Big)I_2(x,t) \\ &\le \Big(\Lambda_\infty\beta_1^*M + \beta_2(x)I_2(x,t)\Big)S(x,t) \end{aligned}$$

so, if we define

$$G(x,t) = \sigma_1^*\Big(\Lambda_\infty\beta_1^*M + \beta_2(x)I_2(x,t)\Big)S(x,t)$$

where $\sigma_1^* = \max\Big\{1,\sigma^*\Big\}$, then

$$f_j(x,t) \le G(x,t), \quad j=2,3.$$

Then (2.10), (2.7) and (2.4) imply the boundedness of $\|G(\cdot,t)\|_{p,\Omega\times(\tau,T_m)}$ for each $1 \le p < \infty$. Let Ψ_1 be the unique nonnegative solution to

$$\begin{cases} \Psi_{1_t}(x,t) &= d_{I_1}(-\Delta)^{\alpha_{I_1}}\Psi_1(x,t) + G(x,t), \Omega\times(0,T_m), \\ \mathcal{N}_{\alpha_{I_1}}\Psi_1 &= 0, \qquad\qquad \mathbb{R}^n\setminus\Omega\times(0,T_m), \\ \Psi_1 &= I_{1,0}(x), \qquad\qquad \Omega\times\{0\}, \end{cases}$$

Then comparison principle for fractional parabolic initial boundary value problems implies that $I_1 \le \Psi_1$ and from the $L^p(\Omega\times(0,T_m))$ bound on $G(x,t)$ for $p > \frac{n+2\alpha_{I_1}}{2\alpha_{I_1}}$, $\|\Psi_1\|_{\infty,\Omega\times(0,T_m)}$ is bounded.

A similar argument can be applied to the unique nonnegative solution Ψ_2 of the parabolic system

$$\begin{cases} \Psi_{2_t}(x,t) &= d_{I_2}\Delta\Psi_2(x,t) + G(x,t), \Omega\times(0,T_m), \\ \frac{\partial\Psi_2}{\partial\eta} &= 0, \qquad\qquad \partial\Omega\times(0,T_m), \\ \Psi_2 &= I_{2,0}(x), \qquad\qquad \Omega\times\{0\}. \end{cases}$$

Then $I_2 \le \Psi_2$ and for any $p > \frac{n+2}{2}$, $\|\Psi_2\|_{\infty,\Omega\times(0,T_m)}$ is bounded.

Therefore, $\|I_1(\cdot,t)\|_{\infty,\Omega\times(\tau,T_m)}$ and $\|I_2(\cdot,t)\|_{\infty,\Omega\times(\tau,T_m)}$ are bounded. So, there exists a constant $C_5, C_6 > 0$ such that

$$\|I_1(\cdot,t)\|_{L^\infty(\Omega)} \le C_5 \qquad \text{for all } t>0, \tag{2.11}$$

$$\|I_2(\cdot,t)\|_{L^\infty(\Omega)} \le C_6 \qquad \text{for all } t>0. \tag{2.12}$$

This along with (2.4) proves the theorem. □

2.2 Proof of Theorem 2

Lemma 2.1 *Assume that the hypotheses of Theorem 1 hold. If* $\xi = \min\Big\{\lambda_{1,0}+\mu_0, \lambda_{2,0}+\mu_0, \delta_0+\gamma_0\Big\} > C_1(\sigma^*+1)\zeta^*$ *where* $\zeta^* := \Lambda_\infty\beta_1^*|\Omega| + \beta_2^*$, *then*

$$\lim_{T\to\infty}\int_T^{T+1}\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)\,dx\,dt = 0.$$

Moreover, for every $1 \le q < \infty$,

$$\|I_j\|_{L^q(\Omega\times(T,T+1))} \to 0 \qquad \text{as } T\to\infty, \quad j=1,2.$$

Proof: If $\xi > C_1\zeta$ then solving (2.6) and after that integrating respect to time from T to $T+1$ we can obtain

$$\lim_{T\to\infty}\int_T^{T+1}\int_\Omega \Big(I_1(x,t)+I_2(x,t)\Big)dxdt = 0. \tag{2.13}$$

Now, for $\xi > C_1(\sigma^*+1)\zeta^* > C_1\zeta$, (2.9) is true. Then integrating (2.9) with respect to time from T to $T+1$ instead of from τ to T we get,

$$\|I_j\|_{p,\Omega\times(T,T+1)} \le C_p, \qquad \forall\, T\ge 0,\ \ 1<p<\infty,\ \ j=1,2,\quad C_p>0. \tag{2.14}$$

Consequently, if $q>1$ then

$$\int_T^{T+1}\int_\Omega I_j(x,t)^q dxdt \le \sqrt{\int_T^{T+1}\int_\Omega I_j(x,t)dxdt}\sqrt{\int_T^{T+1}\int_\Omega I_j(x,t)^{2q-1}dxdt},\quad j=1,2$$

using (2.13), (2.14) and the nonnegativity of I_j for $j=1,2$ we conclude that

$$\|I_j\|_{q,\Omega\times(T,T+1)} \to 0 \text{ whenever } T\to\infty\ \forall\ q\ge 1. \qquad \square$$

Now, we assume $S^*\in D\Big((-\Delta)^{\alpha_S}\Big)\cap L^\infty(\Omega)$ is the disease-free steady state which is the unique positive solution of

$$\begin{cases} d_S(-\Delta)^{\alpha_S}S^*(x)+b(x)-\mu(x)S^*(x)=0, & x\in\Omega,\\ \mathcal{N}_{\alpha_S}S^*=0, & x\in\mathbb{R}^n\setminus\Omega. \end{cases} \tag{2.15}$$

Since $b(x)\ge 0$, $b\not\equiv 0$, and $\mu(x)\ge\mu_0>0$, it follows from the positivity of the resolvent associated with $d_S(-\Delta)^\alpha-\mu(x)$ that

$$S^*(x)>0 \qquad \text{for all } x\in\Omega.$$

Lemma 2.2 *Assume that the hypotheses of Theorem 1 hold and that the condition of Lemma 2.1 is satisfied. Let S^* be the disease-free steady state satisfying (2.15) with $S_0(x)\le S^*(x)$ in Ω, then the susceptible population converges to the disease-free steady state S^* on sliding time intervals. More precisely,*

$$\|S-S^*\|_{q,\Omega\times(T,T+1)}\to 0 \qquad \text{as } T\to\infty,$$

for every $1\le q<\infty$.

Proof: Since S^* satisfies (2.15), we have

$$-b(x) = d_S(-\Delta)^{\alpha_S}S^*(x)-\mu(x)S^*(x)$$

Define

$$w(x,t)=S^*(x)-S(x,t) \text{ with } w(x,0)=S^*(x)-S(x,0)=S^*(x)-S_0(x)\ge 0.$$

Since S^* is independent of t, then using the first equation of the system (1.1), we get,

$$\begin{aligned} w_t(x,t) &= -S_t(x,t) = -d_S(-\Delta)^{\alpha_S}S(x,t)-b(x)+\mu(x)S(x,t)+g(x,t)S(x,t)\\ &= -d_S(-\Delta)^{\alpha_S}S(x,t)+d_S(-\Delta)^{\alpha_S}S^*(x)-\mu(x)S^*(x)+\mu(x)S(x,t)+g(x,t)S(x,t)\\ &= d_S(-\Delta)^{\alpha_S}(S^*(x)-S(x,t))-\mu(x)(S^*(x)-S(x,t))+g(x,t)S(x,t)\\ &= d_S(-\Delta)^{\alpha_S}w(x,t)-\mu(x)w(x,t)+g(x,t)S(x,t) \end{aligned} \tag{2.16}$$

Since $w(x,0)\ge 0$, $S, I_1, I_2, \beta_1, \beta_2\ge 0$, and the semigroup generated by $d_S(-\Delta)^{\alpha_S}-\mu(x)$ is positive, the comparison principle gives

$$w(x,t)\ge 0 \qquad \text{for all } x\in\Omega,\ t\ge 0.$$

Hence,

$$S(x,t) \le S^*(x) \qquad \text{for all } x \in \Omega,\ t \ge 0.$$

Now integrate the equation for w over Ω to obtain

$$\begin{aligned}\frac{d}{dt}\int_\Omega w(x,t)dx &= d_S \int_\Omega (-\Delta)^{\alpha_S} w(x,t)dx - \int_\Omega \mu(x) w(x,t)dx + \int_\Omega g(x,t)S(x,t)dx \\ &\le 0 - \mu_0 \int_\Omega w(x,t)dx + C_1 \int_\Omega g(x,t)dx\end{aligned}$$

using (2.5), we can write

$$\frac{d}{dt}\int_\Omega w(x,t)dx + \mu_0 \int_\Omega w(x,t)dx \le C_1\zeta \int_\Omega (I_1(x,t) + I_2(x,t))dx.$$

Define

$$W(t) = \int_\Omega w(x,t)\,dx = \int_\Omega \Big(S^*(x) - S(x,t)\Big)dx$$

then using the estimate obtained from (2.6) (i.e. $\int_\Omega \Big(I_1(x,t) + I_2(x,t)\Big)\,dx \le e^{-(\xi - C_1\zeta)t}\int_\Omega \Big(I_{1,0}(x) + I_{2,0}(x)\Big)dx$) we can write,

$$W'(t) + \mu_0 W(t) \le C_1\zeta e^{-(\xi - C_1\zeta)t}\int_\Omega \Big(I_{1,0}(x) + I_{2,0}(x)\Big)dx$$

Multiplying by $e^{\mu_0 t}$ and integrating from 0 to t, we obtain

$$W(t) \le e^{-\mu_0 t}W(0) + C_1\zeta \|I_{1,0} + I_{2,0}\|_{1,\Omega}\int_0^t e^{-\mu_0(t-\tau)}e^{-(\xi - C_1\zeta)\tau}d\tau.$$

Since $\xi > C_1(\sigma^* + 1)\zeta^* > C_1\zeta$ (Lemma 2.1) and $\mu_0 > 0$, the right-hand side tends to 0 as $t \to \infty$. Hence

$$W(t) \to 0.$$

Since, $w(x,t) \ge 0$, we have

$$\|S(\cdot,t) - S^*\|_{1,\Omega} = \int_\Omega |S(x,t) - S^*(x)|\,dx = \int_\Omega w(x,t)\,dx = W(t).$$

Consequently,

$$\int_T^{T+1}\int_\Omega |S(x,t) - S^*(x)|\,dxdt = \int_T^{T+1} W(t)dt \to 0, \qquad \text{as } T \to \infty. \tag{2.17}$$

Now, since S is uniformly bounded by (2.4), $S^* \in L^\infty(\Omega)$, and Ω is bounded, it follows for every $1 \le p < \infty$ there exists $C_p^* > 0$ such that

$$\|S - S^*\|_{p,\Omega\times(T,T+1)} \le \|S\|_{p,\Omega\times(T,T+1)} + \|S^*\|_{p,\Omega\times(T,T+1)} \le C_p^*, \qquad \forall\, T \ge 0$$

Using the above estimate along with (2.17) and following the same interpolation argument as in Lemma 2.1, we get

$$\|S - S^*\|_{q,\Omega\times(T,T+1)} \to 0 \qquad \text{as } T \to \infty,$$

for every $1 \le q < \infty$. □

Proof of Theorem 2: By Theorem 1, the solution is global, nonnegative, and uniformly bounded in $L^\infty(\Omega)$. Moreover, by Lemma 2.1, for every $1 \le p < \infty$,

$$\|I_j\|_{L^p(\Omega\times(T,T+1))} \to 0 \qquad \text{as } T \to \infty, \qquad j = 1, 2.$$

Since S is uniformly bounded in $L^\infty(\Omega)$, we obtain

$$\|f_2\|_{L^p(\Omega\times(T,T+1))} \le C_1\sigma^*\zeta\left(\|I_1\|_{L^p(\Omega\times(T,T+1))} + \|I_2\|_{L^p(\Omega\times(T,T+1))}\right) + (\lambda_1^* + \mu^*)\|I_1\|_{L^p(\Omega\times(T,T+1))} \to 0$$
$$\|f_3\|_{L^p(\Omega\times(T,T+1))} \le C_1\sigma_1^*\zeta\left(\|I_1\|_{L^p(\Omega\times(T,T+1))} + \|I_2\|_{L^p(\Omega\times(T,T+1))}\right) + (\lambda_2^* + \mu^* + \delta^* + \gamma^*)\|I_2\|_{L^p(\Omega\times(T,T+1))} \to 0$$

as $T \to \infty$, $\sigma_1^* = \max\left\{1, \sigma^*\right\}$. Hence,

$$\|f_j\|_{L^p(\Omega\times(T,T+1))} \to 0, \qquad \text{as } T \to \infty, \qquad j = 1, 2.$$

By the classical L^p-regularity theory for linear parabolic equations with homogeneous Neumann boundary conditions [33, 35], there exists a constant $K_p > 0$, independent of T, such that

$$\|I_2\|_{W_p^{2,1}(\Omega\times(T,T+1))} \le K_p.$$

For $p > \frac{n+2}{2}$, the parabolic Sobolev embedding theorem yields $W_p^{2,1}(\Omega \times (T, T+1)) \hookrightarrow C^{\epsilon,\epsilon/2}(\overline{\Omega} \times [T, T+1])$ for some $\epsilon \in (0, 1)$. Consequently, I_2 is uniformly Hölder continuous on $\overline{\Omega} \times [T, T+1]$, with Hölder constants independent of T.

Combining this uniform Hölder continuity with the decay of the L^p-norms on sliding time intervals, and applying the standard contradiction argument of Fitzgibbon and Morgan [21], we conclude that

$$\lim_{t\to\infty} \|I_2(\cdot, t)\|_{\infty,\Omega} = 0.$$

Next, we consider the asymptomatic class I_1. Applying the maximal L^p-regularity theory for fractional parabolic equations with homogeneous nonlocal Neumann boundary conditions, there exists a constant $K_p > 0$, independent of T, such that

$$\|I_1\|_{L^p((T,T+1);W^{2\alpha_{I_1},p}(\Omega))} + \|\partial_t I_1\|_{L^p(\Omega\times(T,T+1))} \le K_p.$$

Equivalently,

$$\|I_1\|_{W_p^{2\alpha_{I_1},1}(\Omega\times(T,T+1))} \le K_p.$$

Choose $p > \frac{n+2\alpha_{I_1}}{2\alpha_{I_1}}$. Then the fractional parabolic Sobolev embedding theorem yields

$$W_p^{2\alpha_{I_1},1}(\Omega \times (T, T+1)) \hookrightarrow C^{\varepsilon,\varepsilon/(2\alpha_{I_1})}\Big(\overline{\Omega} \times [T, T+1]\Big),$$

for some $\varepsilon \in (0, 1)$ [38, 49]. Hence, I_1 is uniformly Hölder continuous on $\overline{\Omega}\times[T, T+1]$, with Hölder constants independent of T.

Combining this uniform Hölder continuity with the decay of the L^p-norms on sliding time intervals and applying the same contradiction argument, we conclude that

$$\lim_{t\to\infty} \|I_1(\cdot, t)\|_{\infty,\Omega} = 0.$$

It remains to prove the convergence of S. Let S^* be the disease-free steady state as in Lemma 2.2, and define

$$w(x, t) := S^*(x) - S(x, t).$$

We observe that $f_1 \in L^p(\Omega \times (T, T+1))$, therefore, using Lemma 2.2 and (2.16), for every $1 \le p < \infty$ we get

$$\|w\|_{L^p(\Omega\times(T,T+1))} \to 0 \qquad \text{as } T \to \infty.$$

Similarly, choosing $p > \frac{n+2\alpha_S}{2\alpha_S}$, applying the same argument as above, we obtain

$$\|w(\cdot,t)\|_{\infty,\Omega} \to 0 \qquad \text{as } t \to \infty.$$

Since $w = S^* - S$, it follows that

$$\lim_{t\to\infty} \|S(\cdot,t) - S^*\|_{\infty,\Omega} = 0.$$

The proof is complete. □

3 Numerical Simulations

In this section, we present numerical simulations to complement the analytical results established in Section 2 and to investigate the effects of spatial diffusion and intermittent treatment on disease dynamics.

The numerical study investigates the mixed reaction–diffusion system (MRDS) (1.1), in which the susceptible and asymptomatic infected populations undergo regional fractional diffusion with distinct fractional exponents α_S and α_{I_1}, while the symptomatic infected and recovered populations undergo classical diffusion. This formulation reflects the fact that different epidemiological classes may exhibit substantially different mobility patterns. In particular, asymptomatic infected individuals often continue their normal daily activities because they are unaware of their infection, potentially undertaking long-range movements that contribute to disease transmission. In contrast, the movement of susceptible individuals is governed primarily by routine travel and social interactions. Allowing separate fractional exponents therefore provides additional flexibility for modeling heterogeneous dispersal behaviors and enables us to investigate how differences in long-range mobility influence epidemic dynamics.

The simulations provide numerical evidence supporting the long-time behavior predicted by Theorem 2 and illustrate the effects of diffusion and intermittent treatment on the temporal and spatial dynamics of the epidemic.

Simulations are performed on the square domain $\Omega = (0,10) \times (0,10)$, using a uniform 128×128 spatial mesh over the time interval $[0, 200]$. The spatial discretization is carried out using a second-order central finite difference approximation on a uniform mesh. Time integration is performed using the explicit forward Euler method. Homogeneous Neumann boundary conditions are implemented through the standard reflection technique. The time step is chosen to satisfy the classical stability restriction associated with the explicit finite difference scheme.

For $\alpha_S, \alpha_{I_1} \in (0,1)$, the regional fractional Laplacian is discretized using the Riesz integral representation

$$(-\Delta)^{\alpha_j} u(x_i, y_j) \approx C_{n,\alpha_j} \sum_{(k,\ell)\neq(i,j)} \frac{u_{i,j} - u_{k,\ell}}{\left[(x_i - x_k)^2 + (y_j - y_\ell)^2\right]^{\frac{n+2\alpha_j}{2}}} \Delta x\, \Delta y, \tag{3.1}$$

where u denotes either S or I_1 with the corresponding fractional exponent α_j, $j \in \{S, I_1\}$. All nonlocal interactions are restricted to the computational domain Ω, consistent with the homogeneous nonlocal boundary condition (1.3) and the regional fractional Laplacian (1.4).

The nonlocal incidence term associated with the asymptomatic infected population is evaluated using the two-dimensional Gaussian kernel with unit variance,

$$\rho(x,y) = \frac{1}{2\pi} \exp\left(-\frac{|x-y|^2}{2}\right).$$

The diffusion coefficients are chosen as

$$d_S = 0.10, \qquad d_{I_1} = 0.06, \qquad d_{I_2} = 0.02, \qquad d_R = 0.05.$$

The initial conditions and spatially heterogeneous coefficient functions used in the simulations are listed in Table 2.

Table 2: Initial conditions and spatially heterogeneous coefficient functions used in the numerical simulations.

Quantity	**Spatial form**
$S(x,y,0)$	$100\left(1+0.20\cos\dfrac{\pi x}{L_x}\cos\dfrac{\pi y}{L_y}\right)$
$I_1(x,y,0)$	$2.00\left(1+0.40\exp\left(-\dfrac{(x-5)^2+(y-5)^2}{3}\right)\right)$
$I_2(x,y,0)$	$0.90\left(1+0.50\exp\left(-\dfrac{(x-3)^2+(y-7)^2}{2}\right)\right)$
$R(x,y,0)$	0
$b(x,y)$	$0.60\left(0.65+0.35\cos^2\dfrac{\pi x}{L_x}\cos^2\dfrac{\pi y}{L_y}\right)$
$\mu(x,y)$	$0.01\left(1+0.40\cos^2\dfrac{\pi x}{L_x}\cos^2\dfrac{\pi y}{L_y}\right)$
$\beta_1(x,y)$	$0.0125\,(0.30+0.70\,\mathcal{R}(x,y))$
$\beta_2(x,y)$	$0.0100\,(0.30+0.70\,\mathcal{R}(x,y))$
$\sigma(x,y)$	$\max\{0.55,\ 0.70\,(0.75+0.25\,\mathcal{R}(x,y))\}$
$\lambda_1(x,y)$	$0.006\left(1+0.30\sin^2\dfrac{\pi y}{L_y}\right)$
$\lambda_2(x,y)$	$0.003\left(1+0.30\cos^2\dfrac{\pi x}{L_x}\right)$
$\delta(x,y)$	$0.001\left(1+0.30\sin^2\dfrac{\pi x}{L_x}\sin^2\dfrac{\pi y}{L_y}\right)$
$\gamma_1(x,y)$	$0.05\left(1+0.75\sin^2\dfrac{\pi x}{L_x}\sin^2\dfrac{\pi y}{L_y}\right)$

Here, $\mathcal{R}(x,y)$ denotes the normalized spatial risk function defined by

$$\mathcal{R}(x,y)=\frac{0.65\,r_1(x,y)+0.35\,r_2(x,y)}{\max\limits_{(x,y)\in\overline{\Omega}}\{0.65\,r_1(x,y)+0.35\,r_2(x,y)\}}.$$

The component functions r_1 and r_2 are given by

$$r_1(x,y)=\frac{\sin(\pi x/L_x)\sin\big(\pi y/L_y\big)}{\max\limits_{(x,y)\in\overline{\Omega}}\Big\{\sin(\pi x/L_x)\sin\big(\pi y/L_y\big)\Big\}},\qquad r_2(x,y)=\frac{\exp\left(-\big((x-7)^2+(y-3)^2\big)/5\right)}{\max\limits_{(x,y)\in\overline{\Omega}}\Big\{\exp\left(-\big((x-7)^2+(y-3)^2\big)/5\right)\Big\}}.$$

The function $\mathcal{R}(x,y)$ is constructed as a weighted combination of a smooth background distribution $r_1(x,y)$ and a localized Gaussian hotspot $r_2(x,y)$. The first component represents large-scale spatial variation across the domain, whereas the second represents a localized region of elevated epidemiological risk.

The numerical values listed in Table 2 are chosen as representative reference values for the computational experiments. They are not calibrated to a particular disease or epidemiological dataset. Rather, they provide a biologically plausible parameter configuration for illustrating the qualitative effects of spatial heterogeneity, asymptomatic transmission, diffusion, and intermittent treatment.

Throughout the simulations, treatment is applied only to the symptomatic infected class I_2. The intermittent treatment function is prescribed by

$$\gamma(x,y,t) = \begin{cases} 0, & kT_\gamma \le t < (k+1)T_\gamma - L_\gamma, \\ \gamma_1(x,y), & (k+1)T_\gamma - L_\gamma \le t < (k+1)T_\gamma, \end{cases} \qquad k = 0,1,2,\ldots. \tag{3.2}$$

In the numerical simulations, we take

$$T_\gamma = 20, \qquad L_\gamma = 10.$$

Thus, treatment is inactive during the first 10 time units of each cycle and active during the final 10 time units, corresponding to a 50% duty cycle. The treatment function enters only the symptomatic infected and recovered equations. Hence, symptomatic individuals removed through treatment are transferred directly to the recovered class. No treatment term is included in the asymptomatic equation, so the class I_1 is affected only indirectly through changes in the force of infection. The periodic on–off protocol represents intermittent treatment programs arising from scheduled intervention campaigns, limited medical resources, temporary treatment availability, or periodic public health measures. The spatial dependence of $\gamma_1(x,y)$ allows the treatment intensity to vary throughout the domain and thereby represents geographical heterogeneity in healthcare availability, treatment coverage, or intervention effectiveness.

First, we present the fractional kernel in Figure 1 for $\alpha = \alpha_S = \alpha_{I_1} = 0.50$ to illustrate the characteristic features of fractional diffusion. We also display the Gaussian kernel used to model the nonlocal asymptomatic transmission. The fractional kernel possesses a singularity at the origin together with a slowly decaying tail, allowing long-range spatial interactions that are absent in the classical diffusion model. In contrast, the Gaussian kernel is smooth and rapidly decaying, representing localized nonlocal transmission. These kernel profiles provide the foundation for the subsequent simulations with and without intermittent treatment.

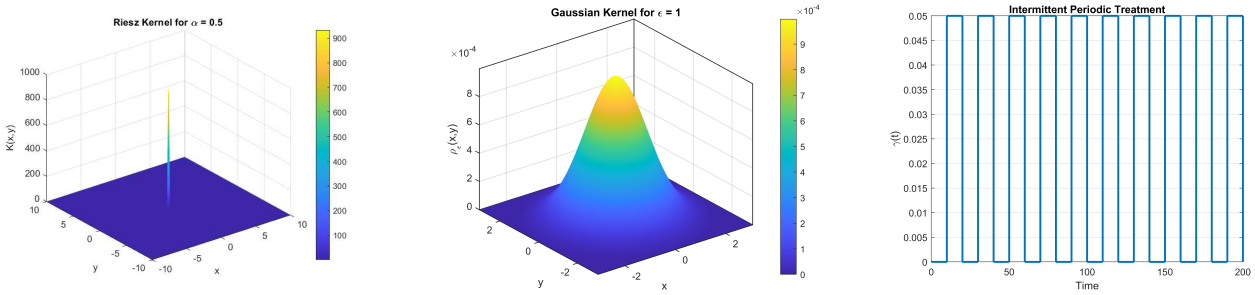


Figure 1: Fractional diffusion kernel associated with the regional fractional Laplacian for $\alpha = 0.50$, together with its logarithmic-scale representation.

To illustrate the qualitative behavior of the proposed model, numerical simulations are performed using the fractional diffusion exponents

$$(\alpha_S, \alpha_{I_1}) = (0.10,\ 0.40),$$

while the Gaussian interaction width in the nonlocal asymptomatic force of infection is fixed at $\epsilon = 1$. Throughout the simulations, the susceptible and asymptomatic infected populations are assumed to diffuse with different fractional orders, allowing the model to capture distinct dispersal behaviors among epidemiological compartments. In particular, the smaller fractional exponent for the susceptible population reflects stronger long-range movement, whereas the larger exponent for the asymptomatic infected population corresponds to comparatively more localized dispersal.

The numerical simulations are intended to demonstrate the spatiotemporal dynamics generated by the proposed spatially heterogeneous fractional epidemic model. They illustrate how heterogeneous epidemiological parameters, nonlocal asymptomatic transmission, fractional diffusion, and intermittent treatment collectively influence the temporal evolution, spatial averages, and spatial distributions of the susceptible, asymptomatic infected, symptomatic infected, and recovered populations. These simulations complement

the theoretical analysis by providing a visual representation of the complex interaction between spatial heterogeneity, long-range dispersal, and disease transmission, thereby highlighting the capability of the model to describe realistic epidemic spread in heterogeneous environments.

Figures 2 show the final spatial distributions of the susceptible, asymptomatic infected, symptomatic infected, and recovered populations for the mixed diffusion model with $(\alpha_S, \alpha_{I_1}) = (0.10, 0.40)$. The results are presented both in the presence and absence of intermittent treatment, illustrating the heterogeneous spatial patterns generated by the combined effects of fractional diffusion, nonlocal transmission, and spatially varying epidemiological parameters.

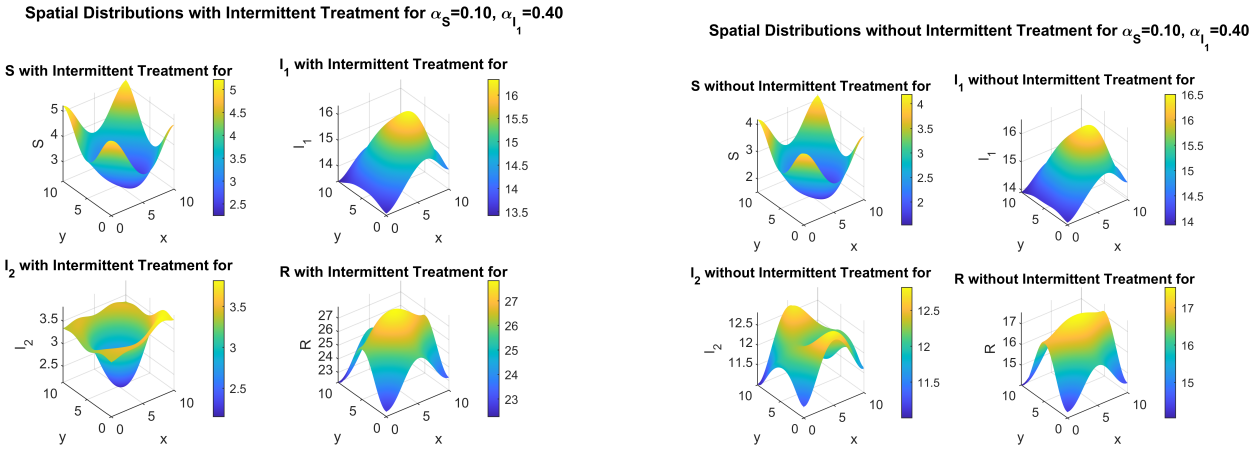


Figure 2: Final spatial distributions of S, I_1, I_2, and R for the mixed reaction–diffusion system with and without intermittent treatment at $t = 200$ for $(\alpha_S, \alpha_{I_1}) = (0.10, 0.40)$.

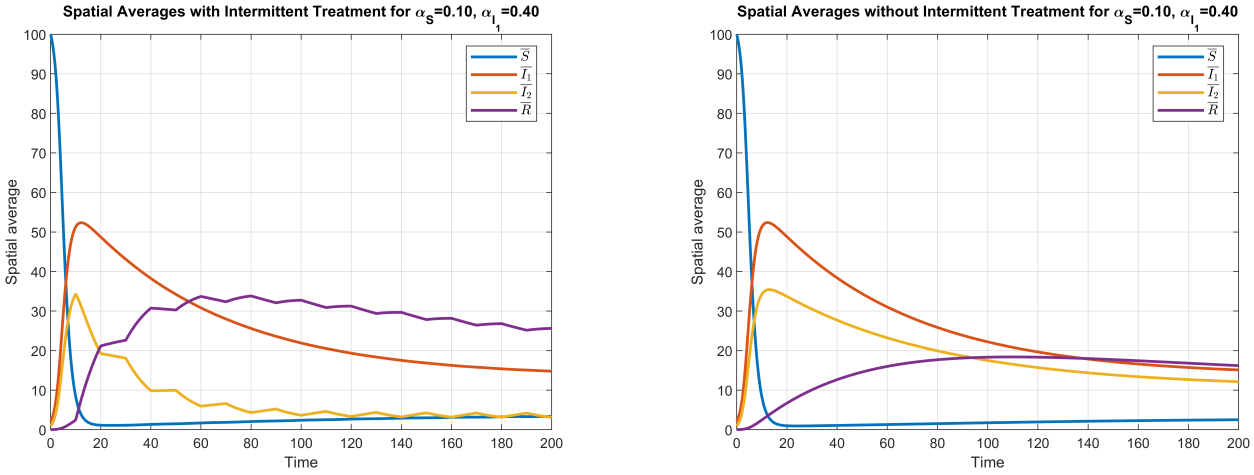


Figure 3: Temporal evolution of the spatial averages $\bar{S}(t)$, $\bar{I}_1(t)$, $\bar{I}_2(t)$, and $\bar{R}(t)$ for the mixed reaction–diffusion system with and without intermittent treatment for $(\alpha_S, \alpha_{I_1}) = (0.10, 0.40)$.

To examine the overall temporal dynamics and evaluate the effectiveness of the intermittent treatment strategy, we compute the spatial averages

$$\bar{U}(t) = \frac{1}{|\Omega|} \int_\Omega U(x, y, t)\, dx\, dy, \qquad U \in \{S, I_1, I_2, R\}. \tag{3.3}$$

These quantities represent the mean population densities over the spatial domain and provide a global measure of the epidemic evolution while averaging out local spatial fluctuations. Figure 3 displays the

temporal evolution of the spatial averages $\overline{S}(t)$, $\overline{I}_1(t)$, $\overline{I}_2(t)$, and $\overline{R}(t)$ for the mixed diffusion model with $(\alpha_S, \alpha_{I_1}) = (0.10, 0.40)$ in the presence and absence of intermittent treatment. Compared with the untreated case, intermittent treatment produces a pronounced reduction in the average symptomatic infected population and a corresponding increase in the average recovered population. Furthermore, the treatment decreases the long-term disease burden and modifies the evolution of the susceptible and asymptomatic infected populations, demonstrating its effectiveness in mitigating disease transmission at the population level.

4 Conclusion

In this work, we investigated a mixed fractional–classical reaction–diffusion epidemic model consisting of susceptible, asymptomatic infected, symptomatic infected, and recovered populations. The model incorporates spatially heterogeneous demographic and epidemiological parameters, nonlocal asymptomatic transmission, and a periodic intermittent treatment strategy. To capture heterogeneous mobility patterns among different epidemiological classes, the susceptible and asymptomatic infected populations were assumed to undergo regional fractional diffusion with distinct fractional exponents, while the symptomatic infected and recovered populations evolve through classical diffusion.

From the analytical perspective, we established the global existence, uniqueness, positivity, and uniform boundedness of classical solutions by combining semigroup theory with suitable L^p-energy estimates. Despite the coexistence of local and regional fractional diffusion operators, together with nonlinear transmission and spatial heterogeneity, we derived sufficient conditions ensuring the extinction of both infected populations and the convergence of the susceptible population to the disease-free equilibrium. These results provide a rigorous characterization of the global dynamics of the proposed epidemic model.

Numerical simulations were carried out to investigate the spatiotemporal dynamics of the proposed mixed reaction–diffusion epidemic model under spatially heterogeneous environments. The results demonstrate the combined effects of fractional diffusion, nonlocal asymptomatic transmission, and heterogeneous epidemiological parameters on the evolution of the susceptible, asymptomatic infected, symptomatic infected, and recovered populations. In particular, the mixed diffusion framework captures the influence of long-range dispersal on the spatial spread of the disease, leading to heterogeneous population distributions over the spatial domain. Furthermore, the numerical results show that intermittent treatment effectively suppresses disease transmission, reduces the infected populations, and promotes recovery, illustrating its potential for mitigating epidemic outbreaks in heterogeneous environments.

The proposed framework extends existing epidemic models by combining regional fractional diffusion with distinct fractional exponents, nonlocal asymptomatic transmission, intermittent treatment, and spatially heterogeneous environments within a unified analytical framework. Future research will focus on the derivation of threshold dynamics through the basic reproduction number, the existence and stability of endemic equilibria, optimal control strategies for treatment and intervention, and the investigation of more general nonlocal diffusion operators, heterogeneous movement kernels, and network-based spatial structures.

Conflict of Interest: There is no conflict of Interest.

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