Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Analysis of a nonlocal SIR model via a diffusive limit approach

Md Shah Alam

malam@htu.edu

Huston–Tillotson University https://orcid.org/0009-0004-3765-4953

Research Article

Keywords: Nonlocal SIR model, Convolution-type diffusion, Intermittent treatment, Global existence, Asymptotic behavior, Diffusive limit

Posted Date: August 13th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10672659/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: The authors declare no competing interests.

Analysis of a nonlocal SIR model via a diffusive limit approach

Md Shah Alam*

Department of Mathematics, Huston–Tillotson University, Austin, TX 78702, USA

Abstract

We investigate a spatially heterogeneous nonlocal SIR epidemic model with convolution-type diffusion and an intermittent treatment strategy. The model incorporates long-range dispersal while allowing the susceptible, infected, and recovered populations to diffuse at distinct rates. Using semigroup theory, we establish the global existence, uniqueness, positivity, and uniform boundedness of classical solutions. Robust L^p-energy estimates are then developed to investigate their asymptotic behavior. Since the nonlocal convolution operator lacks the classical parabolic smoothing property, a diffusive limit approach is employed to recover the required smoothing properties through convergence to the corresponding local reaction–diffusion model. Numerical simulations illustrate the effects of spatial heterogeneity, nonlocal dispersal, increasing interaction radii of the Gaussian kernel, and intermittent treatment on the spatiotemporal dynamics of the epidemic, with the observed trends remaining consistent with the theoretical analysis.

AMS Subject Classification [2020]: 35K57, 35B40, 92D30, 45K05

Keywords: Nonlocal SIR model; Convolution-type diffusion; Intermittent treatment; Global existence; Asymptotic behavior; Diffusive limit.

1 Introduction and Main Results

1.1 Introduction

Since the pioneering work of Kermack and McKendrick [22], mathematical models have become indispensable tools for understanding the transmission dynamics of infectious diseases and for designing effective intervention strategies. Among the numerous compartmental epidemic models proposed over the past century, the susceptible–infected–recovered (SIR) model remains one of the most fundamental and influential frameworks in mathematical epidemiology. The SIR model has served as the foundation for numerous epidemic models incorporating demographic effects, vaccination, treatment, spatial heterogeneity, stochasticity, and network interactions. It has been successfully applied to a wide range of infectious diseases, including influenza, tuberculosis, Ebola, SARS, and COVID-19.

The mathematical theory of epidemic models has developed substantially over the past several decades, encompassing epidemic thresholds, stability, persistence, bifurcation, optimal control, and parameter estimation. Comprehensive treatments of deterministic epidemic models can be found in the monographs of Anderson and May [3], Diekmann and Heesterbeek [14], Hethcote [18], Keeling and Rohani [21], Brauer and Castillo-Chavez [7], and Diekmann *et al.* [13]. These works have established a rigorous mathematical framework for understanding disease transmission and have provided theoretical guidance for modern public health policies.

While classical compartmental models assume spatially homogeneous populations, real epidemic outbreaks typically occur in heterogeneous environments where population density, environmental conditions,

*Email: malam@htu.edu

healthcare accessibility, and human mobility vary across regions. Consequently, incorporating spatial heterogeneity into epidemic models has become an important area of research in mathematical epidemiology. Spatially explicit models provide a natural framework for investigating the temporal and geographical spread of infectious diseases, spatial persistence, invasion fronts, and regional control strategies, particularly for diseases strongly influenced by population movement and environmental variability.

A natural extension of the classical SIR framework incorporates spatial diffusion through reaction–diffusion equations, in which susceptible, infected, and recovered individuals move randomly according to Fickian diffusion represented by the Laplace operator. Such models provide a natural framework for studying the interaction between disease transmission and spatial movement, and have been widely used to investigate epidemic propagation in heterogeneous environments, traveling waves, disease persistence, extinction, spatial pattern formation, and threshold dynamics.

During the past two decades, reaction–diffusion SIR models have been studied extensively from both theoretical and computational perspectives. Allen *et al.* [2] investigated disease dynamics in heterogeneous environments and established several threshold results for spatial epidemic models. Wang and Zhao [36, 37] developed a comprehensive theory for the basic reproduction number of reaction–diffusion epidemic systems and demonstrated its fundamental role in determining disease persistence and extinction. Peng [31] analyzed the global dynamics of diffusive epidemic systems under heterogeneous transmission mechanisms, while Kuniya [25] examined the influence of spatial diffusion on epidemic thresholds and long-term behavior. Other important developments include the works of Capasso and Maddalena [10], Lewis *et al.* [28], and Cantrell and Cosner [9]. Collectively, these studies demonstrate that spatial diffusion can substantially influence the spread, persistence, and control of infectious diseases.

Although reaction–diffusion epidemic models have been highly successful, their diffusion mechanism is based on the classical Laplace operator, which assumes infinitesimally small random displacements resembling Brownian motion. While this assumption is appropriate for predominantly local movement, many contemporary epidemics involve inherently nonlocal mobility. Air transportation, railway networks, highways, daily commuting, and seasonal migration enable infected individuals to travel directly between geographically distant locations, bypassing intermediate regions. Consequently, classical diffusion may underestimate the speed of disease spread and fail to capture important long-range transmission pathways.

Moreover, classical diffusion assumes that movement is driven solely by local density gradients. In practice, human mobility is strongly influenced by transportation infrastructure, socioeconomic activities, employment, education, and healthcare accessibility, leading to interactions between distant locations. Similar long-range movement patterns arise in animal migration, bird dispersal, marine ecology, and vector-borne disease transmission. These observations indicate that the Brownian motion assumption underlying reaction–diffusion models is often too restrictive for describing realistic dispersal processes.

These limitations have motivated increasing interest in nonlocal dispersal models, in which movement is described by integral operators rather than differential operators. By allowing direct interactions between distant locations through dispersal kernels, nonlocal diffusion provides a flexible framework for modeling heterogeneous mobility patterns and establishing a natural connection between microscopic movement mechanisms and macroscopic population dynamics. These biological and mathematical considerations motivate the development of epidemic models based on nonlocal diffusion.

The increasing availability of mobility data and advances in the mathematical theory of nonlocal partial differential equations have stimulated growing interest in epidemic models with nonlocal dispersal. Unlike the classical Laplace operator, nonlocal diffusion is typically represented by an integral operator that describes movement through a dispersal kernel, naturally accounting for long-range interactions and heterogeneous mobility patterns. Over the past two decades, nonlocal diffusion equations have been studied extensively in ecology, population dynamics, image processing, phase transitions, and epidemiology, providing new insights into spatial pattern formation, invasion phenomena, persistence, traveling waves, and asymptotic behavior [4, 6, 8, 11, 12, 15, 16, 20, 33, 38, 39].

A widely adopted formulation of nonlocal diffusion is based on convolution-type integral operators of the form

$$(\mathcal{K}u)(x) = \int_{\Omega} \phi(x, y)\Big(u(y) - u(x)\Big)\, dy,$$

where the kernel ϕ describes the probability of movement from one location to another. The kernel is

typically assumed to be nonnegative, symmetric, normalized, and compactly supported or rapidly decaying. Unlike local diffusion, which depends only on infinitesimal neighborhood interactions, convolution operators allow spatial interactions over finite distances whose magnitude is determined by the support of the kernel. Consequently, different choices of kernels can represent various dispersal mechanisms ranging from short-range random movement to long-distance migration. From the mathematical viewpoint, convolution operators preserve positivity, satisfy comparison principles under suitable assumptions, generate positive semigroups, and converge to the classical Laplace operator under appropriate scaling of the dispersal kernel. These analytical properties, together with their ability to model long-range interactions, have made convolution-type nonlocal diffusion operators a fundamental tool in the study of nonlocal partial differential equations and their applications in biology, ecology, and epidemiology [4, 6, 11, 12, 16, 19, 27].

Besides incorporating realistic dispersal mechanisms, effective disease control strategies constitute another fundamental aspect of epidemic modeling. In practice, treatment and vaccination programs are rarely implemented continuously. Instead, public health interventions are often applied periodically because of limited medical resources, economic constraints, seasonal outbreaks, or logistical considerations. Examples include annual influenza vaccination campaigns, seasonal mosquito-control programs, periodic mass drug administration, and recurrent intervention strategies adopted during the COVID-19 pandemic. Consequently, epidemic models with periodic treatment provide a more realistic description of disease management than models with constant treatment rates.

Among various control strategies, intermittent treatment has attracted considerable attention because it balances disease suppression with implementation cost. By activating treatment only during prescribed time intervals, it reduces resource consumption while maintaining effective disease control. From a mathematical viewpoint, periodic treatment introduces non-autonomous coefficients that lead to rich dynamical behaviors, including periodic solutions, Floquet-type stability, threshold dynamics, and bifurcation phenomena [17, 23, 32].

In the present work, recovery is assumed to occur exclusively through treatment. Consequently, infected individuals enter the recovered compartment only during treatment periods, while no recovery occurs when treatment is inactive. This assumption enables us to investigate the impact of intermittent therapeutic interventions on disease spread and control.

Motivated by the above considerations, we study a spatially heterogeneous nonlocal SIR epidemic model with convolution-type diffusion and intermittent treatment. The proposed framework incorporates long-range dispersal together with periodic disease intervention. We establish the global existence, uniqueness, positivity, and uniform boundedness of classical solutions, and investigate their asymptotic behavior through a diffusive-limit approach. Numerical simulations are presented to investigate the influence of increasing interaction radii of the Gaussian kernel and intermittent treatment on the spatiotemporal dynamics of the epidemic.

To formulate the model, let $n \in \mathbb{N}$ and let $\Omega \subset \mathbb{R}^n$ be a bounded domain with smooth boundary $\partial\Omega$, where $\partial\Omega$ is an $(n-1)$-dimensional $C^{2+\rho}$ $(0 < \rho < 1)$ manifold. We consider the following spatially heterogeneous nonlocal SIR epidemic system:

$$\begin{cases} S_t(x,t) &= d_S\Gamma_S S(x,t) + b(x) - \mu(x)S(x,t) - \beta(x)S(x,t)I(x,t), \quad x \in \Omega,\ t \geq 0, \\ I_t(x,t) &= d_I\Gamma_I I(x,t) + \beta(x)S(x,t)I(x,t) - (\mu(x)+\gamma(x,t))I(x,t), x \in \Omega,\ t \geq 0, \\ R_t(x,t) &= d_R\Gamma_R R(x,t) + \gamma(x,t)I(x,t) - \mu(x)R(x,t), \qquad x \in \Omega,\ t \geq 0, \\ (S,I,R)(x,0) &= (S_0(x), I_0(x), R_0(x)) \geq 0, \qquad x \in \Omega, \end{cases} \tag{1.1}$$

The functions $S(x,t)$, $I(x,t)$, and $R(x,t)$ denote the densities of susceptible, infected, and recovered individuals, respectively, at location $x \in \Omega$ and time $t \geq 0$. The initial data are assumed to be nonnegative and bounded.

The nonlocal diffusion operators Γ_S, Γ_I, and Γ_R describe the spatial movement of the three epidemiological compartments and are defined by

$$\Gamma_i u(x,t) = \int_\Omega \phi_i(x,y)\Big(u(y,t) - u(x,t)\Big)\,dy, \qquad i \in \{S, I, R\},$$

where $\phi_i(x,y) \geq 0$ is a symmetric interaction kernel characterizing movement between locations x and y.

For simplicity, we assume that all compartments share the same interaction kernel,

$$\phi_S(x,y) = \phi_I(x,y) = \phi_R(x,y) =: \phi(x,y).$$

Accordingly, the nonlocal diffusion operators reduce to

$$\Gamma_i u(x,t) = \Gamma u(x,t) = \int_\Omega \phi(x,y)\Big(u(y,t) - u(x,t)\Big)\,dy, \qquad i \in \{S,I,R\}, \tag{1.2}$$

where ϕ satisfies the boundedness assumption in (D1).

Biologically, the operator Γ models nonlocal dispersal by allowing individuals at location x to interact with or move to other locations $y \in \Omega$ according to the interaction kernel $\phi(x,y)$. In contrast to classical diffusion, which is driven by local spatial gradients in infinitesimal neighborhoods, nonlocal diffusion incorporates long-range movement arising from mechanisms such as human travel, commuting, migration, and transportation networks. Consequently, the disease dynamics at a given location depend not only on the local population but also on interactions with individuals across the spatial domain.

The function $b(x) \geq 0$ denotes the spatially dependent recruitment rate of susceptible individuals, while $\mu(x) \geq 0$ represents the natural mortality rate. The treatment function $\gamma(x,t) \geq 0$ is assumed to be bounded, spatially heterogeneous, and periodic in time with period $T > 0$. 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, and a constant $\gamma_0 > 0$ such that $\gamma_1(x,t) \geq \gamma_0$, $(x,t) \in \widehat{\Omega} \times [0,\infty)$, and

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

Thus, during each treatment cycle of length T, no treatment is administered over the interval $kT \leq t < (k+1)T - L$, whereas infected individuals receive treatment at rate $\gamma_1(x,t)$ during the final L-time units of the cycle. This periodic treatment protocol models intermittent intervention strategies arising from limited healthcare resources, seasonal treatment campaigns, or scheduled public health control programs.

We adopt the following notation throughout the paper where all the spatial parameters are continuous and bounded on $\overline{\Omega}$:

$$b^* := \max_{x\in\overline{\Omega}} b(x) \geq 0, \;\; \beta^* := \max_{x\in\overline{\Omega}} \beta(x) \geq 0, \;\; \gamma^* := \max_{(x,t)\in\overline{\Omega}\times[0,T]} \gamma(x,t) \geq 0, \;\; \mu^* := \max_{x\in\overline{\Omega}} \mu(x) \geq 0, \;\; \mu_0 := \min_{x\in\overline{\Omega}} \mu(x) > 0.$$

For notational convenience, we write the reaction terms in system (1.1) as

$$F(x,t,S,I,R) = \begin{pmatrix} F_1(x,t,S,I,R) \\ F_2(x,t,S,I,R) \\ F_3(x,t,S,I,R) \end{pmatrix} = \begin{pmatrix} b(x) - \mu(x)S(x,t) - \beta(x)S(x,t)I(x,t) \\ \beta(x)S(x,t)I(x,t) - (\mu(x) + \gamma(x,t))I(x,t) \\ \gamma(x,t)I(x,t) - \mu(x)R(x,t) \end{pmatrix}. \tag{1.3}$$

Since the reaction terms are polynomial functions of the state variables (S,I,R) with bounded coefficient functions, the mapping F is locally Lipschitz continuous with respect to (S,I,R), uniformly in (x,t), on bounded subsets of $\mathbb{R}^3_+$.

1.2 Statement of the Main Results

Let us assume that

(D1) (Diffusivity) $d_i > 0$ for all $i = 1,2$ and $\phi \in C\left(\overline{\Omega} \times \overline{\Omega}, \mathbb{R}_+\right)$ such that $\phi(x,y) = \phi(y,x)$ for all $x, y \in \Omega$ and there exist $\varepsilon, \lambda \geq 0$ such that

$$0 \leq \varepsilon \leq \int_\Omega \phi(x,y)dy \leq \lambda \;\text{ for all } x \in \Omega. \tag{1.4}$$

(Qp) (Quasi-positivity) For each $F_1(0,I,R), F_2(S,0,R), F_3(S,I,0) \geq 0$ whenever $t \geq 0$ and $(S,I,R) \in \mathbb{R}^3_+$.

Theorem 1.1. *Assume that (D1) and (Qp) hold, and let* $(S_0, I_0, R_0) \in C(\overline{\Omega}; \mathbb{R}^3_+)$. *Then system* (1.1) *admits a unique global component-wise nonnegative classical solution* (S, I, R) *of system* (1.1). *Moreover, each component remains uniformly bounded in the sup norm for all time.*

Although Theorem 1.1 establishes the global existence and uniform boundedness of classical solutions, it does not immediately yield their asymptotic behavior. The main difficulty stems from the fact that convolution-type nonlocal diffusion operators generally do not possess the parabolic smoothing property enjoyed by the classical Laplace operator. Consequently, the standard techniques used for local reaction–diffusion systems cannot be applied directly to the nonlocal model.

To overcome this difficulty, we exploit the diffusive-limit result established in Theorem A.1, whose proof is deferred to Appendix A. This result shows that, under an appropriate scaling of the dispersal kernel, solutions of the nonlocal system converge to those of the corresponding local reaction–diffusion system. Since the uniform estimates derived in Lemmas 2.1–2.4 are independent of the nonlocal kernels, they remain valid in the limiting process. This enables us to transfer the asymptotic properties of the limiting local reaction–diffusion system to the original nonlocal model, leading to the following asymptotic result.

Theorem 1.2. *Assume that the hypotheses of Theorem A.1 hold. Let* $u = (S, I, R)$ *denote the limiting local solution obtained in Theorem A.1. Assume further that* $\mu_0 + \gamma_0 > \beta^* C_3$ *and that* $S_0(x) \le S^*(x)$ *in* Ω, *where* S^* *is the disease-free steady state of the limiting local system. Then*

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

The main analytical contributions of this paper may be summarized as follows.

(i) Unlike many existing studies, our analytical framework allows the susceptible, infected, and recovered populations to possess distinct compartment-dependent nonlocal diffusion coefficients, providing a more realistic description of heterogeneous dispersal while introducing additional mathematical challenges.

(ii) We develop a robust L^p-energy framework that yields uniform *a priori* estimates despite the distinct nonlocal diffusion operators and nonlinear epidemic interactions. These estimates are fundamental to the asymptotic analysis.

(iii) Since convolution-type nonlocal diffusion operators lack the classical parabolic smoothing property, we employ a diffusive-limit approach to establish the asymptotic behavior by proving convergence of the nonlocal model to its corresponding local reaction–diffusion counterpart.

The remainder of this paper is organized as follows. In the next section, we prove Theorems 1.1 and 1.2. Section 3 presents numerical simulations illustrating the effects of spatial heterogeneity, the interaction radius of the Gaussian kernel, and intermittent treatment on the dynamics of system (1.1). Concluding remarks are given in Section 4, while the proof of the diffusive limit result, Theorem A.1, is deferred to Appendix A.

2 Proof of Main Theorems

Since the equation for R is linear with forcing terms depending only on I, it suffices to analyze the subsystem consisting of S and I. The component R is then uniquely determined by the corresponding linear nonhomogeneous nonlocal equation.

2.1 Proof of Theorem 1.1

Lemma 2.1. *Let* (S, I) *be the unique nonnegative maximal solutions of the system (1.1) on the interval* $[0, T_m)$ *with* $T_m \in (0, \infty]$. *Then there exists an* $M_{T_m} > 0$ *such that* $\int_\Omega \big(S + I\big)dx \le M_{T_m}$ *for any* $\mu_0 > 0$.

Proof of Lemma 2.1: Let $u = (u_1, u_2) \equiv (S, I)$. Adding the first two equations of the system (1.1) and then integrating over Ω we get,

$$\begin{aligned}\int_\Omega \sum_{i=1}^{2} u_{i_t}(x,t)dx &\leq \int_\Omega \sum_{i=1}^{2} d_i\Gamma_i u_i(x,t)dx + \int_\Omega b(x)dx - \int_\Omega \mu(x)\sum_{i=1}^{2} u_i(x,t)dx \\ &\leq b^* \int_\Omega dx - \mu_0 \int_\Omega \sum_{i=1}^{2} u_i(x,t)dx \end{aligned} \tag{2.1}$$

here we used $\phi(x,y) = \phi(y,x)$ to get

$$\begin{aligned}\int_\Omega \sum_{i=1}^{2} d_i\Gamma_i u_i(x,t)dx &= \int_\Omega \sum_{i=1}^{2} d_i\Gamma u_i(x,t)dx = \int_\Omega\int_\Omega \sum_{i=1}^{2} d_i\phi(x,y)(u_i(y,t) - u_i(x,t))dydx \\ &= \int_\Omega\int_\Omega \sum_{i=1}^{2} d_i\phi(x,y)u_i(y,t)dydx - \int_\Omega\int_\Omega \sum_{i=1}^{2} d_i\phi(x,y)u_i(x,t)dydx \\ &= \int_\Omega\int_\Omega \sum_{i=1}^{2} d_i\phi(x,y)u_i(y,t)dydx - \int_\Omega\int_\Omega \sum_{i=1}^{2} d_i\phi(x,y)u_i(y,t)dydx \\ &= 0.\end{aligned}$$

Now, using the Grönwall's inequality to the above differential inequality in (2.1) we get,

$$\int_\Omega \Big(S(x,t) + I(x,t)\Big)dx \leq M_{T_m}, \quad t \in [0, T_m)$$

where $M_{T_m} := \max\Big\{\int_\Omega \Big(S_0(x) + I_0(x)\Big)dx, \frac{b^*|\Omega|}{\mu_0}\Big\}$. □

We are now ready to prove Theorem 1.1 and we use semigroup theory [30] for this purpose. Assume that (D1) holds, and set $X = C(\overline{\Omega})$, $X^2_+ = \{z = (z_1, z_2) \in X^2 : z_i \geq 0 \text{ on } \overline{\Omega},\ i = 1, 2\}$. For $i \in \{S, I\}$, let Γ_i be defined by (1.2). Then $\Gamma_i \in \mathcal{L}(X)$ and $\|\Gamma_i\|_{\mathcal{L}(X)} \leq 2\lambda$. Consequently, $A_i z = d_i\Gamma_i z$ generates a uniformly continuous semigroup $\{T_i(t)\}_{t\geq 0}$ on X, for each $i \in \{S, I\}$.

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

$$\mathcal{A} = diag(A_S, A_I)$$

Since each diagonal operator generates semigroup on X, it follows from standard product semigroup theory that $\mathcal{A}$ generates uniformly continuous semigroup on $\mathbb{X}$,see, for instance, [5,30]. 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 1.1: Under the above setting, standard semilinear evolution theory [30] guarantees that system (1.1) admits a unique solution

$$u = (u_i)_{i=1}^2 \in C^1([0, T_{\max}); X^2)$$

defined on a maximal interval $[0, T_m)$, where $T_m \in (0, \infty]$. Let $u = (u_1, u_2) \equiv (S, I)$ and $u_0 = (u_{1,0}, u_{2,0}) \equiv (S_0, I_0)$. Then, for $i = 1, 2$, the variation-of-constants formula is

$$u_i(t) = T_i(t)u_{i,0} + \int_0^t T_i(t-\tau)F_i(\cdot, \tau, u(\tau))\, d\tau. \tag{2.2}$$

Since the reaction terms satisfy the quasi-positivity condition (Qp), standard comparison arguments along with nonnegative initial data imply that the maximal solution remains component-wise nonnegative.

To establish global existence, it remains to exclude finite-time blow-up. By the continuation principle, either $T_m = \infty$, or

$$\limsup_{t \to T_m^-} \|u(t)\|_{X^2} = \infty. \tag{2.3}$$

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

Define $A_{11} : X \to X$, $A_{11}z = d_1\Gamma_1 z - \mu_0 z$, then $A_{11}(z)$ generates $T_{11}(t)$ on X. Since u_i are nonnegative for $i = 1, 2$, the first equation of (1.1) yields

$$u_{1_t}(x,t) \le d_1\Gamma_1 u_1(x,t) + b(x) - \mu(x)u_1(x,t) \le d_1\Gamma_1 u_1(x,t) + b^* - \mu_0 u_1(x,t)$$

Applying the variation-of-constants formula yields

$$u_1(x,t) \le T_{11}(t)u_{1,0}(x) + b^* \int_0^t T_{11}(t-\tau)\mathbf{1}\, d\tau \le \|T_{11}(t)u_{1,0}\|_{\infty,\Omega} + b^* \int_0^t \|T_{11}(t-\tau)\|_{\infty,\Omega}\, d\tau$$

$$\therefore\ \|u_1(\cdot,t)\|_{\infty,\Omega} \le C_3, \quad t \ge 0, \qquad \left[C_3 := \max\left\{\|S_0\|_{\infty,\Omega}, \frac{b^*}{\mu_0}\right\}\right]. \tag{2.4}$$

Now, we add the first two equations of (1.1) to obtain

$$\sum_{i=1}^{2} u_{i_t}(x,t) = \sum_{i=1}^{2} d_i\Gamma_i u_i(x,t) + b(x) - \mu(x)\sum_{i=1}^{2} u_i(x,t) - \gamma(x,t)u_2(x,t) \le \sum_{i=1}^{2} d_i\Gamma_i u_i(x,t) + b^* - \mu_0 \sum_{i=1}^{2} u_i(x,t)$$

But

$$\begin{aligned}
\sum_{i=1}^{2} d_i\Gamma_i u_i(x,t) = \sum_{i=1}^{2} d_i\Gamma u_i(x,t) &= \int_\Omega \phi(x,y)\sum_{i=1}^{2} d_i u_i(y,t)\, dy - \sum_{i=1}^{2} d_i u_i(x,t)\int_\Omega \phi(x,y)\, dy \\
&\le d_{\max}\int_\Omega \phi(x,y)\sum_{i=1}^{2} u_i(y,t)\, dy - d_{\min}\sum_{i=1}^{2} u_i(x,t)\int_\Omega \phi(x,y)\, dy \\
&\le d_{\max}\lambda^*\int_\Omega \sum_{i=1}^{2} u_i(y,t)\, dy - d_{\min}\varepsilon\sum_{i=1}^{2} u_i(x,t) \\
&\le d_{\max}\lambda^* M_{T_m} - d_{\min}\varepsilon\sum_{i=1}^{2} u_i(x,t)
\end{aligned}$$

where $d_{\max} := \max_{i=1,2}\{d_i\}$, $d_{\min} := \min_{i=1,2}\{d_i\}$, $\lambda^* := \|\phi\|_{\infty,\Omega\times\Omega}$. Since $\phi \in C(\overline{\Omega}\times\overline{\Omega}; \mathbb{R}_+)$, it follows that $\lambda^* < \infty$. Here, we used Hölder's inequality together with Lemma 2.1 to estimate the first term, while the second term follows from the lower bound in (1.4). Hence

$$\sum_{i=1}^{2} u_{i_t}(x,t) \le d_{\max}\lambda^* M_{T_m} + b^* - \left(\mu_0 + d_{\min}\varepsilon\right)\sum_{i=1}^{2} u_i(x,t).$$

Applying the variation-of-constants formula to the above differential inequality yields

$$\|u_1(\cdot,t) + u_2(\cdot,t)\|_{\infty,\Omega} \le C_4, \qquad t \in [0, T_m), \tag{2.5}$$

where $C_4 := \max\left\{\|S_0 + I_0\|_{\infty,\Omega}, \frac{d_{\max}\lambda^* M_{T_m} + b^*}{\mu_0 + d_{\min}\varepsilon}\right\}$ Hence the solution remains uniformly bounded on $[0, T_m)$, so the continuation criterion (2.3) precludes finite-time blow-up. Therefore $T_m = \infty$. □

2.2 Proof of Theorem 1.2

The following lemma establishes a robust uniform $L^p(\Omega)$-estimate for solutions of system (1.1) for every $2 \le p < \infty$.

Lemma 2.2. *Assume that the hypotheses of Lemma 2.1 hold. Then, for every $p \in \mathbb{N}$ with $p \ge 2$, there exists a constant $L_p > 0$, independent of kernel $\phi(x,y)$ of nonlocal diffusion operator such that*

$$\|(S,I)(\cdot,t)\|_{L^p(\Omega)} \le L_p, \qquad \forall\, t \ge 0.$$

Proof: To establish the $L^p(\Omega)$-estimates, we introduce the following Lyapunov-type functional, which was originally proposed for two-component systems in [24] and later extended to m-component systems in [1]. We assume $u = (u_1, u_2) \equiv (S, I)$, $\theta = (\theta_1, \theta_2)$, and $a > 0$ (to be determined) to define

$$\mathcal{L}^p(u)(t) = \int_\Omega \sum_{\theta \in \mathbb{Z}^2_+,\ |\theta|=p} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx$$

Differentiating with respect to time we get,

$$\begin{aligned}
\mathcal{L}^p(u)'(t) &= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \Big(\sum_{i=1}^2 a^{2\theta_i+1} u_{i_t}(x,t) \Big) dx \\
&= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \left(\sum_{i=1}^2 a^{2\theta_i+1} \Big(d_i \Gamma_i u_i(x,t) + F_i(x,t,u) \Big) \right) dx \\
&= I + J
\end{aligned} \tag{2.6}$$

where

$$\begin{aligned}
I &= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \sum_{i=1}^2 a^{2\theta_i+1} d_i \Gamma_i u_i(x,t) dx \\
&= \int_\Omega \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \sum_{i=1}^2 a^{2\theta_i+1} d_i \phi(x,y)(u_i(y,t) - u_i(x,t)) dy dx \\
&= \int_\Omega \int_\Omega \phi(x,y) \sum_{i=1}^2 U_i(v(x,t)) \psi_i(x,y,t) dy dx \qquad \Big[\psi_i(x,y,t) = u_i(y,t) - u_i(x,t),\ i = 1,2 \Big]
\end{aligned}$$

where

$$\sum_{i=1}^2 U_i(v(x,t)) = \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \sum_{i=1}^2 a^{2\theta_i+1} d_i \ \text{ and } \ v(\cdot,t) = \Big(S(\cdot,t), I(\cdot,t) \Big)$$

Due to the fact that $\phi(x,y) = \phi(y,x) \ge 0$, for any $\Theta > 0$ we have

$$\int_\Omega \int_\Omega v(x,t)^\Theta \phi(x,y)(v(y,t) - v(x,t)) dx dy = -\frac{1}{2} \int_\Omega \int_\Omega (v(y,t)^\Theta - v(x,t)^\Theta) \phi(x,y)(v(y,t) - v(x,t)) dx dy$$

Using this estimate we can write

$$\begin{aligned}
I &= -\frac{1}{2} \int_\Omega \int_\Omega \phi(x,y) \sum_{i=1}^2 \Big(U_i(v(y,t)) - U_i(v(x,t)) \Big) \psi_i(x,y,t) dy dx \\
&= -\frac{1}{2} \int_\Omega \int_\Omega \phi(x,y) \Big(U(v(y,t),x,y,t) - U(v(x,t),x,y,t) \Big) dy dx
\end{aligned}$$

where we used $U(v,x,y,t) = \sum_{i=1}^{2} U_i(v)\psi_i(x,y,t)$. Now, using Mean Value Theorem (differentiating with respect to $\{S,I\}$) we can write

$$\begin{aligned} I &= -\frac{1}{2}\int_\Omega\int_\Omega \phi(x,y)\sum_{i\in\{S,I\}}\Big(U_i(\tilde{v}(y,t)x,y,t) - U_i(\tilde{v}(x,t),x,y,t)\Big)dxdy \\ &= -\frac{1}{2}\int_\Omega\int_\Omega \phi(x,y)\Big[U_S(\tilde{v}(x,y,t),x,y,t)\psi_S + U_I(\tilde{v}(x,y,t),x,y,t)\psi_I\Big]dxdy \end{aligned} \tag{2.7}$$

where $\tilde{v}(x,y,t) = v(x,t) + K(v(x,t),v(y,t),x,y,t)(v(y,t)-v(x,t))$ and $0 < K(v(x,t),v(y,t),x,y,t) < 1$. Also, $\psi_i = \psi_i(x,y,t),\ i \in \{S,I\}$. Now,

$$U_S(\tilde{v}(x,y,t),x,y,t) = \frac{\partial}{\partial\tilde{S}}\left(\sum_{i=1}^{2} U_i(\tilde{v})\psi_i\right) = \frac{\partial}{\partial\tilde{S}}\left(\sum_{|\theta|=p-1}\binom{p}{\theta}a^{\sum_{i=1}^{2}\theta_i^2}\Pi_{i=1}^{2}\tilde{u_i}(x,t)^{\theta_i}\sum_{i=1}^{2}a^{2\theta_i+1}d_i\psi_i\right) = a_1 + b_1$$

Now,

$$\begin{aligned} a_1 &= \frac{\partial}{\partial\tilde{S}}\left(\sum_{|\theta|=p-1}\binom{p}{\theta}a^{\sum_{i=1}^{2}\theta_i^2}\Pi_{i=1}^{2}\tilde{u_i}(x,t)^{\theta_i}a^{2\theta_1+1}d_S\psi_S\right) \\ &= \sum_{\theta_1+\theta_2=p-1}\binom{p}{\theta_1\ \theta_2}a^{\theta_1^2+\theta_2^2}\theta_1\tilde{S}(x,y,t)^{\theta_1-1}\tilde{I}(x,y,t)^{\theta_2}a^{2\theta_1+1}d_S\psi_S(x,y,t) \\ &= \sum_{\theta_1+\theta_2=p-1}\frac{1}{\theta_1!}\binom{p}{\theta_2}a^{\theta_1^2+\theta_2^2}\theta_1\tilde{S}(x,y,t)^{\theta_1-1}\tilde{I}(x,y,t)^{\theta_2}a^{2\theta_1+1}d_S\psi_S(x,y,t) \\ &= \sum_{\theta_1+\theta_2=p-1}\frac{1}{(\theta_1-1)!}\binom{p}{\theta_2}a^{\theta_1^2+\theta_2^2}\tilde{S}(x,y,t)^{\theta_1-1}\tilde{I}(x,y,t)^{\theta_2}a^{2\theta_1+1}d_S\psi_S(x,y,t) \\ &= \sum_{(\theta_1-1)+\theta_2=p-2}\frac{1}{(\theta_1-1)!}\binom{p}{\theta_2}a^{(\theta_1-1)^2+\theta_2^2}\tilde{S}(x,y,t)^{\theta_1-1}\tilde{I}(x,y,t)^{\theta_2}a^{4(\theta_1-1)+4}d_S\psi_S(x,y,t) \\ &= \sum_{\theta_1+\theta_2=p-2}\frac{1}{\theta_1!}\binom{p}{\theta_2}a^{\theta_1^2+\theta_2^2}\tilde{S}(x,y,t)^{\theta_1}\tilde{I}(x,y,t)^{\theta_2}a^{4\theta_1+4}d_S\psi_S(x,y,t) \\ &= \sum_{\theta_1+\theta_2=p-2}\binom{p}{\theta_1\ \theta_2}a^{\theta_1^2+\theta_2^2}\tilde{S}(x,y,t)^{\theta_1}\tilde{I}(x,y,t)^{\theta_2}a^{4\theta_1+4}d_S\psi_S(x,y,t) \\ \therefore\ a_1 &= \sum_{\theta=0}^{p-2}\binom{p}{\theta}a^{\theta_1^2+\theta_2^2}\tilde{S}(x,y,t)^{\theta_1}\tilde{I}(x,y,t)^{\theta_2}a^{4\theta_1+4}d_S\psi_S(x,y,t) \end{aligned}$$

Using similar technique, we can find b_1 and similar estimates for $U_I(\tilde{v}(x,y,t),x,y,t)$. Then replacing these into (2.7) we can write

$$\begin{aligned} I &= -\frac{1}{2}\int_\Omega\int_\Omega \phi(x,y)\Big[(a_1+b_1)\psi_S + (a_2+b_2)\psi_I\Big]dxdy \\ &= -\frac{1}{2}\int_\Omega\int_\Omega \phi(x,y)\sum_{|\theta|=p-2}\binom{p}{\theta}a^{\theta_1^2+\theta_2^2}\tilde{S}(x,y,t)^{\theta_1}\tilde{I}(x,y,t)^{\theta_2}\begin{pmatrix}\psi_S(x,y,t) & \psi_I(x,y,t)\end{pmatrix} \\ &\begin{pmatrix} d_S a^{4\theta_1+4} & \frac{d_S+d_I}{2}a^{2\theta_1+1}a^{2\theta_2+1} \\ \frac{d_S+d_I}{2}a^{2\theta_1+1}a^{2\theta_2+1} & d_I a^{4\theta_2+4}\end{pmatrix}\begin{pmatrix}\psi_S(x,y,t)\\ \psi_I(x,y,t)\end{pmatrix}dxdy. \end{aligned}$$

If $a > \sqrt{\frac{d_S+d_{I_1}}{2\sqrt{d_S d_{I_1}}}}$, then using Sylvester's Criterion we can write the quadratic term in I above as $z^TAz \geq 0\ \forall$

$z \in \mathbb{R}^2$ where $A = \begin{pmatrix} d_S a^{4\theta_1+4} & \frac{d_S+d_I}{2} a^{2\theta_1+1} a^{2\theta_2+1} \\ \frac{d_S+d_I}{2} a^{2\theta_1+1} a^{2\theta_2+1} & d_I a^{4\theta_2+4} \end{pmatrix}$ and $z = \psi_i(x, y, t)$ for all $i \in \{S, I\}$. Therefore, we have

$$I \leq 0. \tag{2.8}$$

We also have,

$$\begin{aligned} J &= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \left(\sum_{i=1}^2 a^{2\theta_i+1} F_i(x,t,u) \right) dx \\ &= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \Big[(a^{2\theta_1+1} - a^{2\theta_2+1}) F_1(x,t,u) + a^{2\theta_2+1} \Big(F_1(x,t,u) + F_2(x,t,u) \Big) \Big] dx \\ &= J_1 + J_2 \end{aligned} \tag{2.9}$$

If $a > 1$ and $\theta_1 > \theta_2$ then $(a^{2\theta_1+1} - a^{2\theta_2+1}) > 0$. Now,

$$\begin{aligned} J_1 &= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} (a^{2\theta_1+1} - a^{2\theta_2+1}) F_1(x,t,u) dx \\ &= \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} (a^{2\theta_1+1} - a^{2\theta_2+1}) \Big(b(x) - \beta(x) S(x,t) - \mu(x) S(x,t) \Big) dx \\ &\leq \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx \left\| (a^{2\theta_1+1} - a^{2\theta_2+1}) b(x) \right\|_{\infty,\Omega} \\ \therefore\ J_1 &\leq b^* \Big(a^{2\theta_1+1} + a^{2\theta_2+1} \Big) \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx \end{aligned}$$

where we used Hölder's inequality. Similarly,

$$\begin{aligned} J_2 &\leq \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} a^{2\theta_2+1} \Big(b(x) - \mu(x) \sum_{i=1}^2 u_i(x,t) \Big) dx \\ &\leq b^* a^{2\theta_2+1} \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx - \mu_0 a^{2\theta_2+1} \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} \sum_{i=1}^2 u_i(x,t) dx \\ &\leq b^* a^{2\theta_2+1} \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx - a^{2\theta_2+1} \mu_0 p! \int_\Omega \sum_{|\theta|=p} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx \end{aligned}$$

Replacing J_1, J_2 in (2.9) we get,

$$\begin{aligned} J &\leq b^* \Big(a^{2\theta_1+1} + 2a^{2\theta_2+1} \Big) \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx - a^{2\theta_2+1} \mu_0 p! \int_\Omega \sum_{|\theta|=p} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx \\ \therefore\ J &\leq C_1 \int_\Omega \sum_{|\theta|=p-1} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx - a^{2\theta_2+1} \mu_0 p! \int_\Omega \sum_{|\theta|=p} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx \end{aligned}$$

Since $a > 1$, $a > \sqrt{\frac{d_S+d_{I_1}}{2\sqrt{d_S d_{I_1}}}}$, $\theta_1 > \theta_2$, and by considering $a \geq 1$ we can write,

$$\mathcal{L}^p(u)(t) = \int_\Omega \sum_{|\theta|=p} \binom{p}{\theta} a^{\sum_{i=1}^2 \theta_i^2} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx \leq \max\{a\} \int_\Omega \sum_{|\theta|=p} \binom{p}{\theta} \Pi_{i=1}^2 u_i(x,t)^{\theta_i} dx.$$

So, if, $a > \max\left\{1, \sqrt{\frac{d_S+d_{I_1}}{2\sqrt{d_S d_{I_1}}}}\right\}$ and $\theta_1 > \theta_2$, then we get,

$$J \le C_1\mathcal{L}^{p-1}(u)(t) - \frac{p!a^{2\theta_2+1}\mu_0}{\max\{a\}}\mathcal{L}^p(u)(t)$$
$$\therefore\ J \le C_1\mathcal{L}^{p-1}(u)(t) - C_2\mathcal{L}^p(u)(t)$$

Replacing I and J in (2.6) we get,

$$\mathcal{L}^p(u)'(t) \le -I + C_1\mathcal{L}^{p-1}(u)(t) - C_2\mathcal{L}^p(u)(t) \le C_1\mathcal{L}^{p-1}(u)(t) - C_2\mathcal{L}^p(u)(t) \tag{2.10}$$

Taking $p = 2$ we get,

$$\mathcal{L}^2(u)'(t) + C_2\mathcal{L}^p(u)(t) \le C_1\mathcal{L}^1(u)(t) \le C_1 M_{T_m} \qquad \text{(by Lemma 2.1)}$$

using Grönwall's inequality we get,

$$\mathcal{L}^2(u)(t) \le M_2, \quad \forall\ t \in [0, T_m) \text{ and for any } M_2 > 0.$$

Proceeding inductively by taking $p = 3, 4, \ldots$ in (2.10), we conclude that for every $t \ge 0$ and each $2 \le p < \infty$, there exists a constant $M_p > 0$ such that

$$\mathcal{L}^p(u)(t) \le M_p.$$

Moreover, since $\theta \in (0, \infty)^2$, there exist positive constants ε_1 and ε_2, depending only on θ, such that

$$\varepsilon_1\|u(\cdot,t)\|_{L^p(\Omega)} \le \left(\mathcal{L}^p(u)(t)\right)^{\frac{1}{p}} \le \varepsilon_2\|u(\cdot,t)\|_{L^p(\Omega)}, \qquad 2 \le p < \infty.$$

Hence,

$$\|u(\cdot,t)\|_{L^p(\Omega)} \le L_p, \qquad \forall\ t \ge 0,\ 2 \le p < \infty,$$

for some constant $L_p > 0$. □

The proof of the next result follows the same strategy as Lemma 2.1, but additionally exploits the decay induced by the treatment term together with the uniform $L^\infty(\Omega)$-bound established in Theorem 1.1.

Lemma 2.3. *Assume that the hypotheses of Theorem 1.1 and Lemma 2.2 hold. If $\mu_0 + \gamma_0 > \beta^* C_3$ then*

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

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

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

Proof: Integrating the second equation of system (1.1) over Ω we obtain,

$$\begin{aligned}\int_\Omega I_t(x,t)dx &= \int_\Omega d_I\Gamma_I I(x,t)dx + \int_\Omega \beta(x)S(x,t)I(x,t)dx - \int_\Omega \Big(\mu(x)+\gamma(x,t)\Big)I(x,t)dx\\ &\le \beta^* C_3\int_\Omega I(x,t)dx - (\mu_0+\gamma_0)\int_\Omega I(x,t)dx\end{aligned}$$

For $\mu_0 + \gamma_0 > \beta^* C_3$, Grönwall's inequality applied to the above differential inequality yields

$$\int_\Omega I(x,t)\,dx \le e^{-(\mu_0+\gamma_0-\beta^* C_3)t}\int_\Omega I_0(x)\,dx. \tag{2.11}$$

Integrating this estimate over the interval $(T, T+1)$ gives

$$\lim_{T\to\infty} \int_T^{T+1} \int_\Omega I(x,t)dxdt = 0. \tag{2.12}$$

Now, by virtue of Lemma 2.2, the infected population I is uniformly bounded in $L^p(\Omega)$ for every $1 < p < \infty$. Integrating the estimate of Lemma 2.2 over the time interval $(T, T+1)$, we obtain

$$\|I\|_{L^p(\Omega\times(T,T+1))} \le C_p, \qquad \forall\, T \ge 0, \quad 1 < p < \infty, \quad j = 1, 2, \tag{2.13}$$

where $C_p > 0$ is independent of T.

Let $q > 1$. Since

$$I^q = I^{\frac{1}{2}} I^{q-\frac{1}{2}},$$

Hölder's inequality yields

$$\int_T^{T+1} \int_\Omega I(x,t)^q \, dx\, dt \le \left(\int_T^{T+1} \int_\Omega I(x,t)\, dx\, dt \right)^{\frac{1}{2}} \left(\int_T^{T+1} \int_\Omega I(x,t)^{2q-1}\, dx\, dt \right)^{\frac{1}{2}}.$$

Combining (2.12), (2.13), and the nonnegativity of I, we infer that

$$\int_T^{T+1} \int_\Omega I(x,t)^q \, dx\, dt \longrightarrow 0 \qquad \text{as } T \to \infty.$$

Consequently,

$$\|I\|_{L^q(\Omega\times(T,T+1))} \longrightarrow 0 \qquad \text{as } T \to \infty, \quad 1 \le q < \infty. \tag{2.14}$$

This completes the proof. □

We assume that S^* denotes the disease-free steady state, given by the unique nonnegative solution of

$$d_S \Gamma_S S^*(x) + b(x) - \mu(x) S^*(x) = 0, \qquad x \in \Omega. \tag{2.15}$$

Since $b(x) \ge 0$, $b \not\equiv 0$, $\mu(x) \ge \mu_0 > 0$, and $\phi(x, y) \ge 0$, the positivity of the resolvent associated with the operator $d_S\Gamma_S - \mu(x)$ implies that

$$S^*(x) \ge 0, \qquad x \in \Omega.$$

Lemma 2.4. *Assume that the hypotheses of Theorem 1.1, Lemma 2.2 hold and that the condition of Lemma 2.3 is satisfied. If $S_0(x) \le S^*(x)$ in Ω for any S^* satisfying* (2.15)*, then the susceptible population converges to the disease-free steady state S^* on sliding time intervals. More precisely,*

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

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

Proof: We assume $S^*(x)$ is the disease-free steady state satisfying (2.15). Now, from the first equation of system (1.1) we can write,

$$0 = d_S \Gamma_S S^*(x) + b(x) - \mu(x) S^*(x), \quad x \in \Omega.$$

Since, $b(x) \in C(\overline{\Omega})$, $S^* \in L^\infty(\Omega)$ for all $x \in \Omega$ and we define

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

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

$$\begin{aligned} z_t(x,t) &= -S_t(x,t) = -d_S\Gamma_S S(x,t) - b(x) + \mu(x)S(x,t) + \beta(x)S(x,t)I(x,t) \\ &= -d_S\Gamma_S S(x,t) + d_S\Gamma_S S^*(x) - \mu(x)S^*(x) + \mu(x)S(x,t) + \beta(x)S(x,t)I(x,t) \\ &= d_S\Gamma_S(S^*(x) - S(x,t)) - \mu(x)(S^*(x) - S(x,t)) + \beta(x)S(x,t)I(x,t) \\ &= d_S\Gamma_S z(x,t) - \mu(x)z(x,t) + \beta(x)S(x,t)I(x,t) \end{aligned}$$

Since $z(x,0) \geq 0$ along with $S, I, \beta, \mu \geq 0$, and the semigroup generated by $d_S\Gamma_S - \mu(x)$ is positive, the comparison principle gives

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

Hence,

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

Integrating the equation satisfied by z over Ω gives

$$\begin{aligned} \frac{d}{dt}\int_\Omega z(x,t)\,dx &= \int_\Omega d_S\Gamma_S z(x,t)\,dx - \int_\Omega \mu(x)z(x,t)\,dx + \int_\Omega \beta(x)S(x,t)I(x,t)\,dx \\ &\leq -\mu_0 \int_\Omega z(x,t)\,dx + \beta^* C_3 \int_\Omega I(x,t)\,dx \end{aligned}$$

If we consider

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

then for $\mu_0 + \gamma_0 > \beta^* C_3$, using the exponential estimate established in Lemma 2.3, (i.e. $\int_\Omega I(x,t)\,dx \leq e^{-(\mu_0+\gamma_0-\beta^*C_3)t}\int_\Omega I_0(x)\,dx$) we can write,

$$Z'(t) + \mu_0 Z(t) \leq \beta^* C_3 e^{-(\mu_0+\gamma_0-\beta^*C_3)t}\int_\Omega I_0(x)dx$$

Since $(\mu_0 + \gamma_0 - \beta^* C_3) > 0$ and $\mu_0 > 0$, multiplying the above inequality by $e^{\mu_0 t}$, integrating from 0 to t, and sending $t \to \infty$, we get

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

Since $z(x,t) \geq 0$,

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

Consequently,

$$\int_T^{T+1}\int_\Omega |S(x,t) - S^*(x)|\,dxdt = \int_T^{T+1} Z(t)dt \to 0, \qquad \text{as } T \to \infty. \tag{2.16}$$

Since $S^* \in L^\infty(\Omega)$ and Ω is bounded, it follows that $S^* \in L^p(\Omega)$ for every $1 \leq p < \infty$. Therefore, using the $L^p(\Omega)$-bound for S established in Lemma 2.2, we obtain

$$\|S - S^*\|_{L^p(\Omega\times(T,T+1))} \leq \|S\|_{L^p(\Omega\times(T,T+1))} + \|S^*\|_{L^p(\Omega\times(T,T+1))} \leq L_p + \|S^*\|_{L^p(\Omega)} =: C_p^*, \qquad T \geq 0.$$

Combining the uniform L^p-bound above with (2.16) and applying the same interpolation argument as in Lemma 2.3, we conclude that

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

for every $1 \le q < \infty$. □

Proof of Theorem 1.2: Let (S, I) denote the limiting local susceptible and infected components obtained in Theorem A.1. Since the recovered component satisfies a linear parabolic equation driven by the infected population, it can be determined uniquely once I is known. Therefore, it suffices to establish the asymptotic behavior of S and I. Since the nonlocal diffusion operators satisfy

$$\int_\Omega \Gamma_i w(x,t)\,dx = 0,$$

the estimates obtained in Lemma 2.1– 2.4 are independent of the particular choice of the nonlocal kernels. Therefore, they remain valid for the limiting local solution $u = (S, I)$ obtained through Theorem A.1.

The pair (S, I) is the unique global classical solution of the corresponding limiting local susceptible–infected subsystem obtained in Theorem A.1. Moreover, by the global boundedness result for the limiting local system, there exists a constant $M > 0$ such that

$$0 \le S(x,t),\ I(x,t) \le M, \qquad x \in \Omega,\ t \ge 0.$$

We first prove the decay of I. The infected component of the limiting local system satisfies

$$I_t = d_I \Delta I + g(x,t),$$

where

$$g(x,t) = \beta(x)S(x,t)I(x,t) - (\mu(x) + \gamma(x,t))I(x,t).$$

Using the uniform L^∞-bound for S, we obtain

$$|g(x,t)| \le (\beta^* M + \mu^* + \gamma^*)\, I(x,t).$$

Consequently, for every $1 \le p < \infty$,

$$\|g\|_{L^p(\Omega\times(T,T+1))} \le (\beta^* M + \mu^* + \gamma^*)\, \|I\|_{L^p(\Omega\times(T,T+1))} \to 0$$

as $T \to \infty$.

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

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

Equivalently,

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

Choose $p > \frac{n+2}{2}$. Then the parabolic Sobolev embedding theorem implies that

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

for some $\varepsilon \in (0,1)$ [26]. Hence I is uniformly Hölder continuous on $\overline{\Omega} \times [T, T+1]$, with Hölder constants independent of T.

Combining this uniform Hölder continuity with

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

and applying the standard contradiction argument based on the uniform Hölder continuity and the decay of the L^p-norms on sliding time intervals, we obtain

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

Next, define

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

Since S^* is the disease-free steady state of the limiting local system, z satisfies

$$z_t = d_S \Delta z - \mu(x) z + \beta(x) S(x,t) I(x,t).$$

Set

$$\mathcal{G}(x,t) := -\mu(x) z(x,t) + \beta(x) S(x,t) I(x,t).$$

Using the uniform L^∞-bound for S, we have

$$|\mathcal{G}(x,t)| \le \mu^* |z(x,t)| + \beta^* M I(x,t).$$

Therefore, for every $1 \le p < \infty$,

$$\|\mathcal{G}\|_{L^p(\Omega\times(T,T+1))} \le \mu^* \|z\|_{L^p(\Omega\times(T,T+1))} + \beta^* M \|I\|_{L^p(\Omega\times(T,T+1))} \to 0$$

as $T \to \infty$. The same maximal L^p-regularity argument applied to

$$z_t = d_S \Delta z + \mathcal{G}(x,t),$$

there exists a constant $K_p > 0$, independent of T, such that

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

Choosing $p > \frac{n+2}{2}$, the parabolic Sobolev embedding gives uniform Hölder continuity of z on $\overline{\Omega} \times [T, T+1]$. Since

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

the same contradiction argument yields

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

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

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

This completes the proof. □

3 Numerical Simulations

This section presents numerical simulations to complement the theoretical results established in Section 2. The objectives are to illustrate the qualitative behavior of the proposed epidemic model and investigate the effects of intermittent treatment on disease evolution.

Simulations are performed on the square domain $\Omega = (0,10) \times (0,10)$ using a uniform 128×128 spatial mesh over the time interval $[0, 200]$. To ensure a fair and direct comparison of the model dynamics as the interaction radius of the nonlocal kernel increases, all simulations employ the same computational domain, spatial discretization, initial conditions, parameter values, and intermittent treatment protocol.

Time integration is performed using a second-order Crank–Nicolson finite difference scheme for the diffusion terms, while the nonlinear reaction terms are treated explicitly. For the nonlocal model, the classical Laplace operator is replaced by the corresponding convolution-type nonlocal diffusion operator, whereas all remaining components of the numerical implementation are kept unchanged.

Table 1: Diffusion coefficients, initial conditions, and spatially heterogeneous coefficient functions employed in the numerical simulations.

Parameter	**Spatial form**	**Parameter**	**Spatial form**
(d_S, d_I, d_R)	$(0.10,\ 0.06,\ 0.05)$	$\mu(x,y)$	$0.01\left(1+0.75\cos^2\dfrac{\pi x}{L_x}\cos^2\dfrac{\pi y}{L_y}\right)$
$S(x,y,0)$	$100\left(1+0.20\cos\dfrac{\pi x}{L_x}\cos\dfrac{\pi y}{L_y}\right)$	$\gamma_1(x,y)$	$0.05\left(1+0.75\sin^2\dfrac{\pi x}{L_x}\sin^2\dfrac{\pi y}{L_y}\right)$
$I(x,y,0)$	$0.8\left(1+0.30\exp\left(-\dfrac{(x-5)^2+(y-5)^2}{3}\right)\right)+$ $0.3\left(1+0.50\exp\left(-\dfrac{(x-3)^2+(y-7)^2}{2}\right)\right)$	$\mathcal{R}(x,y)$	$\dfrac{0.65\,r_1(x,y)+0.35\,r_2(x,y)}{\max_{\overline{\Omega}}(0.65\,r_1+0.35\,r_2)}$
$R(x,y,0)$	0	$r_1(x,y)$	$\dfrac{\sin(\pi x/L_x)\sin\left(\pi y/L_y\right)}{\max_{\overline{\Omega}}\sin(\pi x/L_x)\sin\left(\pi y/L_y\right)}$
$b(x,y)$	$0.25\left(0.50+0.50\cos^2\dfrac{\pi x}{L_x}\cos^2\dfrac{\pi y}{L_y}\right)$	$r_2(x,y)$	$\dfrac{\exp\left(-\frac{(x-7)^2+(y-3)^2}{5}\right)}{\max_{\overline{\Omega}}\exp\left(-\frac{(x-7)^2+(y-3)^2}{5}\right)}$
$\beta(x,y)$	$0.006\,(0.25+0.75\,\mathcal{R}(x,y))$		

The diffusion coefficients, initial conditions and spatially heterogeneous coefficient functions employed throughout the numerical simulations are summarized in Table 1.

Since recovery in system (1.1) occurs exclusively through the treatment term $\gamma(x,y,t)I$, we choose the initial recovered population to be $R(x,y,0)=0$. Consequently, in the absence of treatment, where $\gamma(x,y,t)\equiv 0$, no individuals transition from the infected to the recovered class, and therefore the recovered population remains identically zero throughout the simulation. In contrast, under intermittent treatment, infected individuals recover during the treatment intervals, resulting in a positive recovered population.

Throughout this section, the intermittent treatment strategy is implemented by periodically activating and deactivating the spatial treatment coefficient according to

$$\gamma(x,y,t)=\begin{cases}0, & kT_\gamma \le t < (k+1)T_\gamma - L_\gamma,\\ \gamma_1(x,y), & (k+1)T_\gamma - L_\gamma \le t < (k+1)T_\gamma,\end{cases} \qquad k=0,1,2,\ldots, \tag{3.1}$$

where the spatially heterogeneous treatment coefficient $\gamma_1(x,y)$ is given in Table 1. Throughout the simulations, we choose the treatment period $T_\gamma = 20$ and treatment duration $L_\gamma = 10$. Consequently, treatment is inactive during the first half of each cycle and active during the final half, corresponding to a 50% duty cycle. The choice of $T_\gamma = 20$ provides a representative intervention cycle over the simulation interval, while $L_\gamma = 10$ allows equal durations of treatment and treatment-free phases. This periodic on–off protocol models intermittent public health interventions arising from limited medical resources, scheduled treatment campaigns, or temporary control measures. At the same time, the spatially varying coefficient $\gamma_1(x,y)$ permits the treatment intensity to differ across the computational domain, thereby accounting for geographical heterogeneity in healthcare resources and intervention effectiveness.

To incorporate spatial heterogeneity in disease transmission, we introduce the normalized spatial risk function $\mathcal{R}(x,y)$, defined in Table 1. The function $\mathcal{R}(x,y)$ combines the background distribution $r_1(x,y)$ with the localized hotspot distribution $r_2(x,y)$ to represent regions of varying transmission intensity. Specifically, $r_1(x,y)$ models gradual large-scale spatial variability, whereas $r_2(x,y)$ captures localized areas of elevated infection risk. Their weighted combination yields a heterogeneous transmission landscape, which is subsequently used to define the spatially varying transmission coefficient $\beta(x,y)$.

To facilitate comparison of the model dynamics for different interaction radii of the nonlocal kernel, we compute the spatial averages of the susceptible, infected, and recovered populations over the spatial

domain, defined by

$$\overline{U}(t) = \frac{1}{|\Omega|}\int_{\Omega} U(x,y,t)\,dx\,dy, \qquad U \in \{S, I, R\}, \tag{3.2}$$

which provide a concise measure of the overall epidemic dynamics while filtering out local spatial variations.

The nonlocal reaction–diffusion system (NRDS) in (1.1) can be written compactly as

$$U_t = D\Gamma U + F(x,t,U),$$

where F is the same reaction vector field and Γ denotes the normalized Gaussian nonlocal diffusion operator.

For $U \in \{S, I, R\}$, the nonlocal operator is implemented as

$$\Gamma U(x,t) = \int_{\Omega} \phi_\sigma(x,y)U(y,t) - U(x,t)\,dy \;\text{ with }\; \phi_\sigma(x,y) = \frac{1}{2\pi\sigma^2}\exp\left(-\frac{|x-y|^2}{2\sigma^2}\right).$$

$\sigma = 0.25, 1.0, 5.0$ are used to represent increasing interaction ranges, from relatively localized dispersal to broader long-range movement. The nonlocal diffusion terms are discretized within the same Crank–Nicolson framework as the local model. Since the convolution-based diffusion operator is applied through FFT-based matrix–vector products rather than being assembled explicitly, the resulting linear systems are solved iteratively using the BiCGSTAB (Bi-Conjugate Gradient Stabilized) method [34, 35].

Figure 1 illustrates the normalized Gaussian interaction kernel for three representative values of the interaction radius σ. As σ increases, the kernel becomes progressively broader and less concentrated around its center, indicating that individuals interact over increasingly larger spatial distances. Consequently, larger values of σ correspond to stronger long-range dispersal, whereas smaller values emphasize localized movement and interactions.

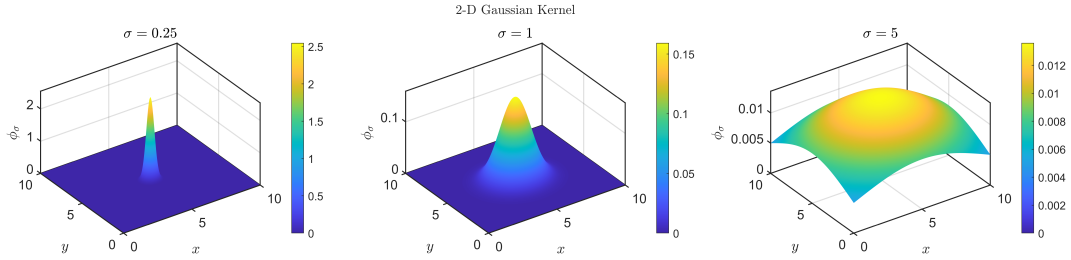


Figure 1: Gaussian interaction kernels corresponding to $\sigma = 0.25$, 1.0, and 5.0.

Figures 2– 4 present the simulation results for the nonlocal model described in system (1.1). Figure 2 and 3 show the final spatial distributions of the nonlocal reaction–diffusion system corresponding to the three interaction radii $\sigma = 0.25$, 1.0, and 5.0 at $t = 200$ of the susceptible, infected, and recovered populations with and without intermittent treatment, respectively.

As the interaction radius increases from $\sigma = 0.25$ to $\sigma = 5$, the final spatial distributions of S, I, and R exhibit noticeable quantitative changes while preserving their overall spatial patterns. In particular, the locations of the peaks and valleys remain similar for all three values of σ, although their magnitudes become less pronounced as σ increases. Thus, increasing the interaction radius influences the amplitude of the spatial distributions without altering their qualitative structure.

A more pronounced difference is observed between the cases with and without intermittent treatment. Without treatment, the infected population remains relatively large throughout the domain, whereas the recovered population is identically zero, as evidenced by the flat surface in the third column of Figure 3. When intermittent treatment is introduced, the infected population is substantially reduced for all values of σ, while a positive recovered population develops across the domain. The susceptible population also attains larger values under treatment than in the untreated case. These observations indicate that the treatment strategy has a considerably greater impact on the long-term spatial distributions than the variation of the interaction radius over the range considered.

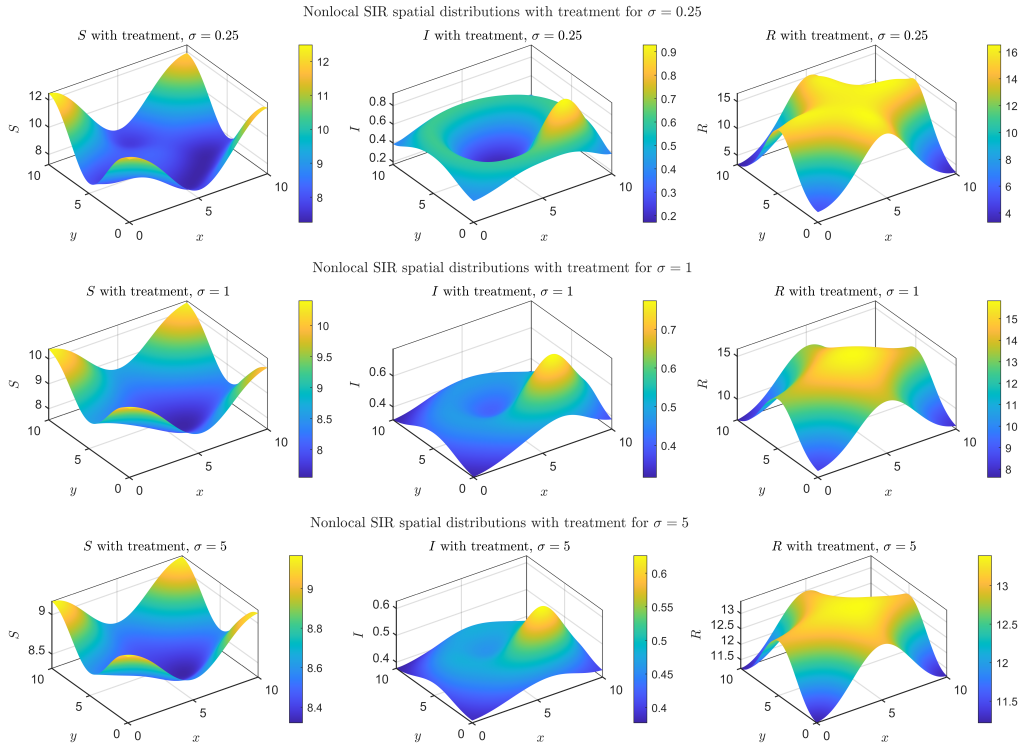


Figure 2: Final spatial profiles of S, I, and R for NRDS with intermittent treatment at $t = 200$ for $\sigma = 0.25$, 1.0, and 5.0.

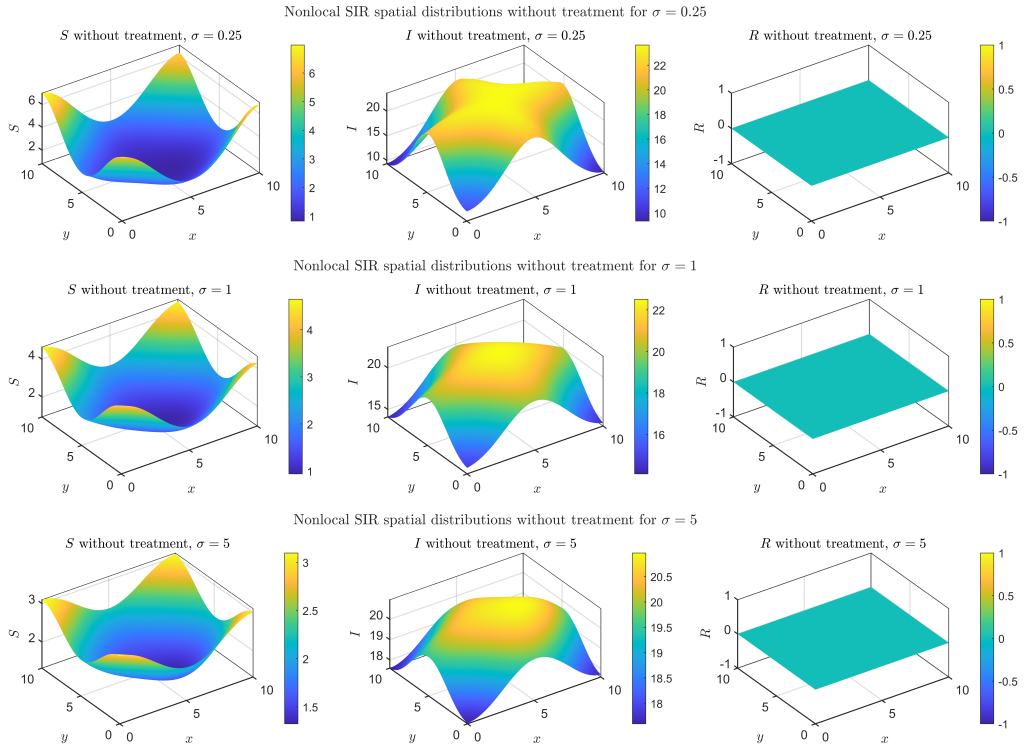


Figure 3: Final spatial profiles of S, I, and R for NRDS without intermittent treatment at $t = 200$ for $\sigma = 0.25$, 1.0, and 5.0.

Figure 4 illustrates the absolute differences in the spatial averages of the nonlocal model (1.1) for kernel radii $\sigma = 1$ and $\sigma = 5$, using the solution corresponding to $\sigma = 0.25$ as the reference. The top row presents the results with intermittent treatment, whereas the bottom row corresponds to the absence of treatment.

For the susceptible and infected populations, the discrepancies increase as the interaction radius becomes larger, indicating that broader nonlocal interactions produce progressively greater changes in the epidemic dynamics. In both treatment scenarios, the largest differences occur during the early stages of disease transmission, when the epidemic evolves most rapidly, and subsequently decrease as the solutions approach their long-time states.

The recovered population exhibits a different behavior. Under intermittent treatment, the discrepancy initially increases with the kernel radius but does not vary monotonically over the entire simulation, reflecting the fact that the recovered population accumulates through the treatment-induced transfer from the infected class. Consequently, its dynamics depend on the cumulative infection history rather than solely on the instantaneous effect of the interaction radius. In contrast, when intermittent treatment is absent, the recovered population remains identically zero throughout the simulation, that is, $R(x,t) \equiv 0$, and therefore the spatial-average differences vanish identically for all values of σ.

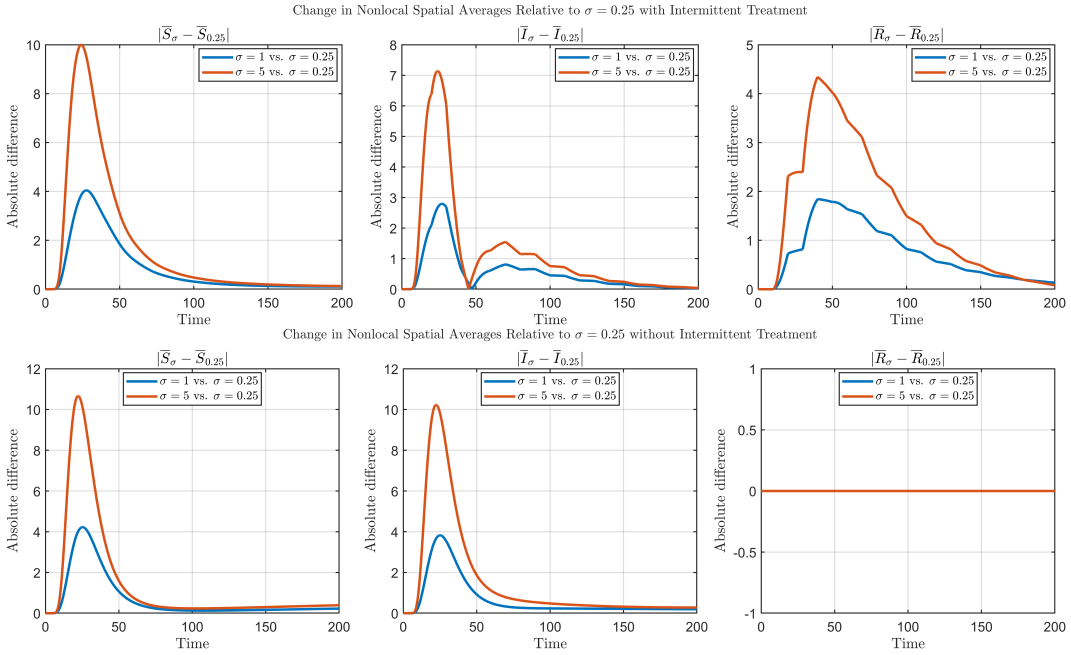


Figure 4: Absolute differences in the spatial averages of S, I, and R for the NRDS with kernel radii $\sigma = 1$ and $\sigma = 5$, measured relative to the reference case $\sigma = 0.25$. The top row corresponds to simulations with intermittent treatment, while the bottom row shows the corresponding results without treatment.

4 Conclusion

In this paper, we studied a spatially heterogeneous nonlocal SIR epidemic model incorporating unequal diffusion coefficients, demographic effects, and an intermittent treatment strategy. The spatial movement of the susceptible, infected, and recovered populations was modeled by convolution-type nonlocal diffusion operators, allowing long-range interactions while preserving the heterogeneous structure of the epidemic process. The presence of distinct diffusion coefficients for the three epidemiological compartments introduces additional analytical challenges that are not encountered in models with identical diffusion rates.

Using semigroup theory together with suitable L^p-energy estimates, we established the global existence, uniqueness, positivity, uniform boundedness, and asymptotic behavior of classical solutions. To overcome the lack of parabolic smoothing for the nonlocal convolution operator, we employed a diffusive limit approach and proved that the nonlocal system converges to its corresponding local reaction–diffusion

counterpart. This convergence allows the asymptotic behavior of the nonlocal model to be deduced from the smoothing properties of the limiting local system. Numerical simulations are presented to illustrate the effects of spatially heterogeneous coefficients, nonlocal dispersal, increasing interaction radii of the Gaussian kernel, and intermittent treatment on the spatiotemporal dynamics of the epidemic model.

Several interesting problems remain open for future investigation. These include establishing direct asymptotic estimates for the fully nonlocal system without appealing to diffusive limit arguments, studying threshold dynamics and persistence, and extending the present framework to more general classes of nonlocal diffusion operators, including fractional and nonlinear nonlocal models.

The analytical framework developed here is sufficiently general to be applicable to a broad class of heterogeneous nonlocal reaction–diffusion systems arising in mathematical biology and related applications.

Conflict of Interest

The author declares that there is no conflict of interest.

References

[1] Abdelmalek, S., and Kouachi, S. Proof of existence of global solutions for m-component reaction–diffusion systems with mixed boundary conditions via the lyapunov functional method. *Journal of Physics A: Mathematical and Theoretical 40*, 41 (2007), 12335–12350.

[2] Allen, L. J. S., Bolker, B. M., Lou, Y., and Nevai, A. L. Asymptotic profiles of the steady states for an sis epidemic reaction-diffusion model. *Discrete and Continuous Dynamical Systems 21*, 1 (2008), 1–20.

[3] Anderson, R. M., and May, R. M. *Infectious diseases of humans: dynamics and control*. Oxford university press, 1991.

[4] Andreu-Vaillo, F. *Nonlocal diffusion problems*. No. 165. American Mathematical Soc., 2010.

[5] Arendt, W. Vector-valued laplace transforms and cauchy problems. *Israel Journal of Mathematics 59*, 3 (1987), 327–352.

[6] Bates, P. W., Fife, P. C., Ren, X., and Wang, X. Traveling waves in a convolution model for phase transitions. *Archive for Rational Mechanics and Analysis 138*, 2 (1997), 105–136.

[7] Brauer, F., and Castillo-Chavez, C. *Mathematical models in population biology and epidemiology*, vol. 2. Springer, 2012.

[8] Bucur, C., Valdinoci, E., et al. *Nonlocal diffusion and applications*, vol. 20. Springer, 2016.

[9] Cantrell, R. S., and Cosner, C. *Spatial ecology via reaction-diffusion equations*. John Wiley & Sons, 2004.

[10] Capasso, V., and Maddalena, L. Convergence to equilibrium states for a reaction-diffusion system modelling the spatial spread of a class of bacterial and viral diseases. *Journal of Mathematical Biology 13*, 2 (1981), 173–184.

[11] Coville, J. On a simple criterion for the existence of a principal eigenfunction of some nonlocal operators. *Journal of Differential Equations 249*, 11 (2010), 2921–2953.

[12] Coville, J., Dávila, J., and Martínez, S. Pulsating fronts for nonlocal dispersion and kpp nonlinearity. In *Annales de l'Institut Henri Poincaré C, Analyse non linéaire* (2013), vol. 30, Elsevier, pp. 179–223.

[13] Diekmann, O., Heesterbeek, H., and Britton, T. *Mathematical tools for understanding infectious disease dynamics*, vol. 7. Princeton University Press, 2013.

[14] Diekmann, O., and Heesterbeek, J. A. P. *Mathematical epidemiology of infectious diseases: model building, analysis and interpretation*, vol. 5. John Wiley & Sons, 2000.

[15] Du, Q., GunzburGer, M., Lehoucq, R. B., and Zhou, K. A nonlocal vector calculus, nonlocal volume-constrained problems, and nonlocal balance laws. *Mathematical Models and Methods in Applied Sciences 23*, 03 (2013), 493–540.

[16] Fife, P. Some nonclassical trends in parabolic and parabolic-like evolutions. *Trends in nonlinear analysis* (2003), 153–191.

[17] Guèrin, C., Girard, R., Selli, J., and Ayzac, L. Intermittent versus continuous renal replacement therapy for acute renal failure in intensive care units: results from a multicenter prospective epidemiological survey. *Intensive Care Medicine 28*, 10 (2002), 1411–1418.

[18] Hethcote, H. W. The mathematics of infectious diseases. *SIAM review 42*, 4 (2000), 599–653.

[19] Ignat, L. I., and Rossi, J. D. A nonlocal convection–diffusion equation. *Journal of Functional Analysis 251*, 2 (2007), 399–437.

[20] Kao, C.-Y., Lou, Y., and Shen, W. Random dispersal vs. non-local dispersal. *Discrete Contin. Dyn. Syst 26*, 2 (2010), 551–596.

[21] Keeling, M. J., and Rohani, P. *Modeling infectious diseases in humans and animals*. Princeton university press, 2008.

[22] Kermack, W. O., and McKendrick, A. G. A contribution to the mathematical theory of epidemics. *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character 115*, 772 (1927), 700–721.

[23] Kirschner, D., and Webb, G. F. A model for treatment strategy in the chemotherapy of aids. *Bulletin of mathematical biology 58*, 2 (1996), 367–390.

[24] Kouachi, S. Existence of global solutions to reaction-diffusion systems via a lyapunov functional. *Electronic Journal of Differential Equations (EJDE)[electronic only] 2001* (2001), 1–10.

[25] Kuniya, T., and Wang, J. Global dynamics of an sir epidemic model with nonlocal diffusion. *Nonlinear Analysis: Real World Applications 43* (2018), 262–282.

[26] Ladyzhenskaia, O. A., Solonnikov, V. A., and Ural'tseva, N. N. *Linear and quasi-linear equations of parabolic type*, vol. 23. American Mathematical Soc., 1968.

[27] Laurençot, P., and Walker, C. A nonlocal gray-scott model: Well-posedness and diffusive limit. *Discrete and Continuous Dynamical Systems-S 16*, 12 (2023), 3709–3732.

[28] Lutscher, F., Lewis, M. A., and McCauley, E. Effects of heterogeneity on spread and persistence in rivers. *Bulletin of mathematical biology 68*, 8 (2006), 2129–2160.

[29] Morgan, J. Global existence for semilinear parabolic systems. *SIAM journal on mathematical analysis 20*, 5 (1989), 1128–1144.

[30] Pazy, A. *Semigroups of Linear Operators and Applications to Partial Differential Equations*, vol. 44 of *Applied Mathematical Sciences*. Springer, 2012.

[31] Peng, R., and Zhao, X.-Q. A reaction–diffusion sis epidemic model in a time-periodic environment. *Nonlinearity 25*, 5 (2012), 1451–1471.

[32] Ross, A., Penny, M., Maire, N., Studer, A., Carneiro, I., Schellenberg, D., Greenwood, B., Tanner, M., and Smith, T. Modelling the epidemiological impact of intermittent preventive treatment against malaria in infants. *PLoS One 3*, 7 (2008), e2661.

[33] Ruan, S. Spatial-temporal dynamics in nonlocal epidemiological models. In *Mathematics for life science and medicine*. Springer, 2007, pp. 97–122.

[34] Saad, Y. *Iterative methods for sparse linear systems*. SIAM, 2003.

[35] Van der Vorst, H. A. Bi-cgstab: A fast and smoothly converging variant of bi-cg for the solution of nonsymmetric linear systems. *SIAM Journal on scientific and Statistical Computing 13*, 2 (1992), 631–644.

[36] Wang, W., and Zhao, X.-Q. Basic reproduction numbers for reaction-diffusion epidemic models. *SIAM Journal on Applied Dynamical Systems 11*, 4 (2012), 1652–1673.

[37] Yang, C., and Wang, J. Basic reproduction numbers for a class of reaction-diffusion epidemic models. *Bulletin of mathematical biology 82* (2020), 1–25.

[38] Yang, F.-Y., Li, Y., Li, W.-T., and Wang, Z.-C. Traveling waves in a nonlocal dispersal kermack-mckendrick epidemic model. *Discrete & Continuous Dynamical Systems-Series B 18*, 7 (2013).

[39] Yang, S., Chu, W., and Kevrekidis, P. An epidemical model with nonlocal spatial infections. *Proceedings of the European Academy of Sciences and Arts 3*, 1 (2025).

A Diffusive Limit

The purpose of this appendix is to justify the diffusive-limit approximation used in the proof of Theorem A.1. Since the nonlocal diffusion operator does not possess the parabolic smoothing property enjoyed by the classical Laplacian, the asymptotic $L^\infty(\Omega)$-behavior cannot be obtained directly from the nonlocal system. Instead, we establish the convergence of the nonlocal model to the corresponding local reaction–diffusion SIR system under a suitable rescaling of the convolution kernel. The desired uniform asymptotic behavior of the limiting local system then follows from the classical L^p-L^∞ smoothing theory for parabolic equations.

Although the analysis in the main text is carried out for the reduced subsystem (S, I), since the recovered population R is uniquely determined by a linear nonhomogeneous equation once I is known, it is technically more convenient to formulate the diffusive-limit argument for the full vector-valued system (S, I, R). This allows the compactness framework to be applied directly to the reaction vector field.

Let us assume that

(D2) (Diffusivity) $d_i > 0$ for all $i = 1, 2, 3$ and $\psi \in C^\infty(\mathbb{R}_+; \mathbb{R}_+)$ is nonincreasing, has compact support, and satisfies $\int_{\mathbb{R}^n} \psi(|z|)\, dz = 1$, $\int_{\mathbb{R}^n} |z|^2 \psi(|z|)\, dz = \mathcal{M} < \infty$.

(Lp) (L^p-continuity) There exists $\tilde{p} \in \mathbb{N}$ such that, for every $p \geq \tilde{p}$, the reaction vector field

$$F : L^p(\Omega; \mathbb{R}^3) \longrightarrow L^2(\Omega; \mathbb{R}^3)$$

is continuous on bounded subsets.

To recover the classical diffusion operator, we introduce the family of rescaled kernels

$$\phi_j(x, y) = j^{n+2} \psi(j|x - y|), \quad j \in \mathbb{N}. \tag{A.1}$$

For each $j \in \mathbb{N}$, let $u_j = (u_{1,j}, u_{2,j}, u_{3,j}) \equiv (S_j, I_j, R_j)$ denote the solution of the nonlocal SIR system associated with the above rescaled kernel which satisfies assumption (D2). For each $j \in \mathbb{N}$ and $i = 1, 2, 3$, we consider the following family of nonlocal SIR systems:

$$\begin{cases} \partial_t u_{i,j}(x,t) = d_i \displaystyle\int_\Omega j^{n+2} \psi(j|x-y|) \Big(u_{i,j}(y,t) - u_{i,j}(x,t) \Big)\, dy + F_i(x,t,u_j), x \in \Omega,\ t \geq 0, \\ u_{i,j}(x,0) = u_{i,0}(x), \qquad\qquad x \in \Omega. \end{cases} \tag{A.2}$$

The corresponding local reaction–diffusion SIR system is

$$\begin{cases} \partial_t u_i = \frac{\mathcal{M} d_i}{2n} \Delta u_i + F_i(x,t,u), \text{ on } \Omega \times (0,\infty),\ i = 1,2,3, \\ \nabla u_i \cdot \eta = 0, \qquad\qquad \text{on } \partial\Omega \times (0,\infty),\ i = 1,2,3, \\ u_i = u_{i,0}, \qquad\qquad\qquad \text{on } \Omega,\ t = 0,\ i = 1,2,3. \end{cases} \tag{A.3}$$

where $u = (u_1, u_2, u_3) \equiv (S, I, R)$. The main objective of this appendix is to show that, under the assumptions above, there exists a subsequence of $\{u_j\}_{j\in\mathbb{N}}$ converging to the solution of (A.3) in $L^p(\Omega\times(0,T);\mathbb{R}^3)$ for every $T > 0$ and $1 \le p < \infty$. The global existence theory of Morgan [29] guarantees the existence of a unique component-wise nonnegative global classical solution to (A.3).

The following theorem establishes the convergence of the family of nonlocal solutions $\{u_j\}_{j\in\mathbb{N}}$ to the corresponding solution of the local reaction–diffusion SIR system as the interaction kernel converges to the Laplacian in the diffusive limit.

Theorem A.1. *Suppose $n \ge 2$, and assume that (D2), (Qp), and (Lp) hold. Then, for each $j \in \mathbb{N}$, the nonlocal system* (A.2) *admits a unique component-wise nonnegative global classical solution u_j, and the local system* (A.3) *admits a unique component-wise nonnegative global classical solution u. Moreover, for every $T > 0$, there exists a subsequence of $\{u_j\}_{j\in\mathbb{N}}$, still denoted by $\{u_j\}_{j\in\mathbb{N}}$, such that*

$$u_j \longrightarrow u \qquad \text{in } L^p(\Omega\times(0,T);\mathbb{R}^3),$$

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

Remark A.1. As in the general diffusive-limit framework, the restriction $n \ge 2$ is imposed because the compactness argument used below relies on estimates that are presently available in this setting. The one-dimensional case is expected to be true for the present SIR system as well, but it would require a separate compactness argument and is not pursued here.

Proof of Theorem A.1

Definition A.1. For each $j \in \mathbb{N}$, define the nonlocal energy functional

$$\mathcal{Y}_j[z] := \int_\Omega\int_\Omega \phi_j(x,y)\Big(z(x)-z(y)\Big)^2\,dydx, \qquad z \in L^2(\Omega), \tag{A.4}$$

where ϕ_j is given by (A.1).

Lemma A.1. *Let $T > 0$, $j \in \mathbb{N}$, and $1 \le p < \infty$. Let $u_j = (u_{1,j}, u_{2,j}, u_{3,j})$ be the unique global classical solution of* (A.2)*. Then there exist constants $C_{p,T} > 0$ and $C_T > 0$, independent of j, such that*

$$\sum_{i=1}^3 \|u_{i,j}(\cdot,t)\|^p_{L^p(\Omega)} \le C_{p,T}, \qquad 0 \le t \le T, \tag{A.5a}$$

$$\int_0^t \sum_{i=1}^3 \mathcal{Y}_j\Big[u_{i,j}(\cdot,s)\Big]\,ds \le C_T, \qquad 0 \le t \le T. \tag{A.5b}$$

Proof: The estimate (A.5a) follows by the same argument as in Lemma 2.2. Indeed, replacing the kernel ϕ by the rescaled kernel ϕ_j does not affect the $L^p(\Omega)$-energy estimate, and therefore the proof carries over verbatim to (A.2).

Now, multiplying the i-th equation of (A.2) by $u_{i,j}(x,t)$, integrating over Ω, and then summing over $i = 1,2,3$, we obtain

$$\begin{aligned}\frac{1}{2}\frac{d}{dt}\sum_{i=1}^3\int_\Omega u_{i,j}(x,t)^2\,dx &= \sum_{i=1}^3 d_i\int_\Omega\int_\Omega u_{i,j}(x,t)\phi_j(x,y)\Big(u_{i,j}(y,t)-u_{i,j}(x,t)\Big)\,dydx + \sum_{i=1}^3\int_\Omega u_{i,j}(x,t)F_i(x,t,u_j(x,t))\,dx\\ &=: A + B.\end{aligned} \tag{A.6}$$

Using the symmetry of ϕ_j, we have

$$\begin{aligned}A &= \sum_{i=1}^3 d_i\int_\Omega\int_\Omega u_{i,j}(x,t)\phi_j(x,y)\Big(u_{i,j}(y,t)-u_{i,j}(x,t)\Big)\,dydx = -\frac{1}{2}\sum_{i=1}^3 d_i\int_\Omega\int_\Omega \phi_j(x,y)\Big(u_{i,j}(y,t)-u_{i,j}(x,t)\Big)^2\,dydx\\ &\le -\frac{d_{\min}}{2}\sum_{i=1}^3 \mathcal{Y}_j[u_{i,j}(\cdot,t)]\end{aligned}$$

where $d_{\min} = \min_{i=1,2,3}\{d_i\}$. Also,

$$\begin{aligned}
B &:= \sum_{i=1}^{3} \int_\Omega u_{i,j}(x,t) F_i(x,t,u_j(x,t))\,dx \\
&= \int_\Omega b(x) u_{1,j}(x,t)dx - \int_\Omega \mu(x) u_{1,j}(x,t)^2 dx - \int_\Omega \beta(x) u_{1,j}(x,t)^2 u_{2,j}(x,t)dx + \int_\Omega \beta(x) u_{1,j}(x,t) u_{2,j}(x,t)^2 dx \\
&\quad - \int_\Omega (\mu(x) + \gamma(x,t)) u_{2,j}(x,t)^2 dx + \int_\Omega \gamma(x,t) u_{2,j}(x,t) u_{3,j}(x,t)dx - \int_\Omega \mu(x) u_{3,j}(x,t)^2 dx \\
&\le b^* \int_\Omega u_{1,j}(x,t)dx + \beta^* C_3 \int_\Omega u_{2,j}(x,t)^2 dx + \gamma^* \left(\int_\Omega u_{2,j}(x,t)^2 dx \right)^{\frac{1}{2}} \left(\int_\Omega u_{3,j}(x,t)^2 dx \right)^{\frac{1}{2}} \\
&\le \tilde{C}
\end{aligned}$$

where we used the uniform L^∞-bound from Theorem 1.1, the L^1-bound from Lemma 2.1, the L^2-bound from Lemma 2.2, and the nonnegativity of $u_{i,j}$, β, μ, and γ. Hence $B \le \tilde{C}$, where $\tilde{C} > 0$ is independent of j. Substituting the estimates for A and B into (A.6), we obtain

$$\frac{1}{2}\frac{d}{dt} \sum_{i=1}^{3} \int_\Omega u_{i,j}(x,t)^2\,dx + \frac{d_{\min}}{2} \sum_{i=1}^{3} \mathcal{Y}_j[u_{i,j}(\cdot,t)] \le \tilde{C}.$$

Integrating from 0 to $t \le T$, we get

$$\frac{1}{2} \sum_{i=1}^{3} \|u_{i,j}(\cdot,t)\|^2_{L^2(\Omega)} + \frac{d_{\min}}{2} \int_0^t \sum_{i=1}^{3} \mathcal{Y}_j[u_{i,j}(\cdot,s)]\,ds \le \frac{1}{2} \sum_{i=1}^{3} \|u_{i,j}(\cdot,0)\|^2_{L^2(\Omega)} + \tilde{C}T.$$

Therefore, using the L^2-boundedness of the initial data, there exists $C_T > 0$, independent of j, such that

$$\int_0^t \sum_{i=1}^{3} \mathcal{Y}_j[u_{i,j}(\cdot,s)]\,ds \le C_T, \qquad 0 \le t \le T.$$

This proves (A.5b). □

The uniform L^p- and nonlocal energy estimates established in Lemma A.1 allow us to estimate the time derivatives of the approximate solutions, a key ingredient for the compactness argument. In particular, we prove that $\partial_t u_{i,j}$ is uniformly bounded in $L^2(0,T;(W^{1,n+1}(\Omega))')$, where $(W^{1,n+1}(\Omega))'$ denotes the dual space of $W^{1,n+1}(\Omega)$, equipped with its usual dual norm.

Lemma A.2. *There exists $C_T > 0$, independent of j, such that*

$$\int_0^t \sum_{i=1}^{3} \left\| \frac{\partial u_{i,j}}{\partial t}(\cdot,s) \right\|^2_{(W^{1,n+1}(\Omega))'} ds \le C_T, \tag{A.7}$$

for all $0 \le t \le T$ and $j \in \mathbb{N}$.

Proof: Fix $i \in \{1,2,3\}$. For any $\zeta \in W^{1,n+1}(\Omega)$, $t > 0$, and $j \in \mathbb{N}$, we have

$$\begin{aligned}
\left| \int_\Omega \zeta(x) \frac{\partial u_{i,j}}{\partial t}(x,t)\,dx \right| &\le \left| d_i \int_\Omega \zeta(x) \int_\Omega \phi_j(x,y) \Big(u_{i,j}(y,t) - u_{i,j}(x,t) \Big) dy dx \right| + \left| \int_\Omega \zeta(x) F_i(x,t,u_j(x,t))\,dx \right| \\
&=: J_1 + J_2.
\end{aligned} \tag{A.8}$$

where

$$\begin{aligned}
J_1 &= \left| d_i \int_\Omega \int_\Omega \zeta(x) \phi_j(x,y) \Big(u_{i,j}(y,t) - u_{i,j}(x,t) \Big) dy dx \right| \\
&= \frac{d_i}{2} \left| \int_\Omega \int_\Omega \phi_j(x,y) \Big(\zeta(x) - \zeta(y) \Big) \Big(u_{i,j}(y,t) - u_{i,j}(x,t) \Big) dy dx \right|
\end{aligned}$$

$$\leq \frac{d_i}{2}\left(\int_\Omega\int_\Omega \phi_j(x,y)\Big(\zeta(x)-\zeta(y)\Big)^2\,dydx\right)^{1/2}\left(\int_\Omega\int_\Omega \phi_j(x,y)\Big(u_{i,j}(y,t)-u_{i,j}(x,t)\Big)^2\,dydx\right)^{1/2}$$
$$= \frac{d_i}{2}\left(\int_\Omega\int_\Omega \phi_j(x,y)\Big(\zeta(x)-\zeta(y)\Big)^2\,dydx\right)^{1/2}\boldsymbol{\mathcal{Y}}_j[u_{i,j}(\cdot,t)]^{1/2}.$$

Since ψ has compact support by assumption (D2), there exists $R>0$ such that

$$\operatorname{supp}(\phi_j)\subset\left\{(x,y)\in\mathbb{R}^{2n}:|x-y|\leq\frac{R}{j}\right\}.$$

Therefore, following the proof of Lemma 4.2 in [27], there exists a constant $C_5>0$, independent of j, such that

$$\left(\int_\Omega\int_\Omega \phi_j(x,y)\Big(\zeta(x)-\zeta(y)\Big)^2\,dydx\right)^{1/2}\leq C_5\|\zeta\|_{W^{1,n+1}(\Omega)}.$$

Consequently,

$$J_1\leq C_5 d_i\|\zeta\|_{W^{1,n+1}(\Omega)}\boldsymbol{\mathcal{Y}}_j[u_{i,j}(\cdot,t)]^{1/2},\qquad 0\leq t\leq T.$$

Also,

$$\begin{aligned}J_2&=\left|\int_\Omega\zeta(x)F_i(x,t,u_j(x,t))\,dx\right|\leq\|\zeta\|_{L^\infty(\Omega)}\int_\Omega|F_i(x,t,u_j(x,t))|\,dx\\&\leq C_6\|\zeta\|_{L^\infty(\Omega)}\\&\leq C_6\|\zeta\|_{W^{1,n+1}(\Omega)},\ \text{ for all } 0\leq t\leq T\end{aligned}$$

Here the uniform L^1-boundedness of $F_i(\cdot,t,u_j(\cdot,t))$, which follows from the L^p-bounds in Lemma A.1 and the structure of the reaction terms, together with the embedding $W^{1,n+1}(\Omega)\hookrightarrow L^\infty(\Omega)$.

Substituting the estimates for J_1 and J_2 into (A.8), we obtain

$$\left|\int_\Omega\zeta(x)\frac{\partial u_{i,j}}{\partial t}(x,t)\,dx\right|\leq C_5 d_i\|\zeta\|_{W^{1,n+1}(\Omega)}\boldsymbol{\mathcal{Y}}_j[u_{i,j}(\cdot,t)]^{1/2}+C_6\|\zeta\|_{W^{1,n+1}(\Omega)}\tag{A.9}$$

Taking the supremum over all $\zeta\in W^{1,n+1}(\Omega)$ satisfying $\|\zeta\|_{W^{1,n+1}(\Omega)}\leq 1$, we obtain

$$\left\|\frac{\partial u_{i,j}}{\partial t}(\cdot,t)\right\|_{(W^{1,n+1}(\Omega))'}\leq C_5 d_i\,\boldsymbol{\mathcal{Y}}_j[u_{i,j}(\cdot,t)]^{1/2}+C_6.$$

Squaring both sides, integrating over $(0,t)$, and using Lemma A.1, we obtain

$$\begin{aligned}\int_0^t\left\|\frac{\partial u_{i,j}}{\partial t}(\cdot,s)\right\|^2_{(W^{1,n+1}(\Omega))'}ds&\leq 2C_5^2d_i^2\int_0^t\boldsymbol{\mathcal{Y}}_j[u_{i,j}(\cdot,s)]\,ds+2C_6^2T\\&\leq C_T,\end{aligned}$$

where $C_T>0$ is independent of j. Summing over $i=1,2,3$ completes the proof. □

The following compactness result is taken from [27].

Lemma A.3. *Let $T>0$. Then the sequence $\{u_j\}_{j\in\mathbb{N}}$ is relatively compact in $L^2(\Omega\times(0,T);\mathbb{R}^3)$. Moreover, every cluster point of $\{u_j\}_{j\in\mathbb{N}}$ belongs to $L^2(0,T;H^1(\Omega;\mathbb{R}^3))$.*

Proof of Theorem A.1. By Lemma A.3, there exists a subsequence $\{u_{j_k}\}_{k\in\mathbb{N}}$ and a component-wise nonnegative function u such that

$$u \in C\Big([0,T];\big(W^{1,n+1}(\Omega)\big)'\Big) \cap L^\infty\Big(0,T;L^2(\Omega)\Big) \cap L^2\Big(0,T;H^1(\Omega)\Big),$$

and

$$\lim_{k\to\infty} \sup_{0\le t\le T} \|(u_{i,j_k} - u_i)(\cdot,t)\|_{(W^{1,n+1}(\Omega))'} = 0, \qquad i = 1,2,3, \tag{A.10}$$

$$\lim_{k\to\infty} \int_0^T \|(u_{i,j_k} - u_i)(\cdot,t)\|^2_{L^2(\Omega)}\, dt = 0, \qquad i = 1,2,3, \tag{A.11}$$

and

$$u_{i,j_k}(x,t) \to u_i(x,t) \quad \text{for a.e. } (x,t) \in \Omega\times(0,T), \qquad i = 1,2,3. \tag{A.12}$$

Since $u_{j_k} \to u$ in $L^2(\Omega\times(0,T);\mathbb{R}^3)$ and $\{u_{j_k}\}$ is uniformly bounded in $L^\infty(0,T;L^p(\Omega;\mathbb{R}^3))$, for every $1 \le p < \infty$, interpolation yields

$$u_{j_k} \longrightarrow u \qquad \text{in} \qquad L^p(\Omega\times(0,T);\mathbb{R}^3), \qquad 1 \le p < \infty.$$

Therefore, by assumption (Lp),

$$F_i(\cdot,\cdot,u_{j_k}) \longrightarrow F_i(\cdot,\cdot,u) \qquad \text{in } L^2(\Omega\times(0,T)), \qquad i = 1,2,3.$$

The desired convergence follows by applying Proposition B.1 in [27] together with a standard diagonalization argument. □