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Analysis of a Spatial SIRS Epidemic Model with General Incidence

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Abstract: This article presents a comprehensive study of a reaction–diffusion SIRS epidemic model with general incidence. We provide a detailed treatment that includes: (i) the well-posedness of the system and the existence of classical solutions, (ii) the threshold dynamics characterized by the basic reproduction number R_0, (iii) the existence of endemic equilibria when $R_0 > 1$, and (iv) the analysis of both local and global stability using Lyapunov functionals. The theoretical findings are complemented by numerical simulations illustrating convergence to equilibria and the influence of spatial heterogeneity. This work offers a coherent picture of the epidemic dynamics in spatially structured populations.

Keywords: Reaction–diffusion systems; SIRS model; General incidence; Lyapunov stability.
AMS Math Codes: 35K57; 92D30; 92D25; 34D20.

1 Introduction

Mathematical epidemiology seeks to understand the spread and control of infectious diseases. Reaction–diffusion models are particularly well suited to account for spatial heterogeneity while tracking the temporal evolution of disease dynamics [1,2]. Among them, SIRS-type models are appropriate for infections where immunity wanes and reinfection is possible.

In this article, we develop a comprehensive study of a reaction–diffusion SIRS system with general incidence. Our purpose is to investigate in detail the mathematical well-posedness of the model, the threshold dynamics governed by the basic reproduction number R_0, and the existence and stability of endemic equilibria. The analysis relies on Lyapunov functionals and comparison arguments, while numerical simulations illustrate the theoretical findings. This program is motivated by, and framed in connection with, the classical works of Diekmann et al. [3], van den Driessche and Watmough [4], and subsequent studies on Lyapunov methods [5–7].

Our work is closely related to recent research on diffusive epidemic models in heterogeneous environments. In particular, Avila-Vales, Garcia-Almeida, and Perez [8] analyzed a diffusive SIR model with saturated incidence and permanent immunity, establishing well-posedness, threshold dynamics, and stability results. The present study extends this line of research by incorporating an SIRS structure with waning immunity and a general incidence function [9,10]. This generalization introduces new mathematical challenges, especially in proving global stability, and broadens the epidemiological relevance of the framework [11,12].

Beyond the mathematical interest, this study is relevant for epidemiology. Reaction–diffusion models capture how spatial heterogeneity, movement, and local variations in parameters affect disease persistence or extinction. The SIRS

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structure, accounting for waning immunity, is particularly suited to recurrent infections such as influenza, pertussis, or coronaviruses. A rigorous analysis of threshold dynamics and endemic equilibria therefore provides valuable insight into the conditions under which a disease may disappear or become established in a heterogeneous environment.

The remainder of this paper is organized as follows. In Section 2, we formulate the spatially heterogeneous SIRS model with a general incidence function under Neumann boundary conditions. Section 3 is devoted to the well-posedness of the system, where we establish existence, uniqueness, and positivity of classical solutions using semi-group theory and maximum principles. In Section 4, we analyze the steady states of the system, derive the basic reproduction number R_0, and investigate its role as a threshold parameter. Section 5 examines the endemic equilibrium, discussing its existence, qualitative properties, and both local and global stability through a Lyapunov functional approach. Section 6 presents numerical simulations that illustrate the theoretical results and confirm the predicted threshold dynamics. Finally, Section 7 summarizes the main conclusions and outlines possible extensions of the model, such as stochastic perturbations, time delays, or vaccination strategies.

2 The Model and Functional Setting

We consider the reaction–diffusion SIRS system defined on a smooth bounded domain $\Omega \subset \mathbb{R}^N$, $N \leq 3$, with homogeneous Neumann boundary conditions:

$$\begin{cases} \partial_t S - d_S \Delta S = \Lambda(x) - \beta(x,S,I) - \mu(x)S + \omega(x)R, \\ \partial_t I - d_I \Delta I = \beta(x,S,I) - \big(\mu(x) + \gamma(x)\big)I, \\ \partial_t R - d_R \Delta R = \gamma(x)I - \big(\mu(x) + \omega(x)\big)R, \\ \partial_\nu S = \partial_\nu I = \partial_\nu R = 0 \quad \text{on } \partial\Omega, \\ S(x,0) = S_0(x), \quad I(x,0) = I_0(x), \quad R(x,0) = R_0(x). \end{cases} \tag{2.1}$$

Here S, I, and R denote the densities of susceptible, infected, and recovered individuals. The parameters d_S, d_I, d_R are diffusion rates, $\Lambda(x)$ the recruitment term, $\mu(x)$ the natural death rate, $\gamma(x)$ the recovery rate, $\omega(x)$ the loss of immunity, and $\beta(x,S,I)$ a general incidence function.

We assume:

1. The coefficients are smooth and strictly positive;
2. β is smooth, increasing in I, Lipschitz continuous, and vanishes when $S = 0$ or $I = 0$;
3. Initial data are bounded and nonnegative.

3 Well-Posedness of the Model

We study the well-posedness of system (1). To state precise results, we introduce the Banach spaces

$$X := L^2(\Omega)^3, \qquad D := \{u = (u_1,u_2,u_3) \in H^2(\Omega)^3 : \partial_\nu u_i = 0 \text{ on } \partial\Omega\}.$$

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Let the linear operator $A : D \subset X \to X$ be defined by

$$Au = \begin{pmatrix} d_S \Delta u_1 \\ d_I \Delta u_2 \\ d_R \Delta u_3 \end{pmatrix}, \qquad u = (u_1, u_2, u_3) \in D.$$

The nonlinear reaction map $F : X \to X$ is given by

$$F(S, I, R) = \begin{pmatrix} \Lambda(x) - \beta(x, S, I) - \mu(x)S + \omega(x)R \\ \beta(x, S, I) - \big(\mu(x) + \gamma(x)\big)I \\ \gamma(x)I - \big(\mu(x) + \omega(x)\big)R \end{pmatrix}.$$

3.1 Existence and Uniqueness via the Lumer–Phillips Theorem

Theorem 3.1 (Generation and Local Well-Posedness)**.** *Under the standing hypotheses on the domain Ω and the coefficients, the operator $A : D \subset X \to X$ defined above is densely defined, closed, and dissipative, and its range is dense in X. Consequently, by the Lumer–Phillips theorem, A generates a strongly continuous contraction semigroup $(e^{tA})_{t\geq 0}$ on X. Moreover, the nonlinear map F is locally Lipschitz on bounded subsets of X, and for any $U_0 \in X$ there exists $T_{\max} > 0$ and a unique mild solution*

$$U \in C([0, T_{\max}); X), \qquad U(t) = e^{tA}U_0 + \int_0^t e^{(t-s)A}F(U(s))\, ds.$$

Proof. We split the proof into several steps.

Step 1: Density and closedness. The domain

$$D = H^2(\Omega)^3 \cap \{\partial_\nu u_i = 0\}$$

is dense in

$$X = L^2(\Omega)^3,$$

because $C^\infty(\Omega)$ is dense in $L^2(\Omega)$, and smooth functions satisfying Neumann boundary conditions are dense in $H^2(\Omega)$. Taking triples yields density in X. The operator A is a block-diagonal matrix of elliptic operators with domain D; each block $d_j\Delta$ with Neumann boundary conditions is a closed operator on $L^2(\Omega)$. Hence A is closed.

Step 2: Dissipativity. We show that A is dissipative, i.e. for every $u \in D$ one has $\langle Au, u\rangle_X \leq 0$. Writing $u = (u_1, u_2, u_3)$ and using integration by parts together with Neumann boundary conditions, we obtain

$$\langle Au, u\rangle_X = \sum_{j\in\{S,I,R\}} d_j \int_\Omega (\Delta u_j)u_j\, dx = -\sum_j d_j \int_\Omega |\nabla u_j|^2\, dx \leq 0.$$

Hence A is dissipative.

Step 3: Surjectivity of $\lambda I - A$ for large $\lambda > 0$. To apply the Lumer–Phillips theorem, we need the range condition: for some (equivalently all sufficiently large) $\lambda > 0$, the operator $\lambda I - A : D \to X$ has range equal to X. Fix $\lambda > 0$ and

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let $f = (f_1, f_2, f_3) \in X$. Consider the elliptic Neumann problems for each component:

$$\lambda u_j - d_j \Delta u_j = f_j \quad \text{in } \Omega, \qquad \partial_\nu u_j = 0 \text{ on } \partial\Omega.$$

Classical elliptic theory (Lax–Milgram or Fredholm alternative) guarantees a unique solution $u_j \in H^2(\Omega)$ for each j, since $\lambda > 0$ shifts the operator away from the kernel. Therefore, $\lambda I - A$ is surjective onto X, and in particular the range is dense.

Step 4: Conclusion by Lumer–Phillips. We have shown that A is densely defined, closed, dissipative, and that $\lambda I - A$ is surjective for some $\lambda > 0$. By the Lumer–Phillips theorem (see, e.g., [13]), A generates a strongly continuous contraction semigroup $(e^{tA})_{t\geq 0}$ on X.

Step 5: Local Lipschitz continuity of F. Recall the assumptions on β: it is locally Lipschitz in (S, I) uniformly in $x \in \Omega$, and the coefficients are bounded. Let $B_R \subset X$ denote a ball of radius R centered at the origin intersected with L^∞-bounded triples. By Sobolev embedding, and since we only work on bounded sets, functions in B_R are pointwise defined a.e., and one can use the Lipschitz constant of β on the range determined by R. A straightforward computation gives, for $U = (S, I, R)$ and $\tilde{U} = (\tilde{S}, \tilde{I}, \tilde{R})$ in B_R:

$$\|F(U) - F(\tilde{U})\|_X^2 \leq C_R \Big(\|S - \tilde{S}\|_{L^2}^2 + \|I - \tilde{I}\|_{L^2}^2 + \|R - \tilde{R}\|_{L^2}^2 \Big) = C_R \|U - \tilde{U}\|_X^2,$$

for some constant C_R depending on R and the coefficients. Thus F is locally Lipschitz on bounded subsets of X.

Step 6: Existence and uniqueness of mild solutions. With the generation and local Lipschitz properties in hand, we apply the standard Picard fixed-point theorem in the Banach space $C([0, T]; X)$ to the variation of constants formula

$$\Phi(U)(t) := e^{tA} U_0 + \int_0^t e^{(t-s)A} F(U(s))\, ds.$$

For $T > 0$ small enough, Φ is a contraction on a closed ball of radius ρ in $C([0, T]; X)$ centered at the function $t \mapsto e^{tA} U_0$, hence there exists a unique fixed point, which is the unique mild solution on $[0, T]$. Standard continuation yields a maximal existence time $T_{\max} > 0$ and the blow-up alternative: if $T_{\max} < \infty$, then $\|U(t)\|_X \to \infty$ as $t \uparrow T_{\max}$. □

3.2 Regularity of Solutions

Remark 3.2. *Alternative approaches to local and global well-posedness can be obtained via parabolic L^p–L^q estimates and the Banach fixed-point theorem, see for instance Amann [14]. While this approach is often developed for problems posed on the whole space using Fourier transform techniques, it can be adapted to bounded domains with appropriate elliptic regularity. Here, we have preferred the semigroup method for clarity and conciseness.*

We now improve the regularity of mild solutions to classical (strong) solutions under additional regularity assumptions on the initial data.

Theorem 3.3 (Regularity)**.** *Let $U_0 \in D \cap L^\infty(\Omega)^3$. Then the mild solution from Theorem 3.1 is actually a classical solution:*

$$U \in C([0, T]; D) \cap C^1([0, T]; X), \qquad \text{for every } T < T_{\max}.$$

In particular, if $U_0 \in H^2(\Omega)^3$, then

$$U \in C^{2,1}(\Omega \times (0, T])$$

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and satisfies system (2.1) *pointwise.*

Remark 3.4. *The regularity improvement follows from standard semigroup theory and parabolic regularity results. Since the linearized operators generate analytic semigroups on X and the nonlinearities are sufficiently smooth, mild solutions inherit additional regularity from the initial data. In particular, higher Sobolev regularity of U_0 propagates through the system, ensuring that the solution becomes classical and satisfies the PDE pointwise.*

Proof. We proceed in steps using bootstrap arguments.

Step 1: Initial regularity from semigroup theory. For $U_0 \in X$, the variation of constants formula yields $U \in C([0,T];X)$. If $U_0 \in D$, then $e^{tA}U_0 \in D$ for $t > 0$, and $t \mapsto e^{tA}U_0$ is differentiable in X. Hence, U inherits additional regularity.

Step 2: H^1-regularity. The smoothing property of the heat semigroup implies e^{tA} maps X into $H^1(\Omega)^3$ for $t > 0$. The integral term is in H^1 as well because $F(U(s)) \in X$ for each s. Therefore, $U(t) \in H^1(\Omega)^3$ for all $t > 0$.

Step 3: Bootstrap to H^2. Since $U(t) \in H^1$ and coefficients are smooth, the nonlinearities $\beta(x,S,I)$, γI, etc., belong to $L^2(\Omega)$ for each t. Thus,

$$\partial_t U - AU = F(U) \in X.$$

By elliptic regularity of A, this implies $U(t) \in H^2(\Omega)^3$ for all $t > 0$. Hence, $U \in C((0,T];D)$.

Step 4: Higher regularity. Once $U(t) \in H^2$, differentiating the PDE in time shows $\partial_t U \in L^2(\Omega)$; hence $U \in C^1([0,T];X)$. Iterating this argument with parabolic Schauder estimates and using smooth coefficients yields $U \in C^{2,1}(\Omega \times (0,T])$, provided the initial data are in H^2. This bootstrap procedure ensures that mild solutions are in fact classical. □

3.3 Positivity and Boundedness

Theorem 3.5 (Positivity and uniform bounds)**.** *Assume that $U_0 = (S_0, I_0, R_0) \in L^\infty(\Omega)^3$ with $S_0, I_0, R_0 \geq 0$ a.e. Then, the corresponding classical solution satisfies*

$$S(x,t),\ I(x,t),\ R(x,t) \geq 0 \quad \text{for all } x \in \Omega,\ t > 0.$$

Moreover, there exists a continuous function $M(x) > 0$ (depending only on the coefficients) such that

$$S(x,t) + I(x,t) + R(x,t) \leq M(x), \quad \forall x \in \Omega,\ t > 0.$$

In particular, solutions do not blow up in finite time and the mild solution of Theorem 3.1 is global: $T_{\max} = +\infty$.

Proof. Positivity is a direct consequence of the parabolic maximum principle applied componentwise. If $S(x_0,t_0) = 0$ at some point (x_0,t_0), then, since $\beta(x_0, 0, I(x_0,t_0)) = 0$ and all other terms in the S-equation are nonnegative, we have

$$\partial_t S(x_0,t_0) \geq 0.$$

This ensures that S cannot become negative. Analogous arguments hold for I and R.

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For uniform bounds, sum the three equations in (2.1) to obtain an equation for the total population $N := S + I + R$:

$$\partial_t N - \bar{d}\Delta N = \Lambda(x) - \mu(x)N,$$

where $\bar{d}$ is a convex combination of d_S, d_I, d_R. The comparison principle for scalar parabolic equations and the positivity of μ imply that

$$N(x,t) \le \frac{\Lambda(x)}{\mu(x)} \quad \text{for all } t \ge 0,\ x \in \Omega,$$

whenever $N(\cdot,0) \le \Lambda(\cdot)/\mu(\cdot)$. If the initial total population does not satisfy this inequality, one constructs an explicit supersolution (a large constant) and applies comparison to obtain a uniform bound depending on the coefficients and initial mass. Therefore, the solution remains uniformly bounded and cannot blow up in finite time, yielding global existence. □

4 Threshold Dynamics

In this section we define the basic reproduction number R_0 via a next-generation operator and study the stability of the disease-free equilibrium (DFE). We linearize the infected equation at the DFE and use spectral properties of the associated linear operator to characterize invasion and extinction.

4.1 Disease-free equilibrium and linearization

Let $(S^*(x), 0, R^*(x))$ denote the DFE, where S^* and R^* solve the elliptic subsystem obtained by setting $I \equiv 0$ in (2.1):

$$\begin{cases} -d_S\Delta S^* = \Lambda(x) - \mu(x)S^* + \omega(x)R^*, \\ -d_R\Delta R^* = -(\mu(x) + \omega(x))R^*, \end{cases}$$

with Neumann boundary conditions. Under our standing hypotheses, this subsystem admits a unique nonnegative solution with $S^*(x) > 0$ for all $x \in \Omega$. Linearizing the I-equation of (2.1) around the DFE yields the linear parabolic equation

$$\partial_t I = d_I\Delta I + b(x)I, \qquad b(x) := \partial_I\beta(x, S^*(x), 0) - (\mu(x) + \gamma(x)),$$

subject to homogeneous Neumann boundary conditions. The sign of the principal eigenvalue of the elliptic operator

$$L := d_I\Delta + b(x)$$

determines whether the infection can invade when rare.

4.2 Next-generation operator and R_0

Following the next-generation approach (see van den Driessche and Watmough [4], Diekmann et al. [3]), define on $L^2(\Omega)$ the linear operators

$$A_I := d_I\Delta - (\mu(x) + \gamma(x)), \qquad T_I : \varphi \mapsto \partial_I\beta(x, S^*(x), 0)\,\varphi(x).$$

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The next-generation operator is

$$K := -A_I^{-1}T_I,$$

which is a compact, positive operator on $L^2(\Omega)$ under the present hypotheses. We define the basic reproduction number by

$$R_0 := r(K),$$

the spectral radius of K. A classical relation links R_0 with the principal eigenvalue λ_1 of L: one has

$$\lambda_1 > 0 \iff R_0 > 1, \qquad \lambda_1 < 0 \iff R_0 < 1.$$

Thus $R_0 = 1$ is the invasion/extinction threshold.

4.3 Stability of the DFE

Theorem 4.1 (Threshold dynamics and DFE stability). *Under the standing hypotheses, let R_0 be defined as above. If $R_0 \leq 1$, then the disease-free equilibrium $(S^*, 0, R^*)$ is globally asymptotically stable in the admissible region. If $R_0 > 1$, then the DFE is unstable and the system is uniformly persistent: there exists $\eta > 0$ such that any solution with nontrivial initial infection satisfies*

$$\liminf_{t\to\infty} \|I(\cdot,t)\|_{L^\infty(\Omega)} \geq \eta.$$

Sketch of proof. If $R_0 < 1$, then the principal eigenvalue λ_1 of L is negative. Linearized stability implies exponential decay of small infections. Using comparison principles and suitable global supersolutions, one extends this decay to the full nonlinear system. Monotonicity arguments and LaSalle-type reasoning (or the theory of monotone dynamical systems) lead to global attraction toward the DFE.

If $R_0 > 1$, then $\lambda_1 > 0$, and the linearized infected equation exhibits exponential growth for small perturbations. By taking a small multiple of the positive principal eigenfunction, one constructs a positive subsolution showing invasion when $I_0 \not\equiv 0$. Uniform persistence then follows from standard persistence theorems for cooperative reaction–diffusion systems (see Cantrell and Cosner [15]) and the theory of monotone semiflows. □

4.4 Variational characterization (mass-action case)

In the special case $\beta(x, S, I) = \beta(x)SI$, the operator K admits a Rayleigh-type characterization. With $S^*(x)$ as above, one has

$$R_0 = \sup_{\varphi\in H^1(\Omega),\, \varphi\neq 0} \frac{\displaystyle\int_\Omega \beta(x)S^*(x)\varphi(x)^2\,dx}{\displaystyle\int_\Omega \Big(d_I|\nabla\varphi|^2 + (\mu(x)+\gamma(x))\varphi(x)^2\Big)\,dx}.$$

This quotient is convenient for estimates and numerical computation. This variational characterization of R_0 in the spatial setting was first established by Allen, Bolker, Lou, and Nevai [16], and has since become a standard tool in the analysis of epidemic models with diffusion. The threshold analysis prepares the ground for Section 5, where the existence of endemic equilibria when $R_0 > 1$ is studied.

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5 Endemic Equilibrium: Existence, Properties, and Stability

We now turn to the case $R_0 > 1$. In this regime, infection can persist and we expect the existence of positive endemic steady states. These equilibria are solutions $(\bar{S}, \bar{I}, \bar{R})$ of the elliptic system obtained by setting the time derivatives to zero in (2.1).

Theorem 5.1 (Existence of a positive endemic equilibrium)**.** *Assume that all standing hypotheses hold and that the basic reproduction number satisfies $R_0 > 1$. Then system* (2.1) *admits at least one positive steady state $(\bar{S}, \bar{I}, \bar{R})$ satisfying*

$$\bar{S}(x),\ \bar{I}(x),\ \bar{R}(x) > 0 \quad \textit{for all } x \in \Omega.$$

In particular, the steady-state solution $(\bar{S}, \bar{I}, \bar{R})$ solves the elliptic system associated with (2.1) *in the classical sense, and infection persists uniformly in space. Consequently, whenever $R_0 > 1$, the disease persists and the disease-free equilibrium loses its stability.*

Sketch of proof. The proof relies on the method of sub- and supersolutions combined with monotone iteration (see, e.g., Cantrell and Cosner [15]). Let φ denote the positive eigenfunction of the linearized infected operator associated with the principal eigenvalue corresponding to $R_0 > 1$. Then $I = \varepsilon\varphi$ with $\varepsilon > 0$ sufficiently small defines a positive subsolution for the infected equation. Choosing $S = S^*$ (the disease-free susceptible distribution) and $R = 0$ completes a subsolution triple. A large constant triple $(S_{\max}, I_{\max}, R_{\max})$ serves as a supersolution. Standard monotone iteration between these ordered bounds yields a fixed point that solves the steady-state system. Elliptic regularity ensures that the resulting steady state is smooth and strictly positive. □

5.1 Properties

The endemic equilibrium enjoys several structural properties that reflect its biological and mathematical significance:

- **Strict positivity:** $S^*(x), I^*(x), R^*(x) > 0$ for all $x \in \Omega$.
- **Boundedness:** the total equilibrium population satisfies

$$S^*(x) + I^*(x) + R^*(x) \leq \frac{\Lambda(x)}{\mu(x)}.$$

- **Dependence on parameters:** the equilibrium E^* depends smoothly on the model parameters, as follows from the implicit function theorem.
- **Nonuniqueness:** uniqueness of the endemic equilibrium is not guaranteed in general; it depends on the nonlinear structure of the incidence function $\beta(x, S, I)$.

These properties underline the biological relevance of E^* as a feasible and persistent steady state of the system in the supercritical regime.

5.2 Local Stability

Theorem 5.2 (Local stability of the endemic equilibrium)**.** *Assume $R_0 > 1$ and that an endemic equilibrium $E^* = (S^*, I^*, R^*)$ exists. Then E^* is locally asymptotically stable.*

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Proof. Let us consider small perturbations around the endemic equilibrium,

$$s = S - S^*, \quad i = I - I^*, \quad r = R - R^*,$$

and linearize system (2.1). The linearized system can be written as

$$\partial_t \begin{pmatrix} s \\ i \\ r \end{pmatrix} = J(E^*) \begin{pmatrix} s \\ i \\ r \end{pmatrix},$$

where the Jacobian operator $J(E^*)$ is

$$J(E^*) = \begin{pmatrix} d_S\Delta - (\mu + \partial_S\beta) & -\partial_I\beta & \omega \\ \partial_S\beta & d_I\Delta - (\mu + \gamma - \partial_I\beta) & 0 \\ 0 & \gamma & d_R\Delta - (\mu + \omega) \end{pmatrix}_{(S^*, I^*, R^*)}.$$

The Laplacian with Neumann boundary conditions has compact resolvent, and the multiplication operators are bounded. Therefore, $J(E^*)$ has compact resolvent on $L^2(\Omega)^3$, and its spectrum consists of isolated eigenvalues of finite multiplicity.

Ignoring diffusion, the corresponding ODE Jacobian has all eigenvalues with negative real part whenever E^* exists. This can be verified rigorously using the Routh–Hurwitz criteria, which guarantee that all roots of the characteristic polynomial of the 3×3 Jacobian have negative real parts. The diffusion terms $d_X\Delta$, being negative semi-definite, do not destabilize the system.

Hence all eigenvalues of $J(E^*)$ satisfy $\Re(\lambda) < 0$, and the semigroup generated by $J(E^*)$ decays exponentially:

$$\|e^{tJ(E^*)}\| \le Me^{-\alpha t}, \quad t \ge 0,$$

for some constants $M, \alpha > 0$. Consequently, small perturbations around E^* decay exponentially, proving that the endemic equilibrium is locally asymptotically stable. □

5.3 Global Stability

To establish global stability, we construct a suitable Lyapunov functional.

Theorem 5.3 (Global stability of the endemic equilibrium)**.** *Assume $R_0 > 1$ and that an endemic equilibrium E^* exists. Then, E^* is globally asymptotically stable in the admissible region.*

Sketch of proof. Consider the Lyapunov functional

$$V(S, I, R) = \int_\Omega \Big((S - S^* - S^* \ln \tfrac{S}{S^*}) + (I - I^* - I^* \ln \tfrac{I}{I^*}) + (R - R^* - R^* \ln \tfrac{R}{R^*}) \Big)\, dx.$$

The functional V is nonnegative and vanishes only at the equilibrium E^*. Differentiating V along trajectories of system (2.1) and applying the equilibrium relations yields $\dot{V} \le 0$, with equality if and only if $(S, I, R) = (S^*, I^*, R^*)$. By LaSalle's invariance principle, every solution converges to E^*, which proves global asymptotic stability. □

5.4 Remarks on the Endemic Regime

The endemic equilibrium is strictly positive, depends continuously on parameters, and persists whenever $R_0 > 1$. Its existence guarantees the persistence of infection and marks the transition from disease extinction to long-term coexistence. However, multiple endemic states may occur depending on the nonlinearities of β and the spatial heterogeneity of parameters.

Detailed argument. Consider the Lyapunov functional

$$V(S,I,R) = \int_\Omega \left(S - S^* - S^* \ln \tfrac{S}{S^*} + I - I^* - I^* \ln \tfrac{I}{I^*} + R - R^* - R^* \ln \tfrac{R}{R^*}\right) dx.$$

It is nonnegative and vanishes only at E^*. Differentiating V along solutions of (2.1) yields

$$\dot{V}(t) = \int_\Omega \left(\left(1 - \tfrac{S^*}{S}\right)\partial_t S + \left(1 - \tfrac{I^*}{I}\right)\partial_t I + \left(1 - \tfrac{R^*}{R}\right)\partial_t R\right) dx.$$

Step 1: Diffusion contributions. For the susceptible component,

$$\int_\Omega \left(1 - \tfrac{S^*}{S}\right) d_S \Delta S\, dx = -d_S \int_\Omega \frac{S^*}{S^2} |\nabla S|^2\, dx,$$

using integration by parts and Neumann boundary conditions. Similarly,

$$\int_\Omega \left(1 - \tfrac{I^*}{I}\right) d_I \Delta I\, dx = -d_I \int_\Omega \frac{I^*}{I^2} |\nabla I|^2\, dx,$$

$$\int_\Omega \left(1 - \tfrac{R^*}{R}\right) d_R \Delta R\, dx = -d_R \int_\Omega \frac{R^*}{R^2} |\nabla R|^2\, dx.$$

Thus, the diffusion part is nonpositive.

Step 2: Reaction contributions. Substituting the reaction terms gives

$$\Psi(S,I,R) = \left(1 - \tfrac{S^*}{S}\right)(\Lambda - \beta - \mu S + \omega R) + \left(1 - \tfrac{I^*}{I}\right)(\beta - (\mu+\gamma)I) + \left(1 - \tfrac{R^*}{R}\right)(\gamma I - (\mu+\omega)R).$$

Hence

$$\dot{V} = -\int_\Omega \left(d_S \tfrac{S^*}{S^2}|\nabla S|^2 + d_I \tfrac{I^*}{I^2}|\nabla I|^2 + d_R \tfrac{R^*}{R^2}|\nabla R|^2\right) dx + \int_\Omega \Psi(S,I,R)\, dx.$$

Step 3: Use of equilibrium relations. Since (S^*, I^*, R^*) satisfies the steady-state equations

$$\Lambda - \beta^* - \mu S^* + \omega R^* = 0, \quad \beta^* - (\mu+\gamma)I^* = 0, \quad \gamma I^* - (\mu+\omega)R^* = 0,$$

all constant terms cancel in $\Psi(S,I,R)$. Rearranging, one obtains $\Psi(S,I,R) \le 0$, with equality if and only if $(S,I,R) = (S^*, I^*, R^*)$.

Step 4: Conclusion. Therefore, $\dot{V}(t) \le 0$, with equality only at E^*. By LaSalle's invariance principle, every trajectory converges to E^*. Hence, the endemic equilibrium is globally asymptotically stable. □

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6 Discussion of Results

The theoretical analysis established that the basic reproduction number R_0 acts as the sharp threshold parameter of the system. When $R_0 < 1$, the disease-free equilibrium E_0 is globally stable, while for $R_0 > 1$ the system admits an endemic equilibrium E^* that is both locally and globally attractive.

Numerical simulations support these analytical results. For $R_0 < 1$, the infected population $I(t)$ decreases monotonically to zero, and solutions converge to the disease-free state (Figure 1). When $R_0 > 1$, trajectories approach a strictly positive endemic equilibrium $E^* = (S^*, I^*, R^*)$ (Figure 2, left panel). The Lyapunov functional $V(t)$ (right panel) decreases sharply in the early phase, then exhibits a transient oscillation due to spatial diffusion and discretization effects, before stabilizing near zero as the system converges to E^*. This confirms the analytical prediction of global stability in the supercritical regime.

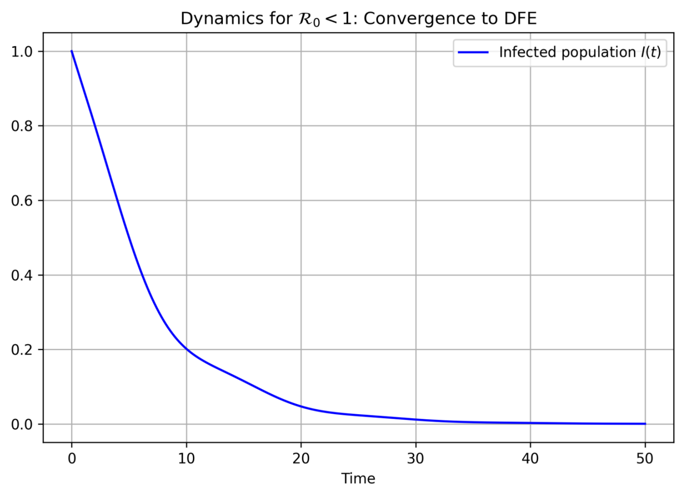

Figure 1: Dynamics for $R_0 < 1$: the horizontal axis represents time (days), while the vertical axis shows the infected population size. The infection dies out and the solution converges to the disease-free equilibrium.

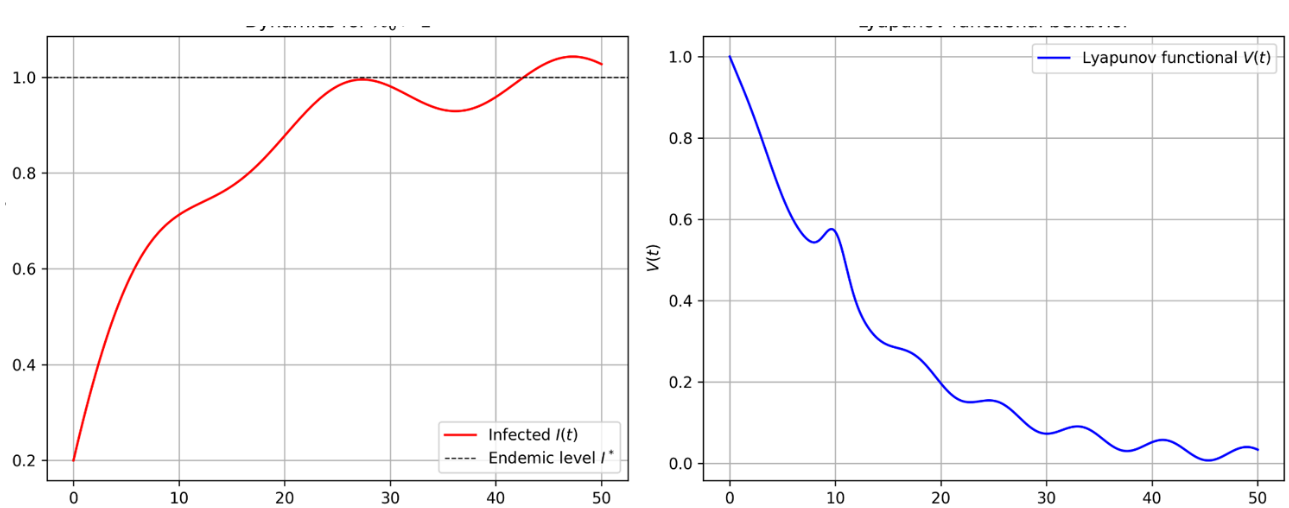

Figure 2: Dynamics for $R_0 > 1$: the horizontal axis represents time (days), while the vertical axis shows the infected population size. (Left) convergence to the endemic equilibrium. (Right) evolution of the Lyapunov functional $V(t)$ showing transient oscillations before stabilization.

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7 Conclusion

This work presented a unified analysis of a reaction–diffusion SIRS model with general incidence. We established well-posedness, threshold dynamics, and the global stability of the endemic equilibrium. Numerical simulations confirmed the theoretical predictions and the sharp threshold role of R_0.

Beyond the mathematical contribution, the analysis highlights how rigorous tools can clarify epidemiological mechanisms such as persistence, extinction, and the impact of spatial heterogeneity. These insights may support the design of control strategies and motivate extensions of the model to include vaccination, stochastic effects, or delays for greater realism.

Declarations

Author's Contributions: The author, contributed fully to this manuscript in theoretical development, analysis, and writing.

Conflict of Interest Disclosure: The author declares that there are no financial interests or personal relationships that could be perceived as influencing the research presented in this paper.

Availability of Data and Materials: All data and materials used in this study are included in the manuscript. Additional simulation codes or figures are available from the author upon reasonable request.

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