

Qualitative Analysis of Infectious Disease Spread in a Spatial SIR Model

Firlya^{1*}, Yudhi¹, Bayu Prihandono¹

¹Department of Mathematics, University of Tanjungpura, Indonesia

h1011221001@student.untan.ac.id

ABSTRACT

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Interpersonal interactions and population mobility play a crucial role in the spread of infectious diseases. However, the classical SIR (Susceptible–Infected–Recovered) model assumes homogeneous mixing and neglects spatial movement. To address this limitation, we investigate disease dynamics using a reaction–diffusion spatial SIR model. We combine analytical and numerical approaches by deriving the basic reproduction ratio ($\mathcal{R}_0$) using the Next Generation Matrix method, determining equilibrium points, and conducting local stability analysis through Jacobian linearization and diffusion-dependent characteristic equations. We further examine spatial disease propagation through traveling wave analysis and derive the minimum wave speed. Numerical simulations based on the Forward Time Centered Space (FTCS) finite difference scheme are performed to validate the analytical results and illustrate spatiotemporal disease dynamics. The results show that $\mathcal{R}_0$ serves as the threshold parameter governing disease persistence. Specifically, $\mathcal{R}_0$ increases with the transmission rate (β) and birth rate (Λ), and decreases with the natural death rate (μ) and recovery rate (γ). The model admits a disease-free equilibrium (E_0) and an endemic equilibrium (E_1). When $\mathcal{R}_0 < 1$, E_0 is locally asymptotically stable, indicating disease eradication. Conversely, when $\mathcal{R}_0 > 1$, E_1 becomes locally asymptotically stable, implying disease persistence. Traveling wave analysis shows that disease invasion occurs only when $\mathcal{R}_0 > 1$, with a minimum wave speed of $c = 2\sqrt{d_2(\gamma + \mu)(\mathcal{R}_0 - 1)}$. FTCS simulations confirm the analytical predictions and demonstrate that increased mobility of infected individuals accelerates spatial disease spread.



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A. INTRODUCTION

Infectious diseases continually challenge public health because they spread rapidly and deeply disrupt communities social and economic lives (Naghavi et al., 2024; Samsudin et al., 2024). Because disease characteristic and human behaviors are highly complex, policymakers cannot rely on a purely medical approach to design effective mitigations strategies. Therefore, researchers now use mathematical modeling and analysis as fundamental tools to understand how epidemics transmit. This approach empowers health officials to predict real-world scenarios, control outbreaks, and prevent future diseases (Brauer, 2017; Buonomo et al., 2018; Sweileh, 2022).

Kermack and McKendrick introduced the SIR compartmental model as an early framework in infectious disease modeling (Diekmann et al., 2025; Mubarez & Mohsini, 2025). This study categorizes the population into three specific groups, applying time-dependent ordinary differential equations (ODEs) to model the transitional flows between them (Brauer et al., 2019;

Kiseleva et al., 2025). Furthermore, the SIR model assumes homogeneous mixing. It treats the population as entirely uniform, ignoring any distance barriers or geographical structures (Porter & Oleson, 2016; Tan et al., 2022; Y. Wang et al., 2024). However, this assumption often fails to reflect real-world conditions because physical space and distance strictly limit population density and human movement (Badr et al., 2020; Kraemer et al., 2020). Consequently, these spatial constraints can skew predictions regarding an outbreak's peak time and the extent of the affected area (Chinazzi et al., 2020; Mendes et al., 2024).

To overcome these limitations and approximate real-world conditions, researchers must develop models that actively integrate spatial variables into disease dynamics. To account for these spatial factors, mathematicians employ systems of partial differential equations, specifically the Reaction-Diffusion model (Davydovych et al., 2023; Viguerie et al., 2020). Consequently, this specific framework produces the Spatial SIR model. Within this formulation, the population density of every subgroup is defined mathematically through both time (t) and physical location (x). Epidemiologists rely on this model because its reaction term captures local biological dynamics such as infection or recovery rates. In contrast, its diffusion term simulates the random movement of individuals from high-density to low-density areas (Kraemer et al., 2020; Wang et al., 2022). By incorporating this diffusion term, the model accurately illustrates how a disease gradually spreads across regions over time, providing a significantly more realistic picture of outbreak dynamics than non-spatial models (Dormy & Soward, 2025; Kraemer et al., 2020).

Previous studies have investigated different aspects of reaction–diffusion epidemic models. For example, Lei et al. (2024) and Xin & Wang (2023) primarily focused on stability properties, threshold dynamics, and long-term behavior of spatial epidemic systems. Meanwhile, Guenad et al. (2025) and Han & Xu (2024) concentrated on the existence of traveling wave solutions and the determination of wave propagation speeds in reaction–diffusion epidemic models. Although these studies have provided important theoretical insights, they generally examine stability analysis and traveling wave behavior separately. Consequently, the relationship between stability thresholds governed by the basic reproduction ratio ($\mathcal{R}_0$) and the emergence of traveling epidemic waves remains insufficiently explored, particularly within the classical spatial SIR framework. Understanding this relationship is essential because stability analysis determines whether a disease can persist, whereas traveling wave analysis characterizes how rapidly the disease invades new susceptible regions.

Motivated by this gap, this study provides an integrated qualitative analysis of a reaction–diffusion spatial SIR model by combining equilibrium analysis, stability analysis, and traveling wave investigation within a unified framework. Unlike previous studies that primarily focus on either threshold dynamics or wave propagation, this research explicitly connects the stability condition governed by the basic reproduction ratio ($\mathcal{R}_0$) with the existence of traveling wave solutions and their minimum propagation speed. This integrated perspective provides a more comprehensive understanding of the interplay between disease persistence and spatial invasion in epidemic systems.

Based on the identified research gap, this study investigates the relationship between equilibrium stability and traveling wave propagation in a reaction–diffusion spatial SIR model. Specifically, we derive the basic reproduction ratio ($\mathcal{R}_0$), analyze the stability properties of the

equilibrium points, and examine the existence of traveling wave solutions together with their minimum propagation speed. To support the analytical findings, we perform numerical simulations using the Forward Time Centered Space (FTCS) finite difference scheme and illustrate the spatiotemporal spread of infectious diseases.

B. METHODS

This study is a theoretical and computational research in mathematical epidemiology. The study investigates the qualitative dynamics of infectious disease transmission using a reaction–diffusion spatial SIR model represented by a system of partial differential equations. The epidemiological system consists of susceptible (S), infected (I), and recovered (R) populations whose dynamics are governed by disease transmission, recovery, demographic processes, and spatial diffusion. The detailed model assumptions are presented in the Model Formulation subsection.

We formulate a reaction–diffusion spatial SIR model based on the underlying epidemiological assumptions and determine its equilibrium points by considering the spatially homogeneous system. Subsequently, we derive the basic reproduction ratio ($\mathcal{R}_0$) using the Next Generation Matrix approach and investigate the local stability of the equilibrium points through Jacobian linearization and characteristic equations. After establishing the stability characteristics of the equilibrium points, we perform a traveling wave analysis to investigate the spatial propagation of infectious diseases. We transform the spatiotemporal reaction–diffusion system into a corresponding system of ordinary differential equations (ODEs) by introducing the traveling wave coordinate $\xi = x + ct$ and converting the resulting second-order equations into a first-order system (Bai & Zhang, 2015). Based on this transformation, we determine the minimum wave speed c^* and analyze the resulting wave propagation profiles to characterize the spatial spread of the disease.

To verify the analytical results and visualize the spatiotemporal behavior of the proposed model, we conduct numerical simulations using the Forward Time Centered Space (FTCS) finite difference scheme to solve the reaction–diffusion system. We discretize the spatial domain $x \in [0,1]$ into 100 grid points with a spatial step size of $\Delta x = 0.1010$ and employ a time step size of $\Delta t = 0.00816$ for temporal discretization. We approximate the first-order time derivatives using forward finite differences and the second-order spatial derivatives using centered finite differences. To ensure numerical stability, we verify the FTCS stability criterion based on the largest diffusion coefficient in the system, and the selected values of Δx and Δt satisfy the stability requirement of the numerical scheme. We then solve the resulting discretized system iteratively subject to the initial conditions and homogeneous Neumann boundary conditions. Finally, we implement all numerical simulations in Python to visualize the spatiotemporal dynamics of the susceptible, infected, and recovered populations, verify the analytical stability results, and illustrate the spatial propagation patterns predicted by the traveling wave analysis. To provide a clear overview of the research procedure, Figure 1 illustrates the overall research framework adopted in this study, including model formulation, equilibrium point determination, basic reproduction ratio calculation, stability analysis, traveling wave analysis, numerical simulation, and interpretation of results.

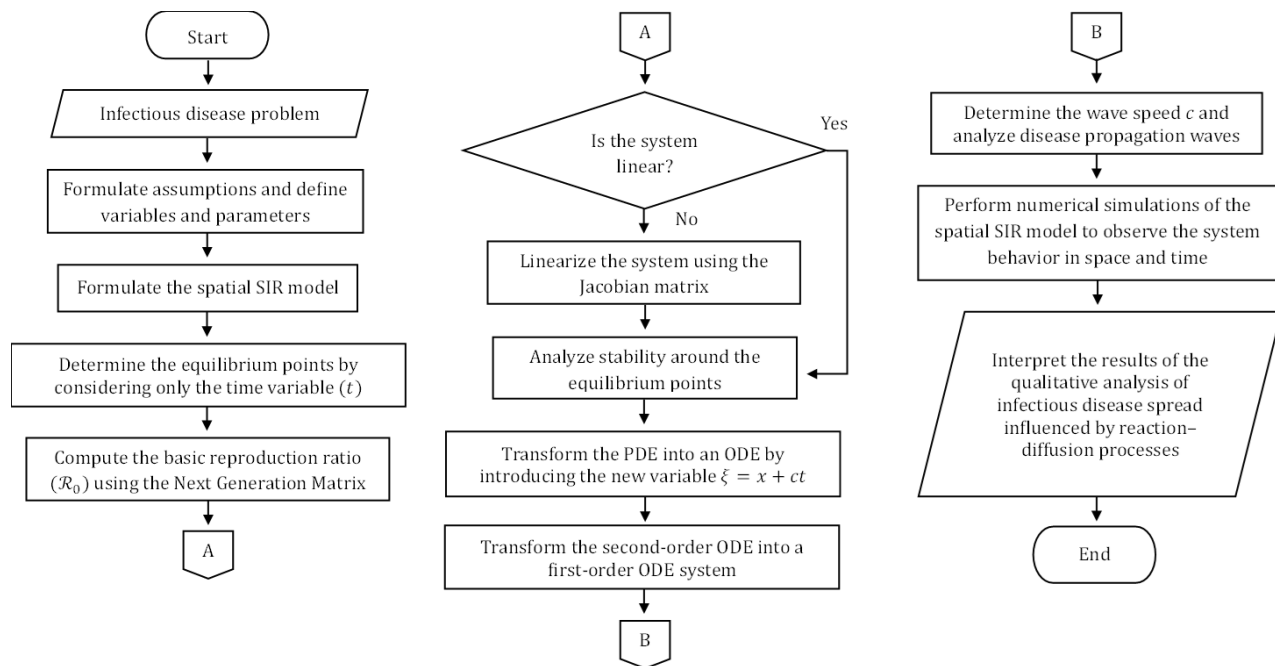


Figure 1. Flowchart of the Qualitative Analysis of the Spatial SIR Model

C. RESULT AND DISCUSSION

1. Formulation of the Epidemic Model

In this study, we construct spatial SIR epidemiological model that applies a reaction-diffusion mechanism to represent disease spread dynamics across time (t) and space (x). Within this formulation, the entire demographic is categorized into three separate epidemiological classes: Susceptible (S), Infected (I), and Recovered (R). To describe the random movement of individuals, we use a diffusion mechanism denoted by the diffusion coefficient d and the Laplacian operator $\nabla^2 = \frac{\partial^2}{\partial x^2}$. Meanwhile, the reaction mechanism captures the biological interactions between individuals. We base this model on the following core assumptions:

- Migration does not occur into or out of the region during the observation period. Only births, natural deaths, and health status transitions influence population changes.
- Susceptible individuals (S) can contract the infection and transition to (I). Infected individuals (I) can recover and transition to (R), acquiring permanent immunity.
- Transmission occurs randomly at a rate β , which directly proportionates to the contact between susceptible and infected individuals, while recovery proceeds at a constant rate γ .
- The model represents individual movement as diffusion using a one-dimensional Laplace operator. Each subpopulation possesses constant, non-negative diffusion coefficients (d_1, d_2, d_3).
- The domain constitutes a bounded region Ω subject to Neumann homogeneous boundary constraints, thereby guaranteeing zero population flow through the boundaries.

We formulated the following dynamical system to describe the propagation of the outbreak.

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

System (1) excludes diffusion effects. Next, by incorporating diffusion effects into each compartment, we obtain the reaction-diffusion model shown in System (2).

$$\left. \begin{aligned} \frac{\partial S(x, t)}{\partial t} &= d_1 \frac{\partial^2 S(x, t)}{\partial x^2} + \Lambda - \beta S(x, t)I(x, t) - \mu S(x, t) \\ \frac{\partial I(x, t)}{\partial t} &= d_2 \frac{\partial^2 I(x, t)}{\partial x^2} + \beta S(x, t)I(x, t) - \gamma I(x, t) - \mu I(x, t) \\ \frac{\partial R(x, t)}{\partial t} &= d_3 \frac{\partial^2 R(x, t)}{\partial x^2} + \gamma I(x, t) - \mu R(x, t) \end{aligned} \right\} \quad (2)$$

For $t > 0$, $x \in \Omega$ with Neumann homogeneous boundary constraints:

$$\frac{\partial S(x, t)}{\partial n} = \frac{\partial I(x, t)}{\partial n} = \frac{\partial R(x, t)}{\partial n} = 0 \quad (3)$$

Equation (3) specifies that the population flux across the domain boundary is zero and initial conditions

$$S(x, 0) = \varphi_1(x) \geq 0, I(x, 0) = \varphi_2(x) \geq 0, R(x, 0) = \varphi_3(x) \geq 0 \quad (4)$$

We define the parameters for system (2) in Table 1 below.

Table 1. Parameters of The System (2)

Parameter	Description	Unit	Conditions
β	Transmission rate of infectious diseases	area/(people $\times$ time)	$\beta > 0$
γ	Recovery rate of infectious diseases	1/time	$\gamma > 0$
Λ	Population recruitment rate	people/(area $\times$ time)	$\Lambda > 0$
μ	Natural population mortality rate	1/time	$\mu > 0$
d_1	Movement rate of susceptible populations in space	area/time	$d_1 \geq 0$
d_2	Movement rate of infected populations in space	area/time	$d_2 \geq 0$
d_3	Movement rate of recovered populations in space	area/time	$d_3 \geq 0$
$\frac{\partial^2 S(x, t)}{\partial x^2}$	Random movement of susceptible populations	people/area ²	$\nabla^2 S(x, t) \geq 0$
$\frac{\partial^2 I(x, t)}{\partial x^2}$	Random movement of infected populations	people/area ²	$\nabla^2 I(x, t) \geq 0$

Parameter	Description	Unit	Conditions
$\frac{\partial^2 R(x, t)}{\partial x^2}$	Random movement of recovered populations	people/area ²	$\nabla^2 R(x, t) \geq 0$

2. Equilibrium Point without Spatial

To determine the equilibrium point of the Spatial SIR model, we assume a spatially homogeneous system that depends only on time, effectively ignoring the diffusion effect (Lotfi et al., 2014). Consequently, we set each spatial derivative to zero, thereby simplifying the model to a system of ODE. We obtain equilibrium points of this infectious disease spread model by setting $\frac{dS(t)}{dt} = \frac{dI(t)}{dt} = \frac{dR(t)}{dt} = 0$. This calculation yields two distinct states: a disease-free equilibrium $(E_0) = (S_0, I_0, R_0) = \left(\frac{\Lambda}{\mu}, 0, 0\right)$ and the endemic disease equilibrium point $(E_1) = (S_1, I_1, R_1) = \left(\frac{\gamma+\mu}{\beta}, \frac{\Lambda}{(\gamma+\mu)} - \frac{\mu}{\beta}, \frac{\gamma}{\mu(\gamma+\mu)} \left(\Lambda - \frac{\mu(\gamma+\mu)}{\beta}\right)\right)$.

3. Basic Reproduction Ratio

We apply Theorem 1 to determine the basic reproduction ratio for system (2).

Theorem 1. (Yang & Wang, 2023) If F is a triangular matrix, then $\mathcal{R}_0^{PDE} = \mathcal{R}_0^{ODE}$. Particularly, if $m = 1$, then $\mathcal{R}_0^{PDE} = \mathcal{R}_0^{ODE}$.

Based on Theorem 1, we calculate $\mathcal{R}_0$ using the Next Generation Matrix because system (2) contains only a single infected compartment. We define the dynamic equation for this infected subpopulation as follows:

$$\frac{\partial I(x, t)}{\partial t} = d_2 \frac{\partial^2 I(x, t)}{\partial x^2} + \beta S(x, t)I(x, t) - \gamma I(x, t) - \mu I(x, t) \quad (5)$$

We separate Equation (5) into the transmission incidence and the removal rate from the infectious class. Consequently, the relationship is formulated as follows:

$$\frac{\partial I(x, t)}{\partial t} = d_2 \frac{\partial^2 I(x, t)}{\partial x^2} + \mathcal{F}(I) - \mathcal{V}(I) \quad (6)$$

With

$$\mathcal{F}(I) = \beta S(x, t)I(x, t), \mathcal{V}(I) = \gamma I(x, t) + \mu I(x, t)$$

Next, we evaluate the partial derivatives of functions $\mathcal{F}(I)$ and $\mathcal{V}(I)$ at E_0 to obtain:

$$F(I) = \frac{\beta\Lambda}{\mu}, V(I) = \gamma + \mu$$

By applying Theorem 1, we obtain the basic reproduction ratio for system (2) to be the following expression:

$$\mathcal{R}_0^{PDP} = \mathcal{R}_0^{PDB} = \frac{\beta\Lambda}{\mu(\gamma+\mu)}$$

From an epidemiological perspective, the basic reproduction ratio acts as a threshold parameter that determines whether an infectious disease can invade the population. When $\mathcal{R}_0 < 1$, each infected individual generates less than one secondary infection on average, causing the disease to gradually disappear from the population. Conversely, when $\mathcal{R}_0 > 1$, the number of new infections exceeds the number of recoveries, allowing the disease to persist and establish an endemic state. These findings highlight the importance of reducing transmission rates or increasing recovery rates to maintain $\mathcal{R}_0$ below unity and prevent sustained outbreaks.

4. Analysis Stability

To analyze the behavior of the system (2) around the equilibrium point and assess the effect of diffusion on stability, we linearize the reaction-diffusion model at the equilibrium point $E^* = (s^*, i^*, r^*)$. In this process, we express s, i , and r as small perturbations from the equilibrium point $E^* = (s^*, i^*, r^*)$. To analyze stability, we apply a small perturbation around the equilibrium point, expressed as

$$u = \begin{pmatrix} s \\ i \\ r \end{pmatrix}$$

We express the linear system resulting from the linearization as follows:

$$\frac{\partial u}{\partial t} = Ju - D \frac{\partial^2 u}{\partial x^2}$$

With

$$J = \begin{pmatrix} \frac{\partial f}{\partial S} & \frac{\partial f}{\partial I} & \frac{\partial f}{\partial R} \\ \frac{\partial g}{\partial S} & \frac{\partial g}{\partial I} & \frac{\partial g}{\partial R} \\ \frac{\partial h}{\partial S} & \frac{\partial h}{\partial I} & \frac{\partial h}{\partial R} \end{pmatrix}_{(s^*, i^*, r^*)} \quad D = \begin{pmatrix} d_1 & 0 & 0 \\ 0 & d_2 & 0 \\ 0 & 0 & d_3 \end{pmatrix}$$

To analyse the system's stability against spatial perturbations, we assume a small perturbation solution around the equilibrium point as follows (Lotfi et al., 2014):

$$u = u^* e^{\lambda t} e^{ikx}$$

From this assumption, we obtain the following derivatives:

$$\frac{\partial u}{\partial t} = \lambda u, \quad \frac{\partial^2 u}{\partial x^2} = -k^2 u$$

Substituting these derivatives into the system yields the following characteristic equation:

$$|J - k^2 D - \lambda I| = 0$$

In the following theorem, we provide a characterization of the local stability for the equilibrium points:

Theorem 2. If $\mathcal{R}_0 < 1$, the disease-free equilibrium points (E_0) of system (2) is locally asymptotically stable. Conversely, if $\mathcal{R}_0 > 1$, the disease-free equilibrium points (E_0) of system (2) is unstable.

Proof. We evaluate the Jacobian matrix at E_0 for system (2), which yields:

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{\beta\Lambda}{\mu} & 0 \\ 0 & \frac{\beta\Lambda}{\mu} - (\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

and we obtain the characteristic polynomial for the matrix $J(E_0)$ as follows:

$$(-\mu - k^2 d_1 - \lambda)(-\mu - k^2 d_3 - \lambda) \left(\frac{\beta\Lambda}{\mu} - (\gamma + \mu) - k^2 d_2 - \lambda \right) = 0$$

Given that all parameters are strictly positive, we obtain the following three eigenvalues:

$$\begin{aligned} \lambda_1 &= -(\mu + k^2 d_1) < 0 \\ \lambda_2 &= -(\mu + k^2 d_3) < 0 \\ \lambda_3 &= \frac{\beta\Lambda}{\mu} - (\gamma + \mu) - k^2 d_2 = -[(1 - \mathcal{R}_0)(\gamma + \mu) + k^2 d_2] \end{aligned}$$

We evaluate two conditions for the eigenvalue λ_3 . First, if $\mathcal{R}_0 < 1$, then $(1 - \mathcal{R}_0) > 0$, so that λ_3 is negative, rendering λ_3 negative. Consequently, all eigenvalues of $J(E_0)$ are strictly negative, proving that disease-free equilibrium point (E_0) of system (2) is locally asymptotically stable when $\mathcal{R}_0 < 1$. Second, if $\mathcal{R}_0 > 1$, then $(1 - \mathcal{R}_0) < 0$, which makes λ_3 positive. Because not all eigenvalues remain negative, Theorem 2 proves that the disease-free equilibrium point (E_0) becomes unstable when $\mathcal{R}_0 > 1$. ■

Theorem 3. If $\mathcal{R}_0 > 1$, then the endemic disease equilibrium points (E_1) of system (2) is locally asymptotically stable.

Proof. We evaluate the Jacobian matrix at E_1 for system (2) to obtain:

$$J(E_1) = \begin{bmatrix} -(\mu + \beta I_1) & -\gamma - \mu & 0 \\ \beta I_1 & 0 & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

which yields the following characteristic equation for the matrix $J(E_1)$:

$$(-\mu - k^2 d_3 - \lambda)(\lambda^2 + a\lambda + b) = 0 \quad (7)$$

where we define the coefficients a and b as:

$$\begin{aligned} a &= k^2(d_1 + d_2) + \mu + I_1 \mathcal{R}_0 \left(\frac{\mu(\gamma + \mu)}{\gamma} \right) \\ b &= k^4 d_1 d_2 + k^2 d_2 \mu + I_1 \mathcal{R}_0 \left(\frac{\mu(\gamma + \mu)}{\gamma} \right) (k^2 d_2 + (\gamma + \mu)) \end{aligned}$$

From characteristic equation (7), we derive two distinct possibilities

$$-\mu - k^2 d_3 - \lambda = 0 \text{ atau } \lambda^2 + a\lambda + b = 0$$

The first possibility yields:

$$-\mu - k^2 d_3 - \lambda = 0 \Leftrightarrow \lambda = -\mu - k^2 d_3$$

Because the parameter μ is strictly positive, this eigenvalue always has a negative real part. Next, the second possibility gives the following quadratic equation:

$$\lambda^2 + a\lambda + b = 0 \quad (8)$$

We analyze the roots of equation (8) using Routh's criterion (Olsder et al., 2011). According to this criterion, the root of equation (8) have negative real parts, which is ensured exactly $a > 0$ and $b > 0$. We observe that the first condition, $a > 0$ is satisfied when $\mathcal{R}_0 > 1$, and the second condition, $b > 0$ is also satisfied when $\mathcal{R}_0 > 1$. Consequently, we prove that if $\mathcal{R}_0 > 1$, the endemic equilibrium points (E_1) of system (2) is locally asymptotically stable. ■

Biologically, we interpret the stability of the disease-free equilibrium as evidence that small introductions of infected individuals cannot trigger large-scale outbreaks when $\mathcal{R}_0$ remains below one. In contrast, we find that the stability of the endemic equilibrium for $\mathcal{R}_0 > 1$ allows the disease to persist despite small perturbations in population density. Therefore, our stability analysis confirms that the epidemic threshold determined by $\mathcal{R}_0$ governs the long-term persistence of the disease in a spatial environment

5. Analysis of Travelling Wave Existence

To examine the dynamics of travelling wave solutions for the model in (2), We reduce the spatiotemporal PDE framework into a corresponding system of ordinary differential equations (ODEs) by defining the wave coordinate variable $\xi = x + ct$. This transformation yields the following system:

$$\left. \begin{aligned} cS' &= d_1 S'' + \Lambda - \beta SI - \mu S \\ cI' &= d_2 I'' + \beta SI - \gamma I - \mu I \\ cR' &= d_3 R'' + \gamma I - \mu R \end{aligned} \right\} \quad (9)$$

Next, we convert the second-order equation into a first-order system. By letting $S' = U$, $I' = V$ and $R' = W$, we obtain:

$$\left. \begin{aligned} S' &= U \\ U' &= \frac{c}{d_1} U - \frac{1}{d_1} (\Lambda - \beta SI - \mu S) \\ I' &= V \\ V' &= \frac{c}{d_2} V - \frac{1}{d_2} (\beta SI - \gamma I - \mu I) \\ R' &= W \\ W' &= \frac{c}{d_3} W - \frac{1}{d_3} (\gamma I - \mu R) \end{aligned} \right\} \quad (10)$$

We evaluate the Jacobian matrix for system (10) at E_0 to obtain:

$$J_0 = \begin{bmatrix} 0 & 1 & 0 & 0 & 0 & 0 \\ \frac{\mu}{d_1} & \frac{c}{d_1} & \frac{\beta\Lambda}{d_1\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & \frac{-\beta\Lambda + \gamma\mu + \mu^2}{d_2\mu} & \frac{c}{d_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & -\frac{\gamma}{d_3} & 0 & \frac{\mu}{d_3} & \frac{c}{d_3} \end{bmatrix} \quad (11)$$

From J_0 , we derive the following characteristic equation:

$$\frac{(-d_3\lambda^2 + c\lambda + \mu)(d_2\lambda^2\mu - c\lambda\mu + \Lambda\beta - \gamma\mu - \mu^2)(-d_1\lambda^2 + c\lambda + \mu)}{\mu d_1 d_2 d_3} = 0$$

Solving this equation yields the following eigenvalues:

$$\begin{aligned} \lambda_{1,2} &= \frac{c \pm \sqrt{c^2 + 4d_1\mu}}{2d_1} \\ \lambda_{3,4} &= \frac{c \pm \sqrt{c^2 + 4d_3\mu}}{2d_3} \\ \lambda_{5,6} &= \frac{c \pm \sqrt{c^2 - 4d_2\left(\frac{\beta\Lambda}{\mu} - \gamma - \mu\right)}}{2d_2} \end{aligned}$$

Given that the wave speed c and all parameters are strictly positive, we observe that λ_1 and λ_3 are positive, while λ_2 and λ_4 are negative. To ensure a biologically meaningful travelling wave solution (without spatial oscillations), all eigenvalues must be real numbers. Consequently, the pair of eigenvalues λ_5 and λ_6 must also be real. Therefore, the value inside the square root must satisfy

$$\begin{aligned} c^2 - 4d_2 \left(\frac{\beta\Lambda}{\mu} - \gamma - \mu \right) &\geq 0 \\ c^2 &\geq 4d_2 \left(\frac{\beta\Lambda}{\mu} - \gamma - \mu \right) \end{aligned} \quad (12)$$

From inequality (12), we derive the minimum speed of the travelling wave as follows:

$$c = \sqrt{4d_2 \left(\frac{\beta\Lambda}{\mu} - \gamma - \mu \right)} = 2\sqrt{d_2(\gamma + \mu)(\mathcal{R}_0 - 1)} \quad (13)$$

Based on equation (13), we observe that if $\mathcal{R}_0 > 1$, the minimum wave speed c takes a positive real value. This condition indicates that the disease can persist and spread spatially as a travelling wave with a minimum speed of c . Conversely, if $\mathcal{R}_0 < 1$, a biologically meaningful wave speed cannot exist, preventing the formation of travelling waves. Furthermore, we note that the minimum wave speed c is directly proportional to the diffusion coefficient d_2 and the difference $\mathcal{R}_0 - 1$. This proportionality implies that increased spatial mobility and higher transmission potential will directly accelerate the rate of disease invasion.

From an epidemiological perspective, the traveling wave solution describes how an infection invades disease-free regions from an initially infected area. The minimum wave speed represents the slowest rate at which the epidemic front advances through space. Consequently, a larger wave speed corresponds to faster geographical disease invasion. This result suggests that increased mobility among infected individuals may enhance disease transmission across neighboring regions, whereas mobility restrictions may help slow the spatial expansion of an outbreak.

6. Numerical Simulation

We conducted numerical simulations to analyze the stability and spatial spread of the disease. Through these simulations, we observed the system's spatial-temporal behavior. For simplicity, we subject system (2) to Neumann homogeneous boundary constraints

$$\frac{\partial S(x, t)}{\partial n} = \frac{\partial I(x, t)}{\partial n} = \frac{\partial R(x, t)}{\partial n} = 0 \quad t > 0 \quad x = 0, 1 \quad (14)$$

and the following initial conditions:

$$S(x, 0) = \begin{cases} 1, & 0 \leq x < 0,5 \\ 1, 1(1 - x), & 0,5 \leq x \leq 1 \end{cases} \quad (15)$$

$$I(x, 0) = \begin{cases} 0,5x, & 0 \leq x < 0,5 \\ 0,5(1 - x), & 0,5 \leq x \leq 1 \end{cases}$$

$$R(x, 0) = \begin{cases} 0,1x, & 0 \leq x < 0,5 \\ 0,1(1 - x), & 0,5 \leq x \leq 1 \end{cases}$$

We conducted the simulations by varying the transmission rate parameter β . Specifically, we set $\beta = 0,2$ to represent the scenario where $\mathcal{R}_0 > 1$, and the $\beta = 0,1$ for $\mathcal{R}_0 < 1$. All other parameter values were taken from Table 2.

Table 2. Numerical Simulation Parameter Values for Disease Spread

Parameter	Value	Unit	Source
γ	0,5	1/month	(Lotfi et al., 2014)
Λ	0,5	people/($\text{km}^2 \times \text{month}$)	(Lotfi et al., 2014)
μ	0,1	1/month	(Lotfi et al., 2014)
d_1	0,1	km^2/month	(Lotfi et al., 2014)
d_2	0,5	km^2/month	(Lotfi et al., 2014)
d_3	0,1	km^2/month	Assumed

Applying the values listed in Table 2 with $\beta = 0,2$, we obtain a basic reproduction ratio of $\mathcal{R}_0 = 1,6 > 1$. This shows that the disease has the potential to spread across the population, eventually creating a spatial propagation wave. Furthermore, using Equation (13), we calculate the minimum traveling wave speed as $c = 0,848 \text{ km/month}$. The simulation results for each subpopulation are as shown in Figure 2, Figure 3, and Figure 4.

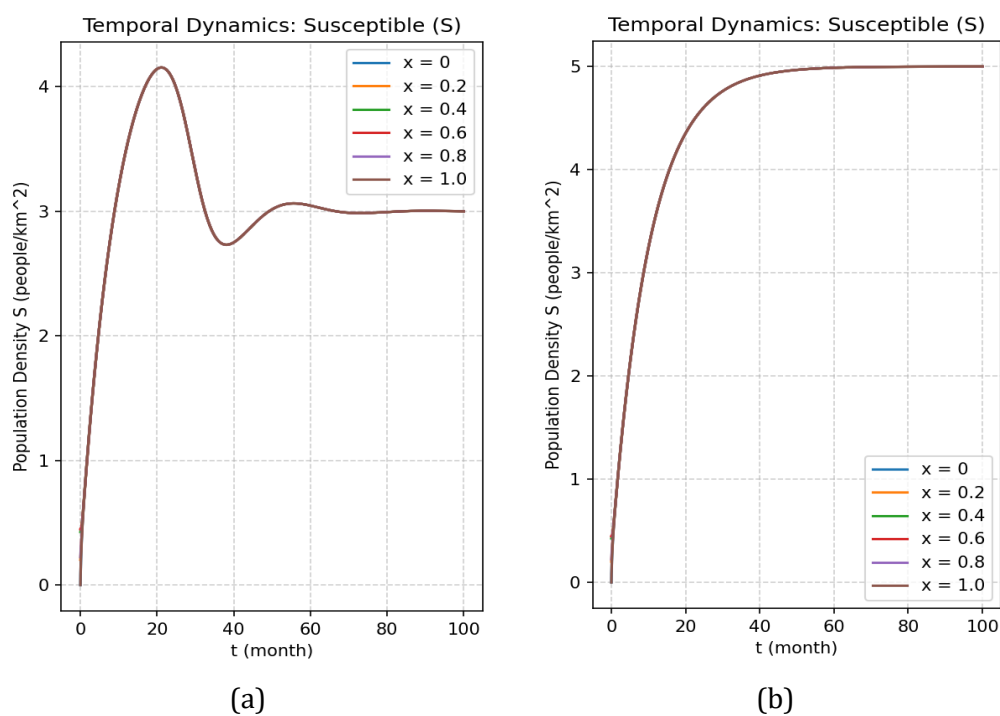


Figure 2. Dynamics of Subpopulation S (a) $\mathcal{R}_0 > 1$, (b) $\mathcal{R}_0 < 1$

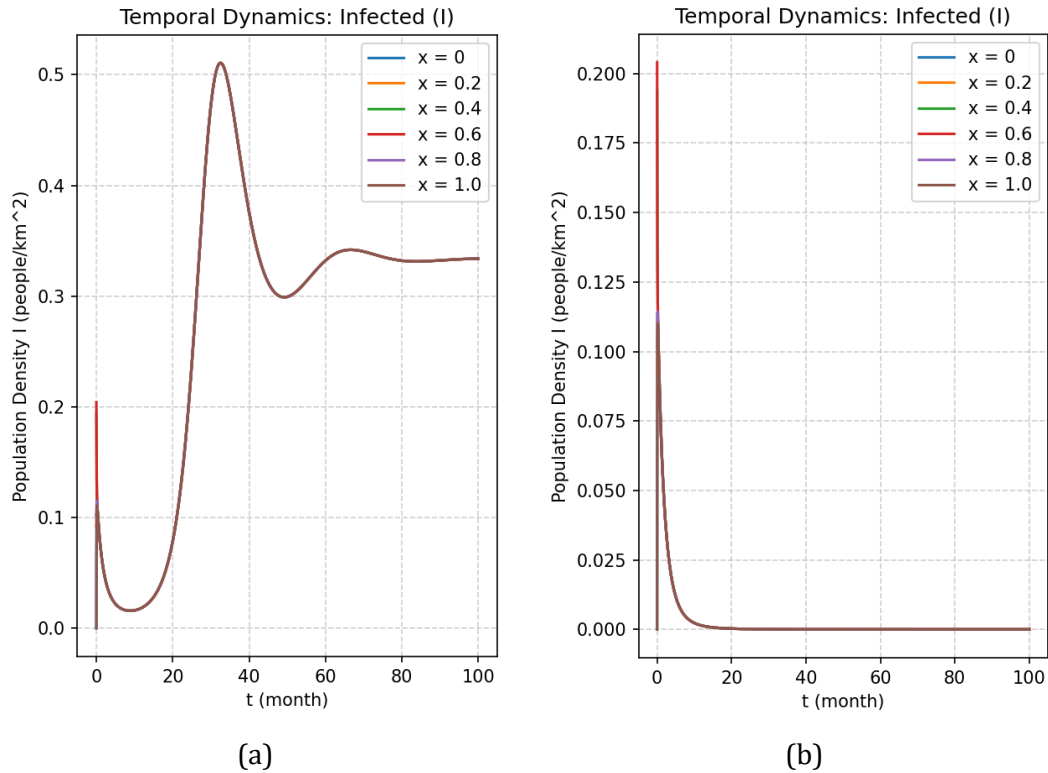


Figure 3. Dynamics of Subpopulation I (a) $\mathcal{R}_0 > 1$, (b) $\mathcal{R}_0 < 1$

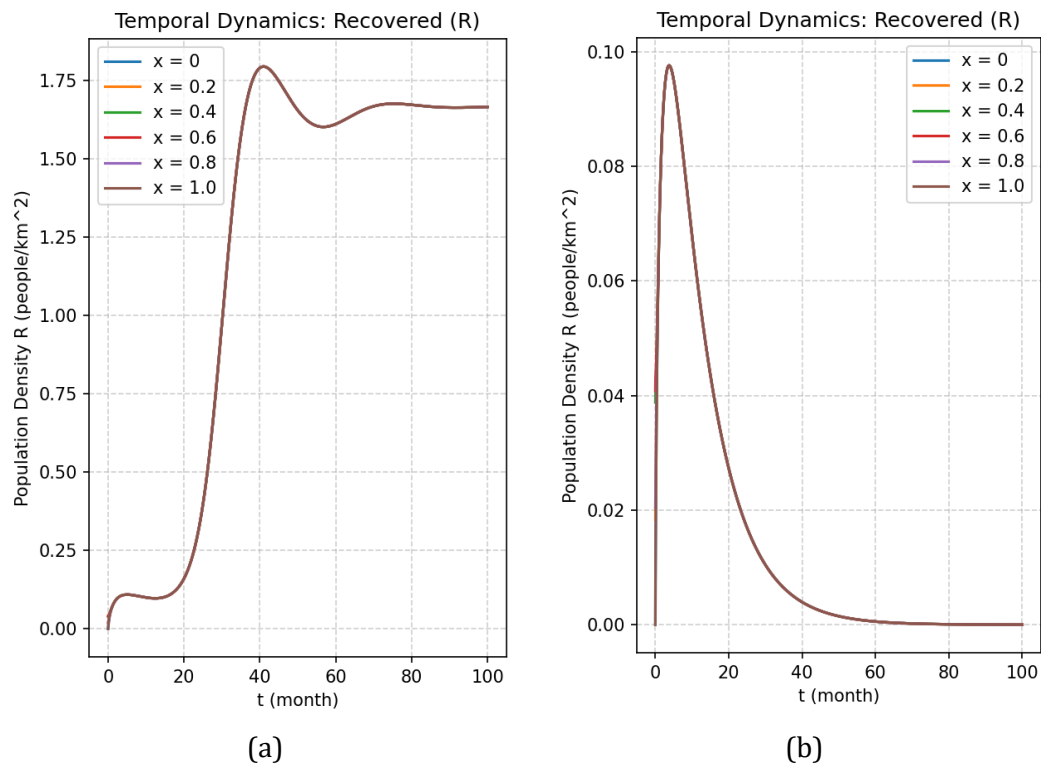


Figure 4. Dynamics of Subpopulation R (a) $\mathcal{R}_0 > 1$, (b) $\mathcal{R}_0 < 1$

The simulations illustrate two distinct dynamical behaviors. When the transmission rate is low ($\mathcal{R}_0 < 1$), the infected population decreases slowly to zero, verifying that the disease will ultimately go extinct. Conversely, when the transmission rate is high ($\mathcal{R}_0 > 1$), it infected population persists and stabilizes at a specific density, which is characteristic of an endemic

state. Furthermore, the spatial visualizations highlight the critical role of diffusion. The initial infection, which is concentrated at the center of the domain, gradually diffuses outward until it is evenly distributed throughout the entire area over time.

Our findings are consistent with those reported by Lei et al. (2024) and Xin & Wang (2023), which identified the basic reproduction ratio ($\mathcal{R}_0$) as the key threshold governing disease persistence and extinction. Likewise, the existence of traveling wave solutions and a minimum wave speed agrees with the results obtained by Guenad et al. (2025) and Han & Xu (2024). However, unlike these studies, we combine stability analysis and traveling wave analysis within a unified spatial SIR framework, providing a more comprehensive understanding of the relationship between epidemic thresholds and spatial disease invasion.

D. CONCLUSION AND SUGGESTIONS

This study investigated a reaction–diffusion spatial SIR model through equilibrium analysis, determination of the basic reproduction ratio, stability analysis, traveling wave analysis, and numerical simulations. The results show that the basic reproduction ratio governs disease persistence or extinction, while traveling wave solutions describe the spatial spread of infection across regions. The main contribution of this work is the integration of stability and traveling wave analyses within a unified spatial SIR framework, providing a comprehensive link between epidemic thresholds and spatial invasion dynamics. However, the model remains theoretical and has not been validated with real epidemiological data, and it assumes homogeneous diffusion without accounting for environmental heterogeneity, human mobility networks, or spatially varying transmission rates. Future research should validate the model using empirical data and extend it to incorporate realistic spatial and demographic factors to improve its applicability for public health planning and epidemic control.

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