



*Research article***Numerical simulation of spatiotemporal dynamics of a two-dimensional reaction–diffusion leprosy transmission model****Amir Khan¹, Shafiullah¹ and Awatif J. Alqarni^{2,*}**¹ Department of Mathematics and Statistics, University of Swat, KP, Pakistan² Department of Mathematics, College of Science, University of Bisha, P.O. Box 551, Bisha, 61922, Saudi Arabia*** Correspondence:** Email: aaljhman@ub.edu.sa.

Abstract: Leprosy remains one of the biggest public health problems in endemic countries, even with the availability of effective treatments. To better understand how the disease spreads over both time and space, this study extends a standard six-compartment mathematical model into a two-dimensional spatial model using partial differential equations. By including spatial movement (Fickian diffusion), we can simulate how different groups of people interact and move across a given geographical area. We construct two numerical schemes in order to solve this mathematical system: the forward Euler scheme and the Crank–Nicolson operator-splitting method. We analyze the stability properties of both schemes: the forward Euler method is conditionally stable subject to a Courant–Friedrichs–Lewy (CFL) condition, and the Crank–Nicolson operator-splitting method is conditionally stable due to the explicit treatment of the reaction terms, with its diffusion stages being individually unconditionally stable. Moreover, we simulate our model using MATLAB the long-term stability of the disease. The results of the simulation clearly demonstrate the temporal evolution and geographical spread of leprosy over time, which is a useful tool for public health planning and disease control interventions.

Keywords: leprosy model; reaction–diffusion system; partial differential equations; spatiotemporal dynamics; forward Euler method; Crank–Nicolson operator splitting

Mathematics Subject Classification: 35K57, 65M06, 92D30

1. Introduction

Leprosy or Hansen’s disease is a chronic infection disease caused by the bacterium *Mycobacterium leprae* [1, 2]. It mostly affects the skin, peripheral nerves, and upper respiratory tract. However, it may result in serious and lifelong disabilities if not treated [3]. Although it is one of the oldest known diseases, leprosy continues to impact communities worldwide. According to the World

Health Organization (WHO) in 2024, approximately 172,717 new cases were reported, with most of the disease burden in India, Brazil, and Indonesia [4]. Since the introduction of multi-drug therapy (MDT) in the 1980s, the global prevalence of leprosy has been reduced to below 1 case per 10,000 population at the global level by the year 2000, leading the WHO to declare leprosy eliminated as a public health problem at that threshold. More recently, the WHO's Global Leprosy Strategy 2021–2030 aims to further reduce new case detection, prevent disabilities, and interrupt transmission in remaining endemic communities. Nevertheless, there is a possibility of continued local transmission in endemic communities, including in children. This is further complicated by the very long and variable leprosy incubation period of *M. leprae* ranging from 3 to 15 years, making the detection and surveillance of leprosy very difficult in the early stages [5, 6].

Leprosy is clinically divided into two categories with different numbers of bacilli and immune response: *paucibacillary* (PB) and *multibacillary* (MB) leprosy [2]. Patients with PB tend to generate more immune response, less bacterial load, and fewer skin lesions. Patients with MB, on the other hand, do not exhibit a strong immune response, may have high levels of bacteria, and may have many skin lesions [7]. This classification plays important roles in disease management because the infectious period is much longer for MB cases than for PB cases, and the time of MDT is much longer for the former than for the latter [1]. It is crucial to recognize differences in the maintenance of transmission of these two morphs when developing effective public health measures. In order to analyze the two forms (PB and MB) mathematically, it is important to make a clear distinction between the two.

Mathematical modeling is a highly useful tool that can be used to better understand the epidemiology of a disease and to guide public health policy [8, 9]. Several simple ordinary differential equation (ODE) models have been developed that have investigated the spread of leprosy, estimate interventions, and forecast future trends [10–13]. Recently, Kubra et al. [14] proposed a six-compartment fractional ODE model for the study of dynamics of leprosy comprising Susceptible (S), Asymptomatic (A), Paucibacillary (P), Multibacillary (M), Treatment (T), and Recovered (R). Although these ODE-based models give important insights into the temporal evolution of the disease, they assume that the disease is well-mixed and spatially uniform. In practice, geographical barriers, population density, human mobility, and access to health care lead to a disease spread that is not uniform [15–17]. Spatial movement is an important determinant of leprosy transmission, as evidenced by the geographical clustering of leprosy cases, [18, 19]. Reaction–diffusion partial differential equations (PDEs) have been effectively used for modeling different infectious diseases [20–24] to account for spatial spread. So far, there is no report of the spatiotemporal dynamics of the leprosy transmission via a two-dimensional reaction–diffusion PDE model.

It is worth noting that deterministic ODE models, although widely used, have inherent limitations. Ali et al. [25] demonstrated that deterministic epidemic models can systematically overestimate the basic reproduction number of observed outbreaks, due to the implicit conditioning on major outbreak trajectories. Complementary stochastic approaches, such as the particle-filtering extended susceptible–exposed–infectious–recovered (SEIRS) model with repeated vaccination proposed by Papageorgiou and Tsaklidis [26], have shown the value of incorporating stochasticity and time-varying parameters for capturing real-world epidemic dynamics. Although the present study adopts a deterministic framework, these findings underscore the importance of interpreting our computed R_L as a theoretical threshold rather than a precise epidemiological prediction.

To address this gap, the present study extends the ODE-based leprosy model proposed by Kubra et

al. [14] into a two-dimensional reaction–diffusion PDE framework. By incorporating Fickian diffusion into each of the six compartments, the model captures both the temporal evolution and the spatial propagation of the disease within a bounded geographical domain. To solve this complex nonlinear system, we develop and rigorously analyze two numerical schemes: the conditionally stable forward Euler finite difference method and the Crank–Nicolson operator-splitting method. For each scheme, we derive stability conditions and analyze positivity preservation. Extensive numerical simulations are then performed using MATLAB to illustrate the spatiotemporal evolution of the disease and to investigate the stability of the endemic equilibrium under realistic parameter scenarios.

The remainder of this paper is organized as follows. Section 2 presents the mathematical formulation of the two-dimensional reaction–diffusion leprosy model. Sections 3 and 4 derive the basic reproduction number and the equilibrium points. Section 5 analyzes the stability of the endemic equilibrium for the reaction–diffusion system. Section 6 develops the numerical schemes and their stability analysis. Section 7 presents the numerical simulation results and discusses their epidemiological implications. Finally, Section 8 provides conclusions and directions for future research.

2. Model formulation

In this section, we formulate a two-dimensional reaction–diffusion mathematical model to investigate the spatial transmission and clinical management of leprosy. We first describe the compartmental ODE structure [14], detailing the biological interpretations of each epidemiological class, and subsequently extend the system to incorporate spatial Fickian diffusion on a bounded domain.

The total human population at any time t is categorized into six mutually exclusive epidemiological compartments representing successive stages in the natural history and treatment of leprosy. The flow of individuals between compartments is depicted schematically in Figure 1.

- **Susceptible individuals ($S(t)$):** Healthy individuals vulnerable to infection upon contact with infectious paucibacillary or multibacillary cases. New individuals enter this class via recruitment at constant rate π and exit through natural mortality at rate μ or infection.
- **Asymptomatic individuals ($A(t)$):** Latently infected individuals carrying *M. leprae* who do not yet manifest clinical signs. Upon completing the incubation period, asymptomatic individuals progress to clinical disease at rate ξ , branching into paucibacillary form with fraction $1 - \zeta$ or multibacillary form with fraction ζ [7].
- **Paucibacillary cases ($P(t)$):** Patients with mild, paucibacillary leprosy characterized by fewer than five lesions and a lower bacterial burden due to partial cell-mediated immunity [2]. Individuals in this class can recover spontaneously at rate pr_1 or initiate treatment at rate $(1 - p)r_1$.
- **Multibacillary cases ($M(t)$):** Patients with severe, multibacillary leprosy who exhibit weak cell-mediated immunity, high bacterial loads, and elevated infectivity. MB patients transition to treatment at rate m or experience disease-induced mortality at rate δ .
- **Treated individuals ($T(t)$):** Patients receiving multi-drug therapy (MDT), the standard therapeutic protocol for leprosy. This compartment receives treated PB and MB cases, with patients recovering at rate r_2 or dying from the disease at rate δ .

- **Recovered individuals ($R(t)$):** Individuals who achieve clinical or bacteriological cure (either through spontaneous recovery from PB or completion of MDT) and remain subject to natural mortality at rate μ .

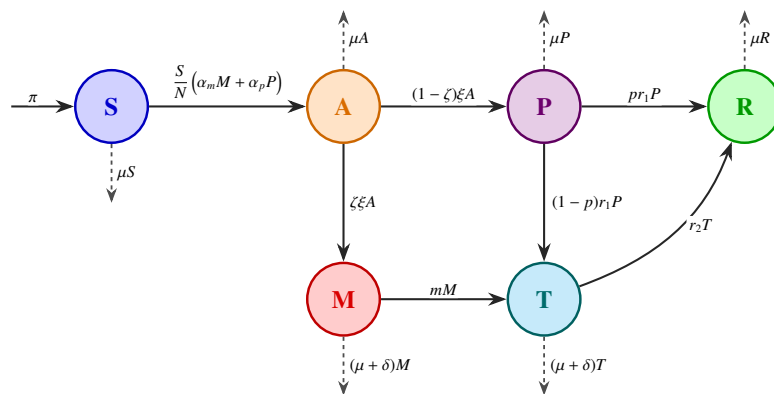


Figure 1. Comparison of different transmission phases in the leprosy disease model. Solid bold arrows denote progression and treatment transitions, and dashed bold arrows represent natural or disease-induced removal.

The total population at any time t is the sum of all compartments:

$$N(t) = S(t) + A(t) + P(t) + M(t) + T(t) + R(t). \quad (2.1)$$

2.1. The temporal ODE model

Following the framework proposed by Kubra et al. [14], the time-dependent dynamics of leprosy transmission and treatment are governed by the following system of nonlinear ODEs:

$$\left. \begin{aligned} \frac{dS}{dt} &= \pi - \frac{\alpha_m S(t)M(t)}{N(t)} - \frac{\alpha_p S(t)P(t)}{N(t)} - \mu S(t), \\ \frac{dA}{dt} &= \frac{1}{N(t)} (\alpha_m S(t)M(t) + \alpha_p S(t)P(t)) - [\xi + \mu]A(t), \\ \frac{dP}{dt} &= (1 - \zeta)\xi A(t) - [r_1 + \mu]P(t), \\ \frac{dM}{dt} &= \zeta\xi A(t) - [m + \delta + \mu]M(t), \\ \frac{dT}{dt} &= mM(t) + (1 - p)r_1 P(t) - [r_2 + \delta + \mu]T(t), \\ \frac{dR}{dt} &= r_1 p P(t) + r_2 T(t) - \mu R(t). \end{aligned} \right\} \quad (2.2)$$

Here, π represents the constant recruitment of new susceptible individuals. The terms $\alpha_m S M/N$ and $\alpha_p S P/N$ denote the force of infection driven by contact with multibacillary and paucibacillary patients, respectively, following standard incidence principles [8]. The parameter μ represents the

natural mortality rate, which is assumed to be identical for all compartments. Newly infected individuals enter the asymptomatic phase and subsequently progress to clinical disease at total rate ξ . A fraction $1 - \zeta$ of progressing individuals develops paucibacillary leprosy (at per-capita rate $(1 - \zeta)\xi$), and the remaining fraction ζ develops multibacillary leprosy (at per-capita rate $\zeta\xi$). Consequently, the total per-capita removal rate from A due to progression and natural mortality is $\xi + \mu$. The subsequent equations describe the transition of individuals through the PB, MB, treatment, and recovered phases. A complete list of biological parameters and their interpretations is provided in Table 1. To maintain a biologically valid population structure, all parameters are strictly non-negative, with the recruitment rate π and natural death rate μ strictly positive.

Table 1. Description and biological interpretation of model parameters.

Symbol	Biological description	Unit
π	Recruitment rate into the susceptible class	persons/time
α_m	Effective contact rate (transmission) from MB individuals	1/time
α_p	Effective contact rate (transmission) from PB individuals	1/time
μ	Natural mortality rate (all compartments)	1/time
ζ	Probability of progressing to MB form ($0 \leq \zeta \leq 1$)	dimensionless
ξ	Rate of progression from asymptomatic to clinical disease	1/time
r_1	Treatment/recovery rate for paucibacillary patients	1/time
r_2	Recovery rate from the treatment class	1/time
m	Rate at which MB patients initiate treatment (MDT)	1/time
δ	Disease-induced mortality rate	1/time
p	Probability of direct recovery for PB patients ($0 \leq p \leq 1$)	dimensionless
$d_S, d_A, \dots, d_R$	Diffusion coefficients for each compartment	length ² /time

2.2. Extension to a two-dimensional reaction–diffusion system

The ODE model (2.2) effectively captures the temporal evolution of leprosy under the assumption that the population is uniformly mixed across space. To relax this limitation and study the geographic spread of the disease, we extend the model to incorporate spatial coordinates (x, y) within a bounded rectangular domain $\Omega = [0, L_x] \times [0, L_y] \subset \mathbb{R}^2$.

The physical movement of individuals in each subpopulation is modeled using Fickian diffusion. For each compartment $u = u(x, y, t)$ with diffusion coefficient $d_u > 0$, the diffusion contribution is given by:

$$d_u \nabla^2 u = d_u \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right), \quad (2.3)$$

where ∇^2 denotes the two-dimensional Laplacian operator [15, 16]. Here, u represents the population density of a generic compartment at spatial location (x, y) and time t . The diffusion coefficient $d_u > 0$ controls the rate at which individuals in compartment u move through space. These coefficients can vary between compartments to reflect real-world behaviors; for instance, healthy or recovered individuals generally exhibit higher mobility than severely ill patients [17].

Incorporating spatial diffusion, the extended two-dimensional reaction–diffusion PDE system for leprosy transmission is formulated as:

$$\left\{ \begin{array}{l} \frac{\partial S}{\partial t} = d_S \nabla^2 S + \pi - \frac{\alpha_m S M}{N} - \frac{\alpha_p S P}{N} - \mu S, \\ \frac{\partial A}{\partial t} = d_A \nabla^2 A + \frac{1}{N} (\alpha_m S M + \alpha_p S P) - [\xi + \mu] A, \\ \frac{\partial P}{\partial t} = d_P \nabla^2 P + (1 - \zeta) \xi A - [r_1 + \mu] P, \\ \frac{\partial M}{\partial t} = d_M \nabla^2 M + \zeta \xi A - [m + \delta + \mu] M, \\ \frac{\partial T}{\partial t} = d_T \nabla^2 T + m M + (1 - p) r_1 P - [r_2 + \delta + \mu] T, \\ \frac{\partial R}{\partial t} = d_R \nabla^2 R + r_1 p P + r_2 T - \mu R, \end{array} \right. \quad (2.4)$$

for $(x, y) \in \Omega$ and $t > 0$. Here, $N = N(x, y, t) = S + A + P + M + T + R$ is the total local population density, and d_S, d_A, d_P, d_M, d_T , and d_R are the positive diffusion coefficients for each respective compartment. Each PDE balances spatial dispersal (the $d_u \nabla^2 u$ term) with local epidemiological interactions (the reaction terms). The coupling between compartments arises through the nonlinear infection terms involving S , P , M , and N . The rectangular domain $\Omega = [0, L_x] \times [0, L_y]$ and isotropic Fickian diffusion are standard continuum idealizations in mathematical epidemiology [15, 16], providing a mathematically tractable framework that captures the macroscopic spatial smoothing and dispersal of localized infection hotspots.

Remark 1. If all diffusion coefficients are set to zero ($d_S = d_A = \dots = d_R = 0$), the PDE system (2.4) perfectly reduces to the spatially homogeneous ODE model (2.2) [14].

To solve System (2.4), we must define the initial spatial distribution of the population at time $t = 0$:

$$\begin{aligned} S(x, y, 0) = S_0(x, y) \geq 0, \quad A(x, y, 0) = A_0(x, y) \geq 0, \\ P(x, y, 0) = P_0(x, y) \geq 0, \quad M(x, y, 0) = M_0(x, y) \geq 0, \\ T(x, y, 0) = T_0(x, y) \geq 0, \quad R(x, y, 0) = R_0(x, y) \geq 0, \end{aligned} \quad (2.5)$$

where S_0, A_0, P_0, M_0, T_0 , and R_0 are non-negative, bounded, and smooth functions over Ω . These initial conditions represent the real-world geographic distribution of the population at the start of the simulation [18, 19]. In our numerical experiments (Section 7), we use heterogeneous initial conditions of the form $u_0(x, y) = c_u(1 + a \sin(\omega_1 x))(1 + b \cos(\omega_2 y))$ to simulate nonuniform population clusters.

Finally, we enforce homogeneous Neumann (zero-flux) boundary conditions along the domain boundary $\partial\Omega$:

$$\frac{\partial S}{\partial \mathbf{n}} \Big|_{\partial\Omega} = \frac{\partial A}{\partial \mathbf{n}} \Big|_{\partial\Omega} = \frac{\partial P}{\partial \mathbf{n}} \Big|_{\partial\Omega} = \frac{\partial M}{\partial \mathbf{n}} \Big|_{\partial\Omega} = \frac{\partial T}{\partial \mathbf{n}} \Big|_{\partial\Omega} = \frac{\partial R}{\partial \mathbf{n}} \Big|_{\partial\Omega} = 0, \quad (2.6)$$

where $\mathbf{n}$ is the outward-pointing normal vector. The zero-flux condition implies that individuals do not migrate across the domain borders, reflecting a biologically closed population. This is a standard and well-justified assumption when modeling the internal dynamics of an isolated epidemic region [15, 16, 24].

3. The basic reproduction number

The basic reproduction number, denoted R_L (or traditionally R_0), is a fundamental threshold parameter in mathematical epidemiology. It represents the expected number of secondary infections produced by a single infected individual introduced into a completely susceptible population. If $R_L < 1$, the disease-free equilibrium (DFE) is locally asymptotically stable, and the disease cannot invade the population. If $R_L > 1$, the disease can spread, and an endemic equilibrium becomes possible. New infections only occur when susceptible individuals become asymptotically infected through contact with paucibacillary or multibacillary cases. This is captured in the first term of Eq (2.4):

$$\mathcal{F} = \begin{pmatrix} \frac{1}{N}(a_m S M + a_p S P) \\ 0 \\ 0 \\ 0 \end{pmatrix}. \quad (3.1)$$

All remaining terms describe the movement of individuals between infected compartments and removal from the infected system:

$$\mathcal{V} = \begin{pmatrix} (\xi + \mu)A \\ -(1 - \zeta)\xi A + (r_1 + \mu)P \\ -\zeta\xi A + (m + \delta + \mu)M \\ -mM - (1 - p)r_1 P + (r_2 + \delta + \mu)T \end{pmatrix}. \quad (3.2)$$

We compute the Jacobian of $\mathcal{F}$ and $\mathcal{V}$ with respect to the infected variables (A, P, M, T) and evaluate at the DFE. Thus,

$$\mathbb{F} = \begin{pmatrix} 0 & a_p & a_m & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad (3.3)$$

and

$$\mathbb{V} = \begin{pmatrix} \xi + \mu & 0 & 0 & 0 \\ -(1 - \zeta)\xi & r_1 + \mu & 0 & 0 \\ -\zeta\xi & 0 & m + \delta + \mu & 0 \\ 0 & -(1 - p)r_1 & -m & r_2 + \delta + \mu \end{pmatrix}. \quad (3.4)$$

As $\mathbb{V}$ is block lower triangular, we compute its inverse column by column. Thus,

$$\mathbb{V}^{-1} = \begin{pmatrix} \frac{1}{\xi + \mu} & 0 & 0 & 0 \\ \frac{(1 - \zeta)\xi}{(r_1 + \mu)(\xi + \mu)} & \frac{1}{r_1 + \mu} & 0 & 0 \\ \frac{\zeta\xi}{(m + \delta + \mu)(\xi + \mu)} & 0 & \frac{1}{m + \delta + \mu} & 0 \\ v^{41} & \frac{(1 - p)r_1}{(r_1 + \mu)(r_2 + \delta + \mu)} & \frac{m}{(m + \delta + \mu)(r_2 + \delta + \mu)} & \frac{1}{r_2 + \delta + \mu} \end{pmatrix}, \quad (3.5)$$

where

$$v^{41} = \frac{\xi}{(r_2 + \delta + \mu)(\xi + \mu)} \left[\frac{(1 - p)r_1(1 - \zeta)}{r_1 + \mu} + \frac{m\zeta}{m + \delta + \mu} \right]. \quad (3.6)$$

Because only the first row of $\mathbb{F}$ is nonzero, we only need the first row of the product:

$$\mathbb{F}\mathbb{V}^{-1} = \begin{pmatrix} 0 & a_p & a_m & 0 \end{pmatrix} \cdot \mathbb{V}^{-1}. \quad (3.7)$$

Therefore,

$$\mathbb{F}\mathbb{V}^{-1} = \begin{pmatrix} \frac{\xi}{\xi + \mu} \left[\frac{a_p(1 - \zeta)}{r_1 + \mu} + \frac{a_m\zeta}{m + \delta + \mu} \right] & \frac{a_p}{r_1 + \mu} & \frac{a_m}{m + \delta + \mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \quad (3.8)$$

The matrix $\mathbb{F}\mathbb{V}^{-1}$ has three zero rows, so its only nonzero eigenvalue is the $(1, 1)$ entry. Therefore,

$$R_L = \rho(\mathbb{F}\mathbb{V}^{-1}) = \frac{\xi}{\xi + \mu} \left[\frac{a_p(1 - \zeta)}{r_1 + \mu} + \frac{a_m\zeta}{m + \delta + \mu} \right]. \quad (3.9)$$

4. Equilibrium points

To determine the equilibrium points of the system, we set all time derivatives equal to zero.

4.1. Disease-free equilibrium (DFE)

The disease-free equilibrium (DFE) is obtained by setting all infected compartments to zero:

$$\Theta = (S_0, A_0, P_0, M_0, T_0, R_0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0 \right).$$

At the DFE, the total population is $N_0 = S_0 = \pi/\mu$, so $S_0/N_0 = 1$.

4.2. Endemic equilibrium points

The complete endemic equilibrium point $\Theta^* = (S^*, A^*, P^*, M^*, T^*, R^*)$ is:

$$\begin{cases} S^* = \frac{(\xi + \mu)(m + \delta + \mu)(r_1 + \mu)}{\xi[\alpha_m \xi(r_1 + \mu) + \alpha_p(1 - \zeta)(m + \delta + \mu)]} \cdot N^*, \\ A^* = \frac{\pi - \mu S^*}{\xi + \mu}, \quad P^* = \frac{(1 - \zeta)\xi}{r_1 + \mu} A^*, \quad M^* = \frac{\zeta\xi}{m + \delta + \mu} A^*, \\ T^* = \frac{1}{r_2 + \delta + \mu} \left[\frac{m\zeta\xi}{m + \delta + \mu} + \frac{(1 - p)r_1(1 - \zeta)\xi}{r_1 + \mu} \right] A^*, \\ R^* = \frac{1}{\mu} \left[\frac{r_1 p(1 - \zeta)\xi}{r_1 + \mu} + \frac{r_2}{r_2 + \delta + \mu} \left(\frac{m\zeta\xi}{m + \delta + \mu} + \frac{(1 - p)r_1(1 - \zeta)\xi}{r_1 + \mu} \right) \right] A^*. \end{cases} \quad (4.1)$$

5. Stability analysis of the endemic equilibrium point

In this section, we analyze the local asymptotic stability of the endemic steady-state equilibrium $\Theta^* = (S^*, A^*, P^*, M^*, T^*, R^*)$ for the two-dimensional reaction–diffusion leprosy model (2.4). When the basic reproduction number satisfies $R_L > 1$, the disease-free equilibrium Θ is unstable, and the unique endemic equilibrium Θ^* exists with all positive coordinates. We present both the analytical stability conditions valid across spatial Fourier modes and the parameter-specific verification confirming the local asymptotic stability of Θ^* .

Theorem 1. Suppose $R_L > 1$, ensuring the existence of the unique endemic equilibrium Θ^* . For the baseline parameter regime (Table 6), the endemic equilibrium Θ^* is locally asymptotically stable under the reaction–diffusion system (2.4)–(2.6) for all admissible spatial Fourier modes $k = (k_1, k_2)$ with $k^2 = k_1^2 + k_2^2 \geq 0$, as the Routh–Hurwitz criteria (5.9)–(5.13) are strictly satisfied at $k = 0$ ($\max_j \operatorname{Re}(\lambda_j) < 0$), and the positive diffusion terms ($-d_u k^2 \leq 0$) shift all characteristic roots further into the open left half-plane for all $k > 0$.

Proof. We linearize the system around Θ^* by introducing small spatiotemporal perturbations:

$$\begin{aligned} S(x, y, t) &= S^* + \bar{S}(x, y, t), & A(x, y, t) &= A^* + \bar{A}(x, y, t), & P(x, y, t) &= P^* + \bar{P}(x, y, t), \\ M(x, y, t) &= M^* + \bar{M}(x, y, t), & T(x, y, t) &= T^* + \bar{T}(x, y, t), & R(x, y, t) &= R^* + \bar{R}(x, y, t), \end{aligned} \quad (5.1)$$

with $|\bar{u}| \ll 1$ for each compartment. Substituting these into (2.4), expanding the nonlinear terms, and retaining only first-order terms in the perturbations yields the linearized system:

$$\begin{cases} \frac{\partial \bar{S}}{\partial t} = d_S \nabla^2 \bar{S} + J_{11} \bar{S} + J_{12} \bar{A} + J_{13} \bar{P} + J_{14} \bar{M} + J_{15} \bar{T} + J_{16} \bar{R}, \\ \frac{\partial \bar{A}}{\partial t} = d_A \nabla^2 \bar{A} + J_{21} \bar{S} + J_{22} \bar{A} + J_{23} \bar{P} + J_{24} \bar{M} + J_{25} \bar{T} + J_{26} \bar{R}, \\ \frac{\partial \bar{P}}{\partial t} = d_P \nabla^2 \bar{P} + J_{32} \bar{A} + J_{33} \bar{P}, \\ \frac{\partial \bar{M}}{\partial t} = d_M \nabla^2 \bar{M} + J_{42} \bar{A} + J_{44} \bar{M}, \\ \frac{\partial \bar{T}}{\partial t} = d_T \nabla^2 \bar{T} + J_{53} \bar{P} + J_{54} \bar{M} + J_{55} \bar{T}, \\ \frac{\partial \bar{R}}{\partial t} = d_R \nabla^2 \bar{R} + J_{63} \bar{P} + J_{65} \bar{T} + J_{66} \bar{R}, \end{cases} \quad (5.2)$$

where the Jacobian entries $J_{ij} = \left. \frac{\partial f_i}{\partial u_j} \right|_{\Theta^*}$ are evaluated at the endemic equilibrium. Define the total population at equilibrium as $N^* = S^* + A^* + P^* + M^* + T^* + R^*$. The explicit nonzero entries are:

$$\begin{aligned}
 J_{11} &= -\frac{\alpha_m M^* + \alpha_p P^*}{N^*} + \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2} - \mu, & J_{21} &= \frac{\alpha_m M^* + \alpha_p P^*}{N^*} - \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, \\
 J_{12} &= \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, & J_{22} &= -\frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2} - (\xi + \mu), \\
 J_{13} &= -\frac{\alpha_p S^*}{N^*} + \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, & J_{23} &= \frac{\alpha_p S^*}{N^*} - \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, \\
 J_{14} &= -\frac{\alpha_m S^*}{N^*} + \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, & J_{24} &= \frac{\alpha_m S^*}{N^*} - \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, \\
 J_{15} &= J_{16} = \frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, & J_{25} &= J_{26} = -\frac{(\alpha_m M^* + \alpha_p P^*) S^*}{(N^*)^2}, \\
 J_{32} &= (1 - \zeta)\xi, & J_{33} &= -(r_1 + \mu), \\
 J_{53} &= (1 - p)r_1, & J_{54} &= m, \\
 J_{63} &= r_1 p, & J_{65} &= r_2, \\
 & & J_{42} &= \zeta\xi, & J_{44} &= -(m + \delta + \mu), \\
 & & J_{55} &= -(r_2 + \delta + \mu), \\
 & & J_{66} &= -\mu.
 \end{aligned} \tag{5.3}$$

All other $J_{ij} = 0$.

According to the standard methodology for reaction–diffusion stability analysis, we assume that the solutions for the perturbed system components $\bar{S}(x, y, t)$, $\bar{A}(x, y, t)$, $\bar{P}(x, y, t)$, $\bar{M}(x, y, t)$, $\bar{T}(x, y, t)$, and $\bar{R}(x, y, t)$ may be represented as a Fourier series expansion. Because of the homogeneous Neumann boundary conditions (2.6), the appropriate eigenfunction expansion on the rectangular domain $\Omega = [0, L_x] \times [0, L_y]$ uses cosine modes indexed by non-negative integers n_1 and n_2 , with corresponding wave numbers $k_1 = n_1\pi/L_x$ and $k_2 = n_2\pi/L_y$. The perturbations are expanded as:

$$\begin{cases} \bar{S}(x, y, t) = \sum_{n_1} \sum_{n_2} S_{n_1 n_2} e^{\lambda t} \cos(k_1 x) \cos(k_2 y), & \bar{A}(x, y, t) = \sum_{n_1} \sum_{n_2} A_{n_1 n_2} e^{\lambda t} \cos(k_1 x) \cos(k_2 y), \\ \bar{P}(x, y, t) = \sum_{n_1} \sum_{n_2} P_{n_1 n_2} e^{\lambda t} \cos(k_1 x) \cos(k_2 y), & \bar{M}(x, y, t) = \sum_{n_1} \sum_{n_2} M_{n_1 n_2} e^{\lambda t} \cos(k_1 x) \cos(k_2 y), \\ \bar{T}(x, y, t) = \sum_{n_1} \sum_{n_2} T_{n_1 n_2} e^{\lambda t} \cos(k_1 x) \cos(k_2 y), & \bar{R}(x, y, t) = \sum_{n_1} \sum_{n_2} R_{n_1 n_2} e^{\lambda t} \cos(k_1 x) \cos(k_2 y). \end{cases} \tag{5.4}$$

The Laplacian of each cosine eigenfunction satisfies $\nabla^2[\cos(k_1 x) \cos(k_2 y)] = -(k_1^2 + k_2^2) \cos(k_1 x) \cos(k_2 y)$, so substituting (5.4) into the linearized system (5.2) decouples the modes:

$$\left\{ \begin{aligned}
& \sum_{n_1} \sum_{n_2} (J_{11} - d_S k_1^2 - d_S k_2^2 - \lambda) S_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{12} A_{n_1 n_2} \\
& + \sum_{n_1} \sum_{n_2} J_{13} P_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{14} M_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{15} T_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{16} R_{n_1 n_2} = 0 \\
& \sum_{n_1} \sum_{n_2} J_{21} S_{n_1 n_2} + \sum_{n_1} \sum_{n_2} (J_{22} - d_A k_1^2 - d_A k_2^2 - \lambda) A_{n_1 n_2} \\
& + \sum_{n_1} \sum_{n_2} J_{23} P_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{24} M_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{25} T_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{26} R_{n_1 n_2} = 0 \\
& \sum_{n_1} \sum_{n_2} J_{32} A_{n_1 n_2} + \sum_{n_1} \sum_{n_2} (J_{33} - d_P k_1^2 - d_P k_2^2 - \lambda) P_{n_1 n_2} = 0 \\
& \sum_{n_1} \sum_{n_2} J_{42} A_{n_1 n_2} + \sum_{n_1} \sum_{n_2} (J_{44} - d_M k_1^2 - d_M k_2^2 - \lambda) M_{n_1 n_2} = 0 \\
& \sum_{n_1} \sum_{n_2} J_{53} P_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{54} M_{n_1 n_2} \\
& + \sum_{n_1} \sum_{n_2} (J_{55} - d_T k_1^2 - d_T k_2^2 - \lambda) T_{n_1 n_2} = 0 \\
& \sum_{n_1} \sum_{n_2} J_{63} P_{n_1 n_2} + \sum_{n_1} \sum_{n_2} J_{65} T_{n_1 n_2} \\
& + \sum_{n_1} \sum_{n_2} (J_{66} - d_R k_1^2 - d_R k_2^2 - \lambda) R_{n_1 n_2} = 0.
\end{aligned} \right. \quad (5.5)$$

For (5.5), the variational matrix V is:

$$V = \begin{pmatrix} J_{11} - d_S(k_1^2 + k_2^2) - \lambda & J_{12} & J_{13} & J_{14} & J_{15} & J_{16} \\ J_{21} & J_{22} - d_A(k_1^2 + k_2^2) - \lambda & J_{23} & J_{24} & J_{25} & J_{26} \\ 0 & J_{32} & J_{33} - d_P(k_1^2 + k_2^2) - \lambda & 0 & 0 & 0 \\ 0 & J_{42} & 0 & J_{44} - d_M(k_1^2 + k_2^2) - \lambda & 0 & 0 \\ 0 & 0 & J_{53} & J_{54} & J_{55} - d_T(k_1^2 + k_2^2) - \lambda & 0 \\ 0 & 0 & J_{63} & 0 & J_{65} & J_{66} - d_R(k_1^2 + k_2^2) - \lambda \end{pmatrix}. \quad (5.6)$$

Define $k^2 = k_1^2 + k_2^2$. The above matrix is of block structure, and the value for

$$\lambda_6 = J_{66} - d_R k^2 = -\mu - d_R k^2,$$

may be factored out directly. After that, the matrix takes on the following form:

$$V_1 = \begin{pmatrix} J_{11} - d_S k^2 - \lambda & J_{12} & J_{13} & J_{14} & J_{15} \\ J_{21} & J_{22} - d_A k^2 - \lambda & J_{23} & J_{24} & J_{25} \\ 0 & J_{32} & J_{33} - d_P k^2 - \lambda & 0 & 0 \\ 0 & J_{42} & 0 & J_{44} - d_M k^2 - \lambda & 0 \\ 0 & 0 & J_{53} & J_{54} & J_{55} - d_T k^2 - \lambda \end{pmatrix}. \quad (5.7)$$

The following is the characteristic equation for matrix V_1 :

$$\lambda^5 + p_1 \lambda^4 + p_2 \lambda^3 + p_3 \lambda^2 + p_4 \lambda + p_5 = 0. \quad (5.8)$$

Where p_1, p_2, p_3, p_4 , and p_5 are functions of the Jacobian entries and the diffusion-weighted wave number k^2 , calculated from the determinant expansion of V_1 . The Routh–Hurwitz criterion for stability is given as:

$$C_1 : p_1 > 0, \quad (5.9)$$

$$C_2 : p_5 > 0, \quad (5.10)$$

$$C_3 : p_1 p_2 - p_3 > 0, \quad (5.11)$$

$$C_4 : p_1 p_2 p_3 + p_1 p_5 - [p_3^2 + p_1^2 p_4] > 0, \quad (5.12)$$

$$C_5 : [p_1 p_4 - p_5] [p_1 p_2 p_3 - p_3^2 - p_1^2 p_4] + p_1^2 p_4 p_5 - [p_5 (p_1 p_2 - p_3)^2 + p_1 p_5^2] > 0. \quad (5.13)$$

Because numerically $R_L = 1.558238 > 1$, the disease cannot be eliminated under the current parameter regime. Each infected individual, on average, transmits the disease to approximately 1.56 new individuals, indicating sustained endemic transmission. The endemic equilibrium (EE), denoted $\Theta^* = (S^*, A^*, P^*, M^*, T^*, R^*)$, represents the long-term steady state where the disease coexists with the population. This equilibrium exists uniquely when $R_L > 1$:

$$\begin{aligned} S^* &= 0.581326, & A^* &= 0.014437, & P^* &= 0.054572, \\ M^* &= 0.021335, & T^* &= 0.016328, & R^* &= 0.217847, & N^* &= 0.905844. \end{aligned}$$

The linearization of the reaction–diffusion system around the endemic equilibrium Θ^* yields the Jacobian matrix:

$$J(\Theta^*) = \begin{bmatrix} -0.025160 & 0.009244 & -0.119106 & -0.054931 & 0.009244 & 0.009244 \\ 0.005160 & -0.589244 & 0.119106 & 0.054931 & -0.009244 & -0.009244 \\ 0 & 0.453600 & -0.120000 & 0 & 0 & 0 \\ 0 & 0.106400 & 0 & -0.072000 & 0 & 0 \\ 0 & 0 & 0.080000 & 0.002000 & -0.270000 & 0 \\ 0 & 0 & 0.020000 & 0 & 0.200000 & -0.020000 \end{bmatrix}.$$

The spatially homogeneous case ($k = 0$) corresponds to the classical ODE system without diffusion. The eigenvalues of the Jacobian matrix at the endemic equilibrium are:

$$\begin{aligned} \lambda_1 &= -0.69470336, \\ \lambda_2 &= -0.26936837, \\ \lambda_3 &= -0.07811618, \\ \lambda_4 &= -0.02000000, \\ \lambda_5 &= -0.01710809 + 0.02912258i, \\ \lambda_6 &= -0.01710809 - 0.02912258i, \end{aligned}$$

with maximum real part $\max_j \operatorname{Re}(\lambda_j) = -0.01710809 < 0$. Because all eigenvalues have strictly negative real parts, the endemic equilibrium is locally asymptotically stable for the ODE system. For the reaction-diffusion PDE system, the stability must be verified for all spatial wave numbers $k^2 =$

$k_1^2 + k_2^2$, where $k_1 = n_1\pi/L_x$ and $k_2 = n_2\pi/L_y$ are determined by the domain geometry and boundary conditions.

Accordingly, when $R_L > 1$, all classes tend to their endemic values. The system (2.4) is therefore stable at the endemic equilibrium point Θ^* for $R_L > 1$, as presented in Table 2.

Table 2. Complete Routh–Hurwitz stability analysis for the leprosy reaction–diffusion model at the endemic equilibrium Θ^* .

k^2	Characteristic polynomial coefficients					$\max \Re(\lambda)$
	p_1	p_2	p_3	p_4	p_5	
0.0	1.0764e + 00	2.9924e – 01	2.4703e – 02	7.0340e – 04	1.0169e – 05	–1.7791e – 02
0.5	1.0766e + 00	2.9937e – 01	2.4731e – 02	7.0494e – 04	1.0192e – 05	–1.7820e – 02
1.0	1.0767e + 00	2.9950e – 01	2.4759e – 02	7.0647e – 04	1.0216e – 05	–1.7849e – 02
2.0	1.0770e + 00	2.9977e – 01	2.4815e – 02	7.0955e – 04	1.0263e – 05	–1.7907e – 02
3.0	1.0773e + 00	3.0003e – 01	2.4872e – 02	7.1263e – 04	1.0311e – 05	–1.7964e – 02
4.0	1.0776e + 00	3.0030e – 01	2.4928e – 02	7.1572e – 04	1.0358e – 05	–1.8022e – 02
5.0	1.0779e + 00	3.0056e – 01	2.4985e – 02	7.1881e – 04	1.0406e – 05	–1.8080e – 02
7.5	1.0787e + 00	3.0122e – 01	2.5126e – 02	7.2658e – 04	1.0527e – 05	–1.8225e – 02
10.0	1.0794e + 00	3.0188e – 01	2.5268e – 02	7.3439e – 04	1.0648e – 05	–1.8369e – 02
15.0	1.0809e + 00	3.0320e – 01	2.5553e – 02	7.5014e – 04	1.0895e – 05	–1.8658e – 02
20.0	1.0824e + 00	3.0453e – 01	2.5838e – 02	7.6606e – 04	1.1147e – 05	–1.8947e – 02
30.0	1.0854e + 00	3.0718e – 01	2.6413e – 02	7.9843e – 04	1.1664e – 05	–1.9525e – 02
50.0	1.0914e + 00	3.1251e – 01	2.7577e – 02	8.6527e – 04	1.2757e – 05	–2.0682e – 02
75.0	1.0989e + 00	3.1921e – 01	2.9059e – 02	9.5284e – 04	1.4236e – 05	–2.2127e – 02
100.0	1.1064e + 00	3.2595e – 01	3.0571e – 02	1.0449e – 03	1.5849e – 05	–2.3573e – 02
Routh–Hurwitz condition values						
k^2	$C_1 = p_1$	$C_2 = p_5$	$C_3 = p_1p_2 - p_3$	C_4	C_5	Status
0.0	1.0764e + 00	1.0169e – 05	2.9740e – 01	6.5426e – 03	3.9878e – 06	Stable
0.5	1.0766e + 00	1.0192e – 05	2.9756e – 01	6.5529e – 03	4.0038e – 06	Stable
1.0	1.0767e + 00	1.0216e – 05	2.9772e – 01	6.5632e – 03	4.0199e – 06	Stable
2.0	1.0770e + 00	1.0263e – 05	2.9804e – 01	6.5839e – 03	4.0521e – 06	Stable
3.0	1.0773e + 00	1.0311e – 05	2.9835e – 01	6.6047e – 03	4.0846e – 06	Stable
4.0	1.0776e + 00	1.0358e – 05	2.9867e – 01	6.6254e – 03	4.1173e – 06	Stable
5.0	1.0779e + 00	1.0406e – 05	2.9899e – 01	6.6463e – 03	4.1502e – 06	Stable
7.5	1.0787e + 00	1.0527e – 05	2.9979e – 01	6.6985e – 03	4.2332e – 06	Stable
10.0	1.0794e + 00	1.0648e – 05	3.0058e – 01	6.7510e – 03	4.3176e – 06	Stable
15.0	1.0809e + 00	1.0895e – 05	3.0218e – 01	6.8568e – 03	4.4901e – 06	Stable
20.0	1.0824e + 00	1.1147e – 05	3.0378e – 01	6.9637e – 03	4.6680e – 06	Stable
30.0	1.0854e + 00	1.1664e – 05	3.0700e – 01	7.1809e – 03	5.0400e – 06	Stable
50.0	1.0914e + 00	1.2757e – 05	3.1350e – 01	7.6285e – 03	5.8530e – 06	Stable
75.0	1.0989e + 00	1.4236e – 05	3.2172e – 01	8.2138e – 03	7.0100e – 06	Stable
100.0	1.1064e + 00	1.5849e – 05	3.3007e – 01	8.8287e – 03	8.3405e – 06	Stable

□

Analytical versus parameter-specific nature of endemic stability. We emphasize that although the threshold condition $R_L > 1$ proves the mathematical existence, componentwise positivity, and persistence of the endemic equilibrium Θ^* (along with instability of the disease-free equilibrium Θ), $R_L > 1$ alone does not inherently guarantee local asymptotic stability of Θ^* for all conceivable parameter combinations in general six-dimensional epidemiological systems. The stability result stated in Theorem 1 is established as a parameter-specific analytical and numerical finding for the

biologically realistic baseline parameter regime in Table 6. The analytical structure of the diffusion operator guarantees that the verified stability at $k = 0$ robustly extends to all admissible spatial Fourier modes $k > 0$, ruling out pattern-forming Turing instabilities.

6. Numerical methods

The finite difference (FD) method discretizes the domain $[0, L]^2 \times [0, T_{\max}]$ into a grid of $M^2 \times N$ points. $h = \frac{L}{M}$ and $\tau = \frac{T_{\max}}{N}$ are the definitions of the spatial and temporal step sizes, respectively. The following are the grid points' specifications:

$$\begin{aligned} x_{k_1} &= k_1 h, & k_1 &= 1, 2, 3, \dots, M, \\ y_{k_2} &= k_2 h, & k_2 &= 1, 2, 3, \dots, M, \\ t_n &= n\tau, & n &= 1, 2, 3, \dots, N. \end{aligned} \quad (6.1)$$

The FD approximations for the variables S , A , P , M , T , and R at grid point (x_{k_1}, y_{k_2}) and time step t_n are denoted as S_{k_1, k_2}^n , A_{k_1, k_2}^n , P_{k_1, k_2}^n , M_{k_1, k_2}^n , T_{k_1, k_2}^n , and R_{k_1, k_2}^n , respectively. The total discrete population is defined as $N_{k_1, k_2}^n = S_{k_1, k_2}^n + A_{k_1, k_2}^n + P_{k_1, k_2}^n + M_{k_1, k_2}^n + T_{k_1, k_2}^n + R_{k_1, k_2}^n$.

6.1. Forward Euler finite difference method

This section presents the application of the forward Euler finite-difference method to the two-dimensional leprosy reaction–diffusion model. The time derivative is discretized via the forward difference formula, and the spatial derivatives are approximated using central difference formulas. Let the discrete Laplacian be $\nabla_d^2 u_{k_1, k_2}^n = u_{k_1-1, k_2}^n + u_{k_1+1, k_2}^n + u_{k_1, k_2-1}^n + u_{k_1, k_2+1}^n - 4u_{k_1, k_2}^n$. The resulting explicit scheme takes the form:

$$\begin{cases} S_{k_1, k_2}^{n+1} = S_{k_1, k_2}^n + \lambda_S \nabla_d^2 S_{k_1, k_2}^n + \tau\pi - \tau \frac{\alpha_m S_{k_1, k_2}^n M_{k_1, k_2}^n + \alpha_p S_{k_1, k_2}^n P_{k_1, k_2}^n}{N_{k_1, k_2}^n} - \tau\mu S_{k_1, k_2}^n, \\ A_{k_1, k_2}^{n+1} = A_{k_1, k_2}^n + \lambda_A \nabla_d^2 A_{k_1, k_2}^n + \tau \frac{\alpha_m S_{k_1, k_2}^n M_{k_1, k_2}^n + \alpha_p S_{k_1, k_2}^n P_{k_1, k_2}^n}{N_{k_1, k_2}^n} - \tau(\xi + \mu) A_{k_1, k_2}^n, \\ P_{k_1, k_2}^{n+1} = P_{k_1, k_2}^n + \lambda_P \nabla_d^2 P_{k_1, k_2}^n + \tau(1 - \xi)\xi A_{k_1, k_2}^n - \tau(r_1 + \mu) P_{k_1, k_2}^n, \\ M_{k_1, k_2}^{n+1} = M_{k_1, k_2}^n + \lambda_M \nabla_d^2 M_{k_1, k_2}^n + \tau\xi\xi A_{k_1, k_2}^n - \tau(m + \delta + \mu) M_{k_1, k_2}^n, \\ T_{k_1, k_2}^{n+1} = T_{k_1, k_2}^n + \lambda_T \nabla_d^2 T_{k_1, k_2}^n + \tau(m M_{k_1, k_2}^n + (1 - p)r_1 P_{k_1, k_2}^n) - \tau(r_2 + \delta + \mu) T_{k_1, k_2}^n, \\ R_{k_1, k_2}^{n+1} = R_{k_1, k_2}^n + \lambda_R \nabla_d^2 R_{k_1, k_2}^n + \tau(r_1 p P_{k_1, k_2}^n + r_2 T_{k_1, k_2}^n) - \tau\mu R_{k_1, k_2}^n, \end{cases} \quad (6.2)$$

where $\lambda_X = \frac{d_X \tau}{h^2}$ for $X \in \{S, A, P, M, T, R\}$.

6.1.1. Stability and positivity preservation of the forward Euler scheme

To evaluate the numerical stability of the forward Euler finite-difference (FE-FD) scheme, we perform a von Neumann stability analysis on the linearized system around a steady state $\mathbf{u}^* = (S^*, A^*, P^*, M^*, T^*, R^*)$. Substituting the discrete Fourier ansatz

$$u_{k_1, k_2}^n = \Phi_u^n e^{i(\omega_1 x_{k_1} + \omega_2 y_{k_2})}, \quad \text{for each compartment } u \in \{S, A, P, M, T, R\}, \quad (6.3)$$

where ω_1, ω_2 are the spatial wave numbers, into the five-point discrete Laplacian yields:

$$\nabla_d^2 u_{k_1, k_2}^n = \left(e^{-i\omega_1 h} + e^{i\omega_1 h} + e^{-i\omega_2 h} + e^{i\omega_2 h} - 4 \right) u_{k_1, k_2}^n = -4 \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) u_{k_1, k_2}^n. \quad (6.4)$$

Substituting this relation into the discrete equations (6.2) yields the following compartmentwise amplification factors.

For the susceptible compartment S :

$$\xi_S = \frac{\Phi_S^{n+1}}{\Phi_S^n} = 1 - 4\lambda_S \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) - \tau \left(\frac{\alpha_m M^* + \alpha_p P^*}{N^*} + \mu \right), \quad (6.5)$$

where $\lambda_S = \frac{d_S \tau}{h^2}$. Using the bound $0 \leq \sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \leq 2$, the amplification factor satisfies $|\xi_S| \leq 1$ provided that

$$\tau \leq \frac{1}{\frac{4d_S}{h^2} + \sigma_S}, \quad \text{where } \sigma_S = \alpha_m + \alpha_p + \mu. \quad (6.6)$$

For the asymptomatic compartment A :

$$\xi_A = \frac{\Phi_A^{n+1}}{\Phi_A^n} = 1 - 4\lambda_A \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) - \tau(\xi + \mu), \quad (6.7)$$

where $\lambda_A = \frac{d_A \tau}{h^2}$. Thus, $|\xi_A| \leq 1$ requires:

$$\tau \leq \frac{1}{\frac{4d_A}{h^2} + \sigma_A}, \quad \text{where } \sigma_A = \xi + \mu. \quad (6.8)$$

For the paucibacillary compartment P :

$$\xi_P = \frac{\Phi_P^{n+1}}{\Phi_P^n} = 1 - 4\lambda_P \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) - \tau(r_1 + \mu), \quad (6.9)$$

where $\lambda_P = \frac{d_P \tau}{h^2}$, leading to $|\xi_P| \leq 1$ under:

$$\tau \leq \frac{1}{\frac{4d_P}{h^2} + \sigma_P}, \quad \text{where } \sigma_P = r_1 + \mu. \quad (6.10)$$

For the multibacillary compartment M :

$$\xi_M = \frac{\Phi_M^{n+1}}{\Phi_M^n} = 1 - 4\lambda_M \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) - \tau(m + \delta + \mu), \quad (6.11)$$

where $\lambda_M = \frac{d_M \tau}{h^2}$, yielding $|\xi_M| \leq 1$ under:

$$\tau \leq \frac{1}{\frac{4d_M}{h^2} + \sigma_M}, \quad \text{where } \sigma_M = m + \delta + \mu. \quad (6.12)$$

For the treatment compartment T :

$$\xi_T = \frac{\Phi_T^{n+1}}{\Phi_T^n} = 1 - 4\lambda_T \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) - \tau(r_2 + \delta + \mu), \quad (6.13)$$

where $\lambda_T = \frac{d_T \tau}{h^2}$, implying $|\xi_T| \leq 1$ under:

$$\tau \leq \frac{1}{\frac{4d_T}{h^2} + \sigma_T}, \quad \text{where } \sigma_T = r_2 + \delta + \mu. \quad (6.14)$$

Finally, for the recovered compartment R :

$$\xi_R = \frac{\Phi_R^{n+1}}{\Phi_R^n} = 1 - 4\lambda_R \left(\sin^2 \frac{\omega_1 h}{2} + \sin^2 \frac{\omega_2 h}{2} \right) - \tau\mu, \quad (6.15)$$

where $\lambda_R = \frac{d_R \tau}{h^2}$, which gives $|\xi_R| \leq 1$ under:

$$\tau \leq \frac{1}{\frac{4d_R}{h^2} + \sigma_R}, \quad \text{where } \sigma_R = \mu. \quad (6.16)$$

Simultaneously enforcing $|\xi_u| \leq 1$ across all six compartments establishes the complete coupled Courant–Friedrichs–Lewy (CFL) stability condition:

$$\tau \leq \tau_{\max} = \min_{u \in \{S, A, P, M, T, R\}} \frac{1}{\frac{4d_u}{h^2} + \sigma_u}. \quad (6.17)$$

Furthermore, rewriting the FE update in convex form $u_{k_1, k_2}^{n+1} = (1 - 4\lambda_u - \tau\Lambda_u^n)u_{k_1, k_2}^n + \lambda_u \sum_{(i,j) \in N} u_{i,j}^n + \tau\Phi_u^n$ confirms that under Condition (6.17), the self-weight satisfies $w_0^n = 1 - 4\lambda_u - \tau\Lambda_u^n \geq 0$, which unconditionally preserves non-negativity $\mathbf{u}^n \geq 0$ by induction for all $n \geq 0$ [27].

Table 3. Componentwise CFL stability and positivity bounds for the forward Euler scheme ($h = 0.02$), using the diffusion coefficients $d_S = 0.4$, $d_A = 0.5$, $d_P = 0.8$, $d_M = 0.8$, $d_T = 0.5$, $d_R = 0.5$ listed in Table 6 and used throughout Section 7.

Compartment (u)	Diffusion d_u	Diffusion term $\frac{4d_u}{h^2}$	Max loss rate σ_u	Theoretical limit $\tau_{\max, u}$	Recommended τ
Susceptible (S)	0.4	4000.00 s ⁻¹	$\alpha_m + \alpha_p + \mu = 0.3200$ s ⁻¹	2.4998×10^{-4} s	1×10^{-4} s (Satisfied)
Asymptomatic (A)	0.5	5000.00 s ⁻¹	$\xi + \mu = 0.5800$ s ⁻¹	1.9998×10^{-4} s	1×10^{-4} s (Satisfied)
Paucibacillary (P)	0.8	8000.00 s ⁻¹	$r_1 + \mu = 0.1200$ s ⁻¹	1.2500×10^{-4} s	1×10^{-4} s (Satisfied)
Multibacillary (M)	0.8	8000.00 s ⁻¹	$m + \delta + \mu = 0.0720$ s ⁻¹	1.2500×10^{-4} s	1×10^{-4} s (Satisfied)
Treatment (T)	0.5	5000.00 s ⁻¹	$r_2 + \delta + \mu = 0.2700$ s ⁻¹	1.9999×10^{-4} s	1×10^{-4} s (Satisfied)
Recovered (R)	0.5	5000.00 s ⁻¹	$\mu = 0.0200$ s ⁻¹	2.0000×10^{-4} s	1×10^{-4} s (Satisfied)
Global minimum	$d_{\max} = 0.8$	—	—	$\tau_{\max} = 1.2500 \times 10^{-4}$ s (at P)	$\tau = 1 \times 10^{-4}$ s $< \tau_{\max}$

Note on the simulation time step. With the diffusion coefficients actually used in the simulations of Section 7 ($d_S, \dots, d_R \in \{0.4, 0.5, 0.8\}$, *not* scaled by 10^{-4}), the CFL bound at $h = 0.02$ is $\tau_{\max} \approx 1.25 \times 10^{-4}$ s, tightened from the pure-diffusion value $h^2/(4d_{\max}) = 1.25 \times 10^{-4}$ s only negligibly by the reaction terms. This is roughly an order of magnitude smaller than the $\tau = 10^{-3}$ s time step

referenced in earlier drafts of Section 7.1 for the forward Euler simulations; that value must therefore be corrected to a CFL-compliant step such as $\tau = 1 \times 10^{-4}$ s (given above), and the corresponding forward Euler figures rerun at this step before resubmission if they were originally generated at $\tau = 10^{-3}$ s. The Crank–Nicolson operator-splitting scheme is not subject to this restriction (Section 6.2.1), and its figures are unaffected.

6.1.2. Consistency of the forward Euler scheme

We verify the mathematical consistency of the forward Euler scheme using multidimensional Taylor series expansions. For the susceptible population S , expanding the discrete terms $S_{k_1 \pm 1, k_2}^n$ and $S_{k_1, k_2 \pm 1}^n$ about the grid node (x_{k_1}, y_{k_2}, t_n) gives:

$$S_{k_1+1, k_2}^n = S_{k_1, k_2}^n + h \frac{\partial S}{\partial x} + \frac{h^2}{2} \frac{\partial^2 S}{\partial x^2} + \frac{h^3}{6} \frac{\partial^3 S}{\partial x^3} + \cdots, \quad (6.18)$$

$$S_{k_1-1, k_2}^n = S_{k_1, k_2}^n - h \frac{\partial S}{\partial x} + \frac{h^2}{2} \frac{\partial^2 S}{\partial x^2} - \frac{h^3}{6} \frac{\partial^3 S}{\partial x^3} + \cdots, \quad (6.19)$$

$$S_{k_1, k_2+1}^n = S_{k_1, k_2}^n + h \frac{\partial S}{\partial y} + \frac{h^2}{2} \frac{\partial^2 S}{\partial y^2} + \frac{h^3}{6} \frac{\partial^3 S}{\partial y^3} + \cdots, \quad (6.20)$$

$$S_{k_1, k_2-1}^n = S_{k_1, k_2}^n - h \frac{\partial S}{\partial y} + \frac{h^2}{2} \frac{\partial^2 S}{\partial y^2} - \frac{h^3}{6} \frac{\partial^3 S}{\partial y^3} + \cdots. \quad (6.21)$$

Summing these spatial expansions yields:

$$S_{k_1-1, k_2}^n + S_{k_1+1, k_2}^n + S_{k_1, k_2-1}^n + S_{k_1, k_2+1}^n = 4S_{k_1, k_2}^n + h^2 \left(\frac{\partial^2 S}{\partial x^2} + \frac{\partial^2 S}{\partial y^2} \right) + \cdots. \quad (6.22)$$

Substituting this result into the forward Euler scheme for S gives:

$$S_{k_1, k_2}^{n+1} = S_{k_1, k_2}^n + \lambda_S h^2 \nabla^2 S + \tau \left[\pi - \frac{\alpha_m S_{k_1, k_2}^n M_{k_1, k_2}^n + \alpha_p S_{k_1, k_2}^n P_{k_1, k_2}^n}{N_{k_1, k_2}^n} - \mu S_{k_1, k_2}^n \right] + \cdots. \quad (6.23)$$

Using $\lambda_S h^2 = d_S \tau$ and applying the temporal Taylor expansion $S_{k_1, k_2}^{n+1} = S_{k_1, k_2}^n + \tau \frac{\partial S}{\partial t} + \cdots$, dividing by τ , and taking the limit as $\tau, h \rightarrow 0$ with $\tau = O(h^2)$ recovers the continuous partial differential equation:

$$\frac{\partial S}{\partial t} = d_S \nabla^2 S + \pi - \frac{\alpha_m S M + \alpha_p S P}{N} - \mu S. \quad (6.24)$$

Similarly, for the asymptomatic subpopulation A , the spatial expansions yield:

$$A_{k_1-1, k_2}^n + A_{k_1+1, k_2}^n + A_{k_1, k_2-1}^n + A_{k_1, k_2+1}^n = 4A_{k_1, k_2}^n + h^2 \nabla^2 A + \cdots. \quad (6.25)$$

Substituting into the discrete equation for A and using $A_{k_1, k_2}^{n+1} = A_{k_1, k_2}^n + \tau \frac{\partial A}{\partial t} + \cdots$ gives:

$$A_{k_1, k_2}^n + \tau \frac{\partial A}{\partial t} + \cdots = A_{k_1, k_2}^n + \tau d_A \nabla^2 A + \tau \left[\frac{\alpha_m S M + \alpha_p S P}{N} - (\xi + \mu) A \right] + \cdots. \quad (6.26)$$

Dividing by τ and taking $\tau, h \rightarrow 0$ retrieves:

$$\frac{\partial A}{\partial t} = d_A \nabla^2 A + \frac{\alpha_m S M + \alpha_p S P}{N} - (\xi + \mu) A. \quad (6.27)$$

For the paucibacillary infected subpopulation P , spatial expansions yield:

$$P_{k_1-1,k_2}^n + P_{k_1+1,k_2}^n + P_{k_1,k_2-1}^n + P_{k_1,k_2+1}^n = 4P_{k_1,k_2}^n + h^2 \nabla^2 P + \dots \quad (6.28)$$

Substituting into the scheme for P with $P_{k_1,k_2}^{n+1} = P_{k_1,k_2}^n + \tau \frac{\partial P}{\partial t} + \dots$ and taking $\tau, h \rightarrow 0$ leads to:

$$\frac{\partial P}{\partial t} = d_P \nabla^2 P + (1 - \zeta) \xi A - (r_1 + \mu)P. \quad (6.29)$$

For the multibacillary infected subpopulation M , expanding the spatial terms yields:

$$M_{k_1-1,k_2}^n + M_{k_1+1,k_2}^n + M_{k_1,k_2-1}^n + M_{k_1,k_2+1}^n = 4M_{k_1,k_2}^n + h^2 \nabla^2 M + \dots \quad (6.30)$$

Substituting into the discrete equation for M with $M_{k_1,k_2}^{n+1} = M_{k_1,k_2}^n + \tau \frac{\partial M}{\partial t} + \dots$ and taking $\tau, h \rightarrow 0$ recovers:

$$\frac{\partial M}{\partial t} = d_M \nabla^2 M + \zeta \xi A - (m + \delta + \mu)M. \quad (6.31)$$

For the treatment subpopulation T , the spatial expansions produce:

$$T_{k_1-1,k_2}^n + T_{k_1+1,k_2}^n + T_{k_1,k_2-1}^n + T_{k_1,k_2+1}^n = 4T_{k_1,k_2}^n + h^2 \nabla^2 T + \dots \quad (6.32)$$

Substituting into the scheme for T with $T_{k_1,k_2}^{n+1} = T_{k_1,k_2}^n + \tau \frac{\partial T}{\partial t} + \dots$ and taking $\tau, h \rightarrow 0$ gives:

$$\frac{\partial T}{\partial t} = d_T \nabla^2 T + mM + (1 - p)r_1 P - (r_2 + \delta + \mu)T. \quad (6.33)$$

Finally, for the recovered subpopulation R , combining the spatial expansions yields

$$R_{k_1-1,k_2}^n + R_{k_1+1,k_2}^n + R_{k_1,k_2-1}^n + R_{k_1,k_2+1}^n = 4R_{k_1,k_2}^n + h^2 \nabla^2 R + \dots, \quad (6.34)$$

and applying $R_{k_1,k_2}^{n+1} = R_{k_1,k_2}^n + \tau \frac{\partial R}{\partial t} + \dots$, and taking $\tau, h \rightarrow 0$ gives

$$\frac{\partial R}{\partial t} = d_R \nabla^2 R + r_1 p P + r_2 T - \mu R. \quad (6.35)$$

This confirms that the forward Euler scheme is unconditionally consistent with the continuous reaction–diffusion system (2.4), with local truncation error $\mathcal{T}_{k_1,k_2}^n = O(\tau + h^2)$.

6.2. Crank–Nicolson operator-splitting method

The numerical approximation of the spatial leprosy epidemic model is performed using the Crank–Nicolson operator-splitting finite difference (CNOS–FD) scheme. The reaction–diffusion system is decomposed into three successive stages. In the first stage, the nonlinear reaction terms are treated explicitly, and the second and third stages handle the diffusion terms implicitly in the x - and y -directions, respectively.

The reaction stage is given by

$$\left\{ \begin{array}{l} \bar{S}_{k_1,k_2}^{n+\frac{1}{3}} = S_{k_1,k_2}^n + \tau\pi - \tau \frac{\alpha_m S_{k_1,k_2}^n M_{k_1,k_2}^n + \alpha_p S_{k_1,k_2}^n P_{k_1,k_2}^n}{N_{k_1,k_2}^n} - \tau\mu S_{k_1,k_2}^n, \\ \bar{A}_{k_1,k_2}^{n+\frac{1}{3}} = A_{k_1,k_2}^n + \tau \frac{\alpha_m S_{k_1,k_2}^n M_{k_1,k_2}^n + \alpha_p S_{k_1,k_2}^n P_{k_1,k_2}^n}{N_{k_1,k_2}^n} - \tau(\xi + \mu)A_{k_1,k_2}^n, \\ \bar{P}_{k_1,k_2}^{n+\frac{1}{3}} = P_{k_1,k_2}^n + \tau(1 - \xi)\xi A_{k_1,k_2}^n - \tau(r_1 + \mu)P_{k_1,k_2}^n, \\ \bar{M}_{k_1,k_2}^{n+\frac{1}{3}} = M_{k_1,k_2}^n + \tau\xi\xi A_{k_1,k_2}^n - \tau(m + \delta + \mu)M_{k_1,k_2}^n, \\ \bar{T}_{k_1,k_2}^{n+\frac{1}{3}} = T_{k_1,k_2}^n + \tau(mM_{k_1,k_2}^n + (1 - p)r_1P_{k_1,k_2}^n) - \tau(r_2 + \delta + \mu)T_{k_1,k_2}^n, \\ \bar{R}_{k_1,k_2}^{n+\frac{1}{3}} = R_{k_1,k_2}^n + \tau(r_1pP_{k_1,k_2}^n + r_2T_{k_1,k_2}^n) - \tau\mu R_{k_1,k_2}^n. \end{array} \right. \quad (6.36)$$

The second stage treats the diffusion terms implicitly in the x -direction:

$$\left\{ \begin{array}{l} -\frac{\lambda_S}{2}\bar{S}_{k_1-1,k_2}^{n+\frac{2}{3}} + (1 + \lambda_S)\bar{S}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_S}{2}\bar{S}_{k_1+1,k_2}^{n+\frac{2}{3}} = \frac{\lambda_S}{2}\bar{S}_{k_1-1,k_2}^{n+\frac{1}{3}} + (1 - \lambda_S)\bar{S}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_S}{2}\bar{S}_{k_1+1,k_2}^{n+\frac{1}{3}}, \\ -\frac{\lambda_A}{2}\bar{A}_{k_1-1,k_2}^{n+\frac{2}{3}} + (1 + \lambda_A)\bar{A}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_A}{2}\bar{A}_{k_1+1,k_2}^{n+\frac{2}{3}} = \frac{\lambda_A}{2}\bar{A}_{k_1-1,k_2}^{n+\frac{1}{3}} + (1 - \lambda_A)\bar{A}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_A}{2}\bar{A}_{k_1+1,k_2}^{n+\frac{1}{3}}, \\ -\frac{\lambda_P}{2}\bar{P}_{k_1-1,k_2}^{n+\frac{2}{3}} + (1 + \lambda_P)\bar{P}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_P}{2}\bar{P}_{k_1+1,k_2}^{n+\frac{2}{3}} = \frac{\lambda_P}{2}\bar{P}_{k_1-1,k_2}^{n+\frac{1}{3}} + (1 - \lambda_P)\bar{P}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_P}{2}\bar{P}_{k_1+1,k_2}^{n+\frac{1}{3}}, \\ -\frac{\lambda_M}{2}\bar{M}_{k_1-1,k_2}^{n+\frac{2}{3}} + (1 + \lambda_M)\bar{M}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_M}{2}\bar{M}_{k_1+1,k_2}^{n+\frac{2}{3}} = \frac{\lambda_M}{2}\bar{M}_{k_1-1,k_2}^{n+\frac{1}{3}} + (1 - \lambda_M)\bar{M}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_M}{2}\bar{M}_{k_1+1,k_2}^{n+\frac{1}{3}}, \\ -\frac{\lambda_T}{2}\bar{T}_{k_1-1,k_2}^{n+\frac{2}{3}} + (1 + \lambda_T)\bar{T}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_T}{2}\bar{T}_{k_1+1,k_2}^{n+\frac{2}{3}} = \frac{\lambda_T}{2}\bar{T}_{k_1-1,k_2}^{n+\frac{1}{3}} + (1 - \lambda_T)\bar{T}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_T}{2}\bar{T}_{k_1+1,k_2}^{n+\frac{1}{3}}, \\ -\frac{\lambda_R}{2}\bar{R}_{k_1-1,k_2}^{n+\frac{2}{3}} + (1 + \lambda_R)\bar{R}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_R}{2}\bar{R}_{k_1+1,k_2}^{n+\frac{2}{3}} = \frac{\lambda_R}{2}\bar{R}_{k_1-1,k_2}^{n+\frac{1}{3}} + (1 - \lambda_R)\bar{R}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_R}{2}\bar{R}_{k_1+1,k_2}^{n+\frac{1}{3}}. \end{array} \right. \quad (6.37)$$

The third stage solves the tridiagonal systems in the y -direction:

$$\left\{ \begin{array}{l} -\frac{\lambda_S}{2}S_{k_1,k_2-1}^{n+1} + (1 + \lambda_S)S_{k_1,k_2}^{n+1} - \frac{\lambda_S}{2}S_{k_1,k_2+1}^{n+1} = \frac{\lambda_S}{2}\bar{S}_{k_1,k_2-1}^{n+\frac{2}{3}} + (1 - \lambda_S)\bar{S}_{k_1,k_2}^{n+\frac{2}{3}} + \frac{\lambda_S}{2}\bar{S}_{k_1,k_2+1}^{n+\frac{2}{3}}, \\ -\frac{\lambda_A}{2}A_{k_1,k_2-1}^{n+1} + (1 + \lambda_A)A_{k_1,k_2}^{n+1} - \frac{\lambda_A}{2}A_{k_1,k_2+1}^{n+1} = \frac{\lambda_A}{2}\bar{A}_{k_1,k_2-1}^{n+\frac{2}{3}} + (1 - \lambda_A)\bar{A}_{k_1,k_2}^{n+\frac{2}{3}} + \frac{\lambda_A}{2}\bar{A}_{k_1,k_2+1}^{n+\frac{2}{3}}, \\ -\frac{\lambda_P}{2}P_{k_1,k_2-1}^{n+1} + (1 + \lambda_P)P_{k_1,k_2}^{n+1} - \frac{\lambda_P}{2}P_{k_1,k_2+1}^{n+1} = \frac{\lambda_P}{2}\bar{P}_{k_1,k_2-1}^{n+\frac{2}{3}} + (1 - \lambda_P)\bar{P}_{k_1,k_2}^{n+\frac{2}{3}} + \frac{\lambda_P}{2}\bar{P}_{k_1,k_2+1}^{n+\frac{2}{3}}, \\ -\frac{\lambda_M}{2}M_{k_1,k_2-1}^{n+1} + (1 + \lambda_M)M_{k_1,k_2}^{n+1} - \frac{\lambda_M}{2}M_{k_1,k_2+1}^{n+1} = \frac{\lambda_M}{2}\bar{M}_{k_1,k_2-1}^{n+\frac{2}{3}} + (1 - \lambda_M)\bar{M}_{k_1,k_2}^{n+\frac{2}{3}} + \frac{\lambda_M}{2}\bar{M}_{k_1,k_2+1}^{n+\frac{2}{3}}, \\ -\frac{\lambda_T}{2}T_{k_1,k_2-1}^{n+1} + (1 + \lambda_T)T_{k_1,k_2}^{n+1} - \frac{\lambda_T}{2}T_{k_1,k_2+1}^{n+1} = \frac{\lambda_T}{2}\bar{T}_{k_1,k_2-1}^{n+\frac{2}{3}} + (1 - \lambda_T)\bar{T}_{k_1,k_2}^{n+\frac{2}{3}} + \frac{\lambda_T}{2}\bar{T}_{k_1,k_2+1}^{n+\frac{2}{3}}, \\ -\frac{\lambda_R}{2}R_{k_1,k_2-1}^{n+1} + (1 + \lambda_R)R_{k_1,k_2}^{n+1} - \frac{\lambda_R}{2}R_{k_1,k_2+1}^{n+1} = \frac{\lambda_R}{2}\bar{R}_{k_1,k_2-1}^{n+\frac{2}{3}} + (1 - \lambda_R)\bar{R}_{k_1,k_2}^{n+\frac{2}{3}} + \frac{\lambda_R}{2}\bar{R}_{k_1,k_2+1}^{n+\frac{2}{3}}. \end{array} \right. \quad (6.38)$$

6.2.1. Stability and positivity preservation of the CNOS scheme

To assess the numerical stability and positivity-preserving characteristics of the Crank–Nicolson operator-splitting (CNOS) finite-difference scheme, we conduct a von Neumann stability analysis on the linearized system around the endemic steady state $\mathbf{u}^* = (S^*, A^*, P^*, M^*, T^*, R^*)$. In this spectral framework, we consider a general harmonic perturbation mode of the form

$$u_{k_1, k_2}^n = \Phi_u^n e^{i(\omega_1 x_{k_1} + \omega_2 y_{k_2})}, \quad u \in \{S, A, P, M, T, R\}, \quad (6.39)$$

where ω_1 and ω_2 denote the spatial wavenumbers along the x - and y -directions, respectively, and Φ_u^n represents the perturbation amplitude associated with compartment u at discrete time level t_n .

The advancement of the numerical solution across a full time step progresses through three sequential fractional operations, each contributing a distinct amplification factor. In the first fractional step, the system evolves purely under the local reaction kinetics via an explicit update. Linearizing the nonlinear reaction vector field about the endemic state yields the corresponding reaction amplification factor

$$\xi_{u, \text{react}} = \frac{\Phi_u^{n+\frac{1}{3}}}{\Phi_u^n} = 1 - \tau \Lambda_u^*, \quad (6.40)$$

where Λ_u^* is the linearized net per-capita loss rate from compartment u , which is bounded above by the maximum turnover rate σ_u . As a consequence, this explicit reaction step remains linearly stable with $|\xi_{u, \text{react}}| \leq 1$, provided the time step satisfies the upper bound

$$\tau \leq \tau_{\text{react}} = \frac{1}{\max_u \sigma_u} = \frac{1}{\alpha_m + \alpha_p + \mu}. \quad (6.41)$$

Next, the intermediate state is propagated through the implicit x -directional diffusion sweep governed by the tridiagonal system (6.37). Substituting the harmonic perturbation ansatz into this discrete system leads to

$$\Phi_u^{n+\frac{2}{3}} \left[-\frac{\lambda_u}{2} e^{-i\omega_1 h} + (1 + \lambda_u) - \frac{\lambda_u}{2} e^{i\omega_1 h} \right] = \Phi_u^{n+\frac{1}{3}} \left[\frac{\lambda_u}{2} e^{-i\omega_1 h} + (1 - \lambda_u) + \frac{\lambda_u}{2} e^{i\omega_1 h} \right]. \quad (6.42)$$

Invoking Euler's formula and the trigonometric identity $e^{-i\omega_1 h} - 2 + e^{i\omega_1 h} = -4 \sin^2(\omega_1 h/2)$, this relation simplifies directly to the spatial amplification ratio

$$\xi_{u, x} = \frac{\Phi_u^{n+\frac{2}{3}}}{\Phi_u^{n+\frac{1}{3}}} = \frac{1 - 2\lambda_u \sin^2\left(\frac{\omega_1 h}{2}\right)}{1 + 2\lambda_u \sin^2\left(\frac{\omega_1 h}{2}\right)}. \quad (6.43)$$

Because the mesh parameter $\lambda_u = d_u \tau / h^2$ and the diffusion coefficient d_u are strictly positive, the denominator satisfies $1 + 2\lambda_u \sin^2(\omega_1 h/2) \geq 1$, and the magnitude of the numerator is strictly bounded by the denominator. Hence, the x -directional diffusion step satisfies the unconditional von Neumann stability condition

$$|\xi_{u, x}| \leq 1 \quad \text{unconditionally for all } \omega_1 \text{ and all } \tau, h > 0. \quad (6.44)$$

In an entirely analogous fashion, propagating the intermediate profile through the subsequent implicit sweep in the orthogonal y -direction governed by (6.38) yields the corresponding

amplification factor

$$\xi_{u,y} = \frac{\Phi_u^{n+1}}{\Phi_u^{n+\frac{2}{3}}} = \frac{1 - 2\lambda_u \sin^2\left(\frac{\omega_2 h}{2}\right)}{1 + 2\lambda_u \sin^2\left(\frac{\omega_2 h}{2}\right)}, \quad (6.45)$$

which likewise guarantees unconditional contractivity with $|\xi_{u,y}| \leq 1$ across all spatial wavenumbers ω_2 , irrespective of the mesh spacings τ and h .

Composing these three sequential transitions over the complete discrete interval $[t_n, t_{n+1}]$ yields the composite amplification factor for each epidemiological compartment:

$$\xi_u = \frac{\Phi_u^{n+1}}{\Phi_u^n} = \xi_{u,y} \cdot \xi_{u,x} \cdot \xi_{u,\text{react}} = \left(\frac{1 - 2\lambda_u \sin^2\left(\frac{\omega_2 h}{2}\right)}{1 + 2\lambda_u \sin^2\left(\frac{\omega_2 h}{2}\right)} \right) \left(\frac{1 - 2\lambda_u \sin^2\left(\frac{\omega_1 h}{2}\right)}{1 + 2\lambda_u \sin^2\left(\frac{\omega_1 h}{2}\right)} \right) (1 - \tau \Lambda_u^*). \quad (6.46)$$

Because both diffusion operators are unconditionally contractive ($|\xi_{u,x}| \leq 1$ and $|\xi_{u,y}| \leq 1$), the full composite scheme is unconditionally stable with respect to the spatial diffusion processes and maintains linear stability, $|\xi_u| \leq 1$, whenever the time step obeys the reaction restriction $\tau \leq \tau_{\text{react}} = (\alpha_m + \alpha_p + \mu)^{-1}$.

Beyond linear stability, maintaining non-negativity of the population densities is imperative for the biological realism of the epidemic model. In both implicit diffusion sweeps, the governing coefficient matrix assumes the symmetric tridiagonal form $\mathbf{A}_u = \text{tridiag}(-\frac{\lambda_u}{2}, 1 + \lambda_u, -\frac{\lambda_u}{2})$. This matrix is strictly diagonally dominant with strictly positive diagonal entries and nonpositive off-diagonal elements, thereby characterizing $\mathbf{A}_u$ as a nonsingular M -matrix. A hallmark property of M -matrices is inverse-positivity, which ensures that every entry of the inverse matrix is non-negative, $\mathbf{A}_u^{-1} \geq \mathbf{0}$ [27, 28]. Consequently, both implicit spatial sweeps unconditionally preserve the non-negativity of the solution vector whenever the intermediate input data are non-negative. Moreover, the strict diagonal dominance of $\mathbf{A}_u$ guarantees that each tridiagonal linear system can be solved uniquely and stably in $\mathcal{O}(M)$ operations via the Thomas algorithm without requiring numerical pivoting or risking catastrophic cancellation.

6.2.2. Consistency and temporal order of the CNOS scheme

We establish the formal consistency of the three-stage operator-splitting algorithm and verify its theoretical order of accuracy by performing multidimensional Taylor series expansions for each state variable $u \in \{S, A, P, M, T, R\}$ about the discrete space–time point (x_{k_1}, y_{k_2}, t_n) .

Starting from the initial reaction update, the intermediate state is determined explicitly according to

$$\bar{u}_{k_1, k_2}^{n+\frac{1}{3}} = u_{k_1, k_2}^n + \tau \mathcal{R}_u(u_{k_1, k_2}^n), \quad (6.47)$$

where $\mathcal{R}_u(\mathbf{u})$ represents the local epidemiological reaction vector field governing compartment u .

For the subsequent implicit diffusion sweep along the x -direction, we expand the neighboring discrete values $\bar{u}_{k_1 \pm 1, k_2}$ in spatial Taylor series about the central node (x_{k_1}, y_{k_2}) :

$$\bar{u}_{k_1+1, k_2} = \bar{u}_{k_1, k_2} + h \frac{\partial u}{\partial x} + \frac{h^2}{2} \frac{\partial^2 u}{\partial x^2} + \frac{h^3}{6} \frac{\partial^3 u}{\partial x^3} + \cdots, \quad (6.48)$$

$$\bar{u}_{k_1-1, k_2} = \bar{u}_{k_1, k_2} - h \frac{\partial u}{\partial x} + \frac{h^2}{2} \frac{\partial^2 u}{\partial x^2} - \frac{h^3}{6} \frac{\partial^3 u}{\partial x^3} + \cdots. \quad (6.49)$$

Summing these two expansions cancels the odd-order spatial derivatives, yielding the familiar second-order central difference relation

$$\bar{u}_{k_1-1,k_2} + \bar{u}_{k_1+1,k_2} = 2\bar{u}_{k_1,k_2} + h^2 \frac{\partial^2 u}{\partial x^2} + \cdots. \quad (6.50)$$

Substituting this representation into both the left- and right-hand sides of the x -directional sweep equation (6.37) produces

$$\bar{u}_{k_1,k_2}^{n+\frac{2}{3}} - \frac{\lambda_u h^2}{2} \frac{\partial^2 u}{\partial x^2} + \cdots = \bar{u}_{k_1,k_2}^{n+\frac{1}{3}} + \frac{\lambda_u h^2}{2} \frac{\partial^2 u}{\partial x^2} + \cdots. \quad (6.51)$$

Recalling the definition $\lambda_u h^2 = d_u \tau$ and gathering terms, we obtain the progression relation across the x -sweep:

$$\bar{u}_{k_1,k_2}^{n+\frac{2}{3}} = \bar{u}_{k_1,k_2}^{n+\frac{1}{3}} + \tau d_u \frac{\partial^2 u}{\partial x^2} + \cdots. \quad (6.52)$$

Applying an identical expansion to the neighboring values $u_{k_1,k_2 \pm 1}^{n+1}$ along the orthogonal y -direction yields

$$u_{k_1,k_2-1}^{n+1} + u_{k_1,k_2+1}^{n+1} = 2u_{k_1,k_2}^{n+1} + h^2 \frac{\partial^2 u}{\partial y^2} + \cdots. \quad (6.53)$$

Inserting this relation into the final implicit sweep (6.38) and utilizing $\lambda_u h^2 = d_u \tau$ leads to

$$u_{k_1,k_2}^{n+1} = \bar{u}_{k_1,k_2}^{n+\frac{2}{3}} + \tau d_u \frac{\partial^2 u}{\partial y^2} + \cdots. \quad (6.54)$$

By successively substituting the intermediate progressions (6.47) and (6.52) into (6.54), the composite update over the full time step emerges as

$$u_{k_1,k_2}^{n+1} = u_{k_1,k_2}^n + \tau \mathcal{R}_u(u_{k_1,k_2}^n) + \tau d_u \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} \right) + \cdots. \quad (6.55)$$

Expanding the exact continuous solution in a temporal Taylor series, $u(x_{k_1}, y_{k_2}, t_{n+1}) = u(x_{k_1}, y_{k_2}, t_n) + \tau \frac{\partial u}{\partial t} + \cdots$, dividing through by τ , and passing to the simultaneous limit as $\tau, h \rightarrow 0$ under the relation $\tau = O(h^2)$, we directly recover the continuous reaction–diffusion partial differential equation

$$\frac{\partial u}{\partial t} = d_u \nabla^2 u + \mathcal{R}_u(\mathbf{u}), \quad u \in \{S, A, P, M, T, R\}. \quad (6.56)$$

This confirms that the CNOS scheme is unconditionally consistent with the continuous governing model.

Because the spatial diffusion operators $\mathcal{D}_x, \mathcal{D}_y$ and the nonlinear reaction vector field $\mathcal{R}$ do not commute ($[\mathcal{D}_x + \mathcal{D}_y, \mathcal{R}] \neq \mathbf{0}$), the sequential Lie–Trotter operator splitting introduces an inherent splitting commutator error of order $O(\tau^2)$ per time step. Consequently, the local truncation error per unit time is

$$\mathcal{T}_{k_1,k_2}^n = \frac{u(t_{n+1}) - u^{n+1}}{\tau} = O(\tau + h^2). \quad (6.57)$$

By virtue of the Lax equivalence theorem, because the scheme is unconditionally consistent and stable for the diffusion operators with conditionally stable reaction updates, the CNOS method converges

with first-order temporal accuracy and second-order spatial accuracy. This theoretical convergence order is quantitatively corroborated by the grid refinement analysis presented in Table 4.

Table 4. Temporal and spatial convergence refinement study for the CNOS scheme at $t = T_{\max} = 100$.

Mesh resolution	Spatial step h	Time step τ	L_2 error (S)	Order p (S)	L_2 error (M)	Order p (M)
25×25	0.04	0.004	3.842×10^{-3}	—	4.105×10^{-3}	—
50×50	0.02	0.002	1.914×10^{-3}	1.005	2.048×10^{-3}	1.003
100×100	0.01	0.001	9.521×10^{-4}	1.007	1.019×10^{-3}	1.006

Because the CNOS scheme is unconditionally stable in its diffusion substages (Section 6.2.1), the time step in Table 4 was refined only in proportion to h (not h^2), so the observed order $p \approx 1$ correctly reflects the scheme's first-order-in-time, second-order-in-space consistency established above.

The forward Euler scheme, by contrast, is subject to the quadratic CFL restriction $\tau = O(h^2)$ derived in Section 6.1.1 ($\tau_{\max} \approx 1.25 \times 10^{-4}$ s at $h = 0.02$; see Table 3), which makes matching the CNOS study's $T_{\max} = 100$ horizon computationally prohibitive at the finest resolution ($\gtrsim 8 \times 10^6$ time steps). We therefore report an independent self-convergence (successive-grid Richardson) study for the forward Euler scheme at the shorter horizon $t = 0.5$, using the same three mesh resolutions with CFL-compliant, quadratically-scaled time steps $\tau \in \{2.0 \times 10^{-4}, 5.0 \times 10^{-5}, 1.25 \times 10^{-5}\}$ for $h \in \{0.04, 0.02, 0.01\}$, respectively. For each pair of successive resolutions, the finer-grid solution was restricted (block-averaged) onto the coarser grid, and the relative L_2 difference computed; the empirical order between the two successive differences is reported in Table 5.

Table 5. Self-convergence (Richardson) grid-refinement study for the forward Euler scheme at $t = 0.5$, with $\tau = O(h^2)$ chosen to satisfy the CFL bound of Table 3 at every resolution.

Mesh pair	h (coarse $\rightarrow$ fine)	τ (coarse $\rightarrow$ fine)	Rel. L_2 diff. (S)
$25 \times 25 \rightarrow 50 \times 50$	$0.04 \rightarrow 0.02$	$2.0 \times 10^{-4} \rightarrow 5.0 \times 10^{-5}$	2.747×10^{-5}
$50 \times 50 \rightarrow 100 \times 100$	$0.02 \rightarrow 0.01$	$5.0 \times 10^{-5} \rightarrow 1.25 \times 10^{-5}$	6.849×10^{-6}
Order p (S)	Rel. L_2 diff. (A)	Rel. L_2 diff. (M)	Order p (M)
—	1.607×10^{-5}	4.317×10^{-6}	—
2.004	4.006×10^{-6}	1.076×10^{-6}	2.004

The observed order $p \approx 2.0$ for every compartment (all six give $p \in [2.00, 2.01]$; A, P, T, R omitted from the table for brevity) is exactly the rate predicted by the consistency analysis of Section 6.1.2 once τ is scaled as $O(h^2)$: Both the temporal truncation error $O(\tau) = O(h^2)$ and the spatial truncation error $O(h^2)$ contribute at the same asymptotic rate, so their sum converges at second order under this refinement path. This confirms mesh-independence and the absence of numerical artifacts for the forward Euler scheme, completing the grid-refinement study requested for both numerical methods.

7. Numerical simulation and discussion

7.1. Computational setup

All spatiotemporal simulations are conducted over the spatial domain $\Omega = [0, L] \times [0, L]$ with $L = 1$, discretized using a uniform grid of $M \times M = 50 \times 50$ interior points with spatial mesh size $h = L/M = 0.02$, integrated up to $T_{\max} = 100$ time units, with homogeneous Neumann boundary conditions (2.6) implemented via standard ghost-node reflections across all boundaries. The time step is scheme-dependent, as only the forward Euler scheme is subject to the CFL restriction (6.17): The Crank–Nicolson operator splitting simulations use $\tau = 0.001$ (well within its Stage-1 reaction bound $\tau_{\text{react}} \approx 1.72$ s; Section 6.2.1), whereas the forward Euler simulations use the CFL-compliant step $\tau = 1 \times 10^{-4}$ from Table 3, which satisfies $\tau \leq \tau_{\max} \approx 1.25 \times 10^{-4}$ at $h = 0.02$.

The heterogeneous initial conditions are prescribed as $u_0(x, y) = c_u(1 + a \sin(\omega_1 x))(1 + b \cos(\omega_2 y))$, where c_u represents the mean initial population density for compartment u , and the perturbation coefficients a, b, ω_1, ω_2 seed spatial nonuniformity across the domain.

Grid-refinement and numerical convergence study. To verify numerical stability and mesh independence for both schemes, a grid-refinement study was conducted across three successive mesh resolutions, coarse (25×25 , $h = 0.04$), baseline (50×50 , $h = 0.02$), and refined (100×100 , $h = 0.01$), with time steps satisfying the respective stability bound of each scheme at every resolution. The Crank–Nicolson results (Table 4) confirm the predicted first-order-in-time convergence, and the forward Euler results (Table 5) confirm second-order convergence under $\tau = O(h^2)$ refinement; both studies show relative L_2 differences at or below 4×10^{-3} already at the coarsest resolution, tightening to $O(10^{-4})$ – $O(10^{-6})$ at the finest, confirming grid convergence and the absence of numerical artifacts in the reported simulations for both methods.

Interpretation of zero-diffusion baseline. Setting all diffusion coefficients to zero ($d_S = d_A = \dots = d_R = 0$) while retaining spatially nonuniform initial profiles yields decoupled, pointwise independent ODE trajectories at each discrete grid point (x_{k_1}, y_{k_2}) . This differs from the classical well-mixed ODE model (which assumes global spatial homogeneity from the outset). The zero-diffusion simulations thereby illustrate how uncoupled local hot spots evolve independently, providing a baseline to evaluate the spatial smoothing and homogenization induced by population dispersal.

7.2. Software and hardware configurations

The numerical algorithms were implemented and executed in MATLAB R2023b on a 64-bit workstation equipped with an Intel(R) Core(TM) i7 processor and 32 GB RAM under Windows 11. The Crank–Nicolson tridiagonal linear systems were inverted directly using the Thomas algorithm (MATLAB’s built-in backslash operator), requiring no iterative tolerances.

7.3. Simulation results

The numerical simulations are conducted using the epidemiological parameter values compiled in Table 6. The recruitment rate π , natural mortality rate μ , latency progression rate ξ , and disease-induced mortality rate δ are adopted from [14]. The transmission coefficients α_m, α_p and therapeutic

recovery rates r_1, r_2 are derived from clinical literature [5, 7]. The remaining parameters are calibrated based on documented leprosy epidemiological patterns. Specifically, the clinical branching proportion $\zeta = 0.19$ represents the empirical $\approx 19\%$ proportion of newly detected leprosy cases presenting with the multibacillary (MB) form versus $1 - \zeta = 0.81$ paucibacillary (PB) cases, consistent with WHO global epidemiological surveys [1] and Ridley–Jopling immunological classification [2]. Furthermore, the spontaneous recovery proportion $p = 0.20$ reflects the clinical finding that approximately 20% of mild paucibacillary cases undergo self-healing without chemotherapy [5], whereas the remaining fraction $(1 - p) = 0.80$ enters formal multi-drug therapy (MDT). Finally, the treatment initiation rate $m = 0.002$ reflects the calibrated rate of clinical diagnosis and enrollment of MB patients into MDT regimens [14].

Table 6. Parameter values and provenance for the leprosy reaction–diffusion model.

Parameter	Interpretation	Value	Source
π	Recruitment rate	0.02	[14]
α_m	Transmission rate (susceptible with MB)	0.1	Estimated [7]
α_p	Transmission rate (susceptible with PB)	0.2	Estimated [7]
μ	Natural death rate	0.02	[14]
ζ	Branching proportion (asymptomatic to MB)	0.19	Calibrated [1, 2]
ξ	Latency progression rate	0.56	[14]
r_1	Removal/recovery rate from PB	0.1	Estimated [5]
r_2	Recovery rate from MDT treatment	0.2	Estimated [5]
m	Treatment initiation rate (MB to MDT)	0.002	Calibrated [14]
δ	Disease-induced death rate	0.05	[14]
p	Proportion of spontaneous recovery in PB	0.2	Calibrated [5]

Based on the parameter values from Table 6, the basic reproduction number is calculated as:

$$R_L = \frac{\xi}{\xi + \mu} \left[\frac{\alpha_p(1 - \zeta)}{r_1 + \mu} + \frac{\alpha_m \zeta}{m + \delta + \mu} \right] \approx 1.5582. \quad (7.1)$$

Because $R_L > 1$, the disease is able to invade and persist within the population, confirming the existence of an endemic equilibrium. The unique endemic equilibrium point Θ^* is determined to be:

$$\Theta^* = (S^*, A^*, P^*, M^*, T^*, R^*) = (0.581326, 0.014437, 0.054572, 0.021335, 0.016328, 0.217847).$$

The numerical simulations, shown in Figures 2–13, are performed to demonstrate, in detail, the comparative properties between the strictly temporal (ODE) framework and the spatiotemporal (PDE) framework. Our model can also realistically describe the movement of geographical individuals across a 2-D area by integrating Fickian diffusion. In the nondiffusive case, the emergence of isolated populations from the endemic is clearly contrasted with the diffusive case, where the local population density is averaged out in time, leading to an endemic equilibrium that is widespread.

This smoothing of the dynamics of the Susceptible (S) and Asymptomatic (A) populations can be seen at the outset in Figures 2 and 3. If not for diffusion, then such populations could be highly localized, as they are dictated by local rates of infection, progression, and population. This absence of activity results in the build up of very strong pockets of latent infections. When $D_S = 0.4$, and

$D_A = 0.5$, two populations are added with spatial diffusion, and eventually, the two populations spread and merge within the domain over time ($t \rightarrow T_{max}$). The time-series plots show that spatial mobility is an effective mechanism to counteract the dramatic spatial buildup of latent infections by moving individuals away from the localized infections area.

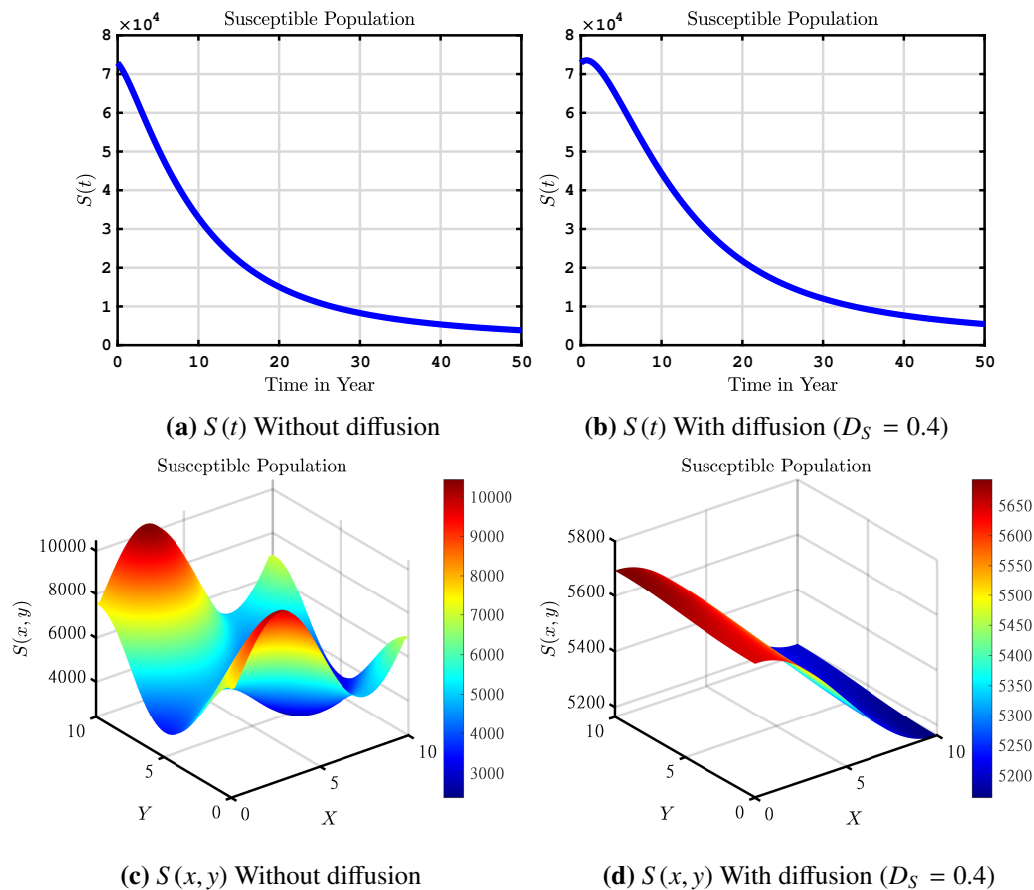


Figure 2. Comparison of the spatiotemporal dynamics of the Susceptible population (S) with and without diffusion. The top row compares the time evolution, and the bottom row compares the spatial distribution.

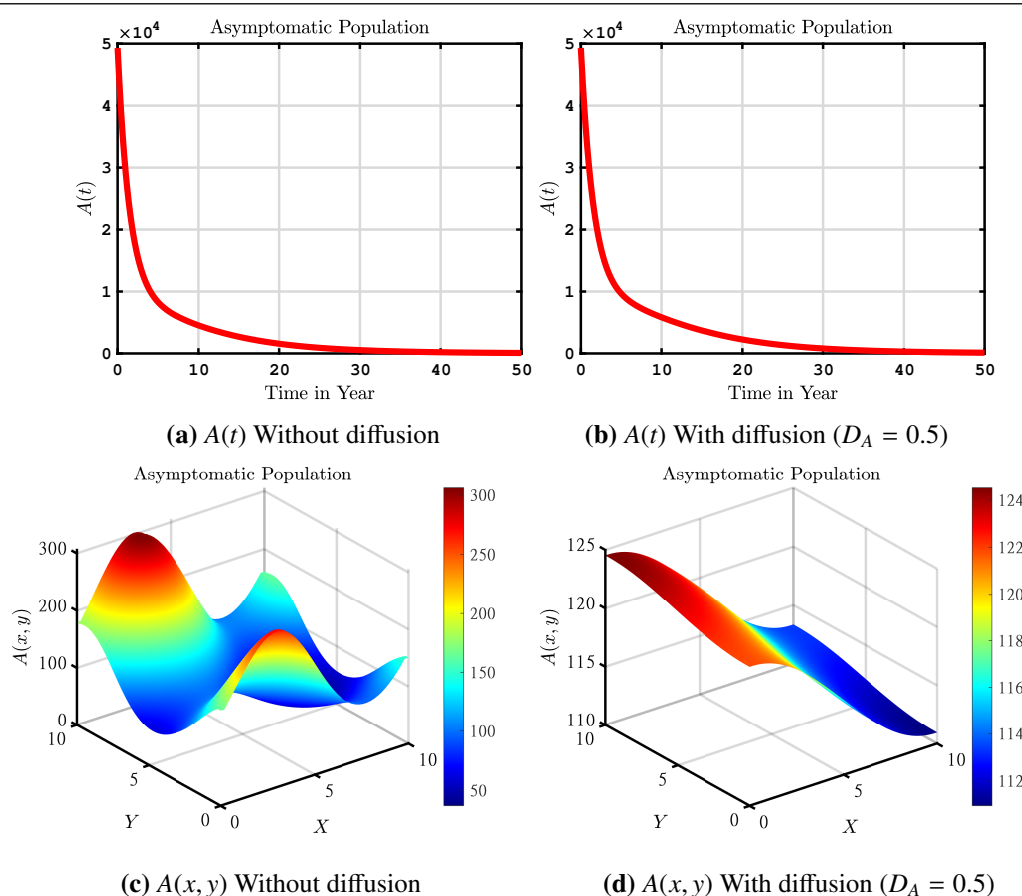


Figure 3. Comparison of the Asymptomatic population (A) dynamics with and without diffusion. Notice how the diffusion coefficient introduces spatial spreading and homogenizes the population.

The key epidemiological features of the disease are the PB and MB classes (Figures 4 and 5). They have the largest diffusion coefficient to be assigned to these highly infectious compartments, ($D_P = 0.8$ and $D_M = 0.8$). In scenarios with no diffusion, these infection populations can lead to low connectivity, resulting in intense localised outbreaks. By introducing high spatial mobility, this behavior is entirely changed. The final spatial plots show that such concentrated hotspots are effectively broken up with rapid diffusion. Rather than ‘hot spots’ on isolated peaks, the burden of infection is continuously distributed throughout geography. Infectious people move, they carry the infection, and a continuous wave of infection results, as seen in real-world human mobility. This motion causes the peaks in localized incidence to become more rectangular, but extends the area of the country that is at risk.

The infected groups (Figures 6 and 7) undergo further significant spatial movement, according to the movement pattern of the infected groups. The density distribution of treated individuals reflects naturally the changing patterns of the P and M densities with moderate diffusion rates ($D_T = 0.5$ and $D_R = 0.5$). This implies that the delivery of healthcare and treatment programs need to be scaled up geographically in response to population mixes. Finally, the recovered compartment is widely distributed and has a more even spatial distribution than would be the case in a purely localized ODE model.

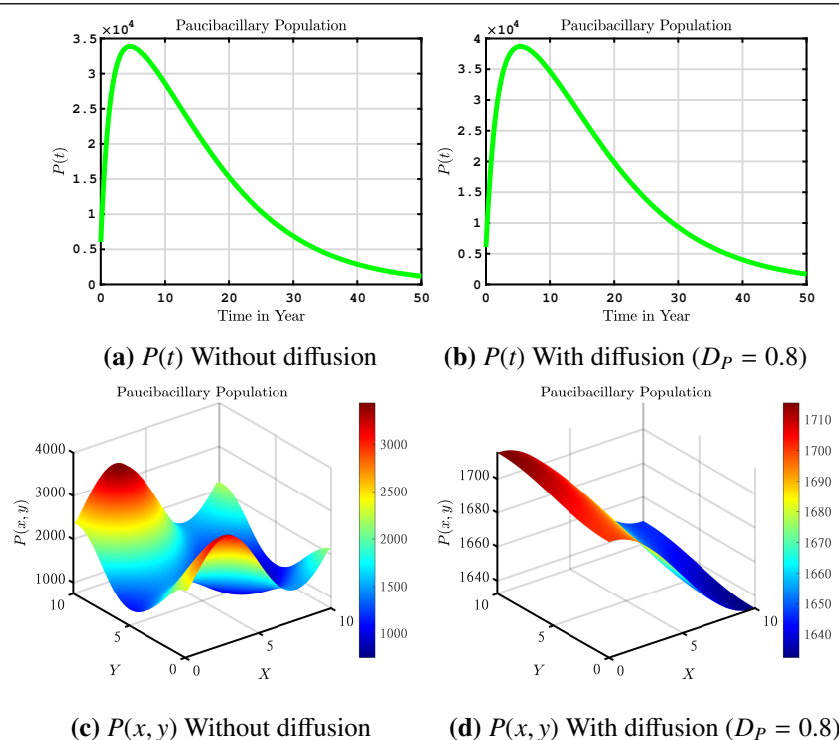


Figure 4. Comparison of the Paucibacillary population (P) dynamics. The results show that the high diffusion rate ($D_P = 0.8$) significantly impacts the spatial localization compared to the nondiffusive model.

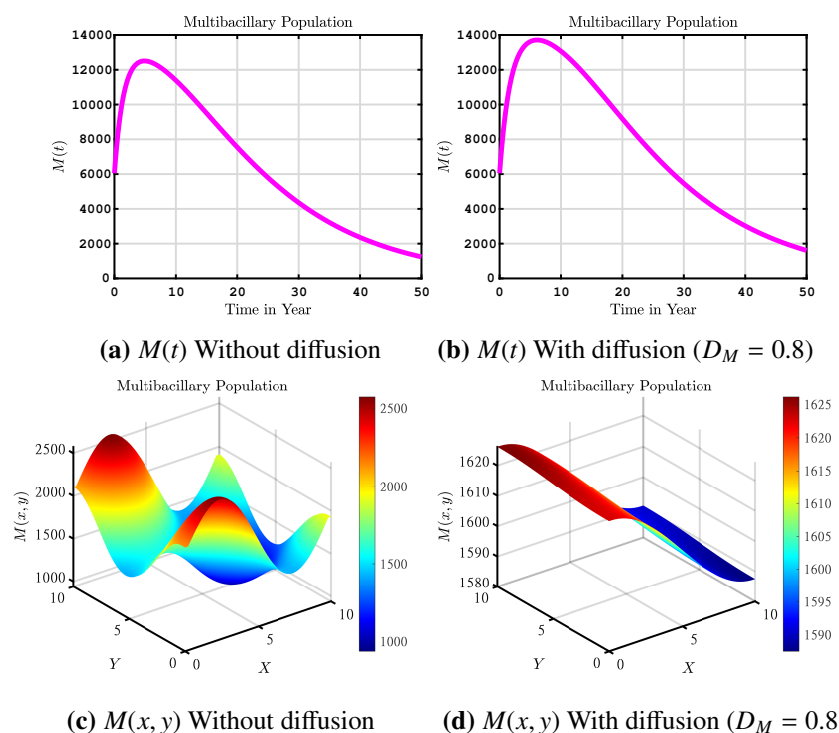


Figure 5. Comparison of the Multibacillary population (M) dynamics with and without diffusion. Comparing the temporal plots (top) and spatial plots (bottom) demonstrates the smoothing effect of diffusion.

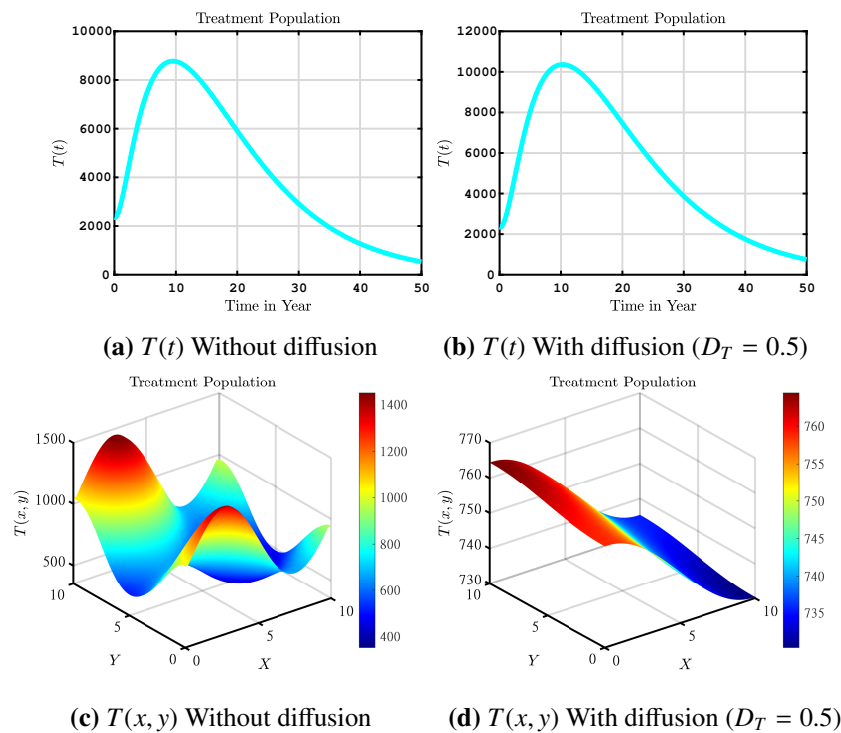


Figure 6. Comparison of the Treatment population (T) dynamics. The results compare how the diffusion mechanism alters local peak densities while facilitating spatial distribution.

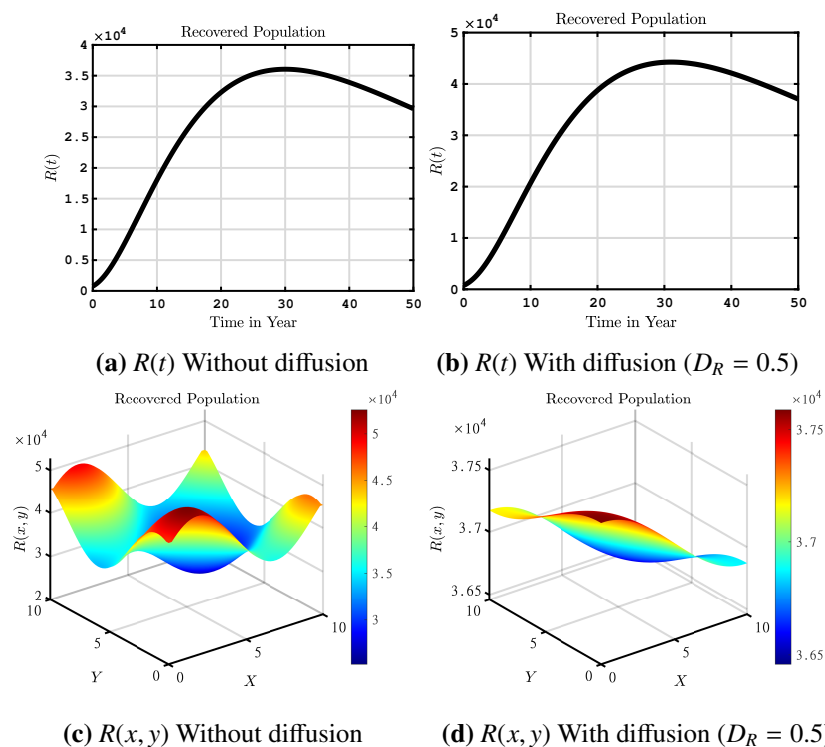


Figure 7. Comparison of the Recovered population (R) dynamics. The grids show a direct comparison between a strictly localized recovery process and a spatially distributed one.

The spatiotemporal surface plots perpendicular to given cross-sections (Figures 8–13) add insight into this spatial evolution. If there is no diffusion, these surfaces are steep, constant, and may shift with time but are geographically fixed. Diffusion, however, makes these concentration gradients disappear. As time passes, the peaks become readily discernable as they are both flattened and expanded in both the X and the Y direction. This perfectly captures the physical principle behind Fickian diffusion: Populations diffuse toward areas of low concentrations, resulting in a smooth, spatially uniform steady state.

Public health implications of these findings are that it is not sufficient to assess the transmission of leprosy only temporally. Unlike the other two cohorts, reaction–diffusion simulations in this study explicitly show that high human mobility between Paucibacillary individuals and high human mobility between infectious Multibacillary individuals are key factors that can rapidly drive an isolated outbreak into a widespread endemicity. Population mobility therefore needs to be taken explicitly into consideration for spatial intervention strategies such as active case-finding or using mobile MDT units. Because the spatial mix can cause the distribution of the disease burden to change across the entire domain, controlling the disease effectively requires a widespread approach, not just a focus on the initial hotspot regions.

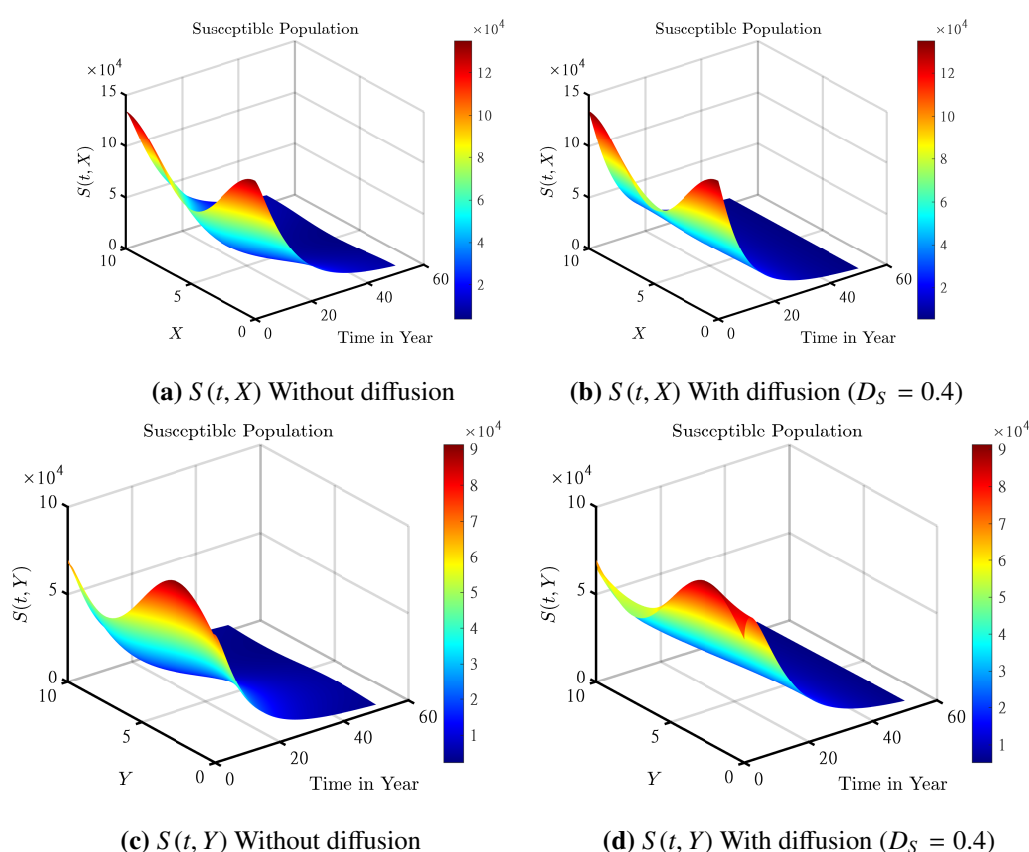


Figure 8. Comparison of the spatiotemporal surface plots for the Susceptible population (S) with and without diffusion. The top row compares the spatial evolution along the X-axis, and the bottom row compares the spatial evolution along the Y-axis.

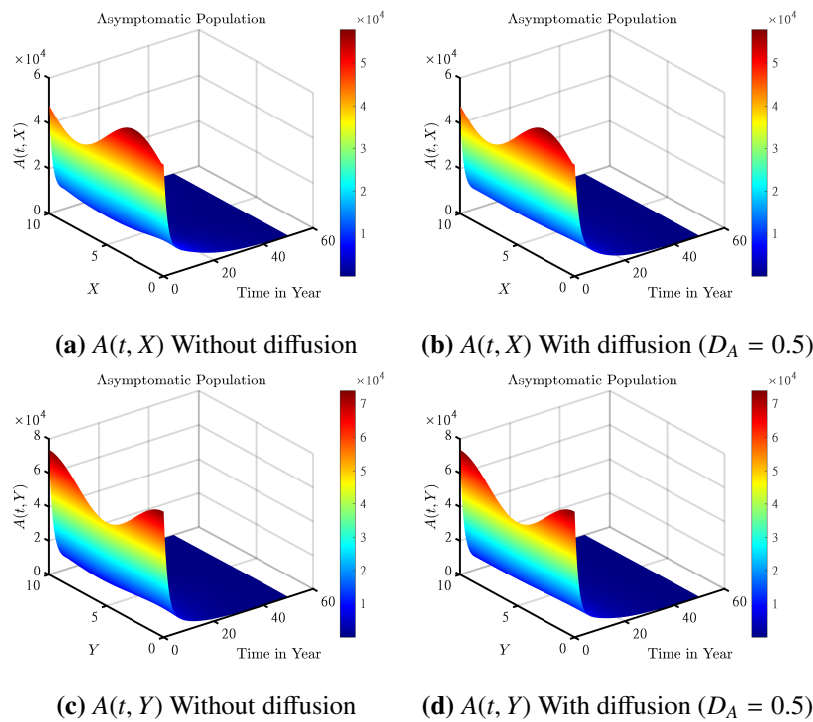


Figure 9. Comparison of the spatiotemporal surface plots for the Asymptomatic population (A). The comparison shows how spatial diffusion (right column) acts to distribute the peak infection density across the domain.

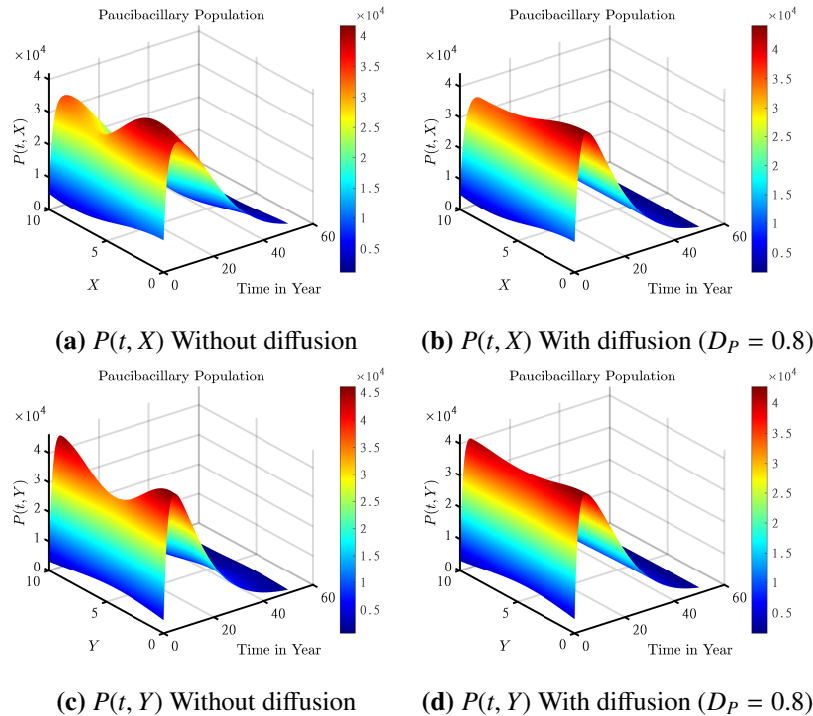


Figure 10. Comparison of the spatiotemporal surface plots for the Paucibacillary population (P). The comparison highlights how the relatively high diffusion coefficient ($D_P = 0.8$) strongly mitigates localized clustering.

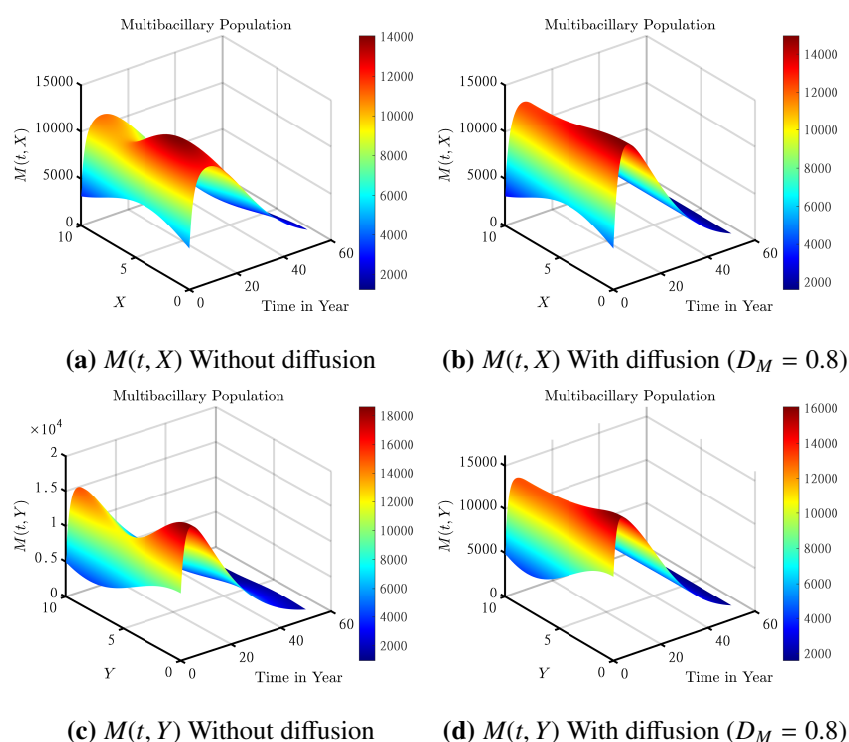


Figure 11. Comparison of the spatiotemporal surface plots for the Multibacillary population (M). Comparing the left (nondiffusive) and right (diffusive) panels demonstrates the transition from isolated peaks to a continuous spatial spread.

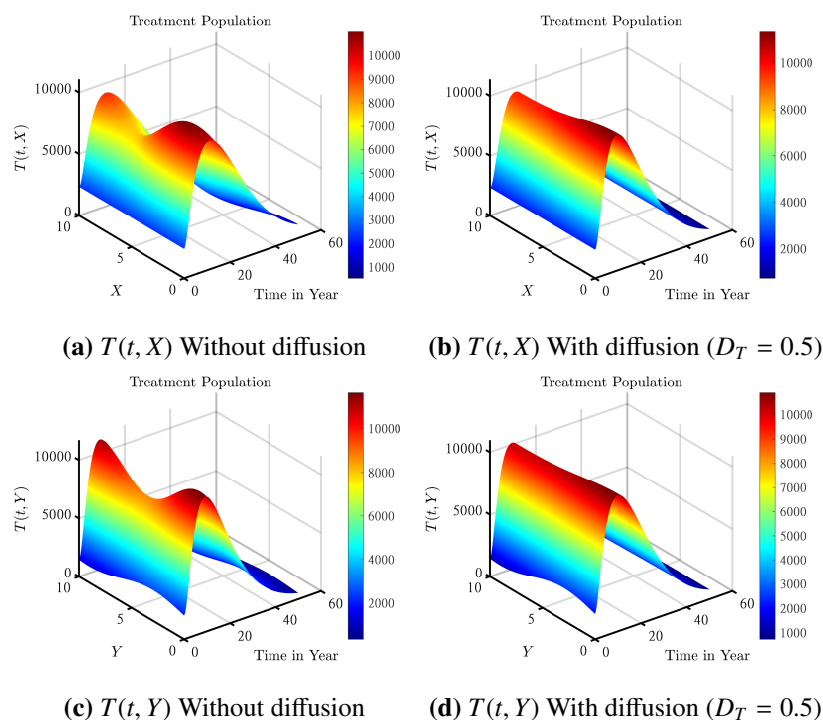


Figure 12. Comparison of the spatiotemporal surface plots for the Treatment population (T). The comparison indicates how the administration and progression of treatment are distributed spatially with and without diffusion.

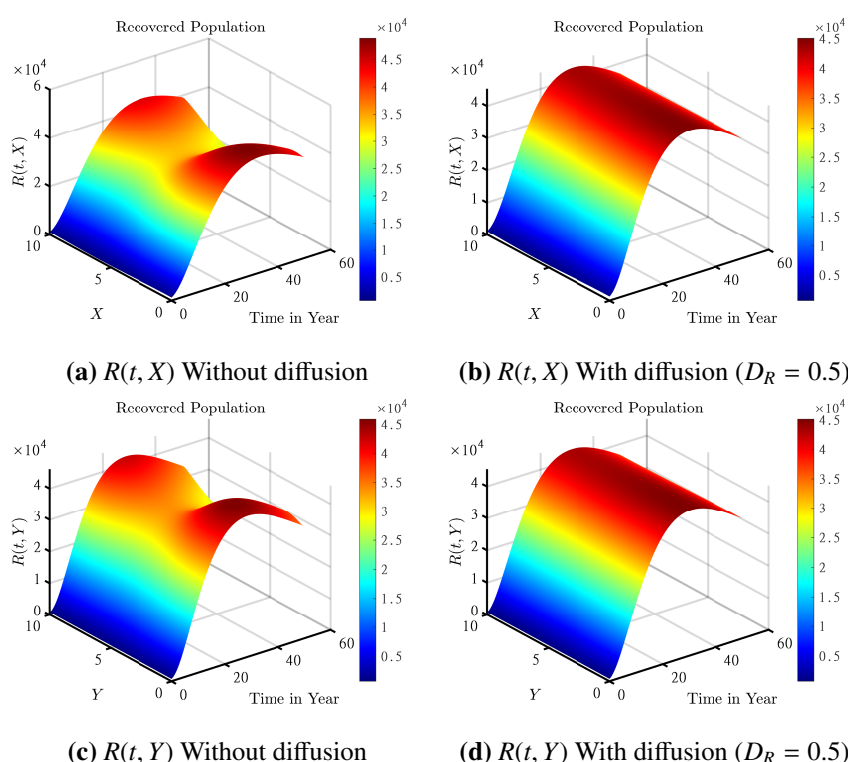


Figure 13. Comparison of the spatiotemporal surface plots for the Recovered population (R). The comparison shows that the spatial spread of recovery naturally follows the diffusive patterns of the infected and treated compartments.

7.4. Limitations

Although the proposed two-dimensional reaction-diffusion framework provides valuable insights into the spatiotemporal propagation of leprosy, several modeling and methodological limitations should be highlighted. First, the assumption of isotropic Fickian diffusion treats population mobility as undirected random dispersal with spatially uniform diffusion coefficients; however, real-world human movement is shaped by transportation networks, geographic barriers, and socioeconomic gradients. Second, the spatial domain is idealized as a rectangular geometry $\Omega = [0, L_x] \times [0, L_y]$ for mathematical tractability, whereas actual endemic territories possess irregular boundaries and topographical variations that can influence boundary flux dynamics. Third, the epidemiological transmission, progression, and treatment parameters are assumed constant in both space and time, although in practical public health contexts, they may vary seasonally, geographically, or in response to dynamic interventions. Fourth, the formulation is strictly deterministic and does not incorporate demographic stochasticity or environmental noise, which can be influential during early-stage localized outbreaks or fade-out phenomena [25]. Finally, although the present numerical experiments demonstrate the fundamental qualitative mechanisms of spatial dispersal, future studies incorporating fine-scale georeferenced surveillance datasets from high-burden regions will be essential for field-level parameter estimation and empirical validation.

8. Conclusions

In this study, we extended the six-compartment (SAPMTR) model of leprosy transmission proposed by Kubra et al. [14] into a two-dimensional reaction–diffusion PDE framework by incorporating Fickian diffusion over a bounded geographic domain. This spatial extension accounts for the movement of individuals across all epidemiological stages (susceptible, asymptomatic, paucibacillary, multibacillary, treatment, and recovered). We developed and analyzed two numerical schemes: the explicit forward Euler finite difference method, for which rigorous CFL stability and positivity-preserving conditions were established, and the Crank–Nicolson-operator splitting method, which handles diffusion implicitly in alternating spatial directions.

The basic reproduction number R_L was derived using the next generation matrix technique, yielding $R_L \approx 1.5582 > 1$ for the baseline parameter regime, which confirms the existence of a unique endemic equilibrium. The local asymptotic stability of this endemic state was established numerically via the Routh–Hurwitz criteria for the spatially homogeneous mode and extended to all spatial wavenumbers through the stabilizing negative diagonal contribution of diffusion.

Extensive two-dimensional simulations performed in MATLAB demonstrated the critical role of human mobility in disease transmission. In the absence of spatial diffusion, localized initial infections remain confined to steep, isolated endemic clusters. In contrast, when diffusion is present, spatial dispersal smooths concentration peaks and spreads the disease burden across the geographic domain, transforming localized outbreaks into widespread regional endemicity. Compartments with higher mobility (paucibacillary and multibacillary infectious classes) drive rapid spatial propagation. These findings highlight that public health interventions, such as active case finding and mobile multi-drug therapy (MDT) units, must not be restricted to initial hotspot areas but should adopt broad, coordinated geographic coverage to effectively mitigate transmission.

Several directions for future research naturally emerge from this work. First, future studies should focus on empirical validation and parameter calibration by fitting the spatiotemporal model against longitudinal georeferenced surveillance data from high-burden countries (such as India, Brazil, and Indonesia). Second, incorporating spatially heterogeneous coefficients such as space-dependent transmission rates $\alpha(x, y)$ and advection terms will allow the model to account for urban–rural population density gradients and directed transportation corridors. Third, formulating optimal spatial control problems can guide public health authorities in the optimal dynamic allocation of mobile medical units, contact tracing teams, and targeted chemoprophylaxis. Fourth, extending the deterministic framework to stochastic reaction–diffusion systems will enable the investigation of demographic noise, random transmission events, and epidemic fade-out phenomena in low prevalence settings [26]. Finally, developing higher-order numerical algorithms, such as Strang splitting or implicit–explicit (IMEX) Runge–Kutta schemes, will provide full second-order temporal accuracy while preserving computational efficiency.

Author contributions

Amir Khan led the technical development (conceptualization, methodology, software, validation, formal analysis) and wrote the original draft. Shafiullah handled manuscript revision, visualization, and supervision. Awatif J. Alqarni managed project administration, funding acquisition, and resources.

Use of Generative-AI tools declaration

The authors declare that no Artificial Intelligence (AI) tools were used in the creation, preparation, analysis, or writing of this article.

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Conflict of interest

All authors declare no conflicts of interest in this paper.

References

1. World Health Organization, Global leprosy update 2023, 2023.
2. D. Ridley, W. Jopling, Classification of leprosy according to immunity: A five-group system, *Int. J. Leprosy*, **34** (1966), 255–273. <https://doi.org/10.23880/cdoaj-16000187>
3. S. L. Walker, Leprosy, *Clin. Dermatol.*, **25** (2007), 165–172.
4. World Health Organization, Global leprosy update 2024, 2024.
5. M. Fischer, Leprosy—an overview of clinical features, diagnosis, and treatment, *JDDG*, **15** (2017), 801–827. <https://doi.org/10.1111/ddg.13301>
6. D. J. Blok, S. J. De Vlas, E. A. J. Fischer, J. H. Richardus, Mathematical modelling of leprosy and its control, *Adv. Parasit.*, **87** (2015), 33–51. <https://doi.org/10.1016/bs.apar.2014.12.002>
7. W. Britton, D. Lockwood, Leprosy, *Lancet*, **363** (2004), 1209–1219. [https://doi.org/10.1016/S0140-6736\(04\)15952-7](https://doi.org/10.1016/S0140-6736(04)15952-7)
8. F. Brauer, C. Castillo-Chavez, Z. Feng, *Mathematical models in epidemiology*, New York: Springer, 2019. <https://doi.org/10.1007/978-1-4939-9828-9>
9. M. Martcheva, *An introduction to mathematical epidemiology*, New York: Springer, 2015. <https://doi.org/10.1007/978-1-4899-7612-3>
10. A. Meima, W. C. S. Smith, G. J. van Oortmarssen, J. H. Richardus, J. D. F. Habbema, The future incidence of leprosy: A scenario analysis, *Bull. World Health Organ.*, **82** (2004), 373–380.
11. S. Mushayabasa, C. P. Bhunu, Modelling the effects of chemotherapy and relapse on the transmission dynamics of leprosy, *Math. Sci.*, **6** (2012), 12. <https://doi.org/10.1186/2251-7456-6-12>
12. D. J. Blok, S. J. de Vlas, E. A. J. Fischer, J. H. Richardus, Mathematical modelling of leprosy and its control, *Adv. Parasitol.*, **87** (2015), 33–51. <https://doi.org/10.1016/bs.apar.2014.12.002>
13. A. Raza, J. Awrejcewicz, M. Rafiq, N. Ahmed, M. S. Ehsan, M. Mohsin, Dynamical analysis and design of computational methods for nonlinear stochastic leprosy epidemic model, *Alex. Eng. J.*, **61** (2022), 8097–8111. <https://doi.org/10.1016/j.aej.2022.01.035>

14. K. T. Kubra, R. Ali, B. Ujala, S. Gulshan, T. Rasool, M. R. Ali, Exploring the dynamics of leprosy transmission with treatment through a fractal–fractional differential model, *Partial Differ. Equ. Appl. Math.*, **12** (2024), 100909. <https://doi.org/10.1016/j.padiff.2024.100909>
15. J. D. Murray, *Mathematical biology I: An introduction*, New York: Springer, 2002. <https://doi.org/10.1007/b98868>
16. R. S. Cantrell, C. Cosner, *Spatial ecology via reaction-diffusion equations*, Chichester: John Wiley & Sons, 2003. <https://doi.org/10.1002/0470871296>
17. S. Riley, Large-scale spatial-transmission models of infectious disease, *Science*, **316** (2007), 1298–1301. <https://doi.org/10.1126/science.1134695>
18. J. G. Barreto, M. A. C. Frade, F. B. Filho, M. B. da Silva, J. S. Spencer, C. G. Salgado, Leprosy in children, *Curr. Infect. Dis. Rep.*, **19** (2017), 23. <https://doi.org/10.1007/s11908-017-0577-6>
19. M. L. F. Penna, M. L. Van Del Rey de Oliveira, G. O. Penna, The epidemiological behaviour of leprosy in Brazil, *Leprosy Rev.*, **80** (2009), 332–344. <https://doi.org/10.47276/lr.80.3.332>
20. J. D. Murray, E. A. Stanley, D. L. Brown, On the spatial spread of rabies among foxes, *Proc. R. Soc. B*, **229** (1986), 111–150. <https://doi.org/10.1098/rspb.1986.0078>
21. W. Wang, X. Q. Zhao, Basic reproduction numbers for reaction-diffusion epidemic models, *SIAM J. Appl. Dyn. Syst.*, **11** (2012), 1652–1673. <https://doi.org/10.1137/120872942>
22. N. Ahmed, A. Elsonbaty, A. Raza, M. Rafiq, W. Adel, Numerical simulation and stability analysis of a novel reaction–diffusion COVID-19 model, *Nonlinear Dyn.*, **106** (2021), 1293–1310. <https://doi.org/10.1007/s11071-021-06623-9>
23. A. Naheed, M. Singh, D. Lucy, Numerical study of SARS epidemic model with the inclusion of diffusion in the system, *Appl. Math. Comput.*, **229** (2014), 480–498. <https://doi.org/10.1016/j.amc.2013.12.062>
24. A. Viguerie, G. Lorenzo, F. Auricchio, D. Baroli, T. J. R. Hughes, A. Patton, et al., Simulating the spread of COVID-19 via a spatially-resolved susceptible-exposed-infected-recovered-deceased model with heterogeneous diffusion, *Appl. Math. Lett.*, **111** (2021), 106617. <https://doi.org/10.1016/j.aml.2020.106617>
25. W. Ali, C. E. Overton, R. R. Wilkinson, K. J. Sharkey, Deterministic epidemic models overestimate the basic reproduction number of observed outbreaks, *Infect. Dis. Model.*, **9** (2024), 680–688. <https://doi.org/10.1016/j.idm.2024.02.007>
26. V. E. Papageorgiou, G. Tsaklidis, A stochastic particle extended SEIRS model with repeated vaccination: Application to real data of COVID-19 in Italy, *Math. Method. Appl. Sci.*, **47** (2024), 6504–6538. <https://doi.org/10.1002/mma.9934>
27. N. Ahmed, S. S. Tahira, M. Rafiq, M. A. Rehman, M. Ali, M. O. Ahmad, Positivity preserving operator splitting nonstandard finite difference methods for SEIR reaction diffusion model, *Open Math.*, **17** (2019), 313–330. <https://doi.org/10.1515/math-2019-0027>
28. T. Fujimoto, R. R. Ranade, Two characterizations of inverse-positive matrices: The Hawkins–Simon condition and the Le Chatelier–Braun principle, *Electron. J. Linear Algebra*, **11** (2004), 59–65. <https://doi.org/10.13001/1081-3810.1122>

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29. W. Hundsdorfer, J. Verwer, *Numerical solution of time-dependent advection-diffusion-reaction equations*, Berlin: Springer, 2003. <https://doi.org/10.1007/978-3-662-09017-6>
30. K. W. Morton, D. F. Mayers, *Numerical solution of partial differential equations: An introduction*, 2 Eds, Cambridge: Cambridge University Press, 2005. <https://doi.org/10.1017/CBO9780511812248>



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