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Stability properties of an epidemic model with cross-diffusion

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Abstract: Mathematical models play a crucial role in understanding the dynamics of epidemics. Reaction-diffusion systems provide a powerful framework for modeling epidemics. We study a spatio-temporal SI epidemic model that includes both self-diffusion and cross-diffusion. The basic reproductive ratio is calculated. The asymptotic stability and Turing instability of both disease-free and endemic equilibria are investigated. We found that the stability properties of both equilibria are preserved in the diffusive model. We investigate the dynamics of the model numerically using a finite difference scheme. We perform numerical simulations for different parameter settings. The simulation results are consistent with the analytical study.

Keywords: SI epidemic model; disease-free equilibrium;endemic equilibrium; stability analysis;reaction-diffusion equations; cross-diffusion; Turing instability

1 Introduction

Epidemics have significant negative impact on public health, socio-economic factors and global health security. Outbreaks of infectious diseases can lead to severe illness, disability and death. Moreover, epidemics can put a strain on healthcare systems. The economic impact of epidemics can be significant, including reduced productivity, increased healthcare expenditures and disruption to trade and travel. The COVID-19 pandemic is the major health threat the whole world has faced in recent years. More than 774 million cases of COVID-19 have been reported in more than 230 countries and more than 7 million deaths [1]. It is important to study the dynamics of an epidemic and how to avoid reaching an endemic situation.

Mathematical models [2–5] provide a powerful tool to understand and predict the dynamics of epidemics. Mathematical models can capture the complex interactions among ineffective agents, hosts and the environment. Mathematical models can simulate the spread of an infection and evaluate the impact of different control measures. Models can help decision-makers and public health officials to make evidence-based decisions, such as implementing vaccinations, campaigns, social distancing measures and treatment protocols. Compartmental models [5–8] are widely used to study the dynamics of infectious diseases. These models classify the population into different compartments based on their disease status. In general, the population is divided into susceptible (S), infective (I) and recovered (R) individuals. These models allow researchers to quantify the spread and control of infectious diseases.

Equilibrium points in epidemic models correspond to steady states in which the prevalence of the disease remains constant over time. Investigating the existence and stability properties of equilibrium points [4, 7] is crucial for understanding the long-term behaviour of an epidemic. Stability analysis of equilibrium points provides insight into important epidemiological quantities such as the basic reproductive ratio [9–11], R_0, which measures the average number of secondary infections generated by a single ineffective individual in a susceptible population.

The disease-free equilibrium of an epidemic model represents a state in which there are no infected individuals in the population. It provides insight into the conditions required for disease elimination. Analyzing the stability properties of the disease-free equilibrium can help in determining thresholds for control measures that must be surpassed to prevent an epidemic. The endemic equilibrium reflects a state in which the infection persists

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in the population indefinitely. Investigating the stability properties of the endemic equilibrium provides insight into the long-term behaviour of the epidemic. Additionally, the endemic equilibrium can help in evaluating the impact of control measures and interventions on the overall burden of an infection.

Temporal models often assume a well-mixed population and neglect spatial heterogeneity and interactions among different compartments. However, spatial aspects and interactions among subpopulations play a crucial role in the spread of infectious diseases [12, 13]. Considering both interaction and spatial heterogeneity requires the framework of reaction-diffusion (RD) systems.

Systems of RD equations are often employed to model the spatio-temporal spread of infectious diseases [13–15]. The diffusion term is a component of spatial heterogeneity that allows for the dispersion and transport of individuals. The RD models offer insight into how spatial factors affect disease transmission and can help in the development of control strategies. Although RD models are valuable tools for studying the spatial spread of infectious diseases, most of them only consider self-diffusion, where individuals can diffuse individually within each compartment. It is also important to include cross-diffusion [16–18] which describes the influence of one compartment on the movement of another. The importance of including both self-diffusion and cross-diffusion in epidemic models arises from the need to capture complex spatial interactions among different compartments.

Turing instability [16, 19, 20] is an important aspect of RD systems. It describes the diffusion-driven instability. Turing instability means the conditions under which small perturbations can grow and form spatial patterns. The presence of Turing instability can lead to the formulation of complex spatial structures that are important for various natural systems [16, 20]. In the context of epidemic modeling, Turing instability is often used to study the spatial spread of infectious diseases [21, 22]. Analyzing Turing instability can improve our understanding of how spatial dynamics influence the spread of disease and the effectiveness of epidemic control measures. This can help policy makers to identify the most effective strategies to contain the spread of infectious diseases.

Elgazzar [8] investigated a temporal SIRS epidemic model taking into account social distancing and community awareness. The study showed that a sufficient level of social distancing, based on R_0, effectively keeps the infection under control even without a vaccine. When a vaccine is available, social distancing can minimize the vaccination rate required to control the disease. Community awareness plays a critical role in eradicating transmission of the infection. Mistakes due to low awareness in the community, such as non-compliance with preventive measures, can hinder control efforts.

Wang et al. [21] investigated the complex dynamics of an SI-RD epidemic model and analyzed its equilibria, boundedness, dissipation and persistence. In addition, they obtained the conditions of Turing instability. Cai and Wang [23] studied the spatio-temporal dynamics of an IR-RD epidemic model with a nonlinear incidence rate. The boundedness, dissipation and stability of positive equilibria were analyzed. Cai et al. [24] investigated the stability of steady states in an SIS epidemic model considering intervention strategies in a spatially heterogeneous environment. They found that, first, R_0 plays a crucial role in determining the persistence or extinction of the disease. Second, intervention strategies play a crucial role in implementing efficient interventions to control disease spread. In Ref. [25], the behavior of an SIR-RD epidemic model with a nonlinear incidence rate was investigated. The stability analysis shows that the disease-free equilibrium is asymptotically stable when $R_0 \leq 1$, which leads to disease eradication. However, when $R_0 > 1$, the disease-free equilibrium becomes unstable and the endemic equilibrium becomes asymptotically stable, indicating the persistence of the disease. Deng and Wu [26] studied an SIS-RD epidemic model. The disease-free equilibrium is found to be globally attractive when $R_0 \leq 1$. Under certain conditions, the endemic equilibrium is globally attractive when $R_0 > 1$.

Li et al. [27] investigated a diffusive SIS epidemic model with cross-diffusion. The study found that the model exhibits threshold-like dynamics based on R_0 even in the presence of cross-diffusion. When $R_0 < 1$, the disease-free equilibrium is stable. However, when $R_0 > 1$, the disease persists and an endemic equilibrium exists, which can be stable under certain conditions. Hu and Wang [28] investigated the dynamics of an SIRS-RD epidemic model with cross-diffusion. They only considered the diffusion of S-individuals away from a higher concentration of I-individuals. They found that a disease cannot be contained if only the diffusion of S-individuals is controlled. Triska et al. [22] studied the effect of cross-diffusion on the Turing instability.

Our aim is to analytically and numerically investigate the stability properties of both disease-free and endemic equilibria of a diffusive SI epidemic model. Our model include both self-diffusion and cross-diffusion. We consider cross-diffusion of both S and I individuals. We study the spatio-temporal dynamics of the model in both cases of absence and presence of diffusion. The structure of the study is as follows. Section 2 gives an overview of the model formulation and the calculation of the basic reproductive ratio. In section 3, we analytically investigate the local asymptotic stability of the equilibria of the model in the absence / presence of diffusion. The Turing instability is analyzed in Section 4. Section 5 is devoted to numerical simulations, where the spatio-temporal dynamics of the model are studied under different settings of the model parameters. Some conclusions are summarized in Section 6.

2 The model

Consider a sufficiently large population located in a bounded domain Ω, with smooth boundary $\partial\Omega$. Let Λ be the number of births and μ be the natural mortality rate. When an infection occurs, the population is categorized into two compartments: susceptible and infective. Let $S(x,t)$ and $I(x,t)$ be the fraction of susceptible individuals and infective individuals, respectively at position $x \in \Omega$ and time t. Let β be the rate of infection due to direct contact between infective and susceptible individuals and γ be the rate at which infective individuals are removed. We assume that no infection can transmitted across the boundary $\partial\Omega$. Hence homogeneous Neumann boundary conditions are applied. We consider two types of diffusion. The first is self-diffusion with coefficients d_1 and d_2 for susceptible and infective individuals, respectively. The second is cross-diffusion of susceptible individuals due to the presence of infective individuals with coefficient d_{12} and infective individuals due to the presence of susceptible individuals with coefficient d_{21}. Susceptible individuals tend to diffuse in the direction of lower density of infective individuals (positive cross-diffusion). While infection diffuses in the direction of high density of susceptibles (negative cross diffusion). Therefore, the model is described by the following dynamical system

$$\begin{aligned}
\frac{\partial S(x,t)}{\partial t} - d_1\Delta S(x,t) - d_{12}\Delta I(x,t) &= \Lambda - \beta S(x,t)I(x,t) \\
-\mu S(x,t) &= f_1(S,I), \\
\frac{\partial I(x,t)}{\partial t} - d_2\Delta I(x,t) + d_{21}\Delta S(x,t) &= \beta S(x,t)I(x,t) \\
-\gamma I(x,t) &= f_2(S,I), \\
\frac{\partial S(x,t)}{\partial \mathbf{n}} = \frac{\partial I(x,t)}{\partial \mathbf{n}} &= 0,
\end{aligned} \tag{1}$$

where $\mathbf{n}$ is the outwards unit vector normal to $\partial\Omega$. The initial conditions are assumed to be

$$S(x,0) = S_0(x) > 0, I(x,0) = I_0(x) \geq 0.$$

It is clear that

$$f_1(0,I) = \Lambda > 0, f_2(S,0) = 0, \forall S, I \geq 0.$$

This ensures the positivity of the solutions of system (1).

Without spatial effects, system (1) has a disease-free equilibrium, $E^0 = (\frac{\Lambda}{\mu}, 0)$. The basic reproductive ratio [9, 10], R_0 is a fundamental concept in epidemic models. It determines the expected number of secondary infections produced by an infective individual. Here, we use the next generation matrix method [9] to determine R_0 of system (1). In the diffusionless case, system (1) is written as follows

$$\frac{dU}{dt} = F(U) - V(U), U = \begin{bmatrix} S \\ I \end{bmatrix}, \tag{2}$$

where F_i is the appearance rate of new infective individuals in compartment i, and

$$V(U) = V^-(U) - V^+(U), \tag{3}$$

where V^- is the transfer rate of individuals out of compartment i, and V^+ is the transfer rate of individuals into compartment i. By applying to system (1), then

$$\frac{dU}{dt} = \begin{bmatrix} 0 \\ \beta SI \end{bmatrix} - \begin{bmatrix} \beta SI + \mu S - \Lambda \\ \gamma I \end{bmatrix}. \tag{4}$$

The Jacobian matrices of F and V are calculated at E^0, then

$$J(F) = \begin{bmatrix} 0 & 0 \\ 0 & \beta\Lambda/\mu \end{bmatrix}, J(V) = \begin{bmatrix} \mu & \beta\Lambda/\mu \\ 0 & \gamma \end{bmatrix}. \tag{5}$$

The next generation matrix is

$$J(F)(J(V))^{-1} = \begin{bmatrix} 0 & 0 \\ 0 & \beta\Lambda/\mu\gamma \end{bmatrix}. \tag{6}$$

Then, the basic reproductive ratio is

$$R_0 = \rho[J(F)(J(V))^{-1}] = \frac{\beta\Lambda}{\mu\gamma}, \tag{7}$$

where $\rho(A)$ is the spectral radius of the square matrix A. Since there is only one infective compartment in system (1), then system (1) has the same R_0 of the spatially homogeneous mode [11]. In the next sections, we investigate the stability properties of both disease-free and endemic equilibria.

3 Local asymptotic stability

In the absence of diffusion, system (1) has two equilibria. The first is the disease-free equilibrium, $E^0 = (\Lambda/\mu, 0)$. The second is the endemic equilibrium, $E^* = (\gamma/\beta, (\Lambda\beta - \mu\gamma)/\gamma\beta)$, provided that $R_0 > 1$. The following theorem determines the local asymptotic stability of these equilibria.

Theorem 1. *In the absence of diffusion, the following statements hold for system (1):*
(i) *The disease-free equilibrium, E^0 is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.*
(ii) *The endemic equilibrium, E^* is locally asymptotically stable when reached.*

Proof. In the diffusionless case, the Jacobin matrix of system (1) is given as follows

$$J(S,I) = \begin{bmatrix} -\beta I - \mu & -\beta S \\ \beta I & \beta S - \gamma \end{bmatrix}. \tag{8}$$

(i) For the disease-free equilibrium,

$$J(E^0) = \begin{bmatrix} -\mu & -\Lambda\beta/\mu \\ 0 & (\Lambda\beta/\mu) - \gamma \end{bmatrix}. \quad (9)$$

It is clear that if $R_0 < 1$, $det(J(E^0)) > 0$ and $tr(J(E^0)) < 0$, leading to local asymptotic stability. Oppositely, if $R_0 > 1$, $det(J(E^0)) < 0$, leading to instability.
(ii) For the endemic equilibrium,

$$J(E^*) = \begin{bmatrix} (-1/\gamma)(\Lambda\beta - \mu\gamma) - \mu & -\gamma \\ (1/\gamma)(\Lambda\beta - \mu\gamma) & 0 \end{bmatrix}. \quad (10)$$

Since E^* is reached only when $R_0 > 1$, then $det(J(E^*)) > 0$, and $tr(J(E^*)) < 0$. Therefore the endemic equilibrium is locally asymptotically stable when reached.

□

The following theorem determines the effect of both self-diffusion and cross-diffusion on the local asymptotic stability of equilibria of system (1).

Theorem 2.*In the presence of both self-diffusion and cross-diffusion, system (1) satisfies the following statements*
(i) *If $R_0 < 1$, the disease-free equilibrium, E^0 is locally asymptotically stable.*
(ii) *If $R_0 > 1$, the endemic equilibrium, E^* is locally asymptotically stable.*

Proof. In the presence of both self-diffusion and cross-diffusion, an equillibrium satisfies the following equations

$$d_1 \triangle S + d_{12} \triangle I + \Lambda - \beta SI - \mu S = 0,$$

$$d_2 \triangle I - d_{21} \triangle S + \beta SI - \gamma I = 0. \quad (11)$$

Let the infinite sequence of eigenvalues of the operator $-\triangle$ be $0 = \lambda_0 < \lambda_1 \leq \lambda_1 \leq \lambda_2 \leq ... \to \infty$, where λ_i has algebraic multiplicity $m_i \geq 1$. Let $(\Phi_{ij})_{j=1,...,m_i}$ be the corresponding sequence of normalized eigenfunctions. The set $(\Phi_{ij})_{j=1,...,m_i}$ forms a complete orthonormal basis in the space of square integrable functions on Ω with Neumann boundary conditions. By linearizing system (11) around an equilibrium point, $E^{\sim} = (S^{\sim}, I^{\sim})$, then the linearizing operator is given by

$$L(E^{\sim}) = \begin{bmatrix} d_1 \triangle - \beta I^{\sim} - \mu & d_{12} \triangle - \beta S^{\sim} \\ -d_{21} \triangle + \beta I^{\sim} & d_2 \triangle + \beta S^{\sim} - \gamma \end{bmatrix}. \quad (12)$$

Let ζ be an eigenvalue of $L(E^{\sim})$, and the corresponding eigenfunction be $(\phi(x), \psi(x))$, then

$$(L - \zeta I) \begin{bmatrix} \phi(x) \\ \psi(x) \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \end{bmatrix}. \quad (13)$$

For simplicity, let

$$\phi = \sum_{i \geqslant 0, 1 \leqslant j \leqslant m_i} a_{ij} \Phi_{ij}, \psi = \sum_{i \geqslant 0, 1 \leqslant j \leqslant m_i} b_{ij} \Phi_{ij}. \quad (14)$$

then Eq. (13) can be written in the following form

$$\sum_{i \geqslant 0, i \leqslant j \leqslant m_i} (J_i(E^{\sim}) - \zeta I) \begin{bmatrix} a_{ij} \\ b_{ij} \end{bmatrix} \Phi_{ij} = \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \quad (15)$$

where

$$J_i(E^{\sim}) = \begin{bmatrix} -d_1\lambda_i - \beta I^{\sim} - \mu & -d_{12}\lambda_i - \beta S^{\sim} \\ d_{21}\lambda_i + \beta I^{\sim} & -d_2\lambda_i + \beta S^{\sim} - \gamma \end{bmatrix}, i \geq 0. \quad (16)$$

(i) For the disease-free equilibrium with $R_0 < 1$,

$$J_i(E^0) = \begin{bmatrix} -d_1\lambda_i - \mu & -d_{12}\lambda_i - (\Lambda\beta)/\mu) \\ d_{21}\lambda_i & -d_2\lambda_i + (\Lambda\beta)/\mu) - \gamma \end{bmatrix}, i \geq 0. \quad (17)$$

Then

$$det(J_i(E^0)) = (-d_1\lambda_i - \mu)(-d_2\lambda_i + ((\Lambda\beta)/\mu) - \gamma) + d_{21}\lambda_i(d_{12}\lambda_i + ((\Lambda\beta)/\mu) > 0, \forall i \geq 0,$$

and

$$tr(J_i(E^0)) = (-d_1\lambda_i - \mu - d_2\lambda_i) + ((1/\mu)\Lambda\beta) - \gamma) < 0, \forall i \geq 0.$$

Therefore, the disease-free equillibrium, E^0 remains locally asymptotically stable in the presence of both self-diffusion and cross-diffusion when $R_0 < 1$.
(ii) For the endemic equilibrium with $R_0 > 1$,

$$J_i(E^*) = \begin{bmatrix} -d_1\lambda_i - (1/\gamma)(\Lambda\beta - \mu\gamma) - \mu & -d_{12}\lambda_i - \gamma \\ d_{21}\lambda_i + (1/\gamma)(\Lambda\beta - \mu\gamma) & -d_2\lambda_i. \end{bmatrix}, i \geq 0. \quad (18)$$

Then

$$det(J_i(E^*)) = (d_1\lambda_i + (1/\gamma)(\Lambda\beta - \mu\gamma) + \mu)(d_2\lambda_i) + (d_{12}\lambda_i + \gamma)(d_{21}\lambda_i + (1/\gamma)(\Lambda\beta - \mu\gamma)) > 0, \forall i \geq 0,$$

and

$$tr(J_i(E^*)) = -d_1\lambda_i - (1/\gamma)(\Lambda\beta - \mu\gamma) - \mu - d_2\lambda_i < 0, \forall i \geq 0.$$

Therefore, the endemic equilibrium, E^* is locally asymptotically stable in the presence of both self-diffusion and cross-diffusion with $R_0 > 1$.

□

Therefore, the local asymptotic stability of equilibria of system (1) is preserved even in the presence of both self-diffusion and cross-diffusion. In order to gain a deeper understanding of how spatial heterogeneity affects the stability of equilibria, a Turing instability analysis is performed in the next section.

4 Turing instability

Turing instability [16,20] is a powerful tool for analyzing the stability of spatially homogeneous equilibria in RD systems. The following theorem determines the necessary and sufficient conditions for Turing (diffusion-driven) instability of a stable spatially uniform equilibrium of system (1).

Theorem 3. *A stable spatially uniform equilibrium* $(S^{\sim},I^{\sim})$ *of system* (1) *is Turing instable if*

$$d_1a_{22}+d_2a_{11}-d_{12}a_{21}+d_{21}a_{12}>2\sqrt{(a_{11}a_{22}-a_{12}a_{21})(d_1d_2+d_{12}d_{21})}>0, \tag{19}$$

where

$$a_{ij}=\frac{\partial f_i(S,I)}{\partial u_j}\mid_{(S^{\sim},I^{\sim})},u_1=S,u_2=I. \tag{20}$$

Proof. We examine the linear stability of $(S^{\sim},I^{\sim})$ in the presence of diffusion. Let

$$\omega=\begin{bmatrix}\omega_1\\ \omega_2\end{bmatrix}=\begin{bmatrix}S-S^{\sim}\\ I-I^{\sim}\end{bmatrix} \tag{21}$$

be a small perturbation, and we linearize system (1) around $(S^{\sim},I^{\sim})$. Then

$$\frac{\partial\omega}{\partial t}=J\omega+D\triangle W, \tag{22}$$

where

$$J=\begin{bmatrix}\frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I}\\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial I}\end{bmatrix},D=\begin{bmatrix}d_1 & d_{12}\\ -d_{21} & d_2\end{bmatrix}. \tag{23}$$

Let $\omega_j=\varepsilon_j exp(ikx+\lambda t), j=1,2$, where $\varepsilon_j, j=1,2$ are constants, k is the wave number and λ is the eigenvalue. The eigenvalues are the roots of the characteristic polynomial

$$\begin{vmatrix}\lambda-a_{11}+d_1k^2 & -a_{12}+d_{12}k^2\\ -a_{21}-d_{21}k^2 & \lambda-a_{22}+d_2k^2\end{vmatrix}=0. \tag{24}$$

Then

$$\lambda^2-g(k^2)\lambda+h(k^2)=0, \tag{25}$$

where

$$g(k^2)=-(d_1+d_2)k^2+a_{11}+a_{22}, \tag{26}$$

$$h(k^2)=\mid D\mid k^4-Bk^2+\mid J\mid, \tag{27}$$

$$B=d_1a_{22}+d_2a_{11}-d_{12}a_{21}+d_{21}a_{12}. \tag{28}$$

If $Re\lambda(k)<0$, then the perturbation $\omega\longrightarrow 0$ as $t\longrightarrow\infty$, and $(S^{\sim},I^{\sim})$ is stable to spatial disturbances. Oppositely, if $Re\lambda(k)>0$ for some $k\neq 0$, then $(S^{\sim},I^{\sim})$ is unstable to diffusion. This happens if

$$g(k^2)>0, \tag{29}$$

or

$$h(k^2)<0,\ \text{for some } k\neq 0. \tag{30}$$

Since $(S^{\sim},I^{\sim})$ is stable spatially uniform equilibrium, then $a_{11}+a_{22}<0$. Hence Eq. (29) cannot be satisfied. On the other hand, $h(k^2)$ can be negative only if

$$B>0. \tag{31}$$

Condition (31) is necessary but not sufficient for $h(k^2)<0$ for some $k\neq 0$. The minimum value of $h(k^2)$ should be negative too. By minimizing $h(k^2)$ with respect to k^2, we get

$$k^2_{min}=\frac{B}{2\mid D\mid}, \tag{32}$$

and

$$h_{min}=\mid J\mid-\frac{B^2}{4\mid D\mid}. \tag{33}$$

Then h_{min} is negative if

$$\frac{B^2}{4\mid D\mid}>\mid J\mid, \tag{34}$$

and

$$\mid D\mid>0. \tag{35}$$

By combining conditions (31), (34) and (35), then the condition (19) is obtained.

□

Now we apply Theorem 3 to both the disease-free and the endemic equilibria.

Proposition 1 *For system* (1)*, both the disease-free equilibrium,* E^0 *when* $R_0<1$ *and the endemic equilibrium,* E^* *when* $R_0>1$ *are Turing stable.*

Proof. In system (1),

$$f_1(S,I)=\Lambda-\beta SI-\mu S,\ \ f_2(S,I)=\beta SI-\gamma I. \tag{36}$$

(i) For E^0,

$$a_{11}=-\mu, a_{12}=\tfrac{-\beta\Lambda}{\mu}, a_{12}=0 \text{ and } a_{22}=(\tfrac{\beta\Lambda}{\mu})-\gamma.$$

Since $R_0<1$, then condition (19) is not satisfied, and E^* is Turing stable.
(ii) For E^*,

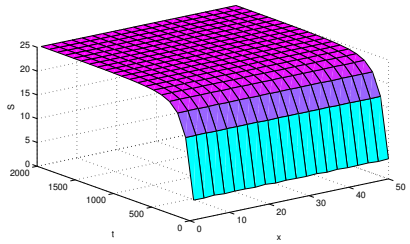
$$a_{11}=(\tfrac{-1}{\gamma})(\Lambda\beta-\mu\gamma)-\mu, a_{12}=-\gamma,$$
$$a_{21}=(\tfrac{1}{\gamma})(\Lambda\beta-\mu\gamma) \text{ and } a_{22}=0.$$

Since $R_0>1$, then condition (19) is not satisfied, and E^* is Turing stable.

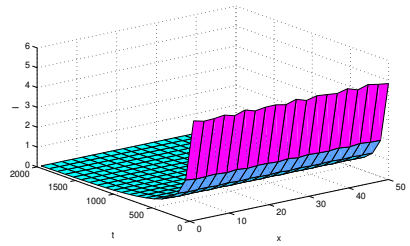
□

Therefore, both disease-free and endemic equilibria of system (1) are Turing Stable. This means that perturbations around an equilibrium of system (1) do not lead to the development of permanent spatial structures, i.e. the system is resistant to the formation of stable spatial patterns. This result suggests that spatial patterns are suppressed, indicating a homogeneous spread of the disease. In this context, the implementation of social distancing measures to reduce contact rates and limit disease transmission can be effective in preventing the formation of spatial patterns and controlling outbreaks [8].

5 Numerical simulations



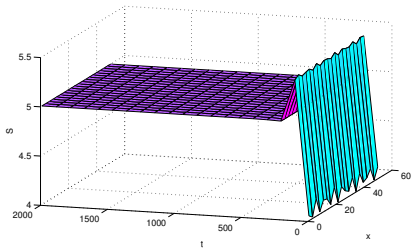
(a)



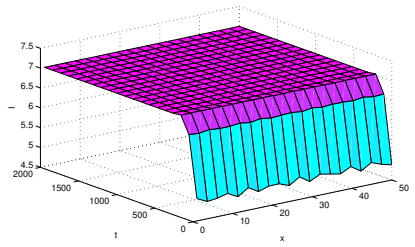
(b)

Fig. 1: (Color online) Spatio-temporal dynamics of the diffusionless case for set 1 of the model parameters: (a) for the susceptible population and (b) for the infective papulation. It is clear that the dynamics of the model converge to the disease-free equilibrium.

In this section, we present numerical simulations of the model (1) to illustrate the analytical results obtained in the previous sections. A finite difference scheme is used. The Laplacian of $S(x,t)$ and $I(x,t)$ are approximated using central differencing formulas. The initial conditions for $S(x,t)$ and $I(x,t)$ are set to $S(x,0) = 4 + \frac{cosx}{10}$ and $I(x,0) = 5 + \frac{sinx}{10}$, respectively, and $\Omega = [0,50]$. Zero-flux boundary conditions are assumed. The model parameters are set to five different sets as shown in Table 1. We investigate the spatio-temporal dynamics of both $S(x,t)$ and $I(x,t)$ for each set of the parameters.



(a)



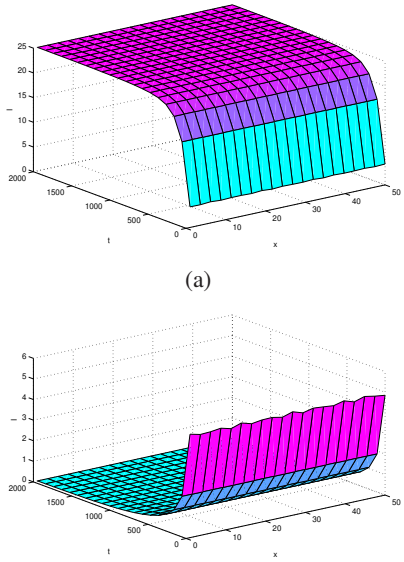
(b)

Fig. 2: (Color online) Spatio-temporal dynamics of the diffusionless case for set 2 of the model parameters: (a) for the susceptible population and (b) for the infective papulation. It is clear that the dynamics of the model converge to the endemic equilibrium.

Table 1: Sets of the model parameters.

set	Λ	β	γ	μ	R_0	d_1	d_2	d_{12}	d_{21}
1	5	0.01	0.3	0.2	5/6	0	0	0	0
2	5	0.1	0.5	0.3	10/3	0	0	0	0
3	5	0.01	0.3	0.2	5/6	2.5	1.0	0	0
4	5	0.1	0.5	0.3	10/3	2.5	1.0	0	0
5	5	0.01	0.3	0.2	5/6	2.5	1.0	2.0	1.5
6	5	0.1	0.5	0.3	10/3	2.5	1.0	2.0	1.5

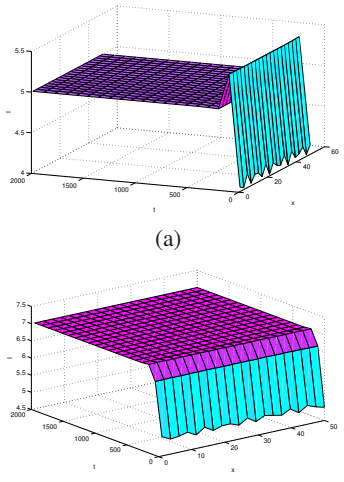
The dynamics of the diffusionless case is illustrated in Fig. 1 for set 1 and Fig. 2 for set 2. As shown in Figs. 1(a) and 1(b), both $S(x,t)$ and $I(x,t)$ go to the stable disease-free equilibrium, E^0, since $R_0 < 1$. In Figs. 2(a) and 2(b) both $S(x,t)$ and $I(x,t)$ go to the stable endemic equilibrium, E^*, since $R_0 > 1$. The same dynamics are observed for the RD system with self-diffusion only, see Fig. 3 for set 3 (the disease-free equilibrium) and Fig. 4 for set 4 (the endemic equilibrium). The stability properties of both E^0 and E^* are preserved in the full diffusive case including both self-diffusion and cross-diffusion. This is clearly observed from Fig. 5 for set 5 and Fig. 6 for set 6. These results are consistent with Theorem 2 and Proposition 1.



(a)

(b)

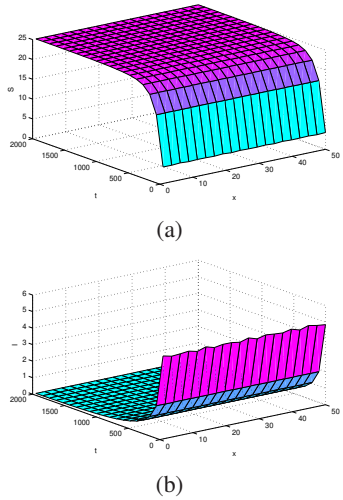
Fig. 3: (Color online) Spatio-temporal dynamics of the model with self-diffusion only for set 3 of the model parameters: (a) for the susceptible population and (b) for the infective papulation. It is clear that the dynamics of the model converge to the disease-free equilibrium.



(a)

(b)

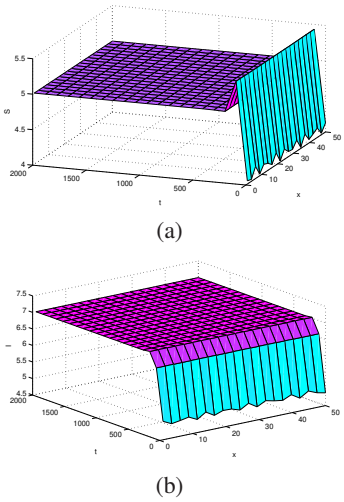
Fig. 4: (Color online) Spatio-temporal dynamics of the model with self-diffusion only for set 4 of the model parameters: (a) for the susceptible population and (b) for the infective papulation. It is clear that the dynamics of the model converge to the endemic equilibrium.



(a)

(b)

Fig. 5: (Color online) Spatio-temporal dynamics of the model including both self-diffusion and cross-diffusion for set 5 of the model parameters: (a) for the susceptible population and (b) for the infective papulation. The dynamics of the model are preserved and converge to the disease-free equilibrium.



(a)

(b)

Fig. 6: (Color online) Spatio-temporal dynamics of the model including both self-diffusion and cross-diffusion for set 6 of the model parameters: (a) for the susceptible population and (b) for the infective papulation. The dynamics of the model are also preserved and converge to the endemic equilibrium.

6 Conclusion

The negative impact of infectious diseases underlines the importance of understanding the dynamics of epidemics and the potential control strategies. Mathematical models play an important role in providing insight into the disease transmission and intervention strategies. Reaction-diffusion equations are a powerful framework

for modeling the spatial spread of infectious diseases. Incorporating both self-diffusion and cross-diffusion in epidemic models provides more realistic representation of epidemic dynamics.

We have studied an SI epidemic model that incorporates both self-diffusion and cross-diffusion. The investigated SI-RD epidemic model shows threshold-like dynamics based on R_0 even in the presence of cross-diffusion. When $R_0 < 1$, the disease-free equilibrium is asymptotically stable and leads to disease eradication. However, when $R_0 > 1$, the endemic equilibrium becomes asymptotically stable, indicating the persistence of the disease. This result is consistent with the results of most temporal [8] and spatio-temporal [24–27] models. Therefore, diffusion does not affect the stability properties of both disease-free and endemic equilibria. A Turing instability analysis is performed and shows that the model is stable against spatial perturbations. Therefore, cross-diffusion acts as a stabilizer, in agreement with Triska et al. [22].

We have applied a finite difference scheme to solve the model equations and investigate the dynamics of both susceptible and infective populations. Simulations have been performed for various parameter settings. The results agree with the analytical calculations and confirm the stability of the model against spatial perturbations. This indicates a homogeneous spread of the disease in the population. Therefore, measures to reduce disease transmission such as social distancing measures can effectively control the spread of the disease and prevent outbreaks. This confirms the important role of social distancing in the control of infectious diseases.

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