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# Modeling precaution, immunity loss and dispersal on disease dynamics: a two-patch SIRS model

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**Abstract**

An SIRS model is developed to account for precautionary measures and immunity loss. The existence and stability of equilibria are studied. It is shown that precautionary measures can induce Hopf bifurcations leading to the occurrence of cyclical behavior. The model is then extended to a two-patch scenario to explore how disease spread patterns are influenced by dispersal (travel). Theoretical analyses establish the stability of the disease-free equilibrium, the basic reproduction number, and thresholds related to travel rates. The persistence of the system, as well as the existence of boundary and endemic equilibria, are also discussed. Using Hopf bifurcation theory, we further examine the interaction between nonlinear incidence functions, travel rates, and precaution delay effects in shaping the stability of the endemic equilibrium. The findings reveal a strong connection between reduced infection rates due to precaution and the emergence of Hopf bifurcations, emphasizing the importance of timely and accurate disease information in curbing the spread of diseases. Additionally, the study highlights the significant impact of different infection force functions on equilibrium stability, underscoring the critical role of precautionary measures in disease transmission mechanisms. The results also show the diverse effects of travel rates on disease spread, suggesting that restricting travel may not always lead to favorable outcomes. This underscores the necessity for governments to consider multiple factors comprehensively in their efforts to prevent and control diseases.

**Mathematics Subject Classification:** 92B05; 92D30; 34K20; 34K18

**Keywords:** SIRS; Patch; Dispersal; Immunity loss; Precaution; Delay

## 1 Introduction

During the outbreak of an infectious disease such as SARS and COVID-19, especially when no vaccines or effective treatments are yet available, non-pharmaceutical interventions (NPIs) are the natural choices in reducing the transmission of the disease [4, 11, 25, 29, 47]. Common NPIs include wearing masks, maintaining social distance, restricting travel, quarantine, isolation, and lockdowns. Studies have demonstrated that NPIs were effective in controlling COVID-19 when case numbers and transmission rates were relatively low. Many studies have demonstrated that timely government intervention

is crucial in controlling the spread of disease, and early action during an outbreak significantly accelerates the transition to a disease-free state [14–16, 37]. Saha et al. [37] demonstrated that government actions, combined with clinical treatments, serve as an effective control pair. Their study illustrated that implementing both strategies together leads to a significant reduction in the disease burden. Dutta et al. [14] highlighted the importance of social and governmental interventions in disease dynamics, emphasizing the value of nonlinear dynamical modeling in epidemiological systems. Their findings showed that government policies and pharmaceutical treatments are seen as the most effective control pair, and their simultaneous implementation significantly reduces the disease burden. Dutta et al. [15] focused on factors such as varying susceptibility, government interventions, social behavior, and public responses. They explored time-based strategies for managing behaviors and treatments by framing the problem as an optimal control challenge. Their analysis of periodic transmission rates revealed the significant impact of vaccination rates and transmission patterns on the long-term dynamics of the disease. Dutta et al. [16] explored the dynamics of transmission of diseases through asymptomatic carriers of the disease to consider a societal and environmental perspective. Their study emphasized the substantial impact that social behavior and governmental action have on disease transmission. However, government intervention and implementing NPIs can be quite challenging in practice due to their economic costs and the influence of social media, which can sometimes disseminate delayed and/or false information.

Timely and accurate media reports can significantly enhance public health knowledge and raise awareness about disease conditions. For example, a study by Chao et al. [8] demonstrated that timely access to public health information from official sources and reduced exposure to new media are beneficial for preventing and controlling COVID-19. Similarly, Iyamu et al. [26] found that individuals using social media are more likely to believe in the effectiveness of masks in preventing the spread of the novel coronavirus compared to those who do not use social media. Wang et al. [45] emphasized that both scientific and non-scientific events can substantially impact health beliefs on Twitter. Additionally, Du et al. [13] highlighted the crucial role of social media in accessing, sharing, and disseminating epidemic information during outbreaks of infectious diseases. This information is vital for raising public awareness of infection risks and for encouraging preventive measures to reduce transmission.

To better understand how media coverage affects the spread of infectious disease, there has been some mathematical modeling work. For instance, Liu et al. [31] used a nonlinear incidence rate function $\beta_0 = \beta e^{-\alpha_1 E - \alpha_2 I - \alpha_3 H}$ to characterize the psychological impact on social behavior for exposed class ($E$), infected class ($I$), and hospitalized class ($H$). Liu and Cui [32] proposed a model using a nonconstant transmission rate $\beta(I) = \beta_1 - \beta_2 \frac{I}{m+I}$. Cui et al. [12] suggested the contact rate as $c(I) = c_1 - c_2 f(I)$, where $c_1$ represents the maximum contact rate between susceptible and infected individuals, $c_2$ represents the reduction in contact rate due to media coverage, and $f(I)$ satisfies the conditions $f(0) = 0, f'(I) \geq 0$, and $\lim_{I\to\infty} f(I) = 1$. Mummert and Weiss [34] studied the impact of social isolation on disease transmission and analyzed the effects of three types of media influence functions (denoted as $f$) and media reporting delays on transmission. Song and Xiao [40] investigated an SIR model that incorporates media delay, employing the nonlinear infectious force $\beta e^{-\alpha I(t-\tau)}$. Their findings suggested that the delayed media significantly impacts the transmission dynamics of infectious diseases. Misra et al. [33] assumed that the growth rate of awareness is

proportional to the number of infected individuals and formulated an *SIAM* model incorporating an awareness variable due to media coverage. Huo and Yang [28] considered factors related to media coverage and integrated the factor $e^{-\alpha M}$ into the characterization of the transmission rate, where this factor represents the extent to which behavioral changes influenced by reading disease-related information impact the transmission rate. Sharma et al. [38] explored the impact of media-driven awareness on reducing disease prevalence through an *SIRS* infectious disease model. Furthermore, Zhou et al. [51] treated the awareness change driven by media coverage as an independent compartment and proposed an ordinary differential equation model parameterized with media data. Their results indicated that the contact rate, transmission probability, and progression rate from the latent period to the symptomatic stage were three key parameters.

The implementation of NPIs, together with the massive media coverage, will greatly raise the awareness of the public about the spread of infection so that the public will become more precautious and less social. Consequently, precaution due to NPIs and media coverage will lead to the behavioral change of the susceptible individuals, and only practically susceptible individuals may make contacts with the infectious individuals and thus become possibly infected [9, 10].

The aforementioned studies have demonstrated that precaution is one important factor that influences the spread, prevention, and control of infectious diseases. For some infectious diseases, such as influenza, COVID-19, and HBV, recovered individuals may only gain temporary immunity, and they may become susceptible again when the immunity is lost [5, 43, 46]. Immunity loss is therefore another important factor to be incorporated into modeling for those infectious diseases, which could result in very complicated disease dynamics [27, 35, 48].

With highly developed transportation and fast growing economy, travel/movements between countries, cities, and communities have become more and more frequent and convenient. Even during the course of an epidemic, banning travel is almost impossible. Thus dispersal is another important factor affecting the spread and control of infectious diseases. Mathematical models concerning different patches via dispersal become the natural choice to explore how dispersal affects disease dynamics. Since Hethcote's work [24] in 1976, which considered a two-patch infectious disease model with population dispersal, numerous studies on patchy infectious diseases have been conducted (see [1–3, 6, 17–21, 30, 36] and the references therein). For example, Saha et al. [36] analyzed the influence of the dispersal of susceptible and recovered individuals on epidemic dynamics using deterministic and fractional order systems in a two-patch environment. They found that higher dispersal towards one patch significantly controls the infection level in the other patch. Their study revealed an increase in the number of recovery cases in both patches through optimal control. Additionally, the implementation of public awareness significantly reduces infection levels, even if people disperse at a comparatively higher rate.

Several disease models incorporating precaution and dispersal have been proposed in the literature. Sun et al. [41] investigated a two-patch SIS model, in which the infection rate $\beta(I_i) = a_i - b_i f_i(I_i)$ was used, where $f_i(I_i)$ represents the reduction in the transmission rate due to media-reported cases in each region. The results indicated that accurate media reporting can alleviate the burden of the epidemic and shorten the duration of disease outbreaks. Sun et al. [42] examined a two-patch SIR model that incorporates travel between

patches and behavioral changes of susceptible individuals due to past disease surveillance information. The analysis suggests that the model demonstrates rich dynamical behavior, influenced by various factors such as patch migration, media surveillance information, and time delays in disease development.

To the best of our knowledge, no work has been carried out to explore the joint impacts of precaution, immunity loss, and dispersal on disease dynamics to help control the spread of infectious diseases. In this work, we will use a two-patch SIRS model to examine how precaution, immunity loss, and dispersal jointly affect the disease dynamics.

The rest of this paper is organized as follows. We propose and study a single-patch SIRS model with precaution in Sect. 2. The model is then extended to a two-patch SIRS model with dispersal between two patches in Sect. 3. Its dynamics is also studied in Sect. 3. Numerical simulations are carried out in Sect. 4. We conclude our work in Sect. 5.

## 2 A single-patch SIRS model and its dynamics

In this section, we present an SIRS (Susceptible-Infectious-Recovered-Susceptible) model that incorporates precaution and immunity loss. The population is divided into three disjoint classes: susceptible ($S$), infectious ($I$), and recovered ($R$). The model assumes that recruitment into the susceptible class occurs at a constant rate $A > 0$. The natural death rate is denoted by $d > 0$, and the disease-induced death rate is $\epsilon \geq 0$. Infectious individuals recover at a constant rate $\gamma > 0$. Additionally, recovered individuals lose their immunity and become susceptible again at a rate $\alpha > 0$. If $\alpha = 0$, the system simplifies to an SIR epidemic model with permanent immunity.

Following the studies by Cui et al. [12] and Sun et al. [41], precaution is characterized by the infection rate $\beta(I) = \hat{\beta} - \tilde{\beta} f(I)$, where $\hat{\beta} \geq \tilde{\beta} > 0$ represents the maximum infection rate and the reduction in infection rate due to precaution, respectively. If the parameter $\tilde{\beta} = 0$, the incidence rate simplifies to a bilinear form, meaning no precautionary measures are present in the model; and as a result, such measures will not influence the model's dynamics. Moreover, we assume that there is a response delay $\tau$, that is, the infection force is modeled by $\beta(I(t-\tau))I$, which depends on the number of infectious individuals at $\tau$ time units ago. Thus, the term $\tau$, along with $\hat{\beta}$, $\tilde{\beta}$, and the function $f(I)$, is used to capture the public's precautionary measures in response to the spread of infection. The model can then be described by the following system:

$$\begin{cases} \frac{dS(t)}{dt} = A - dS(t) - \beta(I(t-\tau))I(t)S(t) + \alpha R(t), \\ \frac{dI(t)}{dt} = \beta(I(t-\tau))I(t)S(t) - (\gamma + \epsilon + d)I(t), \\ \frac{dR(t)}{dt} = \gamma I(t) - \alpha R(t) - dR(t), \end{cases} \tag{2.1}$$

where $\beta(I(t-\tau)) = \hat{\beta} - \tilde{\beta} f(I(t-\tau))$.

Incorporating the notion that people's awareness of disease prevention amplifies as the number of infected individuals increases but the rate of growth for this awareness diminishes as infection cases escalate, we can refine the function $f(I)$ so that $f(0) = 0$, $f'(I) > 0$, $f''(I) \leq 0$, and $\lim_{I\to\infty} f(I) = 1$. Then $\beta(0) = \hat{\beta}$, $\beta'(I) = -\tilde{\beta} f'(I) < 0$, $\beta''(I) = -\tilde{\beta} f''(I) \geq 0$, and

$\lim_{I\to\infty}\beta(I)=\hat{\beta}-\tilde{\beta}$. Typical $f(I)$ may have the following forms:

$$f(I)=1-e^{-hI}, \tag{2.2}$$

$$f(I)=\frac{I^n}{k+I^n}, n>0. \tag{2.3}$$

Let $\mathcal{C}=C([-\tau,0],\mathbb{R})$ be the Banach space consisting of all continuous functions defined on $[-\tau,0]$. Its positive cone is $\mathcal{C}^+=\{\phi\in\mathcal{C}:\phi(\theta)\geq 0,\theta\in[-\tau,0]\}$, where $\phi_t\in\mathcal{C}$ is defined as $\phi_t(\theta)=\phi(t+\theta)$. The associated initial condition is

$$\phi=(\phi_1,\phi_2,\phi_3)\in X:=\mathcal{C}^+\times\mathcal{C}^+\times\mathcal{C}^+. \tag{2.4}$$

**Theorem 2.1** *System* (2.1), *subject to the initial condition* (2.4), *globally possesses a unique solution. Furthermore, the variables* $S(t)$, $I(t)$, *and* $R(t)$ *remain nonnegative for* $t\geq 0$. *Additionally, the total population* $N(t)=S(t)+I(t)+R(t)$ *is bounded and falls within the interval* $[\min\{\frac{A}{d+\epsilon},N(0)\},\max\{\frac{A}{d},N(0)\}]$.

The proof is standard and is thus omitted.

For system (2.1), the disease-free equilibrium (DFE) is $E_0=(\frac{A}{d},0,0)$. By the next generation matrix method [44, 50], we find the basic reproduction number

$$\mathcal{R}_0^1=\frac{\hat{\beta}A}{d(\gamma+\epsilon+d)}. \tag{2.5}$$

**Theorem 2.2** *The DFE* $E_0$ *is globally asymptotically stable* (*GAS*) *if* $\mathcal{R}_0^1<1$, *and it is unstable if* $\mathcal{R}_0^1>1$.

The proof is similar to [9, Theorem 3.1] and is given in Appendix A.

**Theorem 2.3** *If* $\mathcal{R}_0^1>1$, *then there exists a unique endemic equilibrium* $E_*$, *and it is locally asymptotically stable* (*LAS*) *when* $\tau=0$.

*Proof* If $\mathcal{R}_0^1>1$, then it follows directly from the equilibrium equations that there is a unique endemic equilibrium (EE), $E_*=(\frac{\gamma+\epsilon+d}{\beta(I_*)},I_*,\frac{\gamma}{\alpha+d}I_*)$, where $I_*$ is the unique solution of the following equation:

$$A-\left(\gamma+\epsilon+d-\frac{\alpha\gamma}{\alpha+d}\right)I=\frac{d(\gamma+\epsilon+d)}{\beta(I)}.$$

Linearizing (2.1) at $E_*$, we obtain the characteristic equation

$$J(\lambda,\tau)=\lambda^3+p_2\lambda^2+p_1\lambda+p_0-(\lambda+d)(\lambda+\alpha+d)\beta'(I_*)I_*S_*e^{-\lambda\tau}=0, \tag{2.6}$$

where

$$p_2=\alpha+2d+\beta(I_*)I_*,\ p_1=d(\alpha+d)+(d+\alpha+a)\beta(I_*)I_*,\ p_0=\Big((\epsilon+d)(\alpha+d)+d\gamma\Big)\beta(I_*)I_*.$$

At $\tau = 0$, we have

$$J(\lambda, 0) = \lambda^3 + \bar{p}_2\lambda^2 + \bar{p}_1\lambda + \bar{p}_0,$$

where

$$\begin{aligned}
&\bar{p}_2 = \alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_* > 0,\\
&\bar{p}_1 = d(\alpha + d) + (d + \alpha + a)\beta(I_*)I_* - (\alpha + 2d)\beta'(I_*)I_*S_* > 0,\\
&\bar{p}_0 = \Big((\epsilon + d)(\alpha + d) + d\gamma\Big)\beta(I_*)I_* - d(\alpha + d)\beta'(I_*)I_*S_* > 0.
\end{aligned}$$

By a straightforward calculation, we know that $\bar{p}_2\bar{p}_1 - \bar{p}_0 > 0$. (The detailed calculation is presented in Appendix B.) The local stability of $E_*$ then follows from the Routh–Hurwitz criterion. □

Next, we explore the occurrence of possible Hopf bifurcation as $\tau$ increases. Note that $J(0, \tau) = p_0 - d(\alpha + d)\beta'(I_*)I_*S_*e^{-\lambda\tau} > 0$ for $\tau \geq 0$. This implies that $\lambda = 0$ is not an eigenvalue. The EE $E_*$ may lose its stability as $\tau$ increases only if there is a pair of purely imaginary eigenvalues $\lambda = \pm\omega i$ with $\omega > 0$. Substituting $\lambda = \omega i$, $\omega > 0$ into $J(\lambda, \tau) = 0$ gives

$$\begin{aligned}
J(\omega i, \tau) = {} & -p_2\omega^2 + p_0 - \big(d(\alpha + d) - \omega^2\big)\beta'(I_*)I_*S_*\cos(\omega\tau) - (\alpha + 2d)\\
& \times \omega\beta'(I_*)I_*S_*\sin(\omega\tau)\\
& - \omega^3 i + p_1\omega i - (\alpha + 2d)\omega\beta'(I_*)I_*S_*\cos(\omega\tau)i + \big(d(\alpha + d) - \omega^2\big)\\
& \times \beta'(I_*)I_*S_*\sin(\omega\tau)i.
\end{aligned}$$

From which we get

$$\cos(\bar{\omega}\tau) = \frac{(p_0 - p_2\bar{\omega}^2)\big(d(\alpha + d) - \bar{\omega}^2\big) + (p_1 - \bar{\omega}^2)(\alpha + 2d)\bar{\omega}^2}{\big(d(\alpha + d) - \bar{\omega}^2\big)^2 + (\alpha + 2d)^2\bar{\omega}^2}\frac{1}{\beta'(I_*)I_*S_*} := C_1, \tag{2.7}$$

$$\sin(\bar{\omega}\tau) = \frac{(p_0 - p_2\bar{\omega}^2)(\alpha + 2d)\bar{\omega} - (p_1 - \bar{\omega}^2)\big(d(\alpha + d) - \bar{\omega}^2\big)\bar{\omega}}{\big(d(\alpha + d) - \bar{\omega}^2\big)^2 + (\alpha + 2d)^2\bar{\omega}^2}\frac{1}{\beta'(I_*)I_*S_*} := C_2, \tag{2.8}$$

where $\bar{\omega} = \sqrt{x}$ and $x$ is a positive root (if it exists) of the following equation:

$$H(x) := x^3 + q_2x^2 + q_1x + q_0 = 0, \tag{2.9}$$

with

$$\begin{aligned}
&q_2 = (p_2)^2 - 2p_1 - \big(\beta'(I_*)I_*S_*\big)^2,\\
&q_1 = (p_1)^2 - 2p_0p_2 - (\alpha^2 + 2d\alpha + 2d^2)\big(\beta'(I_*)I_*S_*\big)^2,\\
&q_0 = (p_0)^2 - d^2(\alpha + d)^2\big(\beta'(I_*)I_*S_*\big)^2.
\end{aligned}$$

For this $\bar{\omega}$, there exists a sequence $\tau^n, n = 0, 1, 2, \ldots$, given by

$$\tau^n = \tau^0 + \frac{2n\pi}{\bar{\omega}}, \tau^0 = \begin{cases} \frac{\arccos C_1}{\bar{\omega}}, & C_2 \geq 0, \\ \frac{2\pi - \arccos C_1}{\bar{\omega}}, & C_2 < 0, \end{cases} \tag{2.10}$$

such that (2.7) and (2.8) hold. A straightforward calculation also shows that

$$\operatorname{sign}\left(\frac{d\lambda}{d\tau}\Big|_{\tau=\tau^n}\right) = \operatorname{sign}\left(\frac{H(x)}{dx}\Big|_{x=(\bar{\omega})^2}\right).$$

**Theorem 2.4** *If Eq.* (2.9) *has a positive root* $\bar{x}$ *satisfying* $H'(\bar{x}) \neq 0$, *then system* (2.1) *undergoes a Hopf bifurcation at* $E_*$ *when* $\tau = \tau^n$, *where* $\tau^n (n = 0, 1, \ldots)$ *are determined by* (2.10).

## 3 Our two-patch SIRS model

In this section, we couple Model (2.1) with dispersal by extending Model (2.1) to a two-patch setting. More specifically, we assume all individuals traveling between two patches do not change their disease states, that is, susceptibles (infectives/recovered) who traveled from one patch to the other are still susceptible (infectious/recovered). The flow chart is presented in Fig. 1. Patch specified disease states and parameters are labeled with subscript $i$ ($i = 1, 2$). The travel rates from patch $j$ to patch $i$ are assumed to be $m_{ij}^S > 0$, $m_{ij}^I \geq 0$, $m_{ij}^R > 0$, $i, j = 1, 2$.

Our two-patch SIRS model is then described by the following system:

$$\begin{cases} \frac{dS_1(t)}{dt} = A_1 - d_1 S_1(t) - \beta_1(I_1(t-\tau))I_1(t)S_1(t) + \alpha_1 R_1(t) - m_{21}^S S_1(t) + m_{12}^S S_2(t), \\ \frac{dI_1(t)}{dt} = \beta_1(I_1(t-\tau))I_1(t)S_1(t) - (\gamma_1 + \epsilon_1 + d_1)I_1(t) - m_{21}^I I_1(t) + m_{12}^I I_2(t), \\ \frac{dR_1(t)}{dt} = \gamma_1 I_1(t) - \alpha_1 R_1(t) - d_1 R_1(t) - m_{21}^R R_1(t) + m_{12}^R R_2(t), \\ \frac{dS_2(t)}{dt} = A_2 - d_2 S_2(t) - \beta_2(I_2(t-\tau))I_2(t)S_2(t) + \alpha_2 R_2(t) - m_{12}^S S_2(t) + m_{21}^S S_1(t), \\ \frac{dI_2(t)}{dt} = \beta_2(I_2(t-\tau))I_2(t)S_2(t) - (\gamma_2 + \epsilon_2 + d_2)I_2(t) - m_{12}^I I_2(t) + m_{21}^I I_1(t), \\ \frac{dR_2(t)}{dt} = \gamma_2 I_2(t) - \alpha_2 R_2(t) - d_2 R_2(t) - m_{12}^R R_2(t) + m_{21}^R R_1(t), \end{cases} \tag{3.1}$$

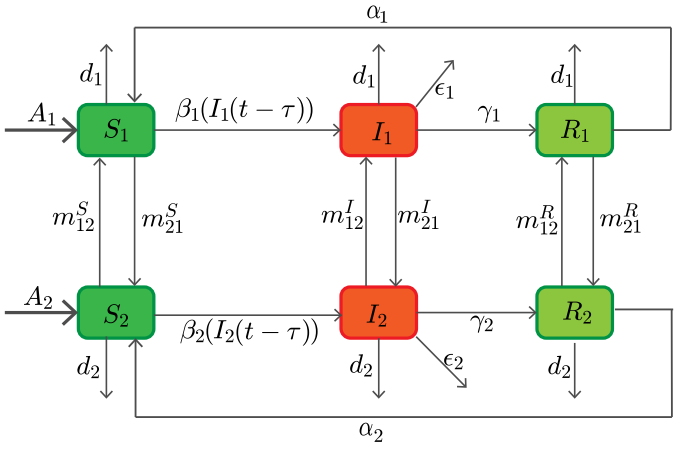


**Figure 1** The flow chart of our two-patch SIRS model

where $\beta_i(I_i(t-\tau)) = \hat{\beta}_i - \tilde{\beta}_i f_i(I_i(t-\tau))$. The associated initial condition is

$$\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6) \in X := \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+. \tag{3.2}$$

The well-posedness of our model is given by the following theorem with proof provided in Appendix C.

**Theorem 3.1** *System (3.1)–(3.2) possesses a unique solution, which is nonnegative. The total population* $N(t) = \sum_{i=1}^{2}(S_i + I_i + R_i)$ *is bounded satisfying*

$$N(t) \in \left[ \min\left\{ \frac{A_1 + A_2}{\max\{d_1 + \epsilon_1, d_2 + \epsilon_2\}}, N(0) \right\}, \max\left\{ \frac{A_1 + A_2}{\min\{d_1, d_2\}}, N(0) \right\} \right].$$

### 3.1 The disease-free equilibrium and the basic reproduction number

The disease-free equilibrium $E^0 = (\bar{S}_1, 0, \bar{R}_1, \bar{S}_2, 0, \bar{R}_2)$ satisfies the following equations:

$$\begin{cases} A_1 - (d_1 + m_{21}^S)\bar{S}_1 + m_{12}^S \bar{S}_2 = -\alpha_1 \bar{R}_1, \\ A_2 - (d_2 + m_{12}^S)\bar{S}_2 + m_{21}^S \bar{S}_1 = -\alpha_2 \bar{R}_2, \\ (\alpha_1 + d_1 + m_{21}^R)\bar{R}_1 = m_{12}^R \bar{R}_2, \\ (\alpha_2 + d_2 + m_{12}^R)\bar{R}_2 = m_{21}^R \bar{R}_1. \end{cases} \tag{3.3}$$

From the last two equations, we deduce that if $\bar{R}_1 = 0$, then $\bar{R}_2 = 0$, and vice versa. Moreover, if $\bar{R}_1 \neq 0$ and $\bar{R}_2 \neq 0$, it follows that

$$(\alpha_1 + d_1 + m_{21}^R)(\alpha_2 + d_2 + m_{12}^R) = m_{12}^R m_{21}^R,$$

which is impossible. Therefore $\bar{R}_1 = \bar{R}_2 = 0$ and hence

$$\begin{cases} A_1 + m_{12}^S \bar{S}_2 = (d_1 + m_{21}^S)\bar{S}_1, \\ A_2 + m_{21}^S \bar{S}_1 = (d_2 + m_{12}^S)\bar{S}_2. \end{cases} \tag{3.4}$$

This yields

$$\bar{S}_1 = \frac{A_1(d_2 + m_{12}^S) + A_2 m_{12}^S}{d_1 d_2 + d_1 m_{12}^S + d_2 m_{21}^S} > 0, \quad \bar{S}_2 = \frac{A_1 m_{21}^S + A_2(d_1 + m_{21}^S)}{d_1 d_2 + d_1 m_{12}^S + d_2 m_{21}^S} > 0. \tag{3.5}$$

Thus, there is a unique disease-free equilibrium (DFE), which is $E^0 = (\bar{S}_1, 0, 0, \bar{S}_2, 0, 0)$ with $\bar{S}_1$ and $\bar{S}_2$ given by (3.5).

To determine the basic reproduction number, using the next generation matrix method [44, 50], we get

$$F = \begin{pmatrix} \hat{\beta}_1 \bar{S}_1 & 0 \\ 0 & \hat{\beta}_2 \bar{S}_2 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} a_1 + m_{21}^I & -m_{12}^I \\ -m_{21}^I & a_2 + m_{12}^I \end{pmatrix},$$

where $a_i = \gamma_i + \epsilon_i + d_i$ and $\beta_i(0) = \beta_{i1}$ for $i = 1, 2$. By a direct calculation, we get

$$FV^{-1} = \frac{1}{a_1a_2 + a_1m_{12}^I + a_2m_{21}^I} \begin{pmatrix} \hat{\beta}_1\bar{S}_1(a_2 + m_{12}^I) & \hat{\beta}_1\bar{S}_1m_{12}^I \\ \hat{\beta}_2\bar{S}_2m_{21}^I & \hat{\beta}_2\bar{S}_{21}(a_1 + m_{21}^I) \end{pmatrix}.$$

Hence, the basic reproduction number $\mathcal{R}_0$ is given by

$$\mathcal{R}_0 = \rho\{FV^{-1}\} = \frac{\hat{\beta}_1\bar{S}_1(a_2 + m_{12}^I) + \hat{\beta}_2\bar{S}_2(a_1 + m_{21}^I) + \sqrt{\Delta}}{2(a_1a_2 + a_1m_{12}^I + a_2m_{21}^I)}, \tag{3.6}$$

where

$$\begin{aligned} \Delta &= \left(\hat{\beta}_1\bar{S}_1(a_2 + m_{12}^I) - \hat{\beta}_2\bar{S}_2(a_1 + m_{21}^I)\right)^2 + 4\hat{\beta}_1\bar{S}_1m_{12}^I\hat{\beta}_2\bar{S}_2m_{21}^I \\ &= \left(\hat{\beta}_1\bar{S}_1m_{12}^I + \hat{\beta}_2\bar{S}_2m_{21}^I\right)^2 + \left(\hat{\beta}_1\bar{S}_1a_2 - \hat{\beta}_2\bar{S}_2a_1\right)^2. \end{aligned}$$

Then from [50] we have the following theorem.

**Theorem 3.2** *The disease-free equilibrium $E^0$ is locally asymptotically stable (LAS) when $\mathcal{R}_0 < 1$, and it is unstable when $\mathcal{R}_0 > 1$.*

**Theorem 3.3** *If the travel rates are the same, $m_{ij}^Y = m$, $Y \in \{S, I, R\}$, $i, j = 1, 2$, $i \neq j$, then the disease-free equilibrium $E^0$ is globally asymptotically stable provided $\mathcal{R}_0 < 1$.*

*Proof* Set $N_i(t) = S_i(t) + I_i(t) + R_i(t)$, $i = 1, 2$. From system (3.1), we have

$$\begin{cases} \frac{dN_1(t)}{dt} = A_1 - d_1N_1(t) - mN_1(t) + mN_2(t) - \epsilon_1I_1, \\ \frac{dN_2(t)}{dt} = A_2 - d_2N_2(t) - mN_2(t) + mN_1(t) - \epsilon_2I_2. \end{cases} \tag{3.7}$$

This implies that

$$\begin{aligned} &\lim_{t\to\infty} \sup S_1(t) \leq \lim_{t\to\infty} \sup N_1(t) \leq \frac{A_1(d_2 + m) + A_2m}{d_1d_2 + d_1m + d_2m} = \bar{S}_1, \\ &\lim_{t\to\infty} \sup S_2(t) \leq \lim_{t\to\infty} \sup N_2(t) \leq \frac{A_2(d_1 + m) + A_1m}{d_1d_2 + d_1m + d_2m} = \bar{S}_2. \end{aligned}$$

Therefore, for sufficiently small $\eta > 0$, there exists $T > 0$ such that $S_i(t) \leq \bar{S}_i + \eta$, $i = 1, 2$. Thus, from the second and fifth equations of system (3.1), we have

$$\begin{pmatrix} I_1'(t) \\ I_2'(t) \end{pmatrix} \leq (F - V + \eta B) \begin{pmatrix} I_1(t) \\ I_2(t) \end{pmatrix},$$

where $B = \operatorname{diag}(\hat{\beta}_1, \hat{\beta}_2)$. From [44], we see that $\mathcal{R}_0 < 1$ implies that both eigenvalues of $F - V$ have negative real parts. Thus, for sufficiently small $\eta > 0$, both eigenvalues of $F - V + \eta B$ also have negative real parts. This implies that the unique equilibrium $(0, 0)$ of the following linear system

$$\begin{pmatrix} i_1'(t) \\ i_2'(t) \end{pmatrix} = (F - V + \eta B) \begin{pmatrix} i_1(t) \\ i_2(t) \end{pmatrix} \tag{3.8}$$

is globally asymptotically stable. Hence

$$\lim_{t\to\infty} i_k(t) = 0,\ k = 1, 2.$$

It then follows from a comparison that $\lim_{t\to\infty} I_k(t) = 0$, $k = 1, 2$.

Thus, the third and sixth equations of system (2.1) lead to a limiting system

$$\begin{cases} \frac{dR_1(t)}{dt} = -\alpha_1 R_1(t) - d_1 R_1(t) - mR_1(t) + mR_2(t), \\ \frac{dR_2(t)}{dt} = -\alpha_2 R_2(t) - d_2 R_2(t) - mR_2(t) + mR_1(t), \end{cases} \tag{3.9}$$

which yields $R_i(t) \to 0$, $i = 1, 2$ as $t \to \infty$.

Therefore, the first and fourth equations of system (2.1) lead to $S_i(t) \to \bar{S}_i$, $i = 1, 2$, as $t \to \infty$, as it has a limiting system

$$\begin{cases} \frac{dS_1(t)}{dt} = A_1 - d_1 S_1(t) - mS_1(t) + mS_2(t), \\ \frac{dS_2(t)}{dt} = A_2 - d_2 S_2(t) - mS_2(t) + mS_1(t). \end{cases} \tag{3.10}$$

By the theory of asymptotically autonomous systems [7], every solution of system (3.1) with a nonnegative initial condition (3.2) converges to $E_0$ as $t \to \infty$, provided that $\mathcal{R}_0^1 < 1$. The proof is complete. □

### 3.2 Existence of boundary equilibria $E_1^B$ and $E_2^B$

In this subsection, we consider the existence of boundary equilibria of system (3.1), denoted by $E_1^B = (\hat{S}_1, 0, \hat{S}_2, \hat{R}_1, \hat{I}_2, \hat{R}_2)$ *and* $E_2^B = (\tilde{S}_1, \tilde{I}_1, \tilde{R}_1, \tilde{S}_2, 0, \tilde{R}_2)$.

From system (3.1), it is easy to find that there are no boundary equilibria if $m_{12}^I > 0$ or $m_{21}^I > 0$. Next we consider the case $m_{12}^I = m_{21}^I = 0$. A direct examination reveals that the equilibrium point $E_1^B$, representing the presence of the disease only in patch 2, is determined by

$$\begin{cases} \hat{S}_2 = \frac{a_2}{\beta_2(\hat{I}_2)}, \\ \hat{R}_1 = \frac{m_{12}^R \gamma_2}{(\alpha_1+d_1)(\alpha_2+d_2)+(\alpha_1+d_1)m_{12}^R+(\alpha_2+d_2)m_{21}^R} \hat{I}_2 := c_1 \hat{I}_2, \\ \hat{R}_2 = \frac{\alpha_1+d_1+m_{21}^R}{m_{12}^R} \hat{R}_1 = \frac{\alpha_1+d_1+m_{21}^R}{m_{12}^R} c_1 \hat{I}_2 := c_2 \hat{I}_2, \\ \hat{S}_1 = \frac{A_1}{d_1+m_{21}^S} + \frac{\alpha_1}{d_1+m_{21}^S} \hat{R}_1 + \frac{m_{12}^S}{d_1+m_{21}^S} \hat{S}_2 = \frac{A_1}{d_1+m_{21}^S} + \frac{\alpha_1 c_1}{d_1+m_{21}^S} \hat{I}_2 + \frac{m_{12}^S}{d_1+m_{21}^S} \frac{a_2}{\beta_2(\hat{I}_2)}, \end{cases} \tag{3.11}$$

where $\hat{I}_2$ is a positive root of the equation

$$A_2 + \frac{A_1 m_{21}^S}{d_1 + m_{21}^S} - \left( a_2 - \alpha_2 c_2 - \frac{\alpha_1 c_1 m_{21}^S}{d_1 + m_{21}^S} \right) \hat{I}_2 = \frac{(d_1 d_2 + d_1 m_{12}^S + d_2 m_{21}^S)}{d_1 + m_{21}^S} \frac{a_2}{\beta_2(\hat{I}_2)}. \tag{3.12}$$

After lengthy calculations, it is found that $a_2 - \alpha_2 c_2 - \frac{\alpha_1 c_1 m_{21}^S}{d_1+m_{21}^S} > 0$. Hence, the left-hand side of (3.12) is a decreasing function of $\hat{I}_2$. Meanwhile, since $\beta_2(\hat{I}_2)$ is a decreasing function of $\hat{I}_2$, the right-hand side of (3.12) is an increasing function of $\hat{I}_2$. Therefore, a solution exists only when

$$A_2 + \frac{A_1 m_{21}^S}{d_1 + m_{21}^S} > \frac{(d_1 d_2 + d_1 m_{12}^S + d_2 m_{21}^S)}{d_1 + m_{21}^S} \frac{a_2}{\beta_2(0)}.$$

That is, $\widetilde{\mathcal{R}}_0^2 > 1$, where

$$\widetilde{\mathcal{R}}_0^2 := \bar{S}_2 \frac{\hat{\beta}_2}{a_2}. \tag{3.13}$$

Define

$$\widetilde{\mathcal{R}}_0^1 := \bar{S}_1 \frac{\hat{\beta}_1}{a_1}. \tag{3.14}$$

Similarly, we can obtain that the condition of the existence and uniqueness of $E_2^B$ is $\widetilde{\mathcal{R}}_0^1 > 1$.

Summarizing the above analysis, we get the following result.

**Theorem 3.4** *If $m_{12}^I = m_{21}^I = 0$, then there exists a unique boundary equilibrium $E_1^B$ if and only if $\widetilde{\mathcal{R}}_0^2 > 1$; while $E_2^B$ exists and is unique if and only if $\widetilde{\mathcal{R}}_0^1 > 1$. Further, there are no boundary equilibria if $m_{12}^I > 0$ or $m_{21}^I > 0$.*

For system (3.1) with $m_{21}^I = m_{12}^I = 0$, (3.6) yields

$$\mathcal{R}_0 = \max\left\{\widetilde{\mathcal{R}}_0^1, \widetilde{\mathcal{R}}_0^2\right\}. \tag{3.15}$$

Then we have the following result.

**Corollary 3.5** *Consider system (3.1) with $m_{21}^I = m_{12}^I = 0$. If $\mathcal{R}_0 \le 1$, then neither $E_1^B$ nor $E_2^B$ exists.*

### 3.3 Existence of the endemic equilibrium

*3.3.1 Uniform persistence of system (3.1) with $m_{12}^I m_{21}^I > 0$*

**Theorem 3.6** *Let $X_0 = \{\phi \in X : \phi_2(0) + \phi_4(0) > 0\}$. If $\mathcal{R}_0 > 1$, then there exists a real number $\delta > 0$ such that any solution $(S_1(t;\phi), I_1(t;\phi), R_1(t;\phi), S_2(t;\phi), I_2(t;\phi), R_2(t;\phi))$ of system (3.1) with $\phi \in X_0$ when $m_{21}^I > 0$ and $m_{12}^I > 0$ satisfies*

$$\liminf_{t\to\infty}(S_1(t,\phi), I_1(t,\phi), R_1(t,\phi), S_2(t,\phi), I_2(t,\phi), R_2(t,\phi)) \ge (\delta,\delta,\delta,\delta,\delta,\delta).$$

*Moreover, system (3.1) has an endemic equilibrium in $X_0$.*

*Proof* Set $\partial X_0 = X \setminus X_0$. Thus $\partial X_0 = \{\phi \in X : \phi_2(0) = \phi_4(0) = 0\}$.

As per convention, let us denote the solution semiflow of system (3.1) as $\Phi(t) : X \to X$. Here, $\Phi(t)\phi = x_t(\cdot;\phi)$ for every $\phi \in X$ and $t \ge 0$. Consequently, $\Phi(t)$ is deemed asymptotically smooth for $t \ge \tau$ [22] and exhibits point dissipativity (cf. Theorem 3.1). Thus, a global attractor exists for $\Phi(t)$ within $X$. Furthermore, deduced from the proof of Theorem 3.1, $\Phi(t)(X_0)$ remains within $X_0$ for $t \ge 0$.

Let $M = \{E_0\}$. It is easy to show that $M$ is an isolated invariant set in $\partial X_0$. By the proof of Theorem 3.3, $\forall \phi \in M_\partial := \{\phi \in \partial X_0 : \Phi(t)(\phi) \in \partial X_0, t \ge 0\}$, its omega limit set $\omega(\phi) \subset M$.

Now, assuming $\mathcal{R}_0 > 1$, by the persistence theory [22], $\Phi(t)$ is uniformly persistent concerning $X_0$ if we can demonstrate that $W^s(M) \cap X_0 = \emptyset$, where $W^s(M)$ represents the stable set associated with $M$.

Let us consider the contrary scenario where there exists $\psi = (\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6) \in X_0$ such that the solution tends to $E_0$ as $t$ approaches infinity. In this case, for any given $\epsilon > 0$, there exists a sufficiently large $T$ such that

$$\begin{aligned}
&\bar{S}_1 - \epsilon < S_1(t;\psi) < \bar{S}_1 + \epsilon, \quad 0 \le I_1(t;\psi) < \epsilon, \quad 0 \le R_1(t;\psi) < \epsilon,\\
&\bar{S}_2 - \epsilon < S_2(t;\psi) < \bar{S}_2 + \epsilon, \quad 0 \le I_2(t;\psi) < \epsilon, \quad 0 \le R_2(t;\psi) < \epsilon
\end{aligned}$$

for all $t > T$. Thus, for $t > T + \tau$,

$$\begin{aligned}
I_1'(t) &\ge \beta_1(\epsilon)(\bar{S}_1 - \epsilon)I_1(t) - a_1 I_1(t) - m_{21}^I I_1(t) + m_{12}^I I_2(t),\\
I_2'(t) &\ge \beta_2(\epsilon)(\bar{S}_2 - \epsilon)I_2(t) - a_2 I_2(t) - m_{12}^I I_2(t) + m_{21}^I I_1(t).
\end{aligned} \tag{3.16}$$

By the continuity of $\beta_i$, for sufficiently small $\epsilon > 0$, $\beta_i(\epsilon)$ is sufficiently close to $\beta_{i1}$. Therefore, if $\mathcal{R}_0 = \rho(FV^{-1}) > 1$, then for those $\epsilon$, $\rho(F_\epsilon V^{-1}) > 1$, where

$$F_\epsilon = \begin{pmatrix} \beta_1(\epsilon)(\bar{S}_1 - \epsilon) & 0 \\ 0 & \beta_2(\epsilon)(\bar{S}_2 - \epsilon) \end{pmatrix}.$$

Consider that $F_\epsilon - V$ possesses two real eigenvalues $\lambda_1 > \lambda_2$. From the condition $\rho(F_\epsilon V^{-1}) > 1$, it follows that $\lambda_1 > 0$. Furthermore, $\lambda_1$ corresponds to an eigenvector $(1, \nu_1)^T$, while $\lambda_2$ corresponds to an eigenvector $(1, -\nu_2)^T$, where $\nu_1 > 0$ and $\nu_2 > 0$.

By comparison, for equation (3.16), we have

$$\begin{pmatrix} I_1'(t) \\ I_2'(t) \end{pmatrix} \ge \begin{pmatrix} i_1'(t) \\ i_2'(t) \end{pmatrix} = (F_\epsilon - V) \begin{pmatrix} i_1(t) \\ i_2(t) \end{pmatrix}$$

with

$$\begin{pmatrix} i_1(0) \\ i_2(0) \end{pmatrix} = \begin{pmatrix} I_1(0) \\ I_2(0) \end{pmatrix}.$$

So,

$$\begin{pmatrix} I_1(t) \\ I_2(t) \end{pmatrix} \ge \begin{pmatrix} i_1(t) \\ i_2(t) \end{pmatrix} = C_1 e^{\lambda_1 t} \begin{pmatrix} 1 \\ \nu_1 \end{pmatrix} + C_2 e^{\lambda_2 t} \begin{pmatrix} 1 \\ -\nu_2 \end{pmatrix}$$

with

$$C_1 = \frac{\nu_2 I_1(0) + I_2(0)}{\nu_1 + \nu_2} > 0, \; C_2 = \frac{\nu_1 I_1(0) - I_2(0)}{\nu_1 + \nu_2}.$$

Consequently, as $t$ approaches infinity, both $I_1(t)$ and $I_2(t)$ diverge towards infinity, leading to a contradiction. Thus, $W^s(M) \cap X_0 = \emptyset$ when $\mathcal{R}_0 > 1$.

To ensure the practical persistence of $\Phi(t)$, inspired by [49, Example 1.3.1], we define a generalized distance function for the semiflow $\Phi(t)$ as follows:

$$p(\phi) = \min\{\phi_2(0), \phi_4(0)\}, \forall \phi \in X.$$

It can be readily verified that $W^s(M) \cap p^{-1}(0,\infty) = \emptyset$. Therefore, according to [49, Theorem 1.3.2], there exists a real number $\delta > 0$ such that $\liminf_{t\to\infty} p(\Phi(t)\phi) \geq (\delta,\delta,\delta,\delta,\delta,\delta)$.

In other words, for any solution $(S_1(t,\phi), I_1(t,\phi), R_1(t,\phi), S_2(t,\phi), I_2(t,\phi), R_2(t,\phi))$ of system (3.1) with $\phi \in X_0$, we have

$$\liminf_{t\to\infty}(S_1(t,\phi), I_1(t,\phi), R_1(t,\phi), S_2(t,\phi), I_2(t,\phi), R_2(t,\phi)) \geq (\delta,\delta,\delta,\delta,\delta,\delta).$$

Moreover, as per [49, Theorem 1.3.11], system (3.1) possesses a coexistence equilibrium in $X_0$, which qualifies as an endemic equilibrium (given $m_{21}^I > 0$ and $m_{12}^I > 0$). □

*3.3.2 Endemic equilibria of system* (3.1) *with* $m_{12}^I = m_{21}^I = 0$

In this section, we consider the case where the infectives do not travel, i.e., $m_{12}^I = m_{21}^I = 0$. We will delve into the existence and uniqueness of the EE, denoted as $E^* = (S_1^*, I_1^*, R_1^*, S_2^*, I_2^*, R_2^*)$ in this particular case. For the EE $E^*$, from the second and fifth equations of system (3.1), we have

$$S_1^* = \frac{a_1}{\beta_1(I_1^*)}, \quad S_2^* = \frac{a_2}{\beta_2(I_2^*)}.$$

And from the third and sixth equations of system (3.1), we get

$$R_1^* = \frac{b_2\gamma_1 I_1^* + m_{12}^R\gamma_2 I_2^*}{b_1 b_2 - m_{12}^R m_{21}^R} := r_{11}I_1^* + r_{12}I_2^*, \quad R_2^* = \frac{m_{21}^R\gamma_1 I_1^* + b_1\gamma_2 I_2^*}{b_1 b_2 - m_{12}^R m_{21}^R} := r_{21}I_1^* + r_{22}I_2^*,$$

where $b_1 = \alpha_1 + d_1 + m_{21}^R$, $b_2 = \alpha_2 + d_2 + m_{12}^R$.

Substituting them into the other two equations of system (3.1), we get

$$\begin{cases} A_1 - d_1\frac{a_1}{\beta_1(I_1^*)} + (\alpha_1 r_{11} - a_1)I_1^* + \alpha_1 r_{12}I_2^* - m_{21}^S\frac{a_1}{\beta_1(I_1^*)} + m_{12}^S\frac{a_2}{\beta_2(I_2^*)} = 0, \\ A_2 - d_2\frac{a_2}{\beta_2(I_2^*)} + (\alpha_2 r_{22} - a_2)I_2^* + \alpha_2 r_{21}I_1^* - m_{12}^S\frac{a_2}{\beta_2(I_2^*)} + m_{21}^S\frac{a_1}{\beta_1(I_1^*)} = 0. \end{cases}$$

That is,

$$\begin{cases} \alpha_2 r_{21}I_1^* + m_{21}^S\frac{a_1}{\beta_1(I_1^*)} = \frac{a_2(d_2+m_{12}^S)}{\beta_2(I_2^*)} + (a_2 - \alpha_2 r_{22})I_2^* - A_2, \\ \alpha_1 r_{12}I_2^* + m_{12}^S\frac{a_2}{\beta_2(I_2^*)} = \frac{a_1(d_1+m_{21}^S)}{\beta_1(I_1^*)} + (a_1 - \alpha_1 r_{11})I_1^* - A_1. \end{cases} \tag{3.17}$$

If $\mathcal{R}_0 \leq 1$, then our next result shows that there is no EE.

**Lemma 3.7** *Consider system* (3.1) *with* $m_{12}^I = m_{21}^I = 0$. *If* $\mathcal{R}_0 \leq 1$, *then system* (3.1) *does not admit an EE.*

*Proof* If there exists an EE, from system (3.1) we obtain

$$d_1 S_1^* + d_2 S_2^* + (\epsilon_1 + d_1)I_1^* + (\epsilon_2 + d_2)I_2^* + d_1 R_1 + d_2 R_2 = A_1 + A_2.$$

This implies that $d_1 S_1^* + d_2 S_2^* < A_1 + A_2$. Meanwhile, by the definitions of $\bar{S}_i, i = 1,2$, we have $d_1\bar{S}_1 + d_2\bar{S}_1 = A_1 + A_2$. When $m_{12}^I = m_{21}^I = 0$, we get

$$\mathcal{R}_0 = \max\left\{\bar{S}_1\frac{\hat{\beta}_1}{a_1}, \bar{S}_2\frac{\hat{\beta}_2}{a_2}\right\}.$$

And hence, if $\mathcal{R}_0 \le 1$, then

$$\bar{S}_1 \le \frac{a_1}{\hat{\beta}_1}, \bar{S}_2 \le \frac{a_2}{\hat{\beta}_2}.$$

As $\beta_i (i = 1, 2)$ are decreasing functions, we have

$$\bar{S}_1 < \frac{a_1}{\beta_1(I_1^*)} = S_1^*, \bar{S}_2 < \frac{a_2}{\beta_2(I_2^*)} = S_2^*.$$

Thus,

$$d_1 S_1^* + d_2 S_2^* > d_1 \bar{S}_1 + d_2 \bar{S}_1 = A_1 + A_2,$$

which is a contradiction. □

To consider the existence of EE when $\mathcal{R}_0 > 1$, we define

$$\mathcal{R}_0^{(1,2)} = \frac{A_1 \hat{\beta}_1}{a_1(d_1 + m_{21}^S)} + \frac{a_2 m_{12}^S \hat{\beta}_1}{a_1(d_1 + m_{21}^S)\hat{\beta}_2}, \tag{3.18}$$

$$\mathcal{R}_0^{(2,1)} = \frac{A_2 \hat{\beta}_2}{a_2(d_2 + m_{12}^S)} + \frac{a_1 m_{21}^S \hat{\beta}_2}{a_2(d_2 + m_{12}^S)\hat{\beta}_1}. \tag{3.19}$$

To better sort out the conditions on the existence of equilibria, we introduce the following lemmas and omit their proof (readers can refer to the proofs of Lemmas 4.8–4.10 in [42]).

**Lemma 3.8** *$\widetilde{\mathcal{R}}_0^1 > \mathcal{R}_0^{(1,2)}$ if and only if $\widetilde{\mathcal{R}}_0^2 > 1$; $\widetilde{\mathcal{R}}_0^2 > \mathcal{R}^{(2,1)}$ if and only if $\widetilde{\mathcal{R}}_0^1 > 1$.*

**Lemma 3.9** *If $\mathcal{R}_0 \le 1$, there are three cases*:

(i) *If $\widetilde{\mathcal{R}}_0^1 > 1$ and $\widetilde{\mathcal{R}}_0^2 \le 1$, then $\mathcal{R}_0^{(1,2)} > 1$ and $\mathcal{R}_0^{(2,1)} < 1$;*
(ii) *If $\widetilde{\mathcal{R}}_0^1 \le 1$ and $\widetilde{\mathcal{R}}_0^2 > 1$, then $\mathcal{R}_0^{(1,2)} < 1$ and $\mathcal{R}_0^{(2,1)} > 1$;*
(iii) *If $\widetilde{\mathcal{R}}_0^1 > 1$ and $\widetilde{\mathcal{R}}_0^2 > 1$, then either $\mathcal{R}_0^{(1,2)} > 1$ or $\mathcal{R}_0^{(2,1)} > 1$.*

Let

$$x_1 = u_1(I_1) := \alpha_2 r_{21} I_1 + m_{21}^S \frac{a_1}{\beta_1(I_1)}, I_1 \in [0, \infty), \tag{3.20}$$

$$x_2 = u_2(I_2) := \alpha_1 r_{12} I_2 + m_{12}^S \frac{a_2}{\beta_2(I_2)}, I_2 \in [0, \infty). \tag{3.21}$$

It is easy to show that $u_1$ and $u_2$ are increasing functions. So, $I_1 = u_1^{-1}(x_1), x_1 \in \left[\frac{m_{21}^S a_1}{\beta_1(0)}, \infty\right)$; and $I_2 = u_2^{-1}(x_2), x_2 \in \left[\frac{m_{12}^S a_2}{\beta_2(0)}, \infty\right)$.

Then, equations (3.17) define the following two functions:

$$x_1 = g_2(x_2) := \frac{a_2(d_2 + m_{12}^S)}{\beta_2(u_2^{-1}(x_2))} + (a_2 - \alpha_2 r_{22}) u_2^{-1}(x_2) - A_2,\ x_2 \in \left[\frac{m_{12}^S a_2}{\beta_2(0)}, \infty\right), \tag{3.22}$$

$$x_2 = g_1(x_1) := \frac{a_1(d_1 + m_{21}^S)}{\beta_1(u_1^{-1}(x_1))} + (a_1 - \alpha_1 r_{11}) u_1^{-1}(x_1) - A_1,\ x_1 \in \left[\frac{m_{21}^S a_1}{\beta_1(0)}, \infty\right). \tag{3.23}$$

The function $x_1 = g_2(x_2)$ passes through the point $\left(\frac{a_2(d_2+m_{12}^S)}{\beta_2(0)} - A_2, \frac{m_{21}^S a_1}{\beta_1(0)}\right)$ in the plane $x_1$-$x_2$. Meanwhile, the function $x_2 = g_1(x_1)$ passes through the point $\left(\frac{m_{12}^S a_2}{\beta_2(0)}, \frac{a_1(d_1+m_{21}^S)}{\beta_1(0)} - A_1\right)$ in the same plane $x_1$-$x_2$.

We can define

$$G_1(x_1) = g_2\big(g_1(x_1)\big) - x_1. \tag{3.24}$$

And for this function, if $g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) \geq \frac{a_2 m_{12}^S}{\beta_2(0)}$ that is equal to $\mathcal{R}_0^{(1,2)} \leq 1$, set $x_1 \in \left[\frac{m_{21}^S a_1}{\beta_1(0)}, \infty\right)$. Otherwise, if $g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < \frac{a_2 m_{12}^S}{\beta_2(0)}$ that is equal to $\mathcal{R}_0^{(1,2)} > 1$, set $x_1 \in \left[g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right), \infty\right)$.

**Lemma 3.10** *$g_1(x_1)$ and $g_2(x_2)$ and $G_1(x_1)$ are all increasing functions.*

The proof of Lemma 3.10 is presented in Appendix D.

Thus, $G_1(x_1) = 0$ has at most one root. Consequently, the two curves $x_1 = g_2(x_2)$ and $x_2 = g_1(x_1)$ have at most one intersection point in the first quadrant of the plane $x_1x_2$. So, in order to have an EE, it is needed that $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < 0$; otherwise, there does not exist an EE. Moreover, for an EE, it is required that $x_1^* = u_1(I_1^*) > \frac{m_{21}^S a_1}{\beta_1(0)}$, $x_2^* = u_2(I_2^*) > \frac{m_{12}^S a_2}{\beta_2(0)}$.

**Lemma 3.11** *Assume $\mathcal{R}_0^{(1,2)} \leq 1$. If $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) \geq 0$, then there does not exist an EE; if $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < 0$, then there exists a unique EE.*

*Proof* If $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < 0$, then set the only intersection point as $\tilde{P} = (\tilde{x}_1, \tilde{x}_2)$ with $\tilde{x}_1 > \frac{m_{21}^S a_1}{\beta_1(0)}$. This implies $g_2(g_1(\tilde{x}_1)) = \tilde{x}_1$ and $\tilde{x}_2 = g_1(\tilde{x}_1)$. Then, according to the monotonicity of $g_1$, we get $\tilde{x}_2 = g_1(\tilde{x}_1) > g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right)$. Note that $g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) = \frac{a_1(d_1+m_{21}^S)}{\beta_1(0)} - A_1$ and $g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) \geq \frac{a_2 m_{12}^S}{\beta_2(0)}$ is equal to $\mathcal{R}_0^{(1,2)} \leq 1$. So, there must be $\tilde{x}_2 > \frac{m_{12}^S a_2}{\beta_2(0)}$. □

Similarly, we can define

$$G_2(x_2) = g_1\big(g_2(x_2)\big) - x_2. \tag{3.25}$$

And, if $g_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) \geq \frac{a_1 m_{21}^S}{\beta_1(0)}$ that is equal to $\mathcal{R}_0^{(2,1)} \leq 1$, then set $x_2 \in \left[\frac{m_{12}^S a_2}{\beta_2(0)}, \infty\right)$. Otherwise, if $g_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) < \frac{a_1 m_{21}^S}{\beta_1(0)}$ that is equal to $\mathcal{R}_0^{(2,1)} > 1$, then set $x_2 \in \left[g_2^{-1}\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right), \infty\right)$.

We can prove that $G_2(x_2)$ is an increasing function. The following lemma also holds.

**Lemma 3.12** *Assume $\mathcal{R}_0^{(2,1)} \leq 1$. If $G_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) \geq 0$, there does not exist an EE; if $G_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) < 0$, there exists a unique EE.*

We further have the following results.

**Lemma 3.13** *If $\mathcal{R}_0^{(1,2)} > 1$ and $\mathcal{R}_0^{(2,1)} > 1$, there exists a unique EE.*

*Proof* If $\mathcal{R}_0^{(1,2)} > 1$ and $\mathcal{R}_0^{(2,1)} > 1$. According to the definitions of $G_1(x_1)$ and $G_2(x_2)$, in this case, we only need to consider under the given conditions $x_1 \geq g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right)$ and $x_2 \geq g_2^{-1}\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right)$.

Firstly, from $\mathcal{R}_0^{(1,2)} > 1$ and $\mathcal{R}_0^{(2,1)} > 1$, that is, $g_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) < \frac{m_{21}^S a_1}{\beta_1(0)}$ and $g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < \frac{m_{12}^S a_2}{\beta_2(0)}$, we get

$$\frac{m_{21}^S a_1}{\beta_1(0)} < g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right).$$

So,

$$\begin{aligned} G_1\left(g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right)\right) &= g_2\left(g_1\left(g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right)\right)\right) - g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) \\ &= g_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) - g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) < 0. \end{aligned}$$

This implies that the two curves $x_1 = g_2(x_2)$ and $x_2 = g_1(x_1)$ have one intersection point denoted by $\tilde{P} = (\tilde{x}_1, \tilde{x}_2)$ with $\tilde{x}_1 > g_1^{-1}\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right)$ and $\tilde{x}_2 > g_2^{-1}\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right)$.

Secondly, note that $g_1$ and $g_2$ are increasing functions. We know that $\tilde{x}_1 = g_2(\tilde{x}_2) > \frac{m_{21}^S a_1}{\beta_1(0)}$ and $\tilde{x}_2 = g_2(\tilde{x}_1) > \frac{m_{12}^S a_2}{\beta_2(0)}$. This completes the proof. □

**Lemma 3.14** *If* $\mathcal{R}_0^{(1,2)} \le 1$ *and* $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) \le 0$, *then* $\mathcal{R}_0^{(2,1)} > 1$. *If* $\mathcal{R}_0^{(2,1)} \le 1$ *and* $G_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) \le 0$, *then* $\mathcal{R}_0^{(1,2)} > 1$.

*Proof* Note that $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) \le 0$ is equivalent to

$$g_2\left(g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right)\right) \le \frac{m_{21}^S a_1}{\beta_1(0)}.$$

Note also that $\mathcal{R}_0^{(1,2)} \le 1 \Leftrightarrow \frac{m_{12}^S a_2}{\beta_2(0)} \le g_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right)$. Thus

$$\frac{a_2(d_2 + m_{12}^S)}{\beta_2(0)} - A_2 = g_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) \le \frac{m_{21}^S a_1}{\beta_1(0)},$$

that is, $\mathcal{R}_0^{(2,1)} > 1$.

Similarly, we can prove the second part of this lemma. □

**Theorem 3.15** *When* $\mathcal{R}_0 > 1$, *there exists a unique EE if one of the following conditions holds*:

(i) $\mathcal{R}_0^{(1,2)} > 1$ *and* $\mathcal{R}_0^{(2,1)} > 1$;
(ii) $\mathcal{R}_0^{(1,2)} \le 1$, $\mathcal{R}_0^{(2,1)} > 1$, *and* $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < 0$;
(iii) $\mathcal{R}_0^{(1,2)} > 1$, $\mathcal{R}_0^{(2,1)} \le 1$, *and* $G_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) < 0$.

With the above results, we summarize the existence of equilibria of system (3.1) with $m_{12}^I = m_{21}^I = 0$ in Table 1.

### 3.4 Stability of $E^*$ at a special case

We have shown that if $m_{12}^I m_{21}^I > 0$ and $\mathcal{R}_0 > 1$, then system (3.1) has at least one endemic equilibrium $E^*$ and the boundary equilibria $E_1^B$ and $E_2^B$ do not exist; If $m_{12}^I = m_{21}^I = 0$ and

**Table 1** The existence of equilibria when $m_{21}^I = m_{12}^I = 0$. Notation is as follows: × (does not exist); ✓ (exists); US (unstable); LAS (locally asymptotically stable). $\widetilde{\mathcal{R}}_0^1$, $\widetilde{\mathcal{R}}_0^2$ are defined in (3.13) and (3.14). $\mathcal{R}_0$, $\mathcal{R}_0^{(1,2)}$, $\mathcal{R}_0^{(2,1)}$, $G_1$, and $G_2$ are defined in (3.6), (3.18), (3.19), (3.24), and (3.25), respectively

| | | | | $E_0$ | $E_1^B$ | $E_2^B$ | $E^*$ |
|---|---|---|---|---|---|---|---|
| $\mathcal{R}_0 < 1$ | $\widetilde{\mathcal{R}}_0^1 < 1, \widetilde{\mathcal{R}}_0^2 < 1$ | | | LAS | × | × | × |
| $\mathcal{R}_0 = 1$ | | | | ✓ | × | × | × |
| $\mathcal{R}_0 > 1$ | $\widetilde{\mathcal{R}}_0^1 > 1, \widetilde{\mathcal{R}}_0^2 \le 1$ | $\mathcal{R}_0^{(1,2)} > 1, \mathcal{R}_0^{(2,1)} < 1$ | | US | × | ✓ | × |
| | $\widetilde{\mathcal{R}}_0^1 \le 1, \widetilde{\mathcal{R}}_0^2 > 1$ | $\mathcal{R}_0^{(1,2)} < 1, \mathcal{R}_0^{(2,1)} > 1$ | | US | ✓ | × | × |
| | $\widetilde{\mathcal{R}}_0^1 > 1, \widetilde{\mathcal{R}}_0^2 > 1$ | $\mathcal{R}_0^{(1,2)} > 1, \mathcal{R}_0^{(2,1)} > 1$ | | US | ✓ | ✓ | ✓ |
| | | $\mathcal{R}_0^{(1,2)} \le 1, \mathcal{R}_0^{(2,1)} > 1$ | $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) \ge 0$ | US | ✓ | ✓ | × |
| | | | $G_1\left(\frac{m_{21}^S a_1}{\beta_1(0)}\right) < 0$ | US | ✓ | ✓ | ✓ |
| | | $\mathcal{R}_0^{(1,2)} > 1, \mathcal{R}_0^{(2,1)} \le 1$ | $G_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) \ge 0$ | US | ✓ | ✓ | × |
| | | | $G_2\left(\frac{m_{12}^S a_2}{\beta_2(0)}\right) < 0$ | US | ✓ | ✓ | ✓ |

$\mathcal{R}_0 > 1$, then boundary equilibria $E_1^B$ and $E_2^B$ exist and there is a unique EE $E^*$ if one of the conditions in Theorem 3.15 is satisfied. The stability analysis of the EE $E^*$ of system (3.1) is quite complex in general. Next we consider the stability of the EE at a special homogeneous case with

$$\begin{aligned} &A_i = A, \qquad \beta_i(I) = \hat{\beta} - \tilde{\beta} f(I), \qquad d_i = d, \qquad \epsilon_i = \epsilon, \qquad \gamma_i = \gamma, \\ &\alpha_i = \alpha, \qquad m_{ij}^Y = m^Y, \qquad Y \in \{S, R\}, \qquad m_{ij}^I = 0. \end{aligned} \tag{3.26}$$

For this special case, we get

$$\widetilde{\mathcal{R}}_0^1 = \widetilde{\mathcal{R}}_0^2 = \mathcal{R}_0 = \mathcal{R}_0^1 = \frac{A\hat{\beta}}{ad} < \frac{A\hat{\beta} + am^S}{ad + am^S} = \mathcal{R}_0^{(1,2)} = \mathcal{R}_0^{(2,1)}.$$

It follows from Theorem 3.15 that there is a unique EE if $\mathcal{R}_0 > 1$.

**Theorem 3.16** *If $\mathcal{R}_0^1 > 1$, then system (3.1) with (3.26) admits a unique endemic equilibrium $E^*$, which is locally asymptotically stable when $\tau = 0$.*

*Proof* Linearizing system (3.1) at $E^*$, we obtain the characteristic equation

$$L(\lambda, \tau) = \hat{L}(\lambda, \tau)\tilde{L}(\lambda, \tau) = 0, \tag{3.27}$$

where

$$\hat{L}(\lambda, \tau) = \lambda^3 + \hat{l}_{12}\lambda^2 + \hat{l}_{11}\lambda + \hat{l}_{10} - (\lambda^2 + \hat{l}_{21}\lambda + \hat{l}_{20})\beta'(I^*)I^*S^*e^{-\lambda\tau}$$

with

$$\begin{aligned} \hat{l}_{12} &= \alpha + 2d + 2m^S + \beta(I^*)I^* + 2m^I + 2m^R > 0, \\ \hat{l}_{11} &= (d + 2m^S)(\alpha + d + 2m^R + 2m^I) + \beta(I^*)I^*(a + 2m^I + \alpha + d + 2m^R) \\ &\quad + 2m^I(\alpha + d + 2m^R) > 0, \end{aligned}$$

$$\hat{l}_{10} = 2m^I(d + 2m^S)(\alpha + d + 2m^R) + \beta(I^*)I^*\left((\epsilon + d + 2m^I)(\alpha + d + 2m^R) + \gamma(d + 2m^R)\right)$$
$$> 0.$$
$$\hat{l}_{21} = \alpha + 2d + 2m^R + 2m^S > 0,$$
$$\hat{l}_{20} = (d + 2m^S)(\alpha + d + 2m^R) > 0$$

and

$$\tilde{L}(\lambda, \tau) = \lambda^3 + \tilde{l}_{12}\lambda^2 + \tilde{l}_{11}\lambda + \tilde{l}_{10} - (\lambda^2 + \tilde{l}_{21}\lambda + \tilde{l}_{20})\beta'(I^*)I^*S^*e^{-\lambda\tau}$$

with

$$\tilde{l}_{12} = \alpha + 2d + \beta(I^*)I^* > 0, \quad \tilde{l}_{11} = d(\alpha + d) + \beta(I^*)I^*(a + \alpha + d) > 0,$$
$$\tilde{l}_{10} = \beta(I^*)I^*\left((\epsilon + d)(\alpha + d) + d\gamma\right) > 0, \quad \tilde{l}_{21} = \alpha + 2d > 0, \quad \tilde{l}_{20} = d(\alpha + d) > 0.$$

When $\tau = 0$, we obtain

$$\hat{L}(\lambda, 0) = \lambda^3 + \hat{l}^0_{12}\lambda^2 + \hat{l}^0_{11}\lambda + \hat{l}^0_{10}, \quad \tilde{L}(\lambda, 0) = \lambda^3 + \tilde{l}^0_{12}\lambda^2 + \tilde{l}^0_{11}\lambda + \tilde{l}^0_{10}$$

with (note that $\beta(\cdot) < 0$)

$$\hat{l}^0_{12} = \hat{l}_{12} - \beta'(I^*)I^*S^* > 0, \quad \hat{l}^0_{11} = \hat{l}_{11} - \hat{l}_{21}\beta'(I^*)I^*S^* > 0, \quad \hat{l}^0_{10} = \hat{l}_{10} - \hat{l}_{20}\beta'(I^*)I^*S^* > 0.$$
$$\tilde{l}^0_{12} = \tilde{l}_{12} - \beta'(I^*)I^*S^* > 0, \quad \tilde{l}^0_{11} = \tilde{l}_{11} - \tilde{l}_{21}\beta'(I^*)I^*S^* > 0, \quad \tilde{l}^0_{10} = \tilde{l}_{10} - \tilde{l}_{20}\beta'(I^*)I^*S^* > 0.$$

By a straightforward calculation, we know that $\hat{l}^0_{12}\hat{l}^0_{11} - \hat{l}^0_{10} > 0$ and $\tilde{l}^0_{12}\tilde{l}^0_{11} - \tilde{l}^0_{10} > 0$. Thus, all roots of $\hat{L}(\lambda, 0) = 0$ and $\tilde{L}(\lambda, 0) = 0$ have negative real parts, and hence $E^*$ is locally asymptotically stable. □

In examining the potential for system (3.1) to undergo a Hopf bifurcation as $\tau$ increases, it is noteworthy that when $\tau \geq 0$,

$$\hat{L}(0, \tau) = \hat{l}_{10} - \hat{l}_{20}\beta'(I^*)I^*S^* > 0, \quad \tilde{L}(0, \tau) = \tilde{l}_{10} - \tilde{l}_{20}\beta'(I^*)I^*S^* > 0.$$

Thus $L(0, \tau) = \hat{L}(0, \tau)\tilde{L}(0, \tau) > 0$, which implies that $\lambda = 0$ is not an eigenvalue. Therefore, our investigation aims to ascertain whether pairs of purely imaginary roots exist for $\hat{L}(\lambda, \tau) = 0$ or $\tilde{L}(\lambda, \tau) = 0$ as $\tau$ increases. To achieve this objective, we substitute $\lambda = \omega i$, $\omega > 0$ into them, respectively.

Set $\mu = \beta'(I^*)I^*S^*$. For $\hat{L}(\omega i, \tau) = 0$, by separating the real and imaginary parts, we obtain

$$\cos(\hat{\omega}\tau) = \frac{(\hat{l}_{10} - \hat{l}_{12}\hat{\omega}^2)\left(\hat{l}_{20} - \hat{\omega}^2\right) + (\hat{l}_{11} - \hat{\omega}^2)\hat{l}_{21}\hat{\omega}^2}{\left(\hat{l}_{20} - \hat{\omega}^2\right)^2 + \hat{l}^2_{21}\hat{\omega}^2}\frac{1}{\mu} := \hat{C}_1, \tag{3.28}$$

$$\sin(\hat{\omega}\tau) = \frac{(\hat{l}_{10} - \hat{l}_{12}\hat{\omega}^2)\hat{l}_{21}\hat{\omega} - (\hat{l}_{11} - \hat{\omega}^2)\left(\hat{l}_{20} - \hat{\omega}^2\right)\hat{\omega}}{\left(\hat{l}_{20} - \hat{\omega}^2\right)^2 + \hat{l}^2_{21}\hat{\omega}^2}\frac{1}{\mu} := \hat{C}_2, \tag{3.29}$$

where $\hat{\omega} = \sqrt{x}$ and $x$ is a positive root (if it exists) of the following equation:

$$\hat{H}(x) := x^3 + \hat{q}_2 x^2 + \hat{q}_1 x + \hat{q}_0 = 0 \tag{3.30}$$

with

$$\hat{q}_2 = \hat{l}_{12}^2 - 2\hat{l}_{11} - \mu^2, \quad \hat{q}_1 = \hat{l}_{11}^2 - 2\hat{l}_{10}\hat{l}_{12} + 2\hat{l}_{20}\mu^2 - \hat{l}_{21}^2\mu^2, \quad \hat{q}_0 = \hat{l}_{10}^2 - \hat{l}_{20}^2\mu^2.$$

Then there exists a sequence $\hat{\tau}^n, n = 0, 1, 2, \dots$, given by

$$\hat{\tau}^n = \hat{\tau}^0 + \frac{2n\pi}{\hat{\omega}}, \hat{\tau}^0 = \begin{cases} \frac{\arccos \hat{C}_1}{\hat{\omega}}, & \hat{C}_2 \geq 0 \\ \frac{2\pi - \arccos \hat{C}_1}{\hat{\omega}}, & \hat{C}_2 < 0 \end{cases} \tag{3.31}$$

at which $\hat{L}(\lambda, \tau) = 0$ admits a pair of purely imaginary roots $\pm\hat{\omega}i$.

Similarly, for $\tilde{L}(\omega i, \tau) = 0$, we obtain

$$\cos(\tilde{\omega}\tau) = \frac{(\tilde{l}_{10} - \tilde{l}_{12}\tilde{\omega}^2)(\tilde{l}_{20} - \tilde{\omega}^2) + (\tilde{l}_{11} - \tilde{\omega}^2)\tilde{l}_{21}\tilde{\omega}^2}{(\tilde{l}_{20} - \tilde{\omega}^2)^2 + \tilde{l}_{21}^2\tilde{\omega}^2} \frac{1}{\mu} := \tilde{C}_1 \tag{3.32}$$

$$\sin(\tilde{\omega}\tau) = \frac{(\tilde{l}_{10} - \tilde{l}_{12}\tilde{\omega}^2)\tilde{l}_{21}\tilde{\omega} - (\tilde{l}_{11} - \tilde{\omega}^2)(\tilde{l}_{20} - \tilde{\omega}^2)\tilde{\omega}}{(\tilde{l}_{20} - \tilde{\omega}^2)^2 + \tilde{l}_{21}^2\tilde{\omega}^2} \frac{1}{\mu} := \tilde{C}_2. \tag{3.33}$$

Squaring and adding both equations, we see that $\tilde{\omega} = \sqrt{x}$ satisfies

$$\tilde{H}(x) := x^3 + \tilde{q}_2 x^2 + \tilde{q}_1 x + \tilde{q}_0 = 0 \tag{3.34}$$

with

$$\tilde{q}_2 = \tilde{l}_{12}^2 - 2\tilde{l}_{11} - \mu^2, \quad \tilde{q}_1 = \tilde{l}_{11}^2 - 2\tilde{l}_{10}\tilde{l}_{12} + 2\tilde{l}_{20}\mu^2 - \tilde{l}_{21}^2\mu^2, \quad \tilde{q}_0 = \tilde{l}_{10}^2 - \tilde{l}_{20}^2\mu^2.$$

If Eq. (3.34) has a positive root, then there exists a sequence $\tilde{\tau}^n, n = 0, 1, 2, \dots$, given by

$$\tilde{\tau}^n = \tilde{\tau}^0 + \frac{2n\pi}{\tilde{\omega}}, \tilde{\tau}^0 = \begin{cases} \frac{\arccos \tilde{C}_1}{\tilde{\omega}}, & \tilde{C}_2 \geq 0, \\ \frac{2\pi - \arccos \tilde{C}_1}{\tilde{\omega}}, & \tilde{C}_2 < 0, \end{cases} \tag{3.35}$$

at which $\tilde{L}(\omega i, \tilde{\tau}^n) = 0$.

Further, we can directly verify the transversality condition by the following lemma.

**Lemma 3.17** $\operatorname{sign}\left(\frac{d\lambda}{d\tau}\Big|_{\tau=\hat{\tau}^n}\right) = \operatorname{sign}\left(\frac{\hat{H}(x)}{dx}\Big|_{x=(\hat{\omega})^2}\right)$ *and* $\operatorname{sign}\left(\frac{d\lambda}{d\tau}\Big|_{\tau=\tilde{\tau}^n}\right) = \operatorname{sign}\left(\frac{\tilde{H}(x)}{dx}\Big|_{x=(\tilde{\omega})^2}\right)$, *where* $\hat{\tau}^n$ *and* $\tilde{\tau}^n$ *are given by* (3.31) *and* (3.35), *respectively.*

**Theorem 3.18** *Consider system* (3.1) *with* (3.26). *If Eq.* (3.30) *has a positive root* $\hat{x}$ *with* $\hat{H}'(\hat{x}) \neq 0$ (*or Eq.* (3.34) *has a positive root* $\tilde{x}$ *satisfying* $\tilde{H}'(\tilde{x}) \neq 0$), *then Hopf bifurcation occurs at* $\tau = \hat{\tau}^n$ (*or* $\tau = \tilde{\tau}^n$), $n = 0, 1, \dots$.

## 4 Numerical simulations

We first carry out some numerical simulations for system (2.1). To facilitate comparison with the findings presented in [40–42], we adopt specific parameters as follows:

$$A = 0.2, \hat{\beta} = 1, d = 0.2, \epsilon = 0.1, \gamma = 0.05, \alpha = 0.06 \tag{4.1}$$

and set $\tilde{\beta} = \theta\hat{\beta}$, $\theta \in (0, 1]$. For convenience, we label Model (2.1) as Model-I for $f$ given in (2.2) and Model-II for $f$ given in (2.3) with $k = \kappa\bar{S}$ and $n = 1$, respectively. That is, in Model-I,

$$\beta(I) = \hat{\beta} - \theta\left(1 - e^{-hI}\right)\hat{\beta},$$

and in Model-II,

$$\beta(I) = \hat{\beta} - \theta\hat{\beta}\frac{I}{\kappa\bar{S} + I}.$$

We examine how precaution related parameters affect the disease dynamics. That is, $\theta$, $h$ for Model-I and $\theta$, $\kappa$ for Model-II. It follows from the expression of $\mathcal{R}_0^1$ that $\tilde{\beta}$ does not affect the value of $\mathcal{R}_0^1$. But as we will see, $\tilde{\beta}$ does influence the stability of the EE.

With (4.1), the basic reproduction number is $\mathcal{R}_0^1 = 2.8571$. For Model-I with $\theta = 0.9$ and $h = 3.5$, we obtain $E_* = (0.6512, 0.2061, 0.0396)$ and a Hopf bifurcation occurs at $\tau = \tau^{0,+} \approx 14.574$. The EE $E^*$ is stable for $\tau \in [0, 14.574)$ and is unstable for $\tau > \tau^{0,+}$ (see the bifurcation diagram presented in the left panel of Fig. 2).

For Model-II with $\theta = 0.9$ and $\kappa = 0.03$, the EE is $E_* = (0.8940, 0.0626, 0.0120)$ and the first Hopf bifurcation value is $\tau \approx 9.759$. The bifurcation diagram presented in the right panel of Fig. 2 indicates that the EE is stable for $\tau \in [0, 9.759)$ and a stable periodic solution may appear for $\tau > 0.759$.

Next we sketch the regions in parameter space to determine whether a Hopf bifurcation occurs for some critical values of $\tau$. We restrict the parameter ranges as $\theta \in [0.6, 1]$, $h \in [2.5, 3.5]$ and $\kappa \in [0.01, 0.45]$. The stability region is depicted in Fig. 3, where the yellow-colored region indicates that $E_*$ is stable for all $\tau \geq 0$, while the blue-colored region represents the case where the EE $E_*$ becomes unstable as $\tau$ surpasses the critical value $\tau^{0,+}$. It is seen from Fig. 3 that a higher value of $\theta$, which corresponds to a higher value of

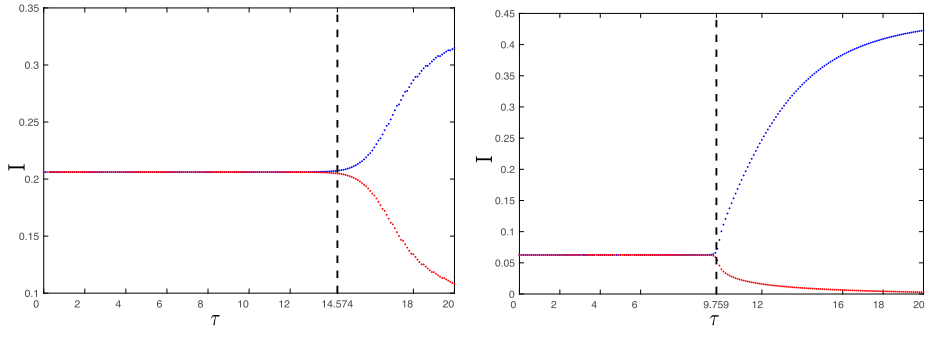


**Figure 2** Left: Bifurcation diagram of Model-I with $h = 3.5$; Right: Bifurcation diagram of Model-II with $\kappa = 0.03$. The parameters are $A = 0.2$, $\hat{\beta} = 1$, $d = 0.2$, $\epsilon = 0.1$, $\gamma = 0.05$, $\alpha = 0.06$, and $\theta = 0.9$, resulting in $\mathcal{R}_0^1 = 2.8571$

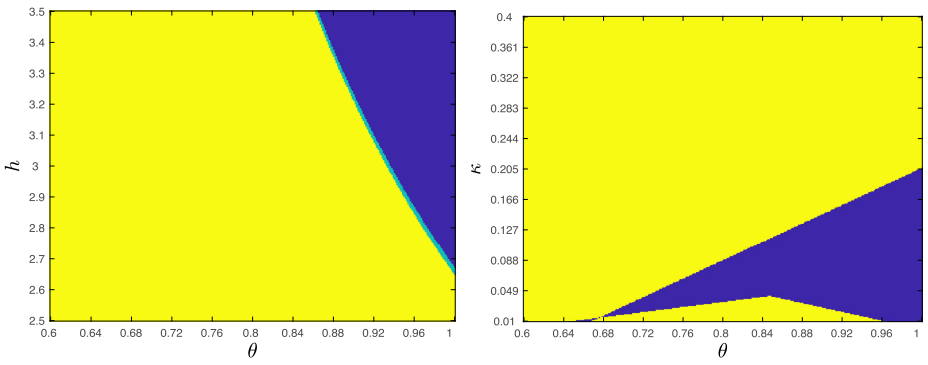


**Figure 3** The stable regions of the endemic equilibrium. Left: Model-I; Right: Model-II. The endemic equilibrium $E_*$ is stable for $\tau \geq 0$ in the yellow-colored region, while in the blue-colored region, the endemic equilibrium is stable for $\tau \in [0, \tau^{0,+})$ and a Hopf bifurcation occurs at $\tau = \tau^{0,+}$

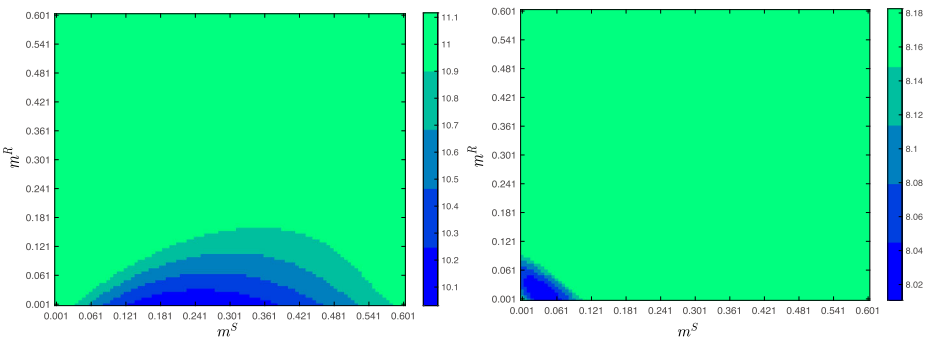


**Figure 4** The influence of travel rates on the Hopf bifurcation values $\tau^{0,+}$. Left: Model-III with $\theta = 0.9$, $h = 3.5$; Right: Model-IV with $\theta = 0.9$ and $\kappa = 0.01$ in the special case (3.26) with parameters given in (4.1) and $m^I = 0$, where both $m^S$ and $m^R$ fall within the range $[0.001, 0.601]$

$\tilde{\beta}$, is associated with a greater likelihood of the occurrence of Hopf bifurcation leading to cyclical behavior in disease incidence.

Next we carry out numerical simulations for Model-III and Model-IV, which correspond to Model-I and Model-II in a two-patch environment, respectively. For the special case (3.26) with parameter values given in (4.1), if $\mathcal{R}_0 > 1$, then $E_1^B$, $E_2^B$, and $E^*$ exist. It can be seen that the travel rates $m^S$ and $m^R$ do not affect the value of $E^*$. However, they do influence the values of $\hat{\tau}^n$ and those of $E_1^B$ and $E_2^B$. Moreover, travel rates also influence the occurrence of Hopf bifurcations. To illustrate this, we set $m^S$, $m^R$ both in the range of $[0.001, 0.601]$ and numerically calculate the values of $\tau^{0,+}$ for Model-III with $\theta = 0.9$, $h = 3.5$, and Model-IV with $\theta = 0.9$ and $\kappa = 0.01$ in Fig. 4. This implies that travel rates do influence the stability of $E^*$ when $\tau > 0$.

Next we fix $m^R = 0.01$ and $\tau = 15$ to explore the impact of $m^S$ on the solutions of system (3.26) for different values of $m^S$, specifically $m^S = 0, 0.02, 0.04, 0.06$. At these parameter settings, both Model-III and Model-IV exhibit periodic oscillations, as depicted in Fig. 5. It is evident that $m^S$ significantly influences both the amplitude and phase, particularly for Model-III.

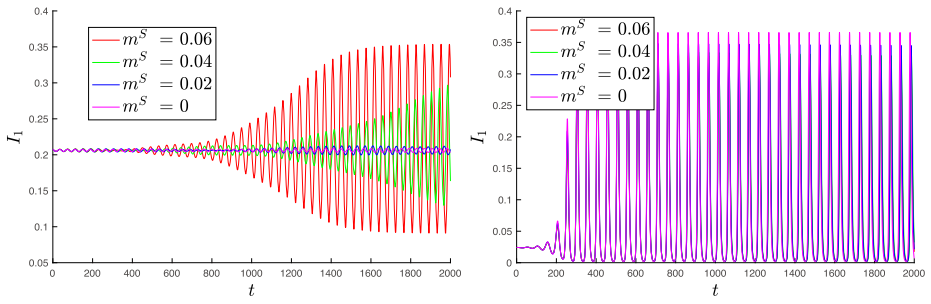


**Figure 5** The solutions of Model-III (left) and Model-IV (right) for $m^S = 0, 0.02, 0.04, 0.06$, while keeping $m^R = 0.01$ and $\tau = 15$ in the special case (3.26) with the parameters of (4.1) and $m^I = 0$

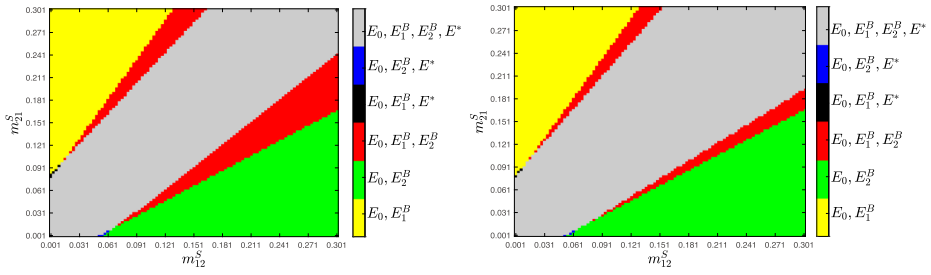


**Figure 6** The existence of equilibria for Model-III (Left) with $h_1 = 1$ and $h_2 = 3$, and Model-IV (Right) with $\kappa_1 = 0.1$ and $\kappa_2 = 0.15$, under the condition of $m_{12}^I = m_{21}^I = 0$. The parameters $m_{21}^S$ and $m_{12}^S$ vary within the ranges [0.001, 0.3001] and [0.001, 0.3001], respectively. Other parameters remain as defined in (4.2)

Next we consider some heterogeneous cases. To this end, we set the baseline parameters as

$$\begin{cases} A_1 = 0.2, \hat{\beta}_1 = 1.1, \theta_1 = 0.9, d_1 = 0.2, \epsilon_1 = 0.1, \gamma_1 = 0.5, \alpha_1 = 0.06; \\ A_2 = 0.2, \hat{\beta}_2 = 1, \theta_2 = 0.8, d_2 = 0.2, \epsilon_1 = 0.1, \gamma_2 = 0.5, \alpha_1 = 0.06; \\ h_1 = 1, h_2 = 3, \kappa_1 = 0.1, \kappa_2 = 0.15. \end{cases} \tag{4.2}$$

First we illustrate the impact of $m_{21}^S$ and $m_{12}^S$ on the existence of equilibria in Fig. 6. There are six different colored regions corresponding to six different scenarios for the existence of equilibria: $E_0$ always exists, $E_1^B$ exists in yellow-, red-, black-, and gray-colored regions, $E_2^B$ exists in green-, red-, blue-, and gray-colored regions, and $E^*$ exists in black-, blue-, and gray-colored regions.

In conclusion, non-pharmaceutical interventions (NPIs) and media coverage can result in behavioral changes among susceptible individuals, meaning that only those who are practically susceptible are likely to come into contact with infectious individuals and thereby face the risk of infection. The varying infection force functions, resulting from precautionary measures, have a considerable influence on the stability of equilibria within the system. It is observed that a higher reduction rate $\tilde{\beta}$ in infection correlates with an increased likelihood of cyclical variations in disease incidence. While the parameters $\hat{\beta}$, $m_{ij}^S$, $m_{ij}^I$, and $m_{ij}^R$ influence the existence of equilibria and associated thresholds, the parameters $\tau$, $\alpha$, $\tilde{\beta}$, and $f(I)$ do not affect these aspects, but instead impact the stability of the equilibria and the occurrence of Hopf bifurcations. The dispersal of recovered individ-

uals ($m_{ij}^R \geq 0$) does not impact the existence or stability of equilibrium points. If infected individuals are allowed to travel between patches ($m_{ij}^I > 0$), the disease will either die out or persist in both patches. If dispersal of infected individuals is restricted ($m_{ij}^I = 0$) and only susceptible individuals can disperse ($m_{ij}^S > 0$), then the system may exhibit complex dynamical behaviors. For instance, boundary equilibrium points can arise and the disease may only persist in one patch.

## 5 Summary and discussions

In this study, we integrated precaution, immunity loss and dispersal (travel between patches) into a two-patch SIRS model and studied its dynamics. From the analysis of the single patch SIRS model, we found that the precaution and its delayed effects can cause oscillatory behavior in endemic disease situation due to the occurrence of Hopf bifurcation. We then analyzed the impact of dispersal (travel) on disease spread patterns. Theoretical analyses were performed to ascertain the stability of disease-free equilibria and to determine the basic reproduction number. We identified several travel-related thresholds to determine the existence of equilibria including the disease-free equilibrium, boundary equilibria (disease persists in one patch only) and the endemic equilibrium (disease persists in both patches). For a special case, we established the stability result for the endemic equilibrium and conducted the Hopf bifurcation analysis.

Numerical simulations were performed to illustrate the impacts of precaution and travel on disease dynamics. The investigation highlighted the considerable influence of varying infection force functions on the stability of equilibria within the system. It is seen that a higher reduction rate in infection due to precaution correlates with an elevated possibility for the observation of cyclical variation in disease incidence. This indicates the crucial role of timely and precise dissemination of disease information in mitigating disease transmission.

The findings also suggest that the travel of individuals in different disease states yields diverse effects on the system. For instance, when infected individuals remain stationary, boundary equilibrium points, indicating the presence of disease in one patch only, can arise. However, the mobility of recovered individuals does not influence the occurrence of disease equilibria. The travel of susceptible individuals exerts intricate effects on the presence and stability of equilibria within the system. Thus, our two-patch model allows us to capture the heterogeneity of disease-related features across different regions and analyze the combined effects of dispersal on disease transmission dynamics. Furthermore, the implementation of public precaution significantly reduces infection levels, even when people disperse at a relatively higher rate. These insights emphasize that restrictive measures targeting individual mobility may not consistently yield beneficial outcomes in curbing disease transmission, thereby highlighting the necessity for governments to holistically consider multiple factors in disease prevention and control efforts.

Certainly, given the myriad factors encompassed in the model and their intricate interplay, numerous unanswered questions remain. For instance, there is a pressing need for comprehensive investigations into the stability analysis of equilibria within heterogeneous environments. Further exploration in areas such as parameter estimation, model analysis, and the development of policies tailored to real-world contexts are also worthy of attempting. To capture the heterogeneous of individuals' immunity and infectiousness in the study

of patchy infectious diseases, it is also crucial to distinguish between susceptible individuals based on their immunity power, as well as between symptomatic and asymptomatic infections. Another challenge is to illustrate how infection force changes under different influencing factors. Future work may consider modeling infection force based on individual behavioral shifts, media influence, spatial heterogeneity, and other factors to more accurately reflect real conditions. Additionally, there is a need for further research into how media reporting influences travel rates and the resulting dynamics of disease within multi-patch environments.

## Appendix A: The proof of Theorem 2.2

*Proof* Summing all three equations of system (2.1), we have

$$\frac{dN(t)}{dt} = A - dN - \epsilon I \leq A - dN.$$

This means

$$\lim_{t\to\infty} \sup S(t) \leq \lim_{t\to\infty} \sup N(t) \leq \frac{A}{d}.$$

When $\mathcal{R}_0^1 < 1$, we can choose a sufficiently small $\eta > 0$ such that

$$\hat{\beta}\left(\frac{A}{d} + \eta\right) < (\gamma + \epsilon + d).$$

Then, for large $T > 0$, we have

$$S(t) \leq \frac{A}{d} + \eta, t > T.$$

When $t > T$, the second equation of system (2.1) has the following linear ordinary differential equation (ODE) as a comparison equation, where all solutions converge to 0 as $t \to \infty$:

$$\frac{dI(t)}{dt} = \left(\hat{\beta}\left(\frac{A}{d} + \eta\right) - (\gamma + \epsilon + d)\right) I(t).$$

Therefore, employing a comparison argument, it can be concluded that the $I(t)$ component of the solution of system (2.1) also tends to 0 as $t \to \infty$.

Thus, the third equation of system (2.1) has a limit equation

$$\frac{dR(t)}{dt} = -(d + \alpha)R(t).$$

This equation exhibits global convergence dynamics with $R(t)$ approaching 0 as $t \to \infty$.

Thus, the first equation of system (2.1) exhibits global convergence dynamics, with $S(t)$ approaching $\frac{A}{d}$ as $t \to \infty$, as it has a limit equation

$$\frac{dS(t)}{dt} = A - dS(t).$$

So, according to the theory of asymptotically autonomous systems, every solution of system (2.1) with a nonnegative initial condition (2.4) converges to $E_0$ as $t \to \infty$, provided that $\mathcal{R}_0^1 < 1$. □

## Appendix B: The characteristic equation of one-patch SIRS

By a straightforward calculation, we know that

$$
\begin{aligned}
\bar{p}_2\bar{p}_1 - \bar{p}_0 &= \Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big) \\
&\quad \times \Big(d(\alpha + d) + (d + \alpha + a)\beta(I_*)I_* - (\alpha + 2d)\beta'(I_*)I_*S_*\Big) \\
&\quad - \Big((\epsilon + d)(\alpha + d) + d\gamma\Big)\beta(I_*)I_* + d(\alpha + d)\beta'(I_*)I_*S_* \\
&= \Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)d(\alpha + d) \\
&\quad + \Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(d + \alpha + a)\beta(I_*)I_* \\
&\quad - \Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(\alpha + 2d)\beta'(I_*)I_*S_* \\
&\quad - \Big((\epsilon + d)(\alpha + d) + d\gamma\Big)\beta(I_*)I_* + d(\alpha + d)\beta'(I_*)I_*S_* \\
&= \Big(\alpha + 2d + \beta(I_*)I_*\Big)d(\alpha + d) - \beta'(I_*)I_*S_*d(\alpha + d) \\
&\quad + (\alpha + 2d)(d + \alpha + a)\beta(I_*)I_* + \Big(\beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(d + \alpha + a)\beta(I_*)I_* \\
&\quad - \beta'(I_*)\Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(\alpha + 2d)I_*S_* \\
&\quad - \Big((\epsilon + d)(\alpha + d) + d\gamma\Big)\beta(I_*)I_* + d(\alpha + d)\beta'(I_*)I_*S_* \\
&= \Big(\alpha + 2d + \beta(I_*)I_*\Big)d(\alpha + d) \\
&\quad + +\Big(\beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(d + \alpha + a)\beta(I_*)I_* \\
&\quad - \beta'(I_*)\Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(\alpha + 2d)I_*S_* \\
&\quad + \Big((\alpha + 2d)(d + \alpha + a) - (\epsilon + d)(\alpha + d) - d\gamma\Big)\beta(I_*)I_* \\
&= \Big(\alpha + 2d + \beta(I_*)I_*\Big)d(\alpha + d) \\
&\quad + +\Big(\beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(d + \alpha + a)\beta(I_*)I_* \\
&\quad - \beta'(I_*)\Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(\alpha + 2d)I_*S_* \\
&\quad + \Big((\alpha + 2d)(d + \alpha + a) - a(\alpha + d) + \alpha\gamma\Big)\beta(I_*)I_* > 0 \\
&= \Big(\alpha + 2d + \beta(I_*)I_*\Big)d(\alpha + d) \\
&\quad + +\Big(\beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(d + \alpha + a)\beta(I_*)I_*
\end{aligned}
$$

$$
\begin{aligned}
&- \beta'(I_*)\Big(\alpha + 2d + \beta(I_*)I_* - \beta'(I_*)I_*S_*\Big)(\alpha + 2d)I_*S_* \\
&+ \Big((\alpha + 2d)(d + \alpha) + ad + \alpha\gamma\Big)\beta(I_*)I_* > 0.
\end{aligned}
$$

## Appendix C: The proof of Theorem 3.1

*Proof* For system (3.1), we know that there exists a unique solution $x(t;\phi)$ for $t \in (0, t_m)$ with $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6) \in X$, as guaranteed by the fundamental theory of functional differential equations [23], where $(0, t_m)$ represents the maximal interval of existence. Additionally, it is straightforward to demonstrate that these solutions are nonnegative, as shown in [39, Proposition A.17]. The nonnegativity implies that the existence holds for all $t \geq 0$.

Moving on, we proceed to demonstrating the boundedness of the solution. For this purpose, we introduce $N_i(t) = S_i(t) + I_i(t) + R_i(t)$ for $i = 1, 2$ and define $N(t) = N_1(t) + N_2(t)$. Then,

$$
\frac{dN}{dt} = \sum_{i=1}^{2}(A_i - \epsilon_i I_i - d_i N_i).
$$

The nonnegativity of the solution implies

$$
A_1 + A_2 - \max\{d_1 + \epsilon_1, d_2 + \epsilon_2\}N \leq \frac{dN}{dt} \leq A_1 + A_2 - \min\{d_1, d_2\}N.
$$

Hence,

$$
N(t) \in \left[\min\left\{\frac{A_1 + A_2}{\max\{d_1 + \epsilon_1, d_2 + \epsilon_2\}}, N(0)\right\}, \max\left\{\frac{A_1 + A_2}{\min\{d_1, d_2\}}, N(0)\right\}\right]. \qquad \square
$$

## Appendix D: The proof of the monotonicity of $g_1$, $g_2$, and $G_1$

*Proof* Firstly, by direct calculations, and noting $\beta_i'(\cdot) < 0$, we have

$$
\begin{aligned}
\frac{d}{dx_1}u_1^{-1}(x_1) &= \left(\frac{d}{dI_1}u_1(I_1)\right)^{-1} = \left(\alpha_2 r_{21} - \frac{a_1 m_{21}^S}{\left(\beta_1(I_1)\right)^2}\frac{d}{dI_1}\beta_1(I_1)\right)^{-1} > 0, \\
\frac{d}{dx_2}u_2^{-1}(x_2) &= \left(\frac{d}{dI_2}u_2(I_2)\right)^{-1} = \left(\alpha_1 r_{12} - \frac{a_2 m_{12}^S}{\left(\beta_2(I_2)\right)^2}\frac{d}{dI_2}\beta_2(I_2)\right)^{-1} > 0,
\end{aligned}
$$

and

$$
\begin{aligned}
g_2'(x_2) &= \frac{d}{dx_2}g_2(x_2) \\
&= -\frac{a_2(d_2 + m_{12}^S)}{\left(\beta_2(u_2^{-1}(x_2))\right)^2}\beta_2'(u_2^{-1}(x_2))\frac{d}{dx_2}u_2^{-1}(x_2) + (a_2 - \alpha_2 r_{22})\frac{d}{dx_2}u_2^{-1}(x_2) > 0,
\end{aligned}
$$

$$
g_1'(x_1) = \frac{d}{dx_1}g_1(x_1)
$$

$$= -\frac{a_1(d_1 + m_{21}^S)}{\left(\beta_1(u_1^{-1}(x_1))\right)^2}\beta_1'(u_1^{-1}(x_1))\frac{d}{dx_1}u_1^{-1}(x_1) + (a_1 - \alpha_1 r_{11})\frac{d}{dx_1}u_1^{-1}(x_1) > 0.$$

So $g_1$ and $g_2$ are increasing functions.

Secondly, because

$$\begin{aligned} a_2 - \alpha_2 r_{22} &= d_2 + \epsilon_2 + \gamma_2 - \frac{\alpha_2(\alpha_1 + d_1 + m_{21}^R)}{(\alpha_1 + d_1)(\alpha_2 + d_2) + m_{21}^R(d_2 + \alpha_2) + m_{12}^R(d_1 + \alpha_1)}\gamma_2 \\ &= d_2 + \epsilon_2 + \frac{d_2\alpha_1 + d_1 d_2 + d_2 m_{21}^R + d_1 m_{12}^R + \alpha_1 m_{12}^R}{(\alpha_1 + d_1)(\alpha_2 + d_2) + m_{21}^R(d_2 + \alpha_2) + m_{12}^R(d_1 + \alpha_1)}\gamma_2 \\ &> \frac{\alpha_1 m_{12}^R}{(\alpha_1 + d_1)(\alpha_2 + d_2) + m_{21}^R(d_2 + \alpha_2) + m_{12}^R(d_1 + \alpha_1)}\gamma_2 \\ &= \alpha_1 r_{12} > 0, \end{aligned}$$

and

$$\begin{aligned} a_1 - \alpha_1 r_{11} &= d_1 + \epsilon_1 + \gamma_1 - \frac{\alpha_1(\alpha_2 + d_2 + m_{12}^R)}{(\alpha_1 + d_1)(\alpha_2 + d_2) + m_{21}^R(d_2 + \alpha_2) + m_{12}^R(d_1 + \alpha_1)}\gamma_1 \\ &= d_1 + \epsilon_1 + \frac{d_1\alpha_2 + d_1 d_2 + d_1 m_{12}^R + d_2 m_{21}^R + \alpha_2 m_{21}^R}{(\alpha_1 + d_1)(\alpha_2 + d_2) + m_{21}^R(d_2 + \alpha_2) + m_{12}^R(d_1 + \alpha_1)}\gamma_1 \\ &> \frac{\alpha_2 m_{21}^R}{(\alpha_1 + d_1)(\alpha_2 + d_2) + m_{21}^R(d_2 + \alpha_2) + m_{12}^R(d_1 + \alpha_1)}\gamma_1 \\ &= \alpha_2 r_{21} > 0, \end{aligned}$$

we get

$$\begin{aligned} &\frac{d}{dx_2}g_2(x_2) \times \frac{d}{dx_1}g_1(x_1) \\ &= \frac{-\frac{a_2(d_2+m_{12}^S)}{\left(\beta_2(I_2)\right)^2}\beta_2'(I_2) + (a_2 - \alpha_2 r_{22})}{\alpha_1 r_{12} - \frac{a_2 m_{12}^S}{\left(\beta_2(I_2)\right)^2}\beta_2'(I_2)} \times \frac{-\frac{a_1(d_1+m_{21}^S)}{\left(\beta_1(I_1)\right)^2}\beta_1'(I_1) + (a_1 - \alpha_1 r_{11})}{\alpha_2 r_{21} - \frac{a_1 m_{21}^S}{\left(\beta_1(I_1)\right)^2}\beta_1'(I_1)} \\ &> \frac{-\frac{a_2\beta_2'(I_2)}{\left(\beta_2(I_2)\right)^2}(d_2 + m_{12}^S) + \alpha_1 r_{12}}{-\frac{a_2\beta_2'(I_2)}{\left(\beta_2(I_2)\right)^2}m_{12}^S + \alpha_1 r_{12}} \times \frac{-\frac{a_1\beta_1'(I_1)}{\left(\beta_1(I_1)\right)^2}(d_1 + m_{21}^S) + \alpha_2 r_{21}}{-\frac{a_1\beta_1'(I_1)}{\left(\beta_1(I_1)\right)^2}m_{21}^S + \alpha_2 r_{21}} \\ &> 1. \end{aligned}$$

Thus,

$$G_1'(x_1) = \frac{d}{dx_1}G_1(x_1) = \frac{d}{dx_2}g_1(x_2) \times \frac{d}{dx_1}g_2(x_1) - 1 > 0.$$

This implies $G_1(x_1)$ is an increasing function. □

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**Availability of data and materials**
All data generated or analysed during this study are included in this published article.

## Declarations

**Competing interests**
The authors declare that they have no competing interests.

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