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Research article

Analysis of a two-patch SIS model with saturating contact rate and one-directing population dispersal

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Abstract: In this paper, a two-patch SIS model with saturating contact rate and one-directing population dispersal is proposed. In the model, individuals can only migrate from patch 1 to patch 2. The basic reproduction number R_0^1 of patch 1 and the basic reproduction number R_0^2 of patch 2 is identified. The global dynamics are completely determined by the two reproduction numbers. It is shown that if $R_0^1 < 1$ and $R_0^2 < 1$, the disease-free equilibrium is globally asymptotically stable; if $R_0^1 < 1$ and $R_0^2 > 1$, there is a boundary equilibrium which is globally asymptotically stable; if $R_0^1 > 1$, there is a unique endemic equilibrium which is globally asymptotically stable. Finally, numerical simulations are performed to validate the theoretical results and reveal the influence of saturating contact rate and migration rate on basic reproduction number and the transmission scale.

Keywords: saturating contact rate; population dispersal; basic reproduction number; global asymptotic stability

1. Introduction

Population migration is a common phenomenon. With the migration of population, infectious diseases can easily spread from one area to another, so it is meaningful to consider population migration when studying the spread of infectious diseases [1–7].

Wang and Mulone [2] established an SIS infectious disease model with standard incidence based on two patches. It is proved that the basic reproduction number is the threshold of the uniform persistence and disappearance of the disease. The dispersal rate of the population will make the infectious disease persist or disappear in all patches. There will be no the phenomena that infectious diseases persists in one patch but disappears in the other.

Sun et al. [3] put forward an SIS epidemic model with media effect in a two patches setting. Under the assumption that the migration matrix is irreducible, it is proved that if the basic reproduction number is greater than 1 then the system persists and solutions converge to an endemic equilibrium

and that if the basic reproduction number is less than 1 then solutions tend to an equilibrium without disease.

Gao et al. [5, 6] studied an SIS multi-patch model with variable transmission coefficients. Their results show that the basic reproduction number R_0 is a threshold parameter of the disease dynamics.

All the patch models referenced above assume that the migration matrix is irreducible. The studies in which the migration matrix is reducible are few. Therefore, based on the case of two patches, we consider that the individuals can only migrate from one patch to the other. In this case, the migration matrix is reducible. It can characterize the phenomenon that individuals migrate in one direction between two regions, such as, from the rural patch to the urban one [8] and from a small community hospital to a large teaching hospital [4].

It is well known that the incidence rate plays an important role in the modeling of infectious disease. Considering the saturation phenomenon for numerous infected individuals, Capasso and Serio [9] first introduce a nonlinear bounded function $g(I)$ to form the interaction term $g(I)S$ in 1978. It can characterize the behavioral changes of individuals, such as wearing masks or reducing their social activities and direct contact with others with the increase of infectious individuals. After that, the saturation incidence rate has attracted much attention and various nonlinear types of incidence rate are employed. The most commonly used types are Holling type II $\frac{\lambda SI}{1+\alpha I}$ [10–12] and $\frac{\beta SI}{1+\alpha S}$ [13], Monod-Haldane type $\frac{\lambda SI}{1+\alpha I^2}$ [14], Beddington-DeAngelis type $\frac{\lambda SI}{1+\alpha S+\beta I}$ [15–17] and Crowley-Martin type $\frac{\lambda SI}{(1+\alpha S)(1+\beta I)}$ [18, 19].

In this paper, we consider infectious disease transmission models with saturation incidence rate. The rest of this paper is organized as follows: In Section 2, we establish a two-patch SIS model with saturating contact rate and one-directing population dispersal. We discuss the existence of disease-free equilibrium, boundary equilibrium and endemic equilibrium and prove the global asymptotic stability of the equilibriums in Section 3. In Section 4, we perform simulations to illustrate the results and analyze the effect of the contact rate and population migration on epidemic transmission. Finally, we discuss in Section 5.

2. Model formulation

In the two patches, the population is divided into two states: susceptible and infective. Thus we can establish a two-patch SIS model with saturating contact rate and one-directing population dispersal

$$\begin{cases} \dfrac{dS_1(t)}{dt} = A_1 - d_1S_1 - \beta_1S_1\frac{I_1}{1+\alpha_1I_1} - mS_1 + \gamma_1I_1, \\ \dfrac{dS_2(t)}{dt} = A_2 - d_2S_2 - \beta_2S_2\frac{I_2}{1+\alpha_2I_2} + mS_1 + \gamma_2I_2, \\ \dfrac{dI_1(t)}{dt} = \beta_1S_1\frac{I_1}{1+\alpha_1I_1} - d_1I_1 - mI_1 - \gamma_1I_1, \\ \dfrac{dI_2(t)}{dt} = \beta_2S_2\frac{I_2}{1+\alpha_2I_2} - d_2I_2 + mI_1 - \gamma_2I_2, \end{cases} \tag{2.1}$$

where S_i is the number of susceptible population in patch i ($i = 1, 2$), I_i is the number of infective population in patch i ($i = 1, 2$), A_i is the recruitment into patch i ($i = 1, 2$), d_i is the natural mortality rate, γ_i is the recovery rate of an infective individual in patch i ($i = 1, 2$), m is the migration rate form patch 1 to patch 2. Since the individuals can migrate from the first patch to the second, patch 1 is the

source patch and patch 2 is the sink patch. The initial conditions is

$$S_i(0) > 0, \;\; I_i(0) \geq 0, \;\; i = 1, 2, \;\; I_1(0) + I_2(0) > 0. \tag{2.2}$$

Denote the population in patch i by N_i. Then $N_i = S_i + I_i$. From system (2.1), the differential equations governing the evolution of N_1 and N_2 are

$$\begin{cases} \dfrac{dN_1(t)}{dt} = A_1 - (d_1 + m)N_1, \\ \dfrac{dN_2(t)}{dt} = A_2 - d_2N_2 + mN_1. \end{cases} \tag{2.3}$$

Obviously, system (2.3) has a unique equilibrium $(N_1^*, N_2^*) = (\frac{A_1}{d_1+m}, \frac{A_2}{d_2} + \frac{mA_1}{d_2(d_1+m)})$ which is globally asymptotically stable for (2.3). So (2.1) is equivalent the following system

$$\begin{cases} \dfrac{dN_1(t)}{dt} = A_1 - (d_1 + -m)N_1, \\ \dfrac{dN_2(t)}{dt} = A_2 - d_2N_2 + mN_1, \\ \dfrac{dI_1(t)}{dt} = \beta_1(N_1 - I_1)\frac{I_1}{1+\alpha_1 I_1} - d_1I_1 - mI_1 - \gamma_1 I_1, \\ \dfrac{dI_2(t)}{dt} = \beta_2(N_2 - I_2)\frac{I_2}{1+\alpha_2 I_2} - d_2I_2 + mI_1 - \gamma_2 I_2. \end{cases} \tag{2.4}$$

Because $\lim\limits_{t\to\infty} N_i(t) \to N_i^*$ $(i = 1, 2)$, system (2.4) leads to the following limit system

$$\begin{cases} \dfrac{dI_1(t)}{dt} = \beta_1(N_1^* - I_1)\frac{I_1}{1+\alpha_1 I_1} - d_1I_1 - mI_1 - \gamma_1 I_1, \\ \dfrac{dI_2(t)}{dt} = \beta_2(N_2^* - I_2)\frac{I_2}{1+\alpha_2 I_2} - d_2I_2 + mI_1 - \gamma_2 I_2. \end{cases} \tag{2.5}$$

3. Mathematical analysis for system (2.5)

3.1. The invariants and equilibriums

Let $\Omega = \{(I_1, I_2)|0 \leq I_1 \leq N_1^*, 0 \leq I_2 \leq N_2^*\}$. Then Ω is invariant region for system (2.5).

Define the basic reproduction number in the two patches respectively by $R_0^1 = \frac{\beta_1 A_1}{(d_1+\gamma_1+m)^2} = \frac{\beta_1}{d_1+\gamma_1+m}N_1^*$, $R_0^2 = \frac{\beta_2}{(d_2+\gamma_2)}N_2^*$. The basic reproduction number R_0^1 gives the expected secondary infections in the source patch produced by a primary infected individual in the source patch when the population is supposed to be in the disease-free equilibrium. The basic reproduction number R_0^2 gives the expected secondary infections in the sink patch produced by a primary infected individual in the sink patch when the population is supposed to be in the disease-free equilibrium. Then we have the following theorem.

Theorem 3.1. *For the system (2.5), we have*
(i) The disease-free equilibrium $E_0 := (0, 0)$ always exists;
(ii) The boundary equilibrium $E_1 := (0, \frac{\beta_2 N_2^* - d_2 - \gamma_2}{(d_2+\gamma_2)\alpha_2+\beta_2})$ exists if $R_0^2 > 1$;
(iii) There is a unique epidemic equilibrium E_* if $R_0^1 > 1$.

Proof. (i) can be easily proved.

Let

$$\beta_1(N_1^* - I_1)\frac{I_1}{1+\alpha_1 I_1} - d_1 I_1 - mI_1 - \gamma_1 I_1 = 0, \tag{3.1}$$

$$\beta_2(N_2^* - I_2)\frac{I_2}{1+\alpha_2 I_2} - d_2 I_2 + mI_1 - \gamma_2 I_2 = 0. \tag{3.2}$$

From Eq (3.1), we can have $I_1 = 0$ always satisfies Eq (3.1). When $I_1 = 0$, from Eq (3.2), we have

$$I_2 = \frac{\beta_2 N_2^* - d_2 - \gamma_2}{(d_2+\gamma_2)\alpha_2 + \beta_2}.$$

If $R_0^2 > 1$, then $I_2 = \frac{\beta_2 N_2^* - d_2 - \gamma_2}{(d_2+\gamma_2)\alpha_2+\beta_2} > 0$. So The boundary equilibrium $E_1 := (0, \frac{\beta_2 N_2^* - d_2 - \gamma_2}{(d_2+\gamma_2)\alpha_2+\beta_2})$ exists if $R_0^2 > 1$. The conclusion (ii) is proved.

If $R_0^1 > 1$, Eq (3.1) has a positive solution $I_1^* = \frac{\beta_1 N_1^* - (d_1+m+\gamma_1)}{(d_1+m+\gamma_1)\alpha_1+\beta_1}$. Solve Eq (3.2), we have

$$I_2 = \frac{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1) \pm \sqrt{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1)^2 + 4[(d_2+\gamma_2)\alpha_2+\beta_2]mI_1}}{2[(d_2+\gamma_2)\alpha_2+\beta_2]}. \tag{3.3}$$

Substituting I_1^* into Eq (3.3), we have

$$I_2^* = \frac{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1^*) \pm \sqrt{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1^*)^2 + 4[(d_2+\gamma_2)\alpha_2+\beta_2]mI_1^*}}{2[(d_2+\gamma_2)\alpha_2+\beta_2]}.$$

Since $I_2^* \geq 0$ is meaning only, we take

$$I_2^* = \frac{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1^*) + \sqrt{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1^*)^2 + 4[(d_2+\gamma_2)\alpha_2+\beta_2]mI_1^*}}{2[(d_2+\gamma_2)\alpha_2+\beta_2]}.$$

So if $R_0^1 > 1$, there is a unique epidemic equilibrium $E_* = (I_1^*, I_2^*)$, where $I_1^* = \frac{\beta_1 N_1^* - (d_1+m+\gamma_1)}{(d_1+m+\gamma_1)\alpha_1+\beta_1}$ and $I_2^* = \frac{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1^*) + \sqrt{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_2 I_1^*)^2 + 4[(d_2+\gamma_2)\alpha_2+\beta_2]mI_1^*}}{2[(d_2+\gamma_2)\alpha_2+\beta_2]}$. The conclusion (iii) is proved.

This completes the proof of the theorem.

From the above analysis, we have the following theorem.

Theorem 3.2. *For the system (2.5), we have*
(i) If $R_0^1 < 1$ and $R_0^2 < 1$, there is the disease-free equilibrium E_0 only;
(ii) If $R_0^1 < 1$ and $R_0^2 > 1$, there are the disease-free equilibrium E_0 and the boundary equilibrium E_1;
(iii) If $R_0^1 > 1$ and $R_0^2 < 1$, there are the disease-free equilibrium E_0 and the epidemic equilibrium E_*;
(iv) If $R_0^1 > 1$ and $R_0^2 > 1$, there are the disease-free equilibrium E_0, the boundary equilibrium E_1 and the epidemic equilibrium E_*.

Remark 3.1. *Define the basic reproduction number R_0 of the system (2.5) by the spectral radius of the next generation matrix [20], we have*

$$R_0 = \rho\begin{pmatrix} \frac{\beta_1}{d_1+\gamma_1+m}N_1^* & 0 \\ -\frac{m\beta_2 N_2^*}{(d_2+\gamma_2)(d_1+\gamma_1+m)} & \frac{\beta_2}{(d_2+\gamma_2)}N_2^* \end{pmatrix},$$

where $\rho(A)$ denotes the spectral radius of a matrix A. So from the above analysis, we know that $R_0 = \max\{R_0^1, R_0^2\}$.

3.2. The stability of the disease-free equilibrium

The next, we shall discuss the local stability of the disease-free equilibrium firstly. Then we discuss the global asymptotical stability.

Theorem 3.3. *For the system (2.5), we have*
(i) If $R_0^1 < 1$ and $R_0^2 < 1$, the disease-free equilibrium E_0 is locally asymptotically stable;
(ii) If $R_0^1 > 1$ or $R_0^2 > 1$, the disease-free equilibrium E_0 is unstable.

Proof. The linearized system of (2.5) at the equilibrium E_0 is

$$\begin{cases} \dfrac{dI_1(t)}{dt} = (\beta_1 N_1^* - d_1 - m - \gamma_1)I_1, \\ \dfrac{dI_2(t)}{dt} = (\beta_2 N_2^* - d_2 - \gamma_2)I_2 + mI_1. \end{cases} \tag{3.4}$$

The associated characteristic equation of the linearized system of (3.4) at the equilibrium E_0 is

$$F(\lambda) = \begin{vmatrix} \lambda - (\beta_1 N_1^* - (d_1 + m + \gamma_1)) & 0 \\ -m & \lambda - (\beta_2 N_2^* - d_2 - \gamma_2) \end{vmatrix} = 0 \tag{3.5}$$

It is easy to see that the two eigenvalues of characteristic Eq (3.5) are

$$\lambda_1 = \beta_1 N_1^* - (d_1 + m + \gamma_1) = (R_0^1 - 1)(d_1 + m + \gamma_1)$$

and

$$\lambda_2 = \beta_2 N_2^* - d_2 - \gamma_2 = (R_0^2 - 1)(d_2 + \gamma_2).$$

So, when $R_0^1 < 1$ and $R_0^2 < 1$, the disease-free equilibrium E^0 is locally asymptotically stable; However, if $R_0^1 > 1$ or $R_0^2 > 1$, the disease-free equilibrium E^0 is unstable.

Remark 3.2. *From Theorem 3.3, we know that for the system (2.5), if $R_0 < 1$ the disease-free equilibrium E_0 is locally asymptotically stable; if $R_0 > 1$, the disease-free equilibrium E_0 is unstable.*

Theorem 3.4. *For the system (2.5), if $R_0^1 < 1$ and $R_0^2 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable.*

Proof. Since $\frac{I_i}{1+\alpha_i I_i} \le I_i$ for $i = 1, 2$, from system (2.5), we can obtain that

$$\begin{cases} \dfrac{dI_1(t)}{dt} \le (\beta_1 N_1^* - d_1 - m - \gamma_1)I_1, \\ \dfrac{dI_2(t)}{dt} \le (\beta_2 N_2^* - d_2 - \gamma_2)I_2 + mI_1. \end{cases} \tag{3.6}$$

Define an auxiliary linear system using the right hand side of (3.6) as follows

$$\begin{cases} \dfrac{dI_1(t)}{dt} = (\beta_1 N_1^* - d_1 - m - \gamma_1)I_1, \\ \dfrac{dI_2(t)}{dt} = (\beta_2 N_2^* - d_2 - \gamma_2)I_2 + mI_1. \end{cases}$$

It can be rewritten as

$$\begin{pmatrix} I_1 \\ I_2 \end{pmatrix}' = \begin{pmatrix} \beta_1 N_1^* - d_1 - m - \gamma_1 & 0 \\ m & \beta_2 N_2^* - d_2 - \gamma_2 \end{pmatrix} \begin{pmatrix} I_1 \\ I_2 \end{pmatrix}. \tag{3.7}$$

if $R_0^1 < 1$ and $R_0^2 < 1$, we can solve (3.7) and know that $\lim_{t\to\infty} I_1(t) = 0$ and $\lim_{t\to\infty} I_2(t) = 0$. By the comparison principle [21], we can conclude that when $R_0^1 < 1$ and $R_0^2 < 1$, all non-negative solutions of (2.5) satisfy $\lim_{t\to\infty} I_i(t) = 0$ for $i = 1, 2$. So the disease-free equilibrium E_0 is globally asymptotically stable.

3.3. The stability of the boundary equilibrium

In this subsection, we will discuss the local stability of the boundary equilibrium firstly. Then discuss the global asymptotical stability.

Theorem 3.5. *For the system (2.5), if $R_0^1 < 1$ and $R_0^2 > 1$, the boundary equilibrium E_1 is globally asymptotically stable.*

Proof. The Jacobian matrix at the boundary equilibrium E_1 of system (2.5) is

$$J = \begin{pmatrix} \beta_1 N_1^* - d_1 - m - \gamma_1 & 0 \\ m & \frac{(d_2+\gamma_2)(1-R_0^2)-(\beta_2\alpha_2+\alpha_2^2)\left(\frac{(d_2+\gamma_2)(R_0^2-1)}{(d_2+\gamma_2)\alpha_2+\beta_2}\right)^2}{\left(1+\alpha_2\frac{(d_2+\gamma_2)(R_0^2-1)}{(d_2+\gamma_2)\alpha_2+\beta_2}\right)^2} \end{pmatrix}.$$

The two eigenvalues of the Jacobian matrix are

$$\lambda_1 = \beta_1 N_1^* - (d_1 + m + \gamma_1) = (R_1^0 - 1)(d_1 + m + \gamma_1)$$

and

$$\lambda_2 = \frac{(d_2 + \gamma_2)(1 - R_2^0) - (\beta_2\alpha_2 + \alpha_2^2)\left(\frac{(d_2+\gamma_2)(R_2-1)}{(d_2+\gamma_2)\alpha_2+\beta_2}\right)^2}{\left(1 + \alpha_2\frac{(d_2+\gamma_2)(R_2-1)}{(d_2+\gamma_2)\alpha_2+\beta_2}\right)^2}.$$

So, when $R_0^1 < 1$ and $R_0^2 > 1$, $\lambda_1 < 0$ and $\lambda_2 < 0$. That is the boundary equilibrium E_1 is locally asymptotically stable.

For every $(I_1(0), I_2(0)) \in \Omega$, assume the solution of the system (2.5) with initial value $(I_1(0), I_2(0))$ is $(I_1(t), I_2(t))$. Since

$$\frac{dI_1(t)}{dt} = \frac{(\beta_1 + (d_1 + \gamma_1)\alpha_1 + m\alpha_1)\left(\frac{\beta_1 N_1^* - d_1 - m - \gamma_1}{\beta_1 + (d_1+\gamma_1)\alpha_1 + m\alpha_1} - I_1\right) I_1}{1 + \alpha_1 I_1},$$

if $R_0^1 < 1$, $\frac{dI_1(t)}{dt} < 0$, then $I_1(t)$ is positive and decreasing and $\lim_{t\to\infty} I_1(t) = 0$. So for sufficiently small positive number ϵ_1, there exists a T, such that $I_1(T) = \epsilon_1$ and $I_1(t) < \epsilon_1$ when $t > T$.

The following, we prove that for any $\epsilon > 0$, there exists a $T^* > T$ such that $|I_2(T^*) - \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}| < \epsilon$. And because $E_1 = (0, \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2})$ is locally asymptotically stable, we have E_1 is globally asymptotically stable.

Since

$$\frac{dI_2(t)}{dt} = \frac{(\beta_2 + (d_2+\gamma_2)\alpha_2)\left(\frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2} - I_2\right) I_2}{1 + \alpha_2 I_2} + mI_1,$$

if $I_2(T) < \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}$, then $\frac{dI_2(t)}{dt} > 0$ for $t > T$. So $I_2(t)$ is increasing and there exists T_1^* such that $|I_2(T_1^*) - \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}| < \epsilon$;

if $I_2(T) > \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}$, there are two cases:

i) $I_2(t)$ is decreasing for $t > T$. In this case, there exists $T_2^* > T$, such that $|I_2(T_2^*) - \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}| < \epsilon$;

ii) There exists $T_1 > T$, such that $\frac{dI_2(T_1)}{dt} > 0$. That is

$$\frac{(\beta_2 + (d_2+\gamma_2)\alpha_2)\left(\frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2} - I_2(T_1)\right) I_2(T_1)}{1 + \alpha_2 I_2(T_1)} + mI_1(T_1) > 0.$$

Since $I_1(t) < \epsilon_1$ for $t > T$, we have

$$\frac{(\beta_2 + (d_2+\gamma_2)\alpha_2)\left(\frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2} - I_2(T_1)\right) I_2(T_1)}{1 + \alpha_2 I_2(T_1)} + m\epsilon_1 > 0.$$

Since $I_2(T) > \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}$, we have

$$\frac{(\beta_2 + (d_2+\gamma_2)\alpha_2)\left(\frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2} - I_2(T_1)\right)}{\alpha_2 + \frac{\beta_2 + (d_2+\gamma_2)\alpha_2}{\beta_2 N_2^* - d_2 - \gamma_2}} + m\epsilon_1 > 0.$$

So

$$I_2(T_1) - \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2} < \frac{\alpha_2 + \frac{\beta_2 + (d_2+\gamma_2)\alpha_2}{\beta_2 N_2^* - d_2 - \gamma_2}}{(d_2+\gamma_2)\alpha_2 + \beta_2} m\epsilon_1.$$

If only $\epsilon_1 < \frac{(d_2+\gamma_2)\alpha_2 + \beta_2}{\left(\alpha_2 + \frac{\beta_2 + (d_2+\gamma_2)\alpha_2}{\beta_2 N_2^* - d_2 - \gamma_2}\right) m}\epsilon$, then $|I_2(T_2^*) - \frac{\beta_2 N_2^* - d_2 - \gamma_2}{\beta_2 + (d_2+\gamma_2)\alpha_2}| < \epsilon$.

It is completed.

3.4. The stability of the epidemic equilibrium

In this subsection, we will discuss the local stability of the epidemic equilibrium firstly and then discuss the global asymptotical stability.

Theorem 3.6. *For the system (2.5), if $R_0^1 > 1$, the epidemic equilibrium E_* is locally asymptotically stable.*

Proof. The Jacobian matrix at the epidemic equilibrium E_* of system (2.5) is

$$J = \begin{pmatrix} -\beta_1 \frac{I_{1*}}{1+\alpha_1 I_1^*} + \frac{\beta_1(N_1^* - I_1^*)}{(1+\alpha_1 I_1^*)^2} - d_1 - m - \gamma_1 & 0 \\ m & \frac{\beta_2 N_2^* - 2\beta_2 I_2^* - 2\beta_2\alpha_2 I_2^{*2} - (d_2+\gamma_2)(1+\alpha_2 I_2^*)^2}{(1+\alpha_2 I_2^*)^2} \end{pmatrix}$$

$$= \begin{pmatrix} (d_1 + m + \gamma_1)\frac{1 - R_0^1 - \left(\frac{\beta_1\alpha_1}{d_1+m+\gamma_1} + \alpha_1^2\right) I_1^{*2}}{(1+\alpha_1 I_1^*)^2} & 0 \\ m & \frac{\beta_2 N_2^* - 2\beta_2 I_2^* - 2\beta_2\alpha_2 I_2^{*^2} - (d_2+\gamma_2)(1+\alpha_2 I_2^*)^2}{(1+\alpha_2 I_2^*)^2} \end{pmatrix}.$$

The two eigenvalues of the Jacobian matrix are

$$\lambda_1 = (d_1 + m + \gamma_1)\frac{1 - R_0^1 - (\frac{\beta_1\alpha_1}{d_1+m+\gamma_1} + \alpha_1^2) I_1^{*2}}{(1 + \alpha_1 I_1^*)^2}$$

and

$$\lambda_2 = \frac{\beta_2 N_2^* - 2\beta_2 I_2^* - 2\beta_2\alpha_2 I_2^{*^2} - (d_2 + \gamma_2)(1 + \alpha_2 I_2^*)^2}{(1 + \alpha_2 I_2^*)^2}.$$

It is easy to see that if $R_0^1 > 1$, $\lambda_1 < 0$. The next, we need only prove the second eigenvalue $\lambda_2 < 0$ if $R_0^1 > 1$. Since $(1 + \alpha_2 I_2^*)^2 > 0$, we need only prove $\beta_2 N_2^* - 2\beta_2 I_2^* - 2\beta_2\alpha_2 I_2^{*^2} - (d_2 + \gamma_2)(1 + \alpha_2 I_2^*)^2 < 0$. Let

$$G(I_2^*) = \beta_2 N_2^* - 2\beta_2 I_2^* - 2\beta_2\alpha_2 I_2^{*^2} - (d_2 + \gamma_2)(1 + \alpha_2 I_2^*)^2.$$

Since $I_2^* \geq \frac{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_1 I_1^*)}{(d_2+\gamma_2)\alpha_2 + \beta_2}$, so

$$\begin{aligned} G(I_2^*) &= \beta_2 N_2^* - 2\beta_2 I_2^* - 2\beta_2\alpha_2 I_2^{*^2} - (d_2 + \gamma_2)(1 + \alpha_2 I_2^*)^2 \\ &\leq \beta_2 N_2^* - (\beta_2 + (d_2 + \gamma_2)\alpha_2)\frac{(\beta_2 N_2^* - d_2 - \gamma_2 + m\alpha_1 I_1^*)}{(d_2 + \gamma_2)\alpha_2 + \beta_2} - d_2 - \gamma_2 \\ &= -m\alpha_1 I_1^* < 0. \end{aligned}$$

This completes the proof.

Theorem 3.7. *For the systeml (2.5), if $R_0^1 > 1$, the epidemic equilibrium E_* is globally asymptotically stable.*

Proof. Since E_* is stable when $R_0^1 > 1$, we need only prove E_* is globally attractive.

Consider the equation

$$\frac{dI_1(t)}{dt} = I_1\left(\frac{\beta_1(N_1^* - I_1)}{1 + \alpha_1 I_1} - d_1 - m - \gamma_1\right).$$

Let

$$f_1(I_1) = \frac{\beta_1(N_1^* - I_1)}{1 + \alpha_1 I_1} - d_1 - m - \gamma_1.$$

Then $f_1'(I_1) = \beta_1\frac{-1 - N_1^*\alpha_1}{(1+\alpha_1 I_1)^2} < 0$. So $f_1(I_1)$ is a monotonic decreasing function for all $I_1 > 0$. Furthermore, $f_1(0) > 0$, $f_1(N_1^*) < 0$ and $f_1(I_1^*) = 0$ when $R_0^1 > 1$. That means if $I_1 \in (0, I_1^*)$, $f_1(I_1) > 0$ and $\frac{dI_1(t)}{dt} > 0$; if $I_1 \in (I_1^*, N_1^*)$, $f_1(I_1) < 0$ and $\frac{dI_1(t)}{dt} < 0$. Hence $\lim_{t\to\infty} I_1(t) = I_1^*$. By Eq (3.3), $\lim_{t\to\infty} I_2(t) = I_2^*$. Thus E_* is globally asymptotically stable.

The results about the existence and stability of equilibria are summarized in Table 1.

Table 1. Existence and stability of equilibria.

Conditions	E_0	E_1	E_*
$R_0^1 < 1$ and $R_0^2 < 1$	Yes (GAS)	No	No
$R_0^1 < 1$ and $R_0^2 > 1$	Yes (Unstable)	Yes (GAS)	No
$R_0^1 > 1$ and $R_0^2 < 1$	Yes (Unstable)	No	Yes (GAS)
$R_0^1 > 1$ and $R_0^2 > 1$	Yes (Unstable)	Yes (Unstable)	Yes (GAS)

Remark 3.3. *From Theorems 3.5 and 3.7, we know that for the system (2.5), if $R_0 > 1$, the infectious disease is uniformly persistent. However, the infectious disease is not always uniformly persistent in every patch. If $R_0 > 1$, but $R_0^1 < 1$, the disease is uniformly persistent in the sink patch, but is extinct in the source patch. If $R_0^1 > 1$, the disease is always uniformly persistent in every patch. This is a different conclusion resulted by the reducible migration matrix.*

Remark 3.4. *From Theorem 3.5, in the case that $R_0^1 < 1$ and $R_0^2 > 1$, the infection does not persist in the source patch but is able to persist in the sink patch. So, in the early stage of the spread of infectious disease, the sink patch should assess the reproduction number R_0^2 reasonably and take control measures timely to prevent the epidemic.*

4. Simulations

In this section, we carry on numerical simulations to verify the theoretical conclusions, reveal the influence of the migration rate form patch 1 to patch 2 on the basic reproduction number, the transmission scale and transmission speed, and discuss the influence of the parameters α_1 and α_2 that measure the inhibitory effect on the basic reproduction number, the transmission scale and transmission speed.

To numerically illustrate the theoretical results, we need to choose some parameter values (see Table 2).

Table 2. Description and values of parameters.

Parameter	Description	Value
A_1	the recruitment rate of the population in patch 1	0.018 (Figures 1 and 2) 0.03 (Figure 3)
A_2	the recruitment rate of the population in patch 2	0.0005 (Figure 1) 0.004 (Figures 2 and 3)
β_1	the transmission rate in patch 1	0.00001
β_2	the transmission rate in patch 2	0.00005
α_1	the parameter that measure the inhibitory effect in patch 1	0.02
α_2	the parameter that measure the inhibitory effect in patch 1	0.02
d_1	the death rate in patch 1	0.0003
d_2	the death rate in patch 2	0.0003
γ_1	the death rate in patch 1	0.0001
γ_2	the death rate in patch 2	0.0001
m	the migration rate form the patch 1 to the patch 2	0.00005

We verify the theoretical conclusions firstly. Denote the density of the infective individuals in patch 1 by $i_1(t) = \frac{I_1(t)}{N_1^*}$. Denote the density of the infective individuals in patch 2 by $i_2(t) = \frac{I_2(t)}{N_2^*}$. Figure 1 shows the evolution of the density of infective individuals in the two patches when $R_0^1 = 0.8889$ and $R_0^2 = 0.7500$. As predicted by the analytic calculation, the infectious disease in the two patches will disappear eventually. Figure 2 shows the evolution of the density of infective individuals in the two patches when $R_0^1 = 0.8889$ and $R_0^2 = 1.8750$. We can see the infectious disease will be endemic in patch 2 and the infectious disease in patch 1 will disappear eventually. Figure 3 shows the evolution of the density of infective individuals in the two patches when $R_0^1 = 1.4815$ and $R_0^2 = 2.2917$. We can see the infectious disease will be endemic in the two patches. And we can see the infectious disease will be endemic in the two patches if $R_0^1 > 1$ from the subfigures (c) and (d) of Figure 4.

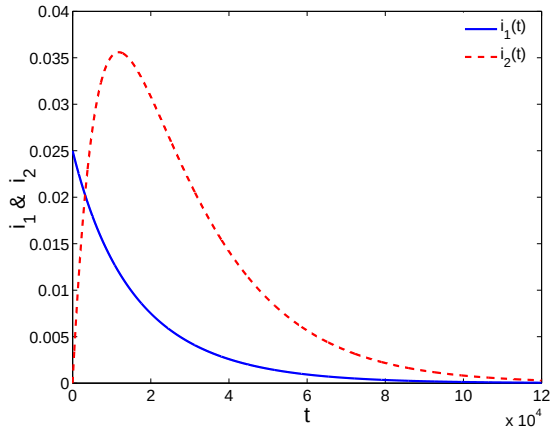

Figure 1. When $R_0^1 = 0.8889$ and $R_0^2 = 0.7500$, the evolution of the density of the infective individuals in the two patches.

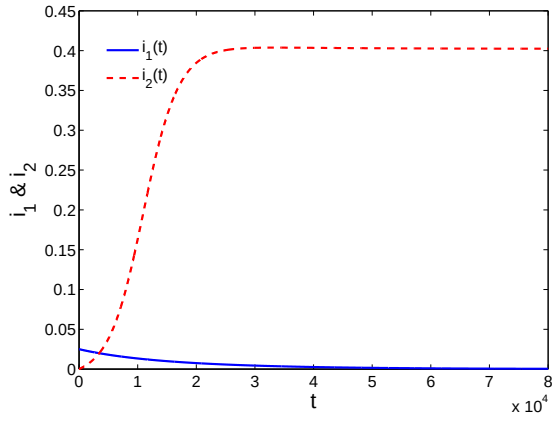

Figure 2. When $R_0^1 = 0.8889$ and $R_0^2 = 1.8750$, the evolution of the density of the infective individuals in the two patches.

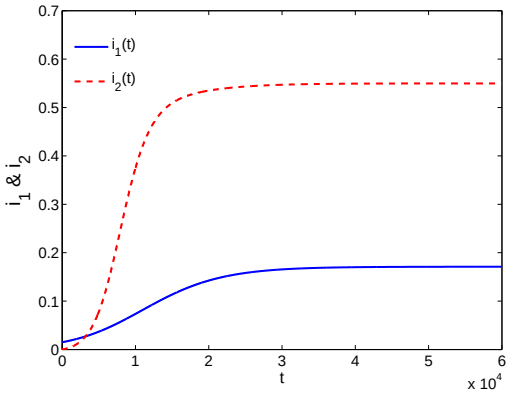

Figure 3. When $R_0^1 = 1.4815$ and $R_0^2 = 2.2917$, the evolution of the density of the infective individuals in the two patches.

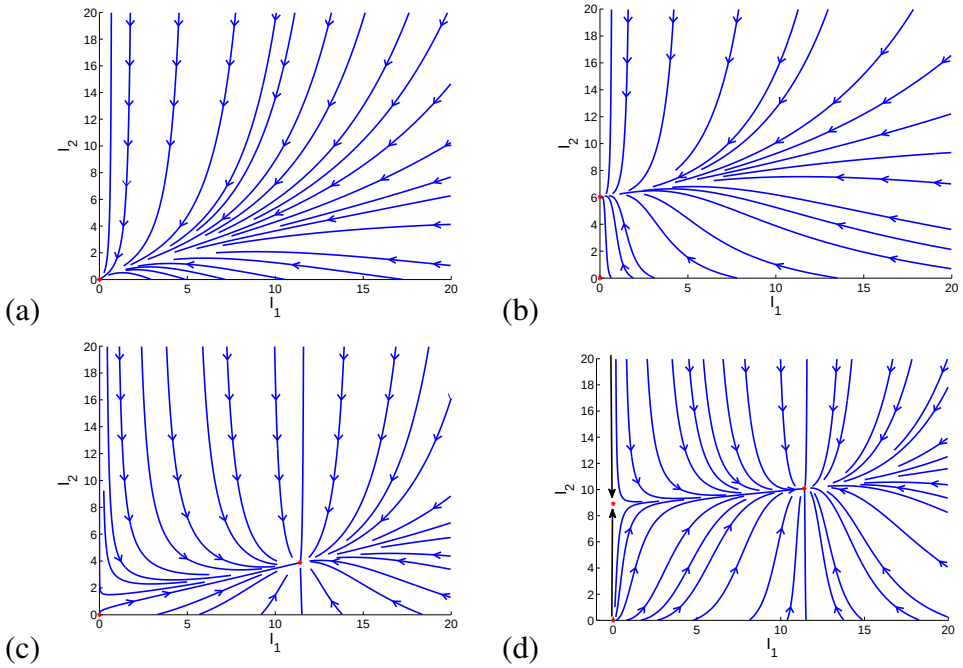

Figure 4. Phase portraits for (a) $R_0^1 < 1$ and $R_0^2 < 1$; (b) $R_0^1 < 1$ and $R_0^2 > 1$; (c) $R_0^1 > 1$ and $R_0^2 < 1$; (d) $R_0^1 > 1$ and $R_0^2 > 1$.

Second, we reveal the influence of the migration rate m on the transmission in Figure 5. With the increasing of m, the density of infective individuals in patch 1 i_1 is decreasing, however the density of infective individuals in patch 2 i_2 is increasing.

Third, we reveal the parameters α_1 and α_2 on the transmission scale and transmission speed. We can see that when α_1 is increasing, the density of infective individuals in patch 1 i_1 is decreasing from Figure 6 and When α_2 is increasing, the density of infective individuals in patch 2 i_1 is decreasing from Figure 7.

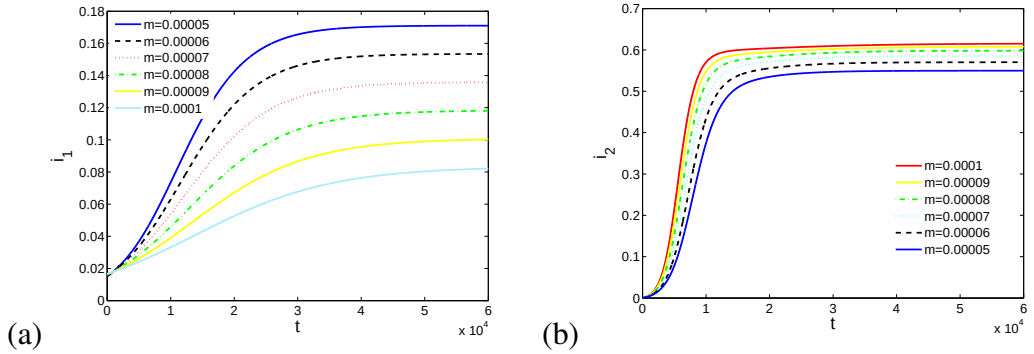

Figure 5. When m is increasing, (a) the density of infective individuals in patch 1 i_1 is decreasing; (b) the density of infective individuals in patch 2 i_2 is increasing.

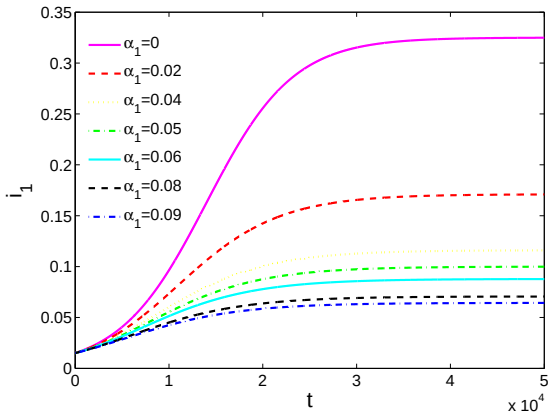

Figure 6. When α_1 is increasing, the density of infective individuals in patch 1 i_1 is decreasing.

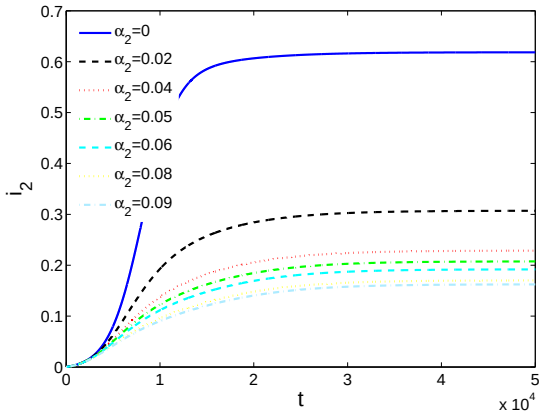

Figure 7. When α_2 is increasing, the density of infective individuals in patch 2 i_1 is decreasing.

5. Discussion and conclusions

Many scholars have studied infectious disease transmission with population migration [1–7], assuming that the migration matrix is irreducible, and found that the propagation dynamics of infectious diseases is determined by the basic reproduction number of the system. When the basic reproduction number is less than 1, the infectious disease eventually becomes extinct; when the basic reproduction number is larger than 1, the infectious disease is epidemic eventually. Since the migration matrix is irreducible, all patches are a connected whole. In all patches, infectious diseases are either extinct or epidemic. That is there is not the phenomenon that infectious diseases are extinct in some patches but epidemic in the others.

Because the studies about the spread of infectious diseases with reducible migration matrix are rare, in this paper, we proposed a two-patch SIS model with saturating contact rate and one-directing population dispersal, discussed the global asymptotic stability of the disease-free equilibrium, the boundary equilibrium and the endemic equilibrium respectively, and revealed the influence of saturating contact rate and migration rate on basic reproduction number and the transmission scale. We have the following main conclusions:

1) If $R_0^1 > 1$ then the system tends to a global endemic equilibrium in which infected individuals are present in both patches provided initially there were infected individuals in the source patch; If $R_0^1 < 1$ and $R_0^2 > 1$ then the system converges to an equilibrium with infected individuals only in the sink patch; If $R_0^1 < 1$ and $R_0^2 < 1$ then the system converges to the disease-free equilibrium.

2) When migration rate is increasing, the density of infective individuals in the source patch is decreasing; but the density of infective individuals in the sink patch is increasing;

3) With the increasing of the parameter α_i $(i = 1, 2)$ in saturating contact rate, the density of infective individuals in patch i $(i = 1, 2)$ is decreasing.

The similar conclusions can be obtained for the two patch SI model

$$\begin{cases} \dfrac{dS_1(t)}{dt} = A_1 - d_1 S_1 - \beta_1 S_1 \frac{I_1}{1+\alpha_1 I_1} - mS_1, \\ \dfrac{dS_2(t)}{dt} = A_2 - d_2 S_2 - \beta_2 S_2 \frac{I_2}{1+\alpha_2 I_2} + mS_1, \\ \dfrac{dI_1(t)}{dt} = \beta_1 S_1 \frac{I_1}{1+\alpha_1 I_1} - d_1 I_1 - mI_1, \\ \dfrac{dI_2(t)}{dt} = \beta_2 S_2 \frac{I_2}{1+\alpha_2 I_2} - d_2 I_2 + mI_1. \end{cases}$$

We can generalize the current model in many aspects to increase realism. For instance, the infection rate can be given by $\beta(I)SI$. We can give the properties on function $\beta(I)$ such that $\beta(I)$ is decreasing and tends to 0 when I tends to infinity. The mortality rates of the susceptible and infected individuals are the same in the current model. In fact, the disease-induced death rate can not be neglected sometimes. So the disease-induced death rate can be considered. It is also significant to consider heterogeneous number of contacts for each individual on complex network. There are many paper on this topic [22, 23]. One can investigate the multi-patch epidemic model with reducible migration matrix.

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Conflict of interest

All authors declare no conflicts of interest in this paper.

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