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Adaptive dispersal effect on the spread of a disease in a patchy environment

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1. Introduction

Because communicable diseases, such as influenza and sexual diseases, can be transmitted easily from one region to another, population dispersal plays a critical role in the spread of such diseases. Hethcote [1] is one of the pioneers who proposed epidemic models with population dispersal between two patches. Subsequently, numerous studies have contributed to investigations on the effect of population migration on the spread of disease, and to determining whether migration can increase disease persistence. When formulating such disease models with migration, the adoption of a constant or adaptive dispersal is considered.

Most studies on the effect of population immigration on the spread of disease have focused on random mobility; in other words, individuals were assumed to travel among patches at constant rates. We examined SIS models with a constant dispersal rate in [2,3,4]. The main results include (1) establishing a threshold below which the disease becomes extinction and above which the disease persists and tends toward a certain level; and (2) identifying the effect of the dispersal rate on this critical value. From the designed examples, they revealed that, depending on other parameters in the system, changing the dispersal rate in a certain patch may either benefit or damage the capacity for disease control. However, the random...
behavior of each individual can be affected by change of the environment or social consideration. Accordingly, the authors in [4] proposed a compelling but unsolved problem in studying an epidemic model with a variable dispersal rate. In epidemiology, [5] is one of the few studies that examined adaptive dispersal among patches by applying mathematical models. Managing wildlife through culling, vaccination, and habitat management is efficient for disease control [6]. Therefore, the authors in [5] developed a mathematical model to describe the effect of increased movement caused by culling individuals. However, from a mathematical perspective, this model involves additional nonlinearity, and leaves the global dynamics of the endemic case unsolved.

Disease information that includes the routes of transmission, the areas of outbreak, and the number of infected and killed populations are currently widely broadcast through strong media coverage. Recent studies [7,8] have taken media coverage within a habitat into consideration to study epidemic problems. The present paper proposes an endemic model with density-dependent dispersal among patches by considering two facts: First, information on disease spread and the characteristics of pathogens is revealed through international organizations, such as the World Health Organization, and quickly communicated through media coverage, such as through television news, newspapers, Internet news, and mobile instant messaging. Education on the latent periods of disease typically results in individuals taking precautions against crowding and adopting social distancing. Second, animals have a natural reaction against disease, which possibly leads to blind escaping from crowding. In addition, the carrying capacity for a species within an area does not change according to the population number, and offspring must establish their own habitat [9].

The remainder of this paper is organized as follows: Section 2 presents a multipatch epidemic model with density-dependent migrations. We discuss how the dispersal rates of individuals depend on the population number in the habitat and the destination, and propose a class of functional responses to formulate the adaptive dispersal rates. Section 3 presents a calculation of the basic reproduction number of the connected patches, and a discussion on how media intensity influences this threshold value. To present a dynamic investigation in one model consistently, we treated a particular case and obtained global dynamics according to different levels of disease spread. In addition to the theoretical results regarding dynamical characteristics, we present examples to discuss changes in the population number concerning the strategy of adaption in Section 4. This paper concludes by presenting a brief comparison of our results with those of other studies as well as a discussion on prediction concerns.

2. Model formulation

An endemic model is considered with multiple patches, which are connected by a population movement. The population of each patch is divided into two groups: for \( i \in P := \{1, 2, \ldots, p\} \). \( S_i(t) \) and \( I_i(t) \) respectively denote the number of susceptible and infectious individuals in \( i \)th patch at time \( t \). We examine a standard proportional incidence of disease transmission [8,10] in each patch, and assume diseases with no immune period; that is, the infectious individuals become susceptible as soon as they recover from the disease. Thus, each isolated patch is equipped with a classic SIS epidemic model.

Next, we introduce an adaptive dispersal in the endemic model with a patchy environment. In the absence of media effect, population disperse randomly; that is, individuals travel among patches with constant dispersal rates. When media coverage is taken into account, the density of population will affect the strategy of migration among patches. Naturally the adaptive dispersal rate is not only related to the alertness to the disease of individuals, but also closely related to the number of total population due to obscure responses in the initial infection. Therefore, we comprehend the population behaviors of escaping from crowds by two factors: the dispersal rate has positive (respectively, negative) functional response to the number of total population in the habitat (respectively, destination). Therefore, we consider an adaptive dispersal rate of susceptible (respectively, infectious) individuals from patch-\( j \) to patch-\( i \), evaluated by \( f_{ij}(N_i, N_j) \) (respectively, \( g_{ij}(N_i, N_j) \)).

\( N_i(t) = S_i(t) + I_i(t) \), which satisfies

\[ (H1) \quad f_{ij}, \quad g_{ij} \in C^1 \quad \text{for} \ i, j \in P, \quad \text{and} \quad \frac{\partial f_{ij}(x_1, x_2)}{\partial x_1} \leq 0, \quad \frac{\partial f_{ij}(x_1, x_2)}{\partial x_2} \geq 0, \quad \text{similarly for} \ g_{ij}, \]

\[ (H2) \quad f_{ij}(N_i, N_j) = - \sum_{j \in P, j \neq i} f_{ji}(N_j, N_i), \quad g_{ij}(N_i, N_j) = - \sum_{j \in P, j \neq i} g_{ji}(N_j, N_i), \quad \text{for} \ i \in P. \]

The assumption \( \frac{\partial f_{ij}(x_1, x_2)}{\partial x_1} \leq 0 \) (respectively, \( \frac{\partial f_{ij}(x_1, x_2)}{\partial x_2} \geq 0 \)) in (H1) indicates that the population of the destination (habitat) reduces (enhances) the dispersal rate to avoid infection. The assumption (H2) means that both the death rate and the birth rate of the individual are neglected during the dispersal process, because of either the fast traffic system or the dispersal between neighboring patches.

A considerable example is

\[ \begin{aligned}
(\mathcal{N}) & \quad \begin{cases}
 f_{ij}(N_i, N_j) = \left( a_{ij} + \frac{b_{ij} N_j}{1 + \alpha_{ij} N_j} \right) \frac{1}{1 + \alpha_{ij} N_i}, \\
 g_{ij}(N_i, N_j) = \left( \tilde{a}_{ij} + \frac{\tilde{b}_{ij} N_j}{1 + \tilde{\alpha}_{ij} N_j} \right) \frac{1}{1 + \tilde{\alpha}_{ij} N_i}.
\end{cases}
\end{aligned} \]
\[ a_{ij}, b_{ij}, c_{ij}, \tilde{a}_{ij}, \tilde{b}_{ij}, \tilde{c}_{ij} \geq 0; \quad a_{ij} + \frac{b_{ij}N_j}{1 + c_{ij}N_j}, \quad \tilde{a}_{ij} + \frac{\tilde{b}_{ij}N_j}{1 + \tilde{c}_{ij}N_j}, \quad \text{and} \quad \frac{1}{1 + c_{ij}N_j}, \quad \frac{1}{1 + \tilde{c}_{ij}N_j} \] indicate the factors of adaption for inner-elusion in Holling Type II and inter-elusion, respectively. \( b_{ij}, \tilde{b}_{ij}, c_{ij} \) and \( \tilde{c}_{ij} \) measure the intensity of media coverage or the level of panic. To explore a positive response in biological mechanisms, saturation or nonsaturation of the response is a trait merits consideration. It is therefore necessary to experiment with both cases. In addition, there may be examples without inter-elusion, i.e., \( c_{ij} = 0 \) for \( i, j \in P \), due to inaccessibility of outer-information in wildlife. Accordingly, we propose two further examples for different types of mental response to media alert:

\[
\begin{align*}
(N') \quad & \begin{cases} 
 f_{ij}(N_i, N_j) = a_{ij} + \frac{b_{ij}N_j}{1 + c_{ij}N_j}, \\
 g_{ij}(N_i, N_j) = \tilde{a}_{ij} + \frac{\tilde{b}_{ij}N_j}{1 + \tilde{c}_{ij}N_j}.
\end{cases}
\end{align*}
\]

\[
\begin{align*}
(L) \quad & \begin{cases} 
 f_{ij}(N_i, N_j) = a_{ij} + b_{ij}N_j, \\
 g_{ij}(N_i, N_j) = \tilde{a}_{ij} + \tilde{b}_{ij}N_j.
\end{cases}
\end{align*}
\]

The functions in type \((N')\) indicate a nonlinear and saturated response to population number while those in type \((L)\) represent linear response and unlimited adaption. A biological reason that the \( p \) patches cannot be separated into two groups such that there is no migration of individuals between different groups suggests that the \( p \times p \) matrices \((f_{ij}(N_i, N_j))\) and \((g_{ij}(N_i, N_j))\) are irreducible whenever \( N_i, N_j \geq 0 \) (see, for example, [11, Appendix A]).

**Remark 1.**

1. The special case of \((N)\) with \( b_{ij} = \tilde{b}_{ij} = c_{ij} = \tilde{c}_{ij} = 0 \) indicates the absence of media coverage and individuals traveling at constant rates. Epidemic models with this migration strategy have been studied in several literature, for example, see [3,4,12].

2. Author in [13] studied a discrete-generation model of a single species with density-dependent migration among a chain of habitat fragments, and proposed a model with migration rates depending increasingly on the number of local population. Assumption \((H1)\) for \( f_{ij} \) and \( g_{ij} \) agrees with this theory of adaptive dispersal rates.

3. If \( b_{ij}, \tilde{b}_{ij} > 0 \) and both are small enough with respect to \( a_{ij} \) and \( \tilde{a}_{ij} \), the dispersal rates \( f_{ij} \) and \( g_{ij} \) are closed to \( a_{ij} \) and \( \tilde{a}_{ij} \), respectively, and it represents weak media effect. As \( b_{ij}, \tilde{b}_{ij} \) are large, media coverage is comprehensive and the public will be more aware of crowds. Hence, an increase of total population in a habitat will result in higher adaption in dispersal rate.

4. When a media coverage arises in patch-\( j \), consideration of spatial heterogeneity allows for different \( b_{ij} \) and \( b_{ij} \) for \( i_1 \neq i_2 \) because they relate to different destinations. The common rule is that stronger media coverage or more severe levels of panic in patch-\( j \) assumes larger \( b_{ij} \) and \( b_{ij} \).

An SIS model with adaptive dispersal is thus described by the following system

\[
\begin{align*}
\frac{dS_i}{dt} &= A_i - d_i S_i - \beta_i \frac{S_i I_i}{N_i} + \gamma_i I_i + \sum_{j \in P} f_{ij}(N_i, N_j) S_j, \\
\frac{dI_i}{dt} &= \beta_i \frac{S_i I_i}{N_i} - (d_i + \nu_i + \gamma_i) I_i + \sum_{j \in P} g_{ij}(N_i, N_j) I_j,
\end{align*}
\]

with non-negative initial conditions

\[
S_i(0) > 0, \quad I_i(0) \geq 0, \quad \text{for} \ i \in P, \quad \sum_{i \in P} I_i(0) > 0.
\]

where \( A_i > 0 \) is the recruitment rate, \( d_i > 0 \) is the natural death rate, \( \gamma_i > 0 \) is the recovery rate, \( \nu_i \geq 0 \) is the disease-induced death rate and \( \beta_i > 0 \) indicates the transmission coefficient.

**3. Mathematical analysis**

We first introduce some notations that will be used throughout this paper. Let \( \mathbb{R}^P_+ = \{ x \in \mathbb{R}^P | x_i \geq 0 \ \text{for} \ i \in P \} \) be the positive orthant in \( \mathbb{R}^P \) and \( \text{Int} \mathbb{R}^P_+ = \{ x \in \mathbb{R}^P | x_i > 0 \ \text{for} \ i \in P \} \) be the interior of \( \mathbb{R}^P_+ \). For \( x, y \in \mathbb{R}^P_+ \), we write \( x \leq y \) whenever \( y - x \in \mathbb{R}^P_+ \), \( x < y \) whenever \( y - x \in \mathbb{R}^P_+ \) and \( y \neq x \), and \( x \ll y \) whenever \( y - x \in \text{Int} \mathbb{R}^P_+ \). Let \( s(M) \) denotes the spectral abscissa of the matrix \( M \), i.e., the maximum real part of all the eigenvalues of \( M \). Two \( p \times p \) matrices \( M = (M_{ij}) \) and \( M' = (M'_{ij}) \) satisfy \( M < M' \) if \( M_{ij} \leq M'_{ij} \) for all \( i, j \) and the strict inequality holds for at least one entry. \( M' \) denotes the transposed matrix of \( M \).
3.1. Basic results

The following theorem shows that model (2.1) is biologically and mathematically well posed.

**Theorem 1.** Consider system (2.1) with non-negative initial conditions (2.2). Then the system has a unique solution defined for all time $t \geq 0$, and the orthant $\mathbb{R}_{+}^{2p}$ is positively invariant under the flow of (2.1), with each $S_j$ remaining positive. Moreover, there is an $N^* > 0$ such that $G := \{(S, I) \in \mathbb{R}_{+}^{2p} | \sum_{i\in P}(S_i + I_i) \leq N^*\}$ attracts all solution orbits of (2.1) in $\mathbb{R}_{+}^{2p}$, and $G$ is positively invariant for (2.1).

**Proof.** The vector field defined by (2.1) and (2.2) is Lipschitzian in each compact set in $\mathbb{R}_{+}^{2p}$, so the initial value problem has a unique solution for all $t \geq 0$ as long as the boundedness of solutions holds [14].

Suppose there exist proper subsets $P_1$, $P_2$ of $P$ and $t_0 \geq 0$ such that $I_i(t_0) = 0$ for $i \in P_1$ and $I_i(t_0) \neq 0$ for $i \in P_2$. From (2.1) and irreducibility of $(g_{ij})$, we have

$$\frac{dI_i(t_0)}{dt} = \sum_{j \in P} g_{ij}(N_i, N_j)I_j > 0,$$

for some $i \in P_1$, which implies that $I_i$ is an increasing function of $t$ at $t_0$. Note that the state without any infectious individual will not change in any one $I_i$. Thus, $I_i$ remains nonnegative for each $i \in P$. Suppose there is one of the first $S_i(t_1) = 0$ for $t_1 \geq 0$. From (2.1), non-negativity of each $I_i$ and irreducibility of the matrix $(f_{ij})$,

$$\frac{dS_i(t_1)}{dt} > A_{i_0} + \sum_{j \in P, j \neq i} f_{ij}(N_i, N_j)S_j > 0.$$

From the initial condition (2.2), each $S_i(t)$ remains positive for $t > 0$. Hence, $\mathbb{R}_{+}^{2p}$ is positively invariant under the flow of (2.1) and (2.2).

Denoting the total population of the system by $N(t) = \sum_{i \in P} N_i(t)$ and $d_* = \min_{i \in P} d_i$, the system (2.1) gives

$$\frac{dN(t)}{dt} = \sum_{i \in P} (A_i - d_i N_i - v_i I_i) \leq \sum_{i \in P} (A_i - d_i N_i) \leq \sum_{i \in P} A_i - d_i N.$$

By a comparison theorem, the total population $N(t)$ is bounded above by $\max\{\sum_{i \in P} A_i/d_*, N(0)\}$. Then the positive invariance and global attractivity of $G$ are proved by the method in [3].

In order to verify that the model (2.1) is equipped with disease-free equilibrium (DFE), we consider

$$\frac{dS_i}{dt} = A_i - d_i S_i + \sum_{j \in P} f_{ij}(S_i, S_j)S_j, \quad i \in P,$$

with initial value $S_i(0) > 0$ for $i \in P$. In [3], the disease-free subsystem of an epidemic model was studied by using the strongly sub-linearity property. In fact, the disease-free subsystem (3.1) obey this property due to the adaption on dispersal rates. Hence, we study (3.1) by using the theory of monotone dynamical systems [15]. Indeed, the following result reveals that model (2.1) always admits a unique DFE, labeled by $E^0 = (S_1^0, S_2^0, \ldots, S_p^0, 0, 0, \ldots, 0)$.

**Lemma 1.** The Eq. (3.1) has a positive equilibrium that is globally asymptotically stable in $\text{Int}\mathbb{R}_{+}^{p}$.

**Proof.** Define $F = (F_1, F_2, \ldots, F_p)$, where $F_i(S) = A_i - d_i S_i + \sum_{j \in P} f_{ij}(S_i, S_j)S_j$ for $S = (S_1, S_2, \ldots, S_p)$. From (H1), there exists $\epsilon = (\epsilon_1, \epsilon_2, \ldots, \epsilon_p)$ such that $F(\epsilon) \gg 0$. Note that system (3.1) is cooperative, and each solution orbit has compact closure in $\{(x_1, x_2, \ldots, x_p) \in \mathbb{R}^p : 0 \leq x_i \leq \sum_{i \in P} A_i/d_*$ for $i \in P\}$. By Lemma 1 in [16], the system (3.1) admits a positive equilibrium, denoted by $S^0 = (S_1^0, S_2^0, \ldots, S_p^0)$.

Next, we claim that the system (3.1) has unique positive equilibrium. Assume, by contrary, that $S^0 = (S_1^0, S_2^0, \ldots, S_p^0)$ is also a positive equilibrium of (3.1). Since the system (3.1) is cooperative and irreducible, the generated semiflow $\Phi_t$ is strongly monotone [15, Theorem 4.11]. Without loss of generality, we say $S^0 \ll \bar{S}$. From (3.1), we have

$$A_i - d_i S_i^0 + \sum_{j \in P} f_{ij}(S_i^0, S_j^0)S_j^0 = 0,$$

$$A_i - d_i S_i^0 + \sum_{j \in P} f_{ij}(S_i^0, S_j^0)S_j^0 = 0,$$

for $i \in P$, and then the assumption (H2) implies

$$\sum_{i \in P} (A_i - d_i S_i^0) = 0 \quad \text{and} \quad \sum_{i \in P} (A_i - d_i S_i^0) = 0.$$

This contradicts to the assumption $S^0 \ll \bar{S}$. Thus, $S^0$ is the unique positive equilibrium of (3.1).

To see that $S^0$ attracts all non-negative orbits of (3.1), we first note that there exists $\epsilon_0 > 0$ small enough such that $F(\bar{\epsilon}) \gg 0$, where $\bar{\epsilon} = (\epsilon, \epsilon, \ldots, \epsilon)$ and $0 < \epsilon \leq \epsilon_0$. For given $\bar{S} \in \text{Int}\mathbb{R}_{+}^{p}$, there exists $r_1, r_2 > 0$ such that $r_1 \epsilon_0 \ll \bar{S} \ll r_2 \bar{S}$. 


We choose positive $r_2$ large enough such that $F(r_2 \hat{S}) \leq 0$. From [15, Proposition 3.2.1], $\Phi_t(r_1,0)$ and $\Phi_t(r_2,0)$ both converge to $S^0$. By the strong monotonicity of the semiflow $\Phi_t$, we have $\Phi_t(r_1,0) \ll \Phi_t(\hat{S}) \ll \Phi_t(r_2,0)$ for $t > 0$, and conclude that $\Phi_t(\hat{S}) \to S^0$ as $t \to \infty$. This completes the proof. 

Remark 2. Previous result is also true for the system (2.1) with other birth rate function, for example, $p_i e^{-q_i N}$, $p_i, q_i > 0$ and $p_i > d_i$, in [3,4]. But, with the birth rate function $\frac{p_i}{\sqrt{r_i^2 + q_i}}$, $p_i > d_i$ and $m > 0$, addressed in [3,4], it fails to show the unique DFE for the system (2.1) by previous manner. In fact, an SIS model incorporated with such a birth rate as well as constant dispersal rates may involve multiple stability [4].

3.2. Basic reproduction number

The basic reproduction number defined in [17,18] is an essential concept in studying infectious diseases via mathematical description. Following their recipe, we linearize the system (2.1) at the disease-free equilibrium $E^0$, and obtain the next generation matrix $\mathcal{F} \mathcal{V}^{-1}$, where

$$\mathcal{F} = (\delta_{ij} \beta_j)_{p \times p},$$

$$\mathcal{V} = (\delta_{ij} (d_i + v_i + \gamma_i) - g_{ij}(S^0_i, S^0_j))_{p \times p},$$

and $\delta_{ij}$ denotes the Kronecker delta (i.e. 1 when $i = j$ and 0 otherwise). By the next generation method [18], the basic reproduction number, denoted by $R_0$, of the system (2.1) is given as the spectral radius of $\mathcal{F} \mathcal{V}^{-1}$; that is,

$$R_0 = \rho(\mathcal{F} \mathcal{V}^{-1}).$$

By van den Driessche and Watmough [18, Theorem 2], the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.

In [3], it was shown that the basic reproduction number, which depends on the natural dispersal rate of infectious individuals, is a threshold value for the uniform persistence and extinction of the disease in a model with constant dispersal rates as well as general birth rate functions. Note that, in the model (2.1), $R_0$ depends not only on the natural dispersal rates $d_{ij}$ but also on the intensity of inner- and inter-elusion, $\tilde{d}_{ij}$ and $\tilde{c}_{ij}$. Therefore, the media coverage certainly affects the control of disease spreading.

To better understand how the basic reproduction number relates to the media coverage, we address an example of (2.1) with a two-patch environment and the simple functional response ($\mathcal{N}$). Fix all parameters except those related to adaptive dispersal, by setting $A_1 = 20$, $d_1 = 0.000036$, $\beta_1 = 0.1$, $v_1 = 0.01$, $\gamma_1 = 0.04$, $a_1 = 0.1$, $A_2 = 15$, $d_2 = 0.000036$, $\beta_2 = 0.03$, $v_2 = 0.001$, $\gamma_2 = 0.04$, $a_2 = 0.1$, and change $b_{21}$, $b_{12}$ to calculate $R_0$. Note that, the basic reproduction number of each isolated patch is $R_0^{(1)} = 1.5$ and $R_0^{(2)} = 0.5882$, respectively. Fig. 1 illustrates the basic reproduction number, $R_0$, of the system (2.1) related to different media intensities $b_{21}$ and $b_{12}$. Obviously, the disease spread cannot be controlled under this level of natural dispersal, and higher media intensity from endemic patch-1 helps annihilating disease in the connected system, while media coverage from disease free patch-2 damages the disease control.

3.3. Threshold dynamics

There are several literature demonstrating the global stability of the DFE in different epidemic models when the basic reproduction number is under a threshold, for example, see [8,19,20]. However, neither the standard comparison principle
used by Sun et al. [8] nor the reduction-like property quoted in [19,20] can be applied to model (2.1) due to its adaptive dispersal. Hereafter, we assume that there is no disease-induced death (i.e., $\nu_i = 0$ for $i \in P$), and susceptible and infectious individuals travel at the identical rates (i.e., $f_{ij} = g_{ij}$ for $i, j \in P$). The system (2.1) becomes

$$\begin{align*}
\frac{dS_i}{dt} &= A_i - dS_i - \beta_i \frac{S_i}{N_i} I_i + \sum_{j \in P} f_{ij}(N_i, N_j) S_j, \\
\frac{dI_i}{dt} &= \beta_i \frac{S_i}{N_i} - (d_i + \gamma_i) I_i + \sum_{j \in P} f_{ij}(N_i, N_j) I_j, \\
\frac{dN_i}{dt} &= A_i - dN_i + \sum_{j \in P} f_{ij}(N_i, N_j) N_j, \\
\frac{dI_i}{dt} &= \beta_i \frac{(N_i - I_i) I_i}{N_i} - (d_i + \gamma_i) I_i + \sum_{j \in P} f_{ij}(N_i, N_j) I_j.
\end{align*}$$

(3.4)

$i \in P$. To study the global dynamics of (3.4), we add the two equations in system (3.4), and obtain the equivalent one

$$\frac{dN_i}{dt} = A_i - dN_i + \sum_{j \in P} f_{ij}(N_i, N_j) N_j,$$

(3.5)

$$\frac{dI_i}{dt} = \beta_i \frac{(N_i - I_i) I_i}{N_i} - (d_i + \gamma_i) I_i + \sum_{j \in P} f_{ij}(N_i, N_j) I_j.$$

(3.6)

$i \in P$. The local stability of the disease-free equilibrium $E^0$ in system (3.4) is fully determined by $R_0$. With a help of Lemma 1, we follow the method in [3] to show the global stability of the DFE when $R_0 < 1$, as in the following theorem.

**Theorem 2.** The disease-free equilibrium $E^0$ of (3.4) is globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$.

**Proof.** From Lemma 1, subsystem (3.5) has a unique equilibrium, denoted by $N^0 = (N^0_1, N^0_2, \ldots, N^0_P)$, which is globally asymptotically stable in $\mathbb{R}^P_+$. Note that $N^0 = S^0$. Since $N_i(t) \rightarrow N^0_i$ as $t \rightarrow \infty$ for $i \in P$, given small $\xi > 0$ there is a large enough $t_2$ such that $N^0_i - \xi < N_i(t) < N^0_i + \xi$ for $t > t_2$ and $i \in P$.

Set

$$V_i = (\delta_i (d_i + \nu_i + \gamma_i) + f_{ij}(S^0_i - \xi, S^0_j + \xi))_{i \in P}.$$

Since (H1), (H2) hold and the spectral radius of a matrix is continuous in each elementary, the spectral radius of $\mathcal{F}V_i^{-1}$ is continuous in $\xi$. Thus, we can choose a positive constant $\xi$ small enough such that $\rho(\mathcal{F}V_i^{-1}) < 1$. For $t \geq t_2$, from (3.6) and (H1), it leads to

$$\frac{dI_i}{dt} \leq \beta_i I_i - (d_i + \gamma_i) I_i + \sum_{j \in P} f_{ij}(S^0_i - \xi, S^0_j + \xi) I_j.$$

This suggests the following comparison linear system for (3.6)

$$\frac{dW_i}{dt} = \beta_i W_i - (d_i + \gamma_i) W_i + \sum_{j \in P} f_{ij}(S^0_i - \xi, S^0_j + \xi) W_j.$$

(3.7)

Since $V$ is a non-singular M-matrix [18], we further choose $\xi > 0$ small enough such that $V_i$ remains as a non-singular M-matrix. Also note that $\mathcal{F}$ is non-negative. Hence, it holds that $s(V_i - \mathcal{F}) < 0$ if and only if $\rho(\mathcal{F}V_i^{-1}) < 1$. All eigenvalues of $V_i - \mathcal{F}$ are negative with real parts, see the proof of [18, Theorem 2] or [3, Lemma 2.1]. Thus, all non-negative solutions of (3.7) satisfies that $\lim_{t \rightarrow \infty} W_i(t) = 0$ for $i \in P$. By a standard comparison principle [11, Theorem B.1] and the non-negativity of each $I_i$, we conclude that all non-negative solutions of (3.4) satisfy $\lim_{t \rightarrow \infty} I_i(t) = 0$ for $i \in P$. This implies that (3.1) performs the limiting system of the $S$ equation in (3.4), which has the dynamics of global convergence to $S^0$. By the theory of asymptotically autonomous systems [21, Theorem 2.3] and [22], we conclude that all the solutions to (3.4) with (2.2) satisfy that $\lim_{t \rightarrow \infty} S_i(t) = S^0_i$ for $i \in P$. Therefore, the disease-free equilibrium $E^0$ of (3.4) is globally asymptotically stable whenever $R_0 < 1$, and hence completes the proof. \( \square \)

The result in Theorem 2 means that $R_0$ measures a sufficient condition for disease extinction. To determine whether it is pandemic in all considered regions, it is necessary to study a criterion for disease persistence. In the case of minor infection, the $S$-equation in (3.4) can be regarded as a perturbed system of (3.1). Substituting the convergent result of such a perturbed system into the $I$-equation in (3.4) facilitates investigating whether the disease persists or not. To this end, we further extend the result in Lemma 1 to the perturbed system

$$\begin{align*}
\frac{dS_i}{dt} &= \bar{A}_i - dS_i + \sum_{j \in P} f_{ij}(S_i, S_j) S_j + \theta \sum_{j \in P, j \neq i} S_j, \\
\frac{dI_i}{dt} &= \beta_i \frac{(N_i - I_i) I_i}{N_i} - (d_i + \gamma_i) I_i + \sum_{j \in P} f_{ij}(N_i, N_j) I_j.
\end{align*}$$

(3.8)

where $\bar{A}_i > 0, \bar{d}_i > 0$ and $\theta$ are all constants, and $|\theta|$ is sufficiently small. Cooperativity is the main property to be used in proving global dynamics of (3.1). However, (3.8) may not be cooperative due to the perturbed term $\theta \sum_{j \in P, j \neq i} S_j$, and then a perturbation theory [23] is necessary to demonstrating a globally stable equilibrium. We denote the perturbed parameters by
\[ \lambda = (\bar{A}, \bar{d}, \theta), \text{ where } \bar{A} = (\bar{A}_1, \bar{A}_2, \ldots, \bar{A}_p) \text{ and } \bar{d} = (\bar{d}_1, \bar{d}_2, \ldots, \bar{d}_p). \] From Lemma 1, the Eq. (3.8) has a globally asymptotically stable equilibrium \( S^0(\lambda_0) \), where \( \lambda_0 = (\bar{A}, \bar{d}, 0). \)

**Lemma 2.** Let \( c := \min_{i \in P} \bar{A}_i / \sum_{i \in P, j \in U} \bar{S}_j \). The perturbed equation (3.8) has a positive equilibrium \( S^0(\lambda) \) that is globally asymptotically stable on \( U \) (respectively, \( \mathbb{R}^+_0 \)) if \(-c < \theta < 0\) (respectively, \( \theta > 0 \)), where \( U := \{S \in \mathbb{R}^0_i \mid S_j \leq \bar{S}_j \text{ for } i \in P\}. \) Moreover, the equilibrium \( S^0(\lambda) \) is continuous in \( \lambda. \)

**Proof.** We shall prove the first case, and the second one can be shown in the same manner and a simpler calculation. Set \( \Lambda = \{(\bar{A}, \bar{d}, \theta) : |\bar{A}, \bar{d} | \geq 0, -c < \theta < 0\}. \) Again, from Lemma 1, the Eq. (3.8) has a globally asymptotically stable equilibrium \( S^0(\lambda_0) \), where \( \lambda_0 = (\bar{A}, \bar{d}, 0) \) and \( \bar{A} = (A_1, A_2, \ldots, A_p). \) Let \( d = (d_1, d_2, \ldots, d_p). \) Next, we show that \( U \) is positively invariant under the solution flow of (3.8) whenever \(-c < \theta < 0\). Suppose there is one of the first \( S_i(t_3) = 0 \) for \( t_3 \geq 0. \) From (3.8) and non-negativity of each \( S_i, f_{ij} \), we obtain

\[
\frac{dS_i(t_3)}{dt} \geq \bar{A}_i + \theta \sum_{i \in P, j \notin i} \bar{S}_j > 0,
\]

which is a contradiction. Hence, each solution of (3.8) starting from the region \( U \) will never leave it from the boundary with at least one zero component. In addition, such a solution satisfies

\[
\frac{dS_i}{dt} \leq \bar{A}_i - \bar{d}_i \bar{S}_i + \sum_{j \in P} f_{ij}(\bar{S}_i, \bar{S}_j)S_j,
\]

since \( \theta < 0. \) As mentioned previously, the upper system

\[
\frac{dS_i}{dt} = \bar{A}_i - \bar{d}_i \bar{S}_i + \sum_{j \in P} f_{ij}(\bar{S}_i, \bar{S}_j)S_j,
\]

has a globally asymptotically stable equilibrium \( S^0(\lambda_0) \). By the comparison theory in [15, Theorem 5.11], \( S_i(t) \leq \bar{S}_i(t) \) for all \( t \geq 0. \) Since \( S^0(\lambda_0) \) is an equilibrium, each solution of (3.8) starting from the region \( U \) will stay therein forever. Therefore, all solutions to the family of perturbed systems (3.8) are eventually uniformly bounded. By Theorem 2.2 and Corollary 2.3 in [23], we prove the existence of a unique equilibrium that varies continuously in the perturbed parameters, and its global stability under the solution flow of (3.8). \( \square \)

With a help of Lemma 2 and the persistence theory in [24–26], we establish the uniform persistence of disease and the existence of endemic equilibrium when \( R_0 > 1. \) Define the following sets

\[
X = \{(S, I) \mid S_i \geq 0, I_i \geq 0, \text{ for all } i \in P\},
\]

\[
X_0 = \{(S, I) \in X \mid I_i > 0, \text{ for all } i \in P\},
\]

\[
X_1 = \{(S, I) \in X \mid I_i > 0, \text{ for some } i \in P\},
\]

\[
\partial X_0 = X \setminus X_0 = \{(S, I) \in X \mid I_i = 0, \text{ for some } i \in P\}.
\]

**Theorem 3.** If \( R_0 > 1, \) then there exists a positive constant \( \epsilon \) such that every solution of (3.4) with \((S(0), I(0)) \in X_1\) satisfies

\[
\liminf_{t \to \infty} I_i(t) > \epsilon, \text{ for } i \in P.
\]

Moreover, the system (3.4) admits at least one endemic equilibrium.

**Proof.** It suffices to show that \( \partial X_0 \) repels uniformly the solutions to the system (3.4) in \( X_0. \) Note that both \( X \) and \( X_0 \) are positively invariant with respect to the solution flow of (3.4), and \( \partial X_0 \) is relatively closed in \( X. \) From Theorem 1, system (3.4) is point dissipative. Define

\[
M_3 = \{(S(0), I(0)) \in \partial X_0 \mid (S(t), I(t)) \text{ satisfies } () \text{ and } (S(t), I(t)) \in \partial X_0, \text{ for all } t \geq 0\}.
\]

We claim that \( M_3 = \{(S(0), I(0)) \mid S \geq 0\} \). Obviously, \( \{(S, 0) \mid S \geq 0\} \subset M_3. \) Choose an initial value \( x_0 = (S(0), I(0)) \in M_3 \setminus \{(S, 0) \mid S \geq 0\}, \) i.e., \( \Sigma_{i \in P} f_{ij}(N_i, N_j) > 0. \) Since the matrix \( f_{ij}(N_i, N_j) \) is irreducible and the \( I \) equation of (3.4) is a non-autonomous cooperative system, each solution satisfies that \( I_i(t) > 0 \) for \( i \in P \) and \( t > 0. \) Thus, \( x \notin M_3, \) and then \( M_3 \subset \{(S, 0) \mid S \geq 0\}. \) Therefore, \( M_3 = \{(S(0), I(0)) \mid S \geq 0\}. \)

Note that the disease-free equilibrium \( E^0 \) is the unique equilibrium in \( M_3. \) Denote the stable manifold of \( E^0 \) by \( W^s(E^0). \) Next, we show that \( W^s(E^0) \cap X_0 = \emptyset \) when \( R_0 > 1. \) Since \( R_0 > 1, \) we can choose positive constants \( \xi, \eta \) small enough such that \( \rho(F_{\xi, \eta}V^{-1}_{\xi, \eta}) > 1. \) where

\[
F_{\xi, \eta} = (\delta_{ij}b_i(1 - \xi))_{p \times p},
\]

\[
V_{\xi, \eta} = (\delta_{ij}(d_i + v_i + \gamma_i) - f_{ij}(\bar{S}_i^0 + \eta + \xi, \bar{S}_j^0 - \eta))_{p \times p}.
\]

We claim that, for any solution of (3.4),

\[
\limsup_{t \to \infty} \max_{i \in P} \{I_i(t)\} > \xi.
\] (3.9)
If not, there exists a \( t_4 > 0 \) such that \( l_i(t) \leq \xi \) and \( \frac{S_i(t)}{N_i(t)} \geq 1 - \xi \), for all \( i \in P \) and \( t \geq t_4 \). Then for \( t \geq t_4 \), we have

\[
\frac{dS_i}{dt} \geq A_i - d_iS_i - \xi \beta_i + \sum_{j \in P, j \neq i} f_{ij}(S_i + \xi, S_j)S_j - \sum_{j \in P, j \neq i} f_{ji}(S_j, S_i + \xi)S_i \\
= A_i - d_iS_i - \xi \beta_i + \sum_{j \in P, j \neq i} \left[ f_{ij}(S_j, S_i)S_j + \frac{\partial f_{ij}(S_j^0, S_i^0)}{\partial x_1} \xi S_j \right] \\
- \sum_{j \in P, j \neq i} \left[ f_{ji}(S_i, S_j)S_j - \frac{\partial f_{ji}(S_i^0, S_j^0)}{\partial x_2} \xi S_j \right] \\
\geq A_i - d_iS_i - \xi \beta_i + \sum_{j \in P} f_{ij}(S_i, S_j)S_j - \kappa \xi \left[ (p - 1)S_i + \sum_{j \in P, j \neq i} S_j \right],
\]

where \( f_{ij} = f_{ij}(x_1, x_2), S_i^0, S_j^0 \in (S_i, S_j + \xi) \) and

\[
\kappa := \max \left\{ \frac{\partial f_{ij}}{\partial x_1}(x_1, x_2), \frac{\partial f_{ij}}{\partial x_2}(x_1, x_2) \mid (x_1, x_2) \in [0, N^+] \right\}
\]

for \( i, j \in P, i \neq j \).

Similarly, there exists a \( t_3 > 0 \) such that for \( t \geq t_3 \)

\[
\frac{dS_i}{dt} \leq A_i - d_iS_i + \xi \gamma_i + \sum_{j \in P} f_{ij}(\bar{S}_i, \bar{S}_j)S_j + \kappa \xi \left[ (p - 1)\bar{S}_i + \sum_{j \in P, j \neq i} \bar{S}_j \right].
\]

Consider the upper and lower systems respectively

\[
\frac{d\bar{S}_i}{dt} = A_i + \xi \gamma_i - d_i\bar{S}_i + \sum_{j \in P} f_{ij}(\bar{S}_i, \bar{S}_j)\bar{S}_j + \kappa \xi \left[ (p - 1)\bar{S}_i + \sum_{j \in P, j \neq i} \bar{S}_j \right], \quad i \in P, \tag{3.10}
\]

\[
\frac{d\bar{S}_i}{dt} = A_i - \xi \beta_i - d_i\bar{S}_i + \sum_{j \in P} f_{ij}(\bar{S}_i, \bar{S}_j)\bar{S}_j - \kappa \xi \left[ (p - 1)\bar{S}_i + \sum_{j \in P, j \neq i} \bar{S}_j \right], \quad i \in P. \tag{3.11}
\]

From Lemma 2, we can choose small \( \xi \) such that (3.10) (respectively, (3.11)) admits a globally asymptotically stable positive equilibrium \( \bar{S}_i^0(\xi) \) (respectively, \( \bar{S}_i^0(\xi) \)) in \( \mathbb{R}^p \) (respectively, \( \mathbb{U} \)). By [15, Theorem 5.11] and the comparison of (3.10), (3.11) with the original \( \bar{S} \) equation, it holds that \( \bar{S}_i^0(\xi) \leq \bar{S}_i^0(\xi) \). Since \( \bar{S}_i^0(\xi) \) and \( \bar{S}_i^0(\xi) \) are continuous in \( \xi \), for given \( \eta > 0 \), we can choose small \( \xi \) such that \( S_i^0 - \eta < \bar{S}_i^0(\xi) \leq S_i^0(\xi) < S_i^0 + \eta \). Again, by the comparison theorem [15, Theorem 5.11], there is a \( \xi \) such that \( S_i^0 - \eta < S_i(t) < S_i^0 + \eta \) for \( t \geq \xi \). Hence, for \( t \geq \xi \), \( \frac{dl_i}{dt} \geq \beta_i(1 - \xi)l_i - (d_i + \gamma_i)l_i + \sum_{j \in P} f_{ij}(N_i^0 + \xi, S_j^0 - \eta)l_j \), \( i \in P \).

Since \( \bar{F} \bar{x} \) is non-negative, \( \forall \epsilon, \eta \) is a non-singular \( M \)-matrix for small enough \( \xi \) and \( \eta \) and \( \rho(\bar{F} \bar{V}^{-1}) \) > 1, the matrix \( M := \bar{F} \bar{V}^{-1} - \bar{V}^{-1} \eta \) has a positive eigenvalue \( s(M) \) associated with a positive eigenvector [18], it follows that \( \lim_{t \to \infty} \frac{l_i(t)}{t} = \infty \) for \( i \in P \), which is a contradiction. Hence, the inequality (3.9) holds.

Consider system (3.4), \( E^0 \) is globally asymptotically stable in \( M_0 \), and then \( (E^0) \) is an isolated invariant set and acyclic in \( M_0 \). Therefore, by Thieme [24, Theorem 4.6], it follows that system (3.4) is uniformly persistent with respect to \( (X_0, \partial X_0) \). Moreover, from [25, Theorem 2.4], system (3.4) has an endemic equilibrium \( (\xi, \bar{\gamma}) \in \mathbb{X} \).

**Remark 3.** Obviously, by using the same manner, we can also show uniform persistence in the general model (2.1) without the restriction in Theorem 3.

Global dynamics of the endemic models with constant dispersal rates has been shown in [4,8,19] when the disease is pandemic. With a help of Lemma 1, we shall investigate this issue in system (3.4), which is equipped with adaptive dispersal, by combining convergence property of strongly sub-linear systems with the theory of asymptotically autonomous systems [21,27].

**Theorem 4.** If \( R_0 > 1 \), then (3.4) admits a unique endemic equilibrium which is globally asymptotically stable related to \( X_1 \).

**Proof.** As in the proof of Theorem 2, we first consider \( N \) equation (3.5) of the equivalent system which has a globally asymptotically stable equilibrium \( N_i^0 \) in the positive orthant. Since \( N_i(t) \to N_i^0 \) as \( t \to \infty \) for \( i \in P \), the system (3.5) and (3.6) has the limiting subsystem

\[
\frac{dl_i}{dt} = \beta_i \left( \frac{N_i^0 - l_i}{N_i^0} \right) - (d_i + \gamma_i)l_i + \sum_{j \in P} f_{ij}(N_i^0, N_j^0)l_j, \quad i \in P. \tag{3.12}
\]
for \( i \in P \). Denote the right hand side of system (3.12) by the function \( \Gamma : \mathbb{R}_+^p \to \mathbb{R}^p \), and let \( \Psi_i \) be the corresponding solution flow. For given \( \alpha \in (0, 1) \) and \( I \in \text{Int} \mathbb{R}_+^p \), it holds that \( \Gamma(\alpha I) > \alpha \Gamma(I) \); that is, \( \Gamma \) is strongly sub-linear on \( \mathbb{R}_+^p \) [27]. In addition, since \( R_0 > 1 \), the Jacobian matrix of (3.12), \( Df(0) = F - \Psi \), satisfies \( s(Df(0)) > 0 \). Therefore, there is a globally asymptotically stable positive equilibrium \( I^* \) with respect to \( \mathbb{R}_+^p \setminus \{0\} \) [27, Corollary 3.2].

Let \( \Psi_i(N(0), I(0)) \) denote the solution flow generated by (3.5) and (3.6). By Theorem 1, the orbit \( \{\Psi_i(N(0), I(0))t \geq 0\} \) is precompact, and the forward limit set of \( N(I) \) exists, denoted by \( \omega_{\Psi_i}(N, I) \). Let \( \omega_{\Psi_i}(N, I) \) be the projection of \( \omega_{\Psi_i}(N, I) \) onto \( \{N(I) \in \mathbb{R}_+^p | N = N^0\} \). From Theorem 3, we obtain \( \omega_{\Psi_i}(N, I) \subseteq \{(N^0, I(t)) | t \geq 0\} \setminus \{N^0, 0\} \). Since \( I^* \) is a globally asymptotically stable equilibrium of (3.12) in \( \mathbb{R}_+^p \setminus \{0\} \), it follows that \( \omega_{\Psi_i}(N, I) \) contains \( I^* \). By the theory of asymptotically autonomous systems [21, Theorem 2.3], we conclude that the equilibrium \( E^* = (N^0, I^*) \) is a globally attractor in \( X_1 \).

Next, we show that the endemic equilibrium \( E^* \) is locally asymptotically stable. It is equivalent to examining the stability of the Jacobian matrix of the system (3.4) at \( E^* \), i.e.,

\[
J(E^*) = \begin{pmatrix}
J_{11} & 0 \\
J_{21} & J_{22}
\end{pmatrix},
\]

where \( J_{11}, J_{21}, J_{22} \) are \( p \times p \) matrices defined as

\[
J_{11} = \begin{pmatrix}
(d_i - \sum_{k \in P} k \bar{f}_{ki}(N_i^*, N_k^*) N_i^* + \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} N_k^* - \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} f_k(N_k^*, N_i^*) & - \delta_{ij} \\
-\delta_{ij} & 0
\end{pmatrix},
\]

\[
J_{21} = \begin{pmatrix}
\beta_i \left( \frac{N_i^*}{N_i^* - 1} - (d_i + \gamma_i) - \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} N_k^* - \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} f_k(N_k^*, N_i^*) (1 - \delta_{ij}) & 0 \\
0 & 0
\end{pmatrix},
\]

\[
J_{22} = \begin{pmatrix}
\beta_i \left( \frac{N_i^*}{N_i^* - 1} - (d_i + \gamma_i) - \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} N_k^* + f_k(N_k^*, N_i^*) (1 - \delta_{ij}) & 0 \\
0 & 0
\end{pmatrix}.
\]

It is sufficient to show the stability of both \( J_{11} \) and \( J_{22} \). From (H1), (H2) and \( d_i > 0 \), \( J_{11} \) is an irreducible and quasi-positive matrix with negative column sum, and then we have \( s(J_{11}) < 0 \) from the Perron–Frobenius Theorem; see, for example, [15, Corollary 4.3.2]. From (3.5), \( J_{22}(I^*) = 0 \), where

\[
J_{22} = \begin{pmatrix}
\beta_i \left( \frac{N_i^*}{N_i^* - 1} - (d_i + \gamma_i) - \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} N_k^* - \sum_{k \in P} \frac{\partial f_{ik}(N_i^*, N_k^*)}{\partial x_k} f_k(N_k^*, N_i^*) (1 - \delta_{ij}) & 0 \\
0 & 0
\end{pmatrix}.
\]

and then \( s(J_{22}) = 0 \). In addition, a direct calculation gives

\[
J_{22} - J_{22} = \begin{pmatrix}
-\beta_i \frac{N_i^*}{N_i^* - 1} \delta_{ij} & 0 \\
0 & 0
\end{pmatrix} < 0.
\]

From [15, Corollary 4.3.2], we have \( s(J_{22}) < 0 \). Therefore, the endemic equilibrium \( E^* \) is locally asymptotically stable, and it completes the proof of the theorem. □

When the threshold \( R_0 > 1 \) in (3.4), the following result shows that the existence and the globally asymptotic stability of the unique endemic equilibrium still hold in (2.1) when the disease has mild effect on the dispersal of infectious individuals, i.e. \( g_{ij}(\cdot, \cdot) \approx f_{ij}(\cdot, \cdot) \) in (2.1), and the disease rarely induces death, i.e. \( v_j \approx 0 \) in (2.1). To study this case, we regard the variable dispersal rate of infectious individuals as the perturbation of the variable dispersal rate of susceptible individuals. By using a perturbation result for a globally asymptotically stable system in [23], we present a proof analogous to those in [4,28]. First, we set further notations to discuss this issue in (2.1). Equip the space of continuous functions \( \mathbb{R}_+^{2p} \to \mathbb{R}_+^{2p} \) with the norm \( \|g\|_{\infty} = \max_{i, j} \|g_{ij}\|_{\infty} \) for \( g = (g_{ij}) \), \( i, j \in P \), where \( \|h\|_{\infty} \) is the essential supremum of the function \( |h| \) for \( h : \mathbb{R}_+^{2p} \to \mathbb{R} \). The neighborhood of the function \( f = (f_{ij}) \), \( i, j \in P \), is defined as

\[
B(f, \varepsilon) = \{g = (g_{ij}) | \|g_{ij} - f_{ij}\|_{\infty} < \varepsilon, \text{ for } i, j \in P\}.
\]

**Theorem 5.** Assume (H1), (H2) hold and \( R_0 > 1 \) for \( f = g \). For this fixed \( f \), there exists an constant \( \bar{\varepsilon} > 0 \) such that for any \( g \in B(f, \varepsilon) \) with \( g_{ij} > 0 \) for \( i, j \in P \), the system (2.1) admits a unique endemic equilibrium \( E^*(g) = (S^*(g), I^*(g)) \), which is globally asymptotically stable with respect to \( X_1 \).

**Proof.** Note that the disease-free equilibrium \( (S^0, 0) \) is independent in functions \( g \). By (H1) and (H2), \( R_0 \) is continuous in the function \( g \). From the assumption, there exists an \( \tilde{\varepsilon} > 0 \) such that \( R_0 > 1 \) for \( g \in B(f, \varepsilon_0) \). Let \( \Pi(g, t) \) denotes the solution flow generated by (2.1). By Theorem 1, the bounded and closed set \( G \) that satisfies that for any \( (S, I) \in X_1 \) and \( g_{ij} \) with \( g \in B(f, \varepsilon_0) \), there exists \( t_0 = t_0(S, I) \) such that \( \Pi(g, t)(S, I) \in G \) for all \( t \geq t_0 \).

There are two criteria in [23] to determine the perturbation result for a globally stable equilibrium: (i) existence of a global attractor \( A_\delta \) of \( \Pi(g, t) \) as \( t \to \infty \) for each \( g \) near the function \( f \), (ii) for all \( t > 0 \), \( \Pi(g, t)A_\delta \) is uniformly bounded by a pre-compact set in \( X_1 \).
First, by Remark 4, for each $g \in B(f, \varepsilon_0)$, $\Pi(g, t)$ is uniformly persistent with respect to $(X_1, \partial X_0)$, and then by Hale and Waltman [29, Theorem 3.2], there is a global attractor $A_g$ of $\Pi(g, t)$: $X_1 \rightarrow X_1$.

From the proof in Theorem 1, $\Pi(g, t)X \rightarrow X$ is compact for all $g \in B(f, \varepsilon_0)$ and $t > 0$. Then for any fixed $t > 0$, $\Pi(\cdot, t)(X) = B(f, \varepsilon_0) \rightarrow X$ is continuous uniformly for $x$ in any bounded $Y \subset X$. By using the method in the proof of [30, Theorem 3.1] and regarding the parameter space as a functional space equipped with the previously defined norm, it shows that $\bigcup_{g \in B(f, \varepsilon_0)} \Pi(g, t)$ is compact in $X$. Let $\omega(x)$ be the omega limit set of $x \in X$ for $\Pi(g, t)$. Since $\omega(x)$ is invariant for $\Pi(g, t)$ and $\omega(x) \subset G$, it leads to $\bigcup_{g \in B(f, \varepsilon_0)} \omega(x) \subset \bigcup_{g \in B(f, \varepsilon_0)} \Pi(g, t) \omega(x) \subset \bigcup_{g \in B(f, \varepsilon_0)} \Pi(g, t) G$, and then $\bigcup_{g \in B(f, \varepsilon_0)} \omega(x)$ is compact in $X$. From Lemma 1, the case $g_{ij} > 0$ for $i, j \in P$ and $R_0 > 1$ implies a unique equilibrium $(S^0, 0)$ in $\partial X_0$ and $\bigcup_{x \in \partial X_0} \omega(x) = \{(S^0, 0)\}$. Clearly, $\{(S^0, 0)\}$ is an acyclic covering of itself. In addition, from Theorem 3 and Remark 4, there exists $\xi = \xi(\varepsilon_0) > 0$ such that for any $g \in B(f, \varepsilon_0)$, we have $\limsup_{t \rightarrow +\infty} \| \Pi(g, t)(x) - (S^0, 0) \| \geq \xi$, for all $x \in X_1$. Then, by the theorem on the uniform persistence uniform in parameters [30, Theorem 4.3 and Remark 4.2], there exist $\varepsilon_1 \in (0, \varepsilon_0)$ and $\tau > 0$, such that $\liminf_{t \rightarrow +\infty} d(\Pi(g, t)(x), \partial X_0) \geq \tau$ for all $x \in X_1$ and $g \in B(f, \varepsilon_1)$ with $g_{ij} > 0$ for $i, j \in P$. Thus, there is a compact and positive invariant subset $G_0$ of $X_1$ such that $A_g \subset G_0$ for all $g \in B(f, \varepsilon_1)$. Therefore, for all $t > 0$,

$$\bigcup_{g \in B(f, \varepsilon_1)} \Pi(g, t) A_g \subset \bigcup_{g \in B(f, \varepsilon_1)} \Pi(g, t) G_0 \subset G_0 \subset X_1,$$

and then $\bigcup_{g \in B(f, \varepsilon_1)} \Pi(g, t) A_g$ is compact in $X_1$.

By Theorem 4, system (2.1) admits an endemic equilibrium which is globally asymptotically stable in $X_1$. From previous results and the perturbation theory in [23, Theorem 2.2], there exists an $\varepsilon \in (0, \varepsilon_1)$ such that for any $g \in B(f, \varepsilon)$ with $g_{ij} > 0$ for $i, j \in P$, the system (2.1) admits an endemic equilibrium $E^*(g) = (S^*(g), I^*(g))$ which is globally asymptotically stable in $X_1$. □

Remark 4. The uniform boundedness in Theorem 1 refers to the general mode (2.1). Therefore, we can consider the model with disease-induced death by regarding the death rate as a perturbed parameter and by using the same theory to show that an endemic equilibrium, if exists, is globally asymptotically stable in the system (2.1).

4. Effect of media coverage

Theoretical results in previous section inform us the global dynamics of (2.1), i.e., all solutions either approach to the disease-free equilibrium or converge to the unique endemic equilibrium, according to the level of the basic reproduction number. In addition, this threshold was numerically shown to depend on the strategy of adaptive dispersal. From the biological viewpoint, there are further questions to be addressed. For examples, (i) How does the media coverage affect the distribution of survival population among connected regions when the disease goes extinction? (ii) If the disease is pandemic, the same question arises for all population, especially for the infectious individuals. (iii) How does the media coverage influence the speed of disease to goes extinction? To investigate these problems, we carry out three examples for (2.1) with two patch.

To explore the problem (ii), we address the first example with adaption in a single patch. Consider the model (2.1) with adaptive dispersal ($\lambda''$) and (L), choose parameters $A_i = 1, d_i = 0.2, \beta_i = 0.8, \gamma_i = 0.2, \nu_i = 0, a_i = 1$ for $i = 1, 2, b_{12} = b_1 = b_2 = 0$, and change the value of $b_{21}$ to see how the adaptive dispersal affects the disease distribution. Fig. 2 depicts the values of $I_1^*$ and $I_2^*$ depending on the inner-elsion rate, $b_{21}$, in the patch-1. First, taking inner-elasion in patch-1 helps inner control of disease spread, but damages the disease control in patch-2. Hence, adopting adaptive dispersal in a patchy disease model represents competition of disease control between different patches. Second, for fixed positive $b_{21}$, the population of infectious individuals $I_1^*$ in patch-1 under linearly adaptive dispersal rate, is smaller than that under the case of nonlinear adaption. Therefore, it is more efficient to control disease by adopting linearly adaptive dispersal rather than nonlinearly.
adaptive dispersal. On the other hand, the linear case also induces more disservice on disease control than the nonlinear one in the other patch-2.

We further simulate the model (2.1) with adaption in both patches to see the competition between patches. In the following, we also choose previous parameters except $b_{12} = kb_{12}$ for positive constant $k$, and the nonlinearly adaptive dispersal ($\mathcal{N}'$). Taking different values of $k$ means different levels of dispersal adaption in two patches. In Fig. 3, when $k = 1$, the same level of adaption are adopted in both patches, and then the intensity of adaption, values of $b_{12} = b_{12}$, does not improve the disease control in both patches. For larger $k = 2$ or 5, patch-1 adapts stronger adaption related to that in patch-2, and then higher level of adaption obviously improves the disease control in patch-1.

To explore the problems (i) and (iii), that is for the case $R_0 < 1$, we investigate in Fig. 4 the effect of media coverage on the time that it takes for a pre-existing epidemic to go extinct and on the distribution of final healthy population. Herein, we simulate the evolution of each solution to system (2.1) with the functional response ($\mathcal{N}'$) by setting $A_1 = 1$. $d_1 = 0.05$. $\beta_1 = 0.2$. $\nu_1 = 0.05$. $\gamma_1 = 0.1$. $a_1 = 0.5$. $d_2 = 0.02$. $\beta_2 = 0.1$. $\nu_2 = 0.05$. $\gamma_2 = 0.1$. $a_2 = 0.5$ and respectively setting the media intensity $b_{21}$, $b_{12}$ as (a) $b_{21} = b_{12} = 0$.

(b) $b_{21} = 6$. $b_{12} = 0$. (c) $b_{21} = 0$. $b_{12} = 6$. (d) $b_{21} = b_{12} = 6$. Without media coverage in both patches, Fig. 4a shows that the natural dispersal can eliminate the disease as the theoretical results in previously mentioned literature. Comparing Fig. 4b or c with a, it reveals that taking the strategy of media coverage would lead to a large reduction in time to extinction of the disease in the local patch. On the other hand, it also greatly reduce the total population of the patch that executes the policy. Finally, Fig. 4d reveals an interesting result that, by taking the same intensity of media coverage, the amounts of both compartments in different patches synchronize in time.

The model (2.1) is a prototype to describe possible adaptive dispersal between different regions due to the threat of an infectious disease. It assumes a constant intensity, $c_{il}$, of media report in each patch. In fact, there may exists complicated process leading to the outbreak of a disease, and the intensity of media varies to reflect different levels of disease spread. Here, we review the spread of severe acute respiratory syndrome (SARS) in 2003 to see how the media report affected the strategy of dispersal between Mainland China and Taiwan, two patches which are connected mainly by airplanes, during the period of epidemic. We refer to the monthly data of the ratio of immigration among the total population reported by Department of Statistics, Ministry of the Interior, Taiwan [31]. Fig. 5a depicts the effect of SARS on the number of immigration from Mainland China (patch-2) into Taiwan (patch-1). The dash curve represents the ratio of immigration for each month in 2002 by natural dispersal from Mainland China into Taiwan $(a_{12})$, and the solid curve is the ratio of each month in 2003 affected by the impact of SARS. In Fig. 5b, we calculate the intensity of adaption according to the data in Fig. 5a and the mode of adaption ($\mathcal{N}'$) with $b_{12} = 0$, for focusing on the role of $c_{12}$. According to the report of World Health Organization, the first onset of SARS in Taiwan happened at 2003 March 13, and the broad spread begun around mid-April and ended at mid-June [32]. The intensity of adaption in Fig. 5b, which reveals larger values from April to July, agrees with the period of outbreak of SARS. Therefore, the intensity of media influence varies to reflect different levels of disease spread and the dispersal rate of population between Mainland China and Taiwan does change and depend on the intensity of media report.

5. Results and discussion

We used the proposed model to investigate the influence of media coverage among spatially distinct patches of habitat by adapting dispersal rates. Our results showed that the basic reproduction number $R_0$ was a threshold parameter of disease spread. Specifically, the epidemic model either has a globally asymptotically stable disease-free equilibrium when $R_0 < 1$, or a globally asymptotically stable endemic equilibrium when $R_0 > 1$, if the disease mildly influences the dispersal of infectious individuals, and if the disease rarely induces death.

In [7,8], media coverage was assumed to affect only the interactions of the local population through the incidence rate, and did not play a role in $R_0$ because the basic reproduction number was evaluated in the absence of disease. By contrast, from (3.2) and (3.3), the assumption in our model on media-induced adaption effects on dispersal affects this threshold value for predicting disease outbreak. In addition to theoretically determining the convergence dynamics under certain cri-
teria, we identified that (i) When the disease spread cannot be controlled under a natural dispersal, applying the strategy of media coverage in an endemic patch can help annihilate the disease in the connected system, whereas media coverage from a disease-free patch exacerbates disease control. (From the discussion in subsection 3.2.) (ii) When the disease is a pandemic (i.e., $R_0 > 1$), increasing the intensity of media coverage can substantially reduce the number of infectious individuals at the endemic equilibrium, and reveals competition for disease control in different patches. (From Fig. 2 and 3) (iii) When $R_0 > 1$, applying the strategy of media coverage results in a considerable reduction in the time for disease extinction in local patch, but substantially reduces the total population of the patch that executes the policy. (From Fig. 4) (iv) By applying the same intensity of media coverage, the amount of both compartments in different patches synchronizes over time. (From Fig. 4)

Unanswered questions concerning the general model (2.1) remain. For example, does the model possibly involve multiple endemic equilibria? Can it exhibit a periodic state through Hopf bifurcation? Time delay is another intrinsic characteristic in biological systems. In [33], oscillations can be induced by disease related death in a model with maturation delay. A question arises in (2.1) that if there exists an oscillation caused by the time delay from the transmission of media information. In addition, diseases without a latent period exist, and individuals move to avoid those with symptoms. In such an epidemic model, setting the dispersal rates depending on the infectious individuals instead of the total population is reasonable; in other words, replacing $f_i(N_i, N_j)$ and $g_{ij}(N_i, N_j)$ with $f_i(l_i, l_j)$ and $g_{ij}(l_i, l_j)$, for $i, j \in P$ in model (2.1). In this case, the basic reproduction number is then independent on the adaption mechanism, and the global dynamics of DFE ($R_0 < 1$) and the persistence of disease ($R_0 > 1$) can be demonstrated by the same manner in this study. However, it is expected to be difficult to deal with the global dynamics of the endemic case. Therefore, we have reserved these problems for our future.

**Fig. 4.** Effect of media coverage on the time to extinction of a pre-existing epidemic and final population distribution, (a) $b_{21} = b_{12} = 0$, (b) $b_{21} = 6$, $b_{12} = 0$, (c) $b_{21} = 0$, $b_{12} = 6$, (d) $b_{21} = b_{12} = 6$. 


study. In addition to the SIS model with contagious transmission, adaptive dispersal can be considered in other epidemic models, including noncontagious diseases such as vector-host models.

Acknowledgments

This work is partially supported by the Ministry of Science and Technology of Taiwan (grant MOST 105-2115-M-153-004).

References

[1] H.W. Hethcote, Qualitative analyses of communicable disease models, Math. Biosci. 28 (1976) 335–356.
[2] J. Arino, P. van den Driessche, A multi-city epidemic model, Math. Popul. Stud. 10 (2003) 175–193.
[3] W. Wang, X.Q. Zhao, An endemic model in a patchy environment, Math. Biosci. 190 (2004) 97–112.
[4] Y. Jin, W. Wang, The effect of population dispersal on the spread of a disease, J. Math. Anal. Appl. 308 (2005) 343–364.
[5] R.A. Lintott, R.A. Norman, A.S. Hoyle, The impact of increased dispersal response to disease control in patchy environments, J. Theor. Biol. 323 (2013) 57–68.
[6] G. Wobeser, Disease management strategies for wildlife, Rev. Sci. Tech. 21 (2002) 159–178.
[7] J. Cui, Y. Sun, H. Zhu, The impact of media on the control of infectious diseases, J. Dyn. Differ. Equ. 20 (2008) 31–53.
[8] C. Sun, W. Yang, J. Arino, K. Khan, Effect of media-induced social distancing on disease transmission in a two patch setting, Math. Biosci. 230 (2011) 87–95.
[9] J.A. Wiens, The landscape context of dispersal, in: Dispersal, Oxford University Press Oxford, United Kingdom, 2001.
[10] H.W. Hethcote, The mathematics of infectious diseases, SIAM Rev. 42 (2000) 599–653.
[11] H.L. Smith, P. Waltman, The Theory of the Chemostat, Cambridge, 1995.
[12] M. Salmani, P. van den Driessche, A model for disease transmission in a patchy environment, Discret. Contin. Dyn. Syst. Ser. B 6 (2006) 185–202.
[13] C.D. Ruxton, Density-dependent migration and stability in a system of linked population, Bull. Math. Biol. 58 (1996) 643–660.
[14] L. Perko, Differential Equations and Dynamical Systems, third ed., Springer-Verlag, New York, 2001.
[15] H.L. Smith, Monotone dynamical systems: an introduction to the theory of competitive and cooperative systems, in: Mathematical Surveys and Monographs, 41, AMS, Providence, RI, 1995.
[16] Z. Lu, Y. Takeuchi, Global asymptotic behavior in single-species discrete diffusion systems, J. Math. Biol. 32 (1993) 67–77.
[17] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, On the definition and the computation of the basic reproduction ratio $r_0$ in models for infectious diseases in heterogeneous populations, J. Math. Biol. 28 (1990) 365–382.
[18] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002) 29–48.
[19] D. Gao, S. Ruan, An SIS patch model with variable transmission coefficients, Math. Biosci. 232 (2011) 110–115.
[20] J.C. Kamgang, G. Sallet, Computation of threshold conditions for epidemiological models and global stability of the disease-free equilibrium (DFE), Math. Biosci. 213 (2008) 1–12.
[21] C. Castillo-Chavez, H. Thieme, O. Arino, M. Kimmel, M. Langlais, Asymptotically autonomous epidemic models, in: D. Axelrod (Ed.), Mathematical Population Dynamics: Analysis of Heterogeneity, Wuerz, 1995, pp. 33–50.
[22] H.R. Thieme, Convergence results and a Poincaré-Bendixson trichotomy for asymptotically autonomous differential equations, J. Math. Biol. 30 (1992) 755–763.
[23] H.L. Smith, P. Waltman, Perturbation of a globally stable steady state, Proc. Amer. Math. Soc. 127 (1999) 447–453.
[24] H.R. Thieme, Persistence under relaxed point-dissipativity (with application to an endemic model), SIAM J. Math. Anal. 24 (1993) 407–435.
[25] X.Q. Zhao, Uniform persistence and periodic coexistence states in infinite dimensional periodic semiflows with applications, Can. Appl. Math. Quart. 3 (1995) 473–495.
[26] X.Q. Zhao, Dynamical Systems in Population Biology, Springer, New York, 2003.
[27] X.Q. Zhao, Z.J. Jing, Global asymptotic behavior in some cooperative systems of functional differential equations, Can. Appl. Math. Quart. 4 (1996) 421–444.
[28] X.Q. Zhao, X. Zou, Threshold dynamics in a delayed SIS epidemic model, J. Math. Anal. Appl. 257 (2001) 282–291.
[29] J.K. Hale, P. Waltman, Persistence in infinite-dimensional systems, SIAM J. Math. Anal. 20 (1989) 388–395.
[30] H.L. Smith, X.-Q. Zhao, Dynamics of a periodically pulsed bio-reactor model, J. Differ. Equ. 155 (1999) 368–404.
[31] Department of Statistics, Ministry of the Interior, Taiwan, Entry persons by sex, age, identification, http://www.moi.gov.tw/stat/english/index.asp.
[32] World health organization, Epidemic curves-Severe Acute Respiratory Syndrome (SARS). http://www.who.int/csr/sars/epicurve/epiindex/en/.
[33] K. Cooke, P. van den Driessche, X. Zou, Interaction of maturation delay and nonlinear birth in population and epidemic models, J. Math. Biol. 39 (1999) 332–352.