EFFECT OF MEDIA-INDUCED MODIFICATION OF TRAVEL RATES ON DISEASE TRANSMISSION IN A MULTIPLE PATCH SETTING*

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Abstract A general SIS epidemic model is formulated that incorporates media-induced modification of travel rates. Basic local properties of solutions to the model are established. In particular, it is shown that the basic reproduction number does not involve parameters related to the effect of media on travel. The general model is subsequently specialised to two-patch models, with two different scenarios regarding patch population size. Qualitative analyses show that the basic reproduction number acts as a sharp threshold between disease persistence and extinction. The concept of uniform weak persistence is used to prove the existence of an endemic equilibrium and disease uniform strong persistence under a certain condition. Numerical investigations are carried out to gain insight into the analytically tractable and intractable cases, highlighting the importance of considering not only the basic reproduction number but also other measures of disease severity.

Keywords Epidemiology, metapopulations, media coverage, nonlinear travel rates, population sizes.

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1. Introduction

Communication in mass media has been sometimes employed as a tool in the effort to control and mitigate epidemics of emerging and re-emerging diseases [9, 34, 38]. Media inform the public of cases of infection during an epidemic and accordingly influence perceptions of the threat of infectious diseases [9, 18, 49]. Media coverage during the 2002-2003 severe acute respiratory syndrome (SARS) [11], the 2009 H1N1 influenza [27] and 2019 novel coronavirus disease (COVID-19) outbreak-

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s [10, 12, 13, 19] delivered preventive health information, alerted the public to take precautionary measures (e.g., wearing masks, washing hands frequently, avoiding crowds) in relation to a disease outbreak. It has been shown to elicit individuals' positive behavior change [9, 18, 22, 43]. This in turn reduces the frequency of potentially infecting contacts and helps lower the probability of disease transmission among the well-informed population.

The impact of media alerts on the propagation of communicable diseases has gained much attention and been studied extensively [9, 18, 22, 34, 38, 43, 49]. Recently, a number of mathematical models have been proposed to tackle this issue. However, existing modelling approaches focus on the effect of media-induced social distancing on disease transmission. For instance, [15,16,30,31,44,47] and references therein incorporated an effect of media coverage into epidemic models by adding a term that directly affects contact rates. Instead of employing different incidence function to describe media effect, [14, 21, 32, 37, 45, 48] suggested adding a mass media compartment, reflecting the involvement of mass media in an epidemic. All mathematical models cited above assume that space is homogeneous and confine the population to a single location or population.

However, infectious diseases usually spread heterogeneously in space and time; integrating spatial heterogeneity into epidemic models provides a more accurate description of reality [2, 4, 7, 8, 28, 35]. To explore the impact of media coverage in a heterogeneous environment, [24] formulated and studied a spatio-temporal SIS reaction-diffusion model, while [23,40] took individual travel into account, and proposed epidemiological models in a multiple patch setting. (A patch here refers to a geographical location; it can be a city, a region or a country.)

Media coverage of an epidemic can affect individuals' travel intentions, resulting in a reduction of the volume of travel. The influence of media-induced modifications of travel rates between patches on the dynamics of disease propagation has not been the object of much attention. A disease outbreak generally does not result in complete interruption of travel between patches, but people tend to avoid areas where incidence of the disease is high; travellers are especially affected by disease-induced panic. One may naturally imagine that the reporting on a disease outbreak by media may amplify the degree of this sort of fear or panic and consequently lead to a drop of the number of travellers. During the 2002-2003 SARS outbreak, the World Health Organization (WHO) issued a recommendation advising visitors to China to consider postponing or cancelling their trips [11]; they issued a similar recommendation regarding travel to Toronto. Most overseas tourist groups to Beijing scheduled for April and May of 2003 were cancelled; in April, the city's foreign tourist arrivals dropped 59.9% compared with the same period in 2002 [11]. During the 2009 H1N1 pandemic, it was, for instance, observed that air traffic to Mexico was affected in the early stages of the epidemic [27].

On the modelling side, in mathematical epidemiology there are very few models with nonlinear movement rates, only ones with variable rates. Wang and Zhao [46] considered a two-patch epidemic model with the coefficients of movement rates being periodic. Dhirasakdanon et al. [17] formulated a general n-patch SEIRS host model, in which individuals may alter their travel rates in terms of the disease status of the host population, and established a sharp threshold to separate disease persistence from the extinction of small disease outbreaks.

In the same spirit, a metapopulation model for the spread of infection within and between patches is considered here to incorporate the adjustment of travel rates to

media alerts and to study its resulting effect on the spatial and temporal spread of an infectious disease. A general SIS (susceptible-infective-susceptible) metapopulation model in a $|\mathcal{P}|$ patch setting is developed, integrating nonlinear media-induced modification of travel rates. Two scenarios in perspective of population size in a two patch setting are considered: in the first scenario, the population levels in both patches are high and they both possess proportional incidence of infection; in the second scenario, motivated by the work of Arino and McCluskey [5], Arino and Portet [6], Fromont et al. [20] and Zhao and Wang [46], the population in one patch is large and incidence is modelled using using proportional incidence, while in the other patch the population is small and mass action incidence is used. The following questions are addressed:

Q1. Does media-induced modification of travel rates affect the epidemiology of a disease outbreak?

Q2. Is there any differentiation in effect of media-induced modification of travel rates for systems with different population sizes?

Q3. Does the population size of coupled patches play a role in facilitating the spread of an infectious disease?

Analytical analyses are conducted to derive a critical threshold of disease persistence and extinction, and to determine sufficient conditions under which an infectious disease is uniformly weakly and strongly persistent, respectively. Numerical simulations are subsequently performed to complement and further extend analytical results.

2. Modelling

2.1. Assumptions

Consider the transmission of a disease conferring no immunity against reinfection that can be modelled using an SIS epidemic model (e.g., common cold or staphylococcus aureus). Set this in a metapopulation setting, i.e., assume that there is a set of locations called *patches*, with the dynamics in each patch described by an SIS model and movement of individuals between the patches; for more details on metapopulation models, see [2] and the references therein. The population in each patch is classified into two categories: individuals who are susceptible to the disease (S) and those who are infected with the disease and infectious to others (I). The simple SIS formalism allows to capture the main characteristics of disease propagation without the burden of additional equations and parameters. Further, this allows to focus on the effect of modification of travel rates between patches led by media coverage, since the dynamics of a classical SIS metapopulation model without media effects, or with effect of media-induced social distancing is well understood.

Let $\mathcal{P}$ be the set of patches. Individuals in patch $p \in \mathcal{P}$ move to patch $q \in \mathcal{P}$ at the *per capita* rate $m_{qp} \geq 0$, with $m_{qp} = 0$ if movement from p to q is not possible. For simplicity of notation, we assume that for all $p \in \mathcal{P}$,

$$m_{pp} = - \sum_{q \in \mathcal{P} \setminus \{p\}} m_{qp}.$$

Before proceeding further, let us briefly discuss the model used in each patch in the absence of mobility. We make the following assumptions for the dynamics in patch $p \in \mathcal{P}$.

H1. Birth occurs at the rate b_p with all births in the susceptible compartment; death rate occurs at the *per capita* rate d_p.

H2. Recovery occurs at the *per capita* rate γ_p. There is no immunity, so upon recovery, individuals are immediately susceptible to the disease again.

H3. Disease transmission from infectious to susceptible individuals is described by a standard (proportional) incidence function $\beta_p S_p I_p / N_p$ where β_p is the effective contact rate in patch p.

The model in each patch, in the absence of movement between patches, then has the flow diagram showed in Figure 1.
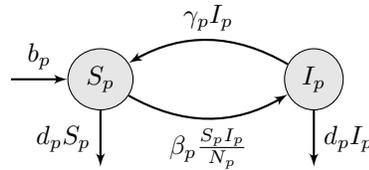
Figure 1. Flow diagram of the SIS model in an isolated patch $p \in \mathcal{P}$.

Media coverage may exert negative effects over the travel of individuals between patches. The following assumptions are made regarding these effects.

H4. Both susceptible and infectious individuals travel.

H5. Knowledge of the presence of disease leads to modification of the rate of travel between patches for all susceptible individuals. If $m_{pq} \geq 0$ is the rate of travel from patch $q \in \mathcal{P}$ to patch $p \in \mathcal{P}$, then the revised travel rate for individuals takes the form

$$\left(1 - \frac{\sigma_p I_p}{\alpha_p + \sigma_p I_p}\right) m_{pq}, \tag{2.1}$$

where $\alpha_p > 0$ describes the insensitivity of response to knowledge of cases and $\sigma_p \in [0, 1]$ is the fraction of cases known in location p.
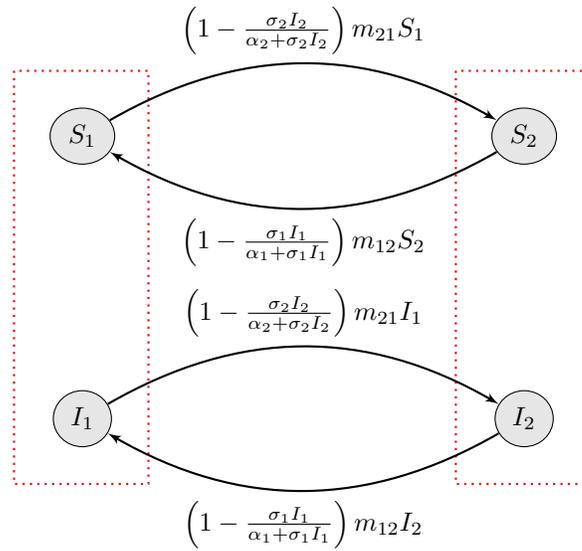
2.2. General model

For all $p \in \mathcal{P}$, the general SIS metapopulation model with media-induced change of travel rates takes the form

$$S_p' = b_p + \gamma_p I_p - \beta_p \frac{S_p I_p}{N_p} - d_p S_p + \sum_{q \in \mathcal{P}} \left(1 - \frac{\sigma_q I_q}{\alpha_q + \sigma_q I_q}\right) m_{pq} S_q, \tag{2.2a}$$

$$I_p' = \beta_p \frac{S_p I_p}{N_p} - (\gamma_p + d_p) I_p + \sum_{q \in \mathcal{P}} \left(1 - \frac{\sigma_q I_q}{\alpha_q + \sigma_q I_q}\right) m_{pq} I_q \tag{2.2b}$$

with non-negative initial conditions $S_p(0) > 0, I_p(0) \geq 0$ for all $p \in \mathcal{P}$ and $\sum_{p \in \mathcal{P}} I_p(0) > 0$.

Figure 2. Flow diagram of the movement component of the SIS model, in the special case of two patches. Flows within patches are not shown for legibility.

3. Mathematical analysis of the general model

3.1. Preliminaries

The vector field of (2.2) is locally Lipschitz so solutions with the prescribed initial conditions uniquely exist. Furthermore, (2.2) is well posed in the following sense.

Theorem 3.1. *Consider* (2.2) *with the initial conditions* $S_p(0) > 0, I_p(0) \geq 0$ *for all* $p \in \mathcal{P}$ *and* $\sum_{p \in \mathcal{P}} I_p(0) > 0$. *Then the positive orthant* $\mathbb{R}_+^{2|\mathcal{P}|}$ *is invariant under the flow of* (2.2), *with* S_p *remaining positive and* I_p *staying non-negative for all* $p \in \mathcal{P}$. *The total population converges to an equilibrium as* $t \to \infty$ *and solutions are bounded.*

Proof. Under the stated initial conditions $S_p(0) > 0, I_p(0) \geq 0$ for all $p \in \mathcal{P}$ and $\sum_{p \in \mathcal{P}} I_p(0) > 0$, suppose, without loss of generality, that I_1 becomes zero at some time t_1 before another I_p becomes zero, $p \in \mathcal{P} \setminus \{1\}$. Then from (2.2b), $I_1'(t_1) = \sum_{q \in \mathcal{P} \setminus \{1\}} \left(1 - \frac{\sigma_q I_q}{\alpha_q + \sigma_q I_q}\right) m_{pq} I_q \geq 0$; thus $I_1(t)$ is a non-decreasing function of t at t_1. Hence, I_1 stays non-negative. Similar reasoning holds for all other I_p, $p \in \mathcal{P}$. Again, without loss of generality, now suppose that at some time t_2, $S_1(t_2) = 0$ before any other S_p goes to zero, $p \in \mathcal{P} \setminus \{1\}$. Then at t_2, from (2.2b), $S_1'(t_2) = b_1 + \gamma_1 I_1 + \sum_{q \in \mathcal{P} \setminus \{1\}} \left(1 - \frac{\sigma_q I_q}{\alpha_q + \sigma_q I_q}\right) m_{pq} S_q > 0$. Thus, there is no time t_2 such that $S_1(t_2) = 0$, considering the contradiction of $S_1(t_2^-) < 0$. Hence, S_1 stays positive for all $t > 0$ when the initial condition $S_p(0) > 0$. By the similar argument, we obtain the positivity of all S_p, $p \in \mathcal{P}$.

Let $N_p(t) = S_p(t) + I_p(t)$ denote the total population of patch $p \in \mathcal{P}$ at time t and $N(t) = \sum_{p \in \mathcal{P}} N_p(t)$ be the total population in the system. Adding up the equations in (2.2) gives $N' = \sum_{p \in \mathcal{P}} b_p - \sum_{p \in \mathcal{P}} d_p N_p$. It follows that $\liminf_{t \to \infty} N(t) \geq (\sum_{p \in \mathcal{P}} b_p)/\max_{p \in \mathcal{P}}\{d_p\}$ and $\limsup_{t \to \infty} N(t) \leq (\sum_{p \in \mathcal{P}} b_p)/\min_{p \in \mathcal{P}}\{d_p\}$. Now

that the positive orthant $\mathbb{R}_+^{2|\mathcal{P}|}$ is invariant under (2.2) and the total population is bounded, the individual components S_p and I_p are also bounded. $\qquad\square$

Moreover, the closed subset of $\mathbb{R}_+^{2|\mathcal{P}|}$,

$$\Gamma = \left\{ (S_p, I_p) \in \mathbb{R}_+^{2|\mathcal{P}|} : \frac{\sum_{p \in \mathcal{P}} b_p}{\max_{p \in \mathcal{P}}\{d_p\}} \le \sum_{p \in \mathcal{P}} (S_p + I_p) \le \frac{\sum_{p \in \mathcal{P}} b_p}{\min_{p \in \mathcal{P}}\{d_p\}} \right\}$$

is also positively invariant under the flow of (2.2).

3.2. Disease-free equilibrium

System (2.2) always has a unique disease-free equilibrium (DFE). To compute it, set $I_p = 0$ for all $p \in \mathcal{P}$ (which we write $\mathbf{I} = \mathbf{0}$) and rewrite the remaining equations (2.2a) in vector form:

$$\mathbf{S}' = \mathbf{b} - \mathbf{d}\mathbf{S} + \mathcal{M}\mathbf{S}, \tag{3.1}$$

where $\mathbf{S} = (S_1, \ldots, S_{|\mathcal{P}|})^T$, $\mathbf{b} = (b_1, \ldots, b_{|\mathcal{P}|})^T$, $\mathbf{d} = \mathsf{diag}(d_1, \ldots, d_{|\mathcal{P}|})$ and

$$\mathcal{M} = \begin{pmatrix} -\sum\limits_{p \in \mathcal{P}\backslash\{1\}} m_{p1} & m_{12} & \cdots & m_{1|\mathcal{P}|} \\ m_{21} & -\sum\limits_{p \in \mathcal{P}\backslash\{2\}} m_{p2} & \cdots & m_{2|\mathcal{P}|} \\ \vdots & \vdots & \ddots & \vdots \\ m_{|\mathcal{P}|1} & m_{|\mathcal{P}|2} & \cdots & -\sum\limits_{p \in \mathcal{P}\backslash\{|\mathcal{P}|\}} m_{p|\mathcal{P}|} \end{pmatrix}. \tag{3.2}$$

Consider equilibria of (3.1). We have

$$\mathbf{S}^\star = (\mathbf{d} - \mathcal{M})^{-1}\mathbf{b},$$

provided that $\mathbf{d} - \mathcal{M}$ is invertible. Since $d_p > 0$ for all $p \in \mathcal{P}$, by [3, Proposition 3(3)], $\mathbf{d} - \mathcal{M}$ is invertible and $(\mathbf{d} - \mathcal{M})^{-1} > \mathbf{0}$. From [3, Proposition 3(4)], if, additionally, $\mathcal{M}$ is irreducible, in other words, if the digraph of patches is strongly connected, then it is not required for the death rates d_p to all be positive in order for $(\mathbf{d} - \mathcal{M})^{-1} \gg \mathbf{0}$, i.e., be entry-wise positive.

Irreducibility of the movement digraph is a reasonable assumption to make, and we assume from now on it holds. We therefore have the DFE

$$E_0^{(2.2)} := (\mathbf{S}^\star, \mathbf{I}^\star) = \left((\mathbf{d} - \mathcal{M})^{-1}\mathbf{b}, \mathbf{0} \right), \tag{3.3}$$

with $\mathbf{S}^\star \gg \mathbf{0}$.

3.3. Basic reproduction number

The stability of the disease-free equilibrium $E_0^{(2.2)}$ is considered in terms of the general basic reproduction number $\mathcal{R}_0^{(2.2)}$, i.e., the basic reproduction number for

the whole system. We follow the next generation matrix method for deterministic compartmental models [42]. Let

$$
\mathbb{F} = \begin{pmatrix} \beta_1 \dfrac{S_1 I_1}{N_1} \\ \vdots \\ \beta_{|\mathcal{P}|} \dfrac{S_{|\mathcal{P}|} I_{|\mathcal{P}|}}{N_{|\mathcal{P}|}} \end{pmatrix}
$$

and

$$
\mathbb{V} = \begin{pmatrix} (d_1 + \gamma_1) I_1 - \sum_{q \in \mathcal{P}} \left(1 - \frac{\sigma_q I_q}{\alpha_q + \sigma_q I_q} \right) m_{1q} I_q \\ \vdots \\ (d_{|\mathcal{P}|} + \gamma_{|\mathcal{P}|}) I_{|\mathcal{P}|} - \sum_{q \in \mathcal{P}} \left(1 - \frac{\sigma_q I_q}{\alpha_q + \sigma_q I_q} \right) m_{|\mathcal{P}|q} I_q \end{pmatrix}
$$

be, respectively, the vectors of new infections and all other flows within the infected compartments (the latter with a negative sign). Taking partial derivatives of $\mathbb{F}$ and $\mathbb{V}$ with respect to $\mathbf{I}$ and evaluating at $E_0^{(2.2)}$ gives $F = \mathsf{diag}(\beta_1, \ldots, \beta_{|\mathcal{P}|})$ and

$$
V = \mathsf{diag}(d_1 + \gamma_1, \ldots, d_{|\mathcal{P}|} + \gamma_{|\mathcal{P}|}) - \mathcal{M},
$$

where F is non-negative and V is an M-matrix. Then FV^{-1} is non-negative. From [42, Theorem 2], we have the following result.

Lemma 3.1. *Let $\mathcal{R}_0^{(2.2)} := \rho(FV^{-1})$, where $\rho(\cdot)$ is the spectral radius. For system (2.2), the disease-free equilibrium $E_0^{(2.2)}$ is locally asymptotically stable if $\mathcal{R}_0^{(2.2)} < 1$ and unstable if $\mathcal{R}_0^{(2.2)} > 1$.*

Note that neither α_q nor σ_q appear in $\mathcal{R}_0^{(2.2)}$. Thus, media coverage does not change the basic reproduction number $\mathcal{R}_0^{(2.2)}$. This is to be expected: computation of $\mathcal{R}_0^{(2.2)}$ takes place where $\mathbf{I} = \mathbf{0}$; when disease is absent, there is no change in travel rates and system (2.2) is reduced to a regular SIS metapopulation model. This is similar to what was observed, for example, in [40].

4. Mathematical analysis of the two-patch model

We now consider the case of only two patches, i.e, where $\mathcal{P} = \{1, 2\}$, which we assume from now on. This allows us to investigate global properties of the model more in detail. It also provides a much easier framework for performing numerical simulations. In this case, model (2.2) takes the form

$$
S_1' = b_1 + \gamma_1 I_1 - \beta_1 \frac{S_1 I_1}{N_1} - d_1 S_1 - m_{21} \left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2} \right) S_1 + m_{12} \left(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1} \right) S_2,
\tag{4.1a}
$$

$$
I_1' = \beta_1 \frac{S_1 I_1}{N_1} - (\gamma_1 + d_1) I_1 - m_{21} \left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2} \right) I_1 + m_{12} \left(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1} \right) I_2,
\tag{4.1b}
$$

$$
S_2' = b_2 + \gamma_2 I_2 - \beta_2 \frac{S_2 I_2}{N_2} - d_2 S_2 - m_{12} \left(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1} \right) S_2 + m_{21} \left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2} \right) S_1,
\tag{4.1c}
$$

$$I_2' = \beta_2 \frac{S_2 I_2}{N_2} - (\gamma_2 + d_2)I_2 - m_{12}\left(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\right)I_2 + m_{21}\left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\right)I_1$$

(4.1d)

with non-negative initial conditions

$$S_p(0) > 0, I_p(0) \geq 0, \sum_{p \in \mathcal{P}} I_p(0) > 0 \tag{4.2}$$

for $p \in \mathcal{P}$. All results established in Section 3 hold for system (4.1), of course. In particular, we have that, at the disease-free equilibrium $E_0^{(4.1)}$,

$$
\begin{aligned}
\mathbf{S}_0^\star &= \begin{pmatrix} d_1 + m_{21} & -m_{12} \\ -m_{21} & d_2 + m_{12} \end{pmatrix}^{-1} \begin{pmatrix} b_1 \\ b_2 \end{pmatrix} \\
&= \begin{pmatrix} \dfrac{(d_2 + m_{12})b_1 + m_{12}b_2}{d_1 m_{12} + d_2 m_{21} + d_1 d_2} \\ \dfrac{m_{21}b_1 + (d_1 + m_{21})b_2}{d_1 m_{12} + d_2 m_{21} + d_1 d_2} \end{pmatrix}.
\end{aligned}
\tag{4.3}
$$

Also, when computing the basic reproduction number, $F = \mathsf{diag}(\beta_1, \beta_2)$ and

$$V = \begin{pmatrix} d_1 + \gamma_1 + m_{21} & -m_{12} \\ -m_{21} & d_2 + \gamma_2 + m_{12} \end{pmatrix} =: \begin{pmatrix} v_{11} & -m_{12} \\ -m_{21} & v_{22} \end{pmatrix}$$

that give the basic reproduction number

$$\mathcal{R}_0^{(4.1)} = \frac{\beta_1 v_{22} + \beta_2 v_{11} + \sqrt{(\beta_2 v_{11} - \beta_1 v_{22})^2 + 4\beta_1 \beta_2 m_{12} m_{21}}}{2(v_{11} v_{22} - m_{12} m_{21})}. \tag{4.4}$$

Note that parameters $\sigma_\mathcal{P}$ and $\alpha_\mathcal{P}$ do not appear in $\mathcal{R}_0^{(4.1)}$, so the introduction of media effect on travel does not change the basic reproduction number. When media coverage does not affect travel rates, i.e., $\sigma_\mathcal{P} = 0$ and or $\alpha_\mathcal{P} \to \infty$, the disease-free equilibrium $E_0^{(4.1)}$ is globally asymptotically stable if $\mathcal{R}_0^{(4.1)} < 1$ [36, Subsection 3.2 and Table 1], and the endemic equilibrium (EE) $E_*^{(4.1)}$ is globally asymptotically stable if $\mathcal{R}_0^{(4.1)} > 1$ [36, Theorem 3.1]. From (4.1b) and (4.1d),

$$(I_1 + I_2)' = \left(\beta_1 \frac{S_1}{N_1} - \gamma_1 - d_1\right)I_1 + \left(\beta_2 \frac{S_2}{N_2} - \gamma_2 - d_2\right)I_2 \tag{4.5a}$$

$$\leq (\beta_1 - \gamma_1 - d_1)I_1 + (\beta_2 - \gamma_2 - d_2)I_2, \tag{4.5b}$$

yielding the following result. (Recall that here, $\mathcal{P} = \{1, 2\}$.)

Theorem 4.1. *The disease-free equilibrium $E_0^{(4.1)}$ is globally asymptotically stable if $\prod_{p \in \mathcal{P}}(\beta_p - \gamma_p - d_p) \geq 0$ and $\sum_{p \in \mathcal{P}}(\beta_p - \gamma_p - d_p) \leq 0$.*

Are conditions in Theorem 4.1 stronger than $\mathcal{R}_0^{(4.1)} < 1$? The answer is positive, as established in the following result.

Theorem 4.2. *The basic reproduction number $\mathcal{R}_0^{(4.1)} < 1$ whenever conditions of Theorem 4.1 hold.*

Proof.   One may compute

$$FV^{-1} = \frac{1}{v_{11}v_{22} - m_{12}m_{21}} \begin{pmatrix} \beta_1 v_{22} & \beta_1 m_{12} \\ \beta_2 m_{21} & \beta_2 v_{11} \end{pmatrix}.$$

By the Gershgorin circle theorem,

$$\left| \lambda_1 - \frac{\beta_1 v_{22}}{v_{11}v_{22} - m_{12}m_{21}} \right| < \frac{\beta_2 m_{21}}{v_{11}v_{22} - m_{12}m_{21}}$$
$$\Rightarrow |\lambda_1| < \frac{\beta_1 v_{22} + \beta_2 m_{21}}{v_{11}v_{22} - m_{12}m_{21}},$$

and

$$\left| \lambda_2 - \frac{\beta_2 v_{11}}{v_{11}v_{22} - m_{12}m_{21}} \right| < \frac{\beta_1 m_{12}}{v_{11}v_{22} - m_{12}m_{21}}$$
$$\Rightarrow |\lambda_2| < \frac{\beta_2 v_{11} + \beta_1 m_{12}}{v_{11}v_{22} - m_{12}m_{21}},$$

where λ_1, λ_2 are the eigenvalues of FV^{-1}.

Since

$$v_{11} := d_1 + \gamma_1 + m_{21}, \ \beta_1 < d_1 + \gamma_1,$$
$$v_{22} := d_2 + \gamma_2 + m_{12}, \ \beta_2 < d_2 + \gamma_2,$$

then

$$\beta_1 v_{22} + \beta_2 m_{21} < (d_1 + \gamma_1)(d_2 + \gamma_2 + m_{12}) + (d_2 + \gamma_2)m_{21}$$
$$= v_{11}v_{22} - m_{12}m_{21}$$
$$\Rightarrow |\lambda_1| < 1.$$

Similarly, one finds that $|\lambda_2| < 1$. Therefore, $\mathcal{R}_0^{(4.1)} = \rho(FV^{-1}) < 1$. □

Next, we discuss for system (4.1) the questions of uniform strong persistence and existence of endemic equilibrium. A system being persistent means that no components within the system approach zero. For example, there is no extinction for any populations that make up a biological system. The definition of *uniform strong persistence* below comes from [1].

Definition 4.1. *A system of differential equations, $dX/dt = F(X,t)$, $X(0) = X_0$, where $X(t) = (x_1(t), x_2(t), \ldots, x_n(t))^T$, is said to be uniformly strongly persistent if for any positive initial condition $X_0 > 0$, there exists $\delta > 0$ such that $\liminf_{t\to\infty} x_i(t) > \delta$ for $i = 1, 2, \ldots, n$.*

An infectious disease is endemic if system (4.1) is uniformly strongly persistent. In other words, the numbers of susceptible and infectious individuals remain above a certain positive level. The following lemma is summarized from [50].

Lemma 4.1. *Let $\phi_t : X \to X$ be a semiflow and $X_0 \subset X$ an open set. Define $\partial X_0 = X \setminus X_0$, and $M_\partial = \{x \in \partial X_0 : \phi_t x \in \partial X_0, \ t \geq 0\}$. Assume that*

(C_1) $\phi_t X_0 \subset X_0$ and ϕ_t has a global attractor A;

(C_2) there exists a finite sequence $\mathcal{M} = \{M_1, \cdots, M_k\}$ of disjoint, compact, and isolated invariant sets in ∂X_0 such that

    (a) $\Omega(M_\partial) := \cup_{x \in M_\partial} \omega(x) \subset \cup_{i=1}^k M_i$;

    (b) no subset of $\mathcal{M}$ forms a cycle in ∂X_0;

    (c) M_i is isolated in X;

    (d) $W^s(M_i) \cap X_0 = \emptyset$, where $W^s(M_i) = \{x \in X_0 : \omega(x) \subset M_i\}$, for each $1 \le i \le k$.

Then ϕ_t is uniformly strongly persistent with respect to $(X_0, \partial X_0)$, i.e., there exists $\delta > 0$, such that $\liminf_{t \to +\infty} d(\phi_t x, \partial X_0) \ge \delta$ for $x \in X_0$.

Using Definition 4.1 and Lemma 4.1, we obtain the following result.

Theorem 4.3. *System* (4.1) *with initial conditions* (4.2) *is uniformly strongly persistent and admits an endemic equilibrium (EE)* $E_*^{(4.1)} := (S_1^*, I_1^*, S_2^*, I_2^*)$ *if* $\mathcal{R}_0^{(4.1)} > 1$.

Proof. Choose $X = \mathbb{R}_+^4$, $X_0 = \{(S_1, I_1, S_2, I_2) \in X, I_1 + I_2 > 0\}$, and $\partial X_0 = X \setminus X_0 = \{(S_1, I_1, S_2, I_2) \in X, I_1 = I_2 = 0\}$. Let ϕ_t be the semiflow induced by the non-negative solutions of (4.1) and $M_\partial \in \partial X_0$ for (4.1). Lemma 3.1 implies $\phi_t X_0 \subset X_0$ when $\mathcal{R}_0^{(4.1)} > 1$. Section 3 has established that ϕ_t is ultimately bounded in X_0. As a consequence, there always exists a global attractor for ϕ_t. It is obvious that $E_0^{(4.1)}$ is the unique boundary equilibrium on ∂X_0, which implies $E_0^{(4.1)}$ is the unique ω-limit set on ∂X_0. Therefore, we have $\mathcal{M} = \{M_1\}$ and $M_1 = E_0^{(4.1)}$. Then $\cup_{x \in M_\partial} \omega(x) = M_1 \in \mathcal{M}$, no subset of $\mathcal{M}$ forms a cycle in ∂X_0 and M_1 is isolated. The instability of $E_0^{(4.1)}$ when $\mathcal{R}_0^{(4.1)} > 1$ leads to the satisfaction of condition (d) in Lemma 4.1. The existence of an endemic equilibrium in $X = \mathbb{R}_+^4$ follows from [50, Theorem 1.3.7]. The proof is complete. $\square$

We now turn to the concept of uniform weak persistence.

Definition 4.2. The disease in system (4.1) is said to be *uniformly weakly persistent* if there exists some $\delta > 0$ such that

$$\limsup_{t \to \infty} \left(\frac{I_1(t)}{N_1(t)} + \frac{I_2(t)}{N_2(t)} \right) \ge \epsilon, \tag{4.6}$$

for all non-negative solutions of (4.1) with initial conditions (4.2).

Obviously, uniform strong persistence implies uniform weak persistence.

Theorem 4.4. *System* (4.1) *with initial conditions* (4.2) *is uniformly weakly persistent if* $\mathcal{R}_0^{(4.1)} > 1$.

5. Mobility between patches with large and small populations

In this section, we consider the interconnection through travel of two patches with very different population sizes, large and small. When the population size is small, it is assumed that each infectious individual may potentially meet almost all susceptible individuals in the population, so that the incidence function for disease transmission is of mass action type. In the case of a large population, each infectious

individual can only contact a proportion of the susceptible individuals, leading to the use of a proportional incidence function. Without loss of generality, we assume that patch 1 is populous and patch 2 is underpopulated. Then system (2.2) becomes the following system of ODE.

$$S_1' = b_1 + \gamma_1 I_1 - \beta_1 \frac{S_1 I_1}{N_1} - d_1 S_1 - m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)S_1 + m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)S_2,$$
$$(5.1a)$$

$$I_1' = \beta_1 \frac{S_1 I_1}{N_1} - (\gamma_1 + d_1)I_1 - m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)I_1 + m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)I_2,$$
$$(5.1b)$$

$$S_2' = b_2 + \gamma_2 I_2 - \beta_2 S_2 I_2 - d_2 S_2 - m_{12}\Big(1 - v\frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)S_2 + m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)S_1,$$
$$(5.1c)$$

$$I_2' = \beta_2 S_2 I_2 - (\gamma_2 + d_2)I_2 - m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)I_2 + m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)I_1.$$
$$(5.1d)$$

5.1. DFE and basic reproduction number

System (5.1) has the same disease-free equilibrium as system (4.1). However, the basic reproduction number $\mathcal{R}_0^{(5.1)}$ for (5.1) is not identical to $\mathcal{R}_0^{(4.1)}$ for (4.1). Moreover, system (5.1) does not possess any boundary equilibria, and might have an endemic equilibrium $E_*^{(5.1)}$. The condition $\mathcal{R}_0^{(5.1)} > 1$ is sharp for disease persistence, and a disease outbreak with low prevalence may eventually dies out if $\mathcal{R}_0^{(5.1)} < 1$ (see [42, Theorem 2]). Again, $\mathcal{R}_0^{(5.1)}$ can be obtained by the next generation matrix method [42] as well, and it presents as follows after a straightforward calculation:

$$\mathcal{R}_0^{(5.1)} = \frac{\beta_1 v_{22} + \beta_2 v_{11} S_2 + \sqrt{(\beta_2 v_{11} S_2 - \beta_1 v_{22})^2 + 4\beta_1 \beta_2 v_{12} v_{21} S_2}}{2(v_{11}v_{22} - v_{12}v_{21})},$$

where S_2 is the number of susceptibles in patch 2 at disease-free equilibrium $E_0^{(5.1)}$, and v_{ij} $(i, j = 1, 2)$ are listed in Section 4.

The parameters α_1, α_2, σ_1 and σ_2 do not appear in $\mathcal{R}_0^{(5.1)}$. This implies that media coverage cannot determine disease elimination and persistence although it modifies individuals' travel rates.

Lemma 5.1. *For any non-negative solutions $(S_1(t), I_1(t), S_2(t), I_2(t))$ of system (5.1), there exists some constant δ such that $\limsup_{t \to \infty} N(t) \le \delta$ and $N(t) \le \max\{\delta, N(0)\}$ for all $t \ge 0$, where $\delta \ge (b_1 + b_2)/\min\{d_1, d_2\}$.*

The proof of Lemma 5.1 is straightforward and is omitted.

5.2. Uniform persistence and existence of endemic equilibrium

Theorem 5.1. *The disease in system (5.1) is uniformly weakly persistent if $\mathcal{R}_0^{(5.1)} > 1$.*

Proof. We introduce the notation $\mathbf{S} := (S_1, S_2)^T$, $\mathbf{I} := (I_1, I_2)^T$, $\|\mathbf{S}(t)\| := S_1(t) + S_2(t)$ and $\|\mathbf{I}(t)\| := I_1(t) + I_2(t)$. The proof is completed in three steps. Suppose the statement is false.

Step 1. There exists some $c > 0$ such that $\limsup_{t\to\infty} \|\mathbf{I}(t)\| \leq c$.

Choose an arbitrarily small $\epsilon > 0$. By Definition 4.2, there exists a solution of (5.1) such that $I_1(0) + I_2(0) > 0$ and $\mathbf{S}(0) \gg \mathbf{0}$, which means $\mathbf{S}(0) \in (0, \infty)^2$, i.e., the vector $\mathbf{S}(0)$ has both coordinates positive, but $\limsup_{t\to\infty}(\frac{I_1(t)}{N_1(t)} + \frac{I_2(t)}{N_2(t)}) < \epsilon$. Then $\mathbf{S}(t) \gg \mathbf{0}$ for all $t \geq 0$ and $\|\mathbf{I}(t)\| > 0$ for all $t > 0$. Shifting forward in time, we may assume

$$\frac{I_1(t)}{N_1(t)} + \frac{I_2(t)}{N_2(t)} < \epsilon, \quad \forall t \geq 0. \tag{5.2}$$

By Lemma 5.1, there exists some positive constant δ such that $\limsup_{t\to\infty} N(t) \leq \delta$. Shifting forward in time and increasing δ, we may assume $N(t) \leq \delta$ for all $t \geq 0$. Then $\|\mathbf{I}(t)\| < \epsilon\delta$ for all $t \geq 0$, which implies $\limsup_{t\to\infty} \|\mathbf{I}(t)\| \leq \epsilon\delta =: c$.

Step 2. There exists some $\delta_1 > 0$ such that $\liminf_{t\to\infty} S_i(t) \geq \delta_1, i = 1, 2$.

To prove the statement above, the following differential inequality derived from (5.1a), (5.1c) and (5.2) is required.

$$\begin{aligned}\|\mathbf{S}(t)\|' &\geq (b_1 + b_2) - (\beta_1\epsilon + d_1)S_1 - (\beta_2\epsilon\delta + d_2)S_2 \\ &\geq (b_1 + b_2) - \max\{\beta_1\epsilon + d_1, \beta_2\epsilon\delta + d_2\}\|\mathbf{S}(t)\|.\end{aligned} \tag{5.3}$$

Then there exists some $t_1 > 0$ such that $\|\mathbf{S}(t)\| > \delta_2$ for all $t \geq t_1$, where

$$0 < \delta_2 < \frac{b_1 + b_2}{\max\{\beta_1\epsilon + d_1, \beta_2\epsilon\delta + d_2\}}.$$

Following the fact that there exists $c_1, c_2 > 0$ and if $\limsup_{t\to\infty} \|\mathbf{I}(t)\| \leq c_1(c_1 \geq c)$, then $\liminf_{t\to\infty} S_i(t)/\|\mathbf{S}(t)\| \geq c_2, i = 1, 2$ for all non-negative solutions of system (5.1) with $(S_1(0) + S_2(0)) > 0$, the statement is then proved, where $\delta_1 \leq c_2\delta_2$.

Step 3. Find the contradiction.

Shifting forward in time and from discussions above, we may assume that $S_i(t) \geq \bar{\delta}_1(\bar{\delta}_1 \leq \delta_1)$, $I_i(t) \leq \bar{c}(\bar{c} \geq c)$ and $I_i(t)/N_i(t) \leq \bar{\epsilon}(\bar{\epsilon} < \epsilon), i = 1, 2$ for all $t \geq 0$, where $\bar{\delta}, \bar{c}, \bar{\epsilon} > 0$. Then $S_i(t)/N_i(t) \geq 1 - \bar{\epsilon}$. From (5.1b) and (5.1d), we deduce that

$$I_1' \geq \beta_1(1 - \bar{\epsilon})I_1 - (\gamma_1 + d_1)I_1 + m_{12}\left(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\right)I_2 - m_{21}\left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\right)I_1, \tag{5.4a}$$

$$I_2' \geq \beta_2\bar{\delta}_1 I_2 - (\gamma_2 + d_2)I_2 + m_{21}\left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\right)I_1 - m_{12}\left(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\right)I_2. \tag{5.4b}$$

By Step 2 and Lemma 5.1, and again shifting forward in time, we may assume $\bar{\delta}_1 \leq S_i(t) \leq N(t) \leq \max\{\delta, N(0)\}$ for all $t \geq 0, i = 1, 2$. Define

$$\hat{m}_{21}(I_2) := m_{21}\left(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\right),$$

$$\hat{m}_{12}(I_1) := m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big).$$

Since the functions $\hat{m}_{21}(I_2)$ and $\hat{m}_{12}(I_1)$ are continuous, then $\hat{m}_{21}(I_2) \to \hat{m}_{21}(0) = m_{21}$ and $\hat{m}_{12}(I_1) \to \hat{m}_{12}(0) = m_{12}$ as $\|\mathbf{I}(t)\| \to 0$. Therefore, for any $\eta \in (0, 1)$, we may choose $\epsilon > 0$ small enough such that for all $t \geq 0$ (bear in mind that $\|\mathbf{I}(t)\| < \epsilon\delta =: c$ for all $t \geq 0$),

$$(1 - \eta)\hat{m}_{21}(0) \leq \hat{m}_{21}(I_2) \leq (1 + \eta)\hat{m}_{21}(0),$$
$$(1 - \eta)\hat{m}_{12}(0) \leq \hat{m}_{12}(I_1) \leq (1 + \eta)\hat{m}_{12}(0).$$

From (5.4) we have $\mathbf{I}' \geq A_\epsilon \mathbf{I}$ where the linear operator A_ϵ is associated with an irreducible quasi-positive matrix. Moreover, $A_\epsilon \to A$ as $\epsilon \to 0$, and A is an operator in the form

$$A\begin{pmatrix} I_1 \\ I_2 \end{pmatrix} = A_1 \begin{pmatrix} I_1 \\ I_2 \end{pmatrix} - A_2 \begin{pmatrix} I_1 \\ I_2 \end{pmatrix},$$

where the positive operator A_1 is represented by a diagonal matrix with all diagonal entries being the capita infection rate $\beta_i > 0$, and the operator A_2 is represented by a matrix with $-\hat{m}_{ij}(0)$ of off-diagonal entries and $\gamma_i + d_i + \hat{m}_{ij}(0)$ of diagonal entries for $i, j = 1, 2$ and $i \neq j$.

Let $s(A)$ denote the spectral bound of a linear bounded operator A, i.e., the largest real part of its eigenvalues. Since $\mathcal{R}_0^{(5.1)} > 1$, then $s(A) > 0$. The eigenvalues depend continuously on the operator or the corresponding representing matrix. Therefore, $s(A_\epsilon) > 0$ for sufficient small $\epsilon > 0$. By the Perron-Frobenius theory (see [39, A] or [41, section A.8]), we may choose a vector $\mathbf{v} \gg 0$ such that $A_\epsilon^* \mathbf{v} = s(A_\epsilon)\mathbf{v}$ where * denotes the dual operator. Then $\langle \mathbf{I}(t), \mathbf{v} \rangle' \geq \langle A_\epsilon \mathbf{I}(t), \mathbf{v} \rangle = \langle \mathbf{I}(t), A_\epsilon^* \mathbf{v} \rangle = s(A_\epsilon)\langle \mathbf{I}(t), \mathbf{v} \rangle$. Since $\mathbf{v} \gg \mathbf{0}$, $\langle \mathbf{I}(t), \mathbf{v} \rangle > 0$, first for $t = 0$, and then for all $t \geq 0$. Since $s(A_\epsilon) > 0$, then $\langle \mathbf{I}(t), \mathbf{v} \rangle \to \infty$ as $t \to \infty$. Without loss of generality, we choose $\mathbf{v} = \{v_1, v_2\}^T$ such that $v_1, v_2 \leq 1$, which implies $N(t) \geq \langle \mathbf{I}(t), \mathbf{v} \rangle \to \infty$ as $t \to \infty$. This contradicts the result in Lemma 5.1. The proof is then complete. $\square$

We next study the uniform strong disease persistence (Definition 4.1) and the existence of an endemic equilibrium of system (5.1).

Lemma 5.2. *In system* (5.1), $S_i(t) > 0$ *(i = 1, 2) for all $t > 0$, and there exist constants $\hat{\delta}_i > 0$ such that* $\liminf_{t \to \infty} S_i(t) \geq \hat{\delta}_i$ *for all non-negative solutions of* (5.1).

Proof. By (5.1a), (5.1c) and Lemma 5.1,

$$S_1' \geq b_1 - \Big[\beta_1 + d_1 + m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)\Big]S_1,$$
$$S_2' \geq b_2 - \Big[\max\{\delta, N(0)\}\beta_2 + d_2 + m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)\Big]S_2.$$

By the fluctuation method ([25], [41, Proposition A.22]),

$$\liminf_{t \to \infty} S_1(t) \geq \frac{b_1}{\beta_1 + d_1 + m_{21} \limsup_{t \to \infty} \Big(1 - \frac{\sigma_2 I_2(t)}{\alpha_2 + \sigma_2 I_2(t)}\Big)},$$

$$\liminf_{t \to \infty} S_2(t) \geq \frac{b_2}{\max\{\delta, N(0)\}\beta_2 + d_2 + m_{12} \limsup_{t \to \infty} \Big(1 - \frac{\sigma_1 I_1(t)}{\alpha_1 + \sigma_1 I_1(t)}\Big)}.$$

Lemma 5.1 indicates that $\limsup_{t\to\infty} \frac{\sigma_i I_i(t)}{\alpha_i + \sigma_i I_i(t)}$ is a positive constant, so there exist constants $\xi_i > 0$, independent of $I_i(t)$, such that $\liminf_{t\to\infty} S_i(t) \geq \frac{b_i}{\xi_i} =: \hat{\delta}_i$ ($i = 1, 2$). This completes the proof. $\square$

Theorem 5.2. *System* (5.1) *with initial conditions* (4.2) *is uniformly strongly persistent, and there exists an endemic equilibrium* $E_*^{(5.1)} := (S_1^*, I_1^*, S_2^*, I_2^*)$ *of* (5.1) *in* $(0, \infty)^4$ *if* $\mathcal{R}_0^{(5.1)} > 1$.

Proof. Let the state space X be as in Theorem 4.3. Define $\rho : X \to \mathbb{R}_+$ by $\rho(S_1, I_1, S_2, , I_2) = \sum \frac{I_i}{N_i}$ where $N_i = S_i + I_i$, and $\tilde{\rho} : X \to \mathbb{R}_+$ by $\tilde{\rho}(S_1, I_1, S_2, , I_2) = I_i, i = 1, 2$. By Lemma 5.1 and Lemma 5.2, the compactness condition of [41, Theorem A.34] is satisfied. Notice that every total orbit $\omega : \mathbb{R} \to X$ of ϕ_t, where ϕ_t is the semiflow induced by the non-negative solutions of (5.1), is associated with a solution of (5.1) that is defined for all $t \in \mathbb{R}$ and takes value in X. By the fact that the matrix below is irreducible,

$$\begin{pmatrix} 0 & m_{12}\left(1 - \dfrac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\right) \\ m_{21}\left(1 - \dfrac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\right) & 0 \end{pmatrix},$$

it follows that $\tilde{\rho}(\omega(0)) > 0$ whenever $\rho(\omega(t)) > 0$ for all $t \in \mathbb{R}$. The claim for $\liminf_{t\to\infty} I_i(t) \geq \bar{\delta}$, a given constant, $i = 1, 2$ now follows from [41, Theorem A.34] which requires $\mathcal{R}_0^{(5.1)} > 1$. For S_1 and S_2, the statement has been shown in Lemma 5.2. The existence of an endemic equilibrium in $(0, \infty)^4$ follows from [50, Theorem 1.3.7]. The proof is then complete. $\square$

Note that $\mathcal{R}_0^{(4.1)} < \mathcal{R}_0^{(5.1)}$. The greater the basic reproduction number is, the harder the epidemic is to control. This implies that the form of incidence function does play an important role in determining disease persistence and extinction.

6. Numerical results

To complement the mathematical analysis performed in previous sections, we now carry out numerical simulations of system (4.1). Choose parameters characteristic of influenza, as detailed in Table 1. The initial total population in each patch is assumed to be $N_1(0) = 80,000$ and $N_2(0) = 100,000$ with initial infectives $I_1(0) = 8000$ and $I_2(0) = 6000$.

Table 1. Parameter values used in simulations

b_1	β_1	m_{12}	d_1	γ_1	σ_1	α_1
3	0.08 or 0.6	0.008 or 0.02	$1/(75 \times 365)$	$1/10$	0.2	200
b_2	β_2	m_{21}	d_2	γ_2	σ_2	α_2
1.2	0.06 or 0.4	0.012 or 0.05	$1/(70 \times 365)$	$1/12$	0.3	250

The effects of media coverage on the prevalence of an epidemic and on the time the epidemic takes to go extinct are illustrated in Fig. 3. The corresponding system, with media alert present in patch 1 and absent in patch 2, can be derived by setting $\sigma_1 = 0$ and $\sigma_2 \neq 0$ in system (4.1). It appears in Fig. 3(a) that the presence of media coverage in patch 2 leads to a respective reduction and increase in the time to extinction of disease in patch 1 and patch 2. And meanwhile, it is shown in Fig. 3(b) that it lowers the magnitude of an epidemic in patch 1 and enhances that in patch 2.
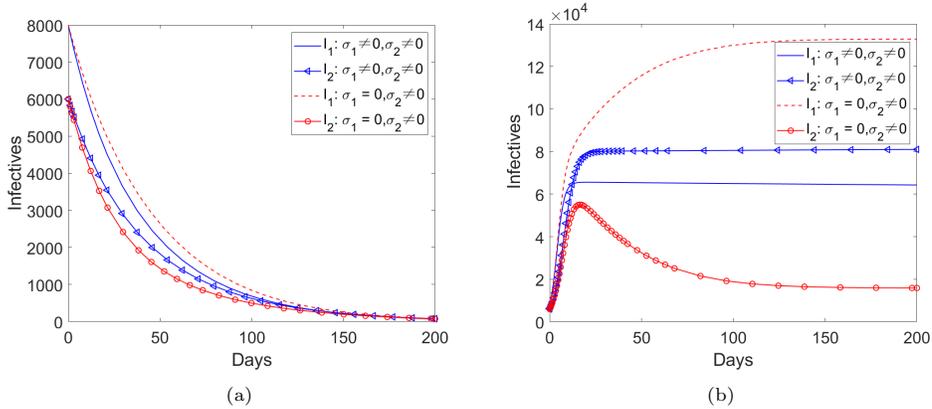

Figure 3. (a) Effect of media coverage on the time to extinction of an existing epidemic when $\mathcal{R}_0^{(4.1)} < 1$. (b) Effect of media coverage on the prevalence of an existing epidemic when $\mathcal{R}_0^{(4.1)} > 1$.
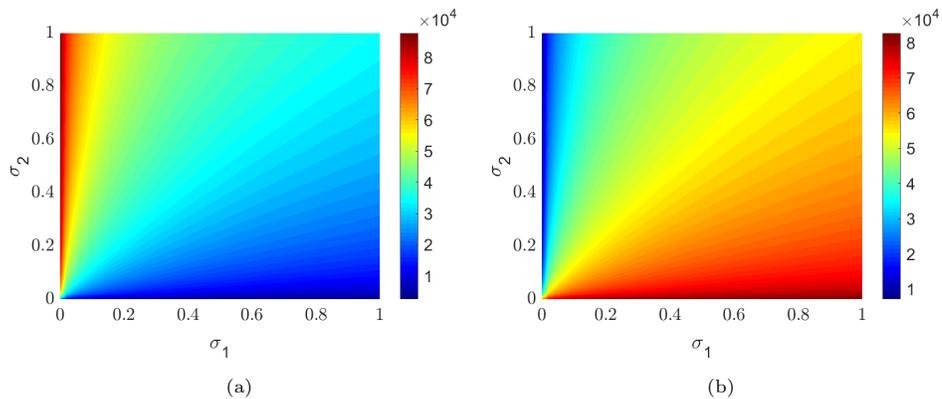
Figure 4. Equilibrium values of (a) I_1^* and (b) I_2^*, as σ_1 and σ_2 are varied, with other parameters as in Table 1 such that $\mathcal{R}_0^{(4.1)} > 1$.

In Fig. 4, we show the trend of I_1 and I_2 at the endemic equilibrium as the fraction of known cases σ_1 and σ_2, characterizing the strength of media coverage in patch 2 and patch 1 respectively, are varied in a simulation $\mathcal{R}_0^{(4.1)} > 1$. Recall from the derivation of $\mathcal{R}_0^{(4.1)}$ in Section 4 that $\mathcal{R}_0^{(4.1)}$ is independent of media coverage. For instance, increasing the intensity of media coverage cannot bring $\mathcal{R}_0^{(4.1)}$ down
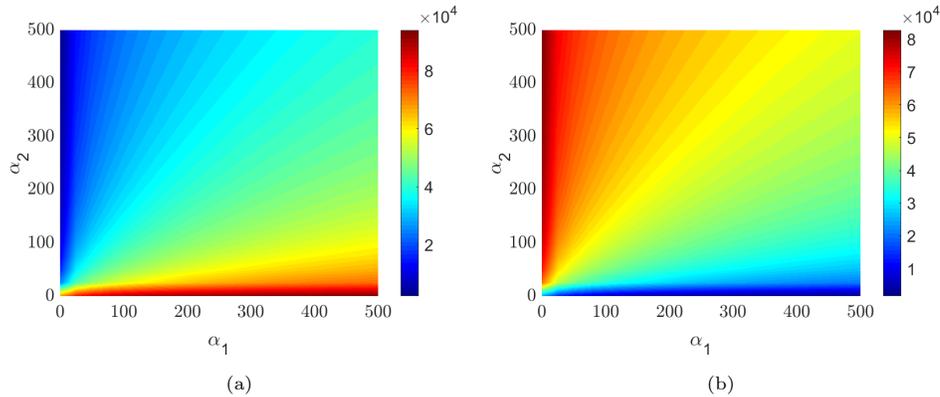
Figure 5. Equilibrium values of (a) I_1^* and (b) I_2^*, as α_1 and α_2 are varied, with other parameters as in Table 1 such that $\mathcal{R}_0^{(4.1)} > 1$.

below unity. It is observed that travel-related media coverage in patch i may respectively exacerbate and reduce the disease burden of patch i and patch j $(i, j = 1, 2$ and $i \neq j)$. Fig. 5 illustrates the endemic equilibrium values for I_1 and I_2 as the insensitivity of response to knowledge of cases covered by media, α_1 and α_2, are varied. And it shows that the insensitivity of travel-related media coverage in patch i may impose positive and negative effects on disease propagation in patch i and patch j, respectively. The results here can be applied to models with border screening.

Fig. 6 presents a sensitivity analysis of the value of $\mathcal{R}_0^{(4.1)}$ to the variation of parameters. Parameters are made to vary in the following ranges: $b_i \in [0.1, 10], \beta_i \in [0.0001, 1], m_{ij} \in [0.00001, 0.1], d_i \in [1/(100 \times 365), 1/(60 \times 365)], \gamma_i \in [1/17, 1/4], \alpha_i \in [0.02, 300]$ and $\sigma_i \in [0.002, 1]$. Sample points are chosen within this range using Latin hypercube sampling. Fig. 6(a) shows the range of values obtained for $\mathcal{R}_0^{(4.1)}$ when 10,000 such sample points are chosen. The red bar shows the median value of $\mathcal{R}_0^{(4.1)}$, the box indicates the interquartile range, while the whiskers show the extent of values not considered to be outlying. Outlying values are not shown here. In Figs. 6(b) and 6(c), the role of individual parameters is investigated. In order to do so, all parameter values are fixed to the values in Table 1, and each of $b_i, \beta_i, m_{ij}, d_i, \gamma_i, \alpha_i$ and σ_i $(i, j = 1, 2, i \neq j)$ is successively made to vary 10,000 times in the ranges indicated above. Obviously, $\mathcal{R}_0^{(4.1)}$ is high, intermediate and low sensitive to parameters β_i, γ_i and m_{ij}, respectively.

Finally, we study in Fig. 7 the effect of population size on the prevalence of an existing epidemic by comparing the disease dynamics numerically between (4.1) and (5.1) when $\mathcal{R}_0^{(4.1)}, \mathcal{R}_0^{(5.1)} > 1$, which is achieved by setting all corresponding parameters, except $\beta_2^{(5.1)} = 3\beta_2^{(4.1)}/N_2(0)$, to be equal in (4.1) and (5.1) with $N_1(0) = 80,000, N_2(0) = 5000, I_1(0) = 8000, I_2(0) = 1000$. It shows that small population incurring frequent contacts of individuals may increase disease prevalence within the patch, but have little effect on that of the destination patch.

(a)

(b)

(c)
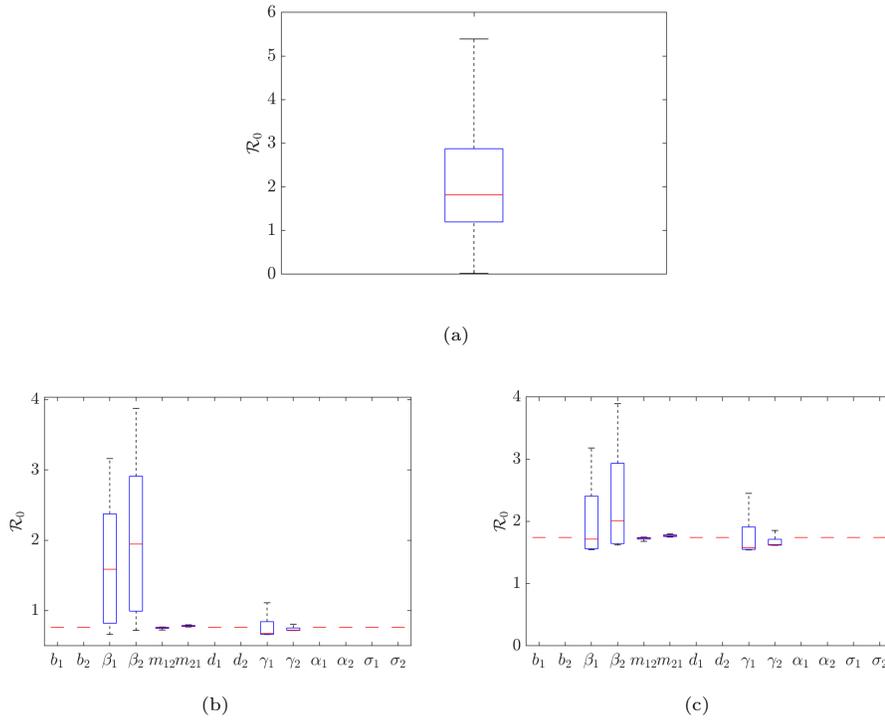
Figure 6. (a) Sensitivity of $\mathcal{R}_0^{(4.1)}$ to variations of parameters, for 10,000 sample points in the parameter region indicated in the text. Sensitivity of $\mathcal{R}_0^{(4.1)}$ to variations of individual parameters. In the absence of variation of any other parameters, (b) $\mathcal{R}_0^{(4.1)} < 1$ and (c) $\mathcal{R}_0^{(4.1)} > 1$.
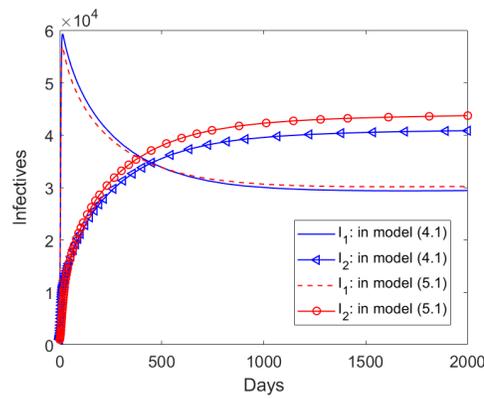
Figure 7. Effect of population size on the prevalence of an existing epidemic when $\mathcal{R}_0^{(4.1)}, \mathcal{R}_0^{(5.1)} > 1$.

7. Discussion

This paper introduces an SIS metapopulation model accounting for media-induced modification (reduction) of travel rates. After brief consideration of local properties of solutions to the general model, the model is specialised to two patches and two scenarios are taken into consideration.

The systems under consideration, (2.2), (4.1) and (5.1), are in essence different from previous models of media effect on disease transmission ([9, 14–16, 21–24, 30–32, 37, 38, 40, 43–45, 47, 48] and references therein). Global stability of system equilibria is still an open problem. Note that neither the standard comparison principle [39, Theorem B.1] in proving the global asymptotic stability of disease-free equilibrium when the basic reproduction number is less than unity, nor the application of LaSalle Invariance Principle by Li and Shuai [29, Theorem 4.1] in proving the uniqueness and global asymptotic stability of endemic equilibrium when the basic reproduction number is greater than unity, are applicable for our model systems. Indeed, the travel rates between patches in our models are variable.

Consider the uncoupled system without travel and denote $\mathcal{R}_0^{(4.1)/i}$ and $\mathcal{R}_0^{(5.1)/i}$, the basic reproduction numbers for patch i ($i = 1, 2$) in (4.1) and (5.1), respectively:

$$\mathcal{R}_0^{(4.1)/i} = \frac{\beta_i}{\gamma_i + d_i}, \quad \mathcal{R}_0^{(5.1)/i} = \frac{\beta_i S_0^{i-1}}{\gamma_i + d_i}.$$

Obviously, $\mathcal{R}_0^{(4.1)/1} = \mathcal{R}_0^{(5.1)/1}$, $\mathcal{R}_0^{(4.1)/2} < \mathcal{R}_0^{(5.1)/2}$. Recall that $\mathcal{R}_0^{(4.1)} < \mathcal{R}_0^{(5.1)}$ and that these quantities do not involve parameters of media coverage. This implies that the differentiation in population size does change the dynamics and potentially accelerate disease transmission. It is then inferred that disease can be persistent in both patches in system (5.1) while it dies down in both patches for system (4.1).

When an epidemic occurs, especially when it is in its early stage, it appears plausible that $S \approx N$. In this case, system (4.1) becomes the following system:

$$S_1' = b_1 - \beta_1 I_1 - d_1 S_1 + \gamma_1 I_1 - m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)S_1 + m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)S_2,$$
$$\text{(7.1a)}$$

$$I_1' = \beta_1 I_1 - \gamma_1 I_1 - d_1 I_1 - m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)I_1 + m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)I_2,$$
$$\text{(7.1b)}$$

$$S_2' = b_2 - \beta_2 I_2 - d_2 S_2 + \gamma_2 I_2 - m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)S_2 + m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)S_1,$$
$$\text{(7.1c)}$$

$$I_2' = \beta_2 I_2 - \gamma_2 I_2 - d_2 I_2 - m_{12}\Big(1 - \frac{\sigma_1 I_1}{\alpha_1 + \sigma_1 I_1}\Big)I_2 + m_{21}\Big(1 - \frac{\sigma_2 I_2}{\alpha_2 + \sigma_2 I_2}\Big)I_1.$$
$$\text{(7.1d)}$$

In this case, analysis of (7.1) can start by considering (7.1b) and (7.1d): the (I_1, I_2) subsystem is independent of S_1 and S_2 as given by (7.1a) and (7.1c). The latter are subordinated to (I_1, I_2) and can then be determined afterwards. It is found that (7.1b) and (7.1d) only have a disease-free equilibrium, no endemic equilibrium or boundary equilibrium. The application of Bendixson-Dulac Theorem [33, Theorem 2, pp. 265], by defining a Dulac function $D := 1/(I_1 I_2)$, shows that the (I_1, I_2)

subsystem has no periodic solutions, homoclinic loops or oriented phase polygons in the first quadrant. Numerical simulations conducted (not shown) on system (7.1) counter-intuitively illustrate that, under the condition $(\beta_1 - \gamma_1 - d_1)(\beta_2 - \gamma_2 - d_2) < 0$, trajectories of (7.1b) and (7.1d) always go to infinity rather than the disease-free equilibrium. Note that the nonnegativity of $S_1(t)$ and $S_2(t)$ in (7.1a) and (7.1c) cannot be guaranteed with $S_1(0), S_2(0) \geq 0$, which may result in the unboundedness of $I_1(t)$ and $I_2(t)$. System (7.1) does not make any sense from biological perspective, which means assuming $S \approx N$ is not always feasible in model formulation.

The models presented and studied here are toy models. The first limitation is that they are based on a simple SIS model, whereas a lot of diseases follow an SLIRS-type progression, where L and R stand for latently infected and recovered individuals, respectively. The second limitation is that intervention strategies such as vaccination and treatment are not included in the model. Moreover, the waning effect of media alert at individual level is not taken into account. Indeed, one may become less and less concerned about the epidemic after initially experiencing high anxiety of getting infected with the disease [26]. Work is in progress to formulate a model addressing such limitations.

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