

## Revisiting a two-patch SIS model with infection during transport

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We incorporate parameter heterogeneity in a two-patch susceptible-infectious-susceptible (SIS) epidemic model with infection during transport and prove that the disease-free and endemic equilibria are globally asymptotically stable when the basic reproduction number  $\mathcal{R}_0 < 1$  and  $\mathcal{R}_0 > 1$ , respectively. We find that infection during transport increases the possibility that the disease persists in both patches and amplifies prevalence when disease is present. We then study the effect of a perfect unilateral exit screening programme. Finally, we compare numerically the effects of using different incidence functions for infection within and while travelling between patches, and find that using mass action incidence to model infection during transport has the effect of maintaining disease prevalence at a higher level compared with when standard incidence is used.

*Keywords:* SIS patch model; infection during transport; basic reproduction number; global asymptotic stability.

### 1. Introduction

Infection with respiratory diseases (tuberculosis (TB), influenza, SARS, common cold, etc.) is usually caused by direct contact, large droplets or airborne transmission. Contact transmission involves direct body-to-body or indirect contacts with a contaminated intermediate object (e.g. a door knob or elevator button during SARS). Large droplets are generated when an infectious individual sneezes, coughs and talks. These droplets are propelled short distances and deposited on a susceptible host's conjunctiva or mucosa. Airborne transmission involves the dissemination of microorganism that can remain in the air for indefinite periods (Tang *et al.*, 2009) and be inhaled into the bronchioles of the recipient's respiratory tract (Desenclos, 2008). See, e.g. Brankston *et al.* (2007) for a review of the mechanisms of transmission of influenza A or Morrison *et al.* (2008) for TB.

The role of prolonged proximity in the spread of infectious pathogens has been known for a long time. For instance, nosocomial (hospital acquired) infections have long plagued hospitals worldwide, both for droplet transmitted and airborne diseases (Eames *et al.*, 2009). Because transportation usually puts individuals in close proximity for extended periods of time, it is likely that the probability of transmission during transport increases, in particular as a function of the duration of transport, so that long distance travel would be transmission prone.

There are many documented cases of disease transmission occurring during transport. WHO confirmed that seven healthy people on a 42-h train journey from Ho Chi Minh City to Hanoi in Vietnam caught Tamiflu-resistant A/H1N1 influenza (Le *et al.*, 2010). For more instances of influenza infection during train transportation, see Furuya (2007). In January 1996, a 22-year-old man was diagnosed with smear- and culture-positive TB infection after he had travelled on 2 US passenger trains (29.1 h) and a bus (5.5 h) over 2 days; of 240 passengers and crew members who underwent a tuberculin skin test (TST), 4 (2%) had a documented TST conversion and 11 (5%) had a single positive TST result (Moore *et al.*, 1999). Propagation is also known to have occurred on-board aircrafts (Moser *et al.*, 1979; Centers for Disease Control and Prevention (CDC), 1995; Olsen *et al.*, 2003; Baker *et al.*, 2010), although some authors claim that these remain rare occurrences because of the nature of ventilation systems on-board airplanes (Leder & Newman, 2005; Byrne, 2007). See in particular the extensive review of Mangili & Gendreau (2005) for details about in-flight transmission. Contamination within cars has also been examined (Knibbs *et al.*, 2012).

To summarize, infection can occur not only within patches (where ‘patch’ refers to a geographical location) but also during transportation between patches. It could therefore be important to take the latter component into consideration. In practice, however, the precise impact of infection during transport is not well understood. This is becoming crucial in the world of today, where travel has become so common. Over three billion passengers travel by air annually (Khan *et al.*, 2009). Overall, it is estimated that humans travelled 23 billion kilometres in 2000 and that this will grow to an annual 105 billion kilometres by 2050 (Schafer & Victor, 2000).

Modelling can contribute to the understanding of the potential consequences of infection during transport on the global spread and burden of an infectious pathogen. However, despite its importance, this has not yet emerged as a very active research area. Some work concerning transmission in enclosed spaces, as for instance Noakes *et al.* (2006), can be adapted to the more specific context of transportation. Most of the work carried out so far specifically about transport and infection has concerned the actual transmission during transport and uses probabilistic or statistical models; see, e.g. Andrews *et al.* (2013) for a model of TB transmission in public transportation in South Africa, Chen *et al.* (2011) for transmission of TB onboard trains and Beggs *et al.* (2003) for a review of some (statistical) models used in the context. Other models are very detailed computational fluid dynamics models that focus on the circulation of air within aircrafts; see, e.g. Gupta *et al.* (2011).

However, there have been few papers considering the effect of infection during dispersal on the global spread of infectious pathogens. Some authors have considered the impact of arrival into a location of individuals already infected: Brauer & van den Driessche (2001) proposed a disease evolution model in a single patch with immigration of infectives and Guo and coauthors have studied problems related to the inflow of individuals infected with TB; see, e.g. Guo & Li (2011, 2012) and Guo & Wu (2011). The previous models consider infection of some migrants as a *fait accompli* and do not postulate about the origin of this infection (whether it is acquired prior to or during travel is irrelevant there). These models pose interesting mathematical problems because the inflow of infectives precludes the existence of a disease-free equilibrium (DFE) and thus the definition of a basic reproduction number in the classical way.

One way to overcome the difficulty linked to the non-existence of a DFE is to consider models set in a metapopulation framework, which allows to describe movement of individuals in any epidemiological status while, usually, retaining the existence of a DFE and therefore, of a basic reproduction number defined the classical way. A few authors have considered the problem in the context of differential equations with a discrete delay, which allow to set a precise travel time between locations; see, e.g. Knipfl *et al.* (2013), Liu *et al.* (2008) and Nakata (2011). In an ordinary differential equations

setting, Cui *et al.* (2006) and Takeuchi *et al.* (2007) formulated susceptible-infectious-susceptible (SIS) patch models with infection during transport and studied the local and global dynamics of those models. However, they assumed that all corresponding parameters in both patches are exactly the same, which results in the number of susceptible individuals at DFE, the number of susceptible and infective individuals at boundary equilibria and endemic equilibrium being identical in two patches regardless of initial conditions. If both susceptible and infective individuals travel in two patches, the population in two patches converges to the same equilibrium in Cui *et al.* (2006) and Takeuchi *et al.* (2007). This implies that their SIS patch model is in some sense equivalent to a model for a single population.

For the consideration of more reality, we allow corresponding parameters in the two patches to differ and revisit the model of Cui *et al.* (2006). We also perform some computational analysis of the problem.

## 2. Model formulation

We make the following assumptions regarding the model.

- The total population in each location is divided into two compartments according to the epidemiological status of hosts: susceptibles,  $S$ , and infectives,  $I$ .
- The population in the two patches and during transport is homogeneously mixing.
- Disease transmission within patch  $i$  is of standard incidence type, i.e. the number of infectives produced by random contact between  $S_i$  susceptible and  $I_i$  infective is given by  $\beta_i S_i I_i / N_i$  with  $N_i = S_i + I_i$ , where  $\beta_i$  is the transmission coefficient in patch  $i$ , representing the number of infecting contacts per infective per unit time.
- $m_i S_i$  susceptibles and  $m_i I_i$  infectives travel from patch  $i$  to patch  $j$  per unit time using a specific type of vehicle (e.g. long distance bus, train or aircraft). Each vehicle approximately has the same number of seats; there are  $n$  vehicles transporting passengers from one patch to the other per unit time; therefore, each vehicle carries approximately  $m_i S_i / n$  susceptibles and  $m_i I_i / n$  infectives. We also assume the vehicle has relatively good ventilation and passengers onboard do not interact much with each other. Then the contacts in each vehicle can be described using standard incidence (if there were more interactions, one could use mass action contacts as is done in Section 7). Thus, the rate at which new infections occur during transport is given by

$$\alpha_i \frac{m_i I_i}{n} \cdot \frac{m_i S_i}{n} \cdot \frac{1}{(m_i S_i / n) + (m_i I_i / n)} = \frac{\alpha_i m_i S_i I_i}{n N_i},$$

where  $0 \leq \alpha_i \leq 1$  is the proportion of contacts between susceptible and infectious individuals that lead to a new infection. Therefore, the total infection ( $n$  vehicles) per unit time during transport from patch  $i$  to patch  $j$  is

$$n \cdot \frac{\alpha_i m_i S_i I_i}{n N_i} = \frac{\alpha_i m_i S_i I_i}{N_i}.$$

(When mass action contacts are assumed,  $\alpha_i$  is not dimensionless, it has units per successful contact.)

- All newly recruited individuals in patch  $i$  are susceptible with constant recruitment rate  $b_i$ . Individuals die at a natural rate  $d_i$  and there is no extra disease-caused mortality.

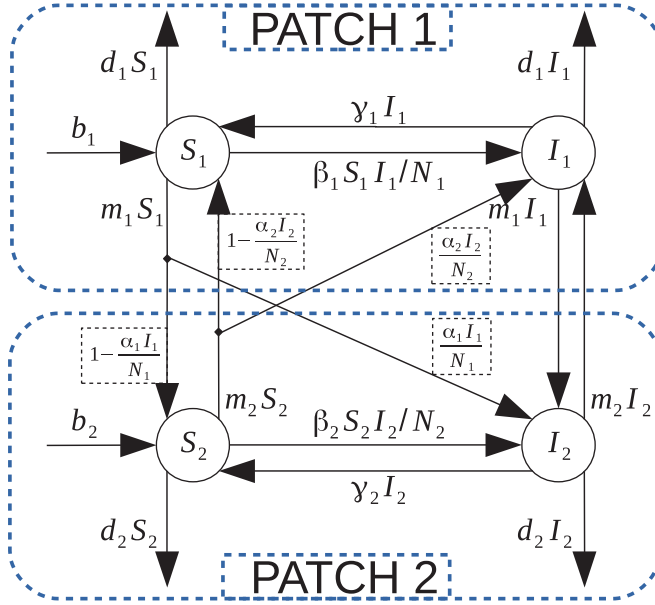


FIG. 1. Flow chart of system (2.1).

- Infectives in patch  $i$  recover at a constant rate  $\gamma_i$  without becoming immune to reinfection. Individuals do not recover during transport (Fig. 1).

The resulting SIS patch model with infection during transport is formulated as follows:

$$\frac{dS_1}{dt} = b_1 - \frac{\beta_1 S_1 I_1}{N_1} + \gamma_1 I_1 - d_1 S_1 - m_1 S_1 + \left(1 - \frac{\alpha_2 I_2}{N_2}\right) m_2 S_2, \quad (2.1a)$$

$$\frac{dI_1}{dt} = \frac{\beta_1 S_1 I_1}{N_1} - d_1 I_1 - \gamma_1 I_1 - m_1 I_1 + \left(1 + \frac{\alpha_2 S_2}{N_2}\right) m_2 I_2, \quad (2.1b)$$

$$\frac{dS_2}{dt} = b_2 - \frac{\beta_2 S_2 I_2}{N_2} + \gamma_2 I_2 - d_2 S_2 + \left(1 - \frac{\alpha_1 I_1}{N_1}\right) m_1 S_1 - m_2 S_2, \quad (2.1c)$$

$$\frac{dI_2}{dt} = \frac{\beta_2 S_2 I_2}{N_2} - d_2 I_2 - \gamma_2 I_2 + \left(1 + \frac{\alpha_1 S_1}{N_1}\right) m_1 I_1 - m_2 I_2, \quad (2.1d)$$

with initial conditions

$$S_i(0) > 0, \quad I_i(0) \geq 0, \quad i = 1, 2, \quad I_1(0) + I_2(0) > 0. \quad (2.2)$$

Next, we present some basic properties of model (2.1).

Define  $f_i(S_i, I_i) = S_i I_i / N_i$ ,  $f_i^{(1)}(S_i, I_i) = S_i / N_i$ ,  $f_i^{(2)}(S_i, I_i) = I_i / N_i$ . For mathematical convenience, assume that

$$f_i^{(1)}(0, 0) = f_i^{(2)}(0, 0) = 0, \quad i = 1, 2. \quad (2.3)$$

Then we have, for  $i = 1, 2$ ,

$$f_i(0, 0) = 0. \quad (2.4)$$

**THEOREM 2.1** Model (2.1) is well posed: (i) (2.1) is globally Lipschitz in  $\mathbb{R}_+^4$  and there exists a unique solution for every initial condition; (ii) all solutions of (2.1) with initial conditions (2.2) stay non-negative for  $t \geq 0$  and (iii) the total population converges to a constant as  $t \rightarrow \infty$ .

*Proof.* (i) *Existence and uniqueness of solutions*

We see that

$$\lim_{(S_i, I_i) \rightarrow (0,0)} f_i(S_i, I_i) = 0,$$

with  $(S_i, I_i) \in \mathbb{R}_+^2$ . Equation (2.4) implies that the right-hand side functions of (2.1) are continuous on  $\mathbb{R}_+^4$ . Straightforward computation using (2.3) shows they are globally Lipschitz on  $\mathbb{R}_+^4$ . Hence, a solution of (2.1) with any initial condition exists and is unique.

(ii) *Non-negativity*

With non-negative initial conditions (2.2), if for instance  $S_1$  becomes zero at time  $t_1$  before  $S_2, I_1, I_2$  become zero, then from (2.1a),  $dS_1/dt = b_1 + \gamma_1 I_1 + m_2 S_2 - \alpha_2 m_2 S_2 I_2 / N_2 > b_1 + \gamma_1 I_1 > 0$  since  $0 \leq \alpha_2 \leq 1$ . Thus,  $S_1$  is an increasing function of  $t$  at  $t_1$ , and therefore  $S_1$  stays positive if  $S_1(0) > 0$ . The same way, we conclude that  $S_2$  is positive if  $S_2(0) > 0$ . Similarly, if  $I_1$  becomes zero at time  $t_2$  before  $S_1, S_2, I_2$  become zero, then from (2.1b),  $dI_1/dt = \alpha_2 m_2 S_2 I_2 / N_2 + m_2 I_2 > 0$  at  $t_2$ . Thus  $I_1$  is an increasing function of  $t$  at  $t_2$ , and therefore  $I_1$  stays non-negative.  $I_2$  is shown to be non-negative the same way.

(iii) *Convergence of the total population*

From model (2.1), the differential equations governing the evolution of  $N_1$  and  $N_2$  are

$$\frac{dN_1}{dt} = b_1 - (m_1 + d_1)N_1 + m_2 N_2, \quad (2.5a)$$

$$\frac{dN_2}{dt} = b_2 + m_1 N_1 - (m_2 + d_2)N_2. \quad (2.5b)$$

A direct calculation shows that the unique positive equilibrium  $(N_1^*, N_2^*)$  of (2.5) is asymptotically stable, where

$$N_1^* = \frac{b_1 d_2 + m_2 (b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1}, \quad (2.6a)$$

$$N_2^* = \frac{b_2 d_1 + m_1 (b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1}. \quad (2.6b)$$

This implies the convergence of total population  $N(t)$

$$\lim_{t \rightarrow \infty} N(t) = N_1^* + N_2^* = \frac{b_1(m_1 + m_2 + d_2) + b_2(m_1 + m_2 + d_1)}{m_1 d_2 + m_2 d_1 + d_1 d_2}. \quad \square$$

Note that it follows directly from this result that solutions to (2.1) are bounded.

In epidemiology, the basic reproduction number  $\mathcal{R}_0$  of an infection is the mean number of secondary cases a typical single infected case will cause in a population with no immunity to the disease and in the absence of interventions to control the infection. If  $\mathcal{R}_0 < 1$ , the infection will generally die out in the long run, while when  $\mathcal{R}_0 > 1$ , the infection will be able to spread in a population. Mathematically,  $\mathcal{R}_0 = 1$  is the hypersurface in parameter space where the DFE loses its local asymptotic stability.

The DFE of (2.1) is

$$(S_1^0, I_1^0, S_2^0, I_2^0) = \left( \frac{b_1 d_2 + m_2(b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1}, 0, \frac{b_2 d_1 + m_1(b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1}, 0 \right) =: E_0. \quad (2.7)$$

Applying the next generation matrix method of [van den Driessche & Watmough \(2002\)](#), we have the following results about the basic reproduction number, where the notation  $\mathcal{R}_0^{(x)}$  indicates the reproduction number for system (x).

**THEOREM 2.2** If  $\mathcal{R}_0^{(2.1)} < 1$ , with  $\mathcal{R}_0^{(2.1)}$  given by (2.8) below, then the DFE  $E_0$  in (2.7) is locally asymptotically stable; if  $\mathcal{R}_0^{(2.1)} > 1$ , then  $E_0$  is unstable.

*Proof.* Using the next generation matrix method of [van den Driessche & Watmough \(2002\)](#), we write equations for the two infected variables  $I_1$  and  $I_2$  as

$$\frac{d}{dt} \begin{pmatrix} I_1 \\ I_2 \end{pmatrix} = \mathcal{F} - \mathcal{V} = \begin{pmatrix} \frac{\beta_1 S_1 I_1}{N_1} + \frac{\alpha_2 m_2 S_2 I_2}{N_2} \\ \frac{\beta_2 S_2 I_2}{N_2} + \frac{\alpha_1 m_1 S_1 I_1}{N_1} \end{pmatrix} - \begin{pmatrix} (d + \gamma_1 + m_1)I_1 - m_2 I_2 \\ -m_1 I_1 + (d + \gamma_2 + m_2)I_2 \end{pmatrix},$$

and compute  $F = D\mathcal{F}$  and  $V = D\mathcal{V}$  evaluated at the DFE  $E_0$ , giving

$$F = \begin{pmatrix} \beta_1 & \alpha_2 m_2 \\ \alpha_1 m_1 & \beta_2 \end{pmatrix}, \quad V = \begin{pmatrix} d_1 + \gamma_1 + m_1 & -m_2 \\ -m_1 & d_2 + \gamma_2 + m_2 \end{pmatrix}.$$

Then

$$FV^{-1} = \begin{pmatrix} \frac{P_1}{\det(V)} & \frac{\beta_1 m_2 + \alpha_2 m_2 (d_1 + \gamma_1 + m_1)}{\det(V)} \\ \frac{\beta_2 m_1 + \alpha_1 m_1 (d_2 + \gamma_2 + m_2)}{\det(V)} & \frac{P_2}{\det(V)} \end{pmatrix},$$

where

$$\begin{aligned} P_1 &:= (d_2 + \gamma_2 + m_2)\beta_1 + \alpha_2 m_1 m_2, \\ P_2 &:= (d_1 + \gamma_1 + m_1)\beta_2 + \alpha_1 m_1 m_2, \\ \det(V) &:= (d_1 + \gamma_1 + m_1)(d_2 + \gamma_2 + m_2) - m_1 m_2. \end{aligned}$$

Therefore, the basic reproduction number of (2.1) is

$$\mathcal{R}_0^{(2.1)} := \rho(FV^{-1}) = \frac{P_1 + P_2 + \sqrt{\Delta}}{2 \det(V)}, \quad (2.8)$$

where

$$\Delta = (P_1 - P_2)^2 + 4[\beta_1 m_2 + \alpha_2 m_2 (d_1 + \gamma_1 + m_1)][\beta_2 m_1 + \alpha_1 m_1 (d_2 + \gamma_2 + m_2)]$$

and  $\rho(X)$  is the spectral radius of matrix  $X$ .

It is easy to verify that hypotheses (H1)–(H5) in [van den Driessche & Watmough \(2002, Theorem 2\)](#) hold and the result follows.  $\square$

In fact, the case  $\mathcal{R}_0^{(2.1)} < 1$  can be shown to make the DFE  $E_0$  globally asymptotically stable (see Section 4 for details).

If there is no travel ( $m_1 = m_2 = 0$ ), then the equations for the two patches decouple. The DFE in patch  $i = 1, 2$  is  $(S_i, I_i) = (b_i/d_i, 0)$ . The basic reproduction number is then given by

$$\mathcal{R}_{0,i} := \frac{\beta_i}{d_i + \gamma_i}, \quad i = 1, 2. \quad (2.9)$$

Define

$$\mathcal{R}_{0,i}^T := \frac{\beta_i}{d_i + \gamma_i + m_i} \quad \text{for } i = 1, 2. \quad (2.10)$$

The term  $1/(d_i + \gamma_i + m_i)$  is the average time spent in compartment  $I_i$  taking travelling between the two patches into account.  $\mathcal{R}_{0,i}^T$  is a modified reproduction number that includes travel of infectives.

### 3. The full system without infection during transport

Assume that infectives in both patches travel and there is no infection during transport (i.e.  $\alpha_i = 0$ ,  $i = 1, 2$ ). Substituting  $\alpha_i = 0$  ( $i = 1, 2$ ) into (2.1) yields the following basic SIS patch model:

$$\frac{dI_1}{dt} = \frac{\beta_1(N_1 - I_1)I_1}{N_1} - d_1I_1 - \gamma_1I_1 - m_1I_1 + m_2I_2, \quad (3.1a)$$

$$\frac{dI_2}{dt} = \frac{\beta_2(N_2 - I_2)I_2}{N_2} - d_2I_2 - \gamma_2I_2 - m_2I_2 + m_1I_1, \quad (3.1b)$$

$$\frac{dN_1}{dt} = b_1 - (d_1 + m_1)N_1 + m_2N_2, \quad (3.1c)$$

$$\frac{dN_2}{dt} = b_2 + m_1N_1 - (d_2 + m_2)N_2, \quad (3.1d)$$

where we use  $N_i = S_i + I_i$  for simplicity.

System (3.1) has the DFE  $E_0$  given by (2.7). Using next generation matrix method, (3.1) has the basic reproduction number

$$\mathcal{R}_0^{(3.1)} := \frac{\bar{P}_1 + \bar{P}_2 + \sqrt{\bar{\Delta}}}{2 \det(\bar{V})}, \quad (3.2)$$

where

$$\begin{aligned} \bar{P}_1 &:= (d_2 + \gamma_2 + m_2)\beta_1, \\ \bar{P}_2 &:= (d_1 + \gamma_1 + m_1)\beta_2, \\ \bar{\Delta} &:= (\bar{P}_1 - \bar{P}_2)^2 + 4\beta_1\beta_2m_1m_2, \end{aligned} \quad (3.3)$$

$$\det(\bar{V}) := (d_1 + \gamma_1 + m_1)(d_2 + \gamma_2 + m_2) - m_1m_2 =: \det(V).$$

For system (3.1), [Salmani & van den Driessche \(2006\)](#) gave a detailed analysis; their main result is the following.

LEMMA 3.1 (Salmani & van den Driessche, 2006) The DFE  $E_0$  in (3.1) is globally asymptotically stable if  $\mathcal{R}_0^{(3.1)} < 1$  and there exists a unique globally asymptotically stable endemic equilibrium  $E_*$  if  $\mathcal{R}_0^{(3.1)} > 1$ .

#### 4. The full system with infection during transport

In this section, we continue studying model (2.1) to complete the preliminary results obtained in Section 2.

THEOREM 4.1 The DFE  $E_0$  of model (2.1) is globally asymptotically stable if  $\mathcal{R}_0^{(2.1)} < 1$ .

*Proof.* It suffices to prove that each positive solution of (2.1) tends to  $E_0$  as  $t \rightarrow \infty$ . Equivalently, this can be done if we prove that each positive solution  $(S_1(t), I_1(t), S_2(t), I_2(t))$  of (2.1) satisfies

$$\lim_{t \rightarrow \infty} (S_1(t), I_1(t), S_2(t), I_2(t)) = \left( \frac{b_1 d_2 + m_2(b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1}, 0, \frac{b_2 d_1 + m_1(b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1}, 0 \right). \quad (4.1)$$

From (2.1b) and (2.1d) with  $S_i(t) \leq N_i(t)$  ( $i = 1, 2$ ) for all  $t$ , we deduce that

$$\frac{dI_1}{dt} \leq \beta_1 I_1 + \alpha_2 m_2 I_2 - d_1 I_1 - \gamma_1 I_1 - m_1 I_1 + m_2 I_2, \quad (4.2a)$$

$$\frac{dI_2}{dt} \leq \beta_2 I_2 + \alpha_1 m_1 I_1 - d_2 I_2 - \gamma_2 I_2 - m_2 I_2 + m_1 I_1. \quad (4.2b)$$

The right-hand side of (4.2) has coefficient matrix  $F - V$ . For  $\mathcal{R}_0^{(2.1)} = \rho(FV^{-1}) < 1$ , each eigenvalue of  $F - V$  lies in the left half plane (van den Driessche & Watmough, 2002, Proof of Theorem 2), thus, following a standard comparison theorem of ODE, each positive solution of (2.1b) and (2.1d) satisfies  $\lim_{t \rightarrow \infty} (I_1, I_2) = (0, 0)$ .

By the theory of asymptotically autonomous systems (Castillo-Chavez & Thieme, 1995, Theorem 2.5), the limit system of (2.1a) and (2.1c) obtained when  $I_i \rightarrow 0$  as  $t \rightarrow \infty$  is written as

$$\frac{dS_1}{dt} = b_1 - d_1 S_1 - m_1 S_1 + m_2 S_2, \quad (4.3a)$$

$$\frac{dS_2}{dt} = b_2 - d_2 S_2 - m_2 S_2 + m_1 S_1. \quad (4.3b)$$

It is easy to verify from (4.3) that

$$\lim_{t \rightarrow \infty} S_i(t) = \frac{b_i d_j + m_j(b_i + b_j)}{d_i d_j + d_i m_j + d_j m_i}, \quad i, j = 1, 2 \text{ and } i \neq j. \quad (4.4)$$

The global asymptotic stability of the DFE  $E_0$  is then proved if  $\mathcal{R}_0^{(2.1)} < 1$ .  $\square$

Theorem 4.1 extends the result of Takeuchi *et al.* (2007, Theorem 3.3) in proving the global asymptotic stability of the DFE when the basic reproduction number is less than unity, without any parameter restrictions.

Discussions in Section 2 tell that the unique positive equilibrium  $(N_1^*, N_2^*)$  is asymptotically stable in the linear system (2.5). Replacing  $N_i$  with  $N_i^*$  ( $i = 1, 2$ ) in sub-system (2.1b) and (2.1d) gives the limit



system

$$\frac{dI_1}{dt} = \beta_1 \frac{(N_1^* - I_1)I_1}{N_1^*} + \alpha_2 m_2 \frac{(N_2^* - I_2)I_2}{N_2^*} + m_2 I_2 - m_1 I_1 - (\gamma_1 + d_1)I_1 =: P(I_1, I_2), \quad (4.5a)$$

$$\frac{dI_2}{dt} = \beta_2 \frac{(N_2^* - I_2)I_2}{N_2^*} + \alpha_1 m_1 \frac{(N_1^* - I_1)I_1}{N_1^*} - m_2 I_2 + m_1 I_1 - (\gamma_2 + d_2)I_2 =: Q(I_1, I_2). \quad (4.5b)$$

Take a Dulac function  $D = 1/(I_1 I_2)$ . Then

$$\begin{aligned} \frac{\partial(DP)}{\partial I_1}(I_1, I_2) &= \frac{\beta_1}{N_1^* I_2} - \alpha_2 m_2 \frac{(N_2^* - I_2)}{N_2^* I_1^2} - \frac{m_2}{I_1^2}, \\ \frac{\partial(DQ)}{\partial I_2}(I_1, I_2) &= \frac{\beta_2}{N_2^* I_1} - \alpha_1 m_1 \frac{(N_1^* - I_1)}{N_1^* I_2^2} - \frac{m_1}{I_2^2} \end{aligned}$$

and

$$\frac{\partial(DP)}{\partial I_1}(I_1, I_2) + \frac{\partial(DQ)}{\partial I_2}(I_1, I_2) < 0.$$

From the Bendixson–Dulac criterion (Dulac, 1937), we have the following result.

**THEOREM 4.2** System (4.5) does not admit any cycle in the invariant region  $\mathcal{D} := \{(I_1, I_2); 0 < I_i \leq N_i^*, i = 1, 2\}$ .

Now we prove the existence and uniqueness of an endemic equilibrium.

**THEOREM 4.3** If  $\mathcal{R}_0^{(2.1)} > 1$ , then there exists a unique endemic equilibrium for system (4.5).

*Proof.* The existence of the endemic equilibrium is confirmed using the properties of ordinary equations in the plane. From Theorem 2.2, if  $\mathcal{R}_0^{(2.1)} > 1$ , then the DFE  $(0, 0)$  is unstable. Since all solutions are ultimately bounded in the plane and there does not exist any cycle in the feasible set, it follows that there must exist endemic equilibrium (equilibria) attracting trajectories of (4.5).

To establish uniqueness, set the right-hand side of (4.5) equal to zero

$$m_2(\alpha_2 + 1)I_2 - \frac{\alpha_2 m_2}{N_2^*} I_2^2 = (m_1 + \gamma_1 + d_1 - \beta_1)I_1 + \frac{\beta_1}{N_1^*} I_1^2, \quad (4.6a)$$

$$m_1(\alpha_1 + 1)I_1 - \frac{\alpha_1 m_1}{N_1^*} I_1^2 = (m_2 + \gamma_2 + d_2 - \beta_2)I_2 + \frac{\beta_2}{N_2^*} I_2^2. \quad (4.6b)$$

Equations (4.6) are two ellipses given by the implicit equations

$$\Phi_1(I_1, I_2) := \frac{(I_1 - x_0)^2}{a^2} + \frac{(I_2 - y_0)^2}{b^2} = 1, \quad (4.7a)$$

$$\Phi_2(I_1, I_2) := \frac{(I_1 - \hat{x}_0)^2}{\hat{a}^2} + \frac{(I_2 - \hat{y}_0)^2}{\hat{b}^2} = 1, \quad (4.7b)$$

where

$$\begin{aligned} x_0 &= \frac{\beta_1 - (m_1 + \gamma_1 + d_1)}{2\beta_1} N_1^* < \frac{N_1^*}{2}, \quad y_0 = \frac{\alpha_2 + 1}{2\alpha_2} N_2^* > N_2^*, \\ \hat{x}_0 &= \frac{\alpha_1 + 1}{2\alpha_1} N_1^* > N_1^*, \quad \hat{y}_0 = \frac{\beta_2 - (m_2 + \gamma_2 + d_2)}{2\beta_2} N_2^* < \frac{N_2^*}{2}, \\ a &= \left( \frac{N_1^*(m_1 + \gamma_1 + d_1 - \beta_1)^2/(4\beta_1) + m_2 N_2^*(\alpha_2 + 1)^2/(4\alpha_2)}{\beta_1/N_1^*} \right)^{1/2}, \\ b &= \left( \frac{N_1^*(m_1 + \gamma_1 + d_1 - \beta_1)^2/(4\beta_1) + m_2 N_2^*(\alpha_2 + 1)^2/(4\alpha_2)}{(\alpha_2 m_2)/N_2^*} \right)^{1/2}, \\ \hat{a} &= \left( \frac{N_2^*(m_2 + \gamma_2 + d_2 - \beta_2)^2/(4\beta_2) + m_1 N_1^*(\alpha_1 + 1)^2/(4\alpha_1)}{\beta_2/N_2^*} \right)^{1/2}, \\ \hat{b} &= \left( \frac{N_2^*(m_2 + \gamma_2 + d_2 - \beta_2)^2/(4\beta_2) + m_1 N_1^*(\alpha_1 + 1)^2/(4\alpha_1)}{(\alpha_1 m_1)/N_1^*} \right)^{1/2}. \end{aligned}$$

The ellipse  $\Phi_1(I_1, I_2)$  has centre  $(x_0, y_0)$  with two radii  $a$  and  $b$ ; the ellipse  $\Phi_2(I_1, I_2)$  has centre  $(\hat{x}_0, \hat{y}_0)$  with two radii  $\hat{a}$  and  $\hat{b}$ ; the major radius is  $\max(a, b)$  and the minor radius is  $\min(a, b)$ ; both  $\Phi_1(I_1, I_2)$  and  $\Phi_2(I_1, I_2)$  pass through the origin. From the existence part of the proof, we know that if  $\mathcal{R}_0^{(2.1)} > 1$ , system (4.5) has at least one positive equilibrium in the feasible set  $\{(I_1, I_2) : 0 < I_1 < N_1^*, 0 < I_2 < N_2^*\} =: \mathcal{D}$ . Next, we show by Fig. 2(a–d) that the two ellipses given by (4.7) have at most one intersection point in  $\mathcal{D}$ . In fact, considering  $\hat{x}_0 > N_1^*$  and  $y_0 > N_2^*$ , then the expected positive equilibrium lies in the region  $\Sigma$  bounded by  $I_1, I_2$  axes, the horizontal line  $I_2 = y_0$  and the vertical line  $I_1 = \hat{x}_0$ .

Therefore, system (4.5) has a unique endemic equilibrium in  $\mathcal{D}$  when the basic reproduction number  $\mathcal{R}_0^{(2.1)} > 1$ . The proof is complete.  $\square$

**REMARK 4.1** In the analysis above, we show that the two ellipses given by (4.7) in  $\mathcal{D}$  have at most one intersection point if it exists. So we omit the cases where the two ellipses may not intersect in the bounded region  $\Sigma$  (see Fig. 3(a,b)), which can never happen if  $\mathcal{R}_0^{(2.1)} > 1$ . Without loss of generality, when drawing Fig. 2(a–d), and Fig. 3(a,b), we assume  $\hat{a}, b$  as the major radii, and  $a, \hat{b}$  as the minor radii for the two ellipses, i.e.  $\hat{a} > \hat{b}$  and  $a < b$ . For the remaining cases,  $a < b$  and  $\hat{a} < \hat{b}$ ;  $a > b$  and  $\hat{a} > \hat{b}$ ;  $a > b$  and  $\hat{a} < \hat{b}$ , the same conclusion can be obtained.

**REMARK 4.2** Under the relations  $x_0 < \hat{x}_0$  and  $y_0 > \hat{y}_0$ , there are in fact additional cases for the two ellipse positions that are not included in Figs 2 and 3. The condition  $\mathcal{R}_0^{(2.1)} > 1$  has no correlation with the conditions  $x_0 < \hat{x}_0$  and  $y_0 > \hat{y}_0$ . When  $\mathcal{R}_0^{(2.1)} > 1$ , system (4.5) is proved to have at least one endemic equilibrium in  $\mathcal{D}$  (i.e. the two ellipses intersect at least one point in  $\mathcal{D}$ ). The four cases in Fig. 2 are all the possibilities that the two ellipses intersect in  $\mathcal{D}$ . As we see, the intersection point in  $\mathcal{D}$  for the four cases of Fig. 2 is unique. Besides  $\mathcal{R}_0^{(2.1)} > 1$ , no other parameter restrictions are needed to guarantee the existence of the unique endemic equilibrium for system (4.5).

Based on Theorem 4.3, we have the following result.

**THEOREM 4.4** If  $\mathcal{R}_0^{(2.1)} > 1$ , then system (2.1) has a unique positive equilibrium that is globally asymptotically stable in the feasible set  $\mathcal{D}$ .

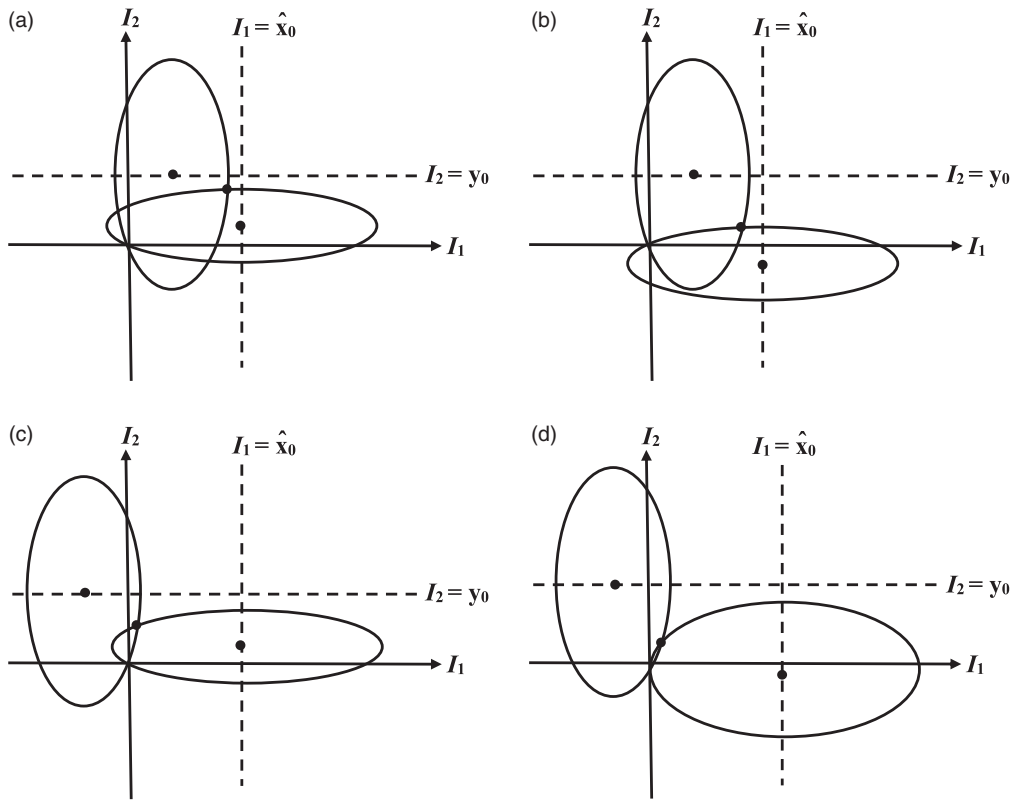


FIG. 2. (a)  $x_0 > 0, \hat{y}_0 > 0$ , (b)  $x_0 > 0, \hat{y}_0 < 0$ , (c)  $x_0 < 0, \hat{y}_0 > 0$  and (d)  $x_0 < 0, \hat{y}_0 < 0$ .

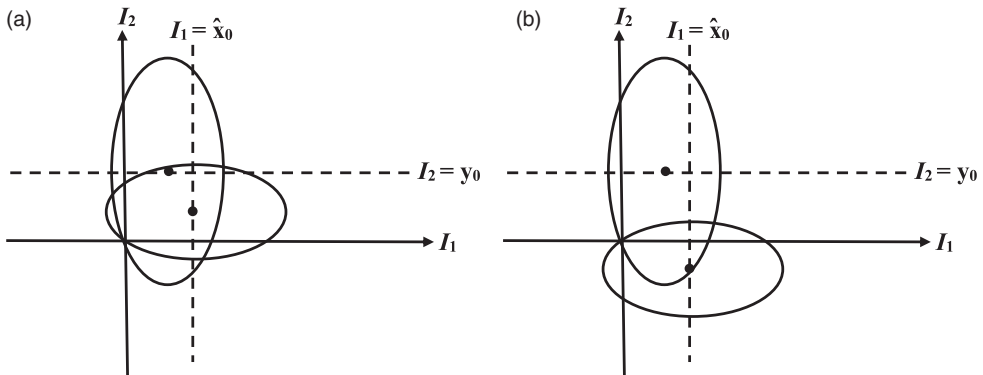


FIG. 3. (a)  $x_0 > 0, \hat{y}_0 > 0$  and (b)  $x_0 > 0, \hat{y}_0 < 0$ .

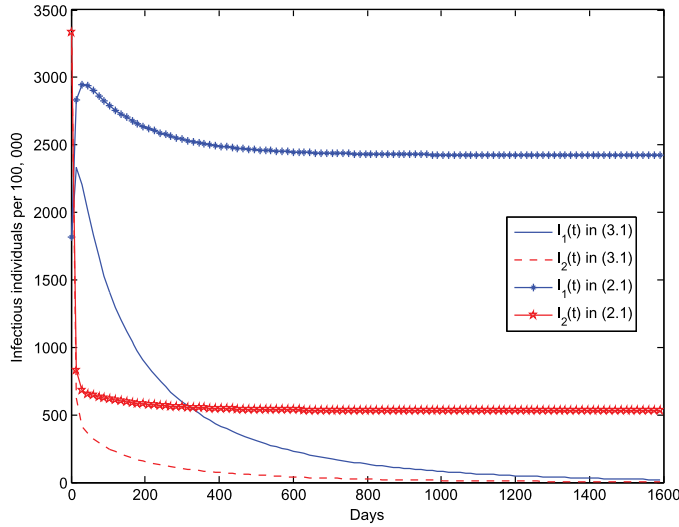


FIG. 4. Behaviour of  $I_1$  and  $I_2$  close to  $\mathcal{R}_0 = 1$  without and with infection during transport.

Theorem 4.4 makes the conjecture of Takeuchi *et al.* (2007) ‘the positive equilibrium is globally asymptotically stable if it exists’ decidable, without extra parameter restrictions. Cui *et al.* (2006) also discuss the simplified system of (2.1). However, all their results are local.

The basic reproduction numbers  $\mathcal{R}_0^{(2.1)}$  is sensitive to the two disease transmission parameters during transport  $\alpha_1$  and  $\alpha_2$ . Straightforward calculations indicate that  $\mathcal{R}_0^{(2.1)}$  increases with respect to both  $\alpha_1$  and  $\alpha_2$ . Moreover,  $\mathcal{R}_0^{(2.1)} = \mathcal{R}_0^{(3.1)}$  only when  $\alpha_1 = \alpha_2 = 0$ . This implies that  $\mathcal{R}_0^{(2.1)} \geq \mathcal{R}_0^{(3.1)}$ . Thus, the risk for disease outbreak is higher in system (2.1) with infection during transport. Even if disease is eradicated for system (3.1), but it can be endemic in both patches for system (2.1).

This is illustrated in Fig. 4, in which  $\mathcal{R}_0^{(2.1)} = 1.0072$ ,  $\mathcal{R}_0^{(3.1)} = 0.9899$ ,  $\beta_1 = 0.235$ ,  $\beta_2 = 0.02$  while other parameters take values as in Table 2.

## 5. Infectives from only one patch travel: perfect exit screening scenario

During an infectious disease outbreak and other public health events, as well as routinely in some countries, passengers are sometimes screened to check if they present symptoms of a disease or if they satisfy certain health requirements such as vaccination against a particular disease. Screening can take place either upon departure from a country (exit screening) or upon arrival into a country (entry screening). For example, with the rapid international spread of SARS from March through May 2003, WHO requested that all affected areas screen departing passengers for SARS symptoms. The Public Health Agency of Canada introduced various measures to screen airplane passengers at selected airports for symptoms and signs of SARS (St John *et al.*, 2005). Many Asian countries hit by SARS set up thermal scanners at airports to screen for passengers who may have been feverish. During the 2009 pH1N1 scare, these countries restarted these checks.

Here, we consider a perfect exit border screening programme implemented asymmetrically in one patch. Without loss of generality, we assume that susceptibles from both patches and infectives from patch 1 travel freely, while infectives from patch 2 are restrained from travelling to patch 1.

Two specific models are presented and studied in this section.

### 5.1 Model without infection during transport

By setting  $\alpha_1 = 0$ ,  $m_2 I_2 = 0$ , we derive from (2.1) the following system of differential equations:

$$\frac{dI_1}{dt} = \frac{\beta_1(N_1 - I_1)I_1}{N_1} - d_1 I_1 - \gamma_1 I_1 - m_1 I_1, \quad (5.1a)$$

$$\frac{dI_2}{dt} = \frac{\beta_2(N_2 - I_2)I_2}{N_2} - d_2 I_2 - \gamma_2 I_2 + m_1 I_1, \quad (5.1b)$$

$$\frac{dN_1}{dt} = b_1 + m_2 N_2 - (d_1 + m_1)N_1 - m_2 I_2, \quad (5.1c)$$

$$\frac{dN_2}{dt} = b_2 - (d_2 + m_2)N_2 + m_1 N_1 + m_2 I_2, \quad (5.1d)$$

which describes a model with a perfect border screening and without infection during transport.

Using the next generation matrix method as in Section 2, the basic reproduction number of (5.1) is calculated, with

$$\mathcal{R}_0^{(5.1)} := \max\{\mathcal{R}_{0,2}, \mathcal{R}_{0,1}^T\}.$$

There possibly exist three non-negative equilibria  $E_0, E_{BE}$  and  $E_*$  for (5.1), which are the disease-free, the boundary and the endemic equilibrium, respectively. The expressions of three equilibria are as follows:

- (i) the DFE  $E_0$  is given by (2.7);
- (ii) the boundary equilibrium (BE) is

$$\begin{aligned} E_{BE} &:= (I_{1BE}, I_{2BE}, N_{1BE}, N_{2BE}) \\ &= (0, (1 - 1/\mathcal{R}_{0,2})N_{2BE}, (b_1 + b_2 - d_2 N_{2BE})/d_1, N_{2BE}) \end{aligned} \quad (5.2)$$

with  $I_{2BE}$  positive if  $\mathcal{R}_{0,2} > 1$ , where

$$N_{2BE} = \frac{(d_1 + m_1)b_2 + m_1 b_1}{d_2(d_1 + m_1) + d_1 m_2 / \mathcal{R}_{0,2}}; \quad (5.3)$$

- (iii) EE  $E_* := (I_{1*}, I_{2*}, N_{1*}, N_{2*}) = ((1 - 1/\mathcal{R}_{0,1}^T)N_{1*}, I_{2*}, N_{1*}, N_{2*})$  with  $d_1 N_{1*} + d_2 N_{2*} = b_1 + b_2$ . The coordinate  $I_{1*}$  is positive if  $\mathcal{R}_{0,1}^T > 1$ . The coordinate  $I_{2*}$  satisfies

$$\frac{\beta_2 I_{2*}^2}{N_{2*}} + (d_2 + \gamma_2 - \beta_2)I_{2*} - m_1 \left(1 - \frac{1}{\mathcal{R}_{0,1}^T}\right) N_{1*} = 0. \quad (5.4)$$

Equation (5.4) has a unique positive solution for  $I_{2*}$  when  $\mathcal{R}_{0,1}^T > 1$ . Indeed, writing (5.1c) and (5.1d) at equilibrium as a linear system in  $(N_{1*}, N_{2*})$ :

$$\begin{pmatrix} -(d_1 + m_1) & m_2 \\ m_1 & -(d_2 + m_2) \end{pmatrix} \begin{pmatrix} N_{1*} \\ N_{2*} \end{pmatrix} = \begin{pmatrix} -b_1 + m_2 I_{2*} \\ -b_2 - m_2 I_{2*} \end{pmatrix}$$

gives

$$\begin{aligned} N_{1*} &= \frac{d_2 b_1 + m_2(b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1} - \frac{d_2 m_2}{d_1 d_2 + d_1 m_2 + d_2 m_1} I_{2*} \\ &=: p_1 - q_1 I_{2*}, \\ N_{2*} &= \frac{d_1 b_2 + m_1(b_1 + b_2)}{d_1 d_2 + d_1 m_2 + d_2 m_1} + \frac{d_1 m_2}{d_1 d_2 + d_1 m_2 + d_2 m_1} I_{2*} \\ &=: p_2 + q_2 I_{2*}, \end{aligned} \tag{5.5}$$

where  $p_1, q_1, p_2, q_2 > 0$  and  $0 < q_2 < 1$ . Define  $c := m_1(1 - 1/\mathcal{R}_{0,1}^T)$ , so  $c > 0$  whenever  $\mathcal{R}_{0,1}^T > 1$ . Equation (5.4) can then be rewritten as

$$\beta_2 I_{2*}^2 + (d_2 + \gamma_2 - \beta_2) I_{2*} N_{2*} - c N_{1*} N_{2*} = 0. \tag{5.6}$$

Substituting  $N_{1*}$  and  $N_{2*}$  in (5.5) into (5.6) yields

$$A I_{2*}^2 + B I_{2*} + C = 0, \tag{5.7}$$

where

$$\begin{aligned} A &= \beta_2 + q_2(d_2 + \gamma_2 - \beta_2) + c q_1 q_2 \\ &= (1 - q_2)\beta_2 + q_2(d_2 + \gamma_2) + c q_1 q_2 > 0, \\ B &= p_2(d_2 + \gamma_2 - \beta_2) - c p_1 q_2 + c p_2 q_1, \\ C &= -c p_1 p_2 < 0. \end{aligned}$$

It is then obvious that (5.4) has a unique solution for  $I_{2*}$  when  $\mathcal{R}_{0,1}^T > 1$ .

Salmani & van den Driessche (2006) presented a more general model for (5.1), in which the disease may cause fatality and travel rates are dependent of disease status, and showed the global asymptotic stability of  $E_0$  if  $\mathcal{R}_{0,2} < 1$  and  $\mathcal{R}_{0,1}^T < 1$ , i.e.  $\mathcal{R}_0^{(5.1)} < 1$ ; local asymptotical stability of  $E_{BE}$  if  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$  (by the Routh–Hurwitz criterion); local asymptotic stability of  $E_*$  (numerical result in Salmani & van den Driessche, 2006) if  $\mathcal{R}_{0,2} < 1$  and  $\mathcal{R}_{0,1}^T > 1$ . The global asymptotic stability of  $E_{BE}$  and analytical results on  $E_*$  remain open. For (5.1), when  $d_1 = d_2$ , Wang & Mulone (2003, Theorem 2.4) showed that the disease is uniformly persistent in two patches if  $\mathcal{R}_{0,1}^T > 1$ .

**THEOREM 5.1** The BE  $E_{BE}$  of (5.1) is globally asymptotically stable if  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$ .

*Proof.* Note from (iii) that when  $\mathcal{R}_{0,1}^T < 1$ , system (5.1) only has disease-free and boundary equilibria. Equation (5.1b) indicates that

$$\frac{dI_1}{dt} \leq \beta_1 I_1 - d_1 I_1 - \gamma_1 I_1 - m_1 I_1 = \beta_1 \left( 1 - \frac{1}{\mathcal{R}_{0,1}^T} \right) I_1.$$

We conclude that  $\lim_{t \rightarrow \infty} I_1 = 0$  if  $\mathcal{R}_{0,1}^T < 1$ . Thus, by the theory of asymptotically autonomous systems (Castillo-Chavez & Thieme, 1995, Theorem 2.5), one only needs to study the limiting system of (5.1):

$$\frac{dI_2}{dt} = \frac{\beta_2(N_2 - I_2)I_2}{N_2} - d_2 I_2 - \gamma_2 I_2, \quad (5.8a)$$

$$\frac{dN_2}{dt} = b_2 - (d_2 + m_2)N_2 + m_1 N_1 + m_2 I_2, \quad (5.8b)$$

$$\frac{dN_1}{dt} = b_1 + m_2 N_2 - (d_1 + m_1)N_1 - m_2 I_2. \quad (5.8c)$$

System (5.8) has two equilibria: the BE  $E'_0$  and the positive equilibrium  $E'_{BE}$ . Here,

$$E'_0 := \left( 0, \frac{b_2 d_1 + m_1(b_1 + b_2)}{d_1 d_2 + d_2 m_1 + d_1 m_2}, \frac{b_1 d_2 + m_2(b_1 + b_2)}{d_1 d_2 + d_2 m_1 + d_1 m_2} \right)$$

and

$$E'_{BE} := ((1 - 1/\mathcal{R}_{0,2})N_{2BE}, N_{2BE}, (b_1 + b_2 - d_2 N_{2BE})/d_1).$$

Salmani & van den Driessche (2006) proved that for (5.8), the BE  $E'_0$  is unstable and the positive equilibrium  $E'_{BE}$  is locally asymptotically stable if  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$ . It is sufficient to prove that the positive equilibrium  $E'_{BE}$  of (5.8) is globally asymptotically stable.

Next, we apply the second compound matrix (Appendix 8) and the Poincaré–Bendixson property (Appendix 8) to prove the global asymptotic stability of  $E'_{BE}$ .

The stability property of  $E'_0$  described above indicates that a similar argument as in Li *et al.* (1999, Proof of Proposition 3.3) may be used to show system (5.8) is uniformly persistent, i.e. there exists a constant  $0 < c < 1$  such that any solution  $(I_2(t), N_2(t), N_1(t))$  with  $(I_2(0), N_2(0), N_1(0)) \in \Sigma$  satisfies

$$\min \left\{ \liminf_{t \rightarrow \infty} I_2(t), \liminf_{t \rightarrow \infty} N_2(t), \liminf_{t \rightarrow \infty} N_1(t) \right\} > c.$$

This implies that system (5.8) has a compact absorbing orbit set  $K \in \Sigma$ , where  $\Sigma$  is the interior of  $\Sigma$  in  $\mathbb{R}^3$ . Moreover,  $E'_{BE}$  is the unique equilibrium in  $\Sigma$ . Hence, condition (1) in Theorem A.2 holds.

The Jacobian matrix of (5.8) at an arbitrary point  $P(I_2, N_2, N_1)$  is

$$J(P) = \begin{pmatrix} \beta_2 - d_2 - \gamma_2 - \frac{2\beta_2 I_2}{N_2} & \frac{\beta_2 I_2^2}{N_2^2} & 0 \\ m_2 & -(d_2 + m_2) & m_1 \\ -m_2 & m_2 & -(d_1 + m_1) \end{pmatrix}.$$

By examining  $J(P)$  and choosing the matrix  $H = \text{diag}(1, -1, 1)$ , one can verify that, when  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$ , the system (5.8) is competitive in the convex region  $\Sigma =: \{(N_1, N_2) | (b_1 + b_2)/\max\{d_1, d_2\} \leq N_1 + N_2 \leq (b_1 + b_2)/\min\{d_1, d_2\}\}$  with respect to the partial ordering defined by the orthant

$\{(I_2, N_2, N_1) \in \mathbb{R}^3 : I_2 \geq 0, N_2 \leq 0, N_1 \geq 0\}$ . By Theorem A.1, System (5.8) satisfies the Poincaré–Bendixson Property and thus condition (3) in Theorem A.2 holds.

The second compound matrix  $J^{[2]}(P)$  (Appendix 8) of the Jacobian matrix  $J(P)$  is

$$\begin{pmatrix} \beta_2 - 2d_2 - \gamma_2 - m_2 - \frac{2\beta_2 I_2}{N_2} & m_1 & 0 \\ m_2 & \beta_2 - d_2 - \gamma_2 - d_1 - m_1 - \frac{2\beta_2 I_2}{N_2} & \frac{\beta_2 I_2^2}{N_2^2} \\ m_2 & m_2 & -(d_1 + m_1 + d_2 + m_2) \end{pmatrix}.$$

Let  $z = (z_1, z_2, z_3)^T$  be the solution of the second compound system  $dz/dt = J^{[2]}(P)z$ , namely,

$$\begin{aligned} \dot{z}_1 &= \left( \beta_2 - 2d_2 - \gamma_2 - m_2 - \frac{2\beta_2 I_2}{N_2} \right) z_1 + m_1 z_2, \\ \dot{z}_2 &= m_2 z_1 + \left( \beta_2 - d_2 - \gamma_2 - d_1 - m_1 - \frac{2\beta_2 I_2}{N_2} \right) z_2 + \frac{\beta_2 I_2^2}{N_2^2} z_3, \\ \dot{z}_3 &= m_2 z_1 + m_2 z_2 - (d_1 + m_1 + d_2 + m_2) z_3. \end{aligned} \quad (5.9)$$

Define the norm of  $z$  as

$$\|z\| = \max\{|z_1| + |z_2|, |z_3|\}. \quad (5.10)$$

If  $\|z\| = |z_1| + |z_2|$ , then

$$\begin{aligned} D_+ \|z\| &= D_+(|z_1| + |z_2|) \\ &\leq \left( \beta_2 - 2d_2 - \gamma_2 - m_2 - \frac{2\beta_2 I_2}{N_2} \right) |z_1| + m_1 |z_2| \\ &\quad + m_2 |z_1| + \left( \beta_2 - d_2 - \gamma_2 - d_1 - m_1 - \frac{2\beta_2 I_2}{N_2} \right) |z_2| + \frac{\beta_2 I_2^2}{N_2^2} |z_3| \\ &\leq \left( \beta_2 - 2d_2 - \gamma_2 - \frac{2\beta_2 I_2}{N_2} \right) |z_1| + \left( \beta_2 - d_2 - \gamma_2 - d_1 - \frac{2\beta_2 I_2}{N_2} \right) |z_2| + \frac{\beta_2 I_2^2}{N_2^2} |z_3| \\ &\leq \left( \beta_2 - 2d_2 - \gamma_2 - \frac{2\beta_2 I_2}{N_2} \right) |z_1| + \left( \beta_2 - d_2 - \gamma_2 - d_1 - \frac{2\beta_2 I_2}{N_2} \right) |z_2| + \frac{\beta_2 I_2}{N_2} |z_3| \\ &\leq \left( \beta_2 - 2d_2 - \gamma_2 - \frac{2\beta_2 I_2}{N_2} \right) |z_1| + \left( \beta_2 - d_2 - \gamma_2 - d_1 - \frac{2\beta_2 I_2}{N_2} \right) |z_2| + \frac{\beta_2 I_2}{N_2} (|z_1| + |z_2|) \\ &\leq \left( \beta_2 - d_2 - \gamma_2 - \frac{\beta_2 I_2}{N_2} \right) (|z_1| + |z_2|) - d_2 |z_1| - d_1 |z_2| \\ &= \frac{I_2}{I_2} (|z_1| + |z_2|) - d_2 |z_1| - d_1 |z_2| \\ &\leq -\min \left\{ d_1 - \frac{I_2}{I_2}, d_2 - \frac{I_2}{I_2} \right\} \|z\|. \end{aligned} \quad (5.11)$$



Suppose that the solution  $\Omega := (I_2(t), N_2(t), N_1(t))$  to (5.8) is periodic with the least positive period  $\omega$  and the initial condition  $(I_2(0), N_2(0), N_1(0)) \in \mathbb{R}_{+0}^3$ . We then have

$$\begin{aligned}
 - \int_{\Omega} \min \left\{ d_2 - \frac{\dot{I}_2}{I_2}, d_1 - \frac{\dot{I}_2}{I_2} \right\} dt &\leq - \int_{\Omega} \min\{d_1, d_2\} dt \\
 &= - \min\{d_1, d_2\} \omega < 0.
 \end{aligned}
 \tag{5.12}$$

On the other hand, if  $\|z\| = |z_3|$ , then

$$\begin{aligned}
 D_+ \|z\| &= D_+ |z_3| \\
 &\leq m_2(|z_1| + |z_2|) - (d_1 + m_1 + d_2 + m_2)|z_3| \\
 &\leq -(d_1 + m_1 + d_2)\|z\|.
 \end{aligned}
 \tag{5.13}$$

Inequalities (5.11)–(5.13) indicate that  $\|z\| \rightarrow 0$  as  $t \rightarrow \infty$ , and this in turn implies that  $(z_1, z_2, z_3) \rightarrow 0$  as  $t \rightarrow \infty$ . Therefore, the linear system (5.9) is asymptotically stable and the periodic solution  $\Omega := (I_2(t), N_2(t), N_1(t))$  of (5.8) is asymptotically orbitally stable. By Theorem A.2, the equilibrium  $E'_{BE}$  is globally asymptotically stable.  $\square$

### 5.2 Model with infection during transport

By setting  $m_2 I_2 = 0$ , we derive from model (2.1) the following system of differential equations:

$$\frac{dI_1}{dt} = \frac{\beta_1(N_1 - I_1)I_1}{N_1} - d_1 I_1 - \gamma_1 I_1 - m_1 I_1,
 \tag{5.14a}$$

$$\frac{dI_2}{dt} = \frac{\beta_2(N_2 - I_2)I_2}{N_2} + \frac{\alpha_1 m_1 (N_1 - I_1)I_1}{N_1} - d_2 I_2 - \gamma_2 I_2 + m_1 I_1,
 \tag{5.14b}$$

$$\frac{dN_1}{dt} = b_1 + m_2 N_2 - (d_1 + m_1)N_1 - m_2 I_2,
 \tag{5.14c}$$

$$\frac{dN_2}{dt} = b_2 - (d_2 + m_2)N_2 + m_1 N_1 + m_2 I_2,
 \tag{5.14d}$$

which describes the model with perfect exit border screening and infection during transport.

The basic reproduction number of (5.14) is  $\mathcal{R}_0^{(5.14)} := \max\{\mathcal{R}_{0,2}, \mathcal{R}_{0,1}^T\}$ . Note that  $\mathcal{R}_0^{(5.14)} = \mathcal{R}_0^{(5.1)}$ . The disease transmission coefficient during transport  $\alpha_1$  has no effect on  $\mathcal{R}_0^{(5.14)}$  when a perfect border screening programme is initiated.

The three possible non-negative equilibria of (5.14) are as follows.

1. DFE  $E_0$  as given by (2.7);
2. BE  $E_{BE}$  as given by (5.2) with  $I_{2BE}$  positive if  $\mathcal{R}_{0,2} > 1$ , where  $N_{2BE}$  has the same form as (5.3);
3. EE  $E_* := ((1 - 1/\mathcal{R}_{0,1}^T)N_{1*}, I_{2*}, N_{1*}, N_{2*})$  with  $d_1 N_{1*} + d_2 N_{2*} = b_1 + b_2$ . The coordinate  $I_{2*}$  satisfies

$$\frac{\beta_2 I_{2*}^2}{N_{2*}} + (d_2 + \gamma_2 - \beta_2)I_{2*} - m_1 \left( 1 + \frac{\alpha_1}{\mathcal{R}_{0,1}^T} \right) \left( 1 - \frac{1}{\mathcal{R}_{0,1}^T} \right) N_{1*} = 0.
 \tag{5.15}$$

Using an argument very similar to that used when considering (5.4), it can be shown that (5.15) has a unique positive solution for  $I_{2*}$  when  $\mathcal{R}_{0,1}^T > 1$ .

**THEOREM 5.2** The DFE  $E_0$  of (5.14) is globally asymptotically stable if  $\mathcal{R}_0^{(5.14)} < 1$ .

*Proof.* Since  $I_1 \leq N_1$  and  $I_2 \leq N_2$ , (5.14a) and (5.14b) satisfy

$$\frac{dI_1}{dt} \leq \beta_1 I_1 - d_1 I_1 - \gamma_1 I_1 - m_1 I_1, \quad (5.16a)$$

$$\frac{dI_2}{dt} \leq \beta_2 I_2 + \alpha_1 m_1 I_1 - d_2 I_2 - \gamma_2 I_2 + m_1 I_1. \quad (5.16b)$$

Define an auxiliary system as

$$\frac{d\bar{I}_1}{dt} = \beta_1 \bar{I}_1 - d_1 \bar{I}_1 - \gamma_1 \bar{I}_1 - m_1 \bar{I}_1, \quad (5.17a)$$

$$\frac{d\bar{I}_2}{dt} = \beta_2 \bar{I}_2 + \alpha_1 m_1 \bar{I}_1 - d_2 \bar{I}_2 - \gamma_2 \bar{I}_2 + m_1 \bar{I}_1. \quad (5.17b)$$

The basic reproduction number  $\mathcal{R}_0^{(5.14)} < 1$  makes all eigenvalues of (5.17) negative, which implies that  $\lim_{t \rightarrow \infty} \bar{I}_1 = \lim_{t \rightarrow \infty} \bar{I}_2 = 0$ . Following a standard comparison theorem of ODE (Smith & Waltman, 1995, Theorem B.1), we have  $\lim_{t \rightarrow \infty} (I_1, I_2) = (0, 0)$ .

By the theory of asymptotically autonomous system (Castillo-Chavez & Thieme, 1995, Theorem 2.5), the limiting system of (5.14c) and (5.14d) is written as

$$\frac{dN_2}{dt} = b_2 - (d_2 + m_2)N_2 + m_1 N_1, \quad (5.18a)$$

$$\frac{dN_1}{dt} = b_1 + m_2 N_2 - (d_1 + m_1)N_1. \quad (5.18b)$$

A direct calculation shows that the positive equilibrium  $(N_1^*, N_2^*)$  of (5.18) is a stable focus.  $\square$

**THEOREM 5.3** The BE  $E_{BE}$  of (5.14) is locally asymptotically stable if  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$ .

*Proof.* At the BE  $E_{BE}$ , (5.1) and (5.14) have the same characteristic equation. Therefore, just as for (5.1), conditions  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$  guarantee the local asymptotic stability of  $E_{BE}$  for system (5.14).  $\square$

Note that in (5.14),

$$\lim_{t \rightarrow \infty} I_1 = 0 \quad \text{if } \mathcal{R}_{0,1}^T < 1, \quad (5.19)$$

which leads to the same limiting system for (5.14) as for (5.8).

**THEOREM 5.4** The BE  $E_{BE}$  of (5.14) is globally asymptotically stable if  $\mathcal{R}_{0,2} > 1$  and  $\mathcal{R}_{0,1}^T < 1$ .

The proof of Theorem 5.4 is similar to that of Theorem 5.1 and we omit it. Analytical results for the endemic equilibrium  $E_*$  are still an open problem. Taking different initial values for  $I_1(t), I_2(t), N_1(t)$  and  $N_2(t)$  and solving system (5.14) numerically gives Fig. 5, which shows the global asymptotic stability of the unique endemic equilibrium  $E_*$  when  $\mathcal{R}_{0,1}^T > 1$ ,  $\mathcal{R}_0^{(5.14)} > 1$ , independent of the value of

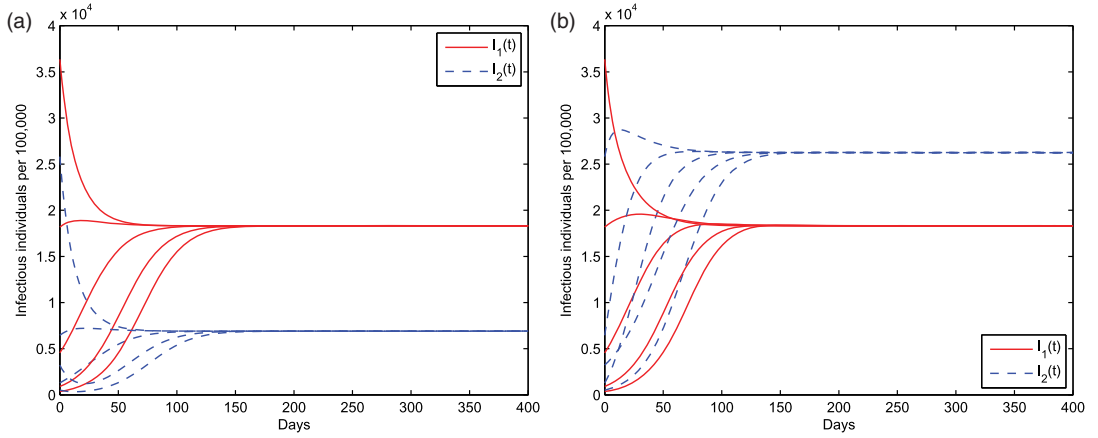


FIG. 5. Time plot showing the trajectories of (5.14) converging to the unique endemic equilibrium  $E_*$ , with parameters as in Table 2 except (a)  $\beta_2 = 0.1$ ,  $\mathcal{R}_{0,2} < 1$ ,  $\mathcal{R}_{0,1}^T > 1$ ,  $\mathcal{R}_0^{(5.14)} > 1$ ; (b)  $\beta_2 = 0.2$ ,  $\mathcal{R}_{0,2} > 1$ ,  $\mathcal{R}_{0,1}^T > 1$ ,  $\mathcal{R}_0^{(5.14)} > 1$ . This seems to indicate that  $E_*$  could be globally asymptotically stable.

$\mathcal{R}_{0,2}$ . As long as the disease becomes endemic in isolated patch 1 and the travel rate  $m_1$  from patch 1 to patch 2 is small, then the disease is endemic in both patches. This indicates that the combination of the infection during transport and the partial (in location) border screening does not help eradicate the disease. Numerical simulations seem to indicate that the endemic equilibrium  $E_*$  is globally asymptotically stable if  $\mathcal{R}_{0,1}^T > 1$  (see Fig. 5) independent of  $\mathcal{R}_{0,2}$ , unlike system (5.1). This implies that infection during transport is more likely to cause a disease outbreak in both patches.

Systems (5.1) and (5.14) have identical basic reproduction numbers, implying that infection during transport does not change the dynamics if infectious individuals only in one patch travel. Moreover, analysis in this section show that if perfect border screening is implemented on patch 2, disease dies out in both patches when  $\mathcal{R}_{0,2} < 1$ ,  $\mathcal{R}_{0,1}^T < 1$ , disease is endemic in patch 2 and dies out in patch 1 when  $\mathcal{R}_{0,2} > 1$ ,  $\mathcal{R}_{0,1}^T < 1$  and disease becomes endemic in both patches when  $\mathcal{R}_{0,2} < 1$ ,  $\mathcal{R}_{0,1}^T > 1$ , whereas disease would not persist in patch 2 in isolation.

Another special case is that a perfect border screening programme is initiated in both patches that results in no travel for infectious individuals. Setting  $m_1 I_1 = m_2 I_2 = 0$ , we derive from model (2.1) the following system of differential equations:

$$\frac{dI_1}{dt} = \frac{\beta_1(N_1 - I_1)I_1}{N_1} - d_1 I_1 - \gamma_1 I_1, \quad (5.20a)$$

$$\frac{dI_2}{dt} = \frac{\beta_2(N_2 - I_2)I_2}{N_2} - d_2 I_2 - \gamma_2 I_2, \quad (5.20b)$$

$$\frac{dN_1}{dt} = b_1 - d_1 N_1 - m_1(N_1 - I_1) + m_2(N_2 - I_2), \quad (5.20c)$$

$$\frac{dN_2}{dt} = b_2 - d_2 N_2 + m_1(N_1 - I_1) - m_2(N_2 - I_2). \quad (5.20d)$$

TABLE 1 *List of reproduction numbers*

Reproduction number	Expression	Corresponding system
$\mathcal{R}_{0,i}$	$\frac{\beta_i}{d_i + \gamma_i}$	Decoupled system without travel
$\mathcal{R}_{0,i}^T$ (modified)	$\frac{\beta_i}{d_i + \gamma_i + m_i}$	Coupled system with travel of infectives
$\mathcal{R}_0^{(2.1)}$	$\frac{P_1 + P_2 + \sqrt{\Delta}}{2 \det(V)}$	System with infection during transport and travel of all infectives
$\mathcal{R}_0^{(3.1)}$	$\frac{\bar{P}_1 + \bar{P}_2 + \sqrt{\bar{\Delta}}}{2 \det(\bar{V})}$	System with no infection during transport and travel of all infectives
$\mathcal{R}_0^{(5.1)}$	$\max\{\mathcal{R}_{0,2}, \mathcal{R}_{0,1}^T\}$	System with no infection during transport and no travel of infectives from patch 2
$\mathcal{R}_0^{(5.14)}$	$\max\{\mathcal{R}_{0,2}, \mathcal{R}_{0,1}^T\}$	System with infection during transport and no travel of infectives from patch 2

The basic reproduction number for (5.20) is  $\mathcal{R}_0^{(5.20)} := \max\{\mathcal{R}_{0,1}, \mathcal{R}_{0,2}\}$ . System (5.20) always has a DFE  $E_0$ ; it has an endemic equilibrium when  $\mathcal{R}_0^{(5.20)} > 1$ . Note that  $\mathcal{R}_0^{(5.20)} \geq \{\mathcal{R}_0^{(5.1)}, \mathcal{R}_0^{(5.14)}\}$ . Then we infer that disease can be endemic in both patches in system (5.20) although in systems (5.1) and (5.14) disease dies out in both patches or is endemic only in one patch.

## 6. Relations between reproduction numbers

For convenience, we list all basic (or modified) reproduction numbers in Table 1. Note that  $\mathcal{R}_{0,i} > \mathcal{R}_{0,i}^T$  for  $i = 1, 2$  and  $\mathcal{R}_0^{(5.1)} = \mathcal{R}_0^{(5.14)}$ . Since  $\mathcal{R}_0^{(2.1)}$  is an increasing function with respect to  $\alpha_1$  and  $\alpha_2$ , and also  $\mathcal{R}_0^{(2.1)} = \mathcal{R}_0^{(3.1)}$  when  $\alpha_1 = \alpha_2 = 0$ , then  $\mathcal{R}_0^{(2.1)} > \mathcal{R}_0^{(3.1)}$ . When  $\mathcal{R}_{0,1}^T \geq \mathcal{R}_{0,2}$ , then  $\mathcal{R}_0^{(3.1)} > \mathcal{R}_0^{(5.1)} = \mathcal{R}_0^{(5.14)}$ . Arino (2009, p. 41) proved that  $\min\{\mathcal{R}_{0,1}, \mathcal{R}_{0,2}\} \leq \mathcal{R}_0^{(3.1)} \leq \max\{\mathcal{R}_{0,1}, \mathcal{R}_{0,2}\}$ . We next compare  $\mathcal{R}_0^{(3.1)}$  with  $\mathcal{R}_{0,i}^T$ .

Equations (3.2) and (3.3) implies  $\mathcal{R}_0^{(3.1)} > \beta_1 \beta_2 (\bar{P}_1 + \bar{P}_2 + |\bar{P}_1 - \bar{P}_2|) / 2\bar{P}_1 \bar{P}_2$ . When  $\bar{P}_1 \geq \bar{P}_2$ , then  $\mathcal{R}_0^{(3.1)} > \beta_1 \beta_2 / \bar{P}_2 = \beta_1 / (d_1 + \gamma_1 + m_1) =: \mathcal{R}_{0,1}^T$ . In addition,  $\bar{P}_1 \geq \bar{P}_2 \Leftrightarrow \beta_1 / (d_1 + \gamma_1 + m_1) \geq \beta_2 / (d_2 + \gamma_2 + m_2) \Leftrightarrow \mathcal{R}_{0,1}^T \geq \mathcal{R}_{0,2}^T$ . Thus, we have  $\mathcal{R}_0^{(3.1)} > \max\{\mathcal{R}_{0,1}^T, \mathcal{R}_{0,2}^T\}$ ; when  $\bar{P}_1 < \bar{P}_2$ , by the similar way, we infer that  $\mathcal{R}_0^{(3.1)} > \max\{\mathcal{R}_{0,1}^T, \mathcal{R}_{0,2}^T\}$ . Thus,  $\mathcal{R}_0^{(3.1)} > \max\{\mathcal{R}_{0,1}^T, \mathcal{R}_{0,2}^T\}$  is always true.

Summarizing the analysis above leads to the following results:

- i.  $\mathcal{R}_{0,i} > \mathcal{R}_{0,i}^T$ ;
- ii.  $\mathcal{R}_0^{(5.1)} = \mathcal{R}_0^{(5.14)}$ ;
- iii.  $\mathcal{R}_0^{(2.1)} > \mathcal{R}_0^{(3.1)} > \max\{\mathcal{R}_{0,1}^T, \mathcal{R}_{0,2}^T\}$ ;
- iv.  $\mathcal{R}_0^{(3.1)} > \mathcal{R}_0^{(5.1)}, \mathcal{R}_0^{(5.14)}$  when  $\mathcal{R}_{0,1}^T \geq \mathcal{R}_{0,2}$ .

TABLE 2 *Parameter values used in simulations*

Parameter	Value	Unit
$b_1$	6	Individuals $\times$ Day $^{-1}$
$b_2$	8	Individuals $\times$ Day $^{-1}$
$\beta_1$	0.3	Day $^{-1}$
$\beta_2$	0.2	Day $^{-1}$
$m_1$	0.045	Day $^{-1}$
$m_2$	0.030	Day $^{-1}$
$\alpha_1$	0.3	Day $^{-1}$
$\alpha_2$	0.6	Day $^{-1}$
$\gamma_1$	1/5	Day $^{-1}$
$\gamma_2$	1/6	Day $^{-1}$
$d_1$	1/(365 $\cdot$ 60)	Day $^{-1}$
$d_2$	1/(365 $\cdot$ 65)	Day $^{-1}$

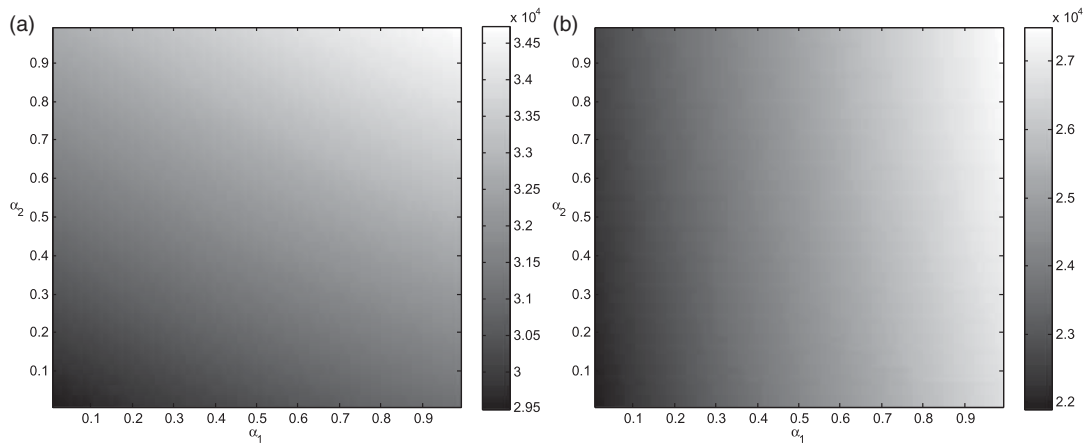


FIG. 6. Effect of  $\alpha_1$  and  $\alpha_2$  on the prevalence in system (2.1), for (a) patch 1 and (b) patch 2. Units are number of infectious individuals per 100,000 people. Other parameters are as in Table 2.

## 7. Numerical results

In this section, we present some numerical simulation results to illustrate and extend analytical results. Parameter values used are summarized in Table 2. Initial values are  $(I_1(0), I_2(0), N_1(0), N_2(0)) = (2000, 4000, 120, 000, 130, 000)$ .

Figure 6 shows the sensitivity of disease prevalence in both patches to variations of  $\alpha_1$  and  $\alpha_2$  when a perfect exit screening programme is implemented in patch 2. It is found that the endemic level in patch 2 ( $I_2$ ) is an increasing function of the disease transmission coefficient for individuals from patch 1,  $\alpha_1$ , but does not depend on  $\alpha_2$ . Indeed, infection during transport from patch 1 to patch 2 contributes to disease prevalence in patch 2, whereas since exit screening is perfect out of patch 2, there is no travel of infectives out of patch 2 and thus no effect of the rate of travel out of patch 2.

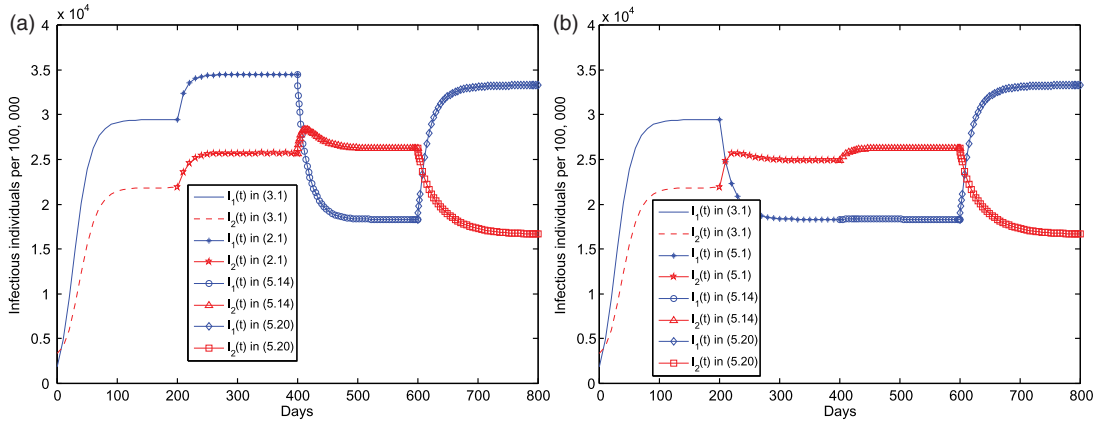


FIG. 7. Comparisons of the effect of different strategies on disease endemicity. In both cases:  $0 \rightarrow 200$  days: no infection during transport nor exit screening;  $400 \rightarrow 600$  days: perfect exit screening in patch 2;  $600 \rightarrow 800$  days: no travel of infectives. Then,  $200 \rightarrow 400$ : (a) infection during transport but no exit screening; (b) no infection during transport.

In Fig. 7, the infection curves in the first 200 days represent the case in which both infectives and susceptibles travel with neither infection during transport nor border screening; infection during transport is incorporated for the second 200-day time period; a perfect border screening programme is then implemented in patch 2 during days 400–600; perfect border screening programmes in both patches follows for the last 200 days. Comparison of infection curves between days  $0 \rightarrow 200$  and days  $200 \rightarrow 400$  indicates that infection during transport increases the endemic level in both patches when the disease is present. Infection curves corresponding to days  $400 \rightarrow 600$  show that a perfect exit screening programme in patch 2 effectively lowers prevalence in patch 1 by forbidding the travel of infectious individuals to patch 1 from patch 2. Furthermore, when a perfect border screening programme is also introduced to patch 1, one can see from the infection curves in days  $600 \rightarrow 800$  that prevalence increases in patch 1 and decreases in patch 2, since perfect border screening in patch 1 interrupts the exportation of infectives to patch 2.

If ventilation is poor in the vehicle or that the vehicle is very crowded, an infective individual might be able to infect a comparatively larger number of travellers. In this case, mass action incidence would more appropriately describe the phenomenon of disease transmission during transport. We assume that there are  $n_i$  vehicles in average per unit time carrying passengers from patch  $i$  to patch  $j$  ( $i, j = 1, 2$  and  $i \neq j$ ). Then, infection during transport per unit time from patch  $i$  to patch  $j$  occurs at the rate

$$n_i \cdot \alpha_i \cdot \frac{m_i S_i}{n_i} \cdot \frac{m_i I_i}{n_i} = \frac{\alpha_i m_i^2 S_i I_i}{n_i}. \quad (7.1)$$

Model (2.1) with mass action incidence during transport then takes the form of the following system of differential equations:

$$\frac{dS_1}{dt} = b_1 - \frac{\beta_1 S_1 I_1}{N_1} + \gamma_1 I_1 - d_1 S_1 - m_1 S_1 + \left(1 - \frac{\alpha_2 m_2 I_2}{n_2}\right) m_2 S_2, \quad (7.2a)$$

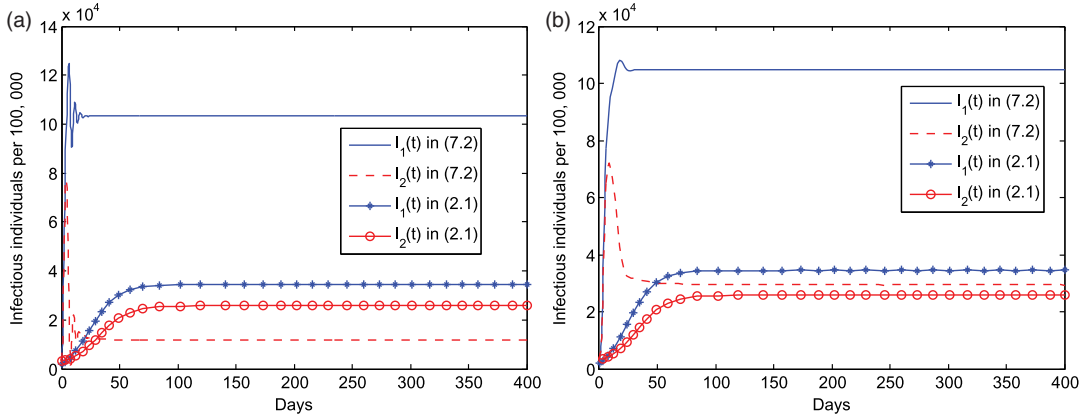


FIG. 8. Comparison of the effect of different incidence functions on disease endemicity: bilinear versus standard type.

$$\frac{dI_1}{dt} = \frac{\beta_1 S_1 I_1}{N_1} - d_1 I_1 - \gamma_1 I_1 - m_1 I_1 + \left(1 + \frac{\alpha_2 m_2 I_2}{n_2}\right) m_2 I_2, \quad (7.2b)$$

$$\frac{dS_2}{dt} = b_2 - \frac{\beta_2 S_2 I_2}{N_2} + \gamma_2 I_2 - d_2 S_2 + \left(1 - \frac{\alpha_1 m_1 I_1}{n_1}\right) m_1 S_1 - m_2 S_2, \quad (7.2c)$$

$$\frac{dI_2}{dt} = \frac{\beta_2 S_2 I_2}{N_2} - d_2 I_2 - \gamma_2 I_2 + \left(1 + \frac{\alpha_1 m_1 I_1}{n_1}\right) m_1 I_1 - m_2 I_2. \quad (7.2d)$$

In Fig. 8(a,b), the values  $n_1 = 40, n_2 = 45$  and  $n_1 = 80, n_2 = 90$  are used, respectively. Figure 8 shows that prevalence in both patches undergoes damped oscillations in the first couple of days; afterwards, the prevalence in both patches for system (7.2) with mass action incidence during transport are higher than those for system (2.1) with standard type incidence during transport, when the number of vehicles travelling per unit time is comparatively large.

Cui *et al.* (2006) showed that the basic reproduction number in the case of subsection 5.2 is identical to that in the case both patches implement a perfect border screening programme (similar to system (5.20)).

## 8. Discussion

In this work, we extend a metapopulation model with infection during transport of Cui *et al.* (2006). The extension concerns the inclusion of different parameters in the two patches and travel-related infection terms.

Cui *et al.* (2006) computed the basic reproduction number  $\mathcal{R}_0$  for the two simplified systems of (5.14) and (5.20), where  $\mathcal{R}_{0,1} = \mathcal{R}_0 = \mathcal{R}_{0,2}$ . However, in our model, we show that the basic reproduction numbers  $\mathcal{R}_{0,1}, \mathcal{R}_{0,2}$  in two isolated patches can be different and  $\mathcal{R}_0^{(5.14)}$  not only depends on  $\mathcal{R}_{0,2}$  but also on the modified basic reproduction number  $\mathcal{R}_{0,1}^T$ . Theorem 5.2 shows that the local stability of the DFE  $E_0$  and the BE  $E_{BE}$  in Cui *et al.* (2006) is in fact global.

Section 5 concerns the effect of border screening. Border screening and travel restrictions have been shown to have little efficiency, both in a deterministic context (Arino *et al.*, 2007) and in a stochastic

one (Scalia Tomba & Wallinga, 2008). In a modern transportation network, this is further compounded by the fact that passengers screened can, sometimes predominantly, simply be connecting through an infected area (Khan *et al.*, 2013). However, another conclusion of Khan *et al.* (2013) is that if border screening is to be implemented, it is best implemented on exit rather than on entry. The situation considered in Section 5 with perfect border screening on exit is, of course, an oversimplification of reality. Performing screening at a level guaranteeing 100% efficacy would require so much time, personnel and resources that it would be infeasible in practice. However, this thought experiment does allow to draw some general principles that would most likely hold in more realistic settings.

Note that the effect of perfect border screening in the model is to reduce the population in the patches. Of course, this would not happen in real life. When using metapopulation models to consider realistic situations, one typically works the following way (see, e.g. Arino & Khan, 2014; Arino & Portet, 2014): since the population in patches, movement rates and death rates are known, the birth rate is chosen so that equilibrium patch population matches the known patch population. Changing the movement rates then simply means that the birth rates have to be adjusted accordingly. This was not done here in the numerics because the example investigated is artificial.

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## Appendix A. Proving global stability using the Poincaré–Bendixson property

DEFINITION A.1 (Li & Wang, 2002) Let  $x \mapsto f(x) \in \mathbb{R}^n$  be a  $C^1$  function for  $x$  in an open set  $\mathcal{D} \in \mathbb{R}^n$ . Consider the differential equation

$$\frac{dx}{dt} = f(x). \quad (\text{A.1})$$

Denote by  $x(t, x_0)$  the solution to (A.1) such that  $x(t, x_0) = x_0$ . A set  $K$  is said to be absorbing in  $\mathcal{D}$  for (A.1) if  $x(t, K_1) \subset K$  for each compact  $K_1 \subset \mathcal{D}$  and  $t$  sufficiently large.

The following two basic assumptions are made:

(H<sub>1</sub>) There exists a compact absorbing set  $K \subset \mathcal{D}$ .

(H<sub>2</sub>) Equation (A.1) has a unique equilibrium  $\bar{x}$  in  $\mathcal{D}$ .

DEFINITION A.2 (Li & Wang, 2002) The differential equation (A.1) is said to be competitive in  $\mathcal{D}$  if, for some diagonal matrix  $H = \text{diag}(\epsilon_1, \dots, \epsilon_n)$ , where each  $\epsilon_i$  is either 1 or  $-1$ ,  $H(\partial f / \partial x)H$  has non-positive off-diagonal elements for all  $x \in \mathcal{D}$ . If  $\mathcal{D}$  is convex, the flow of a competitive system preserves, for  $t < 0$ , the partial ordering in  $\mathbb{R}^n$  defined by the orthant  $K = \{(x_1, \dots, x_n) \in \mathbb{R}^n : \epsilon_i x_i \geq 0, i = 1, \dots, n\}$ .

**THEOREM A.1** (Wang & Mulone, 2003, Chapter 3, Theorem 4.1.) Assume that  $n = 3$  and  $\mathcal{D}$  is convex. Suppose that (A.1) is competitive in  $\mathcal{D}$ . Then it satisfies the Poincaré–Bendixson Property, i.e. any nonempty compact omega limit set of (A.1) that contains no equilibria is a closed orbit.

For higher dimensional systems that satisfy the Poincaré–Bendixson property, Li & Wang (2002) present the following global stability result.

**THEOREM A.2** (Li & Wang, 2002) Assume that

- (1) assumptions  $(H_1)$  and  $(H_2)$  hold;
- (2)  $\bar{x}$  is locally asymptotically stable;
- (3) system (A.1) satisfies the Poincaré–Bendixson Property;
- (4) each periodic orbit of (A.1) in  $\mathcal{D}$  is orbitally asymptotically stable.

Then the unique equilibrium  $\bar{x}$  is globally asymptotically stable in  $\mathcal{D}$ .

### Appendix B. The second additive compound matrix

Let  $A$  be a linear operator on  $\mathbb{R}^n$  and also denote its matrix representation with respect to the standard basis of  $\mathbb{R}^n$ . Let  $\wedge^2 \mathbb{R}^n$  denote the exterior product of  $\mathbb{R}^n$ . Operator  $A$  induces canonically a linear operator  $A^{[2]}$  on  $\wedge^2 \mathbb{R}^n$ : for  $u_1, u_2 \in \mathbb{R}^n$ , define

$$A^{[2]}(u_1 \wedge u_2) =: A(u_1) \wedge u_2 + u_1 \wedge A(u_2)$$

and extend the definition over  $\wedge^2 \mathbb{R}^n$  by linearity. The matrix representation of  $A^{[2]}$  with respect to the canonical basis in  $\wedge^2 \mathbb{R}^n$  is called the second additive compound matrix of  $A$ . This is an  $\binom{n}{2} \times \binom{n}{2}$  matrix and satisfies the property  $(A + B)^{[2]} = A^{[2]} + B^{[2]}$ . In the special case when  $n = 2$ , we have  $A_{2 \times 2}^{[2]} = \text{tr } A$ . In general, each entry of  $A^{[2]}$  is a linear expression of those of  $A$ . For instance, when  $n = 3$ , the second additive compound matrix of  $A = (a_{ij})$  is

$$A^{[2]} = \begin{bmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{bmatrix}.$$

A comprehensive survey and discussion on compound matrices and their relations to differential equations is given in Muldowney (1990).