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# Stochastic and deterministic SIS patch model

T. Yeo <sup>\*†</sup>

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## Abstract

Here, we consider a SIS epidemic model where the individuals are distributed on several distinct patches. We construct a stochastic model and then prove that it converges to a deterministic model as the total population size tends to infinity. Furthermore we show the existence and the global stability of a unique endemic equilibrium provided that the migration rates of susceptible and infectious individuals are equal. Finally we compare the equilibria with those of the homogeneous model, and with those of isolated patches.

Keywords: epidemic patch model . law of large numbers . endemic equilibrium

## 0 Introduction

Early epidemic models were formulated assuming that individuals in the population mix homogeneously [2, 5, 9, 16, 25]. In this consideration, all pairs of individuals in the population have the same probability of coming into contact with each other. But, it is well known that in a large population several groups can be formed due to heterogeneity arising, for example, from social and economic factors. Some people may live in cities while others may live in rural areas. Consequently, demographic and disease parameters may vary for each group, and then the persistence and extinction of infectious diseases in those communities can be different. Furthermore, people may travel between the groups, which leads to the spread of the disease between groups. Then it is clear that spatial heterogeneity, habitat connectivity and rates of movement of individuals play an important role in the outbreak of an infectious

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disease. Those considerations have led to the development of epidemic models that take into account the structure of the population. For this reason, several authors studied heterogeneous epidemic models, by structuring the spatial environment into patches. Fulford et al. [13] developed a Susceptible-Exposed-Infectious (SEI) metapopulation model for the spread of an infectious agent by migration. Salmani & Van den Driessche [19] considered a SEIRS deterministic model in which travel rates were assumed to depend on the disease status. Disease spread in metapopulation models involving discrete patches has been also investigated by Arino et al. [3], Arino & Van den Driessche [4], Jin & Wang [15], Wang & Mulone [23], Wang & Zho [24]. Arino & Van den Driessche developed a general framework for movement of susceptible, exposed, infectious, and recovered individuals (SEIRS model) and define a mobility matrix, an irreducible matrix that defines the spatial arrangement of patches and rates of movement between patches. Wang and colleagues studied uniform persistence and global stability of disease-free and endemic equilibria in Susceptible-Infectious-Susceptible (SIS) metapopulation models. In a similar setting, Allen et al. [1] showed for a SIS deterministic patch model that, while the population is at an endemic level and if infectious individuals travel between the patches but the rate of travel for susceptible individuals approaches zero, then, the endemic equilibrium approaches a disease-free equilibrium.

Deterministic patch models describe the spread of disease under the assumptions of mass action, relying on the law of large numbers. But the most natural way to describe the spread of disease is stochastic. The probabilistic point of view is recent. Let us mention some authors which treated stochastic epidemic models. In 2007, McCormack & Allen [17] studied a SIR and a SIS epidemic model. In this setting both models are deterministic and stochastic. They showed that travel between patches can lead to either disease persistence or extinction in all patches. Considering stochastic model, Clancy [10] proposed a SIR model, and then showed that movement of infectious individuals can decrease the spread of the disease. In the same considerations, Sani et al. [20] introduced a multi-group SIR model for the spread of AIDS. In this study, the authors used Markov process to describe the model. Using an approximating system of the ODEs, they analysed the equilibrium behaviour of the stochastic model. Finally, let's mention that stochastic epidemics in a homogeneous community has been studied recently by Britton & Pardoux [9].

The rest of the paper is organised as follows. In section 1 we introduce a stochastic model on a finite number of patches. Section 2 is devoted to the law of large numbers.

In section 3 we show that the limit deterministic model has an unique endemic equilibrium (EE) which is globally asymptotically stable. Finally we compare this endemic equilibrium with the one of the homogeneous model in the section 4.

## 1 The model

Consider a population consisting of  $N$  individuals, where each individual is located at one of  $j$  geographically distinct patches. Sites (or vertices) represent human communities in which the disease can diffuse and grow. The edges represent links between communities (see figure 1 below). Individuals in that population can be classified according to their ability to transmit the disease to others. Susceptible individuals are those who do not have the disease and who can become infected. Infectious individuals are those who are infected by the disease and can transmit it to susceptible individuals. In this work, attention is given to the SIS model, but the same approach can be developed in the case of the SIRS model and of the SIR model with demography. For any patch  $j$ , the transmission of the disease depends on three factors: the rate of contacts, the probability that a contact is made with a susceptible individual, and the probability that a contact between an infectious and a susceptible individual leads to a successful transmission (see e.g [8, 9]).  $S_j(t)$  (resp.  $I_j(t)$ ) denotes the number of susceptible (resp. infectious) individuals in patch  $j$  at time  $t$ . We formulate a random Markov epidemic model as a Poisson process driven stochastic differential equation (SDE). In what follows the  $P_j$  are mutually independent standard Poisson processes. Infections, healings and migrations of individuals happen according to Poisson processes. In this model

- infections are local;
- when an infectious individual cures, he immediately becomes susceptible again;
- each infectious individual meets other individuals at some rate  $\alpha_j$ . The encounter results in a new infection with probability  $p_j$  if the partner of the encounter is susceptible, which happens with probability  $\frac{S_j(t)}{S_j(t) + I_j(t)}$ , since we assume that individuals in each patch mix homogeneously. Letting  $\lambda_j = \alpha_j p_j$  and summing over the infectious individuals at time  $t$  gives the rate  $\lambda_j \frac{S_j(t)}{S_j(t) + I_j(t)} I_j(t)$  at time  $t$ . Then  $P_j^{inf} \left( \int_0^t \lambda_j \frac{S_j(r) I_j(r)}{S_j(r) + I_j(r)} dr \right)$  counts the number of transitions of type  $S \rightarrow I$  on the patch  $j$  between time 0 and time  $t$ ;

- recovery of an infectious happens at rate  $\gamma_j$ , so  $P_j^{rec} \left( \int_0^t \gamma_j I_j(r) dr \right)$  counts the number of transitions of type I  $\rightarrow$  S on the patch  $j$  between time 0 and time  $t$ .
- The term  $P_{S,j,k}^{mig} \left( \int_0^t \nu_S a_{jk} S_j(r) dr \right)$  counts the number of migrations of susceptible individuals from patch  $j$  to  $k$ , if we assume that each susceptible migrates from  $j$  to  $k$  at rate  $\nu_S a_{jk}$ , and similarly for the compartment I.

Here,  $\nu_S$  and  $\nu_I$  are the diffusion coefficients for susceptible and infectious individuals, respectively.  $a_{ij}$  represents the degree of movement from patch  $i$  into patch  $j$ .

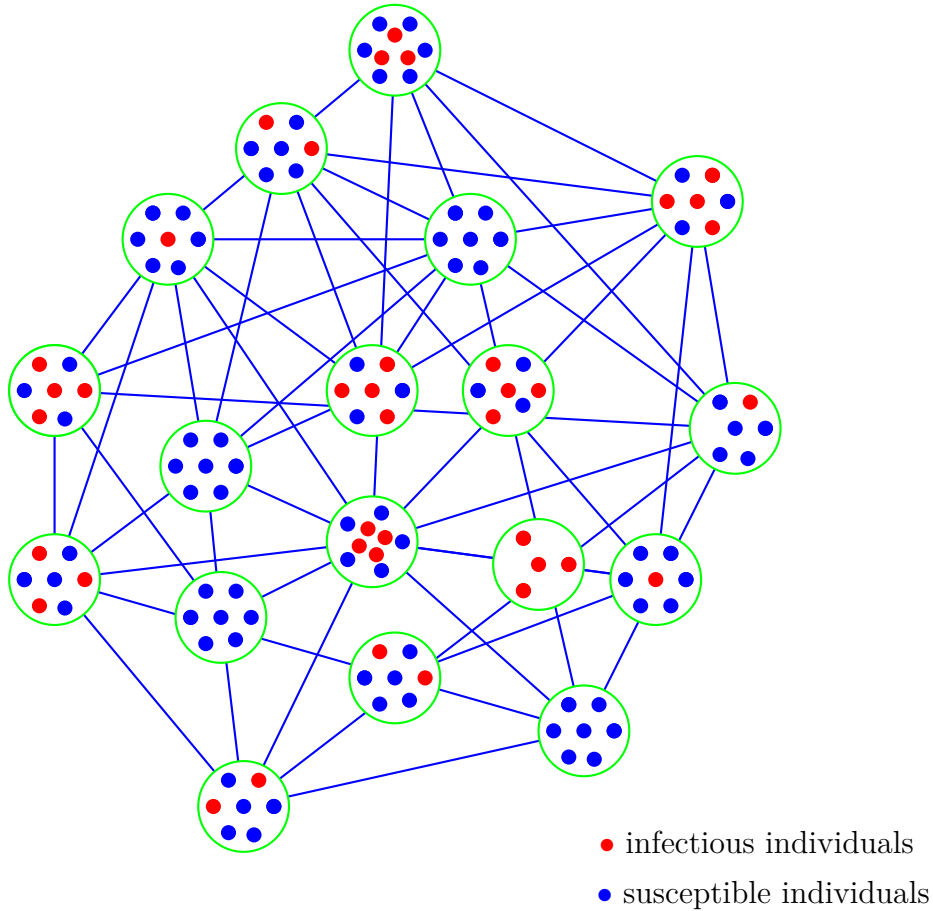


Figure 1: Metapopulation

Then the propagation of the illness can be modeled by the following system of

stochastic differential equations

$$(1.1) \quad \left\{ \begin{array}{l} S_j(t) = S_j(0) - P_j^{inf} \left( \int_0^t \lambda_j \frac{S_j(r)I_j(r)}{S_j(r) + I_j(r)} dr \right) + P_j^{rec} \left( \int_0^t \gamma_j I_j(r) dr \right) \\ \quad - \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{S,j,k}^{mig} \left( \int_0^t \nu_{S a_{jk}} S_j(r) dr \right) + \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{S,k,j}^{mig} \left( \int_0^t \nu_{S a_{kj}} S_k(r) dr \right) \\ I_j(t) = I_j(0) + P_j^{inf} \left( \int_0^t \lambda_j \frac{S_j(r)I_j(r)}{S_j(r) + I_j(r)} dr \right) - P_j^{rec} \left( \int_0^t \gamma_j I_j(r) dr \right) \\ \quad - \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{I,j,k}^{mig} \left( \int_0^t \nu_{I a_{jk}} I_j(r) dr \right) + \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{I,k,j}^{mig} \left( \int_0^t \nu_{I a_{kj}} I_k(r) dr \right) \\ t \in [0, T], \quad j = 1, \dots, \ell. \end{array} \right.$$

In the next section we show that this stochastic model converges to a deterministic epidemic patch model as the total size of population tends to infinity.

## 2 Law of large numbers

We introduce the martingales  $M_j(t) = P_j(t) - t$  and we look instead at the renormalized model by dividing the size of the population in each compartment by  $N$ . Hence by setting

$$S_j^N(t) = \frac{S_j(t)}{N}, \quad I_j^N(t) = \frac{I_j(t)}{N}, \quad S^N(t) = \begin{pmatrix} S_1^N(t) \\ \vdots \\ S_\ell^N(t) \end{pmatrix}, \quad I^N(t) = \begin{pmatrix} I_1^N(t) \\ \vdots \\ I_\ell^N(t) \end{pmatrix}, \text{ and}$$

$\mathcal{Z}^N(t) = \begin{pmatrix} S^N(t) \\ I^N(t) \end{pmatrix}$ , then the stochastic model takes the aggregated form

$$(2.1) \quad \mathcal{Z}^N(t) = \mathcal{Z}^N(0) + \int_0^t b(r, \mathcal{Z}^N(r)) dr + \sum_{j=1}^k \frac{h_j}{N} M_j \left( N \int_0^t \beta_j(\mathcal{Z}^N(r)) dr \right),$$

where  $k$  is the total number of  $P_j$ 's in the system, and

$$(2.2) \quad b(r, \mathcal{Z}^N(r)) = \sum_{j=1}^k h_j \beta_j(\mathcal{Z}^N(r));$$

the vectors  $h_j \in \{-1, 0, 1\}^{2\ell}$  denote the respective jump directions with jump rates

$\beta_j$  . The rates

$$(2.3) \quad \beta.(\mathcal{Z}^N(t)) \in \left\{ \frac{\lambda_j \mathbf{S}_j^N(t) \mathbf{I}_j^N(t)}{\mathbf{S}_j^N(t) + \mathbf{I}_j^N(t)}, \gamma_j \mathbf{I}_j^N(t), \nu_S a_{ij} \mathbf{S}_j^N(t), \right. \\ \left. \nu_I a_{ij} \mathbf{I}_j^N(t), i, j \in \{1, \dots, \ell\} \right\}.$$

Concerning the initial condition, we assume that  $\mathcal{Z}^N(0) = z_N = [Nx]/N$ , for some  $x \in [0, 1]^\ell$ , where  $[Nx]$  is a vector of integers.

We set  $\mathcal{F}_t^N = \sigma\{ \mathcal{Z}_j^N(r), 0 \leq r \leq t, j = 1, \dots, \ell \}$  and we shall assume that the process  $\{\mathcal{Z}^N(t), t \geq 0\}$  is defined on the filtered probability space  $(\Omega, \mathcal{F}, \mathcal{F}_t^N, \mathbb{P})$ . In what follows,  $\|u\|$  denotes the  $L^1$  norm of an  $\ell$ -dimensional vector  $u$ . More precisely,  $\|u\| = \sum_{j=1}^{\ell} |u_j|$ . We shall say that a vector  $u$  is nonnegative (resp. positive) if all its elements are nonnegative (resp. positive), in which case we will write  $u \geq 0$  (resp.  $u > 0$ ). The following theorem shows that the solution of the stochastic model (2.1) converges a.s. locally uniformly in  $t$  to the solution of a deterministic model, as the total population size  $N$  tends to infinity.

**Theorem 2.1 [Law of Large Numbers]**

Let  $\mathcal{Z}^N$  denote the solution of the SDEs (2.1) and  $z$  the unique solution of the system of ordinary differential equations  $\frac{dz}{dt}(t) = b(t, z(t)), z(0) = x$ .

Let us fix an arbitrary  $T > 0$ . Then  $\sup_{0 \leq t \leq T} \|\mathcal{Z}^N(t) - z(t)\| \rightarrow 0$  a.s., as  $N \rightarrow +\infty$ .

Note that the solution  $z(t) = (\mathbf{S}_1(t), \mathbf{I}_1(t), \mathbf{S}_2(t), \mathbf{I}_2(t), \dots, \mathbf{S}_\ell(t), \mathbf{I}_\ell(t))^T$  of the deterministic model satisfy

$$(2.4) \quad \begin{cases} \frac{d\mathbf{S}_j}{dt}(t) = -\lambda_j \frac{\mathbf{S}_j(t) \mathbf{I}_j(t)}{\mathbf{S}_j(t) + \mathbf{I}_j(t)} + \gamma_j \mathbf{I}_j(t) + \nu_S \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{kj} \mathbf{S}_k(t) - a_{jk} \mathbf{S}_j(t)) \\ \frac{d\mathbf{I}_j}{dt}(t) = \lambda_j \frac{\mathbf{S}_j(t) \mathbf{I}_j(t)}{\mathbf{S}_j(t) + \mathbf{I}_j(t)} - \gamma_j \mathbf{I}_j(t) + \nu_I \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{kj} \mathbf{I}_k(t) - a_{jk} \mathbf{I}_j(t)) \\ \mathbf{S}_j(0) \geq 0, \mathbf{I}_j(0) \geq 0 \\ j = 1, \dots, \ell. \end{cases}$$

$\mathbf{S}_j(t)$  (resp.  $\mathbf{I}_j(t)$ ) is the proportion of the total susceptible (resp. infectious) population which is localized on the site  $j$  at time  $t$ .

**Theorem 2.1** ensures that, as the population size  $N$  becomes large, the proportion of susceptible and infectious individuals at each patch is well approximated, on

any bounded interval  $[0, T]$  by the solution of the ODEs (2.4), provided the scaled process starts close to an initial value of the ODEs.

Theorem 2.1 is a special case of Theorem 2.2.7 of Britton & Pardoux [9], where the proof written for the homogeneous model covers our situation as well. One of the earliest references on this convergence result is Ethier & Kurtz [12] (chapter 11, Theorem 2.1). Thus, we do not give details and refer the reader to those papers for a complete proof. We briefly sketch the idea of the proof. First note that  $0 \leq \mathcal{Z}^N(t) \leq 1$ , for all  $t \in [0, T]$ . By using the law of large numbers for Poisson processes and the second Dini Theorem, it follows that

$$\sup_{0 \leq t \leq T} \left| \sum_{j=1}^{\ell} \frac{h_j}{N} M_j \left( N \int_0^t \beta_j(\mathcal{Z}^N(r)) dr \right) \right| \xrightarrow{a.s.} 0.$$

Next, it is not hard to see that  $b(t, z)$  is a globally Lipschitz function of  $z$ , locally uniformly in  $t$ . From this fact, it follows that, for all  $t \in [0, T]$ ,

$$(2.5) \quad \begin{aligned} \left\| \mathcal{Z}^N(t) - z(t) \right\| &\leq \left\| z_N - x \right\| + \left\| \sum_{j=1}^{\ell} \frac{h_j}{N} M_j \left( N \int_0^t \beta_j(\mathcal{Z}^N(r)) dr \right) \right\| \\ &\quad + C \int_0^t \left\| \mathcal{Z}^N(r) - z(r) \right\| dr, \end{aligned}$$

where  $C$  is the Lipschitz constant of  $b$ . Finally, the result follows from Gronwall's Lemma and the fact that the two first terms in the right-hand side of (2.5) tend to zero as  $N \rightarrow \infty$ .

In the following section, we study the equilibria of this system of ODEs.

### 3 Equilibria of the ODEs and their stability

In this section, we consider properties of the disease free equilibrium (DFE) and the endemic equilibrium (EE), including its existence, uniqueness and stability. Throughout this section, we assume that the connectivity matrix  $\mathbb{A} = (a_{ij})_{\substack{1 \leq i \leq \ell \\ 1 \leq j \leq \ell}}$  is irreducible and symmetric. This irreducibility assumption implies that the patches cannot be separated into two disjoint subsets such that there is no migration of individuals from one subset to the other. That is, for any  $j, k \in \{1, \dots, \ell\}$ ,  $j \neq k$ , there exists  $s \geq 2$ , a sequence  $j_1, j_2, \dots, j_s \in \{1, \dots, \ell\}$  such that  $j_1 = j$ ,  $j_s = k$  and  $a_{j_i j_{i+1}} \neq 0$ ,  $\forall i \in \{1, \dots, s-1\}$ . We shall say that a matrix  $M = (m_{ij})_{\substack{1 \leq i \leq \ell \\ 1 \leq j \leq \ell}}$  is



nonnegative (resp. positive) if all its elements are nonnegative (resp. positive), in with case we will write  $M \geq 0$  (resp.  $M > 0$ ). In what follows, we set  $\mathbf{N}_j(t) = \mathbf{S}_j(t) + \mathbf{I}_j(t)$ . Let us mention that the system of ODEs (2.4) obtained in [Theorem 2.1](#) has been studied by Allen et al. [1], where the authors studied the asymptotic profiles of the steady states. First, using the irreducibility of the connectivity matrix, they show

**Lemma 3.1 (Allen [1]) [Existence and uniqueness of the DFE]**

*The system (2.4) has a unique disease-free equilibrium which is given by*

$$\hat{z} := \left( \hat{\mathbf{S}}_1, \hat{\mathbf{I}}_1, \hat{\mathbf{S}}_2, \hat{\mathbf{I}}_2, \dots, \hat{\mathbf{S}}_\ell, \hat{\mathbf{I}}_\ell \right) = \left( \frac{1}{\ell}, 0, \frac{1}{\ell}, 0, \dots, \frac{1}{\ell}, 0 \right).$$

The DFE always exists, an important question is whether an outbreak of the disease can occur when the population initially contains a small number of infected individuals. This question may be addressed using stability analysis of the DFE. In fact if  $\mathcal{R}_0$ , the basic reproduction number (the expected number of secondary cases produced, in a completely susceptible population, by a typical infective individual) is less than 1, the DFE is globally asymptotically stable. That is, the trajectory of the ODEs which starts close to the DFE will be attracted towards the DFE. That is the content of the next lemma. Before, we use the next-generation matrix approach of Van den Driessche & Watmough [11] to compute  $\mathcal{R}_0$ . Define the matrices

$$A = \text{diag}(\gamma_j)_{1 \leq j \leq \ell}, \quad F = \text{diag}(\lambda_j)_{1 \leq j \leq \ell} \quad \text{and} \quad D = (d_{ij})_{1 \leq i, j \leq \ell}$$

$$\text{with } d_{ij} = \begin{cases} -\sum_{\substack{k=1 \\ k \neq i}}^{\ell} a_{ik} & \text{if } i = j, \\ a_{ij} & \text{if } i \neq j. \end{cases}$$

A direct application of the result of the above reference yields the following Proposition.

**Proposition 3.1** *The basic reproduction number for (2.4) is the spectral radius of the next-generation matrix:*

$$\mathcal{R}_0 = \rho(-FV^{-1}),$$

where  $V = \nu_I D - A$ .

We have also the

**Lemma 3.2 (Allen et al. [1]) [Stability of the DFE]**

If  $\mathcal{R}_0 < 1$ , then the disease-free equilibrium  $\hat{z}$  is globally asymptotically stable, that is

$$z(t) \longrightarrow \hat{z}, \quad \text{as } t \rightarrow \infty.$$

We will show at the end of this section that, under a specific condition, the disease-free equilibrium is globally asymptotically stable if  $\mathcal{R}_0 = 1$ . The existence and uniqueness of the EE for (2.4) is shown in [1] under the assumption that  $\mathcal{R}_0 > 1$ . In that work, the authors were not able to prove the stability of the EE, but conjectured that this EE attracts all solutions whose initial conditions have a nonzero proportion of infectious (and numerical simulations suggest that this is indeed the case). Here, we employ the approach in Bichara et al. [7] to prove the global stability of the EE, under the assumption that infectious and susceptible individuals have the same diffusion rate  $\nu_S = \nu_I := \nu$ .

Assuming that  $\nu_S = \nu_I := \nu$ , then the system given by (2.4) is equivalent to

$$(3.1) \quad \begin{cases} \frac{d\mathbf{N}_j(t)}{dt} = \nu \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{kj}\mathbf{N}_k(t) - a_{jk}\mathbf{N}_j(t)) \\ \frac{d\mathbf{I}_j(t)}{dt} = \lambda_j \left(1 - \frac{\mathbf{I}_j(t)}{\mathbf{N}_j(t)}\right) \mathbf{I}_j(t) - \gamma_j \mathbf{I}_j(t) + \nu \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{kj}\mathbf{I}_k(t) - a_{jk}\mathbf{I}_j(t)) \\ j = 1, \dots, \ell, \end{cases}$$

which can be written in the form

$$(3.2) \quad \begin{cases} \frac{d\mathbf{N}(t)}{dt} = \nu \mathbf{D}\mathbf{N}(t) \\ \frac{d\mathbf{I}(t)}{dt} = \nu \mathbf{D}\mathbf{I}(t) - \mathbf{A}\mathbf{I}(t) + \left(\mathbb{I}_\ell - \text{diag}(\mathbf{N}_j^{-1}(t))\text{diag}(\mathbf{I}(t))\right) \mathbf{F}\mathbf{I}(t), \end{cases}$$

where  $\mathbf{N} = (\mathbf{N}_1, \dots, \mathbf{N}_\ell)^\top$ ,  $\mathbf{I} = (\mathbf{I}_1, \dots, \mathbf{I}_\ell)^\top$  and  $\mathbb{I}_\ell$  is the identity matrix with dimension  $\ell \times \ell$ . Note that  $\left(\mathbb{I}_\ell - \text{diag}(\mathbf{N}_j^{-1}(t))\text{diag}(\mathbf{I}(t))\right) \mathbf{F}\mathbf{I}(t)$  is the vector of new infections,  $\nu \mathbf{D}\mathbf{I}(t)$  is the vector of migrations of infectious individuals and  $\mathbf{A}\mathbf{I}(t)$  is the vector of transitions of individuals from the compartment  $\mathbf{I}$  to the compartment  $\mathbf{S}$ .

**Lemma 3.3** *The system  $\frac{d\mathbf{N}(t)}{dt} = \nu \mathbf{D}\mathbf{N}(t)$  has a unique global asymptotically stable equilibrium.*

**Proof :** Let  $Q$  be the matrix such that  $q_{ij} = a_{ji}$  for  $i \neq j$  and  $q_{jj} = -\sum_{k=1}^{\ell} a_{kj}$ .

Hence the system  $\frac{d\mathbf{N}(t)}{dt} = \nu D\mathbf{N}(t)$  is equivalent to  $\frac{d\mathbf{N}^T(t)}{dt} = \nu \mathbf{N}^T(t)Q$ . Notice that  $Q$  is the infinitesimal generator of an irreducible Markov process with state space  $\left\{ \frac{n}{\ell}, n = 0, 1, \dots, \ell \right\}^\ell$ . By using Theorem 5.1 of Pardoux [18], it follows that there exists a unique strictly positive equilibrium  $\mathbf{N}^*$  which solves the equation  $(\mathbf{N}^*)^T Q = 0$ . Since the state space is finite, the Markov process associated to the infinitesimal generator  $Q$  is recurrent, and then the global asymptotic stability of  $\mathbf{N}^*$  is guaranteed by the Theorem 6.5 of the above reference. □

Next we treat the existence and stability of the endemic equilibrium for the system given by (3.2). Notice that (3.2) is of triangular form, and hence the theory of asymptotically autonomous systems for triangular systems (Vidyasagar [22]) guarantees that the asymptotic stability of its equilibrium is equivalent to that of the system

$$\frac{d\mathbf{I}(t)}{dt} = \nu D\mathbf{I}(t) - A\mathbf{I}(t) + \left( \mathbb{I}_\ell - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{I}(t)) \right) F\mathbf{I}(t),$$

where  $(\mathbf{N}_1^*, \dots, \mathbf{N}_\ell^*)$  is the unique asymptotically stable equilibrium defined in Lemma 3.3. The Jacobian matrices of  $\left( \mathbb{I}_\ell - \text{diag}(\mathbf{N}_j^{-1}(t)) \text{diag}(\mathbf{I}(t)) \right) F\mathbf{I}(t)$  and  $\nu D\mathbf{I}(t) - A\mathbf{I}(t)$ , respectively, at the disease free equilibrium are  $F$  and  $V$ . For the convenience of the reader, we recall the following result.

**Theorem 3.1 (Vidyasagar[22], Theorem 3.1 and 3.4)**

*Let  $f$  and  $g$  be two functions of class  $\mathcal{C}^1$ . Consider the following system*

$$(3.3) \quad \begin{cases} \dot{x} = f(x) \\ \dot{y} = g(x, y) \\ \text{with an equilibrium point } (x^*, y^*), \text{ i.e.,} \\ f(x^*) = 0 \text{ and } g(x^*, y^*) = 0. \end{cases} \quad x \in \mathbb{R}^n, y \in \mathbb{R}^m$$

*If  $x^*$  is globally asymptotically stable (GAS) in  $\mathbb{R}^n$  for the system  $\dot{x} = f(x)$ , and if  $y^*$  is GAS in  $\mathbb{R}^m$  for the system  $\dot{y} = g(x^*, y)$ , then  $(x^*, y^*)$  is locally asymptotically stable for (3.3). Moreover, if all the trajectories of (3.3) are forward bounded, then  $(x^*, y^*)$  is GAS for (3.3).*

We shall need below the

**Theorem 3.2 (Hirsch [14], Theorem 6.1)**

*Let  $F$  be a  $\mathcal{C}^1$  vector field in  $\mathbb{R}^q$ , whose flow  $\phi$  preserves  $\mathbb{R}_+^q$  for  $t \geq 0$  and is strongly*

monotone in  $\mathbb{R}_+^q$ . Assume that the origin is an equilibrium and that all trajectories in  $\mathbb{R}_+^q$  are bounded. If the matrix-valued map  $\mathcal{D}F : \mathbb{R}^q \rightarrow \mathbb{R}^q \times \mathbb{R}^q$  is strictly decreasing, in the sense that

$$\text{if } x < y \text{ then } \mathcal{D}F(x) > \mathcal{D}F(y),$$

then either all trajectories in  $\mathbb{R}_+^q \setminus \{0\}$  tend to the origin, or there is a unique equilibrium  $p^*$ , ( $p^* \gg 0$ ) in the interior of  $\mathbb{R}_+^q$  and all trajectories in  $\mathbb{R}_+^q \setminus \{0\}$  tend to  $p^*$ .

Now, we are in a position to prove the main result of this section.

**Theorem 3.3 [Existence and stability of the EE]**

Assume that  $\nu_I = \nu_S := \nu$  and  $\mathcal{R}_0 > 1$ . Then the system (2.4) has a unique endemic equilibrium  $z^* = (\mathbf{S}_1^*, \mathbf{I}_1^*, \mathbf{S}_2^*, \mathbf{I}_2^* \cdots, \mathbf{S}_\ell^*, \mathbf{I}_\ell^*)$ , which is globally asymptotically stable.

**Proof :** It follows from Theorem 3.1 that it is sufficient to study the stability of the reduced system

$$\frac{d\mathbf{I}(t)}{dt} = \nu \mathbf{D} \mathbf{I}(t) - \mathbf{A} \mathbf{I}(t) + \left( \mathbb{I}_\ell - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{I}(t)) \right) \mathbf{F} \mathbf{I}(t).$$

Note that the set defined by

$$K = \left\{ \left( (u_1, \dots, u_\ell), (v_1, \dots, v_\ell) \right) \in \mathbb{R}_+^\ell \times \mathbb{R}_+^\ell : 0 \leq v_i \leq u_i, 1 \leq i \leq \ell \text{ and } \sum_{i=1}^\ell u_i = 1 \right\}$$

is a compact positively invariant for the system (3.1). Define

$$\mathcal{L}(\mathbf{I}) = (\mathbf{F} + \mathbf{V})\mathbf{I} - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{I})\mathbf{F}\mathbf{I}.$$

The derivative  $\mathcal{D}\mathcal{L}(\mathbf{I})$  is

$$\begin{aligned} \mathcal{D}\mathcal{L}(\mathbf{I}) &= (\mathbf{F} + \mathbf{V}) - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{I})\mathbf{F} - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{F}\mathbf{I}) \\ &= \nu \mathbf{D} - \mathbf{A} + \mathbf{F} - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{I})\mathbf{F} - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{F}\mathbf{I}). \end{aligned}$$

Notice that  $\mathcal{D}\mathcal{L}(\mathbf{I})$  is an irreducible Metzler matrix. Since  $\mathbf{F} \geq 0$  and  $\mathbf{F} \neq 0$ ,  $\mathcal{D}\mathcal{L}$  is clearly strictly decreasing with respect of  $\mathbf{I}$ . Applying Theorem 3.2, we deduce that either all trajectories in  $K$  tend to the origin, or there is a unique equilibrium in the interior of  $K$  and all trajectories in  $K \setminus ([0, \infty)^\ell \times \{0\}^\ell)$  tend to this equilibrium.

We introduce the stability modulus  $\alpha(M)$  of a matrix  $M$ , which is the largest real

part of the elements of the spectrum  $\text{Spec}(M)$  of  $M$  :

$$\alpha(M) = \max_{\delta \in \text{Spec}(M)} \text{Re}(\delta).$$

From Theorem 3.13 of Varga [21] (chapter 3),  $\mathcal{R}_0 > 1$  is equivalent to  $\alpha(F + V) > 0$ , and the disease free equilibrium is unstable in this case. It then follows from Theorem 3.2 that there exists a unique attracting endemic equilibrium  $\mathbf{I}^* \neq 0$ , which satisfies

$$(3.4) \quad (\nu D - A + F)\mathbf{I}^* - \text{diag}(1/\mathbf{N}_j^*)\text{diag}(\mathbf{I}^*)F\mathbf{I}^* = 0.$$

Since  $F$  is a non-negative matrix and  $\mathbf{I}^* \neq 0$ , by using (3.4), it follows that

$$(3.5) \quad \mathcal{DL}(\mathbf{I}^*)\mathbf{I}^* = -\text{diag}(1/\mathbf{N}_j^*)\text{diag}(F\mathbf{I}^*)\mathbf{I}^* < 0.$$

Using the fact that  $\mathcal{DL}(\mathbf{I}^*)$  is a Metzler matrix, (3.5) implies that it is stable (Berman & Plemmons [6]: criterion  $I_{28}$  of Theorem 6.2.3). The stability modulus then satisfies  $\alpha(\mathcal{DL}(\mathbf{I}^*)) < 0$ . This proves the local asymptotic stability of  $\mathbf{I}^*$ . Since the attractivity of  $\mathbf{I}^*$  is guaranteed by Hirsh's Theorem 3.2, we conclude that the endemic equilibrium  $\mathbf{I}^*$  is globally asymptotically stable if  $\mathcal{R}_0 > 1$ .

□

Let us mention that, under the assumption  $\nu_I = \nu_S := \nu$ , the DFE is globally asymptotically stable when  $\mathcal{R}_0 = 1$ . Indeed,  $\mathcal{R}_0 = 1$  is equivalent to  $\alpha(F + V) = 0$ . But since  $F + V$  is an irreducible Metzler matrix, there exists a positive vector  $v$  such that  $(F + V)^T v = 0$ . Let us consider the Lyapunov function  $L(\mathbf{I}) = \langle v | \mathbf{I} \rangle$ . The derivative of this function is

$$\begin{aligned} \dot{L}(\mathbf{I}) &= \langle v | \dot{\mathbf{I}} \rangle \\ &= \langle v | F + V - \text{diag}(1/\mathbf{N}_j^*)\text{diag}(\mathbf{I}(t))F\mathbf{I}(t) \rangle \\ &= -\langle v | \text{diag}(1/\mathbf{N}_j^*)\text{diag}(\mathbf{I}(t))F\mathbf{I}(t) \rangle \\ &\leq 0. \end{aligned}$$

Moreover,  $\dot{L}(\mathbf{I}) = 0$  only at the DFE. Hence the DFE is GAS if  $\mathcal{R}_0 = 1$ .

## 4 Comparison of the equilibria: connected patches model, homogeneous model, isolated patches

We now consider the deterministic model in an homogeneous community:

$$(4.1) \quad \begin{cases} \frac{d\mathbf{S}}{dt}(t) = -\lambda\mathbf{S}(t)\mathbf{I}(t) + \gamma\mathbf{I}(t) \\ \frac{d\mathbf{I}}{dt}(t) = \lambda\mathbf{S}(t)\mathbf{I}(t) - \gamma\mathbf{I}(t), \end{cases}$$

where  $\lambda$  (resp.  $\gamma$ ) is the rate of the disease transmission (resp. recovery).

The endemic equilibrium of the system of ODEs (4.1) is  $\mathfrak{z}^* = \left(\frac{\gamma}{\lambda}, 1 - \frac{\gamma}{\lambda}\right)$ .

We wish to compare this EE with the one of the deterministic heterogeneous model.

- First, if the disease transmission and recovery rates are the same on all patches, that is for all  $j = 1, \dots, \ell$ ,  $\lambda_j = \lambda$  and  $\gamma_j = \gamma$ , then the EE of the ODEs (2.4) is  $z^* = \left(\frac{\gamma}{\ell\lambda}, \frac{1}{\ell}\left(1 - \frac{\gamma}{\lambda}\right), \dots, \frac{\gamma}{\ell\lambda}, \frac{1}{\ell}\left(1 - \frac{\gamma}{\lambda}\right)\right)$ . In this case, we note that the proportion of the infectious subpopulation in the homogeneous model is equally distributed between all patches.

- We now look at the case where the patches have different disease transmission and recovery rates. In this case it is difficult to obtain the EE, even for a small number of patches. But it can be found relatively simply using any numerical solver when the state space is small. Here, we consider the case of two patches and use the solver "Wolfram Alpha" to compute the EE.

In the below table we give the values of the infectious subpopulation in each patch at the equilibrium for several values of the parameters. We take  $\gamma_1 = \gamma_2 = 1$  and consider three cases. First  $\lambda_1 = 1.5$ ,  $\lambda_2 = 2$ . In this case when the patches are isolated, the value of the infectious subpopulation in patch 1 and patch 2 are  $\mathbf{I}_1^* \approx 0.333$  and  $\mathbf{I}_2^* \approx 0.5$ , respectively. Secondly  $\lambda_1 = 3$ ,  $\lambda_2 = 2.5$ , and  $\mathbf{I}_1^* \approx 0.666$  and  $\mathbf{I}_2^* \approx 0.600$  in isolated patches. Finally, in the case  $\lambda_1 = 1.5$ ,  $\lambda_2 = 1.2$ , we have  $\mathbf{I}_1^* \approx 0.333$  and  $\mathbf{I}_2^* \approx 0.166$ .

| $\lambda_1$ | $\lambda_2$ | $\gamma_1$ | $\gamma_2$ | $\nu_I$ | $\nu_S$ | $\left( \frac{\mathbf{I}_1^*}{\mathbf{S}_1^* + \mathbf{I}_1^*}, \frac{\mathbf{I}_2^*}{\mathbf{S}_2^* + \mathbf{I}_2^*} \right)$ |
|-------------|-------------|------------|------------|---------|---------|---|
| 1.5         | 2           | 1          | 1          | 0.0001  | 0.0001  | (0.332 , 0.507)   |
| 1.5         | 2           | 1          | 1          | 0.0001  | 0.0005  | (0.334, 0.497)  |
| 1.5         | 2           | 1          | 1          | 0.001   | 0.0001  | (0.333, 0.497)  |
| 1.5         | 2           | 1          | 1          | 0.0001  | 0.001   | (0.332, 0.497)  |
|             |             |            |            |         |         |   |
| 3           | 2.5         | 1          | 1          | 0.0001  | 0.0001  | (0.667 , 0.598)   |
| 3           | 2.5         | 1          | 1          | 0.0007  | 0.0001  | (0.666 , 0.599)   |
| 3           | 2.5         | 1          | 1          | 0.001   | 0.0001  | (0.666 , 0.598)   |
| 3           | 2.5         | 1          | 1          | 0.0001  | 0.001   | (0.666 , 0.598)   |
|             |             |            |            |         |         |   |
| 1.5         | 1.2         | 01         | 1          | 0.0001  | 0.0001  | (0.332 , 0.165)   |
| 1.5         | 1.2         | 1          | 1          | 0.0001  | 0.0009  | (0.332 , 0.165)   |
| 1.5         | 1.2         | 1          | 1          | 0.001   | 0.0001  | (0.333 , 0.165)   |
| 1.5         | 1.2         | 1          | 1          | 0.0001  | 0.008   | (0.332 , 0.165)   |

Table 1: proportion of  $\mathbf{I}_1^*$  and  $\mathbf{I}_2^*$  when patches are connected

In the above table, we have the proportions of the infectious subpopulation in each patch at the equilibrium, for some values of the diffusion coefficients. We observe that those proportions are very close to those when patches are isolated.

We have shown that the stochastic model is well approximated by a deterministic patch model. If  $\mathcal{R}_0 > 1$ , the system of ODEs (2.4) has a unique endemic equilibrium which is globally asymptotically stable. Moreover considering the case of two patches, it appears that in the heterogeneity case, the final size of the epidemic in each patch is close to that of isolated patches.

In a future work, we will study the fluctuations of the stochastic model around its deterministic law of large numbers limit.

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