



HAL
open science

Stochastic and deterministic SIS patch model

Ténan Yeo

► **To cite this version:**

Ténan Yeo. Stochastic and deterministic SIS patch model. *Discrete and Continuous Dynamical Systems - Series B*, 2021, 26 (12), pp.6173. 10.3934/dcdsb.2021012 . hal-03663217

HAL Id: hal-03663217

<https://amu.hal.science/hal-03663217>

Submitted on 10 May 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

STOCHASTIC AND DETERMINISTIC SIS PATCH MODEL

TÉNAN YEO

Aix Marseille Univ, Marseille, France
CNRS, Centrale Marseille, I2M, Marseille, France
Univ. F. H. Boigny, UFR-MI, Abidjan, Côte d'Ivoire

(Communicated by Hao Wang)

ABSTRACT. Here, we consider an 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. We next study the equilibria of the deterministic model. Our main contribution is a stability result of the endemic equilibrium in the case $\mathcal{R}_0 > 1$. Finally we compare the equilibria with those of the homogeneous model, and with those of isolated patches.

1. Introduction. Early epidemic models were formulated assuming that individuals in the population mix homogeneously [2, 3, 7, 12, 18]. 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 disease. Those considerations motivate the development of multipatch epidemic models.

In this paper, we consider a deterministic multipatch SIS model, similar to the one studied by Allen et al. [1]. We first show that it is the law of large numbers limit, as the size N of the population tends to infinity, of a continuous time finite population stochastic model. We next study the stability of the equilibria. Our main result is a stability result of the endemic equilibrium in the case $\mathcal{R}_0 > 1$. We also compare the equilibria with those of the homogeneous model, and with those of isolated patches.

Several authors have exploited Lyapunov functions in order to study the stability of the endemic equilibrium in various types of models, see e.g. [10, 15, 13, 19]. The same method does not seem to be applicable in our case.

The paper is organised as follows. In section 2, we introduce both deterministic and stochastic model on a finite number of patches. Section 3 is devoted to the

2020 *Mathematics Subject Classification.* Primary: 60J75, 34D23; Secondary: 60J20.

Key words and phrases. epidemic patch model, law of large numbers, endemic equilibrium.

law of large numbers. In section 4, we prove that the endemic equilibrium (EE) of the deterministic model is globally asymptotically stable. Finally we compare this endemic equilibrium with the one of the homogeneous model and with the one when patches are isolated, in section 5.

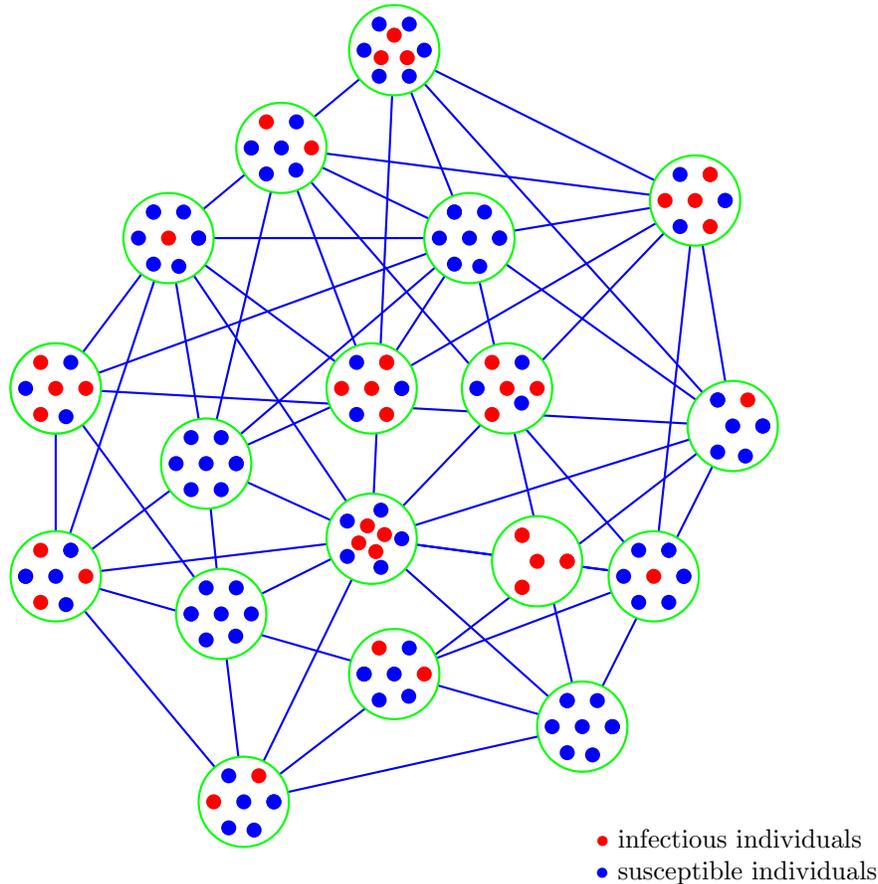


Figure 1: Metapopulation

2. The models.

2.1. The deterministic 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 above). 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 [4, 7]). $\mathbf{S}_j(t)$ (resp. $\mathbf{I}_j(t)$) denotes the number of susceptible (resp. infectious) individuals in patch j at time t . Here, we assume that

- infections are local;
- when an infectious individual cures, he immediately becomes susceptible again;
- each infectious individual meets other individuals at some rate α_j . The encounter results in a new infection with probability p_j if the partner of the encounter is susceptible, which happens with probability $\frac{\mathbf{S}_j(t)}{\mathbf{S}_j(t) + \mathbf{I}_j(t)}$, 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 $\lambda_j \frac{\mathbf{S}_j(t)}{\mathbf{S}_j(t) + \mathbf{I}_j(t)} \mathbf{I}_j(t)$ at time t ;
- each infectious on site j recovers at rate γ_j , so the total recovery rate at time t is $\gamma_j \mathbf{I}_j(t)$;
- each susceptible migrates from j to k at rate $\nu_S a_{jk}$, and similarly for the compartment \mathbf{I} .

ν_S and ν_I are the diffusion coefficients for susceptible and infectious individuals, respectively. a_{ij} represents the degree of movement from patch i into patch j .

Disease spreads between individuals in the same patch by contact between infectious and susceptible individuals, and spread from one patch to another by migration of infectious individuals. Hence the system of ordinary differential equations (ODEs) for the evolution of the number of susceptible and infectious individuals at each patch j is as follows

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

By an obvious homogeneity property of the system of ODEs (1), both the numbers of individuals in each compartment and in each patch, and the proportions of individuals in each compartment and in each patch obey the system (1). In the sequel, we shall use the notation $z(t) = (\mathbf{S}_1(t), \mathbf{I}_1(t), \mathbf{S}_2(t), \mathbf{I}_2(t), \dots, \mathbf{S}_\ell(t), \mathbf{I}_\ell(t))^T$ to denote the proportions.

2.2. The stochastic model. The deterministic models are based on the hypothesis of a population of large size. When it is not the case, the interactions between the individuals are not uniform but possess an intrinsic random character. We are going to expose now a probabilistic version of the previous model. For each given patch, Poisson processes count the number of new infections, removal and migrations between the patches during the time. So the propagation of the illness can be

modeled by the following system of stochastic equations

$$\left\{ \begin{array}{l} S_j(t) = S_j(0) - P_j^{inf} \left(\int_0^t \lambda_j \frac{S_j(r)I_j(r)}{S_j(r) + I_j(r)} dr \right) + P_j^{rec} \left(\int_0^t \gamma_j I_j(r) dr \right) \\ \quad - \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{S,j,k}^{mig} \left(\int_0^t \nu_{S a_{jk}} S_j(r) dr \right) + \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{S,k,j}^{mig} \left(\int_0^t \nu_{S a_{kj}} S_k(r) dr \right) \\ I_j(t) = I_j(0) + P_j^{inf} \left(\int_0^t \lambda_j \frac{S_j(r)I_j(r)}{S_j(r) + I_j(r)} dr \right) - P_j^{rec} \left(\int_0^t \gamma_j I_j(r) dr \right) \\ \quad - \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{I,j,k}^{mig} \left(\int_0^t \nu_{I a_{jk}} I_j(r) dr \right) + \sum_{\substack{k=1 \\ k \neq j}}^{\ell} P_{I,k,j}^{mig} \left(\int_0^t \nu_{I a_{kj}} I_k(r) dr \right) \\ t \in [0, T], \quad j = 1, \dots, \ell. \end{array} \right. \quad (2)$$

where all the P_j 's are mutually independent standard Poisson processes. Here infections, healings and migrations of individuals happen according to Poisson processes whose rates are given by the respective integrands. In system (2)

- $P_j^{inf} \left(\int_0^t \lambda_j \frac{S_j(r)I_j(r)}{S_j(r) + I_j(r)} dr \right)$ 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 γ_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 .

Let mention that such Markov process has been used by Ethier & Kurtz [9] to model both the logistic growth of a population, epidemics and chemical reactions.

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

3. 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

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

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

$$\mathcal{Z}^N(t) = \mathcal{Z}^N(0) + \int_0^t b(r, \mathcal{Z}^N(r)) dr + \sum_{j=1}^k \frac{h_j}{N} M_j \left(N \int_0^t \beta_j(\mathcal{Z}^N(r)) dr \right), \quad (3)$$

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

$$b(r, \mathcal{Z}^N(r)) = \sum_{j=1}^k h_j \beta_j(\mathcal{Z}^N(r)); \quad (4)$$

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

$$\beta.(\mathcal{Z}^N(t)) \in \left\{ \frac{\lambda_j \mathbf{S}_j^N(t) \mathbf{I}_j^N(t)}{\mathbf{S}_j^N(t) + \mathbf{I}_j^N(t)}, \gamma_j \mathbf{I}_j^N(t), \nu_S a_{ij} \mathbf{S}_j^N(t), \right. \\ \left. \nu_I a_{ij} \mathbf{I}_j^N(t), i, j \in \{1, \dots, \ell\} \right\}. \quad (5)$$

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 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. $u > 0$).

Note that the system (1) can be written in the following form

$$\frac{dz}{dt}(t) = b(t, z(t)), \quad x := z(0) \geq 0,$$

where

$$b(t, z(t)) = \begin{pmatrix} -\frac{\lambda_1 \mathbf{S}_1(t) \mathbf{I}_1(t)}{\mathbf{S}_1(t) + \mathbf{I}_1(t)} + \gamma_1 \mathbf{I}_1(t) + \nu_S \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{k1} \mathbf{S}_k(t) - a_{1k} \mathbf{S}_1(t)) \\ \frac{\lambda_1 \mathbf{S}_1(t) \mathbf{I}_1(t)}{\mathbf{S}_1(t) + \mathbf{I}_1(t)} - \gamma_1 \mathbf{I}_1(t) + \nu_I \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{k1} \mathbf{I}_k(t) - a_{1k} \mathbf{I}_1(t)) \\ \vdots \\ -\frac{\lambda_\ell \mathbf{S}_\ell(t) \mathbf{I}_\ell(t)}{\mathbf{S}_\ell(t) + \mathbf{I}_\ell(t)} + \gamma_\ell \mathbf{I}_\ell(t) + \nu_S \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{k\ell} \mathbf{S}_k(t) - a_{\ell k} \mathbf{S}_\ell(t)) \\ \frac{\lambda_\ell \mathbf{S}_\ell(t) \mathbf{I}_\ell(t)}{\mathbf{S}_\ell(t) + \mathbf{I}_\ell(t)} - \gamma_\ell \mathbf{I}_\ell(t) + \nu_I \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{k\ell} \mathbf{I}_k(t) - a_{\ell k} \mathbf{I}_\ell(t)) \end{pmatrix}.$$

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 ℓ -dimensional vector u . More precisely,

$\|u\| = \sum_{j=1}^{\ell} |u_j|$. The following theorem shows that the solution of the stochastic

model (3) converges a.s. locally uniformly in t to the solution of a deterministic model, as the total population size N tends to infinity.

Theorem 3.1. [Law of Large Numbers]

Let \mathcal{Z}^N denote the solution of the SDEs (3) and z the unique solution of the ODEs (1). 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 $N \rightarrow \infty$.

Theorem 3.1 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 (1), provided the scaled process starts close to an initial value of the ODEs.

Theorem 3.1 is a special case of Theorem 2.2.7 of Britton & Pardoux [7], 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 [9] (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 $t \in [0, T]$,

$$\begin{aligned} \left\| \mathcal{Z}^N(t) - z(t) \right\| &\leq \left\| z_N - x \right\| + \left\| \sum_{j=1}^{\ell} \frac{h_j}{N} M_j \left(N \int_0^t \beta_j(\mathcal{Z}^N(r)) dr \right) \right\| \\ &+ C \int_0^t \left\| \mathcal{Z}^N(r) - z(r) \right\| dr, \end{aligned} \quad (6)$$

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 (6) tend to zero as $N \rightarrow \infty$.

In the following section, we study the equilibria of the ODEs (1).

4. 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.

The 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 which case we will write $M \geq 0$ (resp. $M > 0$). In what follows, \mathbf{N}_j denotes the total population size in patch j , i.e. $\mathbf{N}_j = \mathbf{S}_j + \mathbf{I}_j$.

Let us mention that the system of ODEs (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 4.1 (Allen [1]). **[Existence and uniqueness of the DFE]**

The system (1) 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 [8] to compute \mathcal{R}_0 . Define the matrices

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

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

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

Proposition 1. *The basic reproduction number for the system (1) is the spectral radius of the next-generation matrix:*

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

where $V = \nu_I D - A$.

We have also the

Lemma 4.2 (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 the system (1) is shown in Allen et al. [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. [6] to prove the globally 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 sytem given by (1) is equivalent to

$$\begin{cases} \frac{d\mathbf{N}_j(t)}{dt} = \nu \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{kj}\mathbf{N}_k(t) - a_{jk}\mathbf{N}_j(t)) \\ \frac{d\mathbf{I}_j(t)}{dt} = \lambda_j \left(1 - \frac{\mathbf{I}_j(t)}{\mathbf{N}_j(t)}\right) \mathbf{I}_j(t) - \gamma_j \mathbf{I}_j(t) + \nu \sum_{\substack{k=1 \\ k \neq j}}^{\ell} (a_{kj}\mathbf{I}_k(t) - a_{jk}\mathbf{I}_j(t)) \\ j = 1, \dots, \ell, \end{cases} \quad (7)$$

which can be written in the form

$$\begin{cases} \frac{d\mathbf{N}(t)}{dt} = \nu D \mathbf{N}(t) \\ \frac{d\mathbf{I}(t)}{dt} = \nu D \mathbf{I}(t) - A \mathbf{I}(t) + \left(\mathbb{I}_{\ell} - \text{diag}(\mathbf{N}_j^{-1}(t)) \text{diag}(\mathbf{I}(t)) \right) B \mathbf{I}(t), \end{cases} \quad (8)$$

where $\mathbf{N} = (\mathbf{N}_1, \dots, \mathbf{N}_\ell)^\top$, $\mathbf{I} = (\mathbf{I}_1, \dots, \mathbf{I}_\ell)^\top$ and \mathbb{I}_ℓ is the identity matrix with dimension $\ell \times \ell$. Note that $(\mathbb{I}_\ell - \text{diag}(\mathbf{N}_j^{-1}(t))\text{diag}(\mathbf{I}(t)))\mathbf{B}\mathbf{I}(t)$ 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 4.3. *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 $\frac{d\mathbf{N}(t)}{dt} = \nu\mathbf{D}\mathbf{N}(t)$ is equivalent to $\frac{d\mathbf{N}^\top(t)}{dt} = \nu\mathbf{N}^\top(t)Q$. Notice that Q is the infinitesimal generator of an irreducible Markov process with state space $\{\frac{n}{\ell}, n = 0, 1, \dots, \ell\}^\ell$. By using Theorem 5.1 of Pardoux [14], it follows that there exists a unique strictly positive equilibrium \mathbf{N}^* which solves the equation $(\mathbf{N}^*)^\top Q = 0$. 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. \square

Next we treat the existence and stability of the endemic equilibrium for the system given by the system (8). Notice that the system (8) is of triangular form, and hence the theory of asymptotically autonomous systems for triangular systems (Vidyasagar [17]) guarantees that the asymptotic stability of its equilibrium is equivalent to that of the system

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

where $(\mathbf{N}_1^*, \dots, \mathbf{N}_\ell^*)$ is the unique asymptotically stable equilibrium defined in Lemma 4.3. The Jacobian matrices of $(\mathbb{I}_\ell - \text{diag}(\mathbf{N}_j^{-1}(t))\text{diag}(\mathbf{I}(t)))\mathbf{B}\mathbf{I}(t)$ and $\nu\mathbf{D}\mathbf{I}(t) - \mathbf{A}\mathbf{I}(t)$, respectively, at the disease free equilibrium are \mathbf{B} and \mathbf{V} . For the convenience of the reader, we recall the following result.

Theorem 4.4 (Vidyasagar[17], Theorem 3.1 and 3.4).

Let f and g be two functions of class C^1 . Consider the following system

$$\begin{cases} \dot{x} = f(x) \\ \dot{y} = g(x, y) \\ \text{with an equilibrium point } (x^*, y^*), \text{ i.e.,} \\ f(x^*) = 0 \text{ and } g(x^*, y^*) = 0. \end{cases} \quad (9)$$

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 (9). Moreover, if all the trajectories of (9) are forward bounded, then (x^*, y^*) is GAS for (9).*

We shall need below the

Theorem 4.5 (Hirsch [11], Theorem 6.1). *Let F be a C^1 vector field in \mathbb{R}^q , whose flow ϕ 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 4.6. [Existence and stability of the EE]

Assume that $\nu_I = \nu_S := \nu$ and $\mathcal{R}_0 > 1$. Then the system (1) 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 4.4 that it is sufficient to study the stability of the reduced system

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

Note that the set defined by

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

is a compact positively invariant for the system (7). Define

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

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

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

Notice that $\mathcal{D}\mathcal{L}(\mathbf{I})$ is an irreducible Metzler matrix. Since $\mathbf{B} \geq 0$ and $\mathbf{B} \neq 0$, $\mathcal{D}\mathcal{L}$ is clearly strictly decreasing with respect of \mathbf{I} . Applying Theorem 4.5, 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 [16] (chapter 3), $\mathcal{R}_0 > 1$ is equivalent to $\alpha(\mathbf{B} + \mathbf{V}) > 0$, and the disease free equilibrium is unstable in this case. It then follows from Theorem 4.5 that there exists a unique attracting endemic equilibrium $\mathbf{I}^* \neq 0$, which satisfies

$$(\nu \mathbf{D} - \mathbf{A} + \mathbf{B})\mathbf{I}^* - \text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{I}^*) \mathbf{B} \mathbf{I}^* = 0. \quad (10)$$

Since \mathbf{B} is a non-negative matrix and $\mathbf{I}^* \neq 0$, by using (10), it follows that

$$\mathcal{D}\mathcal{L}(\mathbf{I}^*)\mathbf{I}^* = -\text{diag}(1/\mathbf{N}_j^*) \text{diag}(\mathbf{B} \mathbf{I}^*) \mathbf{I}^* < 0. \quad (11)$$

Using the fact that $\mathcal{D}\mathcal{L}(\mathbf{I}^*)$ is a Metzler matrix, (11) implies that it is stable (Berman & Plemmons [5]: criterion I_{28} of Theorem 6.2.3). The stability modulus then satisfies $\alpha(\mathcal{D}\mathcal{L}(\mathbf{I}^*)) < 0$. This proves the local asymptotic stability of \mathbf{I}^* . Since the attractivity of \mathbf{I}^* is guaranteed by Hirsh's Theorem 4.5, we conclude that the endemic equilibrium \mathbf{I}^* is globally asymptotically stable if $\mathcal{R}_0 > 1$. \square

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 $\alpha(\mathbf{B} + \mathbf{V}) = 0$. But, since $\mathbf{B} + \mathbf{V}$ is an irreducible Metzler matrix, there exists a positive vector v such that $(\mathbf{B} + \mathbf{V})^T v = 0$. Let us consider the Lyapunov function $\mathbf{L}(\mathbf{I}) = \langle v | \mathbf{I} \rangle$. The derivative of this function is

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

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

5. Comparison of the equilibria: connected patches model, homogeneous model, isolated patches. We now consider the deterministic model in an homogeneous community:

$$\begin{cases} \frac{d\mathbf{S}}{dt}(t) = -\lambda \mathbf{S}(t) \mathbf{I}(t) + \gamma \mathbf{I}(t) \\ \frac{d\mathbf{I}}{dt}(t) = \lambda \mathbf{S}(t) \mathbf{I}(t) - \gamma \mathbf{I}(t), \end{cases} \quad (12)$$

where λ (resp. γ) is the rate of the disease transmission (resp. recovery).

The endemic equilibrium of the system of ODEs (12) 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 (1) 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 an explicit expression for 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 Table 1, we give the proportion 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 $\mathbf{I}_2^* \approx 0.166$.

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 ODE (1) has a unique endemic equilibrium which is globally asymptotically stable. Moreover, considering the case of two

λ_1	λ_2	γ_1	γ_2	ν_I	ν_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	0.1	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

patches, it appears that in the heterogeneity case, the final size of the epidemic in each patch is close to that of isolated patches, when the diffusion coefficients are small.

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

Acknowledgments. I wish to thank the referee for his valuable advices and suggestions which helped me greatly in the writing of the revised version. I also would like to thank Prof. Etienne Pardoux for careful reading and plentiful suggestions that greatly improved the paper.

Funding. This research was supported by a thesis scholarship from the government of Ivory Coast, and a salary as instructor at University of Aix–Marseille.

REFERENCES

- [1] L. J. S. Allen, B. M. Bolker, Y. Lou and A. L. Nevai, [Asymptotic profiles of the steady states for an SIS epidemic patch model](#), *SIAM Journal on Applied Mathematics*, **67** (2007), 1283–1309.
- [2] H. Andersson and T. Britton, *Stochastic Epidemic Models and their Statistical Analysis*, Vol. **151**, Springer-Verlag, New York, 2000.
- [3] N. T. J. Bailey, *The Mathematical Theory of Infectious Diseases and its Applications*, 2nd edition, Griffin, London, 1975.
- [4] M. Begon, M. Bennett, R. G. Bowers, N. P. French, S. M. Hazel and J. Turner, [A clarification of transmission terms in host-microparasite models: Numbers, densities and areas](#), *Epidemiology & Infection*, **129** (2002), 147–153.
- [5] J. A. Berman and R. J. Plemmons, *Nonnegative Matrices in the Mathematical Sciences*, Society for Industrial and Applied Mathematics, 1994.
- [6] D. Bichara, Y. Kang, C. Castillo-Chavez, R. Horan and C. Perrings, [SIS and SIR epidemic models under virtual dispersal](#), *Bulletin of Mathematical Biology*, **77** (2015), 2004–2034.

- [7] T. Britton and E. Pardoux, [Stochastic epidemic models with inference](#), in *Lecture Notes in Math.* (eds. F. Ball, C. Larédo, D. Sirl and V. C. Tran), **2255**, Springer, (2019), 1–120.
- [8] P. V. D. Driessche and J. Watmough, [Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission](#), *Mathematical Biosciences*, **180** (2002), 29–48.
- [9] S. N. Ethier and T. G. Kurtz, *Markov Processes: Characterization and Convergence*, J. Wiley & Sons, 1986.
- [10] A. Fall et al., [Epidemiological models and Lyapunov functions](#), *Mathematical Modelling of Natural Phenomena*, **2** (2007), 62–83.
- [11] M. W. Hirsch, [The dynamical systems approach to differential equations](#), *Bulletin of the American Mathematical Society*, **11** (1984), 1–64.
- [12] W. O. Kermack and A. G. McKendrick, A contribution to the mathematical theory of epidemics, *Proceedings of the Royal Society of London. Series A*, **115** (1927), 700–721.
- [13] L. Michael and Z. Shuai, Global stability of an epidemic model in a patchy environment, *Canadian Applied Mathematics Quarterly*, **17** (2009), 175–187.
- [14] E. Pardoux, *Markov Processes and Applications: Algorithms, Networks, Genome and Finance*, J. Wiley & Sons, 2008.
- [15] J. Rebaza, [Global stability of a multipatch disease epidemics model](#), *Chaos, Solitons & Fractals*, **120** (2019), 56–61.
- [16] R. Varga, *Matrix Iterative Analysis*, Englewood Cliffs, NJ: Prentice-Hall, Inc, 1962.
- [17] M. Vidyasagar, [Decomposition techniques for large-scale systems with nonadditive interactions: stability and stabilizability](#), *IEEE Transactions Automatic Control*, **25** (1980), 773–779.
- [18] G. H. Weiss and M. Dishon, [On the asymptotic behavior of the stochastic and deterministic models of an epidemic](#), *Mathematical Biosciences*, **11** (1971), 261–265.
- [19] S. Zhisheng and P. Driessche, [Global stability of infectious disease models using Lyapunov functions](#), *SIAM Journal on Applied Mathematics*, **73** (2013), 1513–1532.

Received September 2020; revised November 2020.

E-mail address: yeo.tenan@yahoo.fr