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DYNAMICS OF A MULTI-PATCH AND MULTI-GROUP METAPOPULATION EPIDEMIC MODEL IN PERIODIC ENVIRONMENT

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Abstract. In this paper, we analyse a class of SEIRS multi-patch and multi-group Lagrangian epidemic models in periodic environment. The associated basic reproduction rate, $\mathcal{R}_0(m,n,\mathbb{P})$ has been computed and we have shown that it is the thresholds between the persistence and the extinction of the infection. Precisely, if $\mathcal{R}_0(m,n,\mathbb{P}) \leq 1$, the periodic infection-free equilibrium is globally stable whereas the model exhibits positive periodic solutions when $\mathcal{R}_0(m,n,\mathbb{P}) > 1$. In order to evaluate the effects of periodic environment in the infection transmission, we derive the basic reproduction rate, $[\mathcal{R}_0](m,n,\mathbb{P})$ of the autonomous model and the result of their comparison is explored numerically through a specific case of two patches.

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1. Introduction

1.1. **Concept of heterogeneity.** In the mathematical modelling of infection, classical epidemic models assume that the entire population lives in the same area and well mixing [1–3]. That assumption is in general unrealistic since individuals in the population may be living on isolated islands or in different countries while travelling from one location to another. This spatial heterogeneity can affects the propagation of the infection, [4–7]. To understand the precise impact of heterogeneity on disease dynamics, we need to develop models that account for this heterogeneity. To achieve that, multiple approaches have been used. Mainly, space can be incorporated to epidemic models as variable in

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discrete or continuous form. However, one of the most popular discrete space modelling approaches is the metapopulation, [8,9]. In the recent literature, other discrete methods, including epidemic spatial networks, cellular automata, and lattice epidemic models have been developed to explore the effect of heterogeneity, [10,11].

1.2. **Metapopulation models.** In ecology, we define a metapopulation as a group of populations of the same species that live in spatially isolated areas but interact in some level. Metapopulations occurs when different populations live in fragmented habitats but are connected through migration, [12–14]. Thus, the isolated areas occupied by each population is called patches. In epidemiology, patches may be cities, countries, islands, or other geographical autonomous regions. We define the migration as physical movement of individuals from one area to another. It can be in long-term or in short term. In short term, individuals visit another location, spent a period time before returning in their home patches, [15–17]. In long-term, individuals move in another patch an settle there. These two types of movement are modelled differently and are respectively called Lagrangian and Eulerian movement. The corresponding models in mathematical epidemiology are respectively, Lagrangian and Eulerian metapopulation models, [18–21].

1.3. **Background.** While the concept of modelling epidemiological heterogeneity within a population goes back to Kermack McKendrick [22], the Lagrangian and Eulerian approaches were implemented by Okubo et *al*, [23]. Recently, the use of Lagrangian approach has been explored by several authors, [24–26]. Particularly, Bichara et *al*, [27] have introduced a new general Lagrangian model in order to evaluate how group heterogeneity, patch heterogeneity, mobility patterns and behaviour each alter or mitigate the disease dynamics. In fact, they consider a typical SEIRS epidemic infection which evolutes in a population structured in m groups interacting in n patches. For each group $i(i = 1, \ldots, m)$, we denote by $N_i = S_i + E_i + I_i + R_i$ its population; where S_i, E_i, I_i and R_i represent the susceptible, latent, infectious and recovered populations. Hence, each epidemiological class of such group spend a proportion of times in patch $j(j = 1, \ldots, n)$. Thus, during their short visit, individuals can infect or can be infected. With all necessary assumptions, their model reads as follows:

$$\begin{cases} \dfrac{dS_i}{dt} = \Lambda_i - \displaystyle\sum_{j=1}^{n} \beta_j q_{ij} S_i \frac{\sum_{k=1}^{n} p_{kj} I_k}{\sum_{k=1}^{m} (q_{kj} S_k + m_{kj} E_k + p_{kj} I_k + r_{kj} R_k)} - \mu_i S_i + \gamma_i R_i, \\ \dfrac{dE_i}{dt} = \displaystyle\sum_{j=1}^{n} \beta_j q_{ij} S_i \frac{\sum_{k=1}^{n} p_{kj} I_k}{\sum_{k=1}^{m} (q_{kj} S_k + m_{kj} E_k + p_{kj} I_k + r_{kj} R_k)} - (\mu_i + \tau_i) E_i, \\ \dfrac{dI_i}{dt} = \tau_i E_i - (\mu_i + \alpha_i + \eta) I_i, \\ \dfrac{dR_i}{dt} = \alpha_i I_i - (\mu_i + \gamma_i) R_i(t), \end{cases} \tag{1}$$

where each group i, receives a constant recruitment, Λ_i of susceptible individuals. Any individual leaves the group with natural death rate μ_i and disease induced death rate η_i. While γ_i represents the per capita loss rate of immunity, τ_i denotes the per capita rate at which latent individuals becomes infectious. α_i is the per capita recovery rate of group i. The constant values, $q_{ij}, m_{ij}, p_{ij}, r_{ij}$ represent the proportion of time spent by susceptible, latent, infectious and recovery individuals of group i in patch j during their short visit. β_j is the instantaneous risk of infectious in patch j.

1.4. **Outline of the paper: effects of climate on parameters.** Climate change is increasingly recognized as a key factor influencing the emergence, evolution, and transmission dynamics of infectious diseases. Changes in temperature and precipitation, as well as extreme weather events, are redefining ecological niches, altering the behaviour of hosts and vectors, and disrupting interactions between humans and their environment, thereby creating new selective pressures that favour the adaptation and spread of pathogens, [28,29]. These environmental disturbances can expand the geographic range of disease vectors, alter the seasonality of transmission, and increase the frequency and severity of epidemics, [30,31]. To better understand these complex dynamics, it is essential to combine knowledge from climatology, molecular epidemiology, clinical microbiology, and genomic. Particular attention is given to vulnerable populations, to the accurate modelling of health risks at different spatial and temporal scales, and to the integration of climate variables into predictive frameworks for early warning and response, [32,33].

Thus, realistic mathematical models must integrate climate change in order to capture the real dynamics of an epidemic. Models with climate perturbations give rise to non autonomous differential systems which mathematical analysis are often very complex, [34]. In the literature, there exist more and more contributions that integrate climate change on mathematical models, [35–37]. However, in our knowledge, very few contributions have been proposed for Lagrangian metapopulation models. Hence, in the paper, we propose a rigorous dynamical analysis of a Lagrangian metapopulation models in periodic environment. To address this, we evaluate the model developed by Derdei Bichara *et al.* in periodic environment, that is all biological parameters of such model are assumed to be periodic functions (depend on climate variations). So the model reads as follows:

$$\begin{cases} \dfrac{dS_i}{dt} = \Lambda_i(t) - \displaystyle\sum_{j=1}^{n} \beta_j(t) q_{ij} S_i \frac{\sum_{k=1}^{n} p_{kj} I_k}{\sum_{k=1}^{m} (q_{kj} S_k + m_{kj} E_k + p_{kj} I_k + r_{kj} R_k)} - \mu_i(t) S_i + \gamma_i(t) R_i, \\ \dfrac{dE_i}{dt} = \displaystyle\sum_{j=1}^{n} \beta_j(t) q_{ij} S_i \frac{\sum_{k=1}^{n} p_{kj} I_k}{\sum_{k=1}^{m} (q_{kj} S_k + m_{kj} E_k + p_{kj} I_k + r_{kj} R_k)} - (\mu_i(t) + \tau_i(t)) E_i, \\ \dfrac{dI_i}{dt} = \tau_i(t) E_i - (\mu_i(t) + \alpha_i(t) + \eta(t)) I_i, \\ \dfrac{dR_i}{dt} = \alpha_i(t) I_i - (\mu_i(t) + \gamma_i(t)) R_i(t), \end{cases} \tag{2}$$

where $\Lambda_i, \mu_i, \gamma_i, \alpha_i, \tau_i, \eta_i, \beta_i$ are continuous positive ω-periodic functions.

The remaining parts of the paper are organized as follows. In the next section, we focus on some important basic properties of the model. In section 3, we compute the periodic basic reproduction, $\mathcal{R}_0(m, n, \mathbb{P})$ of the model and derive the average basic reproduction number, $[\mathcal{R}_0](m, n, \mathbb{P})$ of the associated autonomous model. Moreover, we prove that qualitative theory of our model depend strongly on the periodic basic reproduction rate. In Section 4, we show that $\mathcal{R}_0(m, n, \mathbb{P})$ is the threshold dynamics between the persistence and extinction on the infection. Further, some numerical simulations are performed in Section 5 to illustrate our theoretical results. In Section 6, we conclude and give some future investigations.

2. Basic Properties

2.1. **Notations.** For convenience, we denote by $\frac{dy_i}{dt} = \dot{y}_i$, and we consider the following notations for state variables:

$$\mathbf{S} = [S_1, S_2, \ldots, S_m]^T, \mathbf{E} = [E_1, E_2, \ldots, E_m]^T, \ \mathbf{I} = [I_1, I_2, \ldots, I_m]^T, \ \mathbf{R} = [R_1, R_2, \ldots, R_m]^T,$$

and the following notations for parameters:

$$\Lambda(t) = [\Lambda_1(t), \Lambda_2(t), \ldots, \Lambda_m(t)]^T, \ \mu(t) = [\mu_1(t), \mu_2(t), \ldots, \mu_m(t)]^T, \ \tau(t) = [\tau_1(t), \tau_2(t), \ldots, \tau_m(t)]^T,$$
$$\gamma(t) = [\gamma_1(t), \gamma_2(t), \ldots, \gamma_m(t)]^T, \ \mathcal{B}(t) = [\beta_1(t), \beta_2(t), \ldots, \beta_m(t)]^T, \ \eta(t) = [\eta_1(t), \eta_2(t), \ldots, \eta_m(t)]^T,$$
$$\alpha(t) = [\alpha_1(t), \alpha_2(t), \ldots, \alpha_m(t)]^T, \ \delta(t) = \mu(t) + \alpha(t) + \eta(t), \ \theta(t) = \mu(t) + \gamma(t), \ \varrho(t) = \mu(t) + \tau(t).$$

In addition, we denote by $\mathbb{Q} = (q_{ij})$, $\mathbb{P} = (p_{ij})$, $\mathbb{M} = (m_{ij})$ and $\mathbb{L} = (r_{ij})$ the residence time matrices of susceptible, latent, infectious and recovery individuals, respectively. From the above notations, we rewrite the model as follows:

$$\begin{cases} \dot{\mathbf{S}} = \Lambda(t) - \text{diag}(\mathbf{S})\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\mathbf{S} + \mathbb{M}^T\mathbf{E} + \mathbb{P}^T\mathbf{I} + \mathbb{L}^T\mathbf{R})\mathbb{P}^T\mathbf{I} - \text{diag}(\mu(t))\mathbf{S} + \text{diag}(\gamma(t))\mathbf{R}, \\ \dot{\mathbf{E}} = \text{diag}(\mathbf{S})\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\mathbf{S} + \mathbb{M}^T\mathbf{E} + \mathbb{P}^T\mathbf{I} + \mathbb{L}^T\mathbf{R})\mathbb{P}^T\mathbf{I} - \text{diag}(\varrho(t))\mathbf{I}, \\ \dot{\mathbf{I}} = \text{diag}(\tau(t))\mathbf{E} - \text{diag}(\delta(t))\mathbf{I}, \\ \dot{\mathbf{R}} = \text{diag}(\alpha(t))\mathbf{I} - \text{diag}(\theta(t))\mathbf{R}. \end{cases} \tag{3}$$

2.2. **Existence and characterization of solutions.**

Definition 1. *Let* $\mathbf{A} = (A_1, A_2, \ldots, A_m)$ *and* $\mathbf{B} = (B_1, B_2, \ldots, B_m)$ *be two vectors. Then, we have the following definitions:*

(i) $\mathbf{A} \circ \mathbf{B} = (A_1B_1, A_2B_2, \ldots, A_mB_m)$.
(ii) $\frac{\mathbf{1}}{\mathbf{A}} = \left(\frac{1}{A_1}, \frac{1}{A_2}, \ldots, \frac{1}{A_m}\right)$ *if* $\mathbf{A} \gg 0$.

Theorem 2.1. *For any positive initial condition $\phi = (\mathbf{S^0}, \mathbf{E^0}, \mathbf{I^0}, \mathbf{R^0})$, system (3) admits a unique positive solution through ϕ. In addition, all solutions are ultimately bounded.*

Proof. Let $\mathbf{u} = (\mathbf{S}, \mathbf{E}, \mathbf{I}, \mathbf{R})^T$ and $\mathbf{F}(t, \mathbf{u})$ be the Right-Hand side (RHS) of system (3). Then, the Cauchy's problem reads as follows:

$$\begin{cases} \dot{\mathbf{u}}(t) = \mathbf{F}(t, \mathbf{u}), \\ \mathbf{u}(0) = \phi. \end{cases} \tag{4}$$

Thus, for all positive initial condition, $\phi \in \mathbb{R}^{4m}_+$, $\mathbf{F}(t, \phi)$ is continuous and lipschitzian in ϕ in each compact set $\mathbb{R}_+ \times \mathbb{R}^{4m}$. Hence, there exists a unique solution of (3) through $(0, \phi)$. Moreover, from the whole population dynamics, we have:

$$\dot{\mathbf{H}}(t) = \Lambda(t) - \mu(t) \circ \mathbf{H}(t) - \eta(t) \circ \mathbf{I}(t) \leq \Lambda(t) - \mu(t) \circ \mathbf{H}(t). \tag{5}$$

Thus, thanks to the comparison principle [[38], Proposition 1.1, Theorem 1.2], it yields that:

$$\limsup_{t \to \infty} (\mathbf{H}(t) - \tilde{\mathbf{S}}(t)) \leq \mathbf{0} \tag{6}$$

where

$$\tilde{\mathbf{S}}(t) = e^{-\int_0^t \mu(s)ds} \circ \left(\tilde{\mathbf{S}}(0) + \int_0^t \Lambda(s) \circ e^{\int_0^s \mu(\xi)d\xi} ds \right) \tag{7}$$

with

$$\tilde{\mathbf{S}}(0) = \frac{\int_0^\omega \Lambda(s) \circ e^{\int_0^s \mu(\xi)d\xi} ds}{e^{\int_0^\omega \mu(s)ds} - \mathbb{1}^T}, \text{ and } \mathbb{1}^T = [1, 1, \ldots, 1]^T.$$

is the unique positive ω-periodic solution of the following equation:

$$\dot{\mathbf{S}} = \Lambda(t) - \mu(t) \circ \mathbf{S}. \tag{8}$$

Hence, all solutions are ultimately bounded. □

3. Basic Reproduction Number

In this part of the paper, we compute the thresholds dynamics of system (2) according to the theory developed by Wang and Zhao [39] for periodic systems. First, we note that model (2) admits a unique positive ω-periodic disease-free equilibrium given by $\mathbf{e}_0(t) = (\tilde{\mathbf{S}}(t), \mathbf{0}, \mathbf{0}, \mathbf{0})$. Furthermore, by linearising system (2) at $\mathbf{e}_0(t)$, we obtain the following system:

$$\begin{pmatrix} \dot{\mathbf{E}} \\ \dot{\mathbf{I}} \end{pmatrix} = (\mathbb{F}(t) - \mathbb{V}(t)) \begin{pmatrix} \mathbf{E} \\ \mathbf{I} \end{pmatrix}$$

with

$$\mathbb{F}(t) = \begin{pmatrix} \mathbf{0} & \text{diag}(\tilde{\mathbf{S}}(t))\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{S}}(t))\mathbb{P}^T \\ \mathbf{0} & \mathbf{0} \end{pmatrix} \text{ and } \mathbb{V}(t) = \begin{pmatrix} -\text{diag}(\varrho(t)) & \mathbf{0} \\ \text{diag}(\tau(t)) & -\text{diag}(\delta(t)) \end{pmatrix}.$$

For all $t \geq s$, let $\mathbb{W}(t,s)$ be the evolution operator of the linear periodic system

$$\dot{w} = -\mathbb{V}(t)w.$$

For each $s \in \mathbb{R}$, the $2m \times 2m$ matrix $\mathbb{W}(t,s)$ satisfies the equation

$$\dot{\mathbb{W}}(t,s) = -\mathbb{V}(t)\mathbb{W}(t,s), \forall t \geq s, \;\; \mathbb{W}(s,s) = I_{2m}, \tag{9}$$

where I_{2m} is the $2m \times 2m$ identity matrix.

Let C_ω be the ordered Banach space of all ω-periodic functions from $\mathbb{R}$ to $\mathbb{R}^{2m}$ which is equipped with the maximum norm $\|.\|$ and the positive cone

$$\mathrm{C}_\omega^+ := \big\{\phi \in \mathrm{C}_\omega : \phi(t) \geq 0, \forall t \in \mathbb{R}\big\}.$$

Suppose $\phi(s) \in \mathrm{C}_\omega$ is the initial distribution of infectious individuals in the periodic habitat. Then $\mathbb{F}(s)\phi(s) \in \mathrm{C}_\omega$ is the distribution of newly infected individuals at time t produced by infected individuals introduced at time $s \leq t$. Hence, $\mathbb{W}(t,s)\mathbb{F}(s)\phi(s)$ represents the distribution of those infected individuals who were newly infected at time s and remain in the compartment at time s. Thus,

$$\psi(t) = \int_{-\infty}^{t} \mathbb{W}(t,s)\mathbb{F}(s)\phi(s)ds = \int_0^{\infty} \mathbb{W}(t,t-a)\mathbb{F}(t-a)\phi(t-a)da, \; a \in [0,\infty)$$

is the distribution of accumulative new infections at time t, produced by all those infected $\phi(s)$ introduced at the previous time. Thus, the basic reproduction number, $\mathcal{R}_0(m,n,\mathbb{P})$ of system (2) is given by the spectral radius of the following the linear operator:

$$(\mathcal{L}\phi)(t) = \int_0^{\infty} \mathbb{W}(t,t-a)\mathbb{F}(t-a)\phi(t-a)da, \;\; \forall t \in \mathbb{R}, \; \phi \in \mathrm{C}_\omega.$$

Remark 3.1. *If $\mathbb{F}(t) = \mathbb{F}$ and $\mathbb{V}(t) = \mathbb{V}$, that is the periodic coefficients $\Lambda_i, \mu_i, \gamma_i, \alpha_i, \tau_i, \eta_i$ are constants, then the basic reproduction of the associated autonomous model is given by:*

$$\mathcal{R}_0(m,n,\mathbb{P}) = \rho(\mathbb{F}\mathbb{V}^{-1}) = \rho(\mathcal{A}diag(\tau)diag^{-1}\big(\delta \circ \varrho\big)), \tag{10}$$

where $\mathcal{A} = diag(\tilde{\mathbf{S}})\mathbb{Q}diag(\mathcal{B})diag^{-1}(\mathbb{Q}^T\tilde{\mathbf{S}})\mathbb{P}^T$ and $\tilde{\mathbf{S}} = \Lambda \circ \dfrac{1}{\mu}$.

Theorem 3.1. *The following statements are valid.*

i) *Let $\mathbb{P}_1$ and $\mathbb{P}_2$ be two residence matrices of infectious individuals and ζ be positive real number such that $\mathbb{P}_1 = \zeta\mathbb{P}_2$. Then, $\mathcal{R}_0(m,n,\mathbb{P}_1) = \zeta\mathcal{R}_0(m,n,\mathbb{P}_2)$.*

ii) *If there exists a positive real number ς such that $\tilde{\mathcal{B}}(t) = \varsigma\mathcal{B}(t)$, then $\tilde{\mathcal{R}}_0(m,n,\mathbb{P}) = \varsigma\mathcal{R}_0(m,n,\mathbb{P})$; where $\tilde{\mathcal{R}}_0(m,n,\mathbb{P})$ is the basic reproduction number associated to the contact rate $\tilde{\mathcal{B}}(t)$.*

Proof.

i) Suppose that $\mathbb{P}_1 = \zeta\mathbb{P}_2$ and let $\mathcal{L}_1$ and $\mathcal{L}_2$ be the linear operator associated respectively to

$$\mathbb{F}_1(t) = \text{diag}(\tilde{\mathbf{S}}(t))\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{S}}(t))\mathbb{P}_1^T$$

and

$$\mathbb{F}_2(t) = \text{diag}(\tilde{\mathbf{S}}(t))\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{S}}(t))\mathbb{P}_2^T.$$

Then, $\forall\phi \in \mathrm{C}_\omega$ we have:

$$\begin{aligned}\mathcal{L}_1\phi(t) &= \int_0^\infty \mathbb{W}(t,t-a)\mathbb{F}_1(t)\phi(t-a)da,\\ &= \zeta\int_0^\infty \mathbb{W}(t,t-a)\mathbb{F}_2(t)\phi(t-a)da,\\ &= \zeta\mathcal{L}_2\phi(t)\end{aligned}$$

Hence, $\rho(\mathcal{L}_1) = \zeta\rho(\mathcal{L}_2)$. So, $\mathcal{R}_0(m,n,\mathbb{P}_1) = \zeta\mathcal{R}_0(m,n,\mathbb{P}_2)$.

ii) If $\tilde{\mathcal{B}}(t) = \varsigma\mathcal{B}(t)$ and $\tilde{\mathbb{F}}(t) = \text{diag}(\tilde{\mathbf{S}}(t))\mathbb{Q}\text{diag}(\tilde{\mathcal{B}}(t))\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{S}}(t))\mathbb{P}^T$, then

$$\begin{aligned}\tilde{\mathcal{L}} &= \int_0^\infty \mathbb{W}(t,t-a)\tilde{\mathbb{F}}(t-a)\phi(t-a)\\ &= \zeta\int_0^\infty \mathbb{W}(t,t-a)\mathbb{F}(t-a)\phi(t-a) = \zeta\mathcal{L}\end{aligned}$$

That is $\rho(\tilde{\mathcal{L}}) = \zeta\rho(\mathcal{L})$. So, $\tilde{\mathcal{R}}_0(m,n,\mathbb{P}) = \zeta\mathcal{R}_0(m,n,\mathbb{P})$; which conclude the proof.

□

There are a number of results that allow for such a simplification in the study of global stability of systems of this kind. For the convenience of the reader, we recall the following result:

Theorem 3.2. [40] *Consider the following $\mathcal{C}^1$ system:*

$$\begin{cases}\dot{x} = f(x)\\ \dot{y} = g(x,y) \qquad\qquad x\in\mathbb{R}^n, y\in\mathbb{R}^m\\ \textit{with an equilibrium point } (x^0,y^0), i.e.,\\ f(x^0) = 0 \textit{ and } g(x^0,y^0) = 0.\end{cases} \tag{11}$$

If x^0 is globally asymptotically stable (GAS) in $\mathbb{R}^n$ for the system $\dot{x} = f(x)$, and if y^0 is GAS in $\mathbb{R}^m$ for the system $y = g(x^0,y)$, then (x^0,y^0) is (locally) asymptotically stable for (3.2). Moreover, if all the trajectories of (3.2) are forward bounded, then (x^0,y^0) is GAS for (3.2).

Since, $\tilde{\mathbf{H}}(t)$ is globally asymptotically stable for system (8), then thanks to Theorem 3.2, system (3) is asymptotically equivalent to:

$$\begin{cases} \dot{\mathbf{S}} = \Lambda(t) - \text{diag}(\mathbf{S})\text{diag}(\mathcal{B}(t))\mathbb{Q}\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}})\mathbb{P}^T\mathbf{I} - \text{diag}(\mu(t))\mathbf{S} + \text{diag}(\gamma(t))\mathbf{R}, \\ \dot{\mathbf{E}} = \text{diag}(\mathbf{S})\text{diag}(\mathcal{B}(t))\mathbb{Q}\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}})\mathbb{P}^T\mathbf{I} - \text{diag}(\varrho(t))\mathbf{E}, \\ \dot{\mathbf{I}} = \text{diag}(\tau(t))\mathbf{E} - \text{diag}(\delta(t))\mathbf{I}, \\ \dot{\mathbf{R}} = \text{diag}(\alpha(t))\mathbf{I} - \text{diag}(\theta(t))\mathbf{R}. \end{cases} \tag{12}$$

4. Persistence and Extinction of Infection

4.1. Extinction of infection.

Theorem 4.1. *The periodic infection-free equilibrium, $\mathbf{e_0}(t)$ is globally asymptotically stable whenever $\mathcal{R}_0(m, n, \mathbb{P}) \leq 1$.*

Proof. Since the equilibrium $\mathbf{e}_0(t)$ is asymptotically stable when $\mathcal{R}_0(m, n, \mathbb{P}) \leq 1$, it's so sufficient to prove that any solution $\mathbf{u}(t, \phi)$ approaches to $\mathbf{e}_0(t)$.

Indeed, equation (6) implies that for all $\varepsilon = (\varepsilon_1, \varepsilon_2, \ldots, \varepsilon_m)^T > 0$, there exists t_1 such that,

$$\mathbf{S}(t) < \tilde{\mathbf{S}}(t) + \varepsilon, \forall t \geq t_1. \tag{13}$$

Therefore,

$$\dot{\mathbf{E}} \leq \text{diag}(\tilde{\mathbf{S}}(t) + \varepsilon)\text{diag}(\mathcal{B}(t))\mathbb{Q}\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}})\mathbb{P}^T\mathbf{I} - \text{diag}(\varrho(t))\mathbf{E}.$$

Hence, the auxiliary system:

$$\begin{cases} \dot{\bar{\mathbf{E}}} &= \text{diag}(\tilde{\mathbf{S}}(t) + \varepsilon)\text{diag}(\mathcal{B}(t))\mathbb{Q}\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}})\mathbb{P}^T\bar{\mathbf{I}} - \text{diag}(\varrho(t))\bar{\mathbf{E}}, \\ \dot{\bar{\mathbf{I}}} &= \text{diag}(\tau(t))\bar{\mathbf{E}} - \text{diag}(\delta(t))\bar{\mathbf{I}} \end{cases} \tag{14}$$

can be rewritten as follows:

$$\begin{pmatrix} \dot{\bar{\mathbf{E}}} \\ \dot{\bar{\mathbf{I}}} \end{pmatrix} = (\mathbb{F}_\varepsilon(t) - \mathbb{V}(t)) \begin{pmatrix} \bar{\mathbf{E}} \\ \bar{\mathbf{I}} \end{pmatrix} \tag{15}$$

with

$$\mathbb{F}_\varepsilon(t) = \begin{pmatrix} \mathbf{0} & \text{diag}(\tilde{\mathbf{S}}(t) + \varepsilon)\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}}(t))\mathbb{P}^T \\ \mathbf{0} & \mathbf{0} \end{pmatrix}.$$

Furthermore, one can see that the ω-periodic matrix $\mathbb{F}_\varepsilon(t) - \mathbb{V}(t)$ is cooperative, irreducible and continuous. Let $p = \frac{1}{\omega}\ln\left(\rho(\Phi_{\mathbb{F}_\varepsilon - \mathbb{V}}(\omega))\right)$. Then, there exists a ω-periodic function v such that $e^{pt}v(t)$ is solution of system (15), $\forall k > 0$, [19,34]. Since, $ke^{p(t-t_1)}v(t-t_1)$ is also solution of (15), then there exists $t_\varepsilon > t_1$ and $a > 0$ such that $(\dot{\bar{\mathbf{E}}}(t), \dot{\bar{\mathbf{I}}}(t))^T \leq av(0)$, for all $t_\varepsilon > t_1$. Thanks to comparison principle, it yields that $(\mathbf{E}(t), \mathbf{I}(t))^T \leq av(0)e^{p(t-t_1)}v(t-t_1)$ for all $t > t_\varepsilon$. By the continuity of spectral radius,

if $\mathcal{R}_0(m,n,\mathbb{P}) < 1$, then there exists $\varepsilon > 0$ such that $\rho(\Phi_{\mathbb{F}_\varepsilon - \mathbb{V}}(\omega)) < 1$; namely $p < 0$. Moreover, from positivity of solutions and comparison principle, $\mathbf{I}(t) \to 0$ as $t \to \infty$.

It then follows from the theory of asymptotically periodic semi-flow, [41] that $\lim_{t\to\infty}(S(t) - \tilde{H}(t)) = 0$. This complete the proof. □

4.2. **Persistence of infection.** Uniform persistence is an important concept in population dynamics because it characterizes the long-term behaviour of some species in an ecosystem.

Theorem 4.2. *Let $\mathcal{R}_0(m,n,\mathbb{P}) > 1$. Then, system (2)is uniformly persistent and there exists at least one positive equilibrium.*

Proof. Let us consider the following sets:

$$\begin{aligned} \mathbf{X} &:= \mathbb{R}^{3m}_+, \\ \mathbf{X}_0 &:= \{(\mathbf{S},\mathbf{E},\mathbf{I}) \in \mathbf{X} : \mathbf{E} > \mathbf{0}, \mathbf{I} > \mathbf{0}\}, \\ \partial\mathbf{X}_0 &:= \mathbf{X} \setminus \mathbf{X}_0. \end{aligned}$$

We note that $\mathbf{X}_0$ is an open set relative to $\mathbf{X}$ and positively invariant. $\partial\mathbf{X_0}$ is relatively closed in $\mathbf{X}$ and system (2) is point dissipative. Let $\Phi(t)\phi = \mathbf{u}(t,\phi)$ and $P : \mathbf{X} \longrightarrow \mathbf{X}$ be the Poincaré map associated with system (2), that is, $P^r(\phi) = \mathbf{u}(r\omega,\phi), \forall \phi \in \mathbf{X}$. Furthermore, let us consider the following sets:

$$\mathbf{M}_\partial = \{\phi : P^r(\phi) \in \partial\mathbf{X_0}, \forall r \geq 0\} \quad \text{and} \quad \{(\mathbf{S},\mathbf{0},\mathbf{0}) : \mathbf{S} \geq \mathbf{0}\}$$

Claim 4.1.

$$\mathbf{M}_\partial = \{(\mathbf{S},\mathbf{0},\mathbf{0}) : \mathbf{S} \geq \mathbf{0}\}. \tag{16}$$

Proof. Assume $\phi \in \mathbf{M}_\partial$. Let us show that $\mathbf{E}(t) = \mathbf{0} = \mathbf{I}(t), \forall t \geq 0$. Suppose not. Then, $\mathbf{E}^0 > \mathbf{0}$, $\mathbf{I}^0 = \mathbf{0}$ or $\mathbf{I}^0 > \mathbf{0}$, $\mathbf{E}^0 = \mathbf{0}$. Without lost of generality, suppose that $\mathbf{E}^0 > \mathbf{0}$, $\mathbf{I}^0 = \mathbf{0}$. It follows that

$$\frac{d\mathbf{I}}{dt}(0) = \text{diag}(\tau(0))\mathbf{E}(0) > 0.$$

Hence, for all $0 < t << 1$, $\mathbf{E}(t) > 0$ and $\mathbf{I}(t) > 0$. So, $\mathbf{u}(t,\phi) \in \mathbf{X}_0$; since $\mathbf{X}_0$ is invariant, then $\mathbf{u}(t,\phi) \in \mathbf{X}_0, \forall t > 1$. Therefore, $\forall r > 0$ such that $r\omega > 1$, we have $P^r(\phi) = \mathbf{u}(r\omega,\phi) \in \mathbf{X}_0$. Contradiction. Moreover, let $\phi = (\mathbf{S}^0,\mathbf{0},\mathbf{0}) \in X$ and $(\mathbf{S}(t),\mathbf{E}(t),\mathbf{I}(t))$ be the solution through ϕ. Then $(\mathbf{S}(t),\mathbf{0},\mathbf{0})$ with $\mathbf{S}(0) = \mathbf{S}^0$ is a solution that satisfies initial condition. Thus, by uniqueness of solutions, $\mathbf{I}(t) = \mathbf{0} = \mathbf{0}, \forall t \geq 0$. so (16) holds. □

Claim 4.2. *There exists $\kappa > 0$ such that,* $\limsup_{r\to\infty} ||\Phi(r\omega)\phi - \{\mathbf{e_0}(t)\}|| \geq \kappa, \forall \phi \in \mathbf{X}_0$.

Proof. Suppose by contradiction that $\limsup_{r\to\infty} ||\Phi(r\omega)\phi - \{\mathbf{e_0}(t)\}|| < \kappa$ for some $\phi \in \mathbf{X}_0$. then, there exists an integer $p \geq 1$ such that for all $r \geq p$ we have $||\Phi(r\omega)\phi - \{\mathbf{e_0}(t)\}|| < \kappa$. By continuity of solution,

we have $||\mathbf{u}(t,\Phi(r\omega)\phi) - \mathbf{u}(t,\{\mathbf{e_0}(t)\})|| < \varepsilon', \forall t \geq 0$. Moreover, for any $t \geq 0$, let $t = r\omega + t_2$, with $t_2 \in [0,\omega]$ and $r = \mathbb{E}\left[\frac{t}{\omega}\right]$ is the greatest integer less than or equal to $\frac{t}{\omega}$.

It then follows that:

$$\begin{aligned}\|\Phi(t)\phi - \Phi(t)\{\mathbf{e_0}(t)\}\| &= \|\Phi(r\omega+t_2)\phi - \Phi(r\omega+t_2)\{\mathbf{e_0}(t)\}\| \\ &= \|\Phi(t_2)\Phi(r\omega+)\phi - \Phi(t_2)\Phi(r\omega)\{\mathbf{e_0}(t)\}\| \\ &= \|\Phi(t_2)\Phi(r\omega+)\phi - \Phi(t_2)\{\mathbf{e_0}(t)\}\| < \varepsilon'.\end{aligned}$$

Hence, $S_i(t) > \tilde{S}_i(t) - \varepsilon_i',\ 1 \leq i \leq n$, with $\varepsilon' = (\varepsilon_1', \varepsilon_2', \ldots, \varepsilon_m')^T > 0$; which implies that

$$\dot{\mathbf{E}}(t) \geq \text{diag}(\tilde{\mathbf{S}}(\mathbf{t}) - \varepsilon')\text{diag}(\mathcal{B}(t))\mathbb{Q}\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}})\mathbb{P}^T\mathbf{I}(t) - \text{diag}(\varrho(t))\mathbf{I}(t).$$

Let us consider the following auxiliary system:

$$\dot{\mathbf{w}}(t) = (\mathbb{F}_{\varepsilon'}(t) - \mathbb{V}(t))\mathbf{w}(t), \tag{17}$$

with

$$\mathbb{F}_{\varepsilon'}(t) = \begin{pmatrix} \mathbf{0} & \text{diag}(\tilde{\mathbf{S}}(t) - \varepsilon')\mathbb{Q}\text{diag}(\mathcal{B}(t))\text{diag}^{-1}(\mathbb{Q}^T\tilde{\mathbf{H}}(t))\mathbb{P}^T \\ \mathbf{0} & \mathbf{0} \end{pmatrix}. \tag{18}$$

Since $\mathcal{R}_0(m,n,\mathbb{P}) > 1$, then $\rho(\Phi_{\mathbb{F}-\mathbb{V}}(\omega)) > 1$. By the continuity of the spectral radius, we have $\lim_{\varepsilon'\to 0^+}\rho\big(\Phi_{\mathbb{F}_{\varepsilon'}-\mathbb{V}}(\omega)\big) = \rho\big(\Phi_{\mathbb{F}-\mathbb{V}}(\omega)\big) > 1$, [42]. Hence, there exists a positive ω-periodic function $\mathbf{v}_1(t)$ such that $e^{p_1 t}\mathbf{v}_1(t)$ is a solution of (17), with $p_1 = \frac{1}{\omega}\ln\rho(\Phi_{\mathbb{F}_{\varepsilon'}-\mathbb{V}}(\omega))$; then, $p_1 > 0$. Thanks to comparison principle, it yields that $\lim_{t\to\infty}(\mathbf{E}(t)) = \infty = \lim_{t\to\infty}(\mathbf{I}(t))$; which leads to a contradiction. □

Claim 4.2 shows that P is weakly uniformly persistent with respect to $(\mathbf{X}_0, \partial\mathbf{X}_0)$, [[43], Theorem 3.1.1]. Since P has a global attractor, $\tilde{\mathbf{S}}(0)$, it follows that $\{\mathbf{e_0}(t)\}$ is an isolated invariant set in $\mathbf{X}$ and

$$W^S =: \{\phi \in \mathbf{X}_0 : \lim_{r\to\infty}||P^r(\phi) - \{\mathbf{e_0}(t)\} = 0\} \cap \mathbf{X}_0 = \emptyset.$$

That implies that every orbit in M_∂ converges to $\{\mathbf{e_0}(t)\}$ and $\{\mathbf{e_0}(t)\}$ is acyclic. Thanks to aciclity theorem on uniform persistence for maps [44], P is uniformly persistent with respect to $(\mathbf{X}_0, \partial\mathbf{X}_0)$. □

5. Numerical Simulations

In this section, we provide some numerical simulations for $m = n = 2$ in order to highlight the effects of climate on the transmission and to compare our results with previous in term of thresholds dynamics. To that end, we suppose that functions $\Lambda(t) = \Lambda, \mu(t) = \mu, \eta(t) = \eta, \tau(t) = \tau, \alpha(t) = \alpha, \gamma(t) = \gamma,\ \forall t \geq 0$ and we set $\Lambda_1 = \Lambda_2 = 50$; $\mu_1 = \mu_2 = \frac{1}{75\times 365}$ days^{-1}; $\eta_1 = \eta_2 = 2\times 10^{-5}$ days^{-1}, $\tau_1 = \tau_2 = \frac{1}{4}$ days^{-1}, $\alpha_1 = \alpha_2 = \frac{1}{6}$ days^{-1}, $\gamma_1 = \gamma_2 = 0.00137$ days^{-1} and $\omega = 7$ days. Moreover, assuming

that the two patches have different climate conditions, we estimate the function β for each patch as follows:

$$\beta_1(t) = \beta_{01} + \cos\left(\frac{2\pi t}{\omega}\right) \text{ and } \beta_2(t) = \beta_{02} + \cos\left(\frac{2\pi t}{\omega}\right), \tag{19}$$

and the residence time matrices by:

$$\mathbb{Q} = \begin{pmatrix} 0.20 & 0.10 \\ 0.10 & 0.20 \end{pmatrix}, \mathbb{P} = \begin{pmatrix} 0.80 & 0.10 \\ 0.10 & 0.80 \end{pmatrix}, \mathbb{M} = \begin{pmatrix} 0.40 & 0.20 \\ 0.20 & 0.40 \end{pmatrix} \text{ and } \mathbb{L} = \begin{pmatrix} 0.50 & 0.20 \\ 0.20 & 0.50 \end{pmatrix}.$$

Therefore, from (7), the free-infection equilibrium is given by:

$$\mathbf{e}_0(t) = (\tilde{S}_1(t), \tilde{S}_2(t), 0, 0, 0, 0, 0, 0), \text{ with } \tilde{S}_i(t) = \frac{\Lambda_i}{\mu_i},\ i = 1, 2.$$

Furthermore, let $\mathbf{Z}(t, \lambda)$ be the monodromy matrix of the following linear $\omega-$periodic system:

$$\frac{d\mathbf{Z}(t)}{dt} = \left(\frac{1}{\lambda}\mathbb{F}(t) - \mathbb{V}(t)\right)\mathbf{Z}(t), t \in \mathbb{R}, \lambda \in (0, \infty), \tag{20}$$

where

$$\mathbb{V}(t) = \begin{pmatrix} -\varrho_1 & 0 & 0 & 0 \\ 0 & -\varrho_2 & 0 & 0 \\ \tau_1 & 0 & -\delta_1 & 0 \\ 0 & \tau_2 & 0 & -\delta_1 \end{pmatrix} \text{ and } \mathbb{F}(t) = \begin{pmatrix} \mathbf{0}_{2\times 2} & \mathcal{A}(t) \\ \mathbf{0}_{2\times 2} & \mathbf{0}_{2\times 2} \end{pmatrix}$$

with

$$\mathcal{A}(t) = \begin{pmatrix} \dfrac{\beta_1(t)p_{11}\tilde{S}_1 q_{11}}{q_{11}\tilde{S}_1 + q_{21}\tilde{S}_2} + \dfrac{\beta_2(t)p_{12}S_1 q_{12}}{q_{12}\tilde{S}_1 + q_{22}S_2} & \dfrac{\beta_1(t)p_{21}\tilde{S}_1 q_{11}}{q_{11}S_1 + q_{21}\tilde{S}_2} + \dfrac{\beta_2(t)p_{22}\tilde{S}_1 q_{12}}{q_{12}\tilde{S}_1 + q_{22}\tilde{S}_2} \\ \dfrac{\beta_1(t)p_{11}\tilde{S}_2 q_{21}}{q_{11}S_1 + q_{21}\tilde{S}_2} + \dfrac{\beta_2(t)p_{12}\tilde{S}_2 q_{22}}{q_{12}\tilde{S}_1 + q_{22}\tilde{S}_2} & \dfrac{\beta_1(t)p_{21}\tilde{S}_2 q_{21}}{q_{11}\tilde{S}_1 + q_{21}\tilde{S}_2} + \dfrac{\beta_2(t)p_{22}\tilde{S}_2 q_{22}}{q_{12}\tilde{S}_1 + q_{22}\tilde{S}_2} \end{pmatrix}. \tag{21}$$

Since $\mathbb{F}(t)$ is nonnegative and $-\mathbb{V}(t)$ is cooperative, then $\rho(\mathbf{Z}(\omega, \lambda))$ is continuous and non increasing for $\lambda \in (0, \infty)$; and $\lim_{\lambda\to\infty} \rho(\mathbf{Z}(\omega, \lambda)) < 1$. Hence we will use the following result to compute the basic reproduction number of the model.

Theorem 5.1. [39] *The following statements are valid:*

i. *if $\rho(\mathbf{Z}(\omega, \lambda)) = 1$ has a positive solution, λ_0, then λ_0 is an eigenvalue of $\mathcal{L}$, and hence, $\mathcal{R}_0(n, m, \mathbb{P}) > 0$.*
ii. *If $\mathcal{R}_0(n, m, \mathbb{P}) > 0$, then $\lambda_0 = \mathcal{R}_0(n, m, \mathbb{P})$ is the unique solution of $\rho(\mathbf{Z}(\omega, \lambda)) = 1$.*
iii. *$\mathcal{R}_0(n, m, \mathbb{P}) = 0$ if and only if $\rho(\mathbf{Z}(\omega, \lambda)) < 1, \forall\lambda > 0$.*

To compute numerically the basic reproduction, we set λ_0 (initial value) and $\lambda_k = \lambda_0 + kb$ and then, we solve numerically system (20) for each λ_k, such that $\mathbf{Z}(0, 0) = I_{4\times 4}$. Thus, we compute $\rho(\mathbf{Z}(\omega, \lambda_k)) = \ell$ until $\ell \sim 1$.

Remark 5.1. *Let* $[\beta] = \frac{1}{\omega}\int_0^{\omega} \beta(t)dt$ *be the average of function* β*. Then, the basic reproduction of the autonomous system is given by* $[\mathcal{R}_0](2,2,\mathbb{P}) = \rho([\mathbb{F}]\mathbb{V}^{-1}) = \rho\big([\mathcal{A}]diag(\tau)diag^{-1}(\delta \circ \varrho)\big)$.

Using the above values, and thanks to initial conditions: $\mathbf{S}(0) = [985, 890]^T; \mathbf{E}(0) = [5,5]^T; \mathbf{I}(0) = [10,5]^T$ and $\mathbf{R}(0) = [0,0]^T$, we obtain the following figures which illustrate the dependence of the infection with respect to $\beta(t)$.

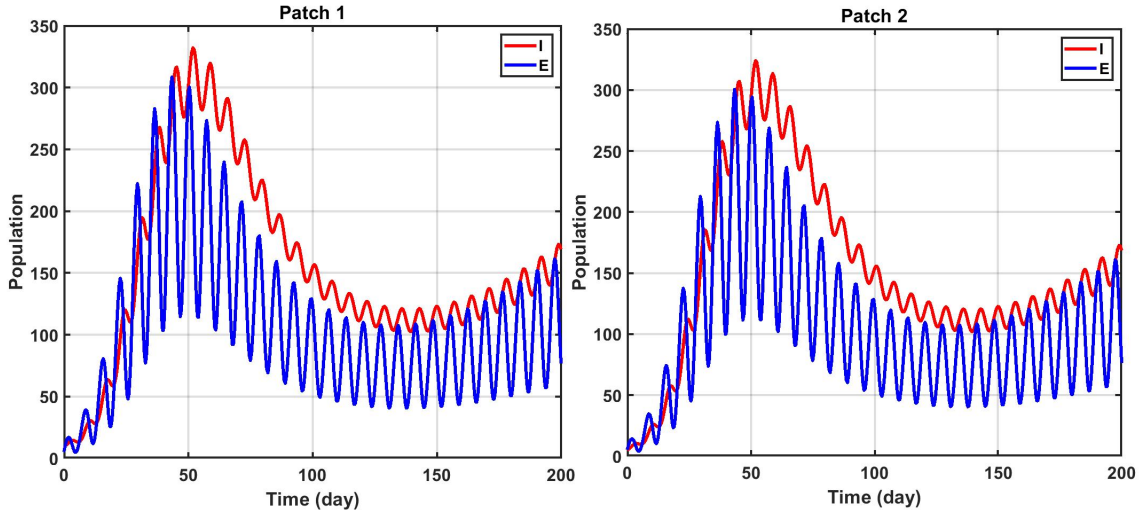


FIGURE 1. Dynamics of infectious hosts with $\beta_{01} = \beta_{02} = 0.6$; $\mathcal{R}_0(2,2,\mathbb{P}) = 3.034$.

Figures 1, 2, 3 indicate the effects of climate variations on the basic reproduction rate, $\mathcal{R}_0(2,2,\mathbb{P})$. Indeed, we notice that $\mathcal{R}_0(2,2,\mathbb{P})$ increases or decreases with respect to β in each patch. Moreover, while infection dies out for $\mathcal{R}_0(2,2,\mathbb{P}) \leq 1$, the model exhibits positive periodic solutions for $\mathcal{R}_0(2,2,\mathbb{P}) > 1$. These numerical examples illustrate our theoretical results.

The contour lines corresponding to periodic and average reproduction numbers allows us to identify regions in the parameters space (β_{01}, β_{02}) where the infection either disappears or persists the threshold value $\mathcal{R}_0(2,2,\mathbb{P}) = 1$ or $[\mathcal{R}_0](2,2,\mathbb{P}) = 1$ plays a key role in this analysis. Thus, Figures 4, 5 show the dependence of the two basic basic reproduction number, $\mathcal{R}_0(2,2,\mathbb{P})$ and $[\mathcal{R}_0](2,2,\mathbb{P})$ with respect to (β_{01}, β_{02}). Thus, while the both basic reproduction numbers increase simultaneous with (β_{01}, β_{02}), the periodic basic reproduction $\mathcal{R}_0(2,2,\mathbb{P})$ is observed to increase rapidly. This observation highlights the impact of climatic fluctuations on the dynamics of infection transmission.

Figure 6 allows us to identify the regions in the parameters space (β_{01}, β_{02}) where the periodic basic reproduction is greater or smaller than the average reproduction number. In fact, we can clearly observe

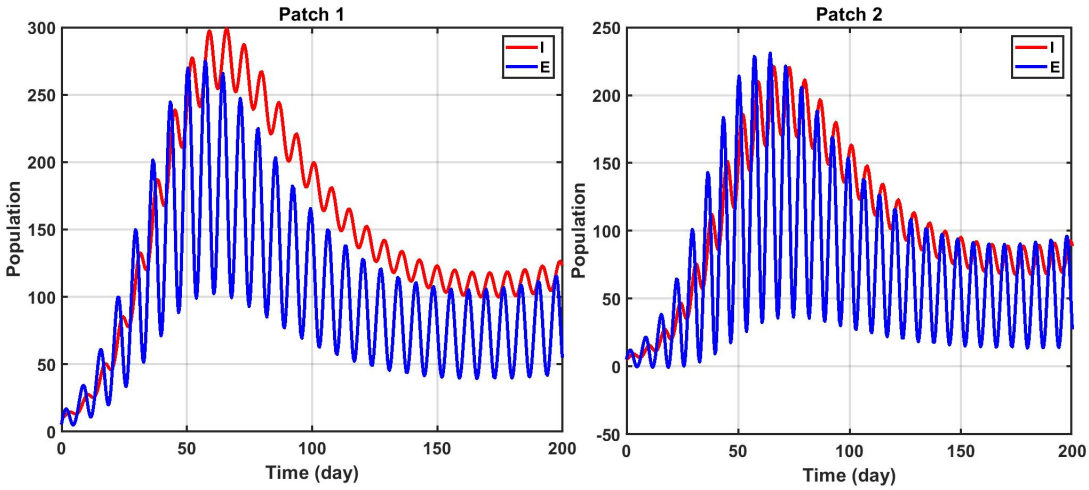


FIGURE 2. Dynamics of infectious hosts with $\beta_{01} = 0.6,\ \beta_{02} = 0.4, \mathcal{R}_0(2,2,\mathbb{P}) = 2.492 > 1$.

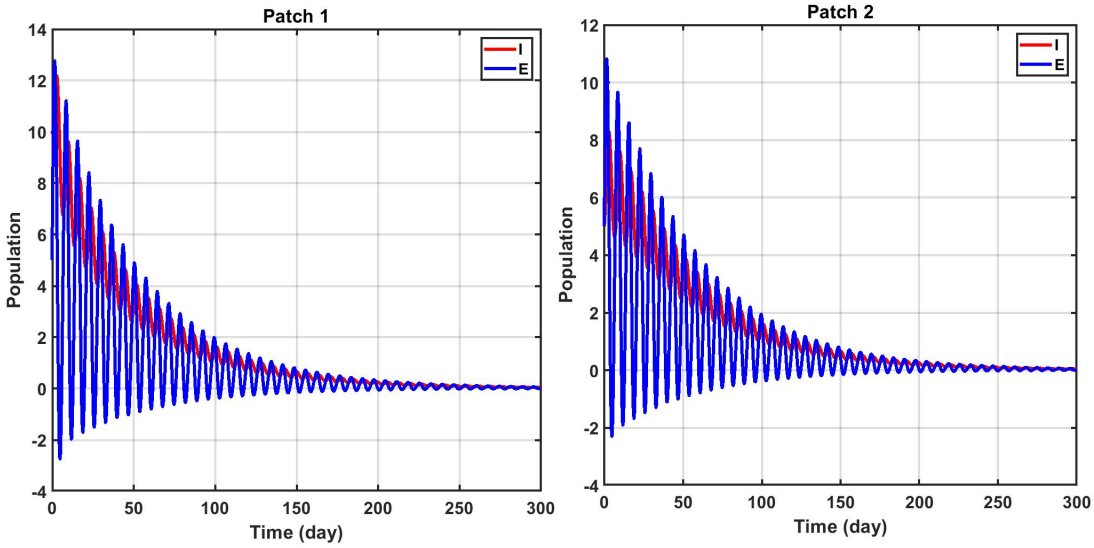


FIGURE 3. Dynamics of infectious hosts with $\beta_{01} = 0.27,\ \beta_{02} = 0.27, \mathcal{R}_0(2,2,\mathbb{P}) = 0.3156$.

that greatest is the values of space (β_{01}, β_{02}), the difference between $\Delta\mathcal{R}_0 = \mathcal{R}_0(2,2,\mathbb{P}) - [\mathcal{R}_0](2,2,\mathbb{P})$ is positive and becomes greater. Hence variations in transmissions leads to a higher estimate of the infection's spread potential. Conversely, when the difference, $\Delta\mathcal{R}_0$ is negative, the periodic model predicts a lower transmission potential.

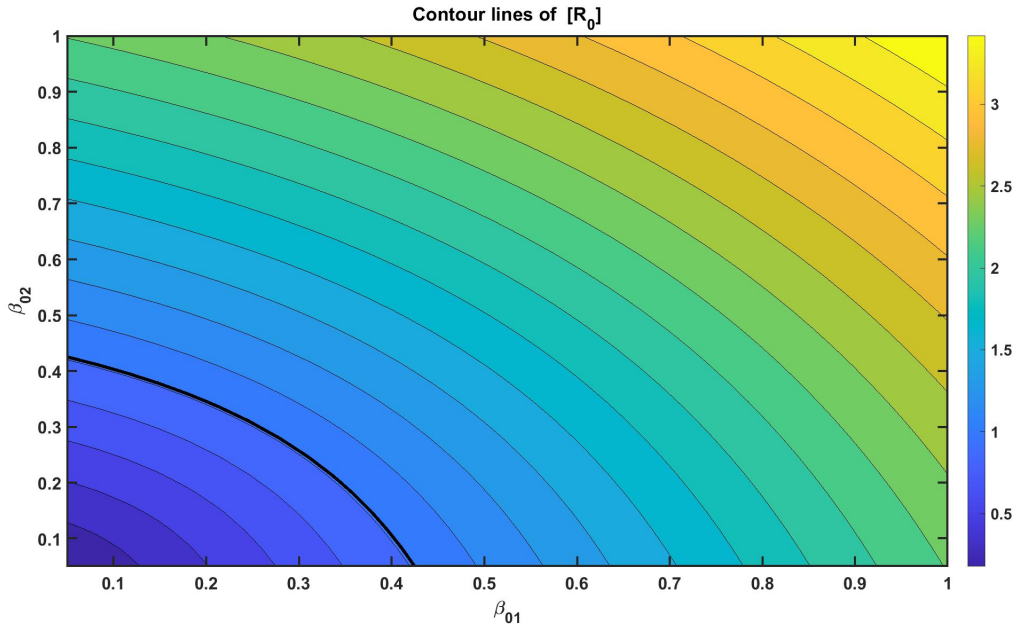


FIGURE 4. Graph of average basic reproduction number when varying β_{01} and β_{02}.

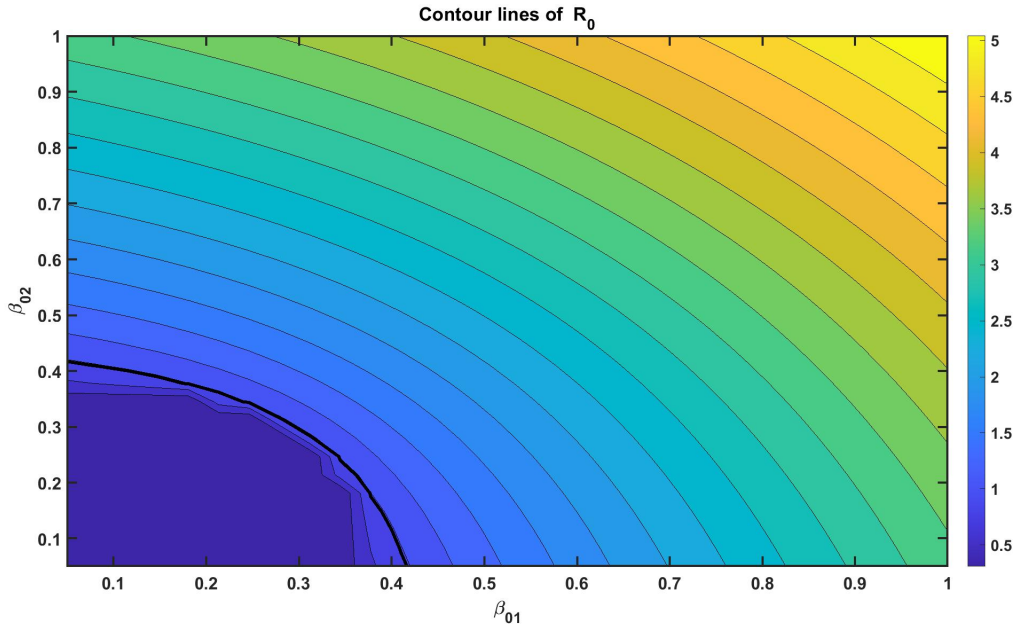


FIGURE 5. Graph of basic reproduction number when varying β_{01} and β_{02}.

6. CONCLUSION

In this paper, we analysed and compared two approaches for characterizing the transmission dynamics of an multi-group and multi-patch SEIRS metapopulation epidemic models. The reproduction number associated with constant transmission parameters and the reproduction number obtained when transmission varies periodically over time. This comparison highlighted the importance of taking

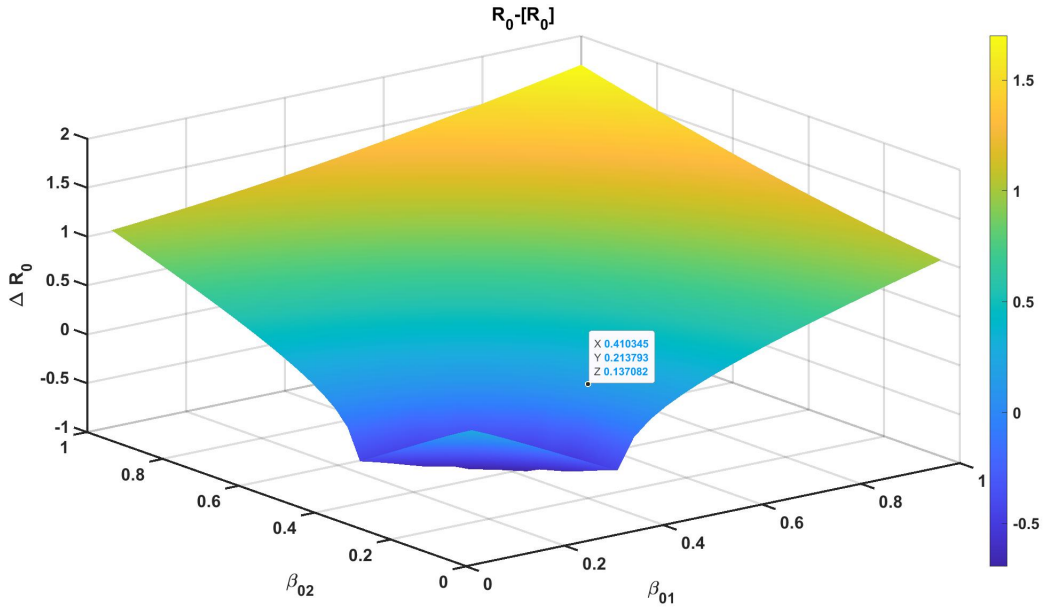


FIGURE 6. Comparison of two basic reproduction numbers.

into account temporal variations and the spatial structure of the population when assessing the risk of an epidemic spreading.

Our results show that the periodic reproduction number may behave differently from the constant reproduction number, even if the average values of the transmission parameters is the same. The difference stems from the fact that periodic transmission accounts for temporal fluctuations in the spread of infection and can better represented real-world situations in which contacts between individuals, mobility, or environment conditions vary over time. Numerical simulations also indicate that variations in transmission parameters directly influence the epidemiological threshold and can alter the conditions under which the disease persists or is eradicated.

Our study also point out the essential role of residence pattern transmission dynamics. Indeed, individual mobility between different patches alters interactions among populations and thus influences the value of reproduction rate. A change in residence patterns can therefore either promote the spread of the infection or help limit it. These results underscore the importance of simultaneously incorporating the spatial structure of the population and temporal variations in epidemiological parameters into the analysis of metapopulation models.

The comparison between constant and periodic reproduction rate shows that using a constant reproduction rate can lead to an oversimplification of the dynamics when epidemiological parameters vary over time. The periodic reproduction number is therefore a more suitable tool for studying systems subject to seasonal variations or regular changes behaviour and transmission conditions. However, the periodic model remains dependent on the assumptions made regarding the shape and amplitude of

temporal variations as well as movement between patches. This study confirms that the joint analysis of the periodicity of epidemiological parameters and spatial mobility is an relevant approach for better understanding the spread of infectious diseases in populations structure into patches. In particular, comparing constant and periodic reproduction rates provides a theoretical framework for evaluation the influence of temporal and spatial fluctuations on the persistence or extinction of an epidemic.

Future investigations can analyse time-dependent control strategies such as periodic vaccination, reduced mobility or decreased contact between individuals in order to identify the most effective interventions for reducing the reproduction number and limiting the spread of infection.

Conflicts of Interest. The authors declare that there are no conflicts of interest regarding the publication of this paper.

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