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Universidad de Oviedo

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Foreword

It is with great pleasure that we present the Proceedings of the 26th Congress of Differential Equations and Applications / 16th Congress of Applied Mathematics (XXVI CEDYA / XVI CMA), the biennial congress of the Spanish Society of Applied Mathematics S$\vec{e}$MA, which is held in Gijón, Spain from June 14 to June 18, 2021.

In this volume we gather the short papers sent by some of the almost three hundred and twenty communications presented in the conference. Abstracts of all those communications can be found in the abstract book of the congress. Moreover, full papers by invited lecturers will shortly appear in a special issue of the S$\vec{e}$MA Journal.

The first CEDYA was celebrated in 1978 in Madrid, and the first joint CEDYA / CMA took place in Málaga in 1989. Our congress focuses on different fields of applied mathematics: Dynamical Systems and Ordinary Differential Equations, Partial Differential Equations, Numerical Analysis and Simulation, Numerical Linear Algebra, Optimal Control and Inverse Problems and Applications of Mathematics to Industry, Social Sciences, and Biology. Communications in other related topics such as Scientific Computation, Approximation Theory, Discrete Mathematics and Mathematical Education are also common.

For the last few editions, the congress has been structured in mini-symposia. In Gijón, we will have eighteen minis-symposia, proposed by different researchers and groups, and also five thematic sessions organized by the local organizing committee to distribute the individual contributions. We will also have a poster session and ten invited lectures. Among all the mini-symposia, we want to highlight the one dedicated to the memory of our colleague Francisco Javier "Pancho" Sayas, which gathers two plenary lectures, thirty-six talks, and more than forty invited people that have expressed their wish to pay tribute to his figure and work.

This edition has been deeply marked by the COVID-19 pandemic. First scheduled for June 2020, we had to postpone it one year, and move to a hybrid format. Roughly half of the participants attended the conference online, while the other half came to Gijón. Taking a normal conference and moving to a hybrid format in one year has meant a lot of efforts from all the parties involved. Not only did we, as organizing committee, see how much of the work already done had to be undone and redone in a different way, but also the administration staff, the scientific committee, the mini-symposia organizers, and many of the contributors had to work overtime for the change.

Just to name a few of the problems that all of us faced: some of the already accepted mini-symposia and contributed talks had to be withdrawn for different reasons (mainly because of the lack of flexibility of the funding agencies); it became quite clear since the very first moment that, no matter how well things evolved, it would be nearly impossible for most international participants to come to Gijón; reservations with the hotels and contracts with the suppliers had to be cancelled; and there was a lot of uncertainty, and even anxiety could be said, until we were able to confirm that the face-to-face part of the congress could take place as planned.

On the other hand, in the new open call for scientific proposals, we had a nice surprise: many people that would have not been able to participate in the original congress were sending new ideas for mini-symposia, individual contributions and posters. This meant that the total number of communications was about twenty percent greater than the original one, with most of the new contributions sent by students.

There were almost one hundred and twenty students registered for this CEDYA / CMA. The hybrid format allows students to participate at very low expense for their funding agencies, and this gives them the opportunity to attend different conferences and get more merits. But this, which can be seen as an advantage, makes it harder for them to obtain a full conference experience. Alfréd Rényi said: "a mathematician is a device for turning coffee into theorems". Experience has taught us that a congress is the best place for a mathematician to have a lot of coffee. And coffee cannot be served online.

In Gijón, June 4, 2021

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Contents

On numerical approximations to diffuse-interface tumor growth models
Acosta-Soba D., Guillén-González F. and Rodríguez-Galván J.R. **8**

An optimized sixth-order explicit RKN method to solve oscillating systems
Ahmed Demba M., Ramos H., Kumam P. and Watthayu W. **15**

The propagation of smallness property and its utility in controllability problems
Apraiz J. **23**

Theoretical and numerical results for some inverse problems for PDEs
Apraiz J., Doubova A., Fernández-Cara E. and Yamamoto M. **31**

Pricing TARN options with a stochastic local volatility model
Arregui I. and Ráfales J. **39**

XVA for American options with two stochastic factors: modelling, mathematical analysis and numerical methods
Arregui I., Salvador B., Ševčovič D. and Vázquez C. **44**

A numerical method to solve Maxwell's equations in 3D singular geometry
Assous F. and Raichik I. **51**

Analysis of a SEIRS metapopulation model with fast migration
Atienza P. and Sanz-Lorenzo L. **58**

Goal-oriented adaptive finite element methods with optimal computational complexity
Becker R., Gantner G., Innerberger M. and Praetorius D. **65**

On volume constraint problems related to the fractional Laplacian
Bellido J.C. and Ortega A. **73**

A semi-implicit Lagrange-projection-type finite volume scheme exactly well-balanced for 1D shallow-water system
Caballero-Cárdenas C., Castro M.J., Morales de Luna T. and Muñoz-Ruiz M.L. **82**

SEIRD model with nonlocal diffusion
Calvo Pereira A.N. **90**

Two-sided methods for the nonlinear eigenvalue problem
Campos C. and Roman J.E. **97**

Fractionary iterative methods for solving nonlinear problems
Candelario G., Cordero A., Torregrosa J.R. and Vassileva M.P. **105**

Well posedness and numerical solution of kinetic models for angiogenesis
Carpio A., Cebrián E. and Duro G. **109**

Variable time-step modal methods to integrate the time-dependent neutron diffusion equation
Carreño A., Vidal-Ferràndiz A., Ginestar D. and Verdú G. **114**

Homoclinic bifurcations in the unfolding of the nilpotent singularity of codimension 4 in R^4
Casas P.S., Drubi F. and Ibánez S. **122**

Different approximations of the parameter for low-order iterative methods with memory
Chicharro F.I., Garrido N., Sarría I. and Orcos L. **130**

Designing new derivative-free memory methods to solve nonlinear scalar problems
Cordero A., Garrido N., Torregrosa J.R. and Triguero P. **135**

Iterative processes with arbitrary order of convergence for approximating generalized inverses
Cordero A., Soto-Quirós P. and Torregrosa J.R. **141**

FCF formulation of Einstein equations: local uniqueness and numerical accuracy and stability
Cordero-Carrión I., Santos-Pérez S. and Cerdá-Durán P. **148**

New Galilean spacetimes to model an expanding universe
De la Fuente D. **155**

Numerical approximation of dispersive shallow flows on spherical coordinates
Escalante C. and Castro M.J. **160**

New contributions to the control of PDEs and their applications
Fernández-Cara E. **167**

Saddle-node bifurcation of canard limit cycles in piecewise linear systems
Fernández-García S., Carmona V. and Teruel A.E. **172**

On the amplitudes of spherical harmonics of gravitational potencial and generalised products of inertia
Floría L. **177**

Turing instability analysis of a singular cross-diffusion problem
Galiano G. and González-Tabernero V. **184**

Weakly nonlinear analysis of a system with nonlocal diffusion
Galiano G. and Velasco J. **192**

What is the humanitarian aid required after tsunami?
González-Vida J.M., Ortega S., Macías J., Castro M.J., Michelini A. and Azzarone A. **197**

On Keller-Segel systems with fractional diffusion
Granero-Belinchón R. **201**

An arbitrary high order ADER Discontinous Galerking (DG) numerical scheme for the multilayer shallow water model with variable density
Guerrero Fernández E., Castro Díaz M.J., Dumbser M. and Morales de Luna T. **208**

Picard-type iterations for solving Fredholm integral equations
Gutiérrez J.M. and Hernández-Verón M.A. **216**

High-order well-balanced methods for systems of balance laws based on collocation RK ODE solvers
Gómez-Bueno I., Castro M.J., Parés C. and Russo G. **220**

An algorithm to create conservative Galerkin projection between meshes
Gómez-Molina P., Sanz-Lorenzo L. and Carpio J. **228**

On iterative schemes for matrix equations
Hernández-Verón M.A. and Romero N. **236**

A predictor-corrector iterative scheme for improving the accessibility of the Steffensen-type methods
Hernández-Verón M.A., Magreñán A.A., Martínez E. and Sukhjit S. **242**

CONTENTS

Recent developments in modeling free-surface flows with vertically-resolved velocity profiles using moments
Koellermeier J. **247**

Stability of a one degree of freedom Hamiltonian system in a case of zero quadratic and cubic terms
Lanchares V. and Bardin B. **253**

Minimal complexity of subharmonics in a class of planar periodic predator-prey models
López-Gómez J., Muñoz-Hernández E. and Zanolin F. **258**

On a non-linear system of PDEs with application to tumor identification
Maestre F. and Pedregal P. **265**

Fractional evolution equations in dicrete sequences spaces
Miana P.J. **271**

KPZ equation approximated by a nonlocal equation
Molino A. **277**

Symmetry analysis and conservation laws of a family of non-linear viscoelastic wave equations
Márquez A. and Bruzón M. **284**

Flux-corrected methods for chemotaxis equations
Navarro Izquierdo A.M., Redondo Neble M.V. and Rodríguez Galván J.R. **289**

Ejection-collision orbits in two degrees of freedom problems
Ollé M., Álvarez-Ramírez M., Barrabés E. and Medina M. **295**

Teaching experience in the Differential Equations Semi-Virtual Method course of the Tecnológico de Costa Rica
Oviedo N.G. **300**

Nonlinear analysis in lorentzian geometry: the maximal hypersurface equation in a generalized Robertson-Walker spacetime
Pelegrín J.A.S. **307**

Well-balanced algorithms for relativistic fluids on a Schwarzschild background
Pimentel-García E., Parés C. and LeFloch P.G. **313**

Asymptotic analysis of the behavior of a viscous fluid between two very close mobile surfaces
Rodríguez J.M. and Taboada-Vázquez R. **321**

Convergence rates for Galerkin approximation for magnetohydrodynamic type equations
Rodríguez-Bellido M.A., Rojas-Medar M.A. and Sepúlveda-Cerda A. **325**

Asymptotic aspects of the logistic equation under diffusion
Sabina de Lis J.C. and Segura de León S. **332**

Analysis of turbulence models for flow simulation in the aorta
Santos S., Rojas J.M., Romero P., Lozano M., Conejero J.A. and García-Fernández I. **339**

Overdetermined elliptic problems in onduloid-type domains with general nonlinearities
Wu J. **344**

XXVI Congreso de Ecuaciones Diferenciales y Aplicaciones
XVI Congreso de Matemática Aplicada
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Analysis of a SEIRS metapopulation model with fast migration

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Abstract

Metapopulation models for the study of a infectious disease in a population with space structure involve a large number of equations. Therefore the mathematical analysis of these models yields only partial results. We propose a model in which, as it is often the case in practical situations, the time scale of the transport of individuals is much master that that of the disease. Then we make use of approximate reduction techniques in order to reduce the system's dimension, and carry out a thorough analysis of the reduced model. In particular we characterize the number and stability of equilibria, provide conditions for the disease to become endemic (resp. die out) and show that certain counter-intuitive behaviors can arise.

1. Introduction

Classical deterministic epidemic models assume an homogeneous spatial distribution of individuals. However, travels of individuals between different regions have proved to have a great influence on the spatial spread of diseases. Therefore, given the characteristics of current society, in which most humans live in cities and travel along defined routes, it seems reasonable to include spatial variation into epidemic models.

The spatial spread of infectious diseases is a complex phenomenon to model. The usual approach in the literature is to use the so called metapopulation models, in which the population is distributed into discrete spatial sites, called patches, amongst which they may migrate. This movement of individuals is captured by a directed graph, in which the vertices represent the geographical regions and the arcs represent the connections between them.

Epidemic metapopulation models have been formulated and discussed in the literature for different diseases, see for example [2–4], yielding systems that consist of a large number of ordinary differential equations. This complexity greatly limits the analytical study that can be carried out, and only partial results have been obtained for these models. In particular, in [2], a SEIRS model with spatial distribution is formulated and studied. The expression of the basic reproduction number of the model is derived, but the existence and stability of endemic equilibria is only considered numerically, as an analytical approach seems unfeasible.

Furthermore, in many practical situations it can be assumed that travel of individuals between patches is much faster than the dynamics of the corresponding disease. For example, in the case of human diseases travel between different cities can be done in the span of a few hours, while the development of an infectious disease may take days or even weeks. This fact justifies the use of two different time scales to formulate the disease dynamics and the movement of individuals between sites.

One can make use of the existence of two different time scales in order to obtain a reduced approximation of the model. Indeed, in [7,8] approximate aggregation techniques are presented for the study of complex population dynamics in which two time scales are considered. Loosely speaking, this method consists in taking the fast process in the original model to its equilibrium value, yielding a system whose dimension can be reduced. These techniques allow us to obtain an approximated simplified model that, under the assumption that migration of individuals is sufficiently faster than the disease dynamics, behave qualitatively similar to the original model, while its dimension and, hence, complexity is highly reduced.

The objective of this work is to formulate a SEIRS metapopulation model, derive its approximate reduced version by means of aggregation techniques and carry out an exhaustive study of this simplified system. We aim to obtain stronger results than those already found in the literature for the case of metapopulation models without a time-scale approach.

In Section 2 the model is formulated, considering a metapopulation of r patches in which the local dynamics of the disease in each site is of SEIRS type, whereas migration of individuals among sites is linear. This model is described by a system of $4r$ differential equations. Next, under the assumption that migration of individuals between regions is fast with respect to the disease dynamics, we make use of approximate aggregation techniques in order to reduce the system's dimension. The resulting system consists of only 4 differential equations.

Section 3 carries out a thorough analytical study of the approximated model. The basic reproduction number $\mathcal{R}_0$ of the model is obtained by the next generation matrix method [1,6]. The analysis carried out shows that the model can only have two behaviours: if $\mathcal{R}_0 < 1$ the disease will die out in every region, whereas if $\mathcal{R}_0 > 1$ the disease will be globally endemic.

Moreover, the reduction on the systems dimension has allowed us to obtain results that can not be found in the literature on epidemic metapopulation models without a time-scale approach. In particular, some conclusions are given regarding the existence of endemic equilibria in relation to the value of the basic reproduction number.

Finally, in Section 4 we study the influence that the parameters of migration have on the behaviour of the disease. The analysis shows that the dynamics the disease would have in the isolated patches can vary under the existence of migration of individuals. In particular, under certain conditions a counter-intuitive scenario can arise: let us assume we have r separated sites and in all of them the disease dies out (resp. is endemic). Then if migration of individuals between them is allowed, then for certain values of the parameters regarding migration, the behaviour of the disease may change, i.e. it might become endemic (resp. die out) globally.

The system formulation and results are also particularized for a SIRS model.

2. Formulation and reduction of the model

We consider a population spread out among r different patches amongst which they can migrate, and affected by a disease. We assume that the local dynamics of the disease in each site follows a classical SEIRS model, whereas migration of individuals among sites is linear, see Figure 1.

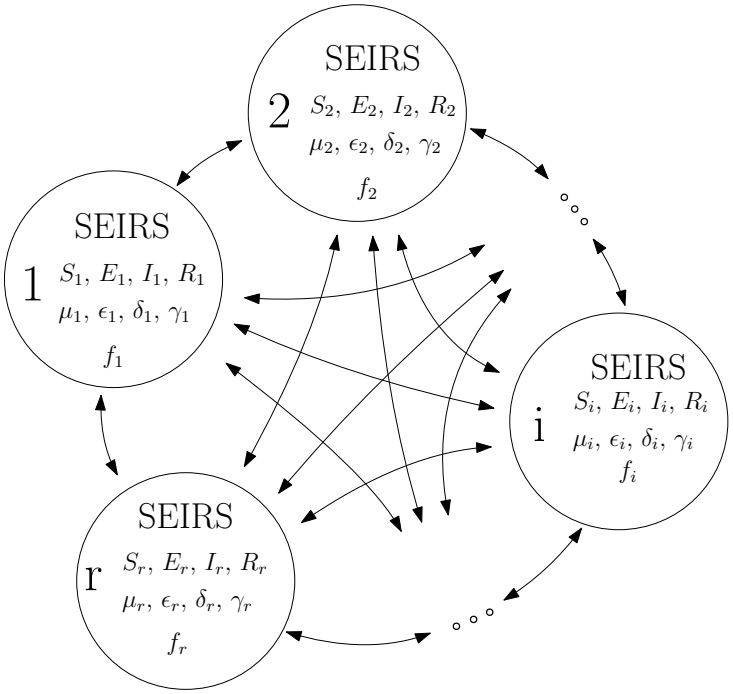

Fig. 1 SEIRS metapopulation model consisting of r interconnected patches.

This setting has been explored in several works [2–4] but the large dimension of the resulting model makes its analytical study very difficult. In our approach we make use of the fact that in many practical situations the time scale of migration is much faster than that of the epidemic process to formulate a two-time scale model.

The "slow time" variable, t, is used to describe the dynamics of the disease, whereas we denote the "fast time "variable as τ. Derivatives of a function g with respect this two different time variables are denoted as $\frac{dg}{dt} := \dot{g}$ and $\frac{dg}{d\tau} := g'$ respectively.

In order to formulate the model, we first consider both phenomena separately, and then combine them into the resulting complete system.

2.1. Local dynamics in each patch

Let S_i, E_i, I_i, and R_i denote the number of susceptible, exposed (infected but can not transmit the disease), infectious (infected and contagious) and recovered individuals who are present in patch i, $i = 1, ..., r$, at time t, respectively. The equations of a classical SEIRS model for patch i are:

$$\begin{aligned}
\dot{S}_i &= -f_i(S_i, E_i, I_i, R_i) + \mu_i N_i - \mu_i S_i + \delta_i R_i \\
\dot{E}_i &= +f_i(S_i, E_i, I_i, R_i) - (\epsilon_i + \mu_i) E_i \\
\dot{I}_i &= \epsilon_i E_i - (\gamma_i + \mu_i) I_i \\
\dot{R}_i &= \gamma_i I_i - (\delta_i + \mu_i) R_i
\end{aligned} \tag{2.1}$$

where the subscript i denotes the patch number each epidemiological variable refers to and the time derivatives are with respect to the slow time t. We denote by f_i the incidence function, over which for the moment we do not impose any condition, μ_i denotes the birth and the death rate of individuals (as it is the case in most models, both are assumed equal so that the total population in each patch remains constant), $1/\epsilon_i$ is the average latency time, $1/\gamma_i$ is the average recovery time for infectious individuals and $1/\delta_i$ is the average time of the immunity period for recovered individuals.

2.2. Migration model

Let us denote $\mathbf{X}(t) := (S_1, ..., S_r, E_1, ..., E_r, I_1, ..., I_r, R_1, ..., R_r)$,

For each $Y \in \{S, E, I, R\}$, let $\mathbf{Y}(t) := (Y_1, ..., Y_r)$ and let

$$Y := \sum_{i=1}^{r} Y_i \tag{2.2}$$

denote the total population in each of the epidemiological classes. We refer to S, E, I and R as "global variables".

We assume that migration between sites is a linear process. The migration rate (with respect to the "fast time") of individuals of each class $\alpha \in \{S, E, I, R\}$ from patch j to i, is denoted by m_{ij}^{α}, $i, j = 1, ..., r$. Therefore, the migration matrix for class α is

$$M^{\alpha} := \begin{pmatrix} -\sum\limits_{i=1, i\neq 1}^{r} m_{i1}^{\alpha} & m_{12}^{\alpha} & \cdots & m_{1r}^{\alpha} \\ m_{21}^{\alpha} & -\sum\limits_{i=1, i\neq 2}^{r} m_{i2}^{\alpha} & \cdots & m_{2r}^{\alpha} \\ \vdots & \vdots & \ddots & \vdots \\ m_{r1}^{\alpha} & m_{r2}^{\alpha} & \cdots & -\sum\limits_{i=1, i\neq r}^{r} m_{ir}^{\alpha} \end{pmatrix},$$

so that it is a Metzler matrix. The matrix characterizing migration for the whole population is then $M = \operatorname{diag}\left(M^S, M^E, M^I, M^R\right)$ and therefore migration dynamics is described by the equation $\mathbf{X}' = M\mathbf{X}$.

We assume that an individual initially present in any patch can (directly or indirectly) travel to any other patch. This assumption results in the fact that that digraph corresponding to the migration is strongly connected and therefore each migration matrix M^{α} is irreducible.

The following result characterizes the asymptotic behavior of migration:

Theorem 2.1 *For each $\alpha \in \{S, E, I, R\}$, let M^{α} be irreducible. Let $\mathbf{v}^{\alpha} > 0$ be the right eigenvector of matrix M^{α} associated to eigenvalue $\lambda = 0$ and normalized so that the sum of its components is 1. Then, if the initial condition $\mathbf{X}(0)$ contains at least an individual in each of the epidemiological clases $\{S, E, I, R\}$, then the dynamics of migration for the whole population tends to the equilibrium*

$$\mathbf{X}_e = \begin{pmatrix} \mathbf{v}^S S \\ \mathbf{v}^E E \\ \mathbf{v}^I I \\ \mathbf{v}^R R \end{pmatrix}, \tag{2.3}$$

where S, E, I and R are given by (2.2).

2.3. Complete model with two time scales

We proceed with the formulation of the complete model, that takes into account the joint effect of the local disease dynamics in each site and inter-site migration. In order to take into account the existence of two different time scales, we define the ratio of characteristic times $\varepsilon := t/\tau$, which under our hypotheses is a small positive number. Putting together the equations regarding the disease dynamics in each site (2.1) with those corresponding to migration, we obtain the following model:

$$\begin{aligned} \mathbf{S}' &= \varepsilon \left[-\mathbf{f}(\mathbf{X}) + D_{\mu}\mathbf{N} - D_{\mu}\mathbf{S} + D_{\delta}\mathbf{R}\right] + M^S\mathbf{S} \\ \mathbf{E}' &= \varepsilon \left[+\mathbf{f}(\mathbf{X}) - \left(D_{\epsilon} + D_{\mu}\right)\mathbf{E}\right] + M^E\mathbf{E} \\ \mathbf{I}' &= \varepsilon \left[D_{\epsilon}\mathbf{E} - \left(D_{\mu} + D_{\gamma}\right)\mathbf{I}\right] + M^I\mathbf{I} \\ \mathbf{R}' &= \varepsilon \left[D_{\gamma}\mathbf{I} - \left(D_{\delta} + D_{\mu}\right)\mathbf{R}\right] + M^R\mathbf{R}, \end{aligned} \tag{2.4}$$

where $\mathbf{f}(\mathbf{X}) := \begin{pmatrix} f_1(\mathbf{X}_1) \\ \vdots \\ f_r(\mathbf{X}_r) \end{pmatrix}$ and $D_{\sigma} := \operatorname{diag}(\sigma_1, ..., \sigma_r)$ for $\sigma \in \{\mu, \delta, \epsilon, \gamma\}$.

2.4. Reduction of the model

In order to carry out the analysis of model (2.4), we make use of the existence of different time scales and apply approximate aggregation techniques (see [7, 8] for a survey and a collection of the main results) in order to reduce its dimension. The reduction procedure consists on replacing the population vector with its equilibrium value for the migration process, i.e., replacing $\mathbf{X}(t)$ with $\mathbf{X}_e$ given by (2.3) and then summing the equations corresponding to each infectious class, in such a way that we obtain an autonomous model in the global variables S, E, I and R.

The resulting reduced or aggregated model is:

$$\begin{aligned}
\dot{S} =& - f(S,E,I,R) + \mu^I I + \mu^E E + \left(\mu^R + \delta\right) R \\
\dot{E} =& + f(S,E,I,R) - \left(\epsilon + \mu^E\right) E \\
\dot{I} =& \epsilon E - \left(\mu^I + \gamma\right) I \\
\dot{R} =& \gamma I - \left(\delta + \mu^R\right) R.
\end{aligned} \tag{2.5}$$

where

$$\begin{aligned}
f(S,E,I,R) &:= \sum_{i=1}^{r} f_i \left(v_i^S S, v_i^E E, v_i^I I, v_i^R R\right) \\
\mu^S &:= \sum_{i=1}^{r} \mu_i v_i^S \in \mathbb{R}\,,\ \mu^E := \sum_{i=1}^{r} \mu_i v_i^E \in \mathbb{R} \\
\mu^R &:= \sum_{i=1}^{r} \mu_i v_i^R \in \mathbb{R}\,,\ \mu^I := \sum_{i=1}^{r} \mu_i v_i^I \in \mathbb{R} \\
\delta &:= \sum_{i=1}^{r} \delta_i v_i^R \in \mathbb{R}\,,\ \gamma := \sum_{i=1}^{r} \gamma_i v_i^I \in \mathbb{R} \\
\epsilon &:= \sum_{i=1}^{r} \epsilon_i v_i^E \in \mathbb{R}
\end{aligned}$$

Note that the reduced model consists of only 4 differential equations, in contrast to the $4r$ equations that constitute the original metapopulation model (2.4).

The results in the field of approximate aggregation techniques allow us to claim that, loosely speaking, if the reduced model has an attractor which is locally structurally stable (in particular this holds for a hyperbolic equilibrium) and ϵ is small enough, i.e., if the separation of time scales between the disease dynamics and migration is large enough, then the original model also has a corresponding attractor which is $O(\epsilon)$-close and has the same stability properties. Therefore, the analysis of the reduced system provides relevant qualitative information about the behavior of the original model.

3. Analysis of the reduced model

In contrast with the original model (2.4), model (2.5) is amenable to an analytical study. In order to do so we will consider, as most epidemiological models do, the case of standard incidence in each patch, i.e., $f_i(S_i, E_i, I_i, R_i) = \beta_i \frac{I_i S_i}{N_i}$, and so we have

$$f(S,E,I,R) = \sum_{i=1}^{r} \beta_i \frac{v_i^S v_i^I}{v_i^S S + v_i^E E + v_i^I I + v_i^R R} IS.$$

It must be taken into account that the total population size ($N := S + E + I + R$) remains constant, so the analysis of the model can be reduced to 3 equations, for example those of E, I, R.

It is straightforward to show that for all non-negative initial conditions system (2.5) has an unique solution for all times and the solution remains non-negative. The study will be reduced to region

$$K := \{(E,I,R) : E \geq 0, I \geq 0, R \geq 0, E + I + R \leq N\}, \tag{3.1}$$

as only solutions inside that region have an epidemiological interpretation. It can be shown taht that K is positively invariant, i.e. solutions lie in K for all positive times given initial conditions inside that region. Axis $\{E = 0\} \cap \{I = 0\}$ is also positively invariant.

	Number of EE	Stability of DFE	Stability of EE	Case number
$\mathcal{R}_0 < 1$	0	GAS	-	1
$\mathcal{R}_0 > 1$	1	US	GAS (*)	2

Tab. 1 Summary of possible scenarios of aggregated model

(*) Not analytically proven.

The basic reproduction number of the model can be calculated by using the next generation matrix approach [1,6], yielding

$$\mathcal{R}_0 = \frac{\epsilon \sum_{i=1}^{r} \beta_i v_i^I}{\left(\epsilon + \mu^E\right)\left(\mu^I + \gamma\right)}. \tag{3.2}$$

Next, we study the stability of the disease free equilibrium (DFE) and the existence of endemic equilibria (EE) in relation to the value of $\mathcal{R}_0$.

Let $\bar{S} := S/N$, $\bar{E} := E/N$, $\bar{I} := I/N$, and $\bar{R} := R/N$ denote the proportions of individuals in each class. We define

$$g(\bar{I}) := \left(1 - \frac{\bar{I}}{H}\right) \sum_{i=1}^{r} \beta_i \frac{v_i^S v_i^I}{v_i^S + \left(v_i^I + b v_i^E + a v_i^R - \frac{v_i^S}{H}\right)\bar{I}}, \tag{3.3}$$

where

$$\sigma := \frac{\epsilon}{\left(\epsilon + \mu^E\right)\left(\gamma + \mu^I\right)}, \tag{3.4}$$

$$H := \frac{\epsilon\left(\delta + \mu^R\right)}{\epsilon\gamma + \left(\delta + \mu^R\right)\left(\epsilon + \gamma + \mu^I\right)}, \tag{3.5}$$

$$a := \frac{\gamma}{\delta + \mu^R}, \tag{3.6}$$

$$b := \frac{\gamma + \mu^I}{\epsilon}. \tag{3.7}$$

Then we have following result:

Theorem 3.1 *Let us consider the reduced model* (2.5)*, for which the basic reproduction number is given by* (3.2)*. Then*:

1. *If* $\mathcal{R}_0 < 1$ *the DFE is hyperbolic and globally asymptotically stable* (*GAS*) *in region K, so the disease will die out asymptotically for any initial condition. No endemic equilibrium exists in this case.*

2. *If* $\mathcal{R}_0 > 1$ *the DFE is hyperbolic and unstable* (*US*)*. There exists a unique endemic equilibrium with the form* $(\bar{E}_e, \bar{I}_e, \bar{R}_e)$*, where* $\bar{I}_e$ *is the only solution to the equation*

$$g(\bar{I}) = \frac{1}{\sigma} \tag{3.8}$$

in the interval $\bar{I}_e \in (0, H)$*, and* $\overline{E}_e = b\bar{I}_e$*,* $\overline{R}_e = a\bar{I}_e$*, where* $g(\bar{I})$*,* σ*, H, a and b are given by* (3.3-3.7)*.*

Furthermore, although we have not been able to showed it analytically, if $\mathcal{R}_0 > 1$ simulations suggest that the EE of the model is GAS in K, and so the disease will be endemic for any initial condition with infected individuals.

Table 1 summarizes the two possible scenarios. For the particular case of a two region metapopulation (i.e. $r = 2$), Figures 2 (a) and (b) show the three dimensional EIR phase diagrams for the two cases $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$.

3.1. Particular case of a SIRS model

The classical SIRS model has the same structure than the SEIRS model, but without latency period, so the exposed class is not considered. Expressions for a SIRS model can be obtained from those of the SEIRS by letting $\epsilon \to \infty$. Therefore, the reduced model is

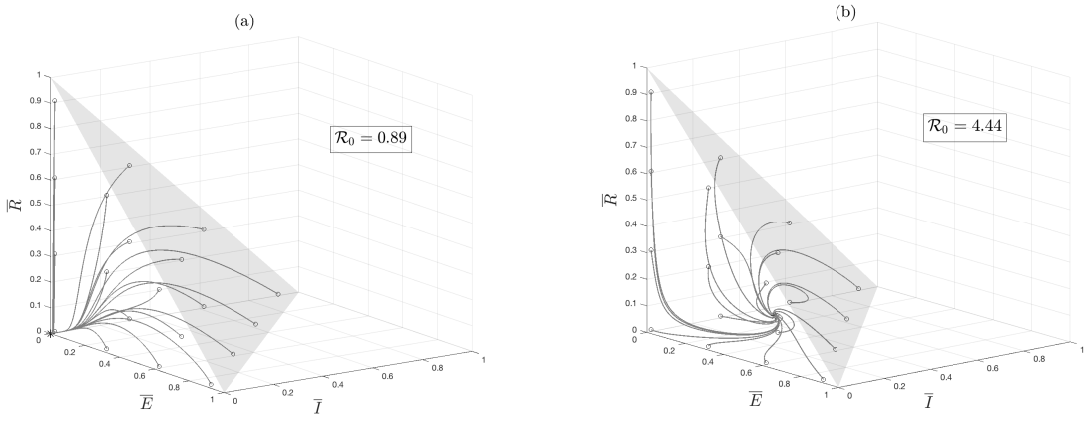

Fig. 2 (a) Case 1: DFE GAS. (b) Case 2: EE GAS.

$$\begin{aligned}
\dot{S} =& - f(S,E,I,R) + \mu^I I + \left(\mu^R + \delta\right) R \\
\dot{I} =& + f(S,E,I,R) - \left(\mu^I + \gamma\right) I \\
\dot{R} =& \gamma I - \left(\delta + \mu^R\right) R.
\end{aligned} \tag{3.9}$$

The basic reproduction number for this model is

$$\mathcal{R}_0 = \frac{\sum_{i=1}^{r} \beta_i v_i^I}{\left(\mu^I + \gamma\right)}, \tag{3.10}$$

and results of Theorem 3.1 apply.

4. Discussion: Influence of migration in the dynamics of the disease

One of the main purposes of this work is to evaluate how migration of individuals between sites can affect the behaviour of the disease. Namely, we aim to explore the possibility that an endemic behaviour of the disease on the isolated sites might be modified by the existence of migration between them.

First of all, it is important to observe that, according to the model, the disease will have the same behaviour in all interconnected regions, i.e., either the disease will be endemic in all of the sites, or it will die out globally. In the first case ($\mathcal{R}_0 > 1$), the asymptotic distribution of individuals of each class α between the different patches can be obtained by the eigenvector $\mathbf{v}^\alpha$, that, as stated in Theorem 2.3, provides the equilibrium distribution for migration of those individuals.

In addition, we want to study the possibility that, provided that the behaviour of the disease is the same in all patches separately, the migration of individuals amongst the sites could alter this behavior. Intuitively, we might consider that this can not happen, but in fact it can be proven that this counter-intuitive scenario may arise. That is, even when the basic reproduction number in every patch separately is $\mathcal{R}_0^i < 1$ (resp. $\mathcal{R}_0^i > 1$), the global basic reproduction number of the aggregated model could be $\mathcal{R}_0 > 1$ (resp. $\mathcal{R}_0 < 1$).

It is important to remark that this counter-intuitive situation is only possible under certain circumstances. In particular, it can not happen if the value of parameters μ_i, γ_i and ε_i is the same for all patches i, i.e., if the birth-death parameters and those regarding latency time, recovery time and incubation time are the same in all sites.

For the particular case of a two region metapopulation, Figure 3 shows an example of this situation, in which the disease dies out in both patches 1 and 2 if they are separated, but it becomes endemic if individuals can travel between the sites. For this case ($r = 2$), this situation arises if and only if the following conditions are satisfied:

$$\begin{aligned}
\max\left\{\frac{\epsilon_1}{\epsilon_1 + \mu_1}, \frac{\gamma_1 + \mu_1}{\beta_1}\right\} &< \frac{\epsilon_2}{\epsilon_2 + \mu_2} \\
\frac{\beta_2}{\beta_1} &< \frac{\gamma_2 + \mu_2}{\gamma_1 + \mu_1}.
\end{aligned}$$

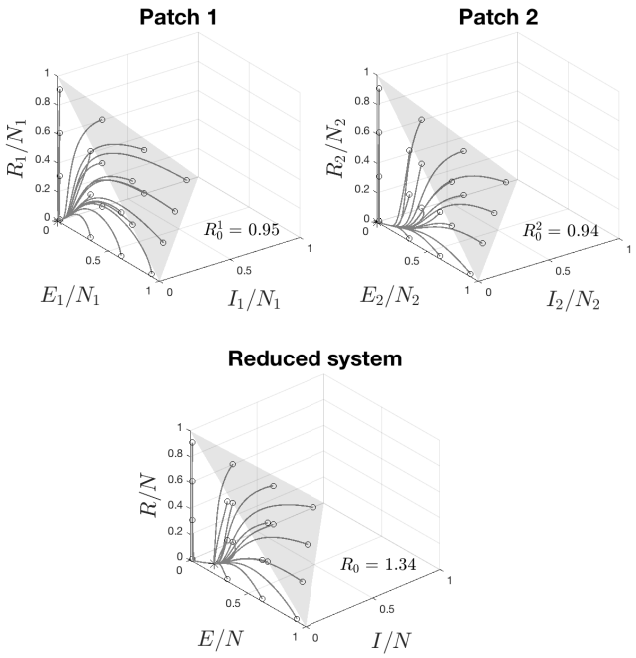

Fig. 3 Counter-intuitive scenario for a two region metapopulation

In the particular case of a SIRS model it can be proven that the counter-intuitive situation presented above cannot occur, i.e., if $\mathcal{R}_0^i < 1$ (resp. $\mathcal{R}_0^i > 1$) for all $i = 1, ..., r$, the global basic reproduction number of the reduced model is $\mathcal{R}_0 > 1$ (resp. $\mathcal{R}_0 < 1$).

In conclusion, our analysis shows that migration of individuals between different sites has a great influence on the behaviour of the disease, and we can quantify how the values of the parameters affecting migration affect this behavior.

Further study will be carried out in this field. In the first place, we want to generalize the study to other forms of incidence functions (amongst them mass action incidence) and analyse the more realistic situation in which the recruitment of individuals is not linear and, correspondingly, the local population in each patch is not necessarily constant.

Additionally, we will extend this analysis of models with time scales to SLIAR models [5], a family of models that can be used for the study of epidemics such as COVID-19.

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