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Local epidemic control through mobility restrictions

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Abstract: Epidemic severity indices that incorporate disease information are essential tools for decision-makers, as these indices allow the design and evaluation of possible control strategies in advance of implementation in susceptible populations. In spatially structured settings, indices that consider human mobility provide valuable information on the spread of infectious diseases and the potential impact of mobility restrictions during outbreaks. In this context, the final epidemic size in metapopulation models serves as an effective measure of outbreak severity in geographical terms. However, the existence and uniqueness of the solution to the corresponding equation have only been established in particular cases. In this study, we derived conditions that guarantee the existence and uniqueness of the solution to the final epidemic size equation in a SIR-type metapopulation model. We also conducted a sensitivity analysis in a two-region, unidirectional infection scenario, which allowed us to examine the effects of mobility between an infected region and a susceptible one. Our results indicate that, under relatively simple conditions, restricting mobility can help contain outbreaks. However, we also identified situations in which mobility is not detrimental and may even be beneficial. These findings provide a preliminary framework for assessing the appropriateness of mobility restrictions during infectious disease outbreaks in spatially structured regions.

Keywords: Mobility in epidemics; epidemic final size; control strategies; severity indices

1. Introduction

One of the main reflections left by the COVID-19 pandemic in modern societies is the need to adopt new social, political, and cultural behaviors to address future high-impact infectious outbreaks that can spread on a large geographic scale. The use of face masks in high-density settings, timely maintenance and renovation of public health infrastructure, vaccination of vulnerable populations, constant monitoring of the evolution of the virus and its various mutations, and the proposal of different pharmacological treatments for sick patients were some of the actions implemented to contain the disease in susceptible populations [1–3].

Furthermore, the high degree of interconnectedness between countries in a completely globalized society, the current population densities in the world's major metropolises, and natural human mobility have generated deep concern among governments about the potential socioeconomic consequences of new epidemic outbreaks that have the capacity to become not only pandemics but endemic diseases that are impossible to eradicate [4–6].

The COVID-19 pandemic also demonstrated the rapidity with which an infectious disease can spread among susceptible populations, even under the application of strict virus control and containment policies (partial and total quarantines) from the early stages of detection [7, 8].

In this sense, it is essential to quantify the severity of the consequences of an epidemic outbreak, not only in the region where it originated, but also in all regions where the epidemic could have spread. This is done in order to determine which areas are most affected and, therefore, which require the most resources to mitigate the consequences of outbreaks caused by this type of infectious disease.

In this context, mathematical modeling (and particularly mathematical epidemiology) has generated a wide range of research focused on analyzing the dynamics of emerging infectious diseases with significant epidemic impact [2, 9–11]. These studies include both theoretical analyses and the use of real databases to estimate key parameters governing disease dynamics. In the case of the COVID-19 pandemic, the mathematical community showed great interest in developing various models that reproduced the dynamics of infectious outbreaks in isolated populations [12–15] as well as in populations connected through human mobility [16–24]. These works contributed to a better understanding of how the disease spread globally and to identifying the most effective control strategies to contain the virus, while considering the socioeconomic costs associated with these measures [25].

In addition to this, a particular interest of mathematical modeling has always been to provide measures that quantify the severity of a high-impact infectious outbreak. An example of this is the interest in calculating the basic reproductive number or equivalent measures [19, 26]. However, one measure that has sparked great interest in mathematical modeling is the epidemic final size, which quantifies how many susceptible people in an isolated region contract the disease at the end of the infectious outbreak. Nevertheless, when we want to observe this phenomenon in spatially structured regions, it is necessary to resort to epidemiological modeling using metapopulation networks [27–29].

Metapopulation models have been used not only as proposals to describe the dynamics of infectious diseases in spatially structured regions [20–25, 27–31] but have also served as a basis for estimating parameters of human mobility between regions with the help of databases and Bayesian inference (see Ramirez et al. [19] and Akuno et al. [17, 18] and some of the references therein). However, theoretical analyses of these types of models are often complex, and proposing and analyzing measures to quantify the severity of infectious outbreaks remain a challenge for the scientific community.

Some research has advanced the theoretical analysis of metapopulation models [19–24, 27–29], where the authors conducted theoretical analyses of the stability and soundness of the proposed metapopulation model. On the other hand, Kühn et al. [16], using a modified SEIR-type metapopulation model, conducted a numerical analysis of the consequences of quarantines and how, in certain situations, without substantial mobility restrictions (only preventing travel by infected individuals), in a highly connected regional environment, epidemic mitigation is possible. This is achieved when testing for infection is carried out appropriately and frequently in populations with high epidemic incidence and when local outbreaks are contained promptly, indicating that there are other determining factors, besides mobility that trigger infectious outbreaks in regions with a specific spatial structure. This control strategy reduces the social and economic costs incurred by total quarantines in modern societies with high human mobility.

In [25, 30, 31], the authors have calculated the epidemic final size for susceptible-infectious-recovered (SIR) diseases in multigroup and metapopulation models, providing us with explicit expressions of this measure for each region of the network. This has made it possible to establish explicit measures that quantify the severity of the epidemic, both in each individual population and in the overall network. Furthermore, these works provide simple iterative algorithms that allow these expressions to be calculated under specific hypotheses and scenarios.

However, studies that calculate the final epidemic size have been limited to performing in-depth theoretical analyses of this equation for specific cases. Some advances in the literature for this case study are presented in [32–35], which demonstrate, in the case of an isolated population, the existence and uniqueness of the solution to this equation for SIR models. On the other hand, the work by Pierre Magal et al. [30], for a multi-group SIR model in an n-dimensional network establishes the conditions for the existence and uniqueness of the solutions to this equation. In particular, the result holds when the infection-rate matrix between susceptible individuals in group i and infectious individuals in group j, defined by $B = (\beta_{ij})_{i,j=1}^{n}$, is nonnegative, nonzero, and irreducible, especially in the case where this matrix is lower triangular. On the other hand, the work presented in [25, 31] presents more general SIR and SEIR metapopulation models, respectively, and demonstrates methodologies that allow the calculation of the value of this measure through an iterative process (based on the works [33, 34, 36]), where convergence is guaranteed when the solution to this equation exists and is unique.

For the reasons stated above, in this work, using fixed-point theory, we propose a test that guarantees the existence and uniqueness of the solution to the final epidemic size equation of the metapopulation model presented in [31]. This is a generalization of the model presented in [20] and includes all possible interactions between individuals in a network of dimension n through human mobility. Only general conditions will be imposed on the parameters used in this model, which extends the usefulness of this measure to more general scenarios. As a result, the calculation of this measure can be guaranteed through the iterative processes shown in [25, 31]. Likewise, a parametric sensitivity analysis was carried out in a particular case that considers two regions unidirectionally connected by human mobility with the aim of evaluating how a disease can invade a susceptible population through contacts established with infected individuals from the issuing region. This provides evidence that human mobility is a relevant factor in the spread of this type of infectious diseases in regions with a spatial structure, which allows us to propose specific control strategies for different local scenarios that both populations may present.

This document is organized as follows. In Section 2, an epidemiological model is presented that

describes the dynamics of an SIR-type infectious disease on an n-dimensional metapopulation network in which the connectivity between regions is determined by human mobility. Likewise, the expression corresponding to the final size of the epidemic in said network is obtained. In Section 3, through fixed-point theory, necessary and sufficient conditions are established that guarantee the existence and uniqueness of the solution to the equation associated with the final size of the epidemic in the proposed model. In Section 4, a sensitivity analysis is carried out with respect to the mobility parameter, considering the particular case of two regions unidirectionally connected by human mobility, with the purpose of determining how the interaction between geographically separated regions influences the spread of this type of disease. Finally, in Section 5, we present our conclusions.

2. Epidemic final size in a networked population

Consider the following system of ordinary differential equations that describes the dynamics of a human-to-human SIR-type infectious disease in an n-patched metapopulation network [30, 31]:

$$\frac{d\mathrm{S}_i(t)}{dt} = -\mathrm{S}_i(t)\sum_{j=1}^{n}\beta_{ij}I_j(t)\,, \tag{2.1}$$

$$\frac{dI_i(t)}{dt} = \mathrm{S}_i(t)\sum_{j=1}^{n}\beta_{ij}I_j(t) - \gamma_i I_i(t)\,, \tag{2.2}$$

$$\frac{dR_i(t)}{dt} = \gamma_i I_i(t)\,, \tag{2.3}$$

where each patch is labeled with the subscript i, and each of them is inhabited by a homogeneously mixed human population with size N_i that can be classified into susceptible residents $S_i(t)$, infected residents $I_i(t)$, and recovered residents $R_i(t)$ such that for any time instant t, it holds that $N_i = S_i(t) + I_i(t) + R_i(t)$. That is, each population remains constant throughout the epidemic. Furthermore, the parameter $\gamma_i > 0$ describes the recovery rate of infected residents in each patch i, while the parameter β_{ij} represents the effective risks of infection between the inhabitants of patches i and j, which is defined by

$$\beta_{ij} = \sum_{k=1}^{n}\beta_k\frac{p_{ik}p_{jk}}{P_k}\,; \quad \forall i, j \in \{1, ..., n\}\,, \tag{2.4}$$

where β_k is the effective infection rate of patch k, while the parameters p_{ij} indicate the fraction of the population of patch i that is located in patch j, thus describing the mobility of humans between patches in the network. Due to their meaning, the p_{ij} should satisfy

$$0 \le p_{ij} \le 1\,; \quad \text{and} \quad \sum_{k=1}^{n} p_{ik} = 1 \quad \forall i, j\,. \tag{2.5}$$

This parameterization corresponds to the Lagrangian description of mobility [37, 38].

Furthermore, due to human mobility between network patches, the effective number of people present in patch i is given by $P_i = \sum_{j=1}^{n} p_{ji}N_j$, where the fraction of residents staying in their own patch is given by $p_{ii}N_i$, while the fraction of neighboring residents visiting patch i daily is given by

$p_{ji}N_j$, with $j = 1, \dots n$. It is worth noting that for the calculation of the mobility parameters p_{ij}, it is assumed that the mobility of infected individuals is the same as that of non-infected individuals.

In the case where β_{ij} remains constant at any time instant t, the metapopulation model (2.1)–(2.3) could be interpreted as a multi-group epidemic model, where β_{ij} is the contact rate between residents of patch i and patch j as analyzed by Pierre Magal et al. [30]. However, for our case study, the interpretation taken by β_{ij} has different meaning, being the effective infection rate of residents of patch i with infected individuals of patch j, considering even the contacts occurring in patches $k \in \{1, \dots, n\}$ with $k \neq i, j$. Because the model assumes that the mobility of infected individuals does not change significantly over time, this model can be applied to diseases for which symptoms do not considerably affect individuals in two scenarios: the first one would be that once they recover from the disease, they acquire permanent immunity and the second if only a single wave of infection is to be quantified, and the recovery gives temporary immunity for a significant time.

In the context of mathematical epidemiology, the final epidemic size is the total number of infected individuals over the entire duration of an infectious outbreak [9, 10, 25, 30, 31, 35]. A common methodology for assessing this is by mathematically expressing the total number of recovered individuals = $R_i(\infty)$, that is, by explicitly solving the recovery compartment (Eq (2.3)) and evaluating the bound as time approaches infinity.

In order to derive an explicit expression for $R_i(\infty)$ in each patch, we sum and integrate the susceptible $S_i(t)$ (Eq (2.1)) and infected $I_i(t)$ (Eq (2.2)) compartments and obtain

$$-\gamma_i^{-1} \int_0^\infty \left(\frac{dS_i(t)}{dt} + \frac{dI_i(t)}{dt} \right) dt = \frac{N_i - S_i(\infty)}{\gamma_i} = \int_0^\infty I_i(t) dt \,, \tag{2.6}$$

where we have used

$$I_i(\infty) = \lim_{t \to \infty} I_i(t) = 0 \,, \qquad R_i(0) = 0 \quad \text{and} \quad N_i = S_i(0) + I_i(0) \quad \forall i. \tag{2.7}$$

On the other hand, from Eq (2.1) and Eq (2.6), we obtain

$$\log \left(\frac{S_i(0)}{S_i(\infty)} \right) = \sum_{j=1}^{n} \beta_{ij} \int_0^\infty I_j(t) dt = \sum_{j=1}^{n} \beta_{ij} \frac{N_j - S_j(\infty)}{\gamma_j} \,, \tag{2.8}$$

then $S_i(\infty) = S_i(0) e^{-\theta_i}$, where the expressions

$$\theta_i(S_1(\infty), \dots, S_n(\infty)) = \sum_{j=1}^{n} \beta_{ij} \frac{N_j - S_j(\infty)}{\gamma_j} \,, \tag{2.9}$$

for each patch $i = 1, \dots, n$, determine the total number of susceptible individuals who did not contract the disease by the end of the infectious outbreak.

Because $N_i = S_i(t) + I_i(t) + R_i(t)$ remains constant all the time, by Eq (2.7), when $t \to \infty$, we obtain $R_i(\infty) = N_i - S_i(\infty)$. Then, the equation satisfies the epidemic final size in a given patch i is

$$R_i(\infty) = N_i - S_i(0) e^{-\theta_i (R_1(\infty), ..., R_n(\infty))} \,, \qquad \text{with} \quad i = 1, \dots, n, \tag{2.10}$$

where

$$\theta_i(R_1(\infty), \dots, R_n(\infty)) = \sum_{j=1}^{n} \frac{\beta_{ij}}{\gamma_j} R_j(\infty). \tag{2.11}$$

Remark: As in the case of an isolated population, in a metapopulation network, although we have an explicit expression for the final disease size, solving this equation represents a very difficult problem due to its implicit dependence on the terms $R_1(\infty), \ldots, R_n(\infty)$.

In a related context, J. Miller [33, 35, 39] derived expressions similar to Eq (2.10) for stochastic SIR models with the main focus on using probability generating functions to study infectious diseases. This approach allows the calculation of the final size distribution in multigroup stochastic models, which aligns with the scope of our proposed SIR model (2.1)–(2.3). On the other hand, P. Magal et. al. [30] managed to establish existence and uniqueness conditions for a solution to this equation in the case of irreducible infectious transmissions, that is, when the matrix whose entries are β_{ij} is irreducible and nonnegative or, in other words, the matrix has no nonzero invariant eigenspaces. In our case, the matrix defined by the coefficients β_{ij} is a nonnegative matrix with always real eigenvalues that may have nontrivial invariant eigenspaces, which may limit the application of this procedure.

Works such as [25, 31] present discrete-time iterative algorithms for solving Eq (2.10) based on the concept of discrete generations (or ranges), introduced by D. Ludwig [32] and extended in later work by L. Pellis et. al. and J. Miller [39, 40]. By constructing a directed graph of all possible transmission events, an individual's range is defined as the shortest transmission path from any initial infection. Despite temporal variations in transmission chains, this rank-based contact process often produces the same end result as the continuous-time epidemic, justifying a discrete time frame. In this sense, the discrete equation associated with θ_i in Eq (2.11) denotes the expected cumulative exposure that a random individual from group i has received according to rank k, while the discrete equation associated with $e^{-\theta_i}$ gives the probability of escaping infection up to that point. Therefore, the iterative method converges to a fixed point that quantifies the final proportion of recoveries that remain uninfected after an infinite number of ranks. This equivalence, rigorously supported by branching process theory and probability generating functions [33, 34], extends to structured and multi-group populations [36].

However, these algorithms depend on the existence of a solution to Eq (2.10), so there is no criterion that guarantees the existence and uniqueness of said solution in cases of metapopulation models where the β_{ij} matrix only has nonnegative entries. Next, we will present a procedure for writing the final epidemic size equation (2.10) as a fixed-point problem. This allows us to impose conditions on the model's epidemiological parameters (2.1)–(2.3) which guarantee the existence and uniqueness of a solution to this equation.

3. Existence and uniqueness of a solution of the final size equation of the epidemic

In this section, we will provide a proof that guarantees the existence and uniqueness of the solution to the final epidemic size equation (Eq (2.10)) associated with the system (2.1)–(2.3) using fixed-point theory applied to an equivalent problem of the form $F(X) = X$.

3.1. Equivalence of problems.

Let us define $g_i = \dfrac{R_i(\infty)}{N_i} \in [0, 1]$. Then, from Eqs (2.10) and (2.11), we have

$$g_i = 1 - \frac{S_i(0)}{N_i} e^{-\sum_{j=1}^{n} \frac{\beta_{ij} N_j}{\gamma_j N_j} R_j(\infty)},$$

$$= 1 - \frac{S_i(0)}{N_i} e^{-\sum_{j=1}^{n} \frac{\beta_{ij} N_j}{\gamma_j} g_j}.$$

Thus, denoting by $x_i = 1 - g_i$, which represents the proportion of individuals who do not contract the disease in patch i, we obtain that

$$\begin{aligned} x_i &= \frac{S_i(0)}{N_i} e^{-\sum_{j=1}^{n} \frac{\beta_{ij} N_j \left(1 - x_j\right)}{\gamma_j}}, \\ &= \frac{S_i(0)}{N_i} e^{-\sum_{j=1}^{n} \frac{\beta_{ij} N_j}{\gamma_j}} e^{\sum_{j=1}^{n} \frac{\beta_{ij} N_j x_j}{\gamma_j}}. \end{aligned}$$

Then, if we define $F : [0,1]^n \to [0,1]^n$ such that for $X = (x_1, \ldots, x_n) \in [0,1]^n$, we have that $F(x_1, \ldots, x_n) = (f_1(x_1, \ldots, x_n), \ldots, f_n(x_1, \ldots, x_n))$, where

$$f_i(x_1, \ldots, x_n) = \alpha_i e^{\sum_{j=1}^{n} \frac{\beta_{ij} N_j x_j}{\gamma_j}}, \tag{3.1}$$

with

$$\alpha_i = \frac{S_i(0)}{N_i} e^{-\sum_{j=1}^{n} \frac{\beta_{ij} N_j}{\gamma_j}}. \tag{3.2}$$

Therefore, the system of equations (2.10) for the epidemic final size can be reduced to a vector equation of the form

$$F(X) = X, \tag{3.3}$$

which tells us that proving the existence and uniqueness of solutions to (2.10) is equivalent to proving the existence and uniqueness of a solution to Eq (3.3), but the advantage of this equation is that we reduce the problem to finding a fixed point for function F.

3.2. Existence and uniqueness of solution to the fixed point problem.

One of the most common ways to guarantee the existence and uniqueness of equations of the form $F(X) = X$ is through contractivity criteria associated with fixed-point theory, which also allows solving this type of equations through iterative processes with initial conditions. This methodology will allow establishing conditions on the model parameters (2.1)–(2.3), where the final size equation (2.10) has a unique solution. To do this, it will be necessary to present the following theorem:

Theorem 3.1. *[41][Theorem 10.6] Let $D = \{(x_1, x_2, \ldots, x_n)^t | \, a_i \le x_i \le b_i \text{ ,for each } i = 1, 2, \ldots, n\}$ for some collection of constants $a_1, a_2, \ldots, a_n$ and $b_1, b_2, \ldots, b_n$. Suppose F is a continuous function from $D \subset \mathbb{R}^n \longrightarrow \mathbb{R}^n$ with the property that $F(X) \in D$ whenever $X \in D$. Then F has a fixed point in D.*

Moreover, suppose that all the component functions of F have continuous partial derivatives, and a constant $q < 1$ exists with

$$\left|\frac{\partial f_i(X)}{\partial x_j}\right| \leq \frac{q}{n}, \qquad \text{whenever } X \in D,$$

for each $j = 1, 2. \ldots, n$ and each component function f_i. Then the sequence $(X^{(k)})_{k=0}^{\infty}$, defined by an arbitrarily selected $X^{(0)}$ in D and generated by

$$X^{(k)} = F(X^{(k-1)}), \qquad \text{for each } k \geq 1,$$

converges to the unique fixed point $X^* \in D$, and

$$\| X^{(k)} - X^* \|_\infty \leq \frac{q^k}{1-q} \| X^{(1)} - X^{(0)} \|_\infty .$$

To apply this result to our case study, let us first define A as the matrix whose entries are the coefficients

$$a_{il} = \beta_{il}\frac{N_l}{\gamma_l}; \quad \forall i, l \in \{1, ..., n\}. \tag{3.4}$$

$$J_F^\top = \begin{pmatrix} a_{11} & a_{21} & \ldots & a_{n1} \\ a_{12} & a_{22} & \ldots & a_{n2} \\ . & . & \ldots & . \\ . & . & \ldots & . \\ . & . & \ldots & . \\ a_{1n} & a_{2n} & \ldots & a_{nn} \end{pmatrix} \begin{pmatrix} f_1 & 0 & \ldots & 0 \\ 0 & f_2 & \ldots & 0 \\ . & . & \ldots & . \\ . & . & \ldots & . \\ . & . & \ldots & . \\ 0 & 0 & \ldots & f_n \end{pmatrix}. \tag{3.5}$$

In a simplified form the previous equation is $J_F^\top = A^\top[F(x)]$, where $[F(x)]$ denotes the matrix in the right side of equation (3.5). Because $\| J_F(X) \|_\infty = \| J_F^\top(X) \|_1$ and $\| F(X) \|_\infty = \| F(X) \|_1$, we can bound the infinity norm of the Jacobian matrix J_F by

$$\| J_F(X) \|_\infty \leq \| A \|_\infty \| F(X) \|_\infty, \qquad \forall X \in [0, 1]^n. \tag{3.6}$$

Now, let us consider the closed set Ω_q as

$$\Omega_q = \{X \in [0, 1]^n : \| A \|_\infty \| F(X) \|_\infty \leq q < 1\}, \tag{3.7}$$

on which it is fulfilled:

$$\| J_F(X) \|_\infty \leq q < 1. \tag{3.8}$$

Assuming Ω_q is not empty, we have the following proposition:

Proposition 1. *The set Ω_q is convex.*

Proof. From the fact that the exponential function is convex, it follows that each component function $f_i(X)$ of the vector function $F(X)$ is also convex. Then for

$$X, Y \in \Omega_q \qquad \text{and} \qquad 0 \leq \lambda \leq 1,$$

we obtain

$$\begin{aligned} \| F(\lambda X + (1-\lambda)Y) \|_\infty &= \max_{1\le i\le n} | f_i(\lambda X + (1-\lambda)Y) |, \\ &\le \max_{1\le i\le n} | \lambda f_i(X) + (1-\lambda) f_i(Y) |, \\ &\le \lambda \max_{1\le i\le n} | f_i(X) | + (1-\lambda) \max_{1\le i\le n} | f_i(Y) |, \\ &\le \lambda \frac{q}{\| A \|_\infty} + (1-\lambda)\frac{q}{\| A \|_\infty}, \\ &\le \frac{q}{\| A \|_\infty}. \end{aligned}$$

Therefore $\lambda X + (1-\lambda)Y \in \Omega_q$, and so Ω_q is convex. □

With this result, the following is concluded:

Proposition 2. *For any $0 < q < 1$, the vector function F is contracting at Ω_q of rate q.*

Finally, by defining the set D_q as:

$$D_q = \left\{ Y \in \mathbb{R}^n_+ : | Y |_\infty \le \frac{q}{\| A \|_\infty} \right\}, \tag{3.9}$$

it is easy to see that Ω_q is the preimage of D_q by the function F; it follows from the fact that in this case we can identify $\| F(X) \|_\infty = | F(X) |_\infty$. Then, if the function F had a fixed point X^*, the fixed point would have to satisfy:

$$| F(X^*) |_\infty \| A \|_\infty \le q,$$

and therefore

$$| X^* |_\infty \| A \|_\infty \le q,$$

which implies that $X^* \in D_q$, which suggests looking for the fixed point of F in the set D_q. With this, we have the following theorem:

Theorem 3.2. *Let us denote by α the vector with components α_i, with $i = 1 \ldots, n$ and suppose that $| \alpha |_\infty \| A \|_\infty < 1/e$. Then*

$$D_q \subset \Omega_q,$$

for any value $0 < q < 1$ such that $| \alpha |_\infty \| A \|_\infty \le q e^{-q}$, and the function

$$F : D_q \longrightarrow D_q,$$

is contracting, so it has a unique fixed point X^* in D_q. Also, for any $X^{(0)} \in D_q$, the sequence of iterates

$$X^{(k)} = F^{(k)}(X^{(0)}) = F(X^{(k-1)})$$

converges to X^*, and it holds that

$$| X^{(k)} - X^* |_\infty \le \frac{q^k}{1-q} | X^{(1)} - X^{(0)} |_\infty .$$

Proof. We know that the function $g(x) = xe^{-x}$ defined on $[0, 1]$ is increasing, $g(0) = 0$, and $g(1) = 1/e$. From the assumption that $\mid \alpha \mid_\infty \parallel A \parallel_\infty < 1/e$, it follows that there exists $q^* \in (0, 1)$ such that $q^* e^{-q^*} = \mid \alpha \mid_\infty \parallel A \parallel_\infty$, and therefore $qe^{-q} \geq \mid \alpha \mid_\infty \parallel A \parallel_\infty, \forall q \in (q^*, 1)$.
Suppose now that $X \in D_q$ for some $q \in (q^*, 1)$. First we note that

$$f_i(X) = \alpha_i e^{\left(\sum_{j=1}^{n} \frac{\beta_{ij} N_j x_j}{\gamma_j}\right)} = \alpha_i e^{\left(\sum_{j=1}^{n} a_{ij} x_j\right)},$$

where a_{ij} are the coefficients of the matrix A.
But because $\left(\sum_{j=1}^{n} a_{ij}\right) X \leq \parallel A \parallel_\infty \mid X \mid_\infty$, and $X \in D_q$, then $\left(\sum_{j=1}^{n} a_{ij}\right) X \leq q$, and therefore

$$\mid f_i(X) \mid \leq \mid \alpha_i \mid e^q;$$

we obtain

$$\mid F(X) \mid_\infty \leq \mid \alpha \mid_\infty e^q. \tag{3.10}$$

With this, we have chosen q such that $\mid \alpha \mid_\infty \parallel A \parallel_\infty \leq qe^{-q}$. So from Eq (3.10), one has

$$\mid F(X) \mid_\infty \leq \frac{\mid \alpha \mid_\infty \parallel A \parallel_\infty}{\parallel A \parallel_\infty} e^q \leq \frac{qe^{-q}e^q}{\parallel A \parallel_\infty} = \frac{q}{\parallel A \parallel_\infty}. \tag{3.11}$$

But from the inequality (3.11), it follows that $X \in \Omega_q$. That is, we have proved that, under the conditions of the theorem imposed on $\mid \alpha \mid_\infty$, we have $D_q \subset \Omega_q$ and, therefore, F transforms D_q into D_q, because $F[\Omega_q] = D_q$.
Applying Theorem (3.1), the proof of this theorem is concluded. □

From Theorem (3.2), we can see that condition $\mid \alpha \mid_\infty \parallel A \parallel_\infty < 1/e$ guarantees $0 \in \Omega_q$ for some $0 < q \leq 1$, because $\alpha = f(0)$. This theorem gives conditions for the existence of at least one fixed point.

Furthermore, the same theorem provides us with a simple algorithm to obtain the fixed point X^*. First, it is necessary to verify that the vector α satisfies condition $\mid \alpha \mid_\infty \parallel A \parallel_\infty < 1/e$, which guarantees that there exist positive values of q close to 1 for which f contracts in D_q. Then, if we start from any $X^{(0)} \in D_q$ and construct the iterations $X^{(k)}$ of $X^{(0)}$, we will know that they will converge to the fixed point X^*.
Remark: Note that if $\parallel A \parallel_\infty < 1$, then we explicitly bound the value of q. In fact, $q \leq max\left\{\frac{S_1}{N_1}, \frac{S_2}{N_2}, \cdots, \frac{S_n}{N_n}\right\} \leq 1$, so we proceed in the same way as above.

It is worth mentioning that this algorithm is equivalent to the one presented in [25, 31], where convergence is guaranteed by branching theories and probability generating functions [33, 34, 36].

In the next section, we will use equation (2.10) to perform a sensitivity analysis for a particular case, which will allow us to understand how human mobility between regions can be an important factor in the spread of infectious diseases through person-to-person contact.

4. Parametric sensitivity analysis

In the study of infectious diseases in spatially structured regions, one of the central questions is determining how human mobility between regions influences the spread of infection [1, 42, 43].

The interest in this issue was increased by the pandemic caused by the SARS-CoV-2 virus, the causative agent of COVID-19. Although early detection of the outbreak allowed the implementation of control measures (including the isolation of infected individuals; the confinement of urban areas; and the suspension of land, sea and air transportation), these proved insufficient to contain the rapid global spread of the virus [1, 7].

Furthermore, mobility restrictions generated high socioeconomic costs, raising questions about the effectiveness and appropriateness of total lockdowns as a mitigation strategy [8, 44]. A remarkable work in this direction is the work developed by Kühn et al. [16], where the authors demonstrated that restricted mobility coupled with continuous surveillance through infection confirmation tests in populations with high incidence effectively helped prevent outbreaks between surrounding regions. On the other hand, in [17–19], using metapopulation models and real-world databases, researchers were able to quantify, through Bayesian inference, mobility matrices that recreated the dynamics observed during the COVID-19 pandemic. This represented a significant advance in understanding these infectious processes in contexts where spatial dynamics play a fundamental role.

Therefore, in this work, we will develop a sensitivity analysis of the mobility parameters p_{12} in a system composed of two regions interconnected by human movement. Our aim is to analyze a scenario where one region experiences an initial epidemic outbreak, while the other is progressively affected by the disease. This simplified framework allows for a more precise assessment of the impact of people's mobility from one region to another on disease transmission, highlighting its relevance as a determining factor in the spread of epidemics in structured spatial environments.

4.1. Parametric sensitivity analysis on the human mobility parameter p_{12}

Let us consider the particular case of a two-patch unidirectional network where we will assume that the recovery rate γ_i and infection rate β_k only depends on the specific region. Furthermore, we assume that travelers moving from one patch to another mix with individuals from the visited patch and maintain the same internal contact rate of the patch they arrive at. We will assume that individuals from patch 2 do not travel to patch 1, so the mobility rates will be given by $p_{11} + p_{12} = 1$ and $p_{22} = 1$, obtaining as densities $P_1 = p_{11}N_1$ for patch 1 and $P_2 = p_{12}N_1 + N_2$ for patch 2. Under these hypotheses, we want to analyze the dynamics under which conditions with an infectious outbreak can spread the disease to another, completely susceptible region through human mobility. Thus, the two-patch unidirectional model is expressed as follows:

$$\frac{dS_1(t)}{dt} = -\frac{\beta_1 p_{11}^2 S_1(t) I_1(t)}{p_{11}N_1} - \frac{\beta_2 p_{12} S_1(t)}{p_{12}N_1 + N_2}\left(p_{12}I_1(t) + I_2(t)\right), \tag{4.1}$$

$$\frac{dI_1(t)}{dt} = \frac{\beta_1 p_{11}^2 S_1(t) I_1(t)}{p_{11}N_1} + \frac{\beta_2 p_{12} S_1(t)}{p_{12}N_1 + N_2}\left(p_{12}I_1(t) + I_2(t)\right) - \gamma_1 I_1(t), \tag{4.2}$$

$$\frac{dR_1(t)}{dt} = \gamma_1 I_1(t), \tag{4.3}$$

$$\frac{dS_2(t)}{dt} = -\frac{\beta_2 S_2(t)}{p_{12}N_1 + N_2}\left(p_{12}I_1(t) + I_2(t)\right), \tag{4.4}$$

$$\frac{dI_2(t)}{dt} = \frac{\beta_2 S_2(t)}{p_{12}N_1 + N_2}\left(p_{12}I_1(t) + I_2(t)\right) - \gamma_2 I_2(t), \tag{4.5}$$

$$\frac{dR_2(t)}{dt} = \gamma_2 I_2(t). \tag{4.6}$$

The term $\frac{\beta_2 p_{12} S_1(t)}{p_{12}N_1 + N_2}\left(p_{12}I_1(t) + I_2(t)\right)$ in Eqs (4.1)–(4.2) indicates that although the infections of susceptible people in patch 1 can be caused in patch 2, these people can return to their place of origin. On the other hand, the term $\frac{\beta_2 S_2(t)}{p_{12}N_1 + N_2}\left(p_{12}I_1(t) + I_2(t)\right)$ in Eqs (4.4)–(4.5) indicates that susceptible people from patch 2 contract the infection through interaction with both infected individuals from the same patch and infected travelers from patch 1.

By using the equation (2.10) to calculate the final size of the epidemic in the two study patches, we obtain that these are given by

$$\tilde{f_1} := N_1 - S_1(0)e^{-\theta_1} \tag{4.7}$$

with

$$\theta_1 = \left[\left(\frac{\beta_1(1-p_{12})}{\gamma_1 N_1} + \frac{\beta_2 p_{12}^2}{\gamma_1(p_{12}N_1 + N_2)}\right)R_1(\infty) + \left(\frac{\beta_2 p_{12}}{\gamma_2(p_{12}N_1 + N_2)}\right)R_2(\infty)\right],$$

and

$$\tilde{f_2} := N_2 - S_2(0)e^{-\theta_2} \tag{4.8}$$

with

$$\theta_2 = \left[\left(\frac{\beta_2 p_{12}}{(\gamma_1(p_{12}N_1 + N_2)}\right)R_1(\infty) + \left(\frac{\beta_2}{(\gamma_2(p_{12}N_1 + N_2)}\right)R_2(\infty)\right].$$

About the Eqs (4.7) and (4.8), we will do the sensitivity analysis with respect to the mobility parameter p_{12}. This is to know how the direct introduction of individuals from one area to another can affect the spread of the disease, this being one of the main causes of the spread of epidemics in regions with spatial structure. For this, the total derivatives of Eqs (4.7) and (4.8) are calculated with respect to the parameter p_{12}; that is

$$\frac{d\tilde{f_1}}{dp_{12}} = \frac{\partial\tilde{f_1}}{\partial p_{12}} + \frac{\partial\tilde{f_1}}{\partial R_1(\infty)}\frac{\partial R_1(\infty)}{\partial p_{12}} + \frac{\partial\tilde{f_1}}{\partial R_2(\infty)}\frac{\partial R_2(\infty)}{\partial p_{12}}, \tag{4.9}$$

and

$$\frac{d\tilde{f_2}}{dp_{12}} = \frac{\partial\tilde{f_2}}{\partial p_{12}} + \frac{\partial\tilde{f_2}}{\partial R_1(\infty)}\frac{\partial R_1(\infty)}{\partial p_{12}} + \frac{\partial\tilde{f_2}}{\partial R_2(\infty)}\frac{\partial R_2(\infty)}{\partial p_{12}}. \tag{4.10}$$

If we consider $R_1(\infty)$ and $R_2(\infty)$ as functions of the parameter p_{12} assuming that the rest of the epidemiological parameters remain constant at any instant of time t, we obtain the following system of ordinary differential equations with variable coefficients $p_{12} \in [0, 1]$:

$$[N_1 - R_1(\infty)]\left[a_1 R_1(\infty) + b_1 R_2(\infty) + c_1\frac{dR_1(\infty)}{dp_{12}} + d_1\frac{dR_2(\infty)}{dp_{12}}\right] = \frac{dR_1(\infty)}{dp_{12}} \tag{4.11}$$

$$[N_2 - R_2(\infty)]\left[a_2R_1(\infty) - b_2R_2(\infty) + c_2\frac{dR_1(\infty)}{dp_{12}} + d_2\frac{dR_2(\infty)}{dp_{12}}\right] = \frac{dR_2(\infty)}{dp_{12}}, \tag{4.12}$$

where the parameters associated with Eq (4.11) are

$$a_1 = \left(\frac{\beta_2 p_{12}(p_{12}N_1 + 2N_2)}{\gamma_1(p_{12}N_1 + N_2)^2} - \frac{\beta_1}{\gamma_1 N_1}\right), b_1 = \left(\frac{\beta_2 N_2}{\gamma_2(p_{12}N_1 + N_2)^2}\right),$$

$$c_1 = \left(\frac{\beta_1(1 - p_{12})}{\gamma_1 N_1} + \frac{\beta_2 p_{12}^2}{\gamma_1(p_{12}N_1 + N_2)}\right), d_1 = \left(\frac{\beta_2 p_{12}}{\gamma_2(p_{12}N_1 + N_2)}\right),$$

and the parameters associated with the Eq (4.12) are

$$a_2 = \left(\frac{\beta_2 N_2}{\gamma_1(p_{12}N_1 + N_2)^2}\right), b_2 = \left(\frac{\beta_2 N_1}{\gamma_2(p_{12}N_1 + N_2)^2}\right),$$

$$c_2 = \left(\frac{\beta_2 p_{12}}{\gamma_1(p_{12}N_1 + N_2)}\right), d_2 = \left(\frac{\beta_2}{\gamma_2(p_{12}N_1 + N_2)}\right).$$

From the system of ordinary differential equations, we can clear the terms $\frac{dR_1(\infty)}{dp_{12}}$ and $\frac{dR_2(\infty)}{dp_{12}}$ with what we obtain:

$$\frac{dR_1(\infty)}{dp_{12}} = \frac{-(a_1R_1(\infty) + b_1R_2(\infty))(d_2 - [N_2 - R_2(\infty)]^{-1}) - d_1(b_2R_2(\infty) - a_2R_1(\infty))}{(c_1 - [N_1 - R_1(\infty)]^{-1})(d_2 - [N_2 - R_2(\infty)]^{-1}) - d_1c_2}, \tag{4.13}$$

and

$$\frac{dR_2(\infty)}{dp_{12}} = \frac{(b_2R_2(\infty) - a_2R_1(\infty))(c_1 - [N_1 - R_1(\infty)]^{-1}) + c_2(a_1R_1(\infty) + b_1R_2(\infty))}{(c_1 - [N_1 - R_1(\infty)]^{-1})(d_2 - [N_2 - R_2(\infty)]^{-1}) - d_1c_2}. \tag{4.14}$$

Remark: Although from the formal point of view Eqs (4.13) and (4.14) make epidemiological sense, interpreting their meanings will depend on the different geographic levels that are being modeled. At a global geographic level, it would be analyzed what happens with the exchange of infected individuals between countries. At the regional geographic level, it would be analyzed what happens with the exchange of individuals between the states of a country or even between the municipalities of a particular state of a country. One of the fundamental differences between the global and regional geographic analysis is the value of the mobility parameter p_{12}. In the global case, this should take values very close to zero, while in the regional case, this would only happen if very severe policies are applied to restrict the mobility of individuals in the affected sectors.

In this work, we will concentrate on performing a global analysis; that is, we consider the mobility parameter $p_{12} \longrightarrow 0$. Under this assumption, we can simplify the parameters of the Eqs (4.13) and (4.14) as follows:

$$a_1 = -\frac{\beta_1}{\gamma_1 N_1}, b_1 = \frac{\beta_2}{\gamma_2 N_2}, c_1 = \frac{\beta_1}{\gamma_1 N_1}, d_1 = 0,$$

and

$$a_2 = \frac{\beta_2}{\gamma_1 N_2}, b_2 = \frac{\beta_2 N_1}{\gamma_2 N_2^2}, c_2 = 0, d_2 = \frac{\beta_2}{\gamma_2 N_2}.$$

We can then simplify Eqs (4.13) and (4.14) obtaining

$$\frac{dR_1(\infty)}{dp_{12}} = \frac{\left(\mathcal{R}_0^{(1)}\frac{R_1(\infty)}{N_1} - \mathcal{R}_0^{(2)}\frac{R_2(\infty)}{N_2}\right)}{\left(\frac{\mathcal{R}_0^{(1)}}{N_1} - [N_1 - R_1(\infty)]^{-1}\right)}, \tag{4.15}$$

and

$$\frac{dR_2(\infty)}{dp_{12}} = \frac{\left(\frac{\mathcal{R}_0^{(2)}N_1}{N_2}\frac{R_2(\infty)}{N_2} - \frac{\mathcal{R}_0^{(1)}\mathcal{R}_0^{(2)}\gamma_1 N_1}{\beta_2 N_2}\frac{R_1(\infty)}{N_1}\right)}{\left(\frac{\mathcal{R}_0^{(2)}}{N_2} - [N_2 - R_2(\infty)]^{-1}\right)}, \tag{4.16}$$

where $\mathcal{R}_0^{(1)} = \beta_1/\gamma_1$ and $\mathcal{R}_0^{(2)} = \beta_2/\gamma_2$ represent the local basic reproductive numbers of patch 1 and patch 2, respectively.

The approach where $p_{12} \to 0$ is performed in the derivative of $R_i(\infty)$ for $i = 1, 2$; that is, the approximation tells us the change in $R_i(\infty)$ when mobility is decreases. This is done at a regime of small mobilities. In other words, the sensitivity analysis is based on changes in $R_i(\infty)$, and studying these changes reveals whether the final size will increase or decrease because of mobility. The values of $R_i(\infty)$ are similar to those without mobility, owing to the regime under consideration. So, when $\mathcal{R}_0^{(i)} > 1$ for $i = 1, 2$, the following inequality makes for $i = 1, 2$, [11]:

$$\frac{\mathcal{R}_0^{(i)}}{N_i} < \frac{1}{S_i(\infty)} \qquad \text{for} \qquad i = 1, 2. \tag{4.17}$$

Therefore, from Eq (4.17) and from the fact that $S_i(\infty) = N_i - R_i(\infty)$ for $i = 1, 2$, it follows that the denominators of Eq (4.15) and Eq (4.16) are always negative in case of $\mathcal{R}_0^{(i)} > 1$ for $i = 1, 2$, which concludes that the signs of these equations only depend on the signs of their numerators.

We observe that the final size of patch 1, $R_1(\infty)$ is increased if the mobility from 1 to 2 is also increased if

$$\mathcal{R}_0^{(2)}\frac{R_2(\infty)}{N_2} > \mathcal{R}_0^{(1)}\frac{R_1(\infty)}{N_1}.$$

As $\mathcal{R}_0^{(2)}$ and $R_2(\infty)$ are measures of the severity of the outbreak in patch 2, this indicates that travelers from a low-severity region to a high-severity outbreak region increase the severity of the epidemics in their own patch; see Figures 1 and 2 (left). Remember the model just takes into account the mobility from patch 1 to patch 2. In contrast, if the outbreak is more severe in region 1,

$$\mathcal{R}_0^{(1)}\frac{R_1(\infty)}{N1} > \mathcal{R}_0^{(2)}\frac{R_2(\infty)}{N_2}, \tag{4.18}$$

the derivative $dR_1(\infty)/dp_{12}$ is negative, indicating that travelers from a high-severity outbreak region to a low-severity region lead to a reduction in the severity of its own region's severity (see Figures 1 and 2, right). Eq (4.16) describes the effect of in-migrants to a particular region. It is concluded that the severity of the disease increases in the region 2 if

$$\mathcal{R}_0^{(1)}\frac{R_1(\infty)}{N_1}\frac{\gamma_1}{\beta_2} > \frac{R_2(\infty)}{N_2}.$$

In order to gain insight from this inequality, we write it in an equivalent form as follows:

$$\mathcal{R}_0^{(1)} \frac{R_1(\infty)}{N_1} \gamma_1 > \mathcal{R}_0^{(2)} \frac{R_2(\infty)}{N_2} \gamma_2. \tag{4.19}$$

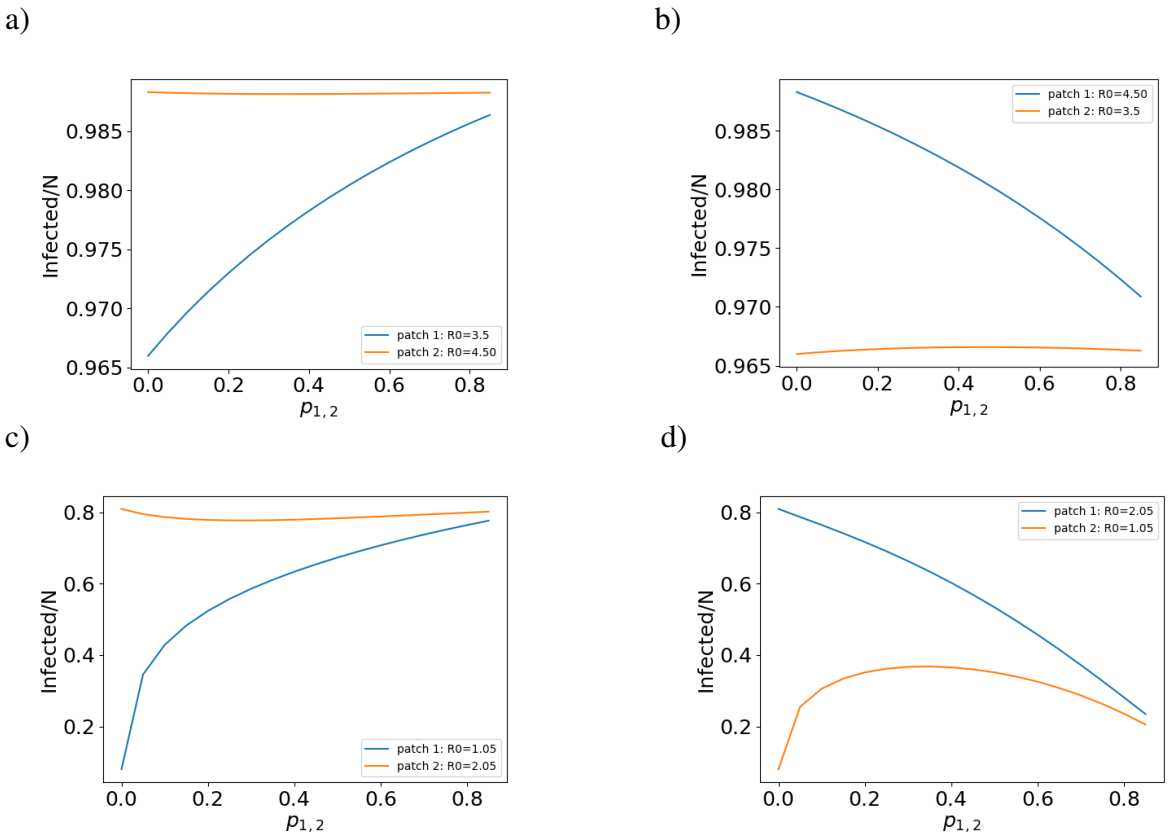

Figure 1. Final size change due to mobility from region 1 to region 2 and when the difference between patches is the contact rate. a) High $\mathcal{R}_0$ case. Traveling from a region of lower-severity to a region of high-severity increases the severity in the original traveler region. b) Traveling from a high-severity region to a lower-severity region reduces the severity in the traveler's region of origin and only increases the severity of the outbreak in the visited region when its severity is low, as seen in d). c) Low $\mathcal{R}_0$ case. Traveling from region of lower-severity to a region of high-severity, increases the severity in the original traveler region.

This means that for a case where $\gamma_1 \simeq \gamma_2$, meaning the recovery rate is similar in both regions, the severity of the disease increases in region 2 if the immigrants come from a region with greater severity. On the other hand, if the immigrants come from a lower-severity region, the size of the epidemic in region 2 can decrease. This phenomenon is due to the dilution of susceptibles in region 2, leading to a noticeable split of contagions between the immigrants and the susceptibles of region 2. The appearance of γ_1 and γ_2 in inequality (4.19) indicates the existence of a possibly uncommon but interesting scenario. If the recovery rate of the immigrants, γ_1, is lower than the recovery rate of the inhabitants of patch 2, γ_2, it could lead to an effective dilution effect that reduces infections in patch 2 even if the severity in patch 1 is higher than in region 2. An interpretation is that, because the recovery

rate γ_1 of the population in 1 is smaller, to keep the same epidemic severity given by $\mathcal{R}_0$ and $R(\infty)$ at a similar level, the contact rate β_1 also must decrease, effectively causing there to be fewer infected individuals at every instant of time even though the epidemic lasts longer. This is another process that also gives rise to the dilution effect.

This same phenomenon gives us special conditions where mobility reduces the overall size of the disease instead of increasing it. As mobility of travelers from a high-severity zone to one of lower severity reduces its local size of the epidemic, and this doesn't depend on the rate of recovering γ_1 (4.18), this rate could be big enough that this mobility doesn't affect or even helps region 2 (4.19), leading to a global reduction in the size of the epidemic; see Figure 2 (bottom right).

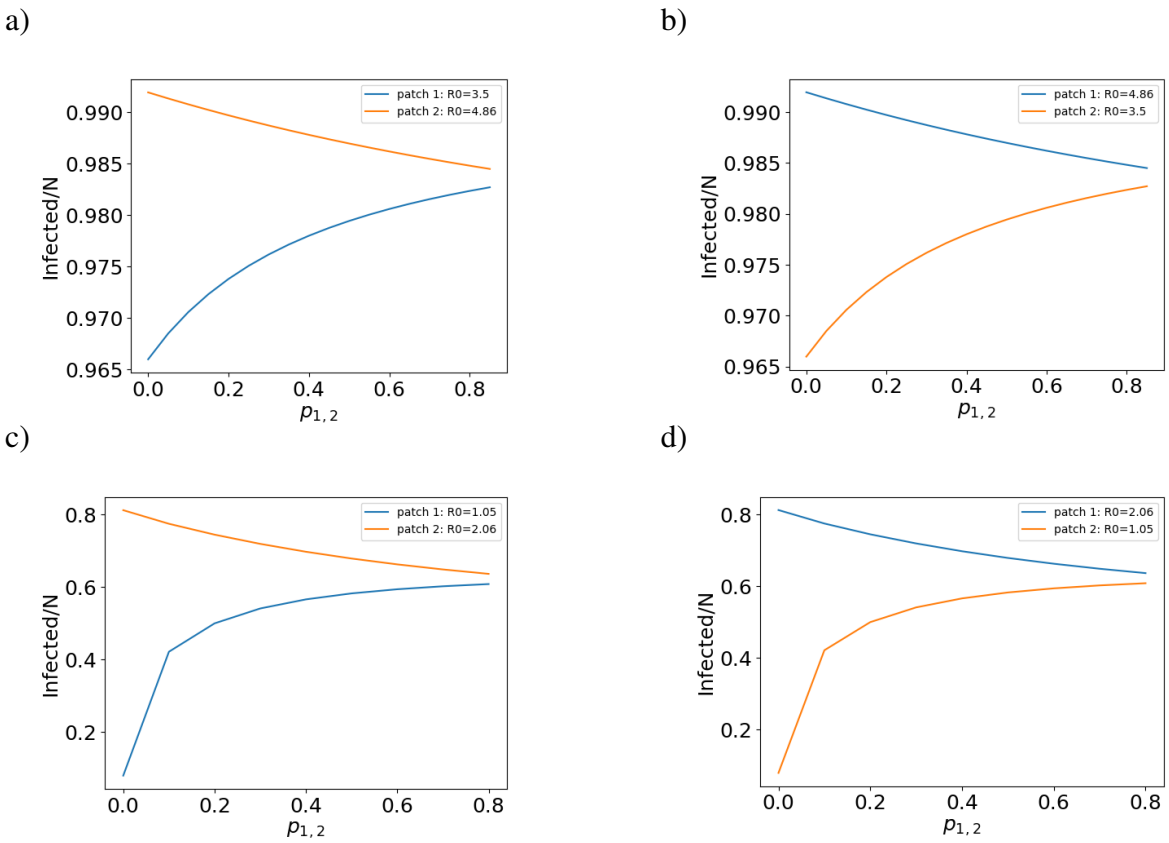

Figure 2. Final size change due to mobility from region 1 to region 2 and when the difference between patches is the recovery or isolation rate. a) and d) High $\mathcal{R}_0$ case. Traveling from a region of lower-severity to a region of high-severity, increases the severity in the original traveler region. b) and d) Traveling from a high-severity region to a lower-severity region, reduces the severity in the traveler's original region and normally increases the severity of the outbreak in the visited region if the contact rates are similar in both regions.

5. Conclusion

To begin, we have shown the existence and uniqueness of the solution to the final size equation of an SIR metapopulation model. This provides further theoretical support for using the final epidemic size as an index of regional and global severity.

This study is intentionally theoretical. The deliberate simplifications introduced in the model are not shortcomings; they are the means by which we isolate and expose the dynamical mechanisms that can produce non-trivial, counterintuitive outcomes when human migration is coupled with epidemic spread. We do not aim to reproduce or forecast any particular outbreak. Instead, we follow the long-standing tradition of conceptual epidemic modeling (Kermack & McKendrick, [45]) whose hallmark is to extract interpretable, mechanistic insight from minimal mathematical structures.

Next, through parametric sensitivity analysis performed on the human mobility parameter, we were able to affirm how travel from a low-severity region to a high-severity region increases the impact of the epidemic in the low-severity region. Conversely, the high-severity region has little impact. On the other hand, travel from a high-severity region to a low-severity region reduces cases in the high-severity zone, while the total number of cases in the lower-severity region is low, unless $\mathcal{R}_0$ is close to 1 (from above) and, therefore, the outbreak is small. This interesting and important result shows that the total number of infected people in a community is primarily due to social behavior acquired during infectious outbreaks and minimally by potentially infected visitors.

This phenomenon can be understood because it assumes that local behavior will be the same among local and foreign individuals. In general, during outbreaks characterized by high $\mathcal{R}_0$, restricting mobility might be useful to prevent the introduction of the disease into specific regions. However, once a local outbreak has begun, visitors have little impact on the final magnitude of the epidemic in that location. However, for epidemics characterized by $\mathcal{R}_0$ close to 1 from above, the impact of immigrants must be considered. This is understandable, as any increase in infections in a small outbreak is noticeable due to the high sensitivity of the relative size of the epidemic.

It is also worth considering a less common, but possible, scenario. In a situation where control measures are primarily based on isolation, thereby increasing the effective recovery rate γ, the consequences of travel are completely different, as can be seen in Figure 2. In this scenario, mobility always increases the final size of the outbreak in the region of lower severity. This is because it was hypothesized that isolation only occurs among local residents, while visitors continue to transmit diseases throughout their stay.

The objective of this study is to uncover the mechanistic relationship between a targeted control rule and the final epidemic size. We used biologically grounded simulations to isolate the causal effect of mobility on the propagation process. Once these mechanisms are formally understood, future field studies can be designed to measure the corresponding empirical quantities. It should be noted that the described effects of mobility on disease spread are the most basic, in the sense that their combination in multiregional systems can lead to more complex overall behavior. Furthermore, average mobility could change over time, and isolation and behavioral changes among infected individuals could reduce their mobility, thereby reducing the effective contact rate.

In summary, this work reinforces the use of the final epidemic size not only as an index of magnitude or classification but also as a guide for designing control strategies. Furthermore, the local analysis of human mobility for each region allows for simple but non-trivial conclusions to be drawn about the effect and relevance of mobility restrictions during an infectious outbreak, facilitating decision-making in these scenarios. General, concise insights of this kind are often more useful to public health authorities than highly specific tactical recommendations: they provide an immediate, qualitative understanding of what can happen, thereby alerting decision-makers to unintended consequences before detailed data or computationally intensive simulations become available.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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