SIAM J. APPL. MATH.
Vol. 85, No. 2, pp. 806–820

VACCINATING ACCORDING TO THE MAXIMAL ENDEMIC EQUILIBRIUM ACHIEVES HERD IMMUNITY*

JEAN-FRANÇOIS DELMAS†, DYLAN DRONNIER†, AND PIERRE-ANDRÉ ZITT‡

Abstract. We consider the simple epidemiological SIS model for a general heterogeneous population introduced by Lajmanovich and Yorke (1976) in finite dimensions, and its infinite-dimensional generalization we introduced in previous works. In this model the basic reproducing number R_0 is given by the spectral radius of an integral operator. If the basic reproducing number R_0 is greater than 1 ($R_0 > 1$), then there exists a maximal endemic equilibrium. In this very general heterogeneous SIS model, we prove that vaccinating according to the profile of this maximal endemic equilibrium ensures herd immunity. Moreover, this vaccination strategy is critical: the resulting effective reproduction number is exactly equal to one. As an application, we estimate in an example from Britton, Ball, and Trapman (2020) that if $R_0 = 2$ in an age-structured community with mixing rates fitted to social activity, applying this strategy would require approximately 29% fewer vaccine doses than the strategy which consists in vaccinating uniformly a proportion $1 - 1/R_0$ of the population. From a dynamical systems point of view, we prove that the nonmaximality of an equilibrium g is equivalent to its linear instability in the original dynamics, and to the linear instability of the disease-free state in the modified dynamics where we vaccinate according to g.

Key words. SIS model, vaccination strategy, effective reproduction number, herd immunity, spectral radius, endemic equilibrium

MSC codes. 92D30, 47B34, 34D20

DOI. 10.1137/23M1558112

1. Introduction.

Increasing the prevalence of immunity from contagious disease in a population limits the circulation of the infection among the individuals who lack immunity. This so-called herd effect plays a fundamental role in epidemiology; for example, it has had a major impact in the eradication of smallpox and rinderpest or the near eradication of poliomyelitis [12]. Our aim is to present a targeted vaccination strategy based on the heterogeneity of the infection spreading in the population which can effectively eradicate the epidemic. We consider for simplicity the deterministic infinite-dimensional SIS model (with S = Susceptible and I = Infectious) and the effect of a perfect vaccine. However, we take into account a very general model for the heterogeneous population based on the infinite-dimensional model introduced in [3], which encompasses the metapopulation SIS models developed by Lajmanovich and Yorke in their pioneer paper [15] or SIS model on graphs; see [9] in this direction. More precisely, the probability $u(t,x)$ of an individual with feature $x \in \Omega$ to be infected at time t is the solution of the (infinite-dimensional) ordinary differential equation

$$\partial_t u(t,x) = (1-u(t,x)) \int_\Omega k(x,y)\, u(t,y)\, \mu(\mathrm{d}y) - \gamma(x) u(t,x) \quad \text{for } t \geq 0 \text{ and } x \in \Omega, \tag{1}$$

*Received by the editors March 10, 2023; accepted for publication (in revised form) January 3, 2025; published electronically April 17, 2025.
https://doi.org/10.1137/23M1558112
Funding: This work was partially supported by Labex Bézout, ANR-10-LABX-58, and by SNF 200020-196999.
†CERMICS, École nationale des ponts et chaussées, 77455 Marne-la-Vallée, France (jean-francois.delmas@enpc.fr, dylan.dronnier@enpc.fr).
‡LAMA, Université Gustave Eiffel, 77420 Champs-sur-Marne, France (pierre-andre.zitt@univ-eiffel.fr).

where k is the *transmission rate* kernel of the disease, γ is the *recovery rate* function, and $\mu(\mathrm{d}y)$ is the probability for an individual taken at random to have feature $y \in \Omega$. In the above equation the term $(1-u)$ in front of the integral corresponds to the *incidence rate* of the disease given by a generalization of the so-called *law of mass action*: infection of subpopulation with feature x from subpopulation with feature y is proportional to $(1-u(t,x))\,u(t,y)$; see [13] for a historical review on the law of mass action. We refer the reader to section 4 for a more precise description of the mathematical framework. For a finite-dimensional setting, see Example 3.2 for an age-structured population and Example 3.3 for subpopulations with a proportionate mixing structure. For the convenience of the reader, we rewrite (1) with $n \geq 1$ subpopulations (see equation (3.3) in [15]): $\Omega = \{1, \ldots, n\}$, μ_i is the relative size of the subpopulation $i \in \Omega$ and $\gamma_i > 0$ its recovery rate, the contact matrix $K = (K_{i,j};\, i,j \in \Omega)$ has nonnegative entries, and we have for $u_i(t)$ the probability (or proportion) of the subpopulation i which is infected at time t:

$$\partial_t u_i(t) = (1-u_i(t)) \sum_{j\in\Omega} K_{ij}\,\mu_j\,u_j(t) - \gamma_i u_i(t) \quad \text{on } [0,\infty) \text{ for } i \in \Omega. \tag{2}$$

In a homogeneous population, the *basic reproduction number* of an infection, denoted by R_0, is defined as the number of secondary cases one individual generates on average over the course of its infectious period, in an otherwise uninfected (susceptible) population. Intuitively, the disease should die out if $R_0 < 1$ and invade the population if $R_0 > 1$. For the heterogeneous generalization of many classical models in epidemiology (including the heterogeneous SIS model), it is still possible to define a meaningful basic reproduction number R_0 as the number of secondary cases generated by a *typical* infectious individual when all other individuals are uninfected and the threshold phenomenon occurs; see [10]. We recall that in the setting of [3], the reproduction number R_0 corresponds to the spectral radius of the next-generation operator defined as the integral operator associated to the kernel $k(x,y)/\gamma(y)$.

After a vaccination campaign, let the vaccination strategy η denote the proportion of the *nonvaccinated* population: η is a $[0,1]$-valued function defined on Ω and $\eta(x)$ denotes the proportion of nonvaccinated individuals of type x. Let the effective reproduction number $R_e(\eta)$ denote the corresponding reproduction number of the nonvaccinated population. (Following [3, section 5.3], the effective reproduction number $R_e(\eta)$ is given by the spectral radius of the effective next-generation operator defined as the integral operator associated to the kernel $k(x,y)\eta(y)/\gamma(y)$, where $\eta(y)$ is the proportion of nonvaccinated individuals with feature y.)

The vaccination strategy η is called *critical* if $R_e(\eta) = 1$. Assuming $R_0 > 1$, suppose now that only a proportion η^{uni} of the population can catch the disease, the rest being perfectly immunized. An infected individual will now only generate $\eta^{\mathrm{uni}} R_0$ new cases, since a proportion $1-\eta^{\mathrm{uni}}$ of previously successful infections will be prevented. Therefore, the new *effective reproduction number* is equal to $R_e(\eta^{\mathrm{uni}}) = \eta^{\mathrm{uni}} R_0$. This fact led to the recognition by Smith in 1970 [18] and Dietz in 1975 [11] of a simple threshold theorem: the incidence of an infection declines if the proportion of nonimmune individuals is reduced below $\eta^{\mathrm{uni}}_{\mathrm{crit}} = 1/R_0$. This effect is called *herd immunity,* and the corresponding proportion $1 - \eta^{\mathrm{uni}}_{\mathrm{crit}}$ of people that have to be vaccinated is called the *herd immunity threshold* [19, 21].

2. Critical vaccination given by the endemic equilibrium. However, herd immunity can also be achieved using a nonuniform vaccination strategy when the population is heterogeneous. Such a heterogeneous vaccination strategy, based on

geographic hospitalization rates, has been implemented by the Norwegian Institute of Public Health for the COVID-19 epidemic; see [2]. See also the study [20] on the Lassa vaccination campaigns in West Africa, where the most effective preventive vaccination strategy targets the WHO-classified "endemic" districts. In another example, the discussion of vaccination control of gonorrhea in [14, section 4.5] suggests that it may be better to prioritize the vaccination of people that have already caught the disease: this leads us to consider a vaccination strategy guided by the equilibrium state. For the SIS model in a heterogeneous population with $R_0 > 1$, there exists a maximal endemic equilibrium, say $\mathfrak{g}$, where $\mathfrak{g}(x)$ represents the fraction of infected people in the group with feature x. In other words, the function $\mathfrak{g}$ is the maximal $[0,1]$-valued solution g of

$$(1-g(x))\int_\Omega k(x,y)\,g(y)\,\mu(\mathrm{d}y) = \gamma(x)g(x) \quad \text{for } x\in\Omega. \tag{3}$$

Let us mention that if there exist isolated subpopulations, it is possible to have other endemic equilibria, i.e., solutions to (3) that are not equal to 0 for all x. Irreducibility conditions on the kernel k ensure, however, the uniqueness of the endemic equilibrium [3, 15]. Consider the vaccination strategy, denoted by η^{equi}, corresponding to vaccinating a fraction $\mathfrak{g}(x)$ of people in the group with feature x, for all groups. In our mathematical framework, this amounts to setting

$$\eta^{\mathrm{equi}}(x) = 1-\mathfrak{g}(x) \quad \text{for} x\in\Omega. \tag{4}$$

The following result ensures that this strategy reaches herd immunity; see Theorem 5.1 in section 5 for a precise mathematical statement.

THEOREM. *In the heterogeneous SIS model with nonzero maximal endemic equilibrium, the vaccination strategy* η^{equi} *is critical:*

$$R_e(\eta^{\mathrm{equi}}) = 1. \tag{5}$$

Let us stress that implementing the critical vaccination strategy η^{equi} relies on the estimation of the maximal endemic equilibrium and thus can be achieved without estimating the transmission rate kernel and the recovery rate.

The proof of the theorem relies on the study of the spectral bound of the linearized operator associated to (1) near an equilibrium. When $R_0 > 1$, this spectral bound is nonpositive at the maximal equilibrium and positive at all other equilibria; see Proposition 5.5 (ii). Thus, the nonmaximality of an equilibrium is equivalent to its linear instability in the original dynamics. We also prove the linear instability of the disease-free state in the modified dynamics where we vaccinate according to a nonmaximal equilibrium; see Proposition 5.5 (iv).

3. Discussion. We expect the results obtained here for the SIS model to be generic in the sense that similar behaviors should also be observed in more realistic and complex models in epidemiology for nonhomogeneous populations: when an endemic equilibrium exists, vaccinating the population according to the maximal endemic profile should protect the population from the disease.

We refer the reader to [6] for a general framework for cost comparison of vaccination strategies and the notions of "best" and "worst" vaccination strategies; see also [4, 5, 7] for further comments and various examples of optimal vaccinations.

Consider a general cost function C which measures the cost for the society of a vaccination strategy (production and diffusion). A simple and natural choice is the uniform cost given by the overall proportion of vaccinated individuals:

$$C(\eta) = \int_\Omega (\mathbb{1} - \eta)\,\mathrm{d}\mu = 1 - \int_\Omega \eta\,\mathrm{d}\mu. \tag{6}$$

The cost $C(\eta_{\mathrm{crit}}^{\mathrm{uni}})$ is equal to the herd immunity threshold $1 - 1/R_0$, while the cost $C(\eta^{\mathrm{equi}})$ is equal to $\int_\Omega \mathfrak{g}\,\mathrm{d}\mu$, which is the proportion of infected people in the endemic state for the SIS infection.

It is not possible to determine which strategy is cheaper in general; we shall prove in a forthcoming paper on two subpopulations that all configurations are possible. However, we can state some partial results on the comparison of the strategies $\eta_{\mathrm{crit}}^{\mathrm{uni}}$, η^{equi} and their cost under the following assumption of the kernel k on Ω defined as $\mathrm{k}(x,y) = \gamma(x)^{-1}k(x,y)$: constant degree kernel and monotone kernel. In the constant degree case, that is, the maps $x \mapsto \int_\Omega \mathrm{k}(x,y)\,\mathrm{d}y$ and $x \mapsto \int_\Omega \mathrm{k}(y,x)\,\mathrm{d}y$ are constant and thus equal, we get that $\eta_{\mathrm{crit}}^{\mathrm{uni}} = \eta^{\mathrm{equi}}$, and we refer the reader to section 5 (and also sections 6–7 for particular examples) in [4] to get the minimality or maximality of their cost among the costs of the critical vaccination strategies according to some properties of k. In the forthcoming paper [8], under monotonicity assumptions on k, we get that $C(\eta^{\mathrm{equi}}) < C(\eta_{\mathrm{crit}}^{\mathrm{uni}})$, and furthermore there exists a critical strategy with cost strictly less than $C(\eta^{\mathrm{equi}})$. In Example 3.3 below on a proportionate mixing structure with two subpopulations, which is commonly considered in epidemiological literature, the kernel satisfies in particular the monotonicity conditions, and we provide explicit computations here for the reader's convenience. Notice that in the age structured population from Example 3.2, we also get that $C(\eta^{\mathrm{equi}}) \le C(\eta_{\mathrm{crit}}^{\mathrm{uni}})$ even though there the kernel does not have constant degrees, nor is it monotone.

Example 3.1 (homogeneous mixing). If the population is homogeneous (which corresponds to the one-dimensional SIS model where Ω is a singleton), then the maximal equilibrium is constant equal to $1 - 1/R_0$. It follows that $C(\eta_{\mathrm{crit}}^{\mathrm{uni}}) = C(\eta^{\mathrm{equi}})$.

Example 3.2 (age and activity structure). In [1], Britton, Ball, and Trapman study an SEIR model, where immunity can be obtained through infection. Using parameters derived from real-world data, these authors noticed that the disease-induced herd immunity level can, for some models, be substantially lower than the classical herd immunity threshold $1 - 1/R_0$. This can be reformulated in terms of targeted vaccination strategies: prioritizing the individuals that are more likely to get infected in an SEIR epidemic may be more efficient than distributing uniformly the vaccine in the population.

We use the same age and activity structures to determine which strategy between η^{equi} and $\eta_{\mathrm{crit}}^{\mathrm{uni}}$ is less costly. More precisely, the community is categorized into six age groups, and contact rates between them are derived from an empirical study of social contacts [22]. For the activity structure, individuals are categorized into three different activity levels, which are arbitrary and chosen for illustration purposes: 50% of each age cohort have normal activity, 25% have low activity corresponding to half as many contacts compared with normal activity, and 25% have high activity corresponding to twice as many contacts as normal activity. Note that when the population is only structured by activity, the mixing is proportionate. Assuming that the recovery rate is constant equal to 1, we solved numerically equation (3) and computed in Table 1 the cost of the uniform and the equilibrium strategies for different values of R_0 and different population structures. We observe that $C(\eta^{\mathrm{equi}}) < C(\eta_{\mathrm{crit}}^{\mathrm{uni}})$ as soon as the

TABLE 1

Cost of the equilibrium vaccination $C(\eta^{\text{equi}})$ for different population structures; it is equal to the herd immunity level $C(\eta^{\text{uni}}_{\text{crit}})$ when the population is homogeneous. Numbers correspond to percentage.

	$R_0 = 2$	$R_0 = 2.5$	$R_0 = 3$
Homogeneous $(= C(\eta^{\text{uni}}_{\text{crit}}))$	50	60	66.7
Age structure	46.6	56.7	63.9
Activity structure	40.1	50	57
Age and activity structure	35.7	45.2	52.2

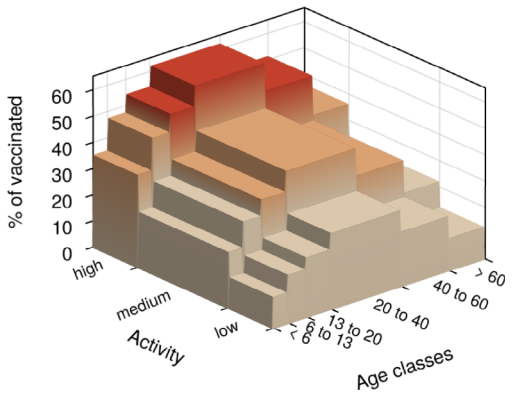


FIG. 1. *Fraction of vaccinated individuals in different groups for the strategy η^{equi} from Example 3.2. The population structure includes both age and activity. These values assume that $R_0 = 2$, so that the uniform critical vaccination consists in vaccinating 50% of the population: only three categories, indicated in deep red, require more vaccine in the targeted strategy than in the uniform strategy. Numbers on the vertical axis correspond to percentage; lengths of the blocks are proportional to the population sizes.*

population is not homogeneous. In Figure 1, we represent the fractions of vaccinated individuals in the different age activity groups when following the strategy η^{equi}. This is done by assuming $R_0 = 2$. Note that in this case, only three subpopulations (in red) need to be vaccinated at a level higher than $1 - 1/R_0 = 50\%$.

Example 3.3 (proportionate mixing structure with two subpopulations). The proportionate mixing is a classical mixing structure introduced by [17] and used in many different epidemiological models. It assumes that the number of adequate contacts between two subpopulations is proportional to the relative activity levels of the two subpopulations. Thus individuals in more active subpopulations will have more adequate contacts. Let us consider the simple case where there are only two subpopulations (that is, (2) with $n = 2$). Then the contact matrix is given by

$$K = \begin{pmatrix} a^2 & ab \\ ab & b^2 \end{pmatrix},$$

where a and b are positive constants that correspond to the activity levels of the first and second subpopulations, respectively. Denote by μ_1 and μ_2 their respective relative size, suppose that the recovery rate γ is equal to 1 for both subpopulations, and assume without loss of generality that $a \geq b$. In this case, we get that

$$R_0 = a^2\mu_1 + b^2\mu_2, \quad R_e(\eta) = a^2\eta_1\mu_1 + b^2\eta_2\mu_2, \quad \text{and} \quad C(\eta) = 1 - (\eta_1\mu_1 + \eta_2\mu_2)$$

for the vaccination strategy $\eta = (\eta_1, \eta_2)$. If $R_0 > 1$, then the (unique) nonzero equilibrium satisfies

$$(1 - \mathfrak{g}_i) \sum_{j=1}^{2} K_{i,j}\, \mathfrak{g}_j\, \mu_j = \mathfrak{g}_i \quad \text{for } i = 1, 2,$$

and the corresponding vaccination strategy $\eta^{\text{equi}} = 1 - \mathfrak{g}$ is given by

$$\eta^{\text{equi}} = \left(\frac{1}{1+ac}, \frac{1}{1+bc} \right),$$

where $c \in [(R_0 - 1)/a, (R_0 - 1)/b]$ is the unique positive solution of the second-order equation $R_e(\eta^{\text{equi}}) = 1$. It is elementary to check that in this case $C(\eta^{\text{equi}}) \le C(\eta^{\text{uni}}_{\text{crit}})$, with an equality if and only if $a = b$. However, the critical vaccination strategy with minimal cost, say η^{opt}, corresponds to vaccinating in priority the population with the highest activity rate, that is, if $a > b$,

$$\eta^{\text{opt}} = \left(\frac{\max(0, 1 - b^2\mu_2)}{a^2\mu_1}, \frac{1}{\max(1, b^2\mu_2)} \right);$$

thus either $\eta_1^{\text{opt}} = 0$ or $\eta_2^{\text{opt}} = 1$. So, for $\mu_1\mu_2 > 0$ and $a > b > 0$, we easily get

$$C(\eta^{\text{opt}}) < C(\eta^{\text{equi}}) < C(\eta^{\text{uni}}_{\text{crit}}) \quad \text{and} \quad R_e(\eta^{\text{opt}}) = R_e(\eta^{\text{equi}}) = R_e(\eta^{\text{uni}}_{\text{crit}}) = 1.$$

An instance is represented in Figure 2, where the solid red line corresponds to the "best" vaccination strategies and the dashed red line to the "worst" vaccination strategies; see [6] for more details.

Assuming the subpopulations have the same size (that is, $\mu_1 = \mu_2 = 1/2$), we represent in Figure 3 the costs of the critical optimal/uniform/endemic vaccination strategy as a function of the activity parameters (a, b) when $R_0 = (a^2 + b^2)/2 > 1$. We also compare in Figure 4 the nonnegative quantities $C(\eta^{\text{equi}}) - C(\eta^{\text{opt}})$ and $C(\eta^{\text{uni}}_{\text{crit}}) - C(\eta^{\text{equi}})$. Using as parameters the mean intensity $i = (a + b)/2$ and the asymmetry measured by the ratio $\alpha = b/a \ge 1$, we observe three regions of interest.

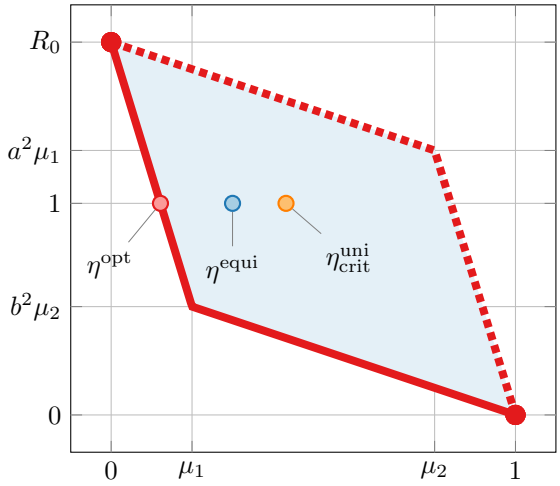
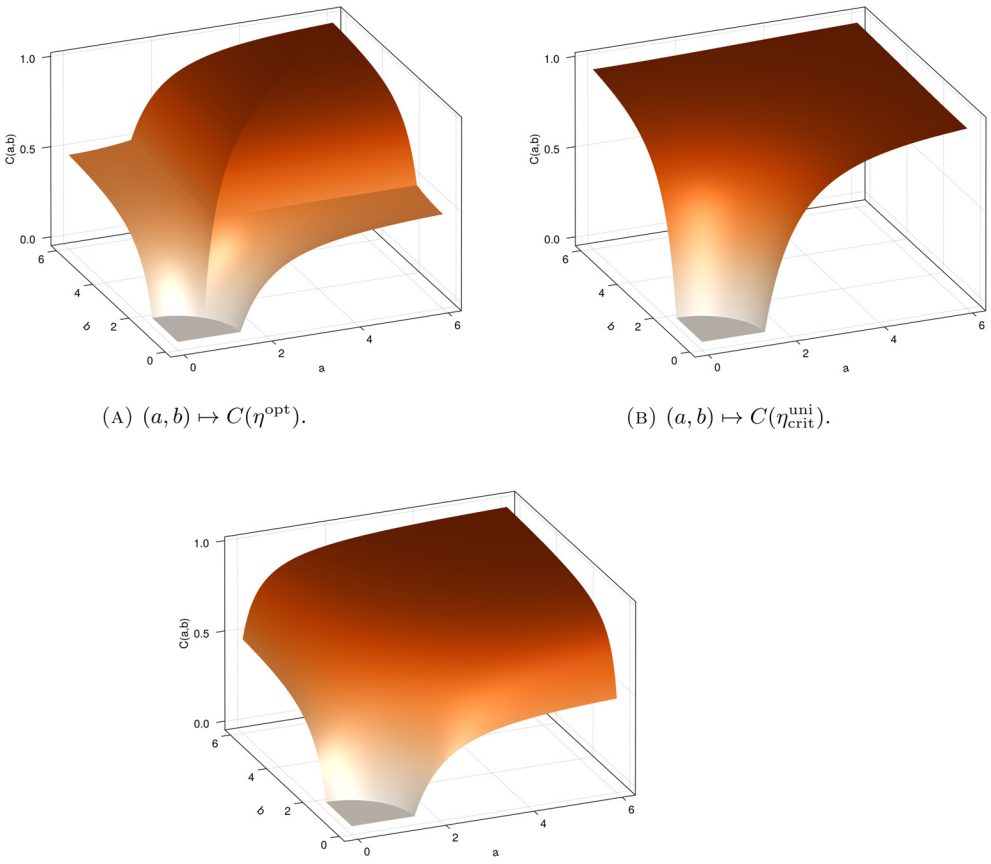


FIG. 2. *Three critical vaccinations from Example* 3.3 *with* $\mu_1 = 0.2 = 1 - \mu_2$, $a = 2.5$, *and* $b = 0.8$. *The horizontal axis corresponds to the cost and the vertical axis to the effective reproduction number for a vaccination strategy. The blue zone represents all the possible outcomes for* $(C(\eta), R_e(\eta))$ *where* η *runs over the set* Δ *of vaccination strategies.*



(A) $(a,b) \mapsto C(\eta^{\mathrm{opt}})$.

(B) $(a,b) \mapsto C(\eta^{\mathrm{uni}}_{\mathrm{crit}})$.

(C) $(a,b) \mapsto C(\eta^{\mathrm{equi}})$.

FIG. 3. *Cost of the critical optimal/uniform/endemic vaccination strategy as a function of the activity parameter* (a,b) *for the proportionate mixing model from Example* 3.3 *with two subpopulations with the same size.*

- If both i and α are large, the optimal vaccination concentrates on one subpopulation and needs almost half as much vaccine as the other two strategies.
- If i is large but α is small, the three strategies require vaccinating almost the whole population.
- If i is small but the asymmetry is large, then the cost of η^{equi} is comparable to the optimal one, but much smaller than the cost of $\eta^{\mathrm{uni}}_{\mathrm{crit}}$.

4. General framework. We recall the differential equations governing the epidemic dynamics in metapopulation SIS models which were developed in the paper [15] in finite dimension and generalized in [3].

4.1. The heterogeneous SIS model. Let $(\Omega, \mathscr{F}, \mu)$ be a probability space, where $x \in \Omega$ represents a feature and the probability measure $\mu(\mathrm{d}x)$ represents the fraction of the population with feature x. The parameters of the SIS model are given by a *recovery rate function* γ, which is a positive bounded measurable function defined on Ω, and a *transmission rate kernel* k, which is a nonnegative measurable function

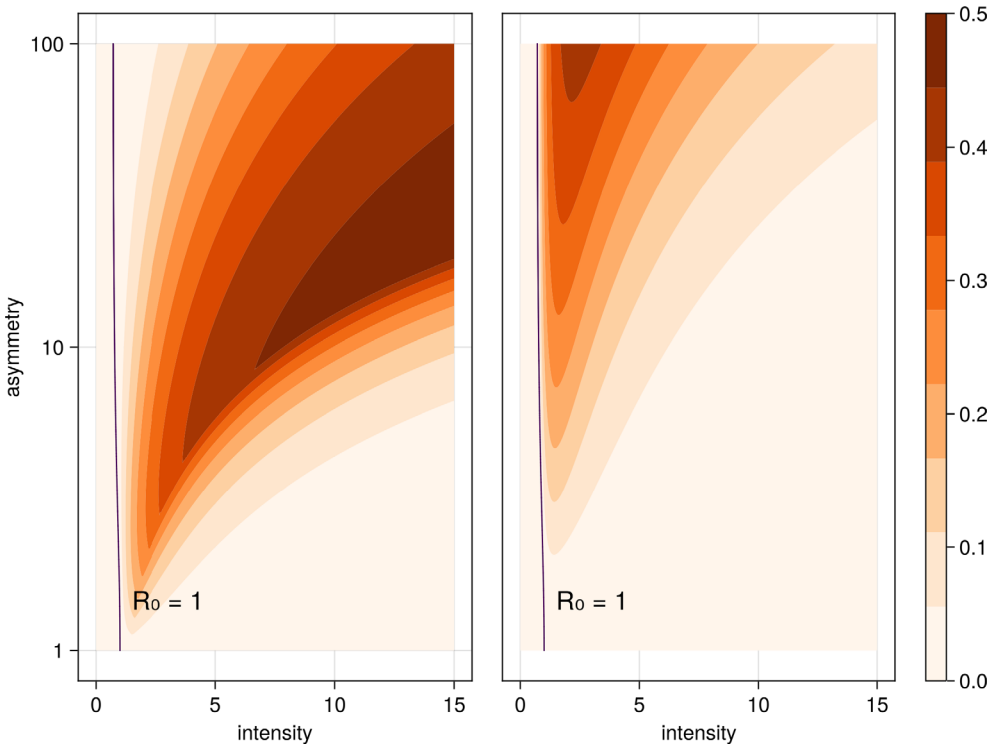


FIG. 4. *Difference between costs for different strategies, for the proportionate mixing model from Example* 3.3 *with two subpopulations with the same size. For clarity we parametrize by the mean intensity* $i = (a+b)/2$*, on the x-axis, and the ratio between the two parameters* $\alpha = b/a$*, on the y-axis (in log scale). The admissible values of* i *and* $\alpha \geq 1$ *corresponding to* $R_0 > 1$ *are delimited by the black curve* $i = (1+\alpha)/\sqrt{2(1+\alpha^2)}$*. On the left:* $C(\eta^{\mathrm{equi}}) - C(\eta^{\mathrm{opt}})$*. On the right:* $C(\eta^{\mathrm{uni}}) - C(\eta^{\mathrm{equi}})$*.*

defined on Ω^2. We shall also consider that the *incidence rate* of the disease is given by the so-called *law of mass action*; see also [16] for a survey on other incidence rates.

In accordance with [3], we consider for a kernel k on Ω and $q \in (1, +\infty)$ its norm:

$$\|\mathrm{k}\|_{\infty,q} = \sup_{x\in\Omega} \left(\int_\Omega \mathrm{k}(x,y)^q \, \mu(\mathrm{d}y) \right)^{1/q}.$$

For a kernel k on Ω such that $\|\mathrm{k}\|_{\infty,q}$ is finite for some $q \in (1, +\infty)$, we define the integral operator $\mathcal{T}_\mathrm{k}$ on the set $\mathscr{L}^\infty$ of bounded measurable real-valued function on Ω by

$$\mathcal{T}_\mathrm{k}(g)(x) = \int_\Omega \mathrm{k}(x,y) g(y) \, \mu(\mathrm{d}y) \quad \text{for } g \in \mathscr{L}^\infty \text{ and } x \in \Omega. \tag{7}$$

By convention, for f, g two nonnegative measurable functions defined on Ω and k a kernel on Ω, we denote by $f\mathrm{k}g$ the kernel on Ω defined by

$$f\mathrm{k}g : (x,y) \mapsto f(x)\,\mathrm{k}(x,y)g(y). \tag{8}$$

We shall consider the kernel $\mathbf{k} = k/\gamma$ (corresponding to $k\gamma^{-1}$, which differs in general from $\gamma^{-1}k$), which is thus defined by

$$\boxed{\mathbf{k}(x,y) = k(x,y)\,\gamma(y)^{-1}.} \tag{9}$$

We shall assume that

$$\boxed{\|\mathbf{k}\|_{\infty,q} < \infty \quad \text{for some } q \in (1,+\infty).} \tag{10}$$

The integral operator $\mathcal{T}_{\mathbf{k}}$ is the so-called *next-generation operator.*

Let $\Delta = \{f \in \mathscr{L}^\infty : 0 \le f \le 1\}$ be the subset of nonnegative functions bounded by 1, and let $\mathbb{1} \in \Delta$ be the constant function equal to 1. The SIS dynamics considered in [3] follows the vector field F defined on Δ by

$$\boxed{F(g) = (\mathbb{1} - g)\mathcal{T}_k(g) - \gamma g.} \tag{11}$$

More precisely, we consider $u = (u_t, t \in \mathbb{R})$, where $u_t \in \Delta$ for all $t \in \mathbb{R}_+$, and u solves (1), that is,

$$\boxed{\partial_t u_t = F(u_t)} \quad \text{for } t \in \mathbb{R}_+, \tag{12}$$

with initial condition $u_0 \in \Delta$. The value $u_t(x) = u(t,x)$ models the probability that an individual of feature x is infected at time t; it is proved in [3] that such a solution u exists and is unique. We also recall (see [3, Proposition 2.10]) that

$$F(u_0) \ge 0 \quad \Longrightarrow \quad \text{the map } t \mapsto u_t \text{ is pointwise nondecreasing.} \tag{13}$$

An *equilibrium* of (12) is a function $g \in \Delta$ such that $F(g) = 0$. According to [3], there exists a maximal equilibrium $\mathfrak{g}$, i.e., an equilibrium such that all other equilibria $h \in \Delta$ are dominated by $\mathfrak{g}$: $h \le \mathfrak{g}$. It is towards this maximal equilibrium that the process stabilizes when started from a situation where all the population is infected, that is, if $u_0 = \mathbb{1}$, then we have

$$\boxed{\lim_{t\to\infty} u_t = \mathfrak{g}.}$$

More generally (see [3, Proposition 2.13]), for $u_0 \in \Delta$, if $\lim_{t\to\infty} u_t$ exists pointwise, then

$$\lim_{t\to\infty} u_t \quad \text{is an equilibrium.} \tag{14}$$

For T a bounded operator on $\mathscr{L}^\infty$ endowed with its usual supremum norm, we define by $\|T\|_{\mathscr{L}^\infty}$ its operator norm and its spectral radius is given by

$$\rho(T) = \lim_{n\to\infty} \|T^n\|_{\mathscr{L}^\infty}^{1/n}.$$

The *reproduction number* R_0 associated to the SIS model given by (12) is the spectral radius of the next-generation operator:

$$\boxed{R_0 = \rho(\mathcal{T}_{\mathbf{k}}).} \tag{15}$$

If $R_0 \le 1$ (subcritical and critical cases), then u_t converges pointwise to 0 when $t \to \infty$. In particular, the maximal equilibrium $\mathfrak{g}$ is equal to 0 everywhere. If $R_0 > 1$ (supercritical case), then 0 is still an equilibrium but different from the maximal equilibrium $\mathfrak{g}$, as $\int_\Omega \mathfrak{g}\,\mathrm{d}\mu > 0$.

4.2. Vaccination strategies. A *vaccination strategy* η of a vaccine with perfect efficiency is an element of Δ, where $\eta(x)$ represents the proportion of *nonvaccinated* individuals with feature x. Notice that $\eta\,\mathrm{d}\mu$ corresponds in a sense to the effective population. In particular, the "strategy" that consists in vaccinating no one (resp., everybody) corresponds to $\eta = \mathbb{1}$, the constant function equal to 1 (resp., $\eta = \mathbb{0}$, the constant function equal to 0).

Recall the definition of the kernel $f\mathrm{k}g$ from (8). For $\eta \in \Delta$, the kernel $\mathbf{k}\eta = k\eta/\gamma$ has finite norm $\|\cdot\|_{\infty,q}$, so we can consider the bounded positive operators $\mathcal{T}_{\mathbf{k}\eta}$ and $\mathcal{T}_{k\eta}$ on $\mathscr{L}^\infty$. According to [3, section 5.3], the SIS equation with vaccination strategy η is given by $u^\eta = (u_t^\eta, t \geq 0)$ solution to (12), with F replaced by F_η, defined by

$$\boxed{F_\eta(g) = (\mathbb{1} - g)\mathcal{T}_{k\eta}(g) - \gamma g.} \tag{16}$$

Then the quantity $u_t^\eta(x) = u^\eta(t,x)$ represents the probability for a nonvaccinated individual of feature x to be infected at time t; so at time t among the population of feature x, a fraction $1 - \eta(x)$ is vaccinated, a fraction $\eta(x)\,u_t^\eta(x)$ is not vaccinated and infected, and a fraction $\eta(x)\,(1 - u_t^\eta(x))$ is not vaccinated and not infected.

We define the *effective reproduction number* $R_e(\eta)$ associated to the vaccination strategy η as the spectral radius of the effective next-generation operator $\mathcal{T}_{\mathbf{k}\eta}$:

$$\boxed{R_e(\eta) = \rho(\mathcal{T}_{\mathbf{k}\eta}).} \tag{17}$$

For example, for the trivial vaccination strategies we get $R_e(\mathbb{1}) = R_0$ and $R_e(\mathbb{0}) = 0$.

We denote by $\mathfrak{g}_\eta$ the corresponding maximal equilibrium:

$$F_\eta(\mathfrak{g}_\eta) = 0. \tag{18}$$

In particular, we have

$$R_e(\mathbb{1}) = R_0 \quad \text{and} \quad \mathfrak{g} = \mathfrak{g}_{\mathbb{1}}.$$

4.3. Critical vaccination strategies. If $R_0 \geq 1$, then a vaccination strategy η is called *critical* if it achieves precisely herd immunity, that is, $R_e(\eta) = 1$.

As the spectral radius is positively homogeneous (that is, $\rho(\lambda t) = \lambda\rho(T)$ for $\lambda \geq 0$), we also get, when $R_0 \geq 1$, that the uniform strategy that corresponds to the constant function

$$\boxed{\eta_{\mathrm{crit}}^{\mathrm{uni}} = \frac{1}{R_0}\mathbb{1}}$$

is critical, as $R_e(\eta_{\mathrm{crit}}^{\mathrm{uni}}) = 1$. This is consistent with results obtained in the homogeneous model.

5. Proof of the main theorem. We now restate the main result of the paper with precise hypothesis and gives its proof in the next subsections. As hinted in [14, section 4.5] for vaccination control of gonorrhea, it is interesting to consider vaccinating people with feature x with probability $\mathfrak{g}(x)$. This corresponds to the strategy based on the maximal equilibrium:

$$\boxed{\eta^{\mathrm{equi}} = \mathbb{1} - \mathfrak{g}.}$$

The following result entails that this strategy is critical and thus achieves herd immunity. Recall that in the (infinite-dimensional) SIS model (11) on the probability space $(\Omega, \mathscr{F}, \mu)$ the recovery rate function γ is positive and bounded, the transmission rate k is nonnegative, and the norm $\|\,\mathbf{k}\,\|_{\infty,q}$ of the kernel $\mathbf{k} = k/\gamma$ is finite for some $q \in (1, +\infty)$.

THEOREM 5.1 (the maximal equilibrium yields a critical vaccination). *Consider the SIS model* (11) *under the boundedness assumption* (10). *If* $R_0 \geq 1$, *then the vaccination strategy* η^{equi} *is critical, that is,* $R_e(\eta^{\mathrm{equi}}) = 1$.

We recall in sections 5.1 and 5.2 some known results on positive operators and SIS models which can be found in [3] and also give some technical complements. Then Theorem 5.1 is part of Proposition 5.5 and is proved in section 5.3

5.1. Technical results on positive operators. For an operator A, we denote by $A^\top$ its adjoint. For the convenience of the reader, we collect here (an adaptation of) a well-known result of results on positive operators and refer the reader to [3] for a detailed proof. We shall consider the cone $\mathscr{L}^\infty_+ = \{f \in \mathscr{L}^\infty : \ f \geq 0\}$ of nonnegative measurable functions defined on Ω. Let k be a (nonnegative) kernel on Ω with finite norms $\|\cdot\|_{\infty,q}$ for some $q \in (1, +\infty)$. Notice that $\mathcal{T}_{\mathrm{k}}$ is a positive operator as $\mathcal{T}_{\mathrm{k}}(\mathscr{L}^\infty_+) \subset \mathscr{L}^\infty_+$.

- *Existence of Perron eigenvector.* According to Krein–Rutman theorem, and more precisely [3, Lemma 3.7 (v)], there exists $v \in L^q_+ \setminus \{0\}$, seen as an element of the topological dual of $\mathscr{L}^\infty$, being a left Perron eigenfunction of $\mathcal{T}_{\mathrm{k}}$, such that $(\mathcal{T}_{\mathrm{k}})^\top(v) = \rho(\mathcal{T}_{\mathrm{k}})v$.
- *Collatz–Wielandt inequality.* If there exist $g \in \mathscr{L}^\infty_+ \setminus \{0\}$ and $\lambda > 0$ such that $\mathcal{T}(g) \geq \lambda g$, then we have $\rho(\mathcal{T}_{\mathrm{k}}) \geq \lambda$; see [3, Proposition 3.6].
- *Monotonicity of the spectral radius.* Let k be a (nonnegative) kernel on Ω with finite norms $\|\cdot\|_{\infty,q}$. We have that

$$\mathrm{k} \geq \mathrm{k}' \implies \rho(\mathcal{T}_{\mathrm{k}}) \geq \rho(\mathcal{T}_{\mathrm{k}'}), \tag{19}$$

as the operator $\mathcal{T}_{\mathrm{k}} - \mathcal{T}_{\mathrm{k}'}$ is positive; see, for example, [3, Theorem 3.5(i)].

We also recall that for two bounded operators T and S on $\mathscr{L}^\infty$,

$$\rho(TS) = \rho(ST). \tag{20}$$

We shall use the following extension of the Collatz–Wielandt inequality.

LEMMA 5.2. *Let* k *be a nonnegative kernel on* Ω *such that* $\|\mathrm{k}\|_{\infty,q}$ *is finite for some* $q \in (1, +\infty)$ *and consider the positive bounded linear integral operator* $\mathcal{T}_{\mathrm{k}}$ *on* $\mathscr{L}^\infty$. *If there exists* $g \in \mathscr{L}^\infty_+$, *with* $\int_\Omega g \, \mathrm{d}\mu > 0$ *and* $\lambda > 0$ *satisfying*

$$\mathcal{T}_{\mathrm{k}}(g)(x) > \lambda g(x), \quad \textit{for all} \quad x \textit{ such that } g(x) > 0,$$

then we have $\rho(\mathcal{T}_{\mathrm{k}}) > \lambda$.

Proof. We simply write $\mathcal{T}$ for $\mathcal{T}_{\mathrm{k}}$. Let $A = \{g > 0\}$ be the support of the function g. Let $\mathcal{T}'$ be the bounded operator defined by $\mathcal{T}'(f) = \mathbb{1}_A \mathcal{T}(\mathbb{1}_A f)$. Since $\mathcal{T}'(g) = \mathbb{1}_A \mathcal{T}(\mathbb{1}_A g) = \mathbb{1}_A \mathcal{T}(g) > \lambda g$, we deduce from the Collatz–Wielandt inequality (with k replaced by $\mathbb{1}_A \mathrm{k} \mathbb{1}_A$) that $\rho(\mathcal{T}') \geq \lambda > 0$. Let $v \in L^q_+ \setminus \{0\}$ be a left Perron eigenfunction of $\mathcal{T}'$: $(\mathcal{T}')^\top(v) = \rho(\mathcal{T}')v$. In particular, we have $v = \mathbb{1}_A v$ and thus $\int_A v \, \mathrm{d}\mu > 0$ and $\int_\Omega vg \, \mathrm{d}\mu > 0$. We obtain

$$(\rho(\mathcal{T}') - \lambda)\langle v, g\rangle = \langle v, \mathcal{T}'(g) - \lambda g\rangle > 0.$$

As $\mathcal{T}' = \mathcal{T}_{\mathrm{k}'}$ with $\mathrm{k}' = \mathbb{1}_A \mathrm{k} \mathbb{1}_A \leq \mathrm{k}$, we deduce from (19) that $\rho(\mathcal{T}) \geq \rho(\mathcal{T}') > \lambda$. ∎

5.2. Technical results on SIS models. We consider the SIS model from section 4.1 and thus assume that (10) holds. We first state a direct consequence of the monotony property (13).

LEMMA 5.3. *Consider the SIS model* (11) *under the boundedness assumption* (10). *Let* $\eta, g \in \Delta$. *If* $F_\eta(g) \geq 0$, *then we have* $g \leq \mathfrak{g}_\eta$.

Proof. Consider the solution u_t^η of the SIS model $\partial_t u_t^\eta = F_\eta(u_t^\eta)$ with vaccination η and initial condition $u_0^\eta = g$. According to (13) (applied to F_η instead of F), this solution is nondecreasing since $F_\eta(g) \geq 0$. According to (14), the pointwise limit of u_t^η is an equilibrium. As this limit is dominated by the maximal equilibrium $\mathfrak{g}_\eta$ and since u_t^η is nondecreasing, this proves that $g \leq \mathfrak{g}_\eta$. ∎

We recall [3, Proposition 4.2] on equivalent conditions for the supercritical regime. Recall that the spectral bound $s(T)$ of a bounded operator T is defined by

$$s(T) = \sup\{\mathrm{Re}(\lambda) : \ \lambda \quad \text{in the spectrum of } T\}.$$

PROPOSITION 5.4. *Consider the SIS model* (11) *under the boundedness assumption* (10). *The following properties are equivalent:*

(i) $s(\mathcal{T}_k - \gamma) > 0$.
(ii) $R_0 > 1$.
(iii) *There exist* $\lambda > 0$ *and* $g \in \mathscr{L}^\infty_+ \setminus \{0\}$ *such that* $\mathcal{T}_k(g) - \gamma g = \lambda g$.

5.3. Proof of Theorem 5.1. The next result characterizes the maximal equilibrium $\mathfrak{g}$ among all equilibria by various spectral properties; Theorem 5.1 may be viewed as a corollary to this characterization. Recall that $R_0 = R_e(\mathbb{1})$, and that the vector field F is defined by (11). Let $DF[h]$ denote the bounded linear operator on $\mathscr{L}^\infty$ of the derivative of the map $f \mapsto F(f)$ defined on $\mathscr{L}^\infty$ at point h:

$$DF[h](g) = (1-h)\mathcal{T}_k(g) - (\gamma + \mathcal{T}_k(h))g \quad \text{for } h, g \in \mathscr{L}^\infty.$$

PROPOSITION 5.5 (equivalent conditions for maximality). *Consider the SIS model* (11) *under the boundedness assumption* (10). *Let* h *in* Δ *be an equilibrium, that is,* $F(h) = 0$. *The following properties are equivalent:*

(i) $h = \mathfrak{g}$.
(ii) $s(DF[h]) \leq 0$.
(iii) $R_e((1-h)^2) \leq 1$.
(iv) $s(DF_{(1-h)}[\mathbb{0}]) \leq 0$.
(v) $R_e(1-h) \leq 1$.

Furthermore, $\mathfrak{g} = \mathbb{0}$ *if and only if* $R_0 \leq 1$, *and if* $\mathfrak{g} \neq \mathbb{0}$, *then it is critical:* $R_e(1-\mathfrak{g}) = 1$.

Remark 5.6 (on stability). From a dynamical systems point of view, this proposition links together two different stability properties. The (classically equivalent) conditions (ii) and (iii) state that for the original dynamics given by (12) with vector field F, the equilibrium h is not linearly unstable. Similarly, conditions (iv) and (v) both state that in the vaccinated dynamics given by the modified vector field F_{1-h} defined by (16), the disease-free equilibrium 0 is not linearly unstable.

In particular, in the original dynamics given by (12), equilibria that are not maximal are necessarily linearly unstable.

Proof. Let $h \in \Delta$ be an equilibrium, that is, $F(h) = 0$.

The equivalence between (iv) and (v) is a direct consequence of Proposition 5.4.

Let us show the equivalence between (ii) and (iii). According to Proposition 5.4 again, we have $s(DF[h]) \leq 0$ if and only if

$$\rho(\mathcal{T}_\mathrm{k}) \leq 1 \quad \text{with} \quad \mathrm{k}(x,y) = (1-h(x))\frac{k(x,y)}{\gamma(y) + \mathcal{T}_k(h)(y)}.$$

Since $F(h)=0$, we have $(1-h)/\gamma = 1/(\gamma+\mathcal{T}_k(h))$. This gives

$$\mathsf{k}(x,y) = (1-h(x))\frac{k(x,y)(1-h(y))}{\gamma(y)} \tag{21}$$

and thus $\mathcal{T}_{\mathrm{k}} = M_{1-h}\,\mathcal{T}_{k/\gamma}\,M_{1-h}$, where M_f is the multiplication operator by f. Recall the definition (17) of R_e. We deduce from (20) that

$$\rho\left(\mathcal{T}_{\mathrm{k}}\right) = \rho\left(\mathcal{T}_{k/\gamma}M_{(1-h)^2}\right) = R_e((1-h)^2). \tag{22}$$

This gives the equivalence between (ii) and (iii).

We prove that (i) implies (v). Suppose that $R_e(1-h)>1$. Thanks to (20), we have $\rho(M_{1-h}\mathcal{T}_{k/\gamma}) = \rho(\mathcal{T}_{k/\gamma}M_{1-h}) = R_e(1-h)>1$. Let $v\in L^q_+\setminus\{0\}$ be a left Perron eigenfunction of $\mathcal{T}_{(1-h)k/\gamma}$, that is, $\mathcal{T}^{\top}_{(1-h)k/\gamma}(v) = R_e(1-h)v$. Using $F(h)=0$, and thus $(1-h)\mathcal{T}_k(h)=\gamma h$, for the last equality, we have

$$R_e(1-h)\langle v,\gamma h\rangle = \langle v,(1-h)\mathcal{T}_{k/\gamma}(\gamma h)\rangle = \langle v,\gamma h\rangle.$$

We get $\langle v,\gamma h\rangle = 0$ and thus $\langle v,\mathbb{1}_A\rangle = 0$, where $A=\{h>0\}$ denotes the support of the function h. Since $\mathcal{T}^{\top}_{(1-h)k/\gamma}(v) = R_e(1-h)v$ and setting $v' = (1-h)v$ (so that $v'=v$ μ-almost surely on A^c), we deduce that

$$\mathcal{T}^{\top}_{k'/\gamma}(v') = R_e(1-h)v',$$

where $k' = \mathbb{1}_{A^c}\,k\,\mathbb{1}_{A^c}$. This implies that $\rho(\mathcal{T}_{k'/\gamma})\geq R_e(1-h)$. Since $k' = (1-h)k'$ and $k-k'\geq 0$, we get that $\mathcal{T}_{k/\gamma}-\mathcal{T}_{k'/\gamma}$ is a positive operator. Using (19) for the inequality as $(1-h)k'/\gamma \leq (1-h)k/\gamma$, we deduce that $\rho(\mathcal{T}_{k'/\gamma}) = \rho(M_{1-h}\mathcal{T}_{k'/\gamma}) \leq \rho(M_{1-h}\mathcal{T}_{k/\gamma}) = R_e(1-h)$. Thus, the spectral radius of $\mathcal{T}_{k'/\gamma}$ is equal to $R_e(1-h)$. According to Proposition 5.4, since $\rho(\mathcal{T}_{k'/\gamma})>1$, there exist $w\in\mathscr{L}^{\infty}_+\setminus\{0\}$ and $\lambda>0$ such that

$$\mathcal{T}_{k'}(w) - \gamma w = \lambda w.$$

This also implies that $w=0$ on $A=\{h>0\}$, that is, $wh=0$ and thus $w\mathcal{T}_k(h)=0$ as $\mathcal{T}_k(h) = \gamma h/(1-h)$. Using that $F(h)=0$, $\mathcal{T}_k(w) = \mathcal{T}_{k'}(w) = (\gamma+\lambda)w$ and $h\mathcal{T}_k(w)=0$, we obtain

$$F(h+w) = w(\lambda - \mathcal{T}_k(w)).$$

Taking $\varepsilon>0$ small enough so that $\varepsilon\mathcal{T}_k(w)\leq\lambda/2$ and $\varepsilon w\leq 1$, we get $h+\varepsilon w\in\Delta$ and $F(h+\varepsilon w)\geq 0$. Then we use Lemma 5.3 to deduce that $h+\varepsilon w\leq\mathfrak{g}$ and thus $h\neq\mathfrak{g}$.

To see that (v) implies (iii), notice that $(1-h)\geq(1-h)^2$, and then use (19) to deduce that $\rho(\mathcal{T}_{\mathbf{k}(1-h)})\geq\rho(\mathcal{T}_{\mathbf{k}(1-h)^2})$ and thus $R_e(1-h)\geq R_e((1-h)^2)$.

We prove that (iii) implies (i). Notice that $F(g)=0$ and $g\in\Delta$ implies that $g<1$. Assume that $h\neq\mathfrak{g}$. Notice that $\gamma/(1-h) = \gamma+\mathcal{T}_k(h)$, so that $\gamma(\mathfrak{g}-h)/(1-h)\in\mathscr{L}^{\infty}_+$. An elementary computation, using $F(h)=F(\mathfrak{g})=0$ and k defined in (21), gives

$$\mathcal{T}_{\mathrm{k}}\left(\gamma\frac{\mathfrak{g}-h}{1-h}\right) = (1-h)\mathcal{T}_k\left(\mathfrak{g}-h\right) = \gamma\frac{\mathfrak{g}-h}{1-\mathfrak{g}} = \frac{1-h}{1-\mathfrak{g}}\,\gamma\frac{\mathfrak{g}-h}{1-h}.$$

Since $h\neq\mathfrak{g}$ and $h\leq\mathfrak{g}$, we deduce that $(1-h)/(1-\mathfrak{g})\geq 1$, with strict inequality on $\{\mathfrak{g}-h>0\}$ which is a set of positive measure. We deduce from Lemma 5.2 (with k replaced by $\mathrm{k}\gamma$) that $\rho\left(\mathcal{T}_{\mathsf{k}}\right)>1$. Then we use (22) to conclude.

To conclude notice that $\mathfrak{g} = 0 \iff R_0 \leq 1$ is a consequence of the equivalence between (i) and (v) with $h = 0$ and $R_0 = R_e(\mathbb{1})$.

Using that $F(\mathfrak{g}) = 0$, we get $\mathcal{T}_k(\mathfrak{g}) = \gamma\mathfrak{g}/(1-\mathfrak{g})$. We deduce that $\mathcal{T}_{k(1-\mathfrak{g})/\gamma}(\mathcal{T}_k(\mathfrak{g})) = \mathcal{T}_k(\mathfrak{g})$. If $\mathfrak{g} \neq 0$, we get $\mathcal{T}_k(\mathfrak{g}) \neq 0$ (on a set of positive μ-measure). This implies that $R_e(1-\mathfrak{g}) \geq 1$. Then use (v) to deduce that $R_e(1-\mathfrak{g}) = 1$ if $\mathfrak{g} \neq 0$. ∎

REFERENCES

[1] T. Britton, F. Ball, and P. Trapman, *A mathematical model reveals the influence of population heterogeneity on herd immunity to SARS-CoV-2*, Science, 369 (2020), pp. 846–849, https://doi.org/10.1126/science.abc6810.

[2] L. Y. H. Chan, G. Rø, J. E. Midtbø, F. D. Ruscio, S. S. V. Watle, L. K. Juvet, J. Littmann, P. Aavitsland, K. M. Nygård, A. S. Berg, G. Bukholm, A. B. Kristoffersen, K. Engø-Monsen, S. Engebretsen, D. Swanson, A. D.-L. Palomares, J. C. Lindstrøm, A. Frigessi, and B. F. de Blasio, *Modeling geographic vaccination strategies for COVID-19 in Norway*, PLoS Comput. Biol., 20 (2024), e1011426, https://doi.org/10.1371/journal.pcbi.1011426.

[3] J.-F. Delmas, D. Dronnier, and P.-A. Zitt, *An infinite-dimensional metapopulation SIS model*, J. Differential Equations, 313 (2022), pp. 1–53, https://doi.org/10.1016/j.jde.2021.12.024.

[4] J.-F. Delmas, D. Dronnier, and P.-A. Zitt, *Optimal vaccination: Various (counter) intuitive examples*, J. Math. Biol., 86 (2023), pp. 1–57, https://doi.org/10.1007/s00285-022-01858-5.

[5] J.-F. Delmas, D. Dronnier, and P.-A. Zitt, *Optimal Vaccinations: Cordons Sanitaires, Reducible Population and Optimal Rays*, preprint, arXiv: 2209.07381, 2022.

[6] J.-F. Delmas, D. Dronnier, and P.-A. Zitt, *Targeted Vaccination Strategies for an Infinite-Dimensional SIS Model*, preprint, arXiv:2103.10330, 2021.

[7] J.-F. Delmas, D. Dronnier, and P.-A. Zitt, *The effective reproduction number: Convexity, concavity and invariance*, J. Eur. Math. Soc., (2024), https://doi.org/10.4171/jems/1431.

[8] J.-F. Delmas, D. Dronnier, and P.-A. Zitt, *Vaccinating highly connected people is (sometimes) optimal*, Forthcoming.

[9] J.-F. Delmas, P. Frasca, F. Garin, V. C. Tran, A. Velleret, and P.-A. Zitt, *Individual-based SIS models on (Not So) dense large random networks*, ALEA, Lat. Am. J. Probab. Math. Stat., 21 (2024), pp. 1375–1405, https://doi.org/10.30757/ALEA.v21-52.

[10] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, *On the definition and the computation of the basic reproduction ratio in models for infectious diseases in heterogeneous populations*, J. Math. Biol., 28 (1990), pp. 365–382, https://doi.org/10.1007/BF00178324.

[11] K. Dietz, *Transmission and control of arbovirus diseases*, Epidemiology, 104 (1975), pp. 104–121.

[12] P. Fine, K. Eames, and D. L. Heymann, *Herd immunity: A rough guide*, Clin. Infect. Dis., 52 (2011), pp. 911–916, https://doi.org/10.1093/cid/cir007.

[13] H. Heesterbeek, *The law of mass-action in epidemiology: A historical perspective*, in Ecological Paradigms Lost: Routes of Theory Change, Academic Press, 2005, pp. 81–104.

[14] H. W. Hethcote and J. A. Yorke, *Gonorrhea Transmission Dynamics and Control*, S. Levin, ed., Lecture Notes in Biomath. 56, Springer-Verlag, 1984.

[15] A. Lajmanovich and J. A. Yorke, *A deterministic model for gonorrhea in a nonhomogeneous population*, Math. Biosci., 28 (1976), pp. 221–236, https://doi.org/10.1016/0025-5564(76)90125-5.

[16] H. McCallum, N. Barlow, and J. Hone, *How should pathogen transmission be modelled?*, Trends Ecol. Evol., 16 (2001), pp. 295–300, https://doi.org/10.1016/S0169-5347(01)02144-9.

[17] A. Nold, *Heterogeneity in disease-transmission modeling*, Math. Biosci., 52 (1980), pp. 227–240, https://doi.org/10.1016/0025-5564(80)90069-3.

[18] C. E. G. Smith, *Prospects for the control of infectious disease*, Proc. Roy. Soc. Med., 63 (1970), pp. 1181–1190, https://doi.org/10.1177/003591577006311P206.

[19] P. G. Smith, *Concepts of herd protection and immunity*, Procedia Vaccinol., 2 (2010), pp. 134–139, https://doi.org/10.1016/j.provac.2010.07.005.

[20] D. R. M. Smith, J. Turner, P. Fahr, L. A. Attfield, P. R. Bessel, C. A. Donnelly, R. Gibb, K. E. Jones, D. W. Redding, D. Asogun, O. O. Ayodeji, et al., *Health and economic impacts of Lassa vaccination campaigns in West Africa*, Nature Med., 30 (2024), pp. 3568–3577, https://doi.org/10.1038/s41591-024-03232-y.

[21] M. Somerville, K. Kumaran, and R. Anderson, *Public Health and Epidemiology at a Glance,* 2nd ed., John Wiley & Sons, 2016.

[22] J. Wallinga, P. Teunis, and M. Kretzschmar, *Using data on social contacts to estimate age-specific transmission parameters for respiratory-spread infectious agents*, Amer. J. Epidemiol., 164 (2006), pp. 936–944, https://doi.org/10.1093/aje/kwj317.