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Homophily and infections: Static and dynamic effects

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ABSTRACT

We analyze the effect of homophily in the diffusion of a harmful state between two groups of agents that differ in immunization rates. Homophily has a very different impact on the steady state infection level (that is increasing in homophily when homophily is small, and decreasing when high), and on the cumulative number of infections generated by a deviation from the steady state (that, instead, is decreasing in homophily when homophily is small, and increasing when high). If immunization rates are endogenous, homophily has the opposite impact on the infection level of the two groups. However, the sign of the group-level impact is reversed if immunization is motivated by infection risk or peer pressure. If motivations are group-specific, homophily can decrease immunization in both groups.

1. Introduction

It is well known that diffusion processes in social networks are crucially affected by homophily, namely the tendency of members of a group to interact among themselves more than across groups. In this paper, our goal is to show that homophily can have a very different impact on the long-run steady state and on the diffusion dynamics. Specifically, we study the diffusion of a harmful state in a population where immunization is available. We can think of the harmful state as an infectious disease, a harmful behavior (tobacco use, as in Galeotti and Rogers, 2013[1]), or a rumor the belief in which may be harmful (as in Merlino et al., 2023). In the following, we use for simplicity the terminology of the diffusion of a disease, referring to immunization as vaccination.

We study a stylized environment with two heterogeneous groups of agents, one with a high vaccination rate, the other with a low vaccination rate, and we focus on the case of a non-zero stable steady state for infection. At time 0 there is a random shock to the number of infected (which we sometimes refer to as an *outbreak*), that generates a dynamic adjustment. After that time, the system converges back to the stable steady state.

We analyze the impact of homophily on two measures: the steady-state number of infections, and the cumulative number of infected-person-periods generated during an outbreak (which we refer to as "cumulative infection", for simplicity). Our main contributions are two: first, we show that the effect of an increase in homophily on the *steady state* infection level can be diametrically opposite to the effect on cumulative infection. Second, we show that homophily has an opposite effect on vaccination rates depending

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[1] In the example of tobacco use, immunization "is interpreted as a commitment to avoid the temptation of smoking" (Galeotti and Rogers, 2013).

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on whether the motivation for infection is a classic "rational" cost-benefit analysis, or instead depends on peer pressure (an important factor according to some recent evidence, reviewed in the literature section).

Empirically observed social networks exhibit a high degree of homophily, and homophily is one of the network characteristics crucially affecting diffusion and contagion (see, e.g. Jackson, 2008). Moreover, certain policies that affect vaccination uptake may also affect homophily. For example, there is evidence that in the US private and charter schools have a higher level of non-vaccinated children, and this is driven by a larger number of families that use the possibility of religious or philosophical exemptions (see the discussion and references in Section 4.2.). Another example, focusing on the tobacco use interpretation, is that smokers likely prefer to spend time in spaces that either allow smoking, or have spaces to do it: for example some types of bars, or clubs. Once again, different enforcement of smoking regulations can have the effect of increasing homophily of interaction between people with similar immunization (commitment) rates.

To model the diffusion of the harmful state, we adopt the SIS ("susceptible-infected-susceptible") model, following Galeotti and Rogers (2013) and Jackson (2008).[2] The network is stylized and composed of two groups, with heterogeneous vaccination rates. Vaccination is perfectly effective and is not subject to waning. In the basic model, vaccination rates are exogenous. We later endogenize vaccination rates and microfound the discrepancy across groups, by assuming that agents in a group perceive a higher cost of vaccination. With this in mind, we label the two groups "*vaxxers*" and "*anti-vaxxers*".[3] The homophily of contacts between the two groups is modeled by a parameter $h \in (0,1)$, which is the percentage of social contacts that people have exclusively with others in their same group, while the rest of contacts is with a fraction of agents drawn at random from the population.

We consider deviations from the steady state of an amount that is stochastic and has zero mean. For tractability, we focus on the linear approximation of the dynamics around the steady state. We study the effect of homophily on two measures of infection: a static one, the *steady-state infection*; and a dynamic one, which is the discounted sum of each period's infections due to a deviation from the steady state, that we call *cumulative infection*. This cumulative measure of infections can be seen as a reduced form of various utilitarian welfare functions that have been used in the recent literature. All of these examples are connected to the idea that each infection in each time period has a cost, and these costs must be summed: the most similar being Rowthorn and Toxvaerd (2012), Farboodi et al. (2021), and Toxvaerd and Rowthorn (2022). An example in this sense is discussed in detail in Remark 1.[4] The key characteristic of the cumulative infection is that, contrary to the steady-state level of infection, it contains information on the way the epidemic evolves over time across groups, how long it lasts, and society's discount rate. If infections are fixed at the steady state, this discounted sum is the same as the steady-state prevalence. However, in an outbreak, when the infection level can vary over time, the two objects can be very different.

Our first main contribution is to show that the effect of homophily on infections may be opposite on the steady state and on the cumulative infection. In particular, we find that the steady-state total infection level is increasing for small homophily h and decreasing for large homophily h (Proposition 2). Instead, cumulative infection follows the opposite behavior: it is decreasing in homophily for small h and increasing for h large (Proposition 3). The key intuition behind the result is as follows. In the steady state, a change in homophily has a direct effect of increasing infections in the group with less vaccinated agents, because they meet non-vaccinated people more often, and decrease them in the other, for the symmetric reason. Then, there are indirect effects due to the impact of the steady-state levels on the dynamics. The key determinants of these indirect effects are (i) a (negative) *size* effect: a higher infection decreases susceptibles, hence decreases future infections; and (ii) a (positive) *contagion* effect: higher infection increases future infections, increasing contagion probability. When homophily is small, the size effect is symmetric, hence the sign of the impact of homophily is determined by the contagion effect and is positive; on the other hand, when homophily is large, the contagion effect is symmetric, so the sign is determined by the size effect and is negative. In Section 3.2, we provide further discussion of the mechanisms, among which we also show that homophily decreases the convergence rate of the system (Proposition 4).

In the main result we do not adopt a specific utility interpretation, because our point is descriptive. However, when in Section 4.1.1 we introduce agents' preferences, to endogenize vaccination choices, we show how a modification of a utilitarian welfare function, with the addition of a health care cost that must be financed through uniform taxes, gives rise to a welfare function that depends both on the steady state level of infection *and* the cumulative infection.[5]

To study the effect of vaccination rates that adjust when homophily changes we explore a model in which agents trade-off a heterogeneous vaccination cost with their perceived benefit of vaccination. Furthermore, we assume that the two groups differ only in (possibly) size and in their judgment about the real cost of vaccination, which is deemed higher by anti-vaxxers. This can be thought of as a psychological cost, a sheer mistake, or any phenomenon that may lead to a difference in perceived cost: we remain agnostic on the cause of it as our aim is to study its consequences.[6] We explore two different possibilities for the motivation of vaccinations:

[2] In the Online Appendix B we show that the main insight remains true if we model the epidemic process using a SIR, instead of a SIS, model: indeed, in the SIR model the steady state has zero infections, so homophily has no effect on the steady state, but it does affect the dynamics and the cumulative infection.

[3] Actually, our main results of Section 2 (for exogenous vaccination rates) only depend on the heterogeneity between the number of susceptibles in each group, and not on the fact that this heterogeneity comes from vaccination (even if it is a natural example). It could also, for example, come from a difference in infectivity across age or ethnic groups: our result on the impact of homophily would be the same.

[4] Moreover, as we discuss in Section 3.2, this dynamic component can be thought of as the Bonacich (1987) centrality of each group in the network composed by the two groups, where the strength of the connection between groups depends on the probability of contagion.

[5] We thank an anonymous referee for suggesting this interpretation.

[6] In recent years many people either refuse drastically any vaccination scheme or reduce (or delay) the prescribed vaccination. The phenomenon has become more pronounced in the last decades, especially in Western Europe and in the US. See Larson et al. (2016) for a general cross country comparison, Phadke et al. (2016) for the US and Funk (2017) for measles in various European countries. With the model in the main text we mean to capture not the extremists that would never take a vaccine, but the more general phenomenon of *vaccine hesitancy*, which is more widespread and, so, potentially more dangerous (Trentini et al., 2017).

vaccinations motivated by avoiding the risk of infections, and vaccinations motivated by peer pressure. In the former, agents evaluate the benefit of vaccination as the negative of the infection rate: the gain in utility if they do not get the illness. In the latter, agents receive a high benefit from vaccination if many other agents in their neighborhood are vaccinated, through a peer effect channel. The cost-benefit model is classic, a variation of the one studied in Galeotti and Rogers (2013). We explore the peer effects model because there is a lot of evidence suggesting that peer effects affect decision making, and specifically decisions related to health and insurance. In Section 4.2 we review the empirical evidence. Real decision making likely involves a mix of peer effects and cost-benefit calculation, so these two cases should be thought of as the two extreme cases in which only one of the two components is visible, for the sake of illustrating the mechanisms.

Our second main contribution is to show that the effect of homophily on vaccination rates is opposite in the two vaccination models. Indeed, if vaccinations are motivated by the risk of infection, an increase in homophily has the effect of increasing risk, hence vaccinations, among anti-vaxxers, and decreasing them among vaxxers (Proposition 5). Instead, if vaccinations are motivated by peer pressure the mechanism is the opposite: an increase in homophily increases the peer pressure in the group with more vaccinations (the vaxxer group), hence increasing vaccination among the vaxxers, and decreasing them among the anti-vaxxers (Proposition 7). Homophily is the most harmful to vaccinations in a hybrid model in which vaxxers vaccinate according to the risk of infection, while anti-vaxxers according to peer pressure. In such a case homophily unambiguously decreases vaccinations both among vaxxers (because it reduces risk), and among anti-vaxxers (because it decreases the peer pressure).

Moreover, even in the endogenous vaccination models, the basic insight that steady-state and cumulative infection measures may have opposite behavior for the extremes values of homophily survives. In the rational model, this is true when the vaccination cost is high enough (Proposition 6). In the peer-effects based model, it is always true (Proposition 7). This happens, essentially, because vaccination rates adjust in opposite directions, hence the additional effect is never too strong.

1.1. Related literature

We contribute to three lines of literature: the literature on epidemics in economics, the literature on contagion and diffusion in networks, and the literature on strategic immunization.

Our contribution to the literature on epidemics in economics is first to study how homophily impacts infections, and more generally to highlight how different risk and time preferences used to evaluate the welfare impact of an epidemic may give different weights to the steady state and to the cumulative infection. Our cumulative measure of infections can be seen as a reduced form of various utilitarian welfare functions that have been used in the recent literature: the most similar being Rowthorn and Toxvaerd (2012), Farboodi et al. (2021), and Toxvaerd and Rowthorn (2022). Other papers use richer models, studying the tradeoffs between economic activity and deaths (both absent from our model): Acemoglu et al. (2021), Brotherhood et al. (2024), Bognanni et al. (2020). All of these papers do not consider the effect of the social network. Bisin and Gottardi (2021) consider health and economic trade-offs, but do not consider dynamics or homophily. The structure of the cross-country network is considered in Chandrasekhar et al. (2021); in their paper, the social planner tries to minimize what they call the “number of infected-person-periods”, a measure analogous to cumulative infection. None of these papers studies the homophily of interactions.

Our contribution to the literature on contagion is to highlight how the effect of homophily of interactions can be radically different when focusing on the cumulative number of infections over time, rather than the steady state. The closest paper, from which we adapt the basic setting, is Galeotti and Rogers (2013). That paper was the first to study in depth the effect of homophily on infection rates. However, the authors focus on the steady-state level of infections, rather than the dynamic measure of cumulative infection we focus on. It is well known that homophily can facilitate the diffusion of a disease, as illustrated, e.g., in Jackson and López-Pintado (2013). However, they do not study the impact of homophily on steady-state levels or the dynamic measure of cumulative infections. Izquierdo et al. (2018) and Burgio et al. (2022) study the steady state and find a non-monotonic effect of homophily similar to our result, but they do not study the dynamic cumulative infection. A different strand of literature has argued that the whole time evolution of the dynamics is important beyond the steady state, studying departures from the standard mean-field approximation to allow stochastic fluctuations, as in Nakamura and Martinez (2019) and Esen et al. (2022). These papers do not study the effect of homophily.

Our contribution to the literature on strategic immunization models is to show that the impact of homophily on group-level vaccinations can be the opposite if vaccination is motivated by a rational cost-benefit analysis or by peer pressure. Our model of vaccinations motivated by infection risk is analogous to Galeotti and Rogers (2013). However, the endogenous vaccination model in Galeotti and Rogers (2013) generates symmetric vaccination in the two groups, because it assumes a homogeneous vaccination cost, while we use heterogeneous vaccination costs precisely to microfound and study different vaccination rates. Goyal and Vigier (2015) study the interaction between the endogenous level of interaction and vaccinations, again in a steady state. The fact that vaxxers tend to vaccinate less when homophily increases is similar to the risk compensation effect studied in Talamàs and Vohra (2020), who show that a partially effective vaccination can decrease welfare. Again, our focus is rather on the static-dynamic trade-offs. Chen and Toxvaerd (2014) argue that the market mechanism yields inefficiently low levels of vaccination. None of these papers explores vaccination driven by peer pressure.[7]

The paper is organized as follows. The next section presents the model. Section 3 shows the main results for the mechanical model, when all choices are exogenous. Section 4 explores the robustness of the results to endogenous vaccination rates. In Section 4.2 we

[7] There is also a recent literature in applied physics that studies models where the diffusion is simultaneous for the disease and for the vaccination choices. On this, see the review of Wang et al. (2015), and the more recent analysis of Alvarez-Zuzek et al. (2017) and Velásquez-Rojas and Vazquez (2017).

extend the model by considering the case in which vaccination choices are motivated by peer pressure. We conclude in Section 5. In Appendix A we prove the formal results of our paper. The Online Appendix contains further results: in Appendix B we study how our setup applies to the SIR model; in Appendix C we discuss the optimal vaccination rates.

2. The model

We consider a simple SIS model with vaccination and two groups of agents, analogous to the setup in Galeotti and Rogers (2013).

The population is composed of a continuum of agents of mass 1, exogenously partitioned into two groups. Agents in each group are characterized by their attitude towards vaccination. In detail, following a popular terminology, we label the two groups with a, for *anti-vaxxers*, and with v, for *vaxxers*. Thus, the set of the two groups is $G := \{a, v\}$, with $g \in G$ being the generic group. Let $q \in (0,1)$ denote the fraction of *anti-vaxxers* in the society and $1-q$ the fraction of *vaxxers*.

People in the two groups meet each other with an *homophilous* bias. We model this by assuming that an agent of any of the two groups has a probability $h \in (0,1)$ to meet only someone from her own group and a probability $1-h$ to meet someone else randomly drawn from the whole society.[8] This implies that anti-vaxxers meet each other at a rate of $\tilde{q}_a := h + (1-h)q$, while vaxxers meet each other at a rate of $\tilde{q}_v := h + (1-h)(1-q)$. Note that h is the same for both groups, but if $q \neq 1/2$ and $h < 1$, then $\tilde{q}_a \neq \tilde{q}_v$.

For each $g \in G$, let $x_g \in (0,1)$ denote the fraction of agents in group g that are vaccinated against the disease. It is natural to assume, without loss of generality, that $x_a < x_v$, and in the benchmark model this is the only difference between the two groups. The total number of vaccinated (or average vaccination rate) is $x = qx_a + (1-q)x_v$. Sometimes we write $\Delta x = x_v - x_a$ to simplify formulas. We start by taking x_a and x_v as exogenous parameters, and we endogenize them later. We assume $x_g \in (0,1)$ to make the problem nontrivial. Similarly, $\rho_g \in [0, 1-x_g]$ denote the fraction of infected out of the whole population of group g, and $S_g = 1 - x_g - \rho_g$ denote the susceptible: agents who are neither vaccinated, nor infected, and thus susceptible to being infected by other infected agents. The total number of vaccinated is denoted $\rho = q\rho_a + (1-q)\rho_v$. Whenever there is possible ambiguity, steady-state variables are denoted with an SS apex, so that the total number of infections in the steady state is denoted ρ^{SS}. We omit the SS apex whenever the context makes it clear that we are using steady-state values. Let μ be the recovery rate of the disease, whereas its infectiveness is normalized to 1.

We study the stable steady state of the system above and with stochastic, zero-mean, deviations from the steady state. At time 0, there is the realization of the random variables $d\rho_0 = (d\rho_{0,a}, d\rho_{0,v})$, measuring such deviation, where $\mathbb{E}\mathrm{d}\rho_{0,a} = \mathbb{E}\mathrm{d}\rho_{0,v} = 0$. For simplicity, we assume that the deviation is symmetric across the two groups: $d\rho_{0,a} = d\rho_{0,v} = \overline{d\rho_0}$. This is already sufficient to show the difference between the impact of homophily in the steady state and in the dynamics, which is our goal; so we stick to this simplifying assumption.

2.1. The dynamical system

Setting the evolution of the epidemic in continuous time, we study the fraction of infected people in each group.

The differential equations of the system are given by:

$$\begin{aligned}\dot{\rho}_a &= F_a(\rho_a, \rho_v) = S_a\left(\tilde{q}_a\rho_a + (1-\tilde{q}_a)\rho_v\right) - \rho_a\mu;\\ \dot{\rho}_v &= F_v(\rho_a, \rho_v) = S_v\left(\tilde{q}_v\rho_v + (1-\tilde{q}_v)\rho_a\right) - \rho_v\mu,\end{aligned} \tag{1}$$

where $\tilde{\rho}_a := \left(\tilde{q}_a\rho_a + (1-\tilde{q}_a)\rho_v\right)$ and $\tilde{\rho}_v := \left(\tilde{q}_v\rho_v + (1-\tilde{q}_v)\rho_a\right)$ are the shares of infected agents met on average by anti-vaxxers and vaxxers, respectively, and $\rho_a\mu$ and $\rho_v\mu$ are the fractions of recovered agents in each group.

First, in the next proposition we characterize some properties of the steady states, using a classic result in Lajmanovich and Yorke (1976) (see also Observation 1 in Galeotti and Rogers, 2013).

Proposition 1 *(Homophily and endemic disease). The system* (1) *always admits a unique stable steady state. For each* $h \in [0,1]$*, there exists a* $\hat{\mu}(h) > 0$ *such that (i) if* $\mu < \hat{\mu}(h)$*,* $(0,0)$ *is an unstable steady state, and in addition there is another unique stable steady state; whereas (ii) if* $\mu \geq \hat{\mu}(h)$*, the unique steady state is* $(0,0)$*, and is globally asymptotically stable.*

The formal passages of the proof are in Appendix A, as those of the other results that follow. In all the paper, from now on, we focus on the interior steady state, and to ensure that this exists for all h we assume $\mu < 1 - x_v$.

For analytical tractability, in the following we approximate the dynamics of outbreaks away from the steady state with the linearized dynamic of the deviation from the steady state $\mathrm{d}\rho_{i,t} = \rho_{i,t} - \rho_i^{SS}$, for $i = a, v$.

Definition 1 *(The linearized dynamic).* We define the functions $\mathrm{d}\rho_{a,t}$, $\mathrm{d}\rho_{v,t}$ as the time evolutions that satisfy:

$$\begin{pmatrix}\dot{\mathrm{d}}\rho_{a,t}\\ \dot{\mathrm{d}}\rho_{v,t}\end{pmatrix} = J\begin{pmatrix}\mathrm{d}\rho_{t,a}\\ \mathrm{d}\rho_{t,v}\end{pmatrix}, \quad \mathrm{d}\rho_0 = \begin{pmatrix}\mathrm{d}\rho_{0,a}\\ \mathrm{d}\rho_{0,v}\end{pmatrix},$$

[8] h is the *inbreeding homophily* index, as defined in Coleman (1958), Marsden (1987), McPherson et al. (2001) and Currarini et al. (2009). It can be interpreted as the amount of time that agents spend interacting with people in their group, while in the remaining time they meet uniformly at random.

where

$$J = \begin{pmatrix} -\tilde{\rho}_a^{SS} - \mu + S_a^{SS}\tilde{q}_a & \left(1 - S_a^{SS}\right)\tilde{q}_a \\ \left(1 - S_v^{SS}\right)\tilde{q}_v & -\tilde{\rho}_v^{SS} - \mu + S_v^{SS}\tilde{q}_v \end{pmatrix}$$

is the Jacobian matrix of (1) calculated in the steady state, and $\mathrm{d}\rho_0' = (\mathrm{d}\rho_{0,a}, \mathrm{d}\rho_{0,v})'$ is the initial magnitude of the outbreak.

Moreover, we denote: $\mathrm{d}\rho_t = q\mathrm{d}\rho_{a,t} + (1-q)\mathrm{d}\rho_{v,t}$.

If $\mu < \hat{\mu}$, the steady state is stable, implying that J has positive determinant: we denote it by $|J| > 0$. Additionally, $\hat{\mu}(h)$ (explicitly derived in Appendix A) is increasing in h. This highlights the first important role of h in comparative statics: if h increases, a disease that was previously non-endemic (because $\mu > \hat{\mu}(h)$) might become endemic as $\hat{\mu}(h)$ increases with h, reversing the inequality (see also the discussion in Jackson and López-Pintado, 2013).

2.2. Cumulative infection

Suppose that, instead of the number of infected in the steady state, society cares about the total number of infected-person-periods generated by the epidemic, as termed by Chandrasekhar et al. (2021). For example, if each infection has a cost to the healthcare system, this aggregate number would correspond to the total monetary cost of healthcare during an epidemic.

In this section, we precisely define the concept. A key simplification is that the dynamics $\mathrm{d}\rho_{t,a}$ and $\mathrm{d}\rho_{t,v}$ are, by construction, linear in $\mathrm{d}\rho_0$. Therefore, the total cumulative number of infections over time is also linear in $\mathrm{d}\rho_0$.

Definition 2 *(Cumulative infection).* Define $\tilde{CI}$ as the (normalized) cumulative number of infections due to a deviation from the steady state of size $\mathrm{d}\rho_0 = (\overline{d\rho_0}, \overline{d\rho_0})$, discounted with discount rate r:

$$\begin{aligned}\tilde{CI} &:= r\left(q\int_0^\infty e^{-rt}(\rho_a^{SS} + \mathrm{d}\rho_{a,t})\mathrm{d}t + (1-q)\int_0^\infty e^{-rt}(\rho_v^{SS} + \mathrm{d}\rho_{v,t})\mathrm{d}t\right) \\ &= \rho^{SS} + r\int_0^\infty e^{-rt}\mathrm{d}\rho_t\mathrm{d}t\end{aligned}$$

Moreover, thanks to the linearity of $\mathrm{d}\rho_t$, define CI as the coefficient such that:

$$\tilde{CI}\left(\overline{d\rho_0}\right) = \rho^{SS} + CI\,\overline{d\rho_0} \tag{2}$$

Since the number of infected in the steady state and the cumulative number of infections due to the deviation from the steady state are distinct quantities, both potentially of interest for policymakers, in the following we separately analyze the impact of homophily on the two measures: the steady state infection level ρ, and the cumulative infection CI. The idea is that both these statistics may be relevant, depending on the problem studied, but homophily has a very different impact on each.

3. Steady state vs cumulative infection

In this section we analyze the epidemic, taking the vaccination rates x_a and x_v as exogenous. Remember that, in this case, the only difference between the two groups is that $x_a < x_v$. In the following, we drop the superscript SS from steady state variables whenever there is no ambiguity, for ease of notation.

3.1. Homophily in steady state

First, we explore what is the effect of homophily in the steady state. Homophily has the effect of increasing the social contacts among agents of the same group: as a consequence, an increase in homophily h has the effect of increasing the amount of not vaccinated people that anti-vaxxers interact with, and this increases the steady-state infection level among anti-vaxxers. The opposite effect is true for the vaxxers. What is the balance of these effects? The next proposition answers.

Proposition 2 *(Homophily in the steady state). In the interior steady state the total infection ρ is increasing if homophily is sufficiently small, and decreasing if homophily is sufficiently high.*[9]

The intuition for the results on group-level infection rates is the following. An increase in homophily has a direct effect, due to the change in the meeting rates across groups; and an indirect effect, due to the change in the steady state. The direct effects are caused by the homophily changing the probability of infection:

[9] For completeness, we can show it has only one maximum under the assumption that $\mu < (1-x)^2/(1-x_a)$.

$$\partial_h \tilde{\rho}_a = (1-q)\Delta\rho;$$
$$\partial_h \tilde{\rho}_v = -q\Delta\rho. \tag{3}$$

where, for simplicity, we denote the derivative with respect to a variable x as ∂_x. The derivatives of the infection rates have opposite signs: anti-vaxxers meet more frequently other anti-vaxxers hence, ceteris paribus, their probability of infection goes up with homophily. For vaxxers, the opposite happens.

The indirect effects are due to the impact that each infection level has on the dynamic increments $\dot{\rho}_a = F_a(\rho_a, \rho_v)$ and $\dot{\rho}_v = F_v(\rho_a, \rho_v)$. They can be decomposed as such:

$$\begin{aligned} \mathrm{d}F_a =& \partial_{\rho_a}(S_a\tilde{\rho}_a)\mathrm{d}\rho_a + \partial_{\rho_v}(S_a\tilde{\rho}_a)\mathrm{d}\rho_v \\ =& (\underbrace{-\mu}_{\substack{\text{recovery}\\ \text{effect}}} - \underbrace{\tilde{\rho}_a}_{\substack{\text{size}\\ \text{effect}}} + \underbrace{\tilde{q}_a S_a}_{\substack{\text{contagion}\\ \text{effect}}})d\rho_a + \underbrace{(1-\tilde{q}_a)S_a}_{\substack{\text{contagion}\\ \text{effect}}} d\rho_v; \\ \mathrm{d}F_v =& (\underbrace{-\mu}_{\text{recovery}} - \underbrace{\tilde{\rho}_v}_{\text{size}} + \underbrace{\tilde{q}_v S_v}_{\text{contagion}})d\rho_a + \underbrace{(1-\tilde{q}_v)S_v}_{\text{contagion}} d\rho_v. \end{aligned} \tag{4}$$

For example, for group a, an increase in the steady-state level ρ_a generates: (i) an increase of recovered, (ii) a decrease in the pool of susceptible agents (size effect), and (iii) a increase in the probability of infection (contagion effect). The recovery effect is constant, and symmetric across groups. Since the amount of newly infected is the product of the number of susceptible and the probability of infection, each of these two effects are respectively proportional to the level of the other (via the Leibniz differentiation rule). The size effect, that is the reduction in the pool of susceptible agents, is proportional to the infection probability $\tilde{\rho}_a$: hence it is stronger for anti-vaxxers. The size effect is always negative. Finally, there is the contagion effect, due to the increase in the probability of meeting an infected person. This is positive, and its magnitude depends on q, but for $q = 1/2$ it is proportional to the share of susceptible agents, and so the effect is once again stronger for anti-vaxxers group. Considering group a, the recovery and the size effect are only present for a variation of the own steady state level ρ_a, while the contagion effect is present both for the own steady state ρ_a, and for a variation in the steady state of the other group ρ_v.

The indirect effects always have the opposite sign compared to the direct effects, making the overall balance of uncertain sign. The results above indicate that the indirect effects are never strong enough to counterbalance the direct effect in group a, so ρ_a always increases with $\rho_{0,a}$. In group v, however, the derivative can take both signs: it is negative if the direct effect prevails, and positive otherwise. Since cross-group contagion is part of the indirect effect, the direct effect prevails and ρ_v decreases with $\rho_{0,a}$ when h is large, meaning the two groups are almost separated. Conversely, when h is small, the indirect effect may be stronger than the direct one, causing ρ_v to increase with $\rho_{0,a}$. This occurs when the recovery rate μ is large enough, making the contagion effect a more significant driver of infection.[10]

Note that also the effect of homophily on total infections stems from a balance of such direct and indirect effects. The expressions for the derivative in (4) reflect the fact that the variations of steady-state levels are a combination of the direct effects from (3), weighted by the responses of the dynamics to a variation in the steady state, in such a way to leave the dynamics at rest.

Summing up, the sign of $\partial_h \rho$ is determined by

$$S_a(\tilde{\rho}_v + \mu) - S_v(\tilde{\rho}_a + \mu)$$

which represents the balance of the strengths of the contagion effects (whose magnitude are proportional to S_a and S_v) and of the size effects (whose magnitude are $\tilde{\rho}_a$ and $\tilde{\rho}_v$).

When homophily h is low, the infection probabilities are the same $\tilde{\rho}_a \sim \tilde{\rho}_v$, hence the size effects, that are proportional to them, do not matter: the contagion effect, which is positive, dominates and so infections increase in h. Instead, when homophily is high, the amount of susceptible agents is the same in the two groups, $S_a, S_v \to \mu$, hence the contagion effect does not matter, and the result is determined by the size effect, which is negative: homophily decreases total infections.[11]

3.2. Cumulative infection

We now analyze how results are affected once we explicitly model the infection dynamic. First, the linearized dynamic can be explicitly solved as: $(\mathrm{d}\rho_{t,a}, \mathrm{d}\rho_{t,v})' = e^{Jt}\mathrm{d}\rho_0$. Using this formula, we note that the cumulative infection CI is closely related to Bonacich centrality:

[10] Notice that this reasoning holds only when both infection rates do not reach corner solutions: if for example $x_v = 1$, so that all the vaxxers are vaccinated, then $\rho_v = 0$ is a constant and does not change; in this case the only relevant derivative is:

$$\partial_h \rho_a = \partial_h \rho = -\frac{S_a(1-q)\Delta\rho}{J_{11}} > 0$$

[11] One might wonder why the population size q has little effect on the result. Note that the marginal changes in the probability of infection (and hence both $\partial_h \rho_a$ and $\partial_h \rho_v$) depend on the fraction of the population in the *other* group: this is the amount of the change in people met for a unit increase in h. The consequence is that when computing the total infection, the population fractions can be collected, because each term is multiplied by $q(1-q)$, and does not matter anymore.

$$\begin{pmatrix} CI_a \\ CI_v \end{pmatrix} = r \int_0^\infty e^{(-rI+J)t}\mathbf{1}\mathrm{d}t = (I - 1/rJ)^{-1}\mathbf{1},$$

where $\mathbf{1} = (1,1)'$, and the inverse exists because $I - 1/rJ$ has eigenvalues $1 + 1/r\lambda_1, 1 + 1/r\lambda_2 > 0$. We can see that the vector of cumulative infections in the two groups is equal to the Bonacich centrality of each group in the weighted network that has adjacency matrix equal to the Jacobian matrix J. In such a network a link between g and h has a high weight if the number of newly infected in g due to contacts with h is high, for $g, h \in \{a, v\}$. The expression above is going to be useful in making calculations with the cumulative infection. The intuition can be better grasped considering the associated discrete time dynamics, that satisfies:

$$\begin{pmatrix} \mathrm{d}\rho_{a,t+1} \\ \mathrm{d}\rho_{v,t+1} \end{pmatrix} = (J + I)\begin{pmatrix} d\rho_{a,t} \\ d\rho_{v,t} \end{pmatrix}. \tag{5}$$

In such a case, calling the discount factor β, the total infection is simply

$$(1-\beta)\sum_t \beta^t (I+J)^t \mathrm{d}\rho_0 = (I - \beta/(1-\beta)J)^{-1}\mathrm{d}\rho_0.$$

Each step in the time iteration adds a number of infections proportional to the direct and indirect connections in the weighted connection network defined above up to step t. The sum of all the total direct and indirect discounted connections amounts to the total cumulative infection over time, and is equal to the Bonacich centrality. The continuous time result follows a similar logic.

The next Proposition is the main result of this section, showing that CI has opposite behavior with respect to ρ^{SS}.

Proposition 3. *Cumulative infection CI is decreasing in homophily h when h is sufficiently small, and is increasing when h is sufficiently high.*

What is the reason for this discrepancy? The total effect can be decomposed into a direct effect of h on the dynamics, and an indirect effect, due to h affecting also the steady-state levels:

$$\mathrm{d}_h CI = \underbrace{\partial_h CI}_{\text{direct effect}} + \underbrace{\partial_{\rho_a} CI \partial_h \rho_a + \partial_{\rho_v} CI \partial_h \rho_v}_{\text{indirect effect}},$$

where we denote as $d_h CI$ the total derivative with respect to h, and as $\partial_h CI$ the direct effect, net of the effect of the change in the steady state levels ρ_a, ρ_v. In the proof of the above Proposition, we clarify that the direct effect $\partial_h CI$ is positive if h high enough, and the indirect effect has a sign that is exactly opposite to the sign of the effect on the steady state in Proposition 2. In the following paragraphs we try to give intuitions for both.

Intuition: direct effect. To better understand the intuition behind the direct effect of h on the cumulative infection, we turn again to the approximate discrete dynamics (5). Let us analyze a simple two-step discrete version of the dynamics. At $t = 1$ we have:

$$d\rho_{a,1} = (-\tilde{\rho_a} - \mu + \tilde{q}_a S_a)\overline{d\rho_0} + (1 - \tilde{q}_a)S_a\overline{d\rho_0},$$
$$\mathrm{d}\rho_{a,1} = (-\tilde{\rho_v} - \mu + \tilde{q}_v S_a)\overline{d\rho_0} + (1 - \tilde{q}_v)S_v\overline{d\rho_0}.$$

The gap with total infection at the steady state is:

$$d\rho_1 = (-\rho^{SS} - \mu + qS_a + (1-q)S_v)\overline{d\rho_0} = (-2\rho^{SS} - \mu + 1 - x)\overline{d\rho_0},$$

and hence we can see that this is *independent* of homophily h. The fact that the two groups have an identical initial deviation $\overline{d\rho_0}$ means that only the average effects matter: the average of the contagion effect terms is equal to the average (total) number of susceptible agents, while the average size effect is equal to the total number of infections. Hence, after one period, only population-level statistics matter.

However, the two deviations $d\rho_{a,1}$ and $d\rho_{v,1}$ are not identical. After one period, the gap between groups' infections $d\rho_{1,a} - d\rho_{1,v} = \Delta\mathrm{d}\rho$ is:

$$\Delta\mathrm{d}\rho_1 = (\Delta S - h\Delta\rho^{SS})\overline{d\rho_0},$$

which once again, depends on the size and contagion effects previously discussed. Similarly to the derivatives in (2), both effects are stronger for anti-vaxxers, so if h is low, since the size effect is symmetric, the contagion effect dominates and the gap is positive; the opposite happens when h is high. These effects are analogous to the effects driving the impact of homophily on steady state infections.

To compute total infections at period 2, we can decompose the new delta infection rates as deviations from the average number of infected agents: $d\rho_{a,1} = d\rho_1 + (1-q)\Delta\mathrm{d}\rho_1$ and $\mathrm{d}\rho_{v,1} = d\rho_1 - q\Delta\mathrm{d}\rho_1$. Then, we can express the new total infections at period 2 as:

$$d\rho_2 = -\mathrm{d}\rho_1^2 + q(1-q)h(\Delta S - \Delta\rho^{SS})\Delta\mathrm{d}\rho_1. \tag{6}$$

By linearity, we get two additive terms: one derives from the average component $\mathrm{d}\rho_1$, while the other derives from the deviations, proportional to $\Delta\mathrm{d}\rho_1$. The first term implies analogous calculations as the total infection at time 1, when starting from homogeneous initial deviations: hence it is also independent of homophily, conditional on the steady state infection.

The second term is the crucial one, containing the effect of homophily h on the total infections at period 2, conditional on steady state values. Once again, we see that it depends on the balance of size ($\Delta\rho^{SS}$) and contagion (ΔS) effects. However, the important part is that the sign of the effect also depends on the increment at the previous period, whose sign depends itself on h. In particular, when h is large and the size effect dominates, the gap $\Delta\mathrm{d}\rho_1$ is *negative*, hence the overall sign is *positive*, which is the opposite conclusion than what we get in the steady state.[12] The reason is that, as Equation (6) describes, homophily changes, together with the steady state levels, also the intermediate steps of the dynamics.

This example shows a short-run intuition for the discrepancy between the static and dynamic effects, that the Proposition above shows formally in infinite time. The next paragraph shows how we can get similar long-run intuitions analyzing the behavior of the convergence rate, as measured by the smallest eigenvalue (in absolute value).

Intuition: indirect effect. To understand the intuitive connection between CI and the share of infected agents at steady state, ρ^{SS}, it is useful to first focus on the case in which groups are totally separated, namely $h = 1$. In this case, each group follows an independent standard SIS equation (we report the equation for the a group)[13]:

$$\dot{\rho}_a = S_a\rho_a - \mu\rho_a = (1 - \rho_a - x_a)\rho_a - \mu\rho_a.$$

The linearization of this process is given by:

$$\dot{\mathrm{d}\rho}_a = -\rho_a^{SS}\mathrm{d}\rho_a \Longrightarrow \mathrm{d}\rho_a(t) = e^{-\rho_a^{SS}t}\overline{\mathrm{d}\rho}_0$$

so that we can analytically compute: $CI = \frac{r}{\rho_a^{SS}+r}$: the cumulative infection is inversely proportional to the steady state infection. The intuition is that the higher the steady-state infection, the fewer susceptible agents are, so that the deviation from the steady state is smaller and the system goes back to the steady state faster.

If $h \neq 1$, the dynamics is paired and a clear analytical inverse proportionality is lost. However, to clarify the dynamic intuition behind the mechanism, in the next paragraph we show that, following a similar intuition, the convergence rate of the dynamics, measured by the smallest eigenvalue of J, is decreasing in the steady-state levels.

Convergence rate. To formalize the intuitions discussed in the previous two paragraphs, we consider the following classic definition (see, e.g. Gabaix et al., 2016).

Definition 3 *(Convergence rate).* The convergence rate of the system after a deviation of size $\mathrm{d}\rho_0$ is

$$CR = -\lim_{t\to\infty}\frac{\log\|e^{tA}\mathrm{d}\rho_0\|}{t}.$$

A classic property of linear systems is that the speed of convergence can be measured by eigenvalues: we show formally that this is also the case here. We show that, similarly to Golub and Jackson (2012), also in this context homophily decreases the convergence rate, at least when h and μ are large: this also provides a long-run intuition behind the direct effect in Proposition 3. Moreover, we show that the speed of convergence is increasing in both steady-state levels, ρ_a^{SS} and ρ_v^{SS}, consistently with the fact that CI decreases with the steady state infections.

Proposition 4. *The convergence rate is equal to the absolute value of the eigenvalue of smallest modulus of the matrix $J - rI$:*

$$CR = \lambda_2$$

When $h \to 1$, CR is decreasing in h. When $h \to 0$, CR is decreasing if and only if $\mu > \frac{1-x}{2}$. Moreover, CR is increasing in both ρ_a^{SS} and ρ_v^{SS}.

[12] If, instead, h is small, the gap $\Delta\mathrm{d}\rho_1$ is positive, and the sign of the term $\Delta S - \Delta\rho^{SS}$ is uncertain: for $h = 0$ it is positive if and only if $\mu > (1 - x)/2$. This is because when μ is large the contagion effect is more important than past infections.

[13] An analogous result can be obtained for $h = 0$, because in this case we can average the two equations and obtain an equation for the evolution of the total infection ρ directly:

$$\dot{\rho} = (1 - \rho - x)\rho - \mu\rho.$$

4. Vaccination choices: infection risk vs peer effects

Our model so far is agnostic on agents' preferences. However, in reality, vaccination is an endogenous choice, and the fraction of vaccinated agents in the population might itself depend on homophily. So, it might be important to explore how the discussed mechanisms interact with agents' decision to vaccinate or not. Endogenizing vaccination choices requires us to carefully define what are the motivations for vaccinating. Because of the reasons described in the introduction, we explore two alternatives: first, a standard model (a variant of Galeotti and Rogers, 2013) in which agents correctly anticipate the equilibrium probability of infection; and another one, in which agents' choices are driven by peer effects.

4.1. Vaccination based on rational choices

4.1.1. Preferences

Agents might vaccinate paying a cost, or not vaccinate incurring the risk of becoming infected. We assume that agents take vaccination decisions ex-ante, before an epidemic takes place, and cannot update their decision during the diffusion.[14] Vaccination is perfectly effective. In a descriptive spirit, we microfound the discrepancy in vaccination rates assuming that anti-vaxxers have a cost larger than vaxxers of a uniform amount $d > 0$. However, our focus being on the effect of homophily on infections, we do not dig deeper into the motivations for this different cost evaluation. Thus, we assume that, for vaxxers, vaccination costs are $c^v \sim U[0, 1/k]$, whereas for anti-vaxxers $c^a \sim U[d/k, 1/k + d/k]$. The parameter k reflects the distribution of vaccination costs in the population: a high k means that vaccination costs are generally small, whereas a low k means that they are high.[15]

The benefits of vaccinations are weighed against the disutility of infection. Focus on an agent i in group a, denoted as $i \in a$. We define its *infected status* $I_{i,a,t} \in \{0, 1\}$ as 1 if they are infected, and zero otherwise, and analogously for $i \in v$. The expected utility of agent i in group g is:

$$U_{i,g} = \begin{cases} -r\mathbb{E}\int_0^{+\infty} e^{-rt} I_{i,g,t}\, dt & \text{if } i \text{ non vaccinated} \\ -c_i & \text{o.w.} \end{cases}$$

Since $I_{i,g,t}$ is bounded and has finite expectation, we can pass the expectation inside the integral, so that:

$$\mathbb{E}\int_0^{+\infty} e^{-rt} I_{i,g,t}\, dt = \int_0^{+\infty} e^{-rt}\mathbb{E}(I_{i,g,t})\, dt$$

Conditional on the realization of the initial deviation $d\rho_0$, the variables $(I_{i,a,t}, I_{i,v,t})$ follow a continuous time Markov chain. Following Pastor-Satorras et al. (2015), the time evolution of the expectations $\mathbb{E}(I_{i,g,t} \mid d\rho_0)$ satisfies:

$$\frac{d}{dt}\mathbb{E}(I_{i,a,t} \mid d\rho_0) = (1 - \mathbb{E}(I_{i,a,t} \mid d\rho_0))\tilde{\rho}_{a,t} - \mu\mathbb{E}(I_{i,a,t} \mid d\rho_0) \tag{7}$$

$$\frac{d}{dt}\mathbb{E}(I_{i,v,t} \mid d\rho_0) = (1 - \mathbb{E}(I_{i,v,t} \mid d\rho_0))\tilde{\rho}_{v,t} - \mu\mathbb{E}(I_{i,v,t} \mid d\rho_0) \tag{8}$$

This is exactly the same equation satisfied by $\left(\frac{\rho_{a,t}}{1-x_a}, \frac{\rho_{v,t}}{1-x_v}\right)$ (obtained dividing each equation in (1) for, respectively, $1 - x_a$ and $1 - x_v$), and by the uniqueness of the solution, it must be:

$$\mathbb{E}(I_{i,a,t} \mid d\rho_0) = \frac{\rho_{a,t}}{1-x_a}, \quad \mathbb{E}(I_{i,v,t} \mid d\rho_0) = \frac{\rho_{v,t}}{1-x_v}$$

Furthermore, using our linearization, we have $\mathbb{E}\rho_{a,t} = \rho_a^{SS}$ and $\mathbb{E}\rho_{v,t} = \rho_v^{SS}$. So, the ex-ante expected utilities are:

$$U_{i,g} = \begin{cases} -\dfrac{\rho_g^{SS}}{1-x_g} & \text{if } i \text{ non vaccinated} \\ -c_i & \text{o.w.} \end{cases}$$

Since there is a continuum of agents, each individual takes the fraction of vaccinated in the population as given. As in Galeotti and Rogers (2013), when deciding to vaccinate or not, agents compare the fraction of time spent in the infected state and the cost of vaccination. The difference is that we assume that the vaccination cost is heterogeneous across agents. So, an agent i in group a vaccinates if and only if $c_i < \rho_a$. The fraction of agents that vaccinate are thus:

[14] In general, agents might find it optimal to postpone the vaccination, for two reasons: to learn first the size of the shock away from the steady state $d\rho_0$, or to postpone in the future the disutility of paying the one-off vaccination cost, $-re^{-rt}c$. However, if we allow full flexibility to agents, the model becomes intractable, because now agents vaccinate at different times, and we have to keep track of two additional differential equations for the evolution of $x_a(t)$ and $x_v(t)$. So, for the sake of tractability, we simplify the problem by assuming that agents can vaccinate only before the epidemic starts.

[15] Our model would not change dramatically if we attribute the difference in perception to the costs of becoming sick, but we stick to the first interpretation because it makes the computations cleaner.

$$\begin{cases} x_a &= k\dfrac{\rho_a^{SS}}{1-x_a} - d \\ x_v &= k\dfrac{\rho_v^{SS}}{1-x_v}, \end{cases} \tag{9}$$

whenever the solution is satisfied with equality. In this section, we allow $x_a \in [0,1]$ and $x_v \in [0,1]$, since we cannot in general guarantee that they are in the interior solution. We look for a configuration (x_a^*, x_v^*) such that equations (9) are satisfied with equality, and we call it a *vaccination equilibrium*.

Remark 1. Utilitarian welfare

With the individual preferences just defined, the utilitarian welfare would be, supposing each agent i is identified by its vaccination cost c:

$$\begin{aligned} W &= q\int U_{i,a}di + (1-q)\int U_{i,v}di \\ &= -q\left(\int_{d/k}^{x_a/k} kdc + \int_{x_a/k}^{(1+d)/k} k\frac{\rho_a}{1-x_a}dc\right) \\ &\quad -(1-q)\left(\int_{0}^{x_v/k} kcdc + \int_{x_v/k}^{1/k} k\frac{\rho_v}{1-x_v}dc\right) \\ &= -q\frac{1}{2k}\left(x_a^2 - d^2\right) + \frac{d\rho_a}{1-x_a} - (1-q)\frac{x_v^2}{2k} - q\rho_a^{SS} - (1-q)\rho_v^{SS} \end{aligned}$$

Now, in the decentralized equilibrium $x_a = k\dfrac{\rho_a}{1-x_a} - d$, and we can use this to rewrite the above as:

$$W = -\frac{1}{2}\left(q(x_a+d)^2 + (1-q)x_v^2\right) - \rho^{SS}$$

Utilitarian welfare with healthcare costs

Now, suppose that each infection above the steady-state level costs b' to the health care system. This can be interpreted as follows: the health care system is prepared to deal with a number of infected individuals equal to the steady-state ρ^{SS}, but additional infections have a cost in terms of congestion for the health care system: for example because they require building up additional capacity in terms of personnel, equipment, and hospital space.[16] This is what happened, for example, during the COVID-19 pandemic. These additional health care costs have to be financed through taxes. Therefore, the government must raise an amount equal to $b'\mathbb{E}(\overline{d\rho_0} \mid \overline{d\rho_0} \geq 0)CI$, and it does so via a lump-sum tax t levied uniformly on all agents: so this would not affect the vaccination decision of individuals. So, the aggregate welfare would become:

$$W^{\text{congestion}} = -\frac{1}{2}\left(q(x_a+d)^2 + (1-q)x_v^2\right) - \rho^{SS} - bCI \tag{10}$$

where we defined $b := b'\mathbb{E}\left(\overline{d\rho_0} \mid \overline{d\rho_0} \geq 0\right)$, that is positive by definition. This shows a simple rationale for which total welfare depends on both the steady-state infection levels, and the cumulative infection.

The following lemma guarantees the existence and uniqueness of a vaccination equilibrium.

Lemma 1. *For each $h \in (0,1)$ there are thresholds $\overline{d}$ and $\overline{k}$ such that if $d < \overline{d}$ and $k < \overline{k}$ (the detailed conditions are in the proof in the Appendix), there exist a unique vaccination equilibrium, and in this equilibrium $x_a^* < x_v^*$.*

The proof of Lemma 1 uses a version of the global implicit function theorem.

4.1.2. The effect of homophily on vaccination rates

Now we study what is the effect of a change in homophily on the equilibrium values of x_a^* and x_v^*.

Proposition 5. *In the vaccination equilibrium, if $\mu < (1-x)^2/(1-x_a)$, homophily has the opposite effect on the vaccination rates of the two groups: x_a^* is increasing in h, and x_v^* is decreasing in h.*

[16] We thank an anonymous referee for suggesting this interpretation.

The mechanism behind the comparative statics is that, as h increases, the group with more vaccinated people (the vaxxers) is more protected against infection, so the expected cost of infection $k\rho_v$ decreases, and as a result, a smaller fraction of the vaxxers is vaccinated: x_v is decreasing in h. The opposite happens for anti-vaxxers.

4.1.3. The effect of homophily on steady state and cumulative infection

We have seen that homophily has opposite impacts on the fraction of vaccinated agents in the two groups. We now study the balance of these effects on the aggregate infection level. The next Proposition shows that, at least for k and d small enough, the signs of the derivatives with respect to homophily are the same as in the model with exogenous vaccination rates. This indicates that the insight that homophily might have a very different impact on the steady state or throughout the dynamics is robust and does not disappear once we endogenize vaccination rates.

Proposition 6. *There are levels of d and k small enough such that:*

1. *if homophily h is sufficiently small, the steady state infection ρ is increasing in h, and if h is sufficiently large, ρ is decreasing in h;*
2. *if homophily h is sufficiently small, the cumulative infection CI is decreasing in h, and if h is sufficiently large, CI is increasing in h.*

4.2. Vaccination motivated by peer pressure

In this section we explore an alternative scenario in which vaccination decisions are not driven by a correct evaluation of the infection risk. Instead, we consider agents who do not know or consider the infection risk at all but vaccinate purely motivated by peer pressure: they vaccinate if a sufficient fraction of the agents they meet are vaccinated. Under this vaccination model, we show that if homophily is large enough, no one vaccinates, and so infection rates are higher with respect to the case in which vaccination is based on infection risk. Moreover, the behavior of vaccination rates with respect to homophily is the opposite of what happens with vaccination based on the risk of infection: x_a is *decreasing* in h, while x_v is increasing in h. This is because in this case agents are insensitive to risk, and increasing homophily tends to increase the peer pressure to vaccinate for vaxxers, and to decrease it for anti-vaxxers. Despite these differences, we show that in this alternative framework, the steady-state and the cumulative infection display the same qualitative behavior, with respect to a change in homophily for $h \to 1$, as in the baseline framework we presented.

4.2.1. Why peer effects in vaccinations?

There is a lot of evidence concerning the role of peer effects on health-related choices, e.g.: in the use of social insurance (Markussen and Røed, 2015), the adoption of menstrual cups (Oster and Thornton, 2012), HIV testing (Godlonton and Thornton, 2012), retirement plan choice (Duflo and Saez, 2003). Recently, many papers have found evidence of peer effects in vaccination choices. Rao et al. (2017) use randomized assignment of students to dorms to estimate the peer effect in flu vaccination decisions, finding a significant effect. Hoffmann et al. (2019) study randomized allocation of worker shifts to weekdays or weekends and find evidence of peer effects on vaccination decisions, which they argue are driven by the desire to conform to the social norm of the group. In an RCT, Sato and Takasaki (2019) find peer effects in tetanus vaccination take-up in Nigeria. Ibuka et al. (2018) empirically analyze online survey data, finding evidence of positive peer effects in vaccination decisions.

More generally, there is indirect evidence suggesting that health behavior displays clustering in social networks: for example, see Sorensen (2006) on health insurance plan choice. A phenomenon that attracted lot of attention is the fact that vaccination exemptions required by parents on religious grounds tend to concentrate in some types of schools, such as Steiner and Waldorf schools, where they constitute a large fraction of the population. The phenomenon attracted a lot of discussion by medical researchers: Ernst and Jacobs (2012), May and Silverman (2003), Muscat (2011), Zier and Bradford (2020). It is documented for California in Silverman and Yang (2019), and has been connected to a measles outbreak in Manhattan in April 2019 (Pager, 2019, see also Mashinini et al., 2020, and the earlier discussion by Shaw et al., 2014). Recently, similar trends have been reported in Italy (Cicchetti, 2019). There is reason to believe that these more permissive schools have attracted parents that are more skeptical of vaccinations. For example, Sobo (2015) argues that school community norms have an important impact in vaccine skepticism among families of children attending Steiner schools.

Edge et al. (2019) document that vaccination patterns in a network of social contacts of physicians in Manchester hospitals are correlated with being close in the network. Geographic clustering of vaccination behavior is another fact that suggests a role of peer effects: Lieu et al. (2015) show that vaccine-hesitant people are more likely to communicate with each other than with others. Cullen et al. (2023) find that health care providers can affect the vaccination decisions of the social networks of their patients.

4.2.2. Formal model

The agents in the two groups have heterogeneous vaccination costs, whose distribution follows the same assumptions as in Section 4. The key difference is the evaluation of the health effect of being vaccinated. Rather than evaluating the disutility connected to the average infection rate, as in Section 4, agents evaluate the peer pressure stemming from other agents' vaccination decisions. In particular, agents compare the vaccination cost with a peer pressure term equal to the average number of infected individuals in their neighborhood.

Formally, an agent in group a with cost c vaccinates if $c < \tilde{q}_a x_a + (1 - \tilde{q}_a)x_v$, and an agent in group v, analogously, vaccinates if $c < (1 - \tilde{q}_v)x_a + \tilde{q}_v x_v$. Hence, the vaccination rates at interior solutions are defined by:

$$\begin{aligned} x_a &= k(\tilde{q}_a x_a + (1-\tilde{q}_a)x_v) - d, \\ x_v &= k(\tilde{q}_v x_v + (1-\tilde{q}_v)x_a). \end{aligned} \tag{11}$$

When h is not too large, these equations have interior solutions given by:

$$x_a = \frac{d(1-k(1-(1-h)q))}{(k-1)(1-hk)}, \quad x_v = \frac{d(1-h)kq}{(k-1)(1-hk)},$$

while for h high enough the peer effects are so high that either no one vaccinates or everyone vaccinates. In particular, this means that, for $h \to 1$, we have $\partial_h x_a = \partial_h x_v = 0$. So, for $h \to 1$, the effect of h when vaccinations are exogenous is exactly the same as when vaccinations are endogenous, and so the results of Section 3 apply.

The derivatives of the interior solutions with respect to homophily are:

$$\partial_h x_a = -\frac{dk(1-q)}{(hk-1)^2}, \quad \partial_h x_v = \frac{dkq}{(hk-1)^2},$$

where it is immediately clear that the signs are opposite with respect to the signs of the derivatives in the rational model in Section 4. The reason is that, here, homophily magnifies the influence of peer effects within each group. In the a group, where there are fewer vaccinated individuals, an increase in homophily tends to decrease peer pressure to vaccinate, thereby reducing infection levels. This is diametrically opposite to the effect of homophily in the rational model: there, homophily magnifies the risk of infection from individuals of the same group, leading to an increase in vaccinations in the a group. The opposite occurs in the v group.

The next Proposition characterizes all the vaccination equilibria, and formalizes the results discussed above.

Proposition 7. *In the model of vaccination induced by peer pressure:*

1. *No vaccinations ($x_a^* = x_v^* = 0$) is always an equilibrium. Moreover, there is also an interior equilibrium if $k > 1$, $h < 1 - \frac{k-1}{kq}$ and $d < \frac{(k-1)(1-hk)}{kq(1-h)}$ (the complete characterization can be found in the proof in Appendix A.3.4).*
2. *In the range of h such that the vaccination rates are interior, $\partial_h x_a < 0$ and $\partial_h x_v > 0$.*
3. *For h sufficiently large, the steady state infection is decreasing ($d_h \rho^{SS} < 0$) but the cumulative infection is increasing ($d_h CI > 0$), as in Proposition 6.*

4.2.3. A mixed model

Finally, we consider a model with mixed motivations. Specifically, we consider the case where anti-vaxxers, instead of correctly evaluating the risk, rely on peer pressure, whereas vaxxers are more prone to evaluate risks accurately. This example is consistent with empirical evidence showing that anti-vaccine attitudes are more prone to diffusion in online social networks (see, e.g., Puri et al., 2020). Under these assumptions, for some parameter values, an increase in homophily unambiguously decreases vaccination rates in *both groups*: vaxxers vaccinate less due to perceived lower risk, while anti-vaxxers vaccinate less due to peer pressure. To be formal, assume:

$$\begin{cases} x_a &= k(\tilde{q}_a x_a + (1-\tilde{q}_a)x_v) - d, \\ x_v &= k\rho_v. \end{cases}$$

Thanks to the implicit function theorem the derivatives of vaccination rates are the following:

$$\begin{pmatrix} \partial_h x_a \\ \partial_h x_v \end{pmatrix} = -\frac{1}{det} \begin{pmatrix} -(1-k\partial_{x_v}\rho_v)k(1-h)\Delta x - k^2(1-\tilde{q}_a)\partial_h \rho_v \\ -k^2 \partial_{x_a}\rho_v (1-h)\Delta x - (1-k\tilde{q}_a)k\partial_h \rho_v \end{pmatrix}$$

where $det = (1-k\partial_{x_v}\rho_v)(1-k\tilde{q}_a) - k^2(1-\tilde{q}_a)\partial_{x_a}\rho_v$. If $1 > k\tilde{q}_a$, since $\partial_{x_v}\rho_v < 0$ and $\partial_{x_a}\rho_v < 0$ we have $det > 0$. Moreover, since $\partial_h \rho_v < 0$ it follows $\partial_h x_v < 0$. Finally, if Δx is small enough, we also have $\partial_h x_a < 0$.

Hence, when peer pressure is the driver of vaccination in the group with higher cost evaluation, while in the other group is the fear of infection, for some parameter values homophily unambiguously decreases equilibrium vaccinations in both.

5. Conclusion

Our study investigates the effects of homophily on infection dynamics within a population divided into “vaxxers” and “anti-vaxxers.” Our key findings can be summarized as follows.

First, we demonstrate that homophily has a nuanced impact on infection dynamics. Specifically, homophily has a hump-shaped impact on steady-state infection levels but a U-shaped impact on the cumulative number of infections. This finding underscores the importance of considering both static and dynamic effects when evaluating the role of homophily in disease spread.

Second, we explore the differences between endogenous and exogenous vaccination rates. When vaccination rates are endogenous, homophily affects the two groups differently. For anti-vaxxers, increased homophily reduces peer pressure to vaccinate, leading to lower infection levels within this group. Conversely, for vaxxers, homophily enhances vaccination rates due to increased peer pressure, thereby mitigating overall infection rates through higher vaccination levels. We also investigate scenarios where vaccination decisions are influenced by peer pressure rather than a rational evaluation of infection risk. Our results indicate that high levels of homophily

can result in lower vaccination rates in both groups. Vaxxers may perceive a lower risk, while anti-vaxxers are swayed by peer pressure, both contributing to reduced vaccination uptake.

In conclusion, while our findings provide valuable insights into the interplay between homophily and diffusion, they also highlight potential challenges in formulating effective public health strategies. For instance, policies aimed at reducing contact with anti-vaxxers might inadvertently prolong outbreaks and exacerbate cumulative infection rates. However, this point requires careful consideration of broader epidemiological and social factors. By addressing these points, we hope to provide a clearer understanding of the nuanced effects of homophily on vaccination behavior and infection dynamics, which can inform more effective public health strategies.

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Declaration of competing interest

We declare that we have no relevant or material financial interests that relate to the research described in this paper.

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Appendix A. Proofs

A.1. Proofs of Section 2

A.1.1. Proof of Proposition 1 (page 4)

Proof. The Jacobian of the dynamical system is:

$$J = \begin{pmatrix} -\mu - \tilde{\rho}_a + \tilde{q}_a S_a & (1-\tilde{q}_a)S_a \\ (1-\tilde{q}_v)S_v & -\mu - \tilde{\rho}_v + \tilde{q}_v S_v \end{pmatrix} = \begin{pmatrix} A & B \\ C & D \end{pmatrix}$$

In the following, we are denoting the entries of the Jacobian as A, B, C, D, for simplicity.

Define:

$$\lambda_1 = -\frac{1}{2}\left(A + D - \sqrt{(A-D)^2 + 4BC}\right)$$

$$\lambda_2 = -\frac{1}{2}\left(A + D + \sqrt{(A-D)^2 + 4BC}\right)$$

It can be checked that $-\lambda_1$ and $-\lambda_2$ are the two eigenvalues of the matrix J (we define the quantities with the minus because of convenience in later formulas). Since we assume $h \in (0,1)$ and $q \in (0,1)$, we have $\tilde{q}_a < 1$ and $\tilde{q}_v < 1$. Moreover, by Lemma A below, S_a and S_v are positive in the interior steady state. So, $BC > 0$, which means both eigenvalues are real, and from the expression above it follows $\lambda_1 > \lambda_2$.

Now let us focus on the Jacobian computed in the point $(0,0)$:

$$J^0 = \begin{pmatrix} -\mu + \tilde{q}_a(1-x_a) & (1-\tilde{q}_a)(1-x_a) \\ (1-\tilde{q}_v)(1-x_v) & -\mu + \tilde{q}_v(1-x_v) \end{pmatrix}$$

and denote the corresponding eigenvalues as $-\lambda_1^0$ and $-\lambda_2^0$.

By Theorem 3.1 in Lajmanovich and Yorke (1976), for the system (1) there are two possibilities: if $\lambda_2^0 \geq 0$ (so that both the eigenvalues are negative), then there in only the zero steady state, and it is asymptotically stable. If instead $\lambda_2^0 < 0$, then there is also another steady state, and is asymptotically stable for any initial condition $(\rho_a, \rho_v) \neq (0,0)$.

Using the expression above, the condition $\lambda_2^0 < 0$ is equivalent to $\mu < \hat{\mu}(h) := \frac{1}{2}(T + \Delta) \in [0,1]$, where $T := \tilde{q}_a(1-x_a) + \tilde{q}_v(1-x_v)$ and $\Delta := \sqrt{T^2 - 4h(1-x_a)(1-x_v)}$. Notice that this is increasing in h. For this reason, $\inf_h \hat{\mu} = \hat{\mu}(0) = 1 - x$. If $h \to 1$ then the steady state converges to $(1 - x_a - \mu, 1 - x_v - \mu)$. So, to ensure the steady state is interior, we assume $\mu < 1 - x_v$. □

A.2. Proofs of Section 3

A.2.1. Proof of Proposition 2 (page 5)

We need first to establish some useful relations.

Lemma A. *In the interior steady state, we have $\rho_a > \tilde{\rho}_a > \tilde{\rho}_v > \rho_v$, $S_a > S_v > 0$, and $x_v - x_a > \rho_a - \rho_v$. Moreover $\tilde{\rho}_a - \tilde{\rho}_v = h(\rho_a - \rho_v)$, and the diagonal elements of the Jacobian, A and D, are negative.*

If $h \to 1$, we have $\rho_a^{SS} \to 1 - x_a - \mu$, $\rho_v^{SS} \to 1 - x_v - \mu$, $\hat{\mu} \to 1 - x_a$, $S_a \to \mu$ and $S_v \to \mu$.

If $h \to 0$, we have $\rho_a^{SS} \to \frac{(1-x_a)(1-x-\mu)}{1-x}$, $\rho_v^{SS} \to \frac{(1-x_v)(1-x-\mu)}{1-x}$, and $\hat{\mu} \to 1 - x$, where $x = x_a q_a + x_v(1 - q_a)$ is the total number of vaccinated. Moreover $S_a \to \frac{1-x_a}{1-x}\mu$, $S_v \to \frac{1-x_v}{1-x}\mu$.

Proof. In the interior steady state susceptibles cannot be zero, otherwise the derivatives would be negative, and the equations could not be satisfied. Moreover:

$$\frac{S_a}{S_v} = \frac{(1-\tilde{q}_v)\frac{\rho_a}{\rho_v} + \tilde{q}_v}{(1-\tilde{q}_a)\frac{\rho_v}{\rho_a} + \tilde{q}_a}$$

If $\rho_a < \rho_v$ and $x_a < x_v$, then $S_a > S_v$, but the fraction above implies $S_v > S_a$, which is a contradiction. Hence $\rho_a > \rho_v$. From the identity $\tilde{\rho}_a - \tilde{\rho}_v = h(\rho_a - \rho_v)$ it follows $\tilde{\rho}_a > \tilde{\rho}_v$, and since they are averages $\rho_a \geq \tilde{\rho}_a \geq \tilde{\rho}_v \geq \rho_v$. Finally:

$$S_a > S_a \frac{\tilde{\rho}_a}{\rho_a} = S_v \frac{\tilde{\rho}_v}{\rho_v} > S_v$$

The limits for $h \to 0$ and $h \to 1$ follow from solving the system (1) in the respective cases, and from continuity of the solution of a differential equation with respect to the parameters.

Finally, if $\mu < \hat{\mu}(h)$, the diagonal elements of J, computed in the interior steady state, are both negative. We know from stability that $A + D = -\lambda_1 - \lambda_2 < 0$, so at least one of them must be negative; moreover since B and C are nonnegative, and the determinant must be positive: $AD - BC > 0 \implies AD > 0$: so both A and D are negative. □

Now we are ready to prove Proposition 2.

Proof. Using the implicit function theorem we can compute the derivatives of infection rates in the steady state:

$$\partial_h F_a = (1-q) S_a \Delta\rho$$
$$\partial_h F_v = -q S_v \Delta\rho$$
$$\partial_h \rho_a = -\frac{S_a(1-q)\Delta\rho}{|J|}(S_v - \tilde{\rho}_v - \mu)$$
$$\partial_h \rho_v = \frac{S_v q \Delta\rho}{|J|}(S_a - \tilde{\rho}_a - \mu)$$

Moreover, $S_v - \tilde{\rho}_v - \mu = S_v - \tilde{\rho}_v - S_v\frac{\tilde{\rho}_v}{\rho_v} < 0$ since $\tilde{\rho}_v > \rho_v$, so it follows that ρ_a is always increasing in h. From the steady state equation $(1 - x_a - \rho_a)\tilde{\rho}_a/\rho_a = \mu$ it follows that also $\tilde{\rho}_a$ is increasing.

Concerning the derivative of ρ_v, if $h \to 0$, then $S_a - \tilde{\rho}_a - \mu = \mu\frac{1-x_a}{1-x} - (1-x)$. This is negative if and only if $\mu < (1-x)^2/(1-x_a)$. Similarly, if $h \to 1$ $S_a - \tilde{\rho}_a - \mu = S_a - \rho_a - S_a\frac{\tilde{\rho}_a}{\rho_a} = S_a - \rho_a - S_a < 0$. Moreover, by the previous conclusions on ρ_a and $\tilde{\rho}_a$, we get that $\partial_h \rho_v$ can only have one zero (because $S_a - \tilde{\rho}_a$ is decreasing): so ρ_v is either decreasing or hump-shaped with one maximum. From the steady state equation $(1 - x_v - \rho_v)\tilde{\rho}_v/\rho_v = \mu$ again it follows that $\tilde{\rho}_v$ is increasing if and only if ρ_v is.

The total infection is:

$$\partial_h \rho = \frac{q(1-q)\Delta\rho}{|J|}(-S_a(S_v - \tilde{\rho}_v - \mu) + S_v(S_a - \tilde{\rho}_a - \mu))$$
$$= \frac{q(1-q)\Delta\rho}{|J|}(S_a(\tilde{\rho}_v + \mu) - S_v(\tilde{\rho}_a + \mu))$$

If $h = 0$ we get: $\partial_h \rho \propto (\Delta x - \Delta\rho)(\mu + \rho) > 0$, while for $h = 1$ we get (since $S_a = S_v = \mu$):

$$\partial_h \rho \propto -\Delta\rho\mu < 0$$

Moreover, if $\mu < (1-x)^2/(1-x_a)$, the derivative is monotonically decreasing, so the total infection is concave.

Moreover, in the limit for $h \to 1$:

$$\partial_h \rho_a \to -\frac{\mu(1-q)\Delta x}{\rho_a \rho_v}(-\rho_v)$$

$$\partial_h \rho_v = \frac{\mu q \Delta x}{\rho_a \rho_v}(-\rho_a)$$

In the limit for $h \to 0$:

$$\partial_h \rho_a = -\frac{S_a(1-q)\Delta\rho}{|J|}(S_v - \rho - \mu)$$
$$\partial_h \rho_v = \frac{S_v q \Delta\rho}{|J|}(S_a - \rho - \mu)$$

The behavior as a function of the vaccination rates is:

$$\partial_{x_a} F_a = -\tilde{\rho}_a \qquad \partial_{x_a} F_v = 0$$
$$\partial_{x_a} \rho_a = -\frac{1}{|J|}(-D\tilde{\rho}_a) < 0 \qquad \partial_{x_a} \rho_v = -\frac{1}{|J|}(C\tilde{\rho}_a) = -q\mu\frac{1-x_a}{1-x} < 0$$

so, all infection rates are decreasing in x_a, and analogously for the derivative with respect to x_v. □

A.2.2. Proof of Proposition 3 (page 7)

We need the following Lemma.

Lemma B. *Call the vector of group-level cumulative infection values $\vec{CI} = (CI_a, CI_v)$. The derivative of the cumulative infection with respect to a parameter y can be expressed as:*

$$\partial_y \vec{CI} = (rI - J)^{-1} \partial_y J (rI - J)^{-1}\mathbf{1}$$

where $\mathbf{1} = (1,1)'$.

Proof. The vector of the cumulative infections $\vec{CI} = (CI_a, CI_v)$ is the solution of:

$$r\vec{CI} = \mathbf{1} + J\vec{CI}.$$

Differentiating it and solving we obtain:

$$\partial_y \vec{CI} = (rI - J)^{-1} \partial_y J (rI - J)^{-1}\mathbf{1}$$

where $\partial_y J$ is the element by element derivative of J. □

Now we can prove the Proposition.

Proof. The total derivative is $d_h CI = \partial_h CI + \partial_{\rho_a} CI \partial_h \rho_a + \partial_{\rho_v} CI \partial_h \rho_v$.

Now, since:

$$\partial_h J = \begin{pmatrix} -(1-q)\Delta\rho & 0 \\ 0 & q\Delta\rho \end{pmatrix} + \begin{pmatrix} S_a & 0 \\ 0 & S_v \end{pmatrix}\begin{pmatrix} 1-q & -(1-q) \\ -q & q \end{pmatrix},$$

using the Lemma we can explicitly calculate the derivatives for $h = 0$ or $h = 1$, and using:

$$(rI - J)^{-1}\,|_{h\to 1} = \begin{pmatrix} \frac{1}{1-x_a-\mu} & 0 \\ 0 & \frac{1}{1-x_a-\mu} \end{pmatrix}$$

$$(rI - J)^{-1}\,|_{h\to 0} = \frac{1}{(r+1-x)(r+1-x-\mu)}\begin{pmatrix} r+1-x-\frac{(1-q)\mu(1-x_v)}{1-x} & \frac{(1-q)\mu(1-x_a)}{1-x} \\ \frac{q\mu(1-x_v)}{1-x} & r+1-x-\frac{q\mu(1-x_a)}{1-x} \end{pmatrix},$$

we obtain the following.

$$\partial_h CI\,|_{h\to 1} = -\frac{(1-q)q(x_a-x_v)^2(2r+2-\mu-x_a-x_v)}{(-r+\mu+x_a-1)^2(-r+\mu+x_v-1)^2} > 0$$

$$\partial_h CI\,|_{h\to 0} = -\frac{\mu(1-q)q(x_a-x_v)^2(1-x-2\mu)}{(1-x)^2(1-x+r)(1-x-\mu+r)^2} > 0$$

$$\iff \mu > \frac{1-x}{2}.$$

Moreover, the derivative of the Jacobian matrix with respect to infection rates is:

$$\partial_{\rho_a} J = \begin{pmatrix} -2\tilde{q}_a & -(1-\tilde{q}_a) \\ 0 & -(1-\tilde{q}_v) \end{pmatrix} \quad \partial_{\rho_v} J = \begin{pmatrix} -(1-\tilde{q}_a) & 0 \\ -(1-\tilde{q}_v) & -2\tilde{q}_v \end{pmatrix}$$

Since $rI - J$ is an *M-matrix* (that is, with non-positive off-diagonal entries and eigenvalues with nonnegative real parts), the inverse has positive elements. So, we conclude that the derivative of *both* cumulative infections with respect to infection rates are negative. In particular, for $h = 0$ or $h = 1$ we can explicitly write the derivatives of the total CI as:

$$\partial_{\rho_a} CI\,|_{h\to 1} = -\frac{2q}{(-1+x_a-r+\mu)^2}$$
$$\partial_{\rho_v} CI\,|_{h\to 1} = -\frac{2(1-q)}{(-1+x_v-r+\mu)^2}$$
$$\partial_{\rho_a} CI\,|_{h\to 0} = -\frac{2q}{(r+1-x-\mu)^2}$$
$$\partial_{\rho_v} CI\,|_{h\to 0} = -\frac{2(1-q)}{(r+1-x-\mu)^2}$$

For $h \to 1$, since $\frac{\partial \rho_v}{\partial h}\,|_{h\to 1} < 0$, we know that the term:

$$\frac{\partial CI}{\partial \rho_v}\,|_{h\to 1}\,\frac{\partial \rho_v}{\partial h}\,|_{h\to 1} = -\frac{2(1-q)}{(1-x_v-\mu+r)^2}\,\frac{\partial \rho_v}{\partial h}\,|_{h\to 1} > 0$$

is positive. So, it must be larger than $-\frac{2(1-q)}{(1-x_a-\mu+r)^2}\,\frac{\partial \rho_v}{\partial h}\,|_{h\to 1}$, because the denominator is smaller. So it is true that, for $h \to 1$:

$$\begin{aligned}\frac{\partial CI}{\partial \rho_a}\frac{\partial \rho_a}{\partial h} + \frac{\partial CI}{\partial \rho_v}\frac{\partial \rho_v}{\partial h} &= -\frac{2q}{(1-x_a-\mu+r)^2}\frac{\partial \rho_a}{\partial h} - \frac{2(1-q)}{(1-x_v-\mu+r)^2}\frac{\partial \rho_v}{\partial h} \\ &> -\frac{2}{(1-x_a-\mu+r)}\left(q\frac{\partial \rho_a}{\partial h} + (1-q)\frac{\partial \rho_v}{\partial h}\right) \\ &= -\frac{2}{(1-x_a-\mu+r)^2}\frac{\partial \rho}{\partial h} > 0\end{aligned}$$

So, for h high enough, both the direct and indirect effects are positive.

For $h \to 0$, $(\partial_{\rho_a} CI, \partial_{\rho_v} CI) \propto -(q, 1-q)$, and so:

$$\left(\partial_{\rho_a} CI\,\partial_h \rho_a + \partial_{\rho_v} CI\,\partial_h \rho_a\right)|_{h\to 0} \propto -\partial_h \rho\,|_{h\to 0} < 0,$$

so the indirect effect is negative. The direct effect, instead, depends on μ: for $\mu < \frac{1-x}{2}$ they have the same sign, and the derivative is negative. Using the results above we can compute the total effect, that is:

$$\begin{aligned}&\partial_h CI^{\text{out}}\,|_{h=0} + \partial_{\rho_a} CI\,\partial_h \rho_a\,|_{h=0} + \partial_{\rho_v} CI\,\partial_h \rho_a\,|_{h=0} \\ &\quad = \frac{\mu(1-q)q\Delta x^2(-2r+2\mu-3(1-x))}{(1-x)^2(1-x+r)(-r+\mu-(1-x))^2}\end{aligned}$$

that has the same sign as $-2r + 2\mu - 3(1-x) < -2r - (1-x) < 0$, since $\mu < 1 - x$. So the indirect effect dominates. □

A.2.3. *Proof of Proposition 4 (page 8)*

Proof. Remember from Proposition 1 that we define the eigenvalues of J to be $-\lambda_1$ and $-\lambda_2$, and $\lambda_1 > \lambda_2$. Using the results in Bernstein and So (1993) (Theorem 2.2), we can express the exponential matrix as a function of eigenvalues, and directly compute the limit:

$$\begin{aligned}CR &= -\lim_{t\to\infty}\frac{\log\|e^{tJ}\mathrm{d}\rho_0\|}{t} = -\lim_{t\to\infty}\frac{\log\left\|\left(\frac{\lambda_1 e^{-\lambda_2 t} - \lambda_2 e^{-\lambda_1 t}}{\lambda_1-\lambda_2} I + \frac{e^{-\lambda_2 t} - e^{-\lambda_1 t}}{\lambda_1-\lambda_2} J\right)\mathrm{d}\rho_0\right\|}{t} \\ &= -\log e^{-\lambda_2 t}\frac{1}{t} - \lim_{t\to\infty}\frac{\log\left\|\left(\frac{\lambda_1 - \lambda_2 e^{-(\lambda_1-\lambda_2)t}}{\lambda_1-\lambda_2} I + \frac{1-e^{-(\lambda_1-\lambda_2)t}}{\lambda_1-\lambda_2} J\right)\mathrm{d}\rho_0\right\|}{t} \\ &= \lambda_2 - \lim_{t\to\infty}\frac{\log\|(\lambda_1 - \lambda_2 e^{-(\lambda_1-\lambda_2)t})\mathrm{d}\rho_0 + (1-e^{-(\lambda_1-\lambda_2)t})J\mathrm{d}\rho_0\|}{t}\end{aligned}$$

$$= \lambda_2 - \frac{\log\|(\lambda_1 I + J)\mathrm{d}\rho_0\|}{\lim_{t\to\infty} t} = \lambda_2$$

where in the last line we used that $e^{-(\lambda_1-\lambda_2)t} \to 0$ because $\lambda_1 > \lambda_2$.

Concerning the behavior of λ_2 as a function of h, we use a standard result on eigenvalue perturbations (Demmel, 1997, Th 4.4). Since the eigenvalues are simple, the result states that for a simple eigenvalue λ of J:

$$\partial_h \lambda = \frac{v' \partial_h J u}{v' u}$$

where v is the left and u the right eigenvector of J relative to λ, and $u'v \neq 0$. For $\lambda = -\lambda_2$, we can explicitly solve for the eigenvectors:

$$u = \left(\frac{2B}{\sqrt{(A-D)^2 + 4BC} - (A-D)}, 1 \right)$$
$$v = \left(\frac{2C}{\sqrt{(A-D)^2 + 4BC} - (A-D)}, 1 \right)$$

and they have both a nonnegative and a positive component, so $v'u > 0$: hence the sign of the derivative is determined by the numerator.

The derivative $\partial_h J$ was computed in the proof of the previous Proposition. When $h \to 1$, both eigenvectors converge to $u = v = (0,1)'$. So in the limit of $h \to 1$ we get:

$$\partial_h \lambda_2 \,|_{h\to 1} = -\partial_h \lambda \,|_{h\to 1} = -\partial D = -q(\Delta\rho + S_v)\,|_{h\to 1} = -q(\Delta x + \mu) < 0$$

so that for high enough h, the rate of convergence is decreasing in h. Instead, when $h \to 0$, the right and left eigenvectors converge respectively to $(1 - x_a, 1 - x_v)$ and $(q, 1-q)$, and the derivative becomes:

$$\partial_h \lambda_2 \,|_{h\to 0} = \frac{(1-q)q(1 - x - 2\mu)\Delta x^2}{(1-x)^2}$$

that is positive if and only if $\mu < \frac{1-x}{2}$. So, for h small, the convergence rate is decreasing in h only if μ is large enough.

By results of the previous proposition, $\partial_{\rho_a} J$ and $\partial_{\rho_v} J$ have both negative elements, hence the derivatives $\partial_{\rho_a} \lambda_2$ and $\partial_{\rho_a} \lambda_2$ are positive, which is what we wanted to show. □

A.3. Proofs of Section 4

For the proofs of this section, we are going to define $\theta_a = \dfrac{\rho_a}{1 - x_a}$ and $\theta_v = \dfrac{\rho_v}{1 - x_v}$. We can rewrite the system (1) with this parameterization as:

$$\begin{aligned} \dot\theta_a &= (1 - \theta_a)\tilde\rho_a - \mu\theta_a \\ \dot\theta_v &= (1 - \theta_v)\tilde\rho_v - \mu\theta_v \end{aligned} \tag{a}$$

Moreover, we are going to need the following Lemma.

Lemma C. *System (a) has the same stability properties of System (1).*
The derivatives $\partial_{x_a}\theta_a$, $\partial_{x_v}\theta_a$, $\partial_{x_v}\theta_v$, $\partial_{x_v}\theta_a$ are all negative.

Proof. The two systems are related by a change of coordinates: $(\rho_{a,t}, \rho_{v,t}) = \Theta(\theta_{a,t}, \theta_{v,t})$, where the function Θ maps $(\theta_{a,t}, \theta_{v,t})$ into $((1 - x_a)\theta_{a,t}, (1 - x_v)\theta_{v,t})$. Since it is differentiable and has differentiable inverse for $x_a, x_v \in (0,1)$, the two systems are *smoothly equivalent*, and so they have the same steady states, and all the steady states have the same eigenvalues (Meiss (2007), Ch. 4). It follows that the interior steady state is asymptotically stable under the same assumptions.

Now we compute the Jacobians.

$$J_\theta F = \begin{pmatrix} \tilde q_a(1-2\theta_a)(1-x_a) - (1-\tilde q_a)\theta_v(1-x_v) - \mu, & (1-\tilde q_a)(1-\theta_a)(1-x_v) \\ (1-\tilde q_v)(1-\theta_v)(1-x_a), & \tilde q_v(1-2\theta_v)(1-x_v) - (1-\tilde q_v)\theta_a(1-x_a) - \mu \end{pmatrix}$$

Since the two systems are smoothly equivalent, in the interior steady state this must have negative eigenvalues, and so $J_\theta F$ is invertible, has positive determinant, and negative trace. Moreover, as in the proof of Lemma A, since the off-diagonal elements are positive, the diagonal elements must be both negative. So, it follows that $J_\theta F$ is the negative of an *M-matrix*, and so the inverse $J_\theta F^{-1}$ has all negative entries. The derivatives with respect to vaccination rates are collected in the matrix:

$$\partial_x \theta = -J_\theta F^{-1} J_x F$$

where:

$$J_x F = \begin{pmatrix} -(1-\theta_a)\theta_a\tilde{q}_a & -(1-\theta_a)\theta_v(1-\tilde{q}_a) \\ -(1-\theta_v)\theta_a(1-\tilde{q}_v) & -(1-\theta_v)\theta_v\tilde{q}_v \end{pmatrix}$$

Since all the elements of the matrices $J_\theta F^{-1}$ and $J_x F$ are negative, it follows that $\partial_x\theta = -J_\theta F^{-1} J_x F$ has all negative elements, which is what we wanted to show. □

A.3.1. Proof of Lemma 1 (page 10)

Proof. Define the vector function Φ as:

$$\Phi_a(x_a, x_v) = x_a - k\theta_a(x_a, x_v) + d$$

$$\Phi_v(x_a, x_v) = x_v - k\theta_v(x_a, x_v)$$

A vaccination equilibrium is a configuration (x_a^*, x_v^*) such that $\Phi_a(x_a^*, x_v^*) = 0$ and $\Phi_v(x_a^*, x_v^*) = 0$.

To prove existence of a solution, we use the Poincaré-Miranda Theorem (Kulpa, 1997). The theorem guarantees that if the following conditions are satisfied:

$$\Phi_a(0, x_v) = -k\theta_a + d \le 0 \text{ for all } x_v \in [0,1]$$

$$\Phi_a(1, x_v) = 1 - k\theta_a + d \ge 0 \text{ for all } x_v \in [0,1]$$

$$\Phi_v(x_a, 0) = -k\theta_v \le 0 \text{ for all } x_a \in [0,1]$$

$$\Phi_v(x_a, 1) = 1 - k\theta_v \ge 0 \text{ for all } x_a \in [0,1]$$

then there is point (x_a^*, x_v^*) such that $\Phi_a = 0$ and $\Phi_v = 0$, that is a vaccination equilibrium.

The first needs just to be checked at the minimum of θ_a, that is realized for $x_v = 1$. In that case, we can solve explicitly for θ_a, and we get: $\theta_a = 1 - \dfrac{\mu}{\tilde{q}_a}$, where in this case the upper bound on μ is $\hat{\mu} = \tilde{q}_a$, so $1 - \dfrac{\mu}{\tilde{q}_a} > 0$, and so this solution is always feasible. The first condition is thus satisfied if: $d \le \overline{d} := k\left(1 - \dfrac{\mu}{\tilde{q}_a}\right)$. The third equation is always satisfied. The second and fourth need to be checked at the maxima of θ_a and θ_v, that are realized, respectively, for $x_v = 0$ and $x_a = 0$. We can explicitly compute:

$$\lim_{x_a \to 1, x_v \to 0} \theta_a = \frac{(1-\tilde{q}_a)(\tilde{q}_v - \mu)}{(1-\tilde{q}_a)\tilde{q}_v + h\mu}$$

$$\lim_{x_a \to 0, x_v \to 1} \theta_v = \frac{(1-\tilde{q}_v)(\tilde{q}_a - \mu)}{(1-\tilde{q}_v)\tilde{q}_a + h\mu}$$

So, the two bounds become:

$$(1+d)\frac{(1-\tilde{q}_a)\tilde{q}_v + h\mu}{(1-\tilde{q}_a)(\tilde{q}_v - \mu)} > k$$

$$\frac{(1-\tilde{q}_v)\tilde{q}_a + h\mu}{(1-\tilde{q}_v)(\tilde{q}_a - \mu)} > k,$$

where, again, the left-hand sides are positive because in the respective cases the upper bound $\hat{\mu} = \tilde{q}_a$ and $\hat{\mu} = \tilde{q}_v$. If k is low enough, these conditions are always satisfied. The above conditions and $d \le k\left(1 - \dfrac{\mu}{\tilde{q}_a}\right)$ are compatible only if:

$$k\left(1 - \frac{\mu}{\tilde{q}_a}\right) > k\frac{(1-h)(1-q)(\tilde{q}_v - \mu)}{(1-h)(1-q)\tilde{q}_v + h\mu} - 1$$

that, again, is satisfied provided k is low enough. So, for each $h \in (0,1)$, there exist a value $\overline{k}$ such that for $d \le \overline{d}$ and $k \le \overline{k}$ there exist a vaccination equilibrium.

Now we prove uniqueness. The Jacobian of the system is:

$$J_x\Phi = \begin{pmatrix} 1 - k\partial_{x_a}\theta_a & -k\partial_{x_v}\theta_a \\ -k\partial_{x_a}\theta_v & 1 - k\partial_{x_v}\theta_v \end{pmatrix}$$

Given the signs computed in Lemma C, this has positive diagonal and negative off-diagonal elements.

We have to study the sign of the determinant:

$$det J_x\Phi = 1 - k\partial_{x_a}\theta_a - k\partial_{x_v}\theta_v + k^2\left(\partial_{x_a}\theta_a\partial_{x_v}\theta_v - \partial_{x_v}\theta_a\partial_{x_a}\theta_v\right)$$

Because of Lemma C we have $1 - k\partial_{x_a}\theta_a - k\partial_{x_v}\theta_v > 0$. Now the term

$$\partial_{x_a}\theta_a\partial_{x_v}\theta_v - \partial_{x_v}\theta_a\partial_{x_a}\theta_v$$

is the determinant of the matrix $\partial_x\theta$, that by the implicit function theorem is equal to:

$$det\begin{pmatrix}\partial_{x_a}\theta_a & \partial_{x_v}\theta_a\\ \partial_{x_a}\theta_v & \partial_{x_v}\theta_v\end{pmatrix} = -J_\theta F^{-1}J_x F$$

So:

$$\partial_{x_a}\theta_a\partial_{x_v}\theta_v - \partial_{x_v}\theta_a\partial_{x_a}\theta_v = detJ_\theta F^{-1}det(-J_x F)$$

The first determinant is positive by stability of the system; the second is:

$$\begin{aligned}det(-J_x F) &= detJ_x F = \theta_a\theta_v(1-\theta_a)(1-\theta_v)(\tilde{q}_a\tilde{q}_v - (1-\tilde{q}_a)(1-\tilde{q}_v))\\ &= \theta_a\theta_v(1-\theta_a)(1-\theta_v)h \geq 0\end{aligned}$$

So, the determinant of $J_x\Phi$ is positive, and in particular the matrix is invertible, so there is locally a solution of the system. Moreover the determinant is positive, hence the matrix is positive definite, so the solution is unique and global thanks to the global implicit function theorem of Gale and Nikaido (1965).

Finally, if $x_a^* > x_v^*$ it follows from Lemma A that $\rho_a < \rho_v$, and $\tilde{\rho}_a < \tilde{\rho}_v$. From (a) it follows that:

$$\frac{\theta_a}{1-\theta_a} = \tilde{\rho}_a < \tilde{\rho}_v = \frac{\theta_v}{1-\theta_v}$$

and so $\theta_a < \theta_v$, that implies $x_a^* < x_v^*$, which is a contradiction. If $x_a^* = x_v^*$, then $\rho_a = \rho_v$, so $\theta_a = \theta_v$, which implies $x_a^* < x_v^*$, which is again a contradiction. So, we conclude that $x_a^* < x_v^*$. □

A.3.2. Proof of Proposition 5

Proof. We use the implicit function theorem. The derivatives of Φ with respect to homophily are:

$$J_h\Phi = \begin{pmatrix}-k\partial_h\theta_a\\ -k\partial_h\theta_v\end{pmatrix}$$

So, the derivatives of the vaccination rates are:

$$\partial_h x = -J_x\Phi^{-1}J_h\Phi = \frac{1}{detJ_x\Phi}\begin{pmatrix}1-k\partial_{x_v}\theta_v & k\partial_{x_v}\theta_a\\ k\partial_{x_a}\theta_v & 1-k\partial_{x_a}\theta_a\end{pmatrix}\begin{pmatrix}k\partial_h\theta_a\\ k\partial_h\theta_v\end{pmatrix}$$

Now, $detJ_x\Phi > 0$. Moreover, $\partial_h\theta_a = \frac{1}{1-x_a}\partial_h\rho_a > 0$ and $\partial_h\theta_v = \frac{1}{1-x_v}\partial_h\rho_v < 0$ if $\mu < (1-x)^2/(1-x_a)$. So, it follows that if $\mu < (1-x)^2/(1-x_a)$, we have $\partial_h x_a > 0$ and $\partial_h x_v < 0$. □

A.3.3. Proof of Proposition 6 (page 11)

Proof. Part 1: steady state

As clarified in the proof of Lemma 1, to have an interior solution the conditions $d \leq \overline{d}$ and $k \leq \overline{k}$ must be satisfied. Moreover, $\overline{d}$ tends to zero if k goes to zero. So, in the following, when we take the limits for k and d to zero, we always assume the conditions for an interior solutions are satisfied.

We want to compute:

$$d_h\rho = \partial_h\rho + \partial_h x_a\partial_{x_a}\rho + \partial_h x_v\partial_{x_v}\rho$$

Consider first the limit $h \to 1$. First, we calculate the limits of $\partial_{x_a}\rho, \partial_{x_v}\rho_a$ for $h \to 1$, using the expressions for the derivatives obtained in the proof of Proposition 2. In this limit, the off-diagonal elements B and C go to zero, while $|J| \to AD = \rho_a\rho_v$. Hence in this limit $(\partial_{x_a}\rho_a, \partial_{x_a}\rho_v) \to (-1, 0)$, and, analogously, $(\partial_{x_a}\rho_a, \partial_{x_a}\rho_v) \to (0, -1)$. So, we have: $\partial_{x_a}\rho = q\partial_{x_a}\rho_a + (1-q)\partial_{x_a}\rho_v$ tends to $-q$, and analogously $\partial_{x_v}\rho \to -(1-q)$. So:

$$d_h\rho = \partial_h\rho - (q\partial_h x_a + (1-q)\partial_h x_v)$$

Using Proposition 2, the direct effect is:

$$\partial_h\rho = -\frac{\mu(1-q)q(x_a - x_v)^2}{(1-\mu-x_a)(1-\mu-x_v)},$$

To compute the derivatives of the vaccination rates we use the formula in Proposition 5, and use:

$$\partial_{x_a}\theta_a = \partial_{x_a}\rho_a \frac{1}{1-x_a} + \frac{\rho_a}{(1-x_a)^2} < 0$$
$$\partial_{x_a}\theta_v = \partial_{x_a}\rho_v \frac{1}{1-x_v}$$
$$\partial_{x_v}\theta_a = \partial_{x_v}\rho_a \frac{1}{1-x_a}$$
$$\partial_{x_v}\theta_v = \partial_{x_a}\rho_v \frac{1}{1-x_v} + \frac{\rho_a}{(1-x_v)^2} < 0$$

So, for $h \to 1$ they are:

$$\partial_h x = \frac{1}{det J_x \Phi}\begin{pmatrix} 1+k\frac{\mu}{(1-x_a)^2} & 0 \\ 0 & 1+k\frac{\mu}{(1-x_v)^2}\end{pmatrix}\begin{pmatrix} k\partial_h\rho_a \frac{1}{1-x_a} \\ k\partial_h\rho_v \frac{1}{1-x_v}\end{pmatrix}$$
$$= \begin{pmatrix} k\left(1+k\frac{\mu}{(1-x_v)^2}\right)^{-1}\frac{1}{1-x_a}\partial_h\rho_a \\ k\left(1+k\frac{\mu}{(1-x_a)^2}\right)^{-1}\frac{1}{1-x_v}\partial_h\rho_v\end{pmatrix}$$

where $J_x\Phi = \left(1+k\frac{\mu}{(1-x_a)^2}\right)\left(1+k\frac{\mu}{(1-x_v)^2}\right)$. Computing the indirect effect we get:

$$-(q\partial_h x_a + (1-q)\partial_h x_v) = k\mu(1-q)q\Delta x\left(-\frac{1-x_a}{(1-\mu-x_a)((1-x_a)^2+k\mu)} + \frac{1-x_v}{(1-\mu-x_v)((1-x_v)^2+k\mu)}\right)$$

So, the total effect is:

$$d_h\rho = \mu q(1-q)\Delta x\left(-\frac{\Delta x}{(1-\mu-x_a)(1-\mu-x_v)} + k\left(-\frac{1-x_a}{(1-\mu-x_a)((1-x_a)^2+k\mu)} + \frac{1-x_v}{(1-\mu-x_v)((1-x_v)^2+k\mu)}\right)\right)$$
$$= -\mu q(1-q)\Delta x\left(\frac{1}{1-x_v-\mu}\left(1-\frac{k(1-x_v)}{(1-x_v)^2+k\mu}\right) - \frac{1}{1-x_a-\mu}\left(1-\frac{k(1-x_a)}{(1-x_a)^2+k\mu}\right)\right)$$

For $k \to 0$, the term in parentheses converges to: $\frac{1}{1-x_v-\mu} - \frac{1}{1-x_a-\mu} > 0$: so, we conclude that $d_h\rho\,|_{h\to 1} < 0$, as we wanted to show.

Now focus on the limit $h \to 0$. In the limit for $h \to 0$ $|J| \to (1-x)\rho$, and $\tilde{\rho}_a \to \rho$. So, using the calculations in Proposition 2:

$$\partial_{x_a}\rho_a \to \frac{D}{1-x} = \frac{-\mu-\rho+(1-q)S_v}{1-x} = -1+(1-q)\mu\frac{1-x_v}{(1-x)^2}$$
$$\partial_{x_a}\rho_v \to -\frac{C}{1-x} = \frac{-qS_v}{1-x} = -q\mu\frac{1-x_v}{(1-x)^2}$$
$$\partial_{x_v}\rho_a \to -\frac{B}{1-x} = \frac{-(1-q)S_a}{1-x} = -(1-q)\mu\frac{1-x_a}{(1-x)^2}$$
$$\partial_{x_v}\rho_v \to \frac{A}{1-x} = \frac{-\mu-\rho+qS_a}{1-x} = -1+q\mu\frac{1-x_a}{(1-x)^2},$$

that implies:

$$\partial_{x_a}\rho = q\partial_{x_a}\rho_a + (1-q)\partial_{x_a}\rho_v \to -q$$
$$\partial_{x_v}\rho = q\partial_{x_v}\rho_a + (1-q)\partial_{x_v}\rho_v \to -(1-q)$$

so, also in this case we have

$$d_h\rho = \partial_h\rho - (q\partial_h x_a + (1-q)\partial_h x_v).$$

Plugging these in the expressions for the derivatives of vaccination rates we get:

$$\partial_h x = \frac{1}{1+\frac{k\mu}{(1-x)^2}}\begin{pmatrix} 1+k\frac{(1-q)\mu}{(1-x)^2} & -k\frac{(1-q)\mu}{(1-x)^2} \\ -k\frac{q\mu}{(1-x)^2} & 1+k\frac{q\mu}{(1-x)^2}\end{pmatrix}\begin{pmatrix} k\partial_h\rho_a\frac{1}{1-x_a} \\ k\partial_h\rho_v\frac{1}{1-x_v}\end{pmatrix}$$

$$= \frac{1}{1+\frac{k\mu}{(1-x)^2}} \begin{pmatrix} k\partial_h\rho_a \frac{1}{1-x_a} + k^2 \frac{(1-q)\mu}{(1-x)^2}\left(\partial_h\rho_a \frac{1}{1-x_a} - \partial_h\rho_v \frac{1}{1-x_v}\right) \\ k\partial_h\rho_v \frac{1}{1-x_v} + k^2 \frac{q\mu}{(1-x)^2}\left(\partial_h\rho_v \frac{1}{1-x_v} - \partial_h\rho_a \frac{1}{1-x_a}\right) \end{pmatrix}$$

When aggregating, we get a simplification:

$$q\partial_h x_a + (1-q)\partial_h x_v = \frac{k}{1+\frac{k\mu}{(1-x)^2}}\left(q\partial_h\rho_a \frac{1}{1-x_a} + (1-q)\partial_h\rho_v \frac{1}{1-x_v}\right)$$

We can compute this analytically, because, using Proposition 2:

$$q\partial_h\rho_a \frac{1}{1-x_a} + (1-q)\partial_h\rho_v \frac{1}{1-x_v} = \frac{\mu^2(1-q)q(x_v-x_a)^2}{(1-x)^4}$$

So, the total derivative is:

$$\begin{aligned} d_h\rho =& \partial_h\rho - (q\partial_h x_a + (1-q)\partial_h x_v) \\ =& \frac{\mu(1-q)q(x_v-x_a)^2}{(1-x)^2}\left(1 - \frac{k\mu}{(1-x)^2+k\mu}\right) > 0, \end{aligned}$$

which is what we wanted to show.

Part 2: cumulative infection

We want to compute:

$$d_h CI = \partial_h CI + \partial_h\rho_a\partial_{\rho_a} CI + \partial_h\rho_v\partial_{\rho_v} CI + d_{x_a} CI\,\partial_h x_a + d_{x_v} CI\,\partial_h x_v$$

where $d_{x_a} CI\,\partial_h x_a + d_{x_v} CI\,\partial_h x_v$ is the indirect effect through endogenous vaccination rates and

$$d_{x_a} CI = \partial_{x_a} CI + \partial_{x_a}\rho_a\partial_{\rho_a} CI + \partial_{x_a}\rho_v\partial_{\rho_v} CI$$
$$d_{x_v} CI = \partial_{x_v} CI + \partial_{x_v}\rho_a\partial_{\rho_a} CI + \partial_{x_v}\rho_v\partial_{\rho_v} CI$$

Focus on the limit for $h \to 1$.

In this limit, from the previous part, we have $\partial_{x_a}\rho_a = -1$, $\partial_{x_v}\rho_a = 0$, and analogously for ρ_v. Moreover, using Lemma B and

$$\partial_{x_a} J\,|_{h\to 1} = \begin{pmatrix} -1 & 0 \\ 0 & 0 \end{pmatrix}$$
$$\partial_{x_v} J\,|_{h\to 1} = \begin{pmatrix} 0 & 0 \\ 0 & -1 \end{pmatrix}$$

we can easily compute the derivatives with respect to the vaccination rates:

$$\partial_{x_a} CI = -\frac{q}{(1-x_a-\mu+r)^2}$$
$$\partial_{x_v} CI = -\frac{1-q}{(1-x_v-\mu+r)^2}$$

So, using the calculations of Proposition 3:

$$d_{x_a} CI = \partial_{x_a} CI + \partial_{x_a}\rho_a\partial_{\rho_a} CI + \partial_{x_a}\rho_v\partial_{\rho_v} CI = \partial_{x_a} CI - \partial_{\rho_a} CI = \frac{q}{(1-x_a-\mu+r)^2}$$
$$d_{x_v} CI = \partial_{x_v} CI + \partial_{x_v}\rho_a\partial_{\rho_a} CI + \partial_{x_v}\rho_v\partial_{\rho_v} CI = \partial_{x_v} CI - \partial_{\rho_v} CI = \frac{1-q}{(1-x_v-\mu+r)^2}$$

So, the indirect effect is:

$$\begin{aligned} & d_{x_a} CI\,\partial_h x_a + d_{x_v} CI\,\partial_h x_v \\ &= \left(\partial_{x_a} CI - \partial_{\rho_a} CI\right)\partial_h x_a + \left(\partial_{x_v} CI - \partial_{\rho_v} CI\right)\partial_h x_v \\ &= \frac{q}{(1-x_a-\mu+r)^2}\partial_h x_a + \frac{1-q}{(1-x_v-\mu+r)^2}\partial_h x_v \\ &= k\mu(1-q)q\Delta x\left(\frac{1-x_v}{(1-\mu-x_v)\left(k\mu+(1-x_v)^2\right)(\mu-r+x_v-1)^2} - \frac{1-x_a}{(1-x_a-\mu)\left((1-x_a)^2+k\mu\right)(x_a+\mu-r-1)^2}\right) \end{aligned}$$

Using the results of the proof of Proposition 3, we obtain that the total effect is:

$$\begin{aligned}
d_h CI =&(1-q)q\Delta x\left(\frac{\Delta x\left(x_a+\mu-2r+x_v-2\right)}{\left(x_a+\mu-r-1\right)^2\left(\mu-r+x_v-1\right)^2}-\frac{2\mu}{\left(1-x_a-\mu\right)\left(x_a+\mu-r-1\right)^2}+\frac{2\mu}{\left(1-\mu-x_v\right)\left(\mu-r+x_v-1\right)^2}\right)\\
&+k\mu(1-q)q\Delta x\left(\frac{1-x_v}{\left(1-\mu-x_v\right)\left(k\mu+\left(1-x_v\right)^2\right)\left(\mu-r+x_v-1\right)^2}-\frac{1-x_a}{\left(1-x_a-\mu\right)\left(\left(1-x_a\right)^2+k\mu\right)\left(x_a+\mu-r-1\right)^2}\right)\\
&=(1-q)q\Delta x\left[-\frac{\mu}{\left(1-x_a-\mu\right)\left(x_a+\mu-r-1\right)^2}\left(\frac{k\left(1-x_a\right)}{\left(x_a-1\right)^2+k\mu}+2\right)\right.\\
&+\frac{\mu}{\left(1-x_v-\mu\right)\left(x_v+\mu-r-1\right)^2}\left(\frac{k\left(1-x_v\right)}{\left(1-x_v\right)^2+k\mu}+2\right)\\
&\left.-\frac{\left(x_v-x_a\right)\left(x_a+\mu-2r+x_v-2\right)}{\left(x_a+\mu-r-1\right)^2\left(\mu-r+x_v-1\right)^2}\right]
\end{aligned}$$

For $k\to 0$ and $d\to 0$, we have $\Delta x\to 0$, so the term in square brackets is asymptotically equivalent to:

$$-\frac{2\mu}{\left(1-x_a-\mu\right)\left(x_a+\mu-r-1\right)^2}+\frac{2\mu}{\left(1-x_v-\mu\right)\left(x_v+\mu-r-1\right)^2}>0$$

and so we conclude $d_h CI>0$ for sufficiently small k and d.

Now focus on $h\to 0$. Again, using Lemma B and

$$\partial_{x_a} J\,|_{h\to 0}=-\begin{pmatrix} q & 1-q\\ 0 & 0\end{pmatrix}$$

$$\partial_{x_v} J\,|_{h\to 0}=-\begin{pmatrix} 0 & 0\\ q & 1-q\end{pmatrix}$$

we can compute the derivatives of CI with respect to vaccination rates:

$$\partial_{x_a} CI=-\frac{q}{(1-x-\mu+r)^2}$$

$$\partial_{x_v} CI=-\frac{1-q}{(1-x-\mu+r)^2}$$

Proceeding as before, using the calculations of Proposition 3 we get:

$$d_{x_a} CI=\partial_{x_a} CI-\partial_{\rho_a} CI=\frac{q}{(-r+\mu+x-1)^2}$$

$$d_{x_v} CI=\partial_{x_v} CI-\partial_{\rho_v} CI=\frac{1-q}{(-r+\mu+x-1)^2}$$

So, there is a simplification:

$$\begin{aligned}
&d_{x_a} CI\partial_h x_a+d_{x_v} CI\partial_h x_v\\
&=\left(\partial_{x_a} CI-\partial_{\rho_a} CI\right)\partial_h x_a+\left(\partial_{x_v} CI-\partial_{\rho_v} CI\right)\partial_h x_v\\
&=\frac{q}{(-r+\mu+x-1)}\partial_h x_a+\frac{1-q}{(-r+\mu+x-1)^2}\partial_h x_v\\
&=\frac{1}{(-r+\mu+x-1)^2}\left(q\partial_h x_a+(1-q)\partial_h x_v\right)
\end{aligned}$$

Now using the analytical results obtained for the steady state case, we get that the indirect effect is:

$$d_{x_a} CI\partial_h x_a+d_{x_v} CI\partial_h x_v=\frac{1}{(-r+\mu+x-1)^2}\frac{k}{1+\frac{k\mu}{(1-x)^2}}\frac{\mu^2(1-q)q(x_v-x_a)^2}{(1-x)^4}$$

Adding the direct effect computed in Proposition 3, we get:

$$d_h CI\,|_{h\to 0}=-\mu q(1-q)\Delta x^2\left(\frac{(1-x)^2(3(1-x)+2r-2\mu)}{1-x+r}-\frac{k\mu}{\frac{k\mu}{(1-x)^2}+1}\right)$$

that is negative if and only if:

$$\frac{(1-x)^2(3(1-x)+2r-2\mu)}{1-x+r} - \frac{k\mu}{\frac{k\mu}{(1-x)^2}+1} > 0$$

and the second term goes to zero in the limit for d and k small, so the derivative is indeed negative. □

A.3.4. Proof of Proposition 7 (page 12)

Proof. Part 1

The equilibria (including corner ones) are the solutions of the following equations:

$$\begin{aligned} x_a &= \min\{1, \max\{0, k(\tilde{q}_a x_a + (1-\tilde{q}_a)x_v) - d\}\}, \\ x_v &= \min\{1, \max\{k(\tilde{q}_v x_v + (1-\tilde{q}_v)x_a)\}\}. \end{aligned} \tag{b}$$

First we summarize the complete characterization of the equilibria, then we prove it.

Equilibria characterization: For all values of the parameters, there is an equilibrium with $x_a^* = x_v^* = 0$. For $k \le 1$, this is the unique equilibrium. For $h < 1 - \frac{k-1}{kq}$ and $d < \frac{(k-1)(1-hk)}{kq(1-h)}$, there is also an equilibrium where both are interior: $x_a^*, x_v^* \in (0,1)$. If $h \ge 1 - \frac{k-1}{kq}$ and, either $d > k(1-\tilde{q}_a)$, or $k\tilde{q}_a > 1$, then there is an equilibrium with $x_a^* = 0$ and $x_v^* = 1$. If $k \ge d+1$, there is an equilibrium with $x_a^* = x_v^* = 1$. If $1 + d > k \ge 1/\tilde{q}_v$, $k(1-\tilde{q}_a) > d$ and $k\tilde{q}_a < 1$, there is an equilibrium where $x_v^* = 1$ and x_a^* is interior.

If $k < 1$, supposing that the system (11) has interior solution, the linear system can be written as:

$$\left(I - k\begin{pmatrix} \tilde{q}_a & 1-\tilde{q}_a \\ 1-\tilde{q}_v & \tilde{q}_v \end{pmatrix}\right)\begin{pmatrix} x_a \\ x_v \end{pmatrix} = \begin{pmatrix} -d \\ 0 \end{pmatrix},$$

where I is the 2×2 identity matrix. Since the matrix $\begin{pmatrix} \tilde{q}_a & 1-\tilde{q}_a \\ 1-\tilde{q}_v & \tilde{q}_v \end{pmatrix}$ is stochastic, it follows that the maximum eigenvalue is 1. So, using standard results, if $k < 1$ the matrix $\left(I - k\begin{pmatrix} \tilde{q}_a & 1-\tilde{q}_a \\ 1-\tilde{q}_v & \tilde{q}_v \end{pmatrix}\right)$ is invertible and has positive inverse. So, it follows that the unique interior equilibrium would satisfy:

$$\begin{pmatrix} x_a^* \\ x_v^* \end{pmatrix} = \left(I - k\begin{pmatrix} \tilde{q}_a & 1-\tilde{q}_a \\ 1-\tilde{q}_v & \tilde{q}_v \end{pmatrix}\right)^{-1}\begin{pmatrix} -d \\ 0 \end{pmatrix},$$

but, since the inverse is positive, it follows that $x_a^*, x_v^* < 0$, which is not feasible.

If $k = 1$, from the equations we obtain $x_v - x_a = 0$ and $x_a - x_v = -d/(1-\tilde{q}_a)$, that is not feasible. So, for $k \le 1$, there is no interior solution. Moreover, it is also not possible to have a partially interior solution with $x_v^* > 0$ but $x_a^* = 0$. Indeed, if $x_a^* = 0$, then x_v^* satisfies the equation:

$$x_v = k\tilde{q}_v x_v$$

and since $k < 1$ then $k\tilde{q}_v < 1$, so the only solution is $x_v^* = 0$. A similar reasoning works to show that there is no solution with $x_a^* > 0$ and $x_v^* = 0$.

So, we focus on the case of $k > 1$. With a calculation, we find that the interior solution is:

$$x_a^* = \frac{d(1-k(1-(1-h)q))}{(k-1)(1-hk)}, \quad x_v^* = \frac{d(1-h)kq}{(k-1)(1-hk)},$$

This is feasible if $x_a^* > 0$, and if $x_v^* < 1$. It can be checked that this is the case if $1 < k < \frac{1}{\tilde{q}_v}$ and $d < \frac{(k-1)(1-hk)}{kq(1-h)}$. The first condition implies $hk < 1$, so in particular the second bound for d is always nonnegative and $x_v^* - x_a^* = \frac{d}{1-hk} > 0$, so $x_v^* > x_a^*$, as expected.

In addition, there is a corner equilibrium in which $x_a^* = x_v^* = 1$ if $x_a^* = 1 \le k(\tilde{q}_a + 1 - \tilde{q}_a) - d = k - d$, that is $k \ge 1 + d$. This condition immediately implies $x_a = 1$, and this, in turn, implies $k(\tilde{q}_v + 1 - \tilde{q}_v) > 1$, that implies that also $x_v = 1$.

There is a corner equilibrium in which $x_a^* = 0$ and $x_v^* = 1$ if $k\tilde{q}_v \ge 1$ and $k(1-\tilde{q}_a) \le d$. If $1 + d > k \ge 1/\tilde{q}_v$, $k(1-\tilde{q}_a) > d$ and $k\tilde{q}_a < 1$, there is an equilibrium where $x_v^* = 1$ and x_a interior, equal to:

$$x_a^* = \frac{k(1-\tilde{q}_a) - d}{1 - k\tilde{q}_a}$$

Part 2

This part simply follows from the calculation of the derivatives:

$$\partial_h x_a = -\frac{dk(1-q)}{(hk-1)^2} < 0, \quad \partial_h x_v = \frac{dkq}{(hk-1)^2} > 0,$$

Part 3

For $h \to 1$, given the characterization in the proof of Part 1, regardless of the other parameters, the only remaining equilibria are corner equilibria where vaccinations levels are either zero or one. So, the effect of increasing homophily is zero: $\partial_h x_a = \partial_h x_v = 0$. As a consequence, the comparative statics with respect to homophily satisfy $d_h CI > 0$, and $d_h \rho^{SS} < 0$, as in Proposition 6. □

Appendix B. Supplementary material

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.geb.2024.12.007.

Data availability

No data was used for the research described in the article.

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