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Homophily and Infections: Static and Dynamic Effects

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Abstract
We analyze the effect of homophily in an epidemics between two groups of agents that differ in vaccination rates ("vaxxers" and "anti-vaxxers"). The steady state infection rate is hump-shaped in homophily, whereas the cumulative number of agents infected during an outbreak is u-shaped. If vaccination rates are endogenous, homophily has the opposite impact on the two groups, but the qualitative behavior of the aggregate is unchanged. However, the sign of the group-level impact is the opposite if vaccination is motivated by infection risk or by peer pressure. If motivations are group-specific, homophily can be harmful for both groups.

JEL Classification: D62; D85; I12; I18.

Keywords: Homophily; seasonal diseases; vaccination; anti-vaccination movements; SIS-type model.

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1 Introduction

The diffusion of diseases is crucially affected by the homophily between different groups, namely the tendency of members of a group to interact among themselves more than across groups. Such a tendency can be affected by policies aiming at increasing vaccination uptake, when enforcement is imperfect. With this motivation, we study an environment with two heterogeneous groups of agents, one with high vaccination rate, the other with a low vaccination rate. We analyze the impact of a change in homophily on the number of infections in a world facing a disease that is endemic, in the sense of a non-zero long-run prevalence, but subject to outbreaks. This mimics what happens for seasonal flu and for COVID-19. Specifically, we ask what is the impact of homophily on the amount of infections generated throughout the epidemic: our key result is that the effect on *steady state* infection level is diametrically opposite to the effect on the cumulative number of infected-person-periods generated by an outbreak.

It is well known that social networks exhibit a high degree of homophily, and that homophily is one of the network characteristics crucially affecting diffusion and contagion.[1] Moreover, when policy makers have to implement certain policies, such as affecting vaccination uptake, this may affect homophily. For example, there has been a lot of evidence that in the US private and charter schools have a higher level of non-vaccinated children,[2] and this is driven by a larger number of families that use the possibility of religious or philosophical exemptions.[3] There is reason to believe that these more permissive schools have attracted parents that are more skeptical of vaccinations.[4] Another example is what happened during the COVID-19 outbreaks in 2021 and 2022, when governments have implemented strong temporary containment and

[1] See e.g. Jackson (2008).

[2] This phenomenon is documented for California by Silverman and Yang (2019). Recent evidence shows that similar trends happened in Italy and have been considered a cause of a measles outbreak in Manhattan in April 2019. ·, Mashinini et al. (2020), Shaw et al. (2014).

[3] Zier and Bradford (2020)

[4] For example, Sobo (2015) argues that school community norms have an important impact in vaccine skepticism among families of children attending Steiner schools.

quarantine policies, such as vaccination passports and, in Italy, the so-called *green pass*. These measures, as the compulsory green pass to attend social activities such as entering restaurants, are likely to be subject to a wide variety of enforcement levels, depending on the type of social activity, and the interest of the owners. As a consequence, this is another measure that can have the unintended effect of increasing homophily of interaction between people with similar vaccination rates (Bardosh et al., 2022).

We model the spread of a generic disease using a standard SIS model with two groups, that differ in their vaccination rates. Vaccination is perfectly effective. We later endogenize vaccination rates and microfound the discrepancy, using a higher evaluation of costs for a group. With this in mind, we label the two groups "vaxxers" and "anti-vaxxers". The homophily of contacts between the two groups is modeled by a parameter $h \in [0, 1]$, which is the percentage of 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. As the object of interest, we analyze the expected discounted aggregate number of infections across all the periods. This is what we would be interested in if, for example, each period an agent is ill she cannot work, or has to be cured, thus creating a cost for the society. If infections are constant, this number is the same as the steady state prevalence. However, in an outbreak, when infections can vary, the two objects can be very different. Indeed, one of the messages of this paper is that the effect of homophily is very different when taking the cumulative infections into account, rather than the steady state alone.

For tractability, we focus on the linear approximation of the dynamics around the steady state. We decompose the total number of infections in a static component, that is the steady-state infection, and a dynamic component, that depends on the size of a deviation from the steady state, and is the amount of infections due to the outbreak. This dynamic component can be thought of as the Bonacich (1987) centrality in the network composed by the two groups, where the strength of the connection between groups depends on the amount

of probable infections transmitted. The key result is that, while the steady-state total infection level is hump-shaped in homophily - namely increasing for small h and decreasing for large h - cumulative infections are increasing in homophily for large h and decreasing for h small. Since the size of the outbreak is zero on average, the long-run steady state can also be thought of as the *average* number of cumulative infections generated, while the outbreak cumulative infections is a measure of the variance of the total number. Hence, the global effect of homophily depends crucially on the level of risk aversion that society has.

The key intuition behind the result is as follows. In 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 determiants of these indirect effects are (i) a *size* effect: higher infection decreases susceptibles, hence decreases future infections; and (ii) a *contagion* effect: higher infection increases future infections boosting 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 contagion effect (hence is negative).

The effect of homophily on cumulative infections due to an outbreak is also decomposed in a direct and indirect effect. First, homophily has a direct effect of changing the dynamics, that we explore in Section 3.2 through a two-step example. Namely, the level of homophily affects the sign of the gap between the additional infections generated in the two groups: this acts as a reversal of the direct effect of homophily on steady-state levels, thus reversing the behavior when h is large. Second, there is an indirect effect due to the change in steady-state levels: higher steady-state levels mean that there are less susceptible individuals that can be infected, hence outbreaks are smaller. As a consequence, cumulative infections due to the outbreak are *decreasing* in the steady-state levels.

Naturally, vaccination rates are not exogenous. To study the effect of vaccination rates that adjust when homophily changes, we explore a model in which agents trade-off an 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.[5]

We explore two different possibilities for the motivation of vaccinations: 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. Reality is likely a mix of the two, so these cases should be thought of as the two extreme cases in which only one of the two components is visible, for the sake of illustration. We explore these alternatives because there is a well-documented fact about vaccine hesitancy that seems hard to reconcile with strategic models: its geographical and social clustering. Various studies, reviewed, e.g., by Dubé and MacDonald (2016), find that people are more likely to have positive attitudes toward vaccination if their family or peers have. In addition, Lieu et al. (2015) show that vaccine-hesitant people are more likely to communicate together than with other people. 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.

[5] 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).

We find that the basic result of the different behavior of the static and dynamic component carries through, because vaccination rates adjust in opposite directions, hence the additional effect is never too strong. The group-level comparative statics are very different under the two vaccination models though. If vaccinations are motivated by risk of infections, an increase in homophily has the effect of increasing risk, hence vaccinations, among anti-vaxxers, and decreasing them among vaxxers. If vaccination is 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. Homophily is the most harmful to vaccinations in an 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).

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 impact 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 due to an outbreak. Our cumulative measure of infections can be seen as a reduced form of various utilitarian welfare functions that have been employed in the recent literature: the more 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. (2021), Bognanni et al. (2020). All of these papers do not consider the effect

of the social network. Bisin and Gottardi (2021) consider health and economics trade-offs, but they do not consider dynamics or homophily. The structure of the cross-country network is considered in Chandrasekhar et al. (2021), that also consider as the objective of the planner to minimize the number of infected person periods, a measure analogous to cumulative infection. None of these papers study 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 the outbreak rather than the steady state. It is well known that homophily might 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 the steady state levels, nor the dynamic 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.

Our contribution to the literature on strategic immunization models is to show that the impact of homophily on group level vaccinations can be opposite if vaccination is motivated mainly by avoiding the risk of infection, or mainly by peer pressure. Our model of vaccinations motivated by infection risk is analogous to Galeotti and Rogers (2013). This paper focuses on the steady state and not on the dynamic component, which is our focus. In addition, the endogenous vaccination model in Galeotti and Rogers (2013) generates symmetric vaccination across 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) studies the interaction between the endogenous level of interaction and vaccinations, again in 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), that shows that a partially effective vaccination can decrease welfare. Again, our focus is rather on the static-dynamic trade-offs. Chen and Toxvaerd (2014) argues that the market mechanism yields inefficiently low levels of vaccination. No one of these papers explore vaccination

driven by peer pressure.[6]

The paper is organized as follows. The next section presents the model. Section 3 shows results for the mechanical model, when all choices are exogenous. Section 4 explores the robustness of results to endogenous vaccination rates. Section 5 explores some generalizations and extensions. We conclude in Section 6. In Appendix A we analyze the model when the disease is not endemic but has a zero steady state.In Appendix B we prove the formal results of our paper.

2 The Model

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

Our society is composed of a continuum of agents of mass 1, partitioned into two groups. To begin with, in this section this partition is exogenous. Agents in each group are characterized by their attitude towards vaccination. In details, 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 $q_a \in [0, 1]$ denote the fraction of *anti-vaxxers* in the society, and $q^v = 1 - q_a$ the fraction of *vaxxers*. To ease the notation, we write q for q_a, when this does not create ambiguity.

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 to meet only someone from her own group and a probability $1 - h$ to meet someone else randomly drawn from the whole society.[7] This implies that anti-

[6] 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).

[7] 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 in several ways, as an outcome of choices or opportunities. As we assume that h can be affected by groups' choices and by policies, we can interpret it as the amount of time in which agents are kept segregated by group, while in the remaining time they meet uniformly at random.

vaxxers meet each others at a rate of $\tilde{q}_a := h + (1-h)q_a$, while vaxxers meet each others at a rate of $\tilde{q}_v := h + (1-h)q^v = h + (1-h)(1-q_a)$. Note that h is the same for both groups, but if $q \neq 1/2$ and $0 < h < 1$, 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 our generic disease. It is natural to assume, without loss of generality, that $x_a < x_v$, and by now this is actually the only difference characterizing the two groups. The total number of vaccinated (or average vaccination rate) is $x = qx_a + (1-q)x_v$. We start by taking x_a and x_v as exogenous parameters, and we endogenize them later. Similarly, ρ_g and S_g denote respectively the fraction of infected and susceptibles in group g. 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 are going to be concerned with the stable steady state of the system above, and with *outbreaks*, deviations from the steady state. Such outbreaks are stochastic, zero-mean deviations from long-run steady-state values: $d\rho_0 = (d\rho_{0,a}, d\rho_{0,v})$, 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. We denote the variance of such stochastic deviation as $\sigma^2 := Var(d\rho_0)$.

2.1 The dynamical system

Setting the evolution of the epidemic in continuous time, we study the fraction of infected people in each group. For each $i \in G$, let ρ^i be the share of infected agents in group i. Since vaccinated agents cannot get infected, we have $\rho_a \in [0, 1-x_a]$ and $\rho_v \in [0, 1-x_v]$, respectively.

The differential equations of the system are given by:

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

where $S_g = \left(1-\rho^g - x^g\right) \in [0,1]$ are the fraction of agents who are neither vaccinated, nor infected, and thus susceptible of being infected by other infected agents. Moreover, the shares of infected agents met by anti-vaxxers and vaxxers are given by $\tilde{\rho}_a := \Big(\tilde{q}_a\rho_a + (1-\tilde{q}_a)\rho_v\Big)$ and by $\tilde{\rho}_v := \Big(\tilde{q}_v\rho_v + (1-\tilde{q}_v)\rho_a\Big)$, respectively. Finally, $\rho_a\mu$ and $\rho_v\mu$ are the recovered agents in each group.

First, in the next proposition we characterize some properties of the steady states.

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 $\mu < \hat{\mu}(h)$, the stable steady state is interior: $\rho_a^{SS} > 0$, $\rho_v^{SS} > 0$, while there is another (unstable) steady state in $(0,0)$, whereas (ii) if $\mu > \hat{\mu}(h)$, the stable steady state is $\rho^{SS} = (0,0)$.*

The formal passages of the proof are in Appendix B, as those of the other results that follow.

In the main body of the paper, we consider the case in which $\mu < \hat{\mu}(h)$, and show the results concerning the interior steady state. This might be the setting more apt to describe the recent behavior of COVID-19, with a consistent number of baseline cases (the interior steady state), and occasional outbreaks.[8]

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

[8]The case in which $\mu > \hat{\mu}(h)$ is consistent with diseases that are not endemic but show themselves in episodic or seasonal waves. For those diseases, society lays for most of its time in a steady state where no one is infected. However, exogenous shocks increase the number of infected people temporarily. Eventually, the disease dies out, as it happens, for example, for the seasonal outbreaks of flu.

Definition 1 (Outbreak dynamic)**.** *We define the function* $\mathrm{d}\rho_t$ *as the time evolution that satisfies:*

$$\begin{pmatrix} \dot{\mathrm{d}\rho}_{a,t} \\ \dot{\mathrm{d}\rho}_{v,t} \end{pmatrix} = J \begin{pmatrix} \mathrm{d}\rho_{t,a} \\ \mathrm{d}\rho_{t,v} \end{pmatrix}, \quad \mathrm{d}\rho_0 = \begin{pmatrix} \mathrm{d}\rho_{0,a} \\ \mathrm{d}\rho_{0,v} \end{pmatrix}, \tag{2}$$

where

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

is the Jacobian matrix of (1) *calculated in the steady state, and* $(\mathrm{d}\rho_{0,a}, \mathrm{d}\rho_{0,v})'$ *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, hence it follows that J has negative diagonal, and in particular $-J$ is an $M-$matrix. We denote the determinant of J as $|J|$ and note that it is positive. We note also that $\hat{\mu}(h)$ (explicitly derived in Appendix B) is increasing in h, so that we can highlight a first important role for h in the comparative statics. If h increases, it is possible that a disease that was not endemic, because $\mu > \hat{\mu}(h)$, becomes so as $\hat{\mu}(h)$ increases with h, and the sign of the inequality is reversed. Indeed, higher homophily counterbalances the negative effect that the recovery rate μ has on the epidemic outbreak (on this, see also the discussion in Jackson and López-Pintado, 2013).

2.2 Cumulative infection of the outbreak and society's preferences

In the following, we are going to analyze the total cumulative number of infections generated by an epidemic (or the number of infected-person-periods in the terminology of, e.g., Chandrasekhar et al., 2021). This is equal to a baseline steady state level, and oscillations around it due to the outbreaks. A key simplification is that the dynamics $\mathrm{d}\rho_{t,a}$ and $\mathrm{d}\rho_{t,v}$ are by construction linear in $\mathrm{d}\rho_0$: hence, the total cumulative number of infections overtime is also

linear in $\mathrm{d}\rho_0$.

Definition 2 (Cumulative infection)**.** *Define $\tilde{CI}$ as the (normalized) cumulative number of infections due to an outbreak 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:

$$\tilde{CI}(\overline{d\rho_0}) = \rho^{SS} + CI\overline{d\rho_0} \tag{3}$$

Notice that the expectation and variance of $\tilde{CI}$ are:

$$\mathbb{E}(TI) = \rho^{SS} \tag{4}$$

$$StDev(TI) = CI\sigma \tag{5}$$

Hence, $\rho^S S$ and CI measure, respectively, the average infection level and the size of the cumulative deviations along an outbreak. What is the relevance of these measures? Suppose society is risk averse, following a utility function of the type:

$$W = -\mathbb{E}\tilde{CI} - BStDev(\tilde{CI}) = -\rho^{SS} - B\sigma CI$$

In this stylized world, welfare depends only on the steady-state level of infection, the number of infections due to outbreaks, and preference parameters. In particular, B regulates the aversion to risk of the planner. If society is more risk averse, it cares more about the development of outbreaks rather than the long-run steady-state infection level.

When computing the welfare effect of homophily, we calculate:

$$\frac{\partial W}{\partial h} = -\frac{\partial \rho^{SS}}{\partial h} - B\sigma\frac{\partial CI}{\partial h}$$

We can see that the relative contribution of the steady state and the cumulative infection is regulated by the weight B and the standard deviation σ, that are arbitrary parameters, independent of the epidemic model. Hence, in case the two effects countervail each other (which we are going to show is a typical case, especially for h high), the sign of the welfare effect crucially depends on the preference parameters of the planner.

In the following, we analyze separately the behavior of $\frac{\partial \rho^{SS}}{\partial h}$ and $\frac{\partial CI}{\partial h}$, and we show how homophily can have opposite effects on them: hence, the sign of the welfare impact of homophily crucially depends on how these effects are combined through risk aversion and discounting parameters.

3 Steady state vs Cumulative infection

In this section we start analyzing the pure epidemic part of the model, 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 $x_a < x_v$.

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: hence, an increase in homophily h has the effect of increasing the amount of not vaccinated people that anti-vaxxers interact with, with the result of increasing the steady-state infection level. 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 derivatives of the infection rates are:*

$$\begin{aligned} \partial_h \rho_a &= -\frac{S_a(1-q)\Delta\rho}{|J|}(S_v - \tilde{\rho}_v - \mu); \\ \partial_h \rho_v &= \frac{S_v q \Delta\rho}{|J|}(S_a - \tilde{\rho}_a - \mu). \end{aligned} \tag{6}$$

1. *Infection among the anti-vaxxers ρ_a is increasing in homophily;*

2. *Infection among the vaxxers ρ_v is always decreasing in homophily if $\mu < (1-x)^2/(1-x_a)$. Otherwise, is hump-shaped: decreasing if h is large enough, increasing otherwise;*

3. *The total infection ρ is increasing if homophily is small enough, and decreasing if homophily is high enough;*[9]

4. *Infection in all groups is decreasing in x_a, x_v.*

Moreover, $\tilde{\rho}_a$ is also increasing in h, and $\tilde{\rho}_v$ is increasing in h whenever ρ_v is.

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 effect that a change in the steady states have. The direct effects are caused by the homophily changing the probability of infection:

$$\begin{aligned} \partial_h \tilde{\rho}_a &= (1-q)\Delta\rho; \\ \partial_h \tilde{\rho}_v &= -q\Delta\rho. \end{aligned} \tag{7}$$

They have opposite signs: anti-vaxxers meet more frequently anti-vaxxers hence, ceteris paribus, their probability of infection goes up. 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

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

decomposed as such:

$$\begin{aligned}
\mathrm{d}F_a =& \partial_{\rho_a}(S_a\tilde{\rho}_a)\mathrm{d}\rho_a + \partial_{\rho_v}(S_a\tilde{\rho}_a)\mathrm{d}\rho_v \\
=&(\underbrace{-\mu}_{\substack{\text{recovery}\\\text{effect}}} - \underbrace{\tilde{\rho}_a}_{\substack{\text{size}\\\text{effect}}} + \underbrace{\tilde{q}S_a}_{\substack{\text{contagion}\\\text{effect}}})d\rho_a + \underbrace{(1-\tilde{q})S_a}_{\substack{\text{contagion}\\\text{effect}}} d\rho_v; \\
\mathrm{d}F_v =&(\underbrace{-\mu}_{\text{recovery}} - \underbrace{\tilde{\rho}_v}_{\text{size}} + \underbrace{\tilde{q}_v S_v}_{\text{contagion}})d\rho_a + \underbrace{(1-\tilde{q}_v)S_v}_{\text{contagion}} d\rho_v. \qquad (8)
\end{aligned}$$

For example, for group a, an increase in the steady-state level ρ_a generates, for group a: (i) an increase of recovery, (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 increment in infection comes from a product of the amount of susceptible agents and of 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 of 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 have always opposite sign with respect to the direct effects, hence the balance is of uncertain sign. The results above state that the indirect effects are never strong enough to counterbalance the direct effect in group a, and so ρ_a is always increasing in $\rho_{0,a}$. In group v instead, the derivative can take both signs: it is negative if the direct effect prevails, and positive otherwise. Since the cross-group contagion is part of the indirect effect, the direct effect prevails and ρ_v is decreasing in $\rho_{0,a}$ when h is large,

that is, when the two groups are almost separated. Instead, when h is small, the indirect effect may be stronger that the direct one, and ρ_v can be also increasing in $\rho_{0,a}$. This happens when the recovery rate μ is large enough, so that the contagion effect is a more important 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 (8) reflect the fact that the variations of the steady state are a combination of the direct effects from (7), 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 are 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: hence homophily decreases total infections.[11]

[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 do not matter anymore.

3.2 Cumulative infection

We now analyze how results are affected once we explicitly model the infection outbreaks. First, we can note that cumulative infection is closely related to Bonacich centrality:

$$\begin{pmatrix} \tilde{CI}_a \\ \tilde{CI}_v \end{pmatrix} = r \int_0^\infty e^{(-rI+J)t} \mathrm{d}\rho_0 \mathrm{d}t = (I - 1/rJ)^{-1} \mathrm{d}\rho_0.$$

We can see that the vector of cumulative infections in the two groups is equal to the Bonacich centrality in the network defined by the Jacobian matrix J. This expression is going to be useful to make calculations with cumulative infection. The intuition can be better grasped considering the associated discrete time dynamics, in which the outbreak satisfies:

$$\begin{pmatrix} \mathrm{d}\rho_{a,t+1} \\ \mathrm{d}\rho_{v,t+1} \end{pmatrix} = J \begin{pmatrix} d\rho_{a,t} \\ d\rho_{v,t} \end{pmatrix}. \tag{9}$$

In such a case, the cumulative infection is simply

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

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 can be obtained for the step size going to zero.

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

Proposition 3. *The impact of homophily h on CI can be decomposed as:*

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

1. *The direct effect* $\partial_h CI$ *is positive if* h *high enough, while, for* $h \to 0$, $\partial_h CI$ *is positive if and only if* $\mu > \frac{1-x}{2}$*;*

2. *the indirect effect is equal to* $-\frac{1}{detJ}\partial_h \rho$*.*

3. *the total effect* $\mathrm{d}_h CI$ *is positive when* h *is low enough, and negative when* h *is high enough.*

What is the reason for this discrepancy? As discussed above, there is a direct effect of h on the dynamics, and an indirect effect, due to h affecting also the steady-state levels. 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 (9). Let us analyze a simple two-step discrete version of the outbreak 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:

$$d\rho_2 = -\mathrm{d}\rho_1^2 + q(1-q)h(\Delta S - \Delta\rho^{SS})\Delta \mathrm{d}\rho_1. \tag{10}$$

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 (10) 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 infi-

[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.

nite time. The next paragraph shows how we can get similar long run intuitions analyzing the behavior of the convergence time, 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:

$$\dot{d\rho}_a = -\rho_a^{SS} d\rho_a \Longrightarrow \mathrm{d}\rho_a = e^{-\rho_a^{SS} t} \rho_0$$

so that we can analytically compute: $CI = \frac{\rho_0}{\rho_a^{SS} + r}$: 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 outbreak 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 intuitio, the convergence time of the dynamics, as measured by the smallest eigenvalue of J, is decreasing in the steady state levels.

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

[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:

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

Definition 3 (Speed of convergence)**.** *The speed of convergence of the system after an outbreak of size ρ is*

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

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

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

$$CT = \lambda_2$$

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

4 Vaccination choices

The fraction of vaccinated agents in the population itself might depend on homophily once we take into account that vaccination is endogenous. We endogenize infections with a variant of the model in Galeotti and Rogers (2013) with heterogeneous costs, to explore asymmetric equilibria. Agents might vaccinate paying a cost, or not vaccinate incurring the risk of becoming infected. Since in our model, as in Galeotti and Rogers (2013), the fraction of time agents spend infected is the steady state value, the health disutility of being not vaccinated is $-\rho_a^{SS}$ for agents in a, and $-\rho_v^{SS}$ for agents in v. From here on, we drop the SS apex. With a descriptive spirit, we microfound the

discrepancy in vaccination rate assuming that anti-vaxxers have a cost larger than vaxxers of a uniform amount d. However, our focus being the effect of homophily on infections, we do not dig deeper in 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 $c^a \sim U[d/k, 1/k + d/k]$. k is a parameter reflecting 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.[14]

We assume that agents take vaccination decisions ex-ante, before an epidemic actually takes place, and cannot update their decision during the diffusion. This mimics well diseases, like seasonal flu, for which the vaccine takes a few weeks before it is fully effective, and the disease spreads rapidly among the population.

Since there is a continuum of agents, each individual takes as given the fraction of vaccinated in the population as given. Thus, an agent in group a vaccinates if and only if $c < \rho_a$. The fraction of agents that vaccinate is, thus, $x_a = k\rho_a - d$. Similarly, $x_v = k\rho_v$ for vaxxers. The following lemma guarantees existence and uniqueness of the vaccination equilibrium.

Lemma 1. *If $k\rho_a > d$ the equations:*

$$\begin{cases} x_a &= k\rho_a - d \\ x_v &= k\rho_v \end{cases} \tag{11}$$

define a unique equilibrium (x_a^*, x_v^*), whenever the solution is interior. Moreover, $x_a^* < x_v^*$, x_a^* is increasing in h, and x_v^* is decreasing in h.

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

[14] 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.

smaller fraction of vaxxers is vaccinated: x_v is decreasing in h. The opposite happens for anti-vaxxers.

4.1 Impact of homophily

Steady state

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 infection levels. The total derivative of steady state infection is

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

If $h \to 1$, we know that $\partial_{x_a}\rho \to -q$ and $\partial_{x_a}\rho \to -(1-q)$. Moreover, using the expressions obtained above for $\partial_h x_a$ and $\partial_h x_v$, we get:

$$\partial_h x_a \to \frac{k}{1+k}\partial_h\rho_a, \tag{12}$$

$$\partial_h x_v \to \frac{k}{1+k}\partial_h\rho_v. \tag{13}$$

Hence, the total additional effect is $-\frac{k}{1+k}(q\partial_h\rho_a + (1-q)\partial_h\rho_v) = -\frac{k}{1+k}\partial_h\rho > 0$. The decrease in infection among the vaxxers dominates the increase among anti-vaxxers (the effect discussed in the previous section). This, in turn, triggers a decrease in (total) vaccination and an increase in total infection. However, since only a fraction of agents vaccinates, this translates in an increase in infection that is less than proportional to the direct effect, mediated by the parameter k. As a result, the sign of the derivative is the same, even if the magnitude is dampened.

If $h \to 0$, we obtain exactly the same result, with symmetric intuitions. The next proposition sums up the discussion of this section (for details when $h \to 0$ see the proof). Let $d_h\rho\,|_{x\,const}$ be the $d_h\rho$ as in Proposition 2, i.e., with exogenous vaccination choices.

Proposition 5. *If $h \to 0$ or $h \to 1$ the total derivative of the steady state*

total infection with respect to homophily satisfies

$$d_h\rho = \frac{1}{k+1} d_h\rho \mid_{x\, const},$$

The sign of such derivative is the same as with exogenous vaccination rates, while the magnitude is smaller.

Cumulative infection

Concerning the cumulative infection of the outbreak, the total derivative can be decomposed as:

$$d_h CI = d_h CI \mid_{x_a, x_v \text{ const}} + d_{x_a} CI \partial_h x_a + d_{x_v} CI \partial_h x_v$$

where $d_h CI \mid_{x_a, x_v \text{ const}} = \partial_h CI + \partial_{\rho_a} CI \partial_h \rho_a + \partial_{\rho_v} CI \partial_h \rho_v$ is $d_h CI$ we calculated in Proposition 3, with exogenous vaccination choices.

The term $d_{x_a} CI \partial_h x_a + d_{x_v} CI \partial_h x_v$, for $h \to 0$ or $h \to 1$ can be shown to be proportional to $-(\partial_{x_a} \rho \partial_h x_a + \partial_{x_v} \rho \partial_h x_v)$ that, in turn, by the previous proposition, is proportional to $\partial_h \rho$.

Hence, if $h \to 1$, the baseline term $d_h CI \mid_{x_a, x_v \text{ const}}$ is positive, and also the additional terms are positive. Hence, the total derivative is positive, with a magnitude larger than with fixed vaccination rates: $d_h CI > d_h CI \mid_{x\, cost}$. If $h \to 0$, the baseline term is negative and the additional effects are negative, hence the symmetric thing happens. Formally, we can sum up the results in the following proposition.

Proposition 6. *For h large enough, cumulative infection is increasing in homophily, and the sensitivity is larger than with exogenous vaccination rates: $d_h CI > d_h CI \mid_{x\, cost} > 0$. For h small enough, the cumulative infection is decreasing in homophily, and the sensitivity is larger than with exogenous vaccination rates: $0 > d_h CI \mid_{x\, cost} > d_h CI$.*

Hence, we conclude that the difference of behavior between steady-state and dynamic infection is magnified by the presence ofendogenous vaccination rates.

4.2 Vaccination motivated by peer pressure

In this sectgion we explore an alternative scenario in which vaccination decisions are not driven by a correct evaluation of the infection risk. Instead, we consideragents 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 is 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 than what happens with vaccination based on 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 peer pressure 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 $h \to 0$ and $h \to 1$, as in the baseline framework we presented.

Formally, an agent in group a with heterogeneous cost c vaccinates if $c < \tilde{q}_a x_a + (1 - \tilde{q}_a)x_v - d/k$, and the analogous but with $d = 0$ happens for an agent in group v. Hence, the vaccination rates at interior solutions are defined by:

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

This is a linear system, and the interior solution is (whenever feasible):

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

This formulation highlights how the equilibrium vaccination rates are proportional to Bonacich centralities in the network defined by the meeting rates $\begin{pmatrix} \tilde{q}_a & 1 - \tilde{q}_a \\ 1 - \tilde{q}_v & \tilde{q}_v \end{pmatrix}$. If $k < 1$ the inverse matrix is positive, and so there are

no interior solutions, and all the vaccination rates are 0.

When $k > 1$ interior solutions are given by:

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

Thus, if $h > 1/k$ we must have $x_v = 0$, and if $h > 1 - \frac{k-1}{kq}$, then $x_a = 0$. Hence, for h high enough no one vaccinates, and theninfection rates are higher. The derivatives of the interior solutions with respect to homophily are:

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

and we can see that they are identical up to proportionality factors $1-q$, and q. Since, for $h \to 0$, the derivatives of the steady state are proportional to q and $1-q$, the additional effect of endogenizing vaccination rates is 0 for $h \to 0$. It is also null for $h \to 1$, because in that case, as we noted above, both vaccination rates are 0 and so they are not sensitive to homophily. It follows that under this vaccination model the impact of homophily on both the steady-state and the cumulative infection for $h \to 0$ and $h \to 1$ are the same as in the model with exogenous vaccination rates. In particular, the impact on the steady state and the impact on the cumulative infection are still opposite.

4.3 A mixed model

We now consider a model with mixed motivations. We consider the case in which anti-vaxxers, instead of evaluating correctly the risk, rely on peer pressure, whereas vaxxers are more prone to evaluate risks. In this case the effect of homophily is the most negative. In this model for some parameter values an increase in homophily produces unambiguously a decrease in vaccination rates in *both groups*: vaxxers vaccinate less because of lower risk, while anti-vaxxers

vaccinate less because of peer pressure. To be formal, assume:

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

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

5 Generalizations and variations

5.1 Imperfectly effective vaccination

The model can be reinterpreted to explore the effect of an imperfectly effective vaccination. Indeed, if in the baseline model we reinterpret x_a and x_v as the rates of *effectively immunized*, all the results can be applied in a similar way. Indeed, suppose that the rate of success of the vaccination is $\eta \in [0,1]$, and the vaccination rates are $\hat{x}_a$ and $\hat{x}_v$: then the effective fractions of immunized agents are $x_a = \eta\hat{x}_a$, $x_v = \eta\hat{x}_v$: notice that $x_v > x_a$ if and only if $\hat{x}_v > \hat{x}_a$. Hence, all the results on exogenous vaccination rates remain valid. Also the results on endogenous vaccination rates remain valid, though, as long as the rate of success of the vaccination is the same, η, in both groups. Indeed, $\partial_h x_a = \eta\partial_h\hat{x}_a$ and similarly for $\partial_h x_v$, so that all the derivatives would simply

be multiplied by η. Hence, for example, Proposition 5 would be modified as:

$$\mathrm{d}_h\rho = \frac{1+(1-\eta)k}{1+k}\mathrm{d}_h\rho\,|_{x \text{ constant}}$$

This variation offers a particularly interesting case: consider a case in which $\hat{x}_a = 0$ and $\hat{x}_v = 1$. In such a case we can identify the *vaxxers* with the vaccinated and the *anti-vaxxers* with the non-vaccinated. In such a case, if the vaccination is perfectly effective, homophily is unambiguously negative, as it increases contacts between non-vaccinated, hence increasing infection. Instead, if the vaccination is imperfectly effective, for homophily high enough the steady state infection goes down. Hence it follows that perfectly or imperfectly effective infection can change the qualitative impact of homophily.

5.2 Corner solutions for vaccination rates

The effects described in the previous section crucially rely on the fact that both vaccination rates adjust when homophily changes. This breaks down when one of the two groups is at a corner solution: e.g. $x_a = 0$ (which happens when d is high enough), or if $x_v = 1$. These cases have opposite comparative statics: if all vaxxers vaccinate, when homophily increases (of a moderate amount), vaccinations unambiguously decrease, because the increased risk affects only the anti-vaxxers, increasing their vaccination level. Instead if no anti-vaxxers vaccinate, homophily unambiguously increases vaccinations, because the change causes a decrease in risk for vaxxers.

6 Conclusion

The problem of vaccine skepticism is a complex one, and requires an analysis from multiple perspectives, e.g., psychological, medical, and social. The results of this paper might be relevant for a policy maker interested in minimizing infection in a world with vaxxers and anti–vaxxers, having available a policy inducing some degree of segregation, or homophily, h. The key observation

is that reducing contact with anti–vaxxers may be counterproductive both from the perspective of vaxxers and of the society as a whole because it slows down the dynamics of the disease to its steady state, if there is an outbreak. Homophily may actually increase the duration of the outbreaks and, depending on the time preferences of the planner, this might crucially change the impact of the policy.

Appendix A Zero steady state

If μ is high enough, following Proposition 1, then the only steady state is the zero infection steady state $\rho_a^{SS} = \rho_v^{SS} = 0$, it is possible to give a sharper characterization. Indeed, since the steady state infection levels do not vary with h, the change in cumulative infection is driven uniquely by the direct effect of h.

Proposition A. *If $\mu > \hat{\mu}(h)$, there is only one steady state, $\rho_a^{SS} = \rho_v^{SS} = 0$, and is stable.*

If $d\rho_{0,a} = d\rho_{0,v} > 0$, the cumulative infection satisfies the following.

a) $CI = CI$ and CI^a are increasing in h; CI^v is decreasing in h;

b) CI, CI^a and CI^v are decreasing in x_v and x_a;

c) CI, CI^a and CI^v are increasing in q.

A.1 Vaccinations

Since the steady state is zero, if we try to apply directly the vaccination model in the main text, we run into the problem that no agent would have an incentive to vaccinate. In this case we can assume that agents think about the risk of infection using a simple heuristic: they estimate it as being proportional to the fraction of non-vaccinated people that they meet. Agents multiply this fraction of non-vaccinated people by a factor $k > 0$, that represents the perceived damage from the disease, which is the same for the two groups. Thus:

$$x_v = k[\tilde{q}_v(1 - x_v) + (1 - \tilde{q}_v)(1 - x_a)] \ , \tag{a}$$

and similarly:

$$x_a = \max\{k(\tilde{q}_a(1 - x_a) + (1 - \tilde{q}_a)(1 - x_v)) - d, 0\} \ , \tag{b}$$

One big advantage of this functional specification is that it allows readily to solve for the interior equilibrium:

$$\begin{aligned} x_a &= 1 - \frac{1+dq_a}{1+k} - \frac{d(1-q_a)}{1+hk}, \\ x_v &= 1 - \frac{1+dq_a}{1+k} + \frac{dq_a}{1+hk} \quad . \end{aligned} \tag{c}$$

This is true provided $d < \overline{d} = \min\left\{\frac{1}{k^2}, \frac{k}{k+1}\right\}$. We use this interiority condition as a maintained assumption for the remainder of this section.

First of all, we note that (i) $x_v > x_a$ - since vaxxers perceive a lower vaccination costs than anti-vaxxers; (ii) x_a is increasing in h whereas x_v is decreasing in h - since a higher homophily makes vaxxers more in contact with agents who are less susceptible than anti-vaxxers and, as a consequence, $(x_v - x_a)$ is decreasing in h; (iii) x_a and x_v are increasing in q_a - since the higher the share of anti-vaxxers, the more agents are in touch with other subjects at risk of infection; (iv) the total number of vaccinated people is $q_a x_a + (1-q_a)x_v = \frac{k-dq_a}{1+k}$, it is independent of h, but decreasing in q_a. It is possible to characterize analytically the behavior of the cumulative infection, as in the following proposition.

Proposition B. *If $d < \overline{d}$, then CI is increasing in h, though* less *than in the case in which vaccination rates are exogenous.*

A.2 Corner solution

If d is larger than $\overline{d}$ the threshold provided above, instead we have an equilibrium in which no anti-vaxxer vaccinates, obtaining:

$$\begin{aligned} x_a &= 0 \ , \\ x_v &= \frac{k}{(h-1)kq + k + 1} \ , \end{aligned} \tag{d}$$

provided $d < 1/k$ so that $x_v \neq 0$.[15]

From the expression we can immediately conclude that x_v is decreasing in h. Again, the mechanisms are similar as discussed in the Section 5.2: since a change in homophily impacts only the vaccination rate among vaxxers, and, by Proposition A, CI^v is decreasing in h, overall homophily increases CI, and the impact is larger than with exogenous vaccination rates, and in the interior equilibrium.

Appendix B Proofs

Proof of Proposition 1 (page 10)

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 a 2x2 matrix, we can explicitly write the expression of the eigenvalues as a function of the entries:

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

Now since $BC > 0$ it follows that the eigenvalues are real and distinct. The steady state is stable if they are both negative.

Now computing the Jacobian in the $(0,0)$ steady state we find that it is stable if and only if $\mu > \hat{\mu}(h) := \frac{1}{2}(T + \Delta) \in [0,1]$, 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. ■

[15] This is possible if $\frac{hk^2+k}{hkq-kq+k+1} < d < \frac{1}{k}$ and either $k < 1$ or $\left(1 < k < \frac{1}{2}\left(1+\sqrt{5}\right) \wedge 0 < q < \frac{-k^2+k+1}{k} \wedge 0 < h < \frac{-k^2-kq+k+1}{k^3-kq}\right)$

Proof of Proposition 2 (page 14)

We need first to establish some useful relations.

Lemma A. *In any steady state in which $x_a < x_v$, we have $\rho_a \geq \tilde{\rho}_a \geq \tilde{\rho}_v \geq \rho_v$, $S_a > S_v$, and $x_v - x_a \geq \rho_a - \rho_v$. Moreover $\tilde{\rho}_a - \tilde{\rho}_v = h(\rho_a - \rho_v)$.*

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

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 average number of vaccinated.

Proof. In every nonzero steady state:

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

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:

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

□

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 other derivative, 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 is:

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

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 (or hump-shaped).

The behavior as a function of the vaccination rates is:

$$\begin{aligned}\partial_{x_a}F_a &= -\tilde{\rho}_a & \partial_{x_a}F_v &= 0 \\ \partial_{x_a}\rho_a &= -\frac{1}{|J|}(-J_{22}\tilde{\rho}_a) < 0 & \partial_{x_a}\rho_v &= -\frac{1}{|J|}(J_{21}\tilde{\rho}_a) < 0\end{aligned}$$

and analogous for the derivative with respect to x_v. □

Proof of Proposition 3 (page 18)

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:*

$$\partial_y \vec{CI} = (q, 1-q)'(rI - J)^{-1}\partial_y J(rI - J)^{-1}d\rho_0$$

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

$$r\vec{CI} = d\rho_0 + J\vec{CI}$$

where $\partial_y J$ is the element by element derivative of J. Differentiating it and solving we obtain:

$$\partial_y \vec{CI} = (rI - J)^{-1}\partial_y J(rI - J)^{-1}d\rho_0$$

Since $rI - J$ is an M-matrix, the inverse has positive elements.

□

Now we can prove the Proposition.

Proof. For simplicity, write $d\rho_0 = d\rho_{0,a} = d\rho_{0,v}$. 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 $\mu < 1 - x$ we obtain the following.

$$\partial_h CI^{\text{out}} \mid_{h=1} = -\frac{(1-q)q\rho_0(x_a - x_v)^2(-2r + \mu + x_a + x_v - 2)}{(-r + \mu + x_a - 1)^2(-r + \mu + x_v - 1)^2} > 0$$

$$\partial_h CI^{\text{out}} \mid_{h=0}$$

$$= -\frac{\mu(1-q)q\mathrm{d}\rho_0(x_a - x_v)^2(2\mu + q(x_a - x_v) + x_v - 1)}{(q(x_a - x_v) + x_v - 1)^2(-r + q(x_a - x_v) + x_v - 1)(-r + \mu + q(x_a - x_v) + x_v - 1)^2} > 0$$

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

because the denominator $-r + q(x_a - x_v) + x_v - 1$ is negative.

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

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

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:

$$\partial_{\rho_a} CI \mid_{h=1} = -((2q\mathrm{d}\rho_0)/(-1 + x_a - r + \mu)^2)\rho_0$$

$$\partial_{\rho_v} CI \mid_{h=1} = -(2(1-q)\mathrm{d}\rho_0)/(-1 + x_v - r + \mu)^2\rho_0$$

$$\partial_{\rho_a} CI \mid_{h=0} = -\frac{2q\mathrm{d}\rho_0}{(-r + \mu + q(x_a - x_v) + x_v - 1)^2}$$

$$\partial_{\rho_v} CI \mid_{h=0} = -\frac{2(1-q)\mathrm{d}\rho_0}{(-r + \mu + q(x_a - x_v) + x_v - 1)^2}$$

we conclude that $(\partial_{\rho_a} CI, \partial_{\rho_v} CI) \propto -(q, 1-q)$, and so $\partial_{\rho_a} CI \partial_h \rho_a \mid_{h=0} + \partial_{\rho_v} CI \partial_h \rho_a \mid_{h=0} \propto -\partial_h \rho$.

Hence, for h high both the direct and indirect term in the derivative are positive, and so the derivative is positive. For h small, the indirect effect is negative, while the direct depends on μ: for $\mu < \frac{1-x}{2}$ they have the same sign, and the derivative is negative. For $\mu > \frac{1-x}{2}$, using the results above we can

compute the total effect, that is:

$$\begin{aligned}&\partial_h CI^{\text{out}} \mid_{h=0} + \partial_{\rho_a} CI \partial_h \rho_a \mid_{h=0} + \partial_{\rho_v} CI \partial_h \rho_a \mid_{h=0} \\ &= \frac{\mu(1-q)q\mathrm{d}\rho_0 \Delta x^2(-2r+2\mu-3(1-x)}{(1-x)^2(r+(1-x))(-r+\mu-(1-x))^2}\end{aligned}$$

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.

□

Proof of Proposition 4 (page 21)

Proof. Using the results in Bernstein and So (1993), we can express the exponential matrix as a function of eigenvalues, and directly compute the limit:

$$-\lim_{t\to\infty} \frac{\log\|e^{tA}\rho_0\|}{t} = \lambda_2 - \lim_{t\to\infty} \frac{\log\|\left(\frac{\lambda_1-\lambda_2 e^{-(\lambda_1-\lambda_2)t}}{\lambda_1-\lambda_2} I + \frac{1-e^{(\lambda_1-\lambda_2)t}}{\lambda_1-\lambda_2} A\right)\|}{t}$$

$$= \lambda_2 - \lim_{t\to\infty} \frac{\log\|(\lambda_1 - \lambda_2 e^{-(\lambda_1-\lambda_2)t})I + (1-e^{-(\lambda_1-\lambda_2)t})A\|}{t} = \lambda_2$$

Concerning the behavior of λ_2 as a function of h, we use a standard result on eigenvalue perturbations (Demmel, 1997, Th 4.4). Namely, if λ is a simple eigenvalue of $J - rI$:

$$\partial_h \lambda = \frac{v' \partial (J - rI) u}{v'u}$$

where v is the left and u the right eigenvector relative to λ.

In our case both eigenvalues are simple and we can explicitly solve for the eigenvectors. For $\lambda = -\lambda_2$, we obtain:

$$u = \left(\frac{2b}{\sqrt{(a-d)^2+4bc}-(a-d)}, 1\right)$$
$$v = \left(\frac{2c}{\sqrt{(a-d)^2+4bc}-(a-d)}, 1\right)$$

and they have both positive components, so $v'u > 0$: hence the sign of the

derivative is determined by the numerator. 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 negative.

Moreover, when $h \to 1$, both eigenvectors converge to $(0, 1)$. So in the limit of $h \to 1$ we get:

$$\partial_h \lambda_2 = -\partial J_{22} = -q\Delta\rho - qS_v < 0$$

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

$$\partial_h \lambda \mid_{h\to 0} = -\frac{(1-q)q(1-x-2\mu)\Delta x^2}{(1-x)^2}$$

that is positive if and only if $\mu > \frac{1-x}{2}$.

□

Proof of Lemma 1 (page 22)

Proof. Define the system of implicit equations:

$$\begin{cases} F_a &= x_a - k\rho_a + d \\ F_v &= x_v - k\rho_v \end{cases}$$

the jacobian is:

$$J_x F = \begin{pmatrix} 1 - k\partial_{x_a}\rho_a & -k\partial_{x_v}\rho_a \\ -k\partial_{x_a}\rho_v & 1 - k\partial_{x_v}\rho_v \end{pmatrix}$$

Thanks to the calculation in previous propositions we obtain that the diagonal is positive. Moreover, the determinant is:

$$detJF = 1 - k\partial_{x_a}\rho_a - k\partial_{x_v}\rho_v - k\partial_{x_a}\rho_a + k^2\partial_{x_v}\rho_v\partial_{x_a}\rho_a - k^2\partial_{x_v}\rho_a\partial_{x_a}\rho_v$$

now using the expressions found previously:

$$\partial_{x_v}\rho_v\partial_{x_a}\rho_a - \partial_{x_v}\rho_a\partial_{x_a}\rho_v = (ad-bc)\tilde{\rho}_a\tilde{\rho}_v > 0$$

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.

Moreover, if $x_a^* > x_v^*$ it follows from the initial Lemma that $\rho_a < \rho_v$ that implies $x_a^* < x_v^*$, which is a contradiction: hence $x_a^* < x_v^*$.

For the second part:

$$\partial_h x_a = -\frac{1}{detJF}\left(-(1-k\partial_{x_v}\rho_v)k\partial_h\rho_a - k^2\partial_{x_v}\rho_a\partial_h\rho_v\right) > 0 \qquad \text{(e)}$$

$$\partial_h x_v = -\frac{1}{detJF}\left(-(1-k\partial_{x_a}\rho_a)k\partial_h\rho_v - k^2\partial_{x_a}\rho_v\partial_h\rho_a\right) < 0 \qquad \text{(f)}$$

where the signs follow on known properties of the derivatives listed.

□

Proof of Proposition 5 (page 24)

Proof. The proof for $h \to 1$ is in the main text. When $h \to 0$, since again we have $\partial_{x_a}\rho \to -q$ and $\partial_{x_a}\rho \to -(1-q)$, using the expressions obtained in the Lemma, we can compute:

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

while we have:

$$\partial_h\rho = \frac{(1-q)q\Delta x^2\mu}{(1-x)^2}$$

from which the thesis follows. □

Proof of Proposition 6 (page 24)

Proof. The total derivative $d_{x_a}CI$ is:

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

taking into account the fact that also the steady state adjusts when vaccination rates change. Reordering, we have:

$$d_{x_a}CI\partial_h x_a + d_{x_v}CI\partial_h x_v =$$

$$= \partial_{x_a}CI\partial_h x_a + \partial_{x_v}CI\partial_h x_v + \partial_{\rho_a}CI(\partial_{x_a}\rho_a\partial_h x_a + \partial_{x_v}\rho_a\partial_h x_v) + \partial_{\rho_v}CI(\partial_{x_a}\rho_v\partial_h x_a + \partial_{x_v}\rho_v\partial_h x_v)$$

Now both if $h \to 0$ and $h \to 1$ we have $(\partial_{\rho_a}CI, \partial_{\rho_v}CI) \propto -(q, 1-q)$. It follows that (remembering $\rho = q\rho_a + (1-q)\rho_v$):

$$\partial_{\rho_a}CI(\partial_{x_a}\rho_a\partial_h x_a + \partial_{x_v}\rho_a\partial_h x_v) + \partial_{\rho_v}CI(\partial_{x_a}\rho_v\partial_h x_a + \partial_{x_v}\rho_v\partial_h x_v) \propto -\partial_{x_a}\rho\partial_h x_a - \partial_{x_v}\rho\partial_h x_v$$

Proposition 5 shows that both for $h \to 0$ and $h \to 1$ this term is $-\frac{k}{k+1}\partial_h\rho$. The only additional term to analyze is:

$$\partial_{x_a}CI\partial_h x_a + \partial_{x_v}CI\partial_h x_v$$

Now, using the expression for the cumulative infection it is a calculation to show:

$$\partial_{x_a}CI = \begin{cases} -q\frac{\mathrm{d}\rho_0}{(r+\mu+2\rho_a^{SS}+x_a-1)^2} & \text{if } h \to 1 \\ -q\frac{\mathrm{d}\rho_0}{(q(2\rho_a+x_a-2\rho_v-x_v)+r+\mu+2\rho_v+x_v-1)^2} & \text{if } h \to 0 \end{cases}$$

$$\partial_{x_v}CI = \begin{cases} -(1-q)\frac{d\rho_0}{(r+\mu+2\rho_v^{SS}+x_v-1)^2} & \text{if } h \to 1 \\ -(1-q)\frac{d\rho_0}{(q(2\rho_a+x_a-2\rho_v-x_v)+r+\mu+2\rho_v+x_v-1)^2} & \text{if } h \to 0 \end{cases}$$

which shows that $(\partial_{x_a}CI, \partial_{x_v}CI) \propto -(q, 1-q)$, similarly as with the derivatives with respect to ρ_a, ρ_v. With an analogous calculation this yields a term also proportional to $-\partial_h\rho$. □

Proof of Proposition A (page 30)

Proof. To analyze stability, we need to identify the values of parameters for which the Jacobian matrix of the system is negative definite when calculated in $(0, 0)$. The matrix is:

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

We can directly compute the eigenvalues, which are:

$$\begin{aligned} e_1 &= \hat{\mu} - \mu \\ e_2 &= \hat{\mu} - \mu - \Delta \end{aligned}$$

where $\hat{\mu} := \frac{1}{2}(T + \Delta) \in [0, 1]$, $T := \tilde{q}_a(1 - x_a) + \tilde{q}_v(1 - x_v)$, and $\Delta := \sqrt{T^2 - 4h(1 - x_a)(1 - x_v)}$.

The eigenvalues are real and distinct because, given $(x + y)^2 > 4xy$ whenever $x \neq y$, we get

$$\Delta^2 = T^2 - 4h(1 - x_a)(1 - x_v) \geq 4\tilde{q}_a(1 - x_a)\tilde{q}_v(1 - x_v) - 4h(1 - x_a)(1 - x_v)$$

Now $\tilde{q}_a\tilde{q}_v = h^2 + h(1 - h) + (1 - h)^2 q(1 - q) \geq h$, so we conclude $\Delta^2 > 0$.

Since eigenvalues are all distinct, the matrix is diagonalizable, and it is negative definite whenever the eigenvalues are negative. Inspecting the expression, this happens whenever $\mu > \hat{\mu}$. □

We are going to need the following lemma.

Lemma C. *Let $(d\rho_0^a, d\rho_0^v)$ be the infected share for each group at the outbreak.*

Then in the linearized approximation around the (0,0) steady state:

$$CI^a = \frac{2\left[d\rho_0^a\left(\mu-(1-x_v)\tilde{q}_v\right)+d\rho_0^v\left(1-x_a\right)\left(1-\tilde{q}_a\right)\right]}{(T-2\mu-\Delta)(T-2\mu+\Delta)}; \tag{g}$$

$$CI^v = \frac{2\left[\rho_0^a\left(1-x_v\right)\left(1-\tilde{q}_v\right)+\rho_0^v\left(\mu-(1-x_a)\tilde{q}_a\right)\right]}{(T-2\mu-\Delta)(T-2\mu+\Delta)}; \tag{h}$$

$$CI = \frac{2\left[d\rho_0^a\left(\mu+(1-x_v)(1-2\tilde{q}_v)\right)+d\rho_0^v\left(\mu+(1-x_a)(1-2\tilde{q}_a)\right)\right]}{(T-2\mu-\Delta)(T-2\mu+\Delta)}. \tag{i}$$

Proof. The linearized dynamics is:

$$\dot{d\rho}(t) = J\mathrm{d}\rho(t)$$
$$d\rho(0) = d\rho_0$$

where $d\rho_0 = (\rho_{0,a}, d\rho_{0,v})$, that is:

$$\dot{d\rho}(t) = M\mathrm{d}\rho(0)$$
$$d\rho(0) = d\rho_0, \quad M = e^{tJ}$$

and:

$$M_{11} = \frac{1}{\Delta}e^{\frac{1}{2}t(T-2\mu)}\left(\sinh\left(\frac{\Delta t}{2}\right)\left(-x_a\tilde{q}_a+\tilde{q}_a-\mu+\frac{1}{2}(2\mu-T)\right)+\frac{1}{2}\Delta\cosh\left(\frac{\Delta t}{2}\right)\right)$$
$$M_{12} = \frac{1}{\Delta}\left(1-x_a\right)\left(1-\tilde{q}_a\right)\sinh\left(\frac{\Delta t}{2}\right)e^{\frac{1}{2}t(T-2\mu)}$$
$$M_{21} = \frac{1}{\Delta}\left(1-x_v\right)\left(1-\tilde{q}_v\right)\sinh\left(\frac{\Delta t}{2}\right)e^{\frac{1}{2}t(T-2\mu)}$$
$$M_{22} = \frac{1}{\Delta}e^{\frac{1}{2}t(T-2\mu)}\left(\sinh\left(\frac{\Delta t}{2}\right)\left(-x_v\tilde{q}_v+\tilde{q}_v-\mu+\frac{1}{2}(2\mu-T)\right)+\frac{1}{2}\Delta\cosh\left(\frac{\Delta t}{2}\right)\right)$$

The cumulative infection in time in the two groups can be calculated analytically by integration, since it is just a sum of exponential terms. Integration

yield, for CI^v:

$$
\begin{aligned}
CI^v &= \int_0^\infty d\rho_v(t)dt \\
&= \frac{2\left(\rho_0^a\left(1-x_v\right)\left(1-\tilde{q}_v\right)+\rho_0^v\left(\mu-(1-x_a)\tilde{q}_a\right)\right)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)}+ \\
&\lim_{t->\infty} e^{\frac{1}{2}t(T-2\mu)}\left(2\Delta\cosh\left(\frac{\Delta t}{2}\right)\left(\rho_0^a\left(x_v-1\right)\left(\tilde{q}_v-1\right)+\rho_0^v\left(\left(1-x_v\right)\tilde{q}_v+\mu-T\right)\right)+\right. \\
&\left.\sinh\left(\frac{\Delta t}{2}\right)\left(\rho_0^v\left(\left(T-2\mu\right)\left(2\left(x_v-1\right)\tilde{q}_v+T\right)+\Delta^2\right)-2\rho_0^a(T-2\mu)\left(x_v-1\right)\left(\tilde{q}_v-1\right)\right)\right)
\end{aligned}
$$

and the limit is zero if $\mu > \hat{\mu}$ because the leading term is $Exp\left(\frac{1}{2}t(T-2\mu)+\frac{\Delta}{2}\right) = \hat{\mu}-\mu$. An analogous reasoning for CI^a yields:

$$
CI^a = \int_0^\infty d\rho_a(t)dt = \frac{2\left(d\rho_0^a\left(\mu-(1-x_v)\tilde{q}_v\right)+d\rho_0^v\left(1-x_a\right)\left(1-\tilde{q}_a\right)\right)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)} \qquad \text{(j)}
$$

$$
CI^v = \int_0^\infty d\rho_v(t)dt = \frac{2\left(\rho_0^a\left(1-x_v\right)\left(1-\tilde{q}_v\right)+\rho_0^v\left(\mu-(1-x_a)\tilde{q}_a\right)\right)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)} \qquad \text{(k)}
$$

The total CI in the population is $CI = q_a CI^a + (1-q_a)CI^v$

$$
\begin{aligned}
CI = \frac{2}{(-\Delta-2\mu+T)(\Delta-2\mu+T)}&\left(q_a\left(d\rho_0^a\left(\mu-(1-x_v)\tilde{q}_v\right)+d\rho_0^v\left(1-x_a\right)\left(1-\tilde{q}_a\right)\right)+\right. \\
&\left.(1-q_a)\left(d{\rho_0}^a\left(1-x_v\right)\left(1-\tilde{q}_v\right)+d{\rho_0}^v\left(\mu-(1-x_a)\tilde{q}_a\right)\right)\right) \\
&= d\rho_0^a\frac{2\left(q_a\left(\mu-(1-x_v)\tilde{q}_v\right)+(1-q_a)\left(1-x_v\right)\left(1-\tilde{q}_v\right)\right)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)}+ \\
&\quad d\rho_0^v\frac{2(q_a\left(1-x_a\right)\left(1-\tilde{q}_a\right)+(1-q_a)\left(\mu-(1-x_a)\tilde{q}_a\right))}{(-\Delta-2\mu+T)(\Delta-2\mu+T)}
\end{aligned}
$$

If $d\rho_0^a = d\rho_0^v = d\rho_0$ (the case considered in the main part of the paper):

$$
CI^a = d\rho_0\frac{2\left(\left(\mu-(1-x_v)\tilde{q}_v\right)+\left(1-x_a\right)\left(1-\tilde{q}_a\right)\right)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)} \qquad \text{(l)}
$$

$$
CI^v = d\rho_0\frac{2\left(\left(1-x_v\right)\left(1-\tilde{q}_v\right)+\left(\mu-(1-x_a)\tilde{q}_a\right)\right)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)} \qquad \text{(m)}
$$

$$
CI = 2d\rho_0\frac{\mu-(1-x_a)(\tilde{q}_a-q)-(1-x_v)(\tilde{q}_v-1+q)}{(-\Delta-2\mu+T)(\Delta-2\mu+T)} \qquad \text{(n)}
$$

We develop the calculations for generic $d\rho_{0,a}$ and $d\rho_{0,v}$.

First, note that $\mu > \hat{\mu}$ implies:

$$\begin{aligned}
\mu &> 1 - x_a > h(1 - x_a) \\
\mu &> 1 - x_v > h(1 - x_v) \\
\mu &> \frac{h(1 - x_a)}{1 - (1 - h)q} \\
\mu &> \frac{h(1 - x_v)}{1 - hq}
\end{aligned}$$

The expressions of the derivatives are:

$$\begin{aligned}
\frac{\partial CI^a}{\partial h} &= \frac{(q-1)(x_a-1)(\mu+x_v-1)(\rho_0^a(\mu+x_v-1)-\rho_0^v(x_a+\mu-1))}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2} \\
\frac{\partial CI^a}{\partial q_a} &= \frac{(h-1)(x_a-1)(h(x_v-1)+\mu)(\rho_0^a(\mu+x_v-1)-\rho_0^v(x_a+\mu-1))}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2} \\
\frac{\partial CI^a}{\partial x_a} &= \frac{((h-1)q(x_v-1)+\mu+x_v-1)(\mu(h(q-1)-q)(\rho_0^a-\rho_0^v)-h\rho_0^a(x_v-1)-\mu\rho_0^v)}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2} \\
\frac{\partial CI^a}{\partial x_v} &= \frac{(h-1)(q-1)(x_a-1)(\mu((h-1)q(\rho_0^v-\rho_0^a)+\rho_0^v)+h(x_a-1)\rho_0^v)}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2}
\end{aligned}$$

$$\begin{aligned}
\frac{\partial CI^v}{\partial h} &= \frac{q(x_v-1)(x_a+\mu-1)(\rho_0^a(\mu+x_v-1)-\rho_0^v(x_a+\mu-1))}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2} \\
\frac{\partial CI^v}{\partial q_a} &= \frac{(h-1)(x_v-1)(h(x_a-1)+\mu)(\rho_0^a(\mu+x_v-1)-\rho_0^v(x_a+\mu-1))}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2} \\
\frac{\partial CI^v}{\partial x_a} &= -\frac{(h-1)q(x_v-1)(h\rho_0^a(\mu+\mu(-q)+x_v-1)+\mu q\rho_0^a+(h-1)\mu(q-1)\rho_0^v)}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2} \\
\frac{\partial CI^v}{\partial x_v} &= \frac{(h(q-1)(x_a-1)+q(-x_a)-\mu+q)(\mu((h-1)q(\rho_0^v-\rho_0^a)+\rho_0^v)+h(x_a-1)\rho_0^v)}{2(h\mu(-qx_a+x_a+qx_v-1)+h(x_a-1)(x_v-1)+\mu(q(x_a-x_v)+\mu+x_v-1))^2}
\end{aligned}$$

and combining them, we get:

$$\frac{\partial CI}{\partial h} = \frac{\mu(q-1)q\left(x_a - x_v\right)\left(\rho_0^a\left(\mu + x_v - 1\right) - \rho_0^v\left(x_a + \mu - 1\right)\right)}{2\left(h\mu\left(-qx_a + x_a + qx_v - 1\right) + h\left(x_a - 1\right)\left(x_v - 1\right) + \mu\left(q\left(x_a - x_v\right) + \mu + x_v - 1\right)\right)^2}$$

$$\frac{\partial CI}{\partial q_a} = \frac{(h-1)\left(\rho_0^a\left(\mu + x_v - 1\right) - \rho_0^v\left(x_a + \mu - 1\right)\right)\left(h\left(x_a - 1\right)\left(x_v - 1\right) + \mu\left(q\left(x_a - x_v\right) + x_v - 1\right)\right)}{2\left(h\mu\left(-qx_a + x_a + qx_v - 1\right) + h\left(x_a - 1\right)\left(x_v - 1\right) + \mu\left(q\left(x_a - x_v\right) + \mu + x_v - 1\right)\right)^2}$$

$$\frac{\partial CI}{\partial x_a} = -\frac{q\left(h\left(x_v - 1\right) + \mu\right)\left(h\rho_0^a\left(\mu + \mu(-q) + x_v - 1\right) + \mu q\rho_0^a + (h-1)\mu(q-1)\rho_0^v\right)}{2\left(h\mu\left(-qx_a + x_a + qx_v - 1\right) + h\left(x_a - 1\right)\left(x_v - 1\right) + \mu\left(q\left(x_a - x_v\right) + \mu + x_v - 1\right)\right)^2}$$

$$\frac{\partial CI}{\partial x_v} = \frac{(q-1)\left(h\left(x_a - 1\right) + \mu\right)\left(\mu\left((h-1)q\left(\rho_0^v - \rho_0^a\right) + \rho_0^v\right) + h\left(x_a - 1\right)\rho_0^v\right)}{2\left(h\mu\left(-qx_a + x_a + qx_v - 1\right) + h\left(x_a - 1\right)\left(x_v - 1\right) + \mu\left(q\left(x_a - x_v\right) + \mu + x_v - 1\right)\right)^2}$$

Note that all the denominators are positive, so to control the sign from now on we focus on the numerators. In particular, if $d\rho_0^a = d\rho_0^v = d\rho_0$, we can note that CI is increasing in h and CI is increasing in q if and only if $x_v > x_a$.

If initial conditions are symmetric:

$$\frac{\partial CI^a}{\partial h} > 0 \Longleftrightarrow -(q-1)\rho_0^a\left(x_a - 1\right)\left(x_a - x_v\right)\left(\mu + x_v - 1\right) > 0$$

$$\frac{\partial CI^a}{\partial q_a} > 0 \Longleftrightarrow -(h-1)\rho_0^a\left(x_a - 1\right)\left(x_a - x_v\right)\left(h\left(x_v - 1\right) + \mu\right) > 0$$

$$\frac{\partial CI^a}{\partial x_a} > 0 \Longleftrightarrow -\rho_0^a\left(h\left(x_v - 1\right) + \mu\right)\left(\mu - (1-h)(1-q)\left(1 - x_v\right)\right) > 0$$

$$\frac{\partial CI^a}{\partial x_v} > 0 \Longleftrightarrow (h-1)(q-1)\rho_0^a\left(x_a - 1\right)\left(h\left(x_a - 1\right) + \mu\right) > 0$$

Now, using the first four inequalities presented above, we can conclude that $\frac{\partial CI^a}{\partial h} > 0$, $\frac{\partial CI^a}{\partial q_a} > 0$, $\frac{\partial CI^a}{\partial x_a} < 0$ and $\frac{\partial CI^a}{\partial x_v} < 0$. Similarly, if $\rho_{0,a} = 0$:

$$\frac{\partial CI^a}{\partial h} > 0 \Longleftrightarrow -(q-1)\left(x_a - 1\right)\rho_0^v\left(x_a + \mu - 1\right)\left(\mu + x_v - 1\right) > 0$$

$$\frac{\partial CI^a}{\partial q_a} > 0 \Longleftrightarrow -(h-1)\left(x_a - 1\right)\rho_0^v\left(x_a + \mu - 1\right)\left(h\left(x_v - 1\right) + \mu\right) > 0$$

$$\frac{\partial CI^a}{\partial x_a} > 0 \Longleftrightarrow -(1-h)(1-q)\rho_0^v\left(\mu - (1-q)(1-h)(1-x_v)\right) > 0$$

$$\frac{\partial CI^a}{\partial x_v} > 0 \Longleftrightarrow (h-1)(q-1)\left(x_a - 1\right)\rho_0^v\left(h\left(x_a - 1\right) + \mu\left((h-1)q + 1\right)\right) > 0$$

and we conclude that $\frac{\partial CI^a}{\partial h} < 0$, $\frac{\partial CI^a}{\partial q_a} < 0$, $\frac{\partial CI^a}{\partial x_a} < 0$ and $\frac{\partial CI^a}{\partial x_v} < 0$.

If $d\rho_{0,v} = 0$:

$$\frac{\partial CI^a}{\partial h} > 0 \Longleftrightarrow (q-1)\rho_0^a \left(x_a - 1\right)\left(\mu + x_v - 1\right)^2 > 0$$
$$\frac{\partial CI^a}{\partial q_a} > 0 \Longleftrightarrow (h-1)\rho_0^a \left(x_a - 1\right)\left(\mu + x_v - 1\right)\left(h\left(x_v - 1\right) + \mu\right) > 0$$
$$\frac{\partial CI^a}{\partial x_a} > 0 \Longleftrightarrow -\rho_0^a \left(\mu - (1-q)(1-h)(1-x_v)\right)\left(h\left(\mu - (1-x_v)\right) + \mu(1-h)q\right) > 0$$
$$\frac{\partial CI^a}{\partial x_v} > 0 \Longleftrightarrow -(h-1)^2\mu(q-1)q\rho_0^a \left(x_a - 1\right) > 0$$

and we conclude that $\frac{\partial CI^a}{\partial h} > 0$, $\frac{\partial CI^a}{\partial q_a} > 0$, $\frac{\partial CI^a}{\partial x_a} < 0$ and $\frac{\partial CI^a}{\partial x_v} < 0$.

The other cases are analogous. □

Proof of Proposition B

Proof. Using the derivatives computed in Proposition A, we find that the additional term due to the fact that vaccination rates adjust is:

$$\frac{\partial CI}{\partial x_v}\frac{\mathrm{d}x_v}{\mathrm{d}h} + \frac{\partial CI}{\partial x_a}\frac{\mathrm{d}x_a}{\mathrm{d}h} =$$
$$-\frac{d\rho_0 q(1-q)}{2(h\mu(-qx_a + qx_v + x_v - 1) + h(x_a - 1)(x_v - 1) + \mu(\mu + q(x_a - x_v) + x_v - 1))^2} \times$$
$$\frac{dk(1-q)q\left((\mu - h(1-x_v))^2 - (\mu - h(1-x_a))^2\right)}{(hk+1)^2}$$

which is negative because since $x_v > x_a$ we have:

$$\left((\mu - h(1-x_v))^2 - (\mu - h(1-x_a))^2\right) > 0$$

The total derivative instead is positive:

$$\begin{aligned}\frac{\mathrm{d}CI}{\mathrm{d}h} =& \frac{\partial CI}{\partial h} + \frac{\partial CI}{\partial x_v}\frac{\mathrm{d}x_v}{\mathrm{d}h} + \frac{\partial CI}{\partial x_a}\frac{\mathrm{d}x_a}{\mathrm{d}h} \\ =& \frac{d\rho_0 q(1-q)(x_v - x_a)^2(\mu(1-hk) + h^2k(2 - x_a - x_v))}{2(h\mu(-qx_a + qx_v + x_v - 1) + h(x_a - 1)(x_v - 1) + \mu(\mu + q(x_a - x_v) + x_v - 1))^2} > 0\end{aligned}$$

□

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