Demographic Dependence of Vaccine Adoption under Opinion Persuasion

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Abstract: Inspired by contagion models of social belief formation, we develop an *epistemically-informed* modeling framework, SIS-Vo, in which vaccine-related information propagates on a signed opinion network. Our model allows for heterogeneous treatment effects of policy messages across subpopulations through demographic-specific responses. We derive fixed-point characterizations of the healthy (disease-free) and endemic equilibria of this model, and obtain conditions for local stability of the healthy state in terms of the contact network and opinion-dependent vaccination capacities. Using numerical simulations, we illustrate how suitably targeted policy interventions, acting through opinion dynamics, can stabilize the epidemic process by moving the system towards the healthy regime. The SIS-Vo framework thus provides a natural basis for control-theoretic analysis of vaccination policies that remain robust even when misinformation targets specific subgroups.

Keywords: Social networks and opinion dynamics; SIS model; misinformation; vaccination; microtargeting

1. INTRODUCTION

Vaccination is one of the most effective tools for reducing the burden of infectious diseases, yet vaccine hesitancy continues to limit uptake. Recent epidemics have shown that vaccination decisions depend not only on biological and logistical factors, but also on heterogeneous beliefs and trust in institutions across demographic groups. Public health agencies increasingly deploy targeted communication campaigns, but it remains unclear how such interventions propagate through social networks and interact with epidemic dynamics.

Vaccine hesitancy and its impact on epidemic trajectories have been studied using compartmental models with capacity constraints and hesitancy-dependent uptake (Leung et al., 2022, 2023; Bhowmick and Selvaganesan, 2024). Related work incorporates media influence and information flows to capture how news and social media shape risk perception and behaviour (Mitchell and Ross, 2016).

More broadly, coupled epidemic—behaviour models analyse how disease spread is intertwined with awareness, opinion formation, and protective behaviour on networks (Funk et al., 2010; Wang et al., 2015). However, behavioural responses are often modeled as homogeneous functions of prevalence or media signals, and their dependence on social structure, demographics, and targeted policy messages is typically overlooked.

In this paper, we develop an epistemically-informed networked model that links opinion dynamics, vaccination uptake, and epidemic spread. Building on recent work on vaccine hesitancy (Leung et al., 2022, 2023; Bhowmick and Selvaganesan, 2024) and signed opinion dynamics (She et al., 2022), we propose a susceptible-infected-susceptible-vaccinated model with opinions (SIS-Vo) in which vaccination confers permanent protection. Each node represents a subpopulation characterized by a demographic vector. Opinions towards vaccination evolve over a signed interaction network induced by demographic homophily and are influenced by each subpopulation's current infection level, and in turn determine an opinion-dependent carrying capacity for vaccination. A policy message enters as a control input whose perceived impact on each subpopulation depends on its demo-

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graphics, inducing heterogeneous treatment effects across groups.

We derive fixed-point characterizations of the healthy (disease-free) and endemic equilibria of the SIS-Vo model and obtain a sufficient condition for local stability of the healthy state in terms of the contact network and the vaccination carrying capacities. Numerical simulations illustrate how targeted policy messages can shift opinions, alter local carrying capacities, and thereby influence the overall impact of the disease.

The paper is organized as follows. Section 2 presents the epistemic foundations, and Section 3 introduces the SIS-Vo model. Section 4 analyses the equilibria and healthy-state stability. Section 5 reports simulation results, and Section 6 concludes with a summary and directions for future work.

2. EPISTEMIC FOUNDATIONS OF DEMOGRAPHIC-WEIGHTED CONTROL

We develop an epistemically-informed modeling framework that

- (i) represents identity-structured opinion influence by letting social structure and homophily over demographic vectors mediate how groups affect one another's vaccine attitudes, connecting to Fricker (2007)'s account of epistemic injustice (inspired by Quaresmini et al. (2025); Villa et al. (2025)) and to sociological work on homophily (McPherson et al., 2001); and
- (ii) models epistemic dependence in the sense of Hardwig (1985) by allowing certain structurally central nodes in the opinion network to function as influential, and potentially misinforming, sources.

Concretely, each subpopulation i is associated with a demographic vector \mathbf{d}_i , and the signed opinion network has weights $\alpha_{ij} = \mathbf{d}_i^{\top} \mathbf{d}_j$, so that demographic similarity shapes both the sign and the strength of opinion influence. In our model, a policy message \mathbf{u} is a vector whose components correspond to different framings: demographic vectors \mathbf{d}_i encode group identities and determine both who interacts with whom and how strongly each group responds to each component of the control input \mathbf{u} , which can be interpreted as a form of opinion persuasion, via the term $\mathbf{u}^{\top}\mathbf{d}_i$ in the opinion dynamics. This structure generates heterogeneous responses to the same message across demographic groups and network neighbourhoods, and allows us to study how different framings of \mathbf{u} can be targeted to different audiences.

Following Longino (1990), we treat epistemic authority as socially organized: credibility heuristics and institutional structures are represented in the topology and weights of the opinion network and in the demographic weighting of messages. As in empirical work on cultural cognition and trust in science (Kahan et al., 2011; Gauchat, 2012; Sonmez et al., 2023; Sieghart, 2022; Eom et al., 2025), the impact of communication can thus vary systematically with both the communicator's social position and the audience's demographic and cultural profile.

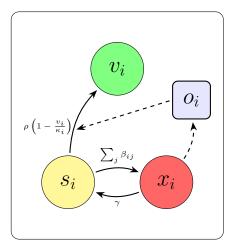


Fig. 1. Node-level epidemic and epistemic dynamics of the SIS-Vo model. The circle compartments represent the epidemic dynamics, while the square represents the opinion dynamics. As illustrated by the dashed arrows, the opinions influence the vaccination adoption and the infection severity influences the opinions.

3. MODEL FORMULATION

We propose a networked susceptible-infected-susceptible-vaccinated model with opinions (SIS-Vo). This model assumes that vaccinated individuals do not lose immunity. Figure 1 shows an overview of the dynamics of the model.

Each node of the system represents a subpopulation that shares some common traits (e.g., demographics and values). Our SIS-Vo model describes the evolution of the epidemics and vaccination, together with the evolution of opinions, within each subpopulation.

Epidemic level. The epidemic level is modeled as a SIS-V model (Leung et al., 2023) where each population i is composed of a susceptible share $s_i \geq 0$, a contaminated share $x_i \geq 0$, and a vaccinated share $v_i \geq 0$, such that $s_i + x_i + v_i = 1$. The infection level x_i is impacted by the current infection level of subpopulation i itself and by the infection levels of its neighbors in the contact network \mathcal{G}_c , whose weighted adjacency matrix is $B \in \mathbb{R}^{n \times n}$ is a nonnegative matrix. Inspired by Leung et al. (2022, 2023); Bhowmick and Selvaganesan (2024), the vaccination adoption is capped by the carrying capacity κ_i , which depends on the opinion of the subpopulation (see below). Altogether, the epidemic level is described by the following set of equations:

$$\dot{s}_i = \gamma_i x_i - s_i \left[\sum_j \beta_{ij} x_j + \rho \left(1 - \frac{v_i}{\kappa_i} \right) \right], \qquad (1)$$

$$\dot{x}_i = s_i \sum_j \beta_{ij} x_j - \gamma_i x_i \,, \tag{2}$$

$$\dot{v}_i = \rho \left(1 - \frac{v_i}{\kappa_i} \right) s_i \,, \tag{3}$$

where γ_i is the recovery rate of subpopulation i, β_{ij} is the (i, j)-th element of matrix B, and ρ is the vaccination rate.

Epistemic level. At the epistemic level, we borrow the opinion structure from She et al. (2022). We model the dynamics of a population's opinion towards the vaccine

by a signed consensus over an interaction network \mathcal{G}_i with weighted adjacency matrix $A \in \mathbb{R}^{n \times n}$ (distinct from B). We include an impact of the infection level of the population x_i , following the rationale that vaccine adoption will increase when faced with the reality of the disease. A population's opinion o_i evolves in the interval (-1/2, 1/2), ranging from opposition to the vaccine $(o_i < 0)$ and approbation $(o_i > 0)$. The opinion dynamics is described as

$$\dot{o}_i = (x_i - o_i - \frac{1}{2}) + \sum_j |\alpha_{ij}| (\operatorname{sign}(\alpha_{ij}) o_j - o_i), \quad (4)$$

where α_{ij} is the (i, j)-th term of matrix A, defined as $\alpha_{ij} = \boldsymbol{d}_i^{\top} \boldsymbol{d}_j$.

Epidemic-epistemic coupling. As seen in Eq. (4), the opinion o_i is influenced by the epidemics. We further couple the vaccination level v_i to the population's opinion o_i through the carrying capacity that we choose to define as

$$\kappa_i(o_i) = \max\left(v_i, \frac{1+2o_i}{1-2o_i}\right). \tag{5}$$

The max function in Eq. (5) ensures that \dot{v}_i in Eq. (3) remains nonnegative (we assume no loss of immunity after vaccination and vaccination levels should not decrease as a function of opinions) and that κ_i remains nonnegative as well.

Notice that our choice for dependence of κ_i on o_i is somewhat arbitrary. Our rationale is that we want κ_i to be monotonous in o_i , taking values between 0 (at $o_i = -1/2$) and 1 (at $o_i = 0$). Eq. (5) is a natural choice, but not the only one.

Demographics. In order to model the discrepancies in the demographics of the subpopulations, for each subpopulation i, we define a demographic vector $\mathbf{d}_i \in \mathbb{R}^k$ such that $\|\mathbf{d}_i\| = 1$, which encodes k distinct demographic characteristics of a population. This demographic vector allows us to naturally define the adjacency matrix of the opinion layer, A, building on the notion of homophily (McPherson et al., 2001). Indeed, it is expected that demographically similar groups will influence each other more positively. From this rationale, we define the interaction between subpopulations i and j as $\alpha_{ij} = \mathbf{d}_i^{\top} \mathbf{d}_j$.

Government intervention. Public health agencies have the ability to broadcast advertisement messages to the population as a whole. Often the same message will be perceived differently by different subpopulations. We model this phenomenon by defining the intervention as an input unit vector $\boldsymbol{u} \in \mathbb{R}^k$ whose impact on subpopulation i can be interpreted as an opinion persuasion attempt and is given by $\boldsymbol{u}^{\top}\boldsymbol{d}_i$. Therefore, public agencies have the freedom to tailor their message towards one subpopulation or another, depending on their objective. We adapt the opinion dynamics in Eq. (4) as

$$\dot{o}_i = (x_i - o_i - \frac{1}{2}) + \sum_j |\alpha_{ij}| (\operatorname{sign}(\alpha_{ij}) o_j - o_i) + \boldsymbol{u}^\top \boldsymbol{d}_i.$$

Notice that under this modification, there is no guarantee that the opinions will remain in (-1/2, 1/2). Nevertheless, the term $\boldsymbol{u}^{\top}\boldsymbol{d}_{i}$ only introduces a shift in the opinions, which remain bounded.

Summary. Since $s_i + x_i + v_i = 1$, one of the epidemic state equations is redundant and thus can be dropped. Therefore, the full SIS-Vo model is

$$\dot{x}_i = (1 - x_i - v_i) \sum_j \beta_{ij} x_j - \gamma_i x_i,$$
 (7)

$$\dot{v}_i = \rho \left(1 - \frac{v_i}{\kappa_i(o_i)} \right) (1 - x_i - v_i), \qquad (8)$$

$$\dot{o}_i = (x_i - o_i - \frac{1}{2}) + \sum_j (\alpha_{ij}o_j - |\alpha_{ij}|o_i) + \boldsymbol{u}^{\top}\boldsymbol{d}_i, \quad (9)$$

with $\kappa_i(o_i)$ given by Eq. (5), but for simplicity we drop the dependence of o_i moving forward.

4. MODEL ANALYSIS

To identify fixed points of the model, we set $\dot{x}_i = \dot{v}_i = \dot{o}_i = 0$. In particular, at the fixed point, $v_i^* = \min(1, \kappa_i^*)$ and x_i^* satisfies

$$x_i^* = (1 - v_i^*) \frac{\sum_j \beta_{ij} x_j^*}{\gamma_i + \sum_j \beta_{ij} x_j^*},$$
 (10)

similarly to the standard networked SIS model (Newman, 2010, Chap. 17). The carrying capacity κ_i^* is given by Eq. (5), where the steady opinion state o_i^* is given in matrix form by

$$o^* = (\tilde{A} + I)^{-1} \left(D^\top u - \frac{1}{2} + x^* \right),$$
 (11)

where

$$\tilde{A}_{ij} = \begin{cases} \sum_{j} |\alpha_{ij}| & \text{if } i = j \\ -\alpha_{ij} & \text{if } i \neq j, \end{cases}$$
 (12)

and D consists of the vectors d_i as columns.

Healthy state. The healthy state where $x_i^* = 0$ for all i always solves Eq. (10). In this case, the opinions o_i^* (and hence, κ_i^* and v_i^*) can be found in closed form from Eq. (11). The healthy state is then of the form

$$(s_i, x_i, v_i, o_i) = \begin{cases} \left(0, 0, 1, \frac{\kappa_i^* - 1}{2(\kappa_i^* + 1)}\right), & \text{if } \kappa_i^* \ge 1, \\ \left(1 - \kappa_i^*, 0, \kappa_i^*, \frac{\kappa_i^* - 1}{2(\kappa_i^* + 1)}\right), & \text{if } \kappa_i^* < 1. \end{cases}$$

$$(13)$$

Linearizing Eq. (2) around $\mathbf{x}^* = 0$ allows us to determine the epidemic threshold. The following lemma provides a sufficient condition for the stability of the healthy state.

Lemma 1. The spectrum of

$$M = (I - \operatorname{diag}(\kappa^*)) B - \operatorname{diag}(\gamma)$$
 (14)

is real and its largest eigenvalue is negative if, for all i,

$$\frac{\lambda_{\max}(B) - \gamma_{\min}}{\lambda_{\max}(B)} < \kappa_i^* < 1, \qquad (15)$$

where $\gamma_{\min} = \min_i \gamma_i$.

Proof. For the sake of readability, let us write $C = I - \text{diag}(\kappa^*)$. The matrix

$$\tilde{M} = C^{1/2}BC^{1/2} - \operatorname{diag}(\boldsymbol{\gamma}), \qquad (16)$$

is symmetric and similar to M. Then by Weyl's inequality (Horn and Johnson, 2012, Theorem 4.3.1),

$$\lambda_i(M) < \lambda_i(C^{1/2}BC^{1/2}) - \gamma_{\min}, \qquad (17)$$

and by similarity the eigenvalues of M are real.

Using the Rayleigh Quotient Theorem (Horn and Johnson, 2012, Theorem 4.2.2), for some unit vector \boldsymbol{v} ,

$$\tilde{\lambda}_1 = \boldsymbol{v}^{\top} C^{1/2} B C^{1/2} \boldsymbol{v} \le \lambda_{\max}(B) \cdot \|C^{1/2} \boldsymbol{v}\|^2 \le \lambda_{\max}(B) (1 - \kappa_{\min}^*),$$
(18)

where the last inequality holds since B is a nonnegative matrix and by the Perron-Frobenius Theorem (Horn and Johnson, 2012, Theorem 8.4.4), $\lambda_{\max}(B) \geq 0$. Therefore, considering the eigenvalues of M, under the assumption (15),

$$\lambda_1 \le \lambda_{\max}(B)(1 - \kappa_{\min}^*) - \gamma_{\min} < 0, \qquad (19)$$

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which concludes the proof.

We now have a sufficient condition for the stability of the healthy state.

Proposition 2. If, for all i,

$$\frac{\lambda_{\max}(B) - \gamma_{\min}}{\lambda_{\max}(B)} < \kappa_i^* < 1 \,,$$

then the healthy state for the SIS-Vo model, defined in Eq. (13), is stable.

Proof. Linearizing Eq. (2) in the healthy state, one sees that $x^* = 0$ is a stable fixed point for Eq. (2) if and only if the largest eigenvalue of M is nonpositive, i.e., if Eq. (15) is satisfied (Lemma 1).

In the disease-free state, o^* is computed in closed form with Eq. (11). This state is stable because all eigenvalues of $-\tilde{A} - I$ are nonpositive by Gershgorin's circle theorem (Horn and Johnson, 2012, Theorem 6.1.1) and any deviation in x_i vanishes due to the previous discussion.

In the disease-free state, the vaccinated share of a population v_i strictly increases as long as $v_i < \kappa_i$ and $v_i < 1$. Indeed, v_i cannot increase above 1 and if $v_i = 1$, then $\dot{v}_i = 0$. Furthermore, if $v_i = \kappa_i$, then $\dot{v}_i = 0$ and if $\kappa_i < v_i < 1$, then $\dot{v}_i < 0$. In summary, in the disease-free case $(\boldsymbol{x}^* = \boldsymbol{0}), v_i$ increases until $\min(\kappa_i^*, 1)$ and stops there. Then s_i^* necessarily take the value $1 - v_i^*$, which concludes the proof.

Note that Lemma 1 and Proposition 2 only hold when o < 0 and opinions towards vaccination are negative (see Eq. (5)). However, if $o_i > 0$, then $\kappa_i > 1$ and then v_i increases to 1, which is a disease-free state.

Endemic state. The set of coupled equations (10) and (11) can be solved numerically. An endemic state is found if there exists a solution with $x_i \neq 0$ for some i. The endemic equilibrium is of the form

$$(s_i, x_i, v_i, o_i) = \left(1 - v_i^* - x_i^*, x_i^*, v_i^*, \frac{\kappa_i^* - 1}{2(\kappa_i^* + 1)}\right), \quad (20)$$

where $v_i^* = \min(1, \kappa_i^*)$. Naturally, when $\kappa_i^* \geq 1$ (i.e., $o_i^* \geq 0$), Eq. (10) tells us that $x_i^* = 0$. Hence, the disease becomes endemic in subpopulations that are negatively opinionated towards vaccination, as illustrated in the simulations (see Fig. 3).

Intervention. There are various natural ways to quantify the effectiveness of an intervention u. Of course, the ideal outcome is the healthy state. However, as seen in Fig. 4, the endemic state may persist and the intervention has a strong impact on the infection levels.

Intuitively, one may want to minimize the total number of infections in the endemic state, namely, minimizing the 1-norm of x^* . Considering that different subpopulations may be composed of significantly different number of individuals, one may want to minimize the weighted norm

$$\|\boldsymbol{x}^*\|_{N,1} = \|\operatorname{diag}(N_1, ..., N_k) \cdot \boldsymbol{x}^*\|_1,$$
 (21)

where N_i is the number of individuals composing subpopulation *i*. Notice that the vulnerabilities of different subpopulations can also easily be incorporated in the minimization by weighting each infection level x_i .

Considering the nonlinear relationship between the different variables at steady state, we do not expect to find a closed form solution for infection minimization. Nevertheless, we can formulate the following optimization problem

minimize
$$\|x^*\|_{N,1}$$

s.t. Eqs. (5), (10), (11). (22)

An intervention is said to be aligned with the demographics of a subpopulation when it points in the same direction as the demographic vector, i.e., $\boldsymbol{u}^{\top}\boldsymbol{d}_{i} > 0$. In this case, the message resonates with the cultural and social profile of that subpopulation, producing a positive drift in the opinion dynamics described by Eq. (6). This synergy leads to larger carrying capacities κ_{i} , higher vaccination levels, and reduced infection prevalence in the endemic equilibrium. Conversely, an anti-aligned intervention sets \boldsymbol{u} in the opposite direction of the demographic vector, so that $\boldsymbol{u}^{\top}\boldsymbol{d}_{i} < 0$. Such a message is poorly received in the subpopulation, shifting opinions accordingly. Fig. 4 illustrates these effects.

5. SIMULATION RESULTS

We illustrate the endemic behavior of the SIS-Vo model with 5 nodes in Fig. 2. Note that for the chosen parameters (including an arbitrary choice of \boldsymbol{u}) and randomly sampled demographic vectors \boldsymbol{d}_i , all but one of the nodes reach an endemic state. The only node that eradicates the virus is the one with a positive opinion state, which results in the whole subpopulation being vaccinated.

To confirm that the numerical solution of (10)-(11) does indeed converge to the steady state solution, we simulate (2)-(4) on a large random network of 200 nodes over 10000 timesteps with dt=0.2 and k=4, plot the simulated steady states for x_i versus o_i , and compare with the result of 1000 steps of fixed-point iteration on (10)-(11) (Fig. 3). Observe that agreement between simulated data and the theoretical steady states is very good, with only a small number of nodes not aligning due to slow convergence of the simulations towards the steady state.

Now using (10)-(11) to explore the effect of different interventions \boldsymbol{u} on the steady state infection proportion x_i^* via opinions o_i^* and demographics \boldsymbol{d}_i , we consider three different types of interventions:

- (i) an intervention that is positively-aligned with demographics $u_i = \operatorname{sign}(\sum d_i)\eta_i$ where $\eta_i \sim U(0,2)$,
- (ii) an anti-aligned intervention $u_i = -\operatorname{sign}(\sum d_i)\eta_i$, and
- (iii) a random intervention $u_i = \zeta_i$ where $\zeta_i \sim U(-2, 2)$.

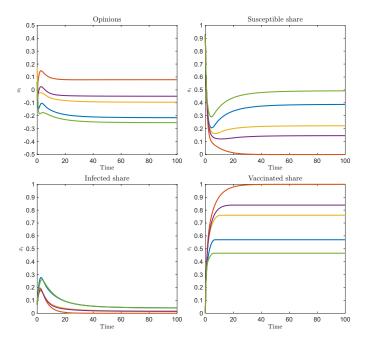


Fig. 2. Example dynamics of the SIS-Vo model with 5 nodes. We see here an endemic state where one community (red) reaches a vanishing infection proportion and a complete vaccination, while other communities reach an endemic situation where the disease survives in the subpopulation. The choice of parameters is $\gamma = 0.5$, $\rho = 0.8$, a symmetric matrix with zero diagonal and off-diagonal entries between 0 and 1, $[\beta_{ij}]$, randomly sampled vectors \mathbf{d}_i (with both positive and negative entries) and an arbitrarily chosen $\mathbf{u} = [0.6, 0.2, 0.4]^{\top}$.

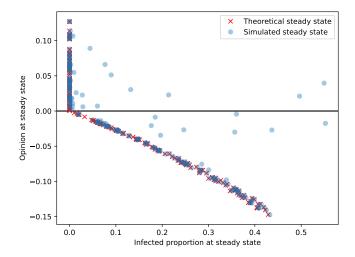


Fig. 3. Steady state proportions x_i versus o_i for a large network (n = 200). The theoretical infection proportions (red crosses) are obtained by numerically solving Eq. (10). In the numerical simulation, the infection proportions converge to the blue dots. We observe a very good agreement between the theory and the simulations even though, due to slow convergence, some simulated infection proportions remain too high.

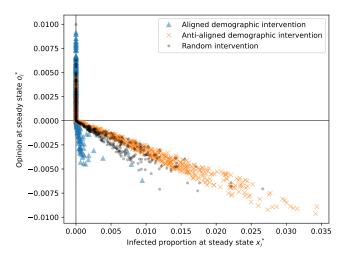


Fig. 4. Opinion o_i compared to the infection proportions x_i under three intervention strategies u. The blue triangles show the outcome of when the intervention u is aligned with the demographics d_i . The orange crosses show the outcome when the intervention is anti-aligned with the demographics vectors. The random intervention (black dots) gives an outcome that is closer to the anti-aligned strategy than to the aligned one, suggesting a strong impact of the customization of the government's strategy.

Note that these definitions do not lead to perfectly-aligned/anti-aligned interventions; however, numerically it leads to over 80% satisfactory cases.

Fig. 4 shows the results where $n=500,\,k=1000,\,$ and all other parameters as for Fig. 3. Observe that the anti-aligned intervention (orange crosses) leads to far more endemic steady states than the aligned intervention (blue triangles), and a stronger dependence of steady state infected proportion on opinions, with the random intervention (black dots) falling between the two.

Interestingly, further simulations (not shown here) confirmed that Prop. 2 is only a sufficient condition, not necessary. Indeed, we were able to find some systems where the healthy state is stable, whereas Eq. (15) is not satisfied.

6. CONCLUSIONS

In order to model vaccine adoption in a population under general media government intervention, we propose a combination of established models of opinion dynamics and epidemic spreading. Opinions and disease are connected through (i) the infection level that impacts a population's opinion towards the disease, and (ii) the carrying capacity that caps the vaccination and directly depends on the population's opinion towards the disease. Public health intervention comes in as a demographic-dependent drift in the opinion dynamics.

As preliminary results, we provide a sufficient condition for the stability of the healthy state (Prop. 2). We further explore numerically how the opinion persuasion impacts the infections in the endemic state. Namely, when the intervention is designed to align with the demographics' reactions, we observe a drastic reduction of the infected

proportion compared to the worst-case scenario (antialigned intervention). Interestingly, random interventions appear to perform slightly better than the worst case, but still quite poorly when compared to the aligned intervention.

For future work, we envision comparing different functional relationships between the opinion o_i and the carrying capacity κ_i . Our current choice, Eq. (5) is arbitrary and it remains unclear if our results are robust against a change in this relationship.

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