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Leveraging Opinions and Vaccination to Eradicate Networked Epidemics

Humphrey C. H. Leung, Zhuocong Li, Baike She, and Philip E. Paré

***Abstract*— We introduce a multi-layer networked compartmental $SIRS - V_o$ model that captures opinion dynamics, disease spread, risk perception, and self-interest vaccine-uptake behavior in an epidemic process. We characterize the target vaccination criterion of the proposed model and conditions that guarantee the criterion is obtainable by influencing opinions on disease prevalence. We leverage this result to design an eradication strategy that leverages opinions and vaccination. Through numerical simulations, we show that the proposed eradication strategy is able to stabilize the epidemic process around a healthy state equilibrium, and the outbreak rebounds after the control signal is relaxed.**

I. Introduction

There has been a long history of studying epidemic mitigation and eradication problems [2]. Various approaches, including network-structured compartmental models [3]–[5], multiplex/multi-layer epidemic spreading networks [6], [7], and game theoretic modeling [8]–[10], have been used to study epidemic spreading over networks. This paper integrates factors including human behavior [11], opinions [12]–[14], and vaccination [15] into epidemic spreading models.

In this work, we incorporate opinion dynamics and imitation dynamics over networks to construct a novel multi-layer networked epidemic model that captures the aforementioned characteristics of epidemic spreading processes. The main results and contributions of this work are:

1) we propose a novel multi-layer network model to connect the opinions towards the disease prevalence, risk perception on the infection, and self-interest vaccination behavior on networked epidemic processes;
2) we show the existence of the healthy state equilibrium and that it is locally stable;
3) we characterize conditions on vaccination levels which ensure disease eradication; and
4) we propose a control strategy for disease eradication through opinions.

While speech censorship, propaganda, and opinion control are characteristics of malfunctioning dystopian states, we hope that through this work we can open up discussions on the role and limitations that opinions play in epidemic control from an analytical and computational perspective.

We organize the paper as follows: in Section II, we introduce our networked $SIRS-V_o$ model with opinion and imitation dynamics and formulate the problem of interest. In Section III, we present the stability analysis of our model and develop a control strategy. Section IV demonstrates the numerical results of our work via simulation. Section V concludes the paper and discusses potential future researches.

Humphrey C. H. Leung, Zhuocong Li, and Philip E. Paré are with the School of Electrical and Computer Engineering at Purdue University. Email: {leung61, li3975, philpare}@purdue.edu. Baike She is affiliated with the Department of Mechanical and Aerospace Engineering at University of Florida. Email: shebaike@ufl.edu. This work is partially supported by the National Science Foundation, grant NSF-ECCS #2032258. The full version of the manuscript [1] is available at: http://arxiv.org/abs/2304.12516.

Notation

Let $[n]$ denote the index set $\{1, 2, ..., n\}$, $\forall\ n \in \mathbb{Z}_{>0}$. We view vectors as column vectors and write $x^\top$ to denote the transpose of a column vector x. We use x_i to denote the ith entry of a vector x. For any matrix $M \in \mathbb{R}^{n\times n}$, we use $[M]_{i,:}$, $[M]_{:,j}$, and $[M]_{ij}$ to denote its ith row, jth column, and ijth entry, respectively. Similarly, we denote $[a_{ij}]_{i,j\in[n]}$ as the $n \times n$ matrix for any $n \times n$ 2-dimensional array a_{ij}. We use $\widetilde{M} = diag\{m_1, ..., m_n\}$ to represent a diagonal matrix $\widetilde{M} \in \mathbb{R}^{n\times n}$ with $[M]_{ii} = m_i,\ \forall i \in [n]$. We use 0_n and 1_n to denote the vectors whose entries all equal 0 and 1, respectively, and I_n to denote the $n \times n$ identity matrix. For a real square matrix M, we use $\alpha(M)$, $\rho(M)$, and $\sigma(M)$ to denote the spectral abscissa (the largest real part among its eigenvalues), spectral radius, and singular value of M, respectively. For any two vectors $v, w \in \mathbb{R}^n$, we write $v \geq w$ if $v_i \geq w_i$, $v > w$ if $v \geq w$ and $v \neq w$, $v \gg w$ when $v_i > w_i$, $\forall i, j \in [n]$. The comparison notations between vectors are used for matrices as well, for instance, for $A, B \in \mathbb{R}^{n\times n}$, $A > B$ indicates that $A_{ij} > B_{ij}$, $\forall i, j \in [n]$. Consider a directed graph $\mathcal{G} = (\mathcal{V}, \mathcal{E})$, with the node set $\mathcal{V} = \{v_1, ..., v_n\}$ and the edge set $\mathcal{E} \subseteq \mathcal{V} \times \mathcal{V}$. Let matrix $A \in \mathbb{R}^{n\times n}$, $[A]_{ij} = a_{ij}$, denote the adjacency matrix of $\mathcal{G} = (\mathcal{V}, \mathcal{E})$, where $a_{ij} \in \mathbb{R}^+$ if $(v_j, v_i) \in \mathcal{E}$ and $a_{ij} = 0$ otherwise, $\forall i \in [n]$. Let $k_i[A] = \sum_{j\in\mathcal{N}_i} a_{ij}$, where $\mathcal{N}_i = \{v_j : (v_i, v_j) \in \mathcal{E}\}$ denotes the neighbor set of v_i. The graph Laplacian of $\mathcal{G}$, whose adjacency matrix is A, is defined as $L[A] \triangleq \widetilde{K}[A] - A$, where $\widetilde{K}[A] \triangleq diag\{k_1[A], ..., k_n[A]\}$.

II. Model And Problem Formulation

In this section, we first introduce a networked ($SIRS-V_o$) model, which integrates the classic networked susceptible-infected-recovered-susceptible ($SIRS$) model with vaccinations (V) and opinion (o). The goal is to incorporate three interacting networks in our compartmental model to capture the potential impact of media on a disease spreading process through the human decision making process. The three interacting networks include a disease transition network that captures the disease spreading through physical interactions; an opinion spreading network that captures agents' perception of the current state of the prevalence of the disease; and a strategy substitution network that describes the dynamics

of agents switching their vaccination strategies to substitute their neighbors' decision of not taking vaccine.

We first consider an epidemic spreading over a network and use a weighted directed graph $\mathcal{G} = (\mathcal{V}, \mathcal{E})$ to capture the physical transmission network. $\mathcal{V} = \{v_1, ..., v_n\}$ represents the set of n nodes and the edge set $\mathcal{E} \subseteq \mathcal{V} \times \mathcal{V}$ represents the disease transmission channels over $\mathcal{V}$. Each edge is weighted by $\beta_{ij} \in \mathbb{R}_{\geq 0}$, which denotes the infection rate from host j to i. Let $x_i(t) \in [0, 1]$, $\forall i \in [n]$, $t \geq t_0$, denote the probability that node i is infected at time t.

We define the network of the opinions of the agents toward the prevalence of the epidemic spread as a directed graph $\bar{\mathcal{G}} = (\mathcal{V}, \bar{\mathcal{E}})$, where $\bar{\mathcal{E}} \subseteq \mathcal{V} \times \mathcal{V}$ represents the opinion exchange over $\mathcal{V}$. Each edge is weighted by $a_{ij} \in \mathbb{R}^+$, denoting the impact of node j on node i in terms of opinions. Let $o_i(t) \in [0, 1]$, $\forall i \in [n]$ at time $t \geq t_0$, denote node i's belief in the seriousness of the epidemic. A higher $o_i(t)$ means that node i considers the epidemic to be more serious, and vice versa. Further, we assume, for node i, that a higher infection level in their neighborhood ($x_j(t)$) will raise their belief in the seriousness of the epidemic.

Lastly, we denote the strategy imitation network as a weighted directed graph $\hat{\mathcal{G}} = (\mathcal{V}, \hat{\mathcal{E}})$, where the edge set $\hat{\mathcal{E}} \subseteq \mathcal{V} \times \mathcal{V}$ represents the set of pairs of agents (i, j) where agent i imitates agent j's vaccine-uptake strategy with a non-zero probability. Furthermore, we assume in this model that each agent i randomly samples another agent j according to some distribution p_{ij}; if the strategy of the sampled agent provides a higher payoff, then the agent will switch to the new strategy with a probability proportional to the expected payoff gain [16], [17].

The weight of each directed edge (v_j, v_i) is $\eta_{ij}(t)$, and it is defined in the following way. Let u_i^v be the probability of serious vaccine side effects, such as allergic reactions, after vaccine-uptake; u_i^x be the probability of significant morbidity after infection of agent i; and p_{ij} be the probability of agent i's decision being influenced by the decision of agent j and $[p_{ij}]_{i,j\in[n]}$ is a row stochastic matrix. Let m_i^v and m_i^x represent the severity of serious vaccine side effects and significant morbidity after infection, respectively, and c be the government compensation for serious vaccine side effects such that $c > m_i^v u_i^v$ for all $i \in [n]$. Then the payoff of getting vaccinated is $c - m_i^v u_i^v$, and the *perceived payoff* of i not getting vaccinated is $-o_i(t)(m_i^x u_i^x)$, where $o_i(t)$ is the perceived disease prevalence of node i at time t. Note that c can be interpreted as the sum of the expected payoff of vaccine-uptake and the expected cost of remaining unprotected that is not captured by $m_i^x u_i^x$ and $m_i^v u_i^v$, such as the inconvenience of mask wearing. The above interpretation only suggests one scenario to interpret these parameters. The above *non-vaccine-uptake* payoff formulation assumes that each agent determines its payoff evaluation on the current perceived disease prevalence. We further assume that the vaccine has perfect efficacy; an agent takes the vaccine once it switches its strategy to *vaccine-uptake*; and the agent cannot switch back except through the loss of immunity. Therefore, the *perceived payoff gain* for agent i at time t is $g_i(t) = o_i(t)(m_i^x u_i^x) + (c - m_i^v u_i^v)$.

Finally, let μ_i be the sampling rate of agent i and q be an arbitrary constant. Then, we define the *strategy transition matrix* $H(o(t)) \in \mathbb{R}^{n\times n}$ such that $[H(o(t))]_{ij} = \eta_{ij}(t) = q\mu_i g_i(t) p_{ij}(t)$. We denote $\eta_{ij}^{min} = q\mu_i(c - m_i^v u_i^v)p_{ij}$, $\Delta\eta_{ij} = q\mu_i(m_i^x u_i^x)p_{ij}$, and $\eta_{ij}(t) = o_i(t)\Delta\eta_{ij} + \eta_{ij}^{min}$. We now formally introduce the networked $(SIRS - V_o)$ model:

$$\dot{o}_i(t) = \sum_{j\in\mathcal{N}_i} a_{ij}(o_j(t) - o_i(t)) + a_{ij}(x_j(t) - o_i(t)), \tag{1a}$$

$$\dot{s}_i(t) = \delta_i v_i(t) - s_i(t) \sum_{j\in\mathcal{N}_i} [\beta_{ij} x_j(t) + \eta_{ij}(t) v_j(t)] + \omega_i r_i(t), \tag{1b}$$

$$\dot{x}_i(t) = s_i(t) \sum_{j\in\mathcal{N}_i} \beta_{ij} x_j(t) - \gamma_i x_i(t), \tag{1c}$$

$$\dot{r}_i(t) = \gamma_i x_i(t) - \omega_i r_i(t) - r_i(t) \sum_{j\in\mathcal{N}_i} \eta_{ij}(t) v_j(t), \tag{1d}$$

$$\dot{v}_i(t) = (s_i(t) + r_i(t)) \sum_{j\in\mathcal{N}_i} \eta_{ij}(t) v_j(t) - \delta_i v_i(t), \tag{1e}$$

where s_i, x_i, r_i, and v_i represent the percentage of susceptible, infected, removed, and vaccinated populations at node i. Note that $\eta_{ij}(t) = ((1 - o_i(t))\eta_{ij} + o_i(t)\eta_{ij}^{\min})$ is a convex combination of η_{ij} and $\eta_{ij}^{\min}$. To further simplify the model, we express (1) in a compact form:

$$\dot{o}(t) = A(x(t) - o(t)) - 2L[A]o(t), \tag{2a}$$

$$\dot{s}(t) = \widetilde{D}v(t) - \widetilde{S}(t)(Bx(t) + H(o(t))v(t) + \widetilde{W}r(t), \tag{2b}$$

$$\dot{x}(t) = \widetilde{S}(t)Bx(t) - \widetilde{G}x(t), \tag{2c}$$

$$\dot{r}(t) = \widetilde{G}x(t) - \widetilde{W}r(t) - \widetilde{R}(t)H(o(t))v(t), \tag{2d}$$

$$\dot{v}(t) = (\widetilde{S}(t) + \widetilde{R}(t))H(o(t))v(t) - \widetilde{D}v(t), \tag{2e}$$

where $\widetilde{S}(t) = diag(s(t))$, $\widetilde{R}(t) = diag(r(t))$, and $L[A]$ is the Laplacian matrix of the opinion spreading graph $\bar{\mathcal{G}}$. $\widetilde{G}$ and $\widetilde{D}$ are diagonal matrices, with $[\widetilde{G}]_{ii} = \gamma_i$ and $[\widetilde{D}]_{ii} = \delta_i$, $\forall i \in [n]$. We define $H(o(t))$ as a function of $o(t)$, where $H(o(t)) = \widetilde{O}(t)\Delta H + H_{\min}$, and $[H_{\min}]_{ij} = \eta_{ij}^{\min}$ if $\eta_{ij} > 0$, otherwise, $[H_{\min}]_{ij} = 0$.

To investigate the potential impact of media on epidemic spreading through human decision processes, we introduce the media actuator $m(t) = [m_i(t)]_{i=1...n}$ and augment (2a) by including the media nodes set:

$$\dot{o}(t) = A(x(t) - o(t)) - 2L[A]o(t) + \widetilde{M}(t)(1 - o(t)), \tag{3}$$

where $\widetilde{M}(t) = diag(M(t))$. The problems that we will answer in this paper include:

1) Show the existence and uniqueness of a healthy state equilibrium of system (2) and the local stability condition of its infection subsystem.
2) Construct an eradication algorithm stabilizing 2c around $x = 0_n$ through the control of a media actuator $g(t)$, and provide the assumptions and conditions that guarantee the validity of the algorithm.

Solutions to these problems will be provided in Section III-A and Section III-B, respectively.

III. Main Results

This section explores the stability conditions, the properties of the opinion dynamics, and the strategies to mitigate

the networked epidemic process in (2). The results are developed under the following assumptions.

Assumption 1: All parameters are real and non-negative.

Assumption 2: The initial conditions at t_0 should obey $(o_i(t_0), s_i(t_0), x_i(t_0), r_i(t_0), v_i(t_0)) \in [0,1]$ and $s_i(t_0) + x_i(t_0) + r_i(t_0) + v_i(t_0) = 1,\ \forall i \in [n]$.

Assumption 3: We assume that the opinion spreading graph $\bar{\mathcal{G}}$, disease transmission graph $\mathcal{G}$, and strategy imitation graph $\hat{\mathcal{G}}$ are strongly connected.

First, one can show that the model in (2) is well defined by checking the derivative of a state being always pointing inside the set $[0,1]$, when the state hits the boundary of $[0,1]$. Thus, we omit the proof by giving the following statements.

Lemma 1: For all $t > t_0$ and $i \in [n]$, $s_i(t) + x_i(t) + r_i(t) + v_i(t) = 1$ and $(s_i(t),\ x_i(t),\ r_i(t),\ v_i(t)) \in [0,1]$.

Lemma 2: If $o(t_0) \in [0,1]^n$, $o_i(t)$ varies in the range of [0,1], $\forall i \in [n]$ and $t > t_0$.

Proposition 1: Without the loss of vaccine immunity, the vaccinated population monotonically increases.

Let $(o^*, s^*, x^*, r^*, v^*)$ denote the equilibria of the $SIRS{-}V_o$ model. We start to analyze the equilibria of the $SIRS - V_o$ model through the following definition.

Definition 1: (Healthy State Equilibrium). *A healthy state equilibrium* is an equilibrium with the steady-state vector of infected populations $x^* = 0_n$, where $\dot{s}^* = \dot{x}^* = \dot{r}^* = \dot{v}^* = \dot{o}^* = 0_n$.

A. Healthy State Equilibrium

In this subsection, we prove that the healthy state equilibrium of the coupled networked system in (2) exists and derive the stability condition of the infection subsystem around any healthy state equilibrium. First, we notice the following features of our model:

Lemma 3: At any healthy state equilibrium, where $x^* = 0_n$, the recovered population $r^* = 0_n$. Furthermore, $s^* + v^* = 1_n$.

Lemma 4: At any healthy state equilibrium, where $x^* = 0_n$, the opinions will reach consensus at $o_i^* = 0$, $\forall i \in [n]$.

The above lemmas outline some important characteristics about the healthy state equilibrium when it exists, which will be useful when proving the following proposition.

Proposition 2: If $\alpha(-D + H_{min}) \leq 0$, then $(0_n, 1_n, 0_n, 0_n, 0_n)$ is the unique healthy state equilibrium of system (2). If $\alpha(-D + H_{min}) > 0$, then system (2) has two healthy state equilibria, $(0_n, 1_n, 0_n, 0_n, 0_n)$ and $(0_n, 1_n - v^*, 0_n, 0_n, v^*)$, where $v^* \gg 0_n$.

Proposition 2 implies that the networked model in (2) has a unique healthy equilibrium when $\alpha(-D + H_{\min}) < 0$, where the susceptible and vaccinated population within the community will reach a balance at a unique ratio, which is determined by the model parameters.

Lemma 5: The infection subsystem (2c) is locally asymptotically stable around $x^* = 0$, if $\rho(\widetilde{G}^{-1}\widetilde{S}^* B) < 1$.

Since $S^* \leq 1_n$, $\rho(\widetilde{G}^{-1}B) < 1$ implies $\rho(\widetilde{G}^{-1}\widetilde{S}^* B) < 1$ and in turn implies local asymptotic stability of the infection subsystem (2c). We define $\mathcal{R}_0 = \rho(\widetilde{G}^{-1}B)$ as the *basic reproduction number* and $\mathcal{R}_t = \rho(\widetilde{G}^{-1}\widetilde{S}(t)B)$ as the *effective reproduction number* of the subsystem (2c).

B. Control Strategy

In this section, we consider a fixed opinion media actuator, which connects to each node i in the opinion network with strength $m_i(t) \in [0, \infty)$. Equation (1a) can be rewritten as:

$$\dot{o}_i(t) = m_i(t)(1 - o_i(t)) + \sum_{j \in \mathcal{N}_i} a_{ij}(o_j(t) - o_i(t)) + a_{ij}(x_j(t) - o_i(t))$$

to include the influence of the actuator, and the corresponding matrix form of the system with the actuator input is:

$$\dot{o}(t) = A(x(t) - o(t)) - 2L[A]o(t) + \widetilde{M}(t)(1_n - o(t)), \quad (4)$$

where $\widetilde{M}(t) = diag(m(t))$.

Intuitively, if $m_i(t)$ are maintained on a sufficiently high level, then the epidemic will eventually die out because individuals will be motivated to take the vaccination due to their perception of the high disease prevalence. The main goal of this section is to derive a lower bound on $m(t)$ that is sufficient to guarantee the local stability of the infection subsystem (2c) around the healthy state equilibrium. To achieve this goal, we will introduce two concepts which we will formally characterize for system (2) with the media actuator in (4) later in the section. They are the *target vaccination criterion* and the *target opinion criterion*:

Definition 2 (Target Vaccination Criterion): We call v_c a target vaccination criterion, if for every $v(t)$ that satisfies the target vaccination criterion v_c, that is, $v_i(t) > v_c$ for all $i \in [n]$ and $t \geq T$ for some $T \in \mathbb{R}_{\geq 0}$, the healthy state equilibrium is locally asymptotically stable for the corresponding infection subsystem (2c).

In other words, if the vaccination levels of all n nodes stay above the target vaccination criterion, v_c, when time t is sufficiently large, then the epidemic will die out. Furthermore, since the eradication solution is trivial if $v_c \leq 0$ and infeasible if $v_c = 1$, only the case $v_c \in (0,1)$ is considered.

Definition 3 (Target Opinion Criterion): We define $\underline{o}_i^{v_c}$ as a target opinion criterion with respect to v_c if $v_i(t) > v_c$ when $o_i(t) \geq \underline{o}_i^{v_c}$ for all $i \in [n]$ and $t \geq T$, where $T \geq 0$.

The target opinion criterion is a lower bound on the opinion that guarantees the vaccination level at node i to remain above the target vaccination criterion. The control criterion $\underline{m}^{v_c}$ will be expressed as a lower bound of $m(t)$ in terms of the target opinion criterion $\underline{o}_i^{v_c}$ such that the vaccination level at each node $v_i(t)$ remains above v_c despite loss of immunity. Before introducing the key results, we make the following assumption for the control strategy.

Assumption 4: The infection transmission matrix B is symmetric, i.e. $B = B^\top$.

The following theorem characterizes the target vaccination criterion of (2).

Theorem 1: Under Assumptions 3 and 4,

$$v_c = 1 - \frac{1}{\rho(\widetilde{G}^{-1})\rho(B)} \quad (5)$$

is a target vaccination criterion.

Proof: Recall that under Assumption 3, B is strongly connected. Since B is non-negative and irreducible, $\rho(B) > 0$, by the Perron–Frobenius theorem [18]. Therefore, the inverse of $\rho(B) \in \mathbb{R}_{>0}$ exists and $1 - \frac{1}{\rho(\widetilde{G}^{-1})\rho(B)}$ is well-defined.

After showing that v_c is well-defined, we will show that for any arbitrary $v(t)$ satisfying the target vaccination criterion, the healthy state equilibrium is locally asymptotically stable for the corresponding infection subsystem (2c), consistent with Definition 2. Consider an arbitrary $v(t)$ such that for all $i \in [n]$:

$$v_i(t) > 1 - \frac{1}{\rho(\widetilde{G}^{-1})\rho(B)}.$$

Therefore, for all $i \in [n]$,

$$\Big(\rho(\widetilde{G}^{-1})\rho(B)\Big)^{-1} > 1 - v_i(t). \tag{6}$$

Since (6) holds for all $i \in [n]$,

$$\begin{aligned}&\Big(\rho(\widetilde{G}^{-1})\rho(B)\Big)^{-1} > \max\{1 - v_i(t) : i \in [n]\}\\ \Rightarrow &\Big(\rho(\widetilde{G}^{-1})\rho(B)\Big)^{-1} > \rho(I_n - \widetilde{V}(t)).\end{aligned}$$

By Lemma 1, $\widetilde{S}(t) \leq I_n - \widetilde{V}(t)$. Therefore,

$$1 > \rho(\widetilde{G}^{-1})\rho(\widetilde{S}(t))\rho(B).$$

By the log-majorization theorem [19, Thm 5.12], we have $\sigma(A)\sigma(B) \geq \sigma(AB)$, where $\sigma(M)$ is the singular value of M. Also note that $\rho(A) \leq \sigma(A)$ and equality holds if A is symmetric. Therefore, since $\widetilde{G}^{-1}$ and $\widetilde{S}^*$ are diagonal and hence symmetric, $\rho(\widetilde{G}^{-1})\rho(\widetilde{S}(t)) = \sigma(\widetilde{G}^{-1})\sigma(\widetilde{S}(t)) \geq \sigma(\widetilde{G}^{-1}\widetilde{S}(t)) = \rho(\widetilde{G}^{-1}\widetilde{S}(t))$. Similarly, since B is symmetric under Assumption 4,

$$\begin{aligned}\rho(\widetilde{G}^{-1}S(t))\rho(B) &= \sigma(\widetilde{G}^{-1}S(t))\sigma(B)\\ &\geq \sigma(\widetilde{G}^{-1}\widetilde{S}(t)B)\\ &\geq \rho(\widetilde{G}^{-1}\widetilde{S}(t)B),\end{aligned}$$

leading to the following conclusion:

$$1 > \rho(\widetilde{G}^{-1}\widetilde{S}(t)B), \tag{7}$$

which is the effective reproduction number of the infection subsystem for all time $t \geq T$. It follows from Lemma 5 that the sub-system (2c) is locally asymptotically stable around $x^* = 0_n$ if (7) holds for all $t \geq T$ for some $T \in \mathbb{R}_{\geq 0}$. Therefore, v_c is a target vaccination criterion. ∎

Note that in the single-node case ($n = 1$), $v_c = 1 - \frac{1}{\mathcal{R}_0}$, which is consistent with the literature [20].

In the next lemma, we introduce the target opinion criterion $\underline{o}^{v^*}$, corresponding to some target vaccination goal v^* and its corresponding control signal $\underline{m}^{v^*}$. Notice that the target vaccination goal v^* can be any constant such that $v_c \leq v^* < 1$.

Lemma 6: If

$$\underline{m}^{v^*} = (I - \widetilde{\underline{O}}^{v^*})^{-1}(A + 2L[A])\underline{o}^{v^*}, \tag{8}$$

where:

$$\underline{o}^{v^*} = k^{-1}[\Delta H](\frac{\delta}{1 - v^*} - k[H_{\min}]), \tag{9}$$

then $(\underline{o}^{v^*}, (1 - v^*)1_n, 0_n, 0_n, v^*1_n)$ is the unique healthy state equilibrium of system (2) with the media actuator in (4).

Note that Lemma 6 does not specify that the value of v^* needs to be greater than v_c. Instead, the lemma introduces a tracking scheme for any vaccination goal $v^* \in [0, 1]$. A natural consequence of this lemma is that the system will return to its original equilibrium when the control signal is relaxed. Furthermore, we will see that not all vaccination goals are feasible via opinion control. The feasibility of the control signal depends on the upper and lower bounds of the opinions. that is, $\underline{o}_i^{v^*} \in [0, 1]\ \forall i \in [n]$. The next lemma characterizes the constraints in terms of the system parameters and the vaccination goal v^*.

Lemma 7: We have $0_n \leq \underline{o}_i^{v^*} \leq 1_n$ if and only if $(1 - v^*)k_i(H_{\min}) \leq \delta_i \leq (1 - v^*)k_i(H)$.

Lemma 7 shows that only a subset of systems of the form in (2) can be controlled via opinions, that is, there exist parameterizations of (2) such that the vaccination goal $v^* \geq v_c$ can be achieved only if $\underline{o}_i^{v^*} > 1$ or $\underline{o}_i^{v^*} < 0$. While there is flexibility in choosing v^*, the fundamental constraint lies in the relationship between the rate of loss of immunity from vaccination δ_i, the target vaccination criterion v_c, and the maximum rate of vaccine-uptake $k_i[H]$. If the ratio between the rate of loss of immunity from vaccination and the maximum rate of vaccine-uptake is greater than $1 - v_c$, then no control signals of the form in (4) are guaranteed to eradicate the disease, unless we have a more relaxed v_c that guarantees stability around the healthy state equilibrium of the system.

The next theorem shows that the suggested control law can provide local stability around the healthy state equilibrium of system (2) with a media actuator (4).

Theorem 2: If $0_n \ll \underline{o}^{v^*} \ll 1_n$ and $m(t) = \underline{m}^{v^*}$ as defined in (9) and (8), respectively, then for all $v^* \geq v_c$ (2) with media actuator (4) is locally asymptotically stable around the healthy state equilibrium $(\underline{o}^{v^*}, (1 - v^*)1_n, 0_n, 0_n, v^*1_n)$.

Proof: Since $s(t) = 1_n - x(t) - r(t) - v(t)$ by Lemma 1, we can linearize (2) around the healthy state equilibrium as follows, without including the dynamics of $s(t)$:

$$\begin{bmatrix}\dot{x}\\ \dot{o}\\ \dot{r}\\ \dot{v}\end{bmatrix} = \begin{bmatrix}J_x & 0 & 0 & 0\\ A & J_o & 0 & 0\\ \widetilde{G} & 0 & J_r & 0\\ 0 & v^*(1 - v^*)I & v^*\widetilde{K}[H(\underline{o})] & J_v\end{bmatrix}\begin{bmatrix}x\\ o\\ r\\ v\end{bmatrix}$$

where $J_o = -(A + 2L[A] + \widetilde{\underline{M}}^{v^*})$, $J_x = (1 - v_c)B - \widetilde{G}$, $J_r = -(\widetilde{W} + v^*K[H(\underline{o}^{v^*})])$, and $J_v = (1 - v^*)H(\underline{o}^{v^*}) - \widetilde{D}$.

The linearized system is stable if and only if each diagonal block is stable because the Jacobian is lower triangular. By Theorem 1, the infection subsystem J_x is locally asymptotically stable around $x^* = 0_n$ for all $v^* \geq v_c$. Moreover, we notice that each Gershgorin disc of J_o has a center

at $-(\underline{m}_i^{v^*} + 2k_i[A])$ with radius $k_i[A]$, and $\underline{m}_i^{v^*}$ is non-negative. Therefore, the spectral abscissa of J_o lies on the open left half plane by the Gershgorin circle theorem. Furthermore, J_r is locally asymptotically stable by observing that $-(\widetilde{W} + v^* K[H(\underline{o}^{v^*})])$ is a negative diagonal matrix.

Lastly, an upper bound of the spectral abscissa of J_v can be obtained by replacing $\widetilde{D}$ with $(1-v^*)k_i[H_{\min}]$, because Lemma 7 states that $\widetilde{D} \gg (1-v^*)\widetilde{K}(H_{\min})$ if $\underline{o}^{v^*} \gg 0_n$. By assumption, $\underline{o}^{v^*} \gg 0_n$ holds. Then, by shifting the center of each Gershgorin disc to the left, we obtain:

$$\begin{aligned}\alpha(J_v) &= \alpha((1-v^*)H(\underline{o}^{v^*}) - \widetilde{D}) \\ &< \alpha((1-v^*)H(\underline{o}^{v^*}) - (1-v^*)\widetilde{K}[H_{\min}]) \\ &= \alpha\left((1-v^*)\left[H(\underline{o}^{v^*}) - \widetilde{K}[H_{\min}]\right]\right). \end{aligned} \tag{10}$$

By replacing each term with its corresponding definition: $H(\underline{o}^{v^*}) := \widetilde{O}^{v^*}\Delta H + (H_{\min} - K[H_{\min}])$ and $\widetilde{O}^{v^*} := \widetilde{K}^{-1}[\Delta H](\frac{1}{1-v^*}\widetilde{D} - \widetilde{K}[H_{\min}])$, we have the following:

$$H(\underline{o}^{v^*}) = \left(\frac{1}{1-v^*}\widetilde{D} - \widetilde{K}[H_{\min}]\right)\widetilde{K}^{-1}[\Delta H] + H_{\min}. \tag{11}$$

Notice that by replacing $\widetilde{D}$ with $(1-v^*)\widetilde{K}(H_{\min})$ in (11), terms cancel out and we are left with $H(\underline{o}^{v^*}) = H_{\min}$. Therefore, if we substitute $H(\underline{o}^{v^*})$ from (11) into (10) and apply the inequality $\widetilde{D} \gg (1-v^*)$, we have:

$$\begin{aligned}\alpha(J_v) &< \alpha\left((1-v^*)\left[H(\underline{o}^{v^*}) - \widetilde{K}[H_{\min}]\right]\right) \\ &< \alpha\left((1-v^*)\left[H_{\min} - \widetilde{K}[H_{\min}]\right]\right).\end{aligned}$$

By the Gershgorin circle theorem, $\alpha\left((1-v^*)\left[H_{\min} - \widetilde{K}[H_{\min}]\right]\right) \leq 0$, which completes the proof. ■

IV. Simulation

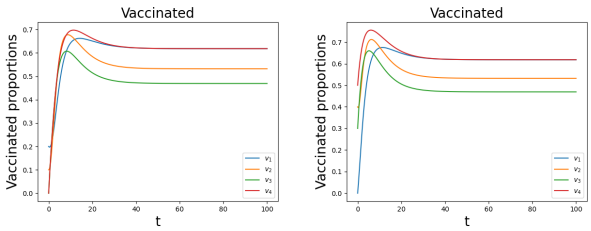
In this section, we illustrate and visualize the results in Section III. There are 4 nodes in our simulated system, where each node represents an individual or a community. The key difference between a network of communities and a network of individuals in a networked epidemic model is that whether we allow self-loops in the disease transmission network. When modelling interaction between individuals, it is most natural to assume that each individual does not induce sickness to themselves, while the opposite is true when modelling interaction between communities.

The infectious network, the strategy immitation network, and the opinion network are characterized by the following matrices:

$$B = \begin{bmatrix} 0 & 0.4 & 0.35 & 0.3 \\ 0.4 & 0 & 0.2 & 0.25 \\ 0.35 & 0.2 & 0 & 0.2 \\ 0.3 & 0.25 & 0.2 & 0 \end{bmatrix}, \quad H = \begin{bmatrix} 0 & 0.4 & 0.2 & 0.5 \\ 0.7 & 0 & 0.8 & 1 \\ 1 & 0.5 & 0 & 0.7 \\ 1 & 0.8 & 0.5 & 0 \end{bmatrix}.$$

Other parameters are defined as follows:

- $\gamma = [0.4, 1.6, 0.8, 0.3]$;
- $\omega = [0.4, 0.5, 0.6, 0.7]$;
- $\delta = [0.1, 0.2, 0.2, 0.1]$;



(a) $v(t)$ with the original initial condition.

(b) $v(t)$ with the second initial condition.

Fig. 1: The existence and uniqueness of the healthy state equilibrium, illustrating Proposition 2. Only the vaccination levels over time are shown.

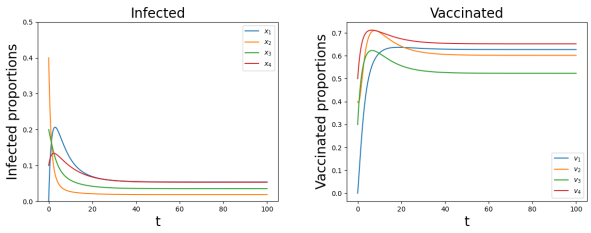


Fig. 2: The system with an endemic state equilibrium.

- $\eta_{ij}^{\min} = 0.1$, $\forall i \neq j \in [n]$;
- $\alpha_{ij} = 0.1$, $\forall i, j \in [n]$.

The initial conditions of the system:

$$(s(t_0), x(t_0), r(t_0), v(t_0), o(t_0)),$$

are $((0, 0.2, 0.3, 0.2)$, $(0.2, 0.4, 0.2, 0.3)$, $(0.6, 0.3, 0.5, 0.5)$, $(0.2, 0.1, 0, 0)$, $(0, 0.1, 0.9, 1))$, respectively, where $t_0 = 0$ in the following simulations.

Figure 1 illustrates the existence of the healthy state equilibrium with a non-zero vaccination equilibrium which is formally presented in Proposition 2. We change the initial conditions (Figure 1 (b)) to: $s(t_0) = (0.9, 0.2, 0.5, 0.4)$, $x(t_0) = (0, 0.4, 0.2, 0.1)$, $r(t_0) = (0.1, 0, 0, 0)$, $v(t_0) = (0, 0.4, 0.3, 0.5)$, and $o(t_0) = (0, 0.1, 0.9, 1)$. Figure 1 shows that $v^* \gg 0$ is invariant to a single perturbation in the initial states. According to Lemma 3, at a healthy equilibrium, if v^* is unique, s^*, r^*, and x^* are also unique.

To show the impact of the proposed controller, we first construct a system with an endemic state equilibrium, which is as an equilibrium where $x^* > 0_n$, by setting $\gamma = [0.2, 0.8, 0.4, 0.15]$.

Other parameters are the same as in the previous setting. Note B is symmetric in the original setup. Figure 2 shows the dynamics of the system without the control strategy.

Then, we apply the control strategy based on Theorem 2. If there exists a t_1^i such that $o_i(t_1^i) < \underline{o}_i^{v_c}$ defined in (9), then $m_i(t) = \underline{m}_i^{v_c}$ defined in (8), $\forall t > t_1^i$, and $m_i(t) = 0$, $\forall t < t_1^i$, where t_1^i represents when the control applied on Node i starts. In our case, as shown by Figure 3, $t_1^{[1-3]}$ are 0, 0, 2.29, respectively. Node 4 is never connected to the actuator, since $o_4(t) \geq \underline{o}_i^{v_c}$, $\forall t > t_0$. In Figure 3, we can see that the

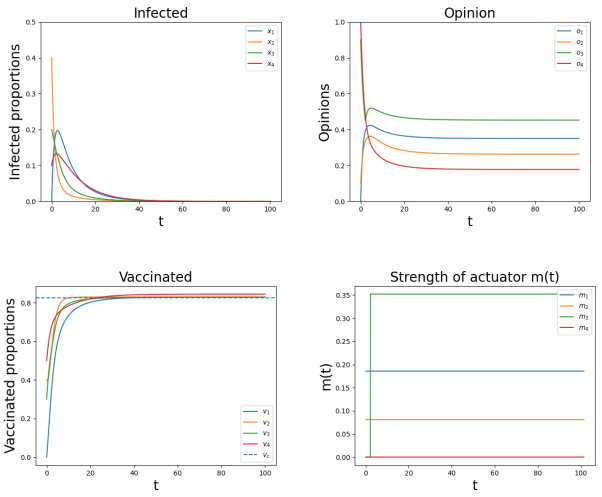


Fig. 3: The system with the control strategy applied.

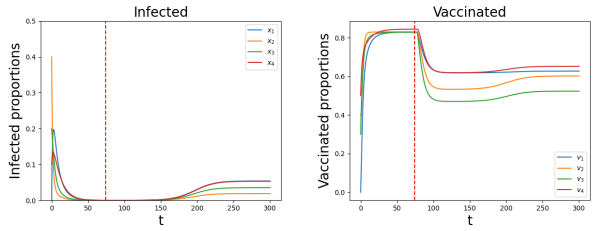


Fig. 4: The healthy state equilibrium is unstable after ending the control. The vertical red dotted line indicates the time when the control signal is lifted.

system with the applied control strategy results in a healthy state equilibrium. Note, we need to maintain the control input in order to ensure that the healthy state equilibrium remains stable. Figure 4 shows that the healthy state equilibrium is no longer stable if we remove the media actuator at $t_2 = 74$, and the system returns to its natural endemic equilibrium. As indicated by the red dashes in Figure 4, the system no longer maintains the healthy state equilibrium $\forall t > t_2$.

V. Conclusion

In this work, we propose a networked $SIRS{-}V_o$ model to study the interaction between opinion dynamics, self-interest decision-making, vaccine uptake, and disease spreading process. We provide in this paper a proof on the existence of the healthy state equilibrium of the networked $SIRS - V_o$ model, and show that the problem can be reduced to the equilibrium analysis of a single viral networked SIS model. We then characterize the local stability condition of the infection subsystem of the networked multi-layer epidemic model around the healthy state equilibria. Building on this model, we develop target vaccination and opinion criteria which guarantee disease eradication. These criteria provide sufficient conditions that leverage opinions and vaccination to eradicate the spread of an epidemic. We then show analytically that the family of control signals which satisfies the target vaccination and opinion criteria stabilizes the networked $SIRS - V_o$ model locally around the healthy state equilibrium under some mild assumptions. We also note that the infection subsystem returns to its natural equilibrium after the control algorithm is relaxed, both analytically and in simulations. In the future, we will extend the control analysis to study robust government-subsidized vaccination programs for disease control under adversarial attacks on opinions through fake news or other mediums.

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