Centrality in Epidemic Networks with Time-Delay: 
A Decision-Support Framework for Epidemic Containment

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\section*{Abstract}
During an epidemic, infectious individuals might not be detectable until some time after becoming infected. The studies show that carriers with mild or no symptoms are the main contributors to the transmission of a virus within the population. The average time it takes to develop the symptoms causes a delay in the spread dynamics of the disease. When considering the influence of delay on the disease propagation in epidemic networks, depending on the value of the time-delay and the network topology, the peak of epidemic could be considerably different in time, duration, and intensity. Motivated by the recent worldwide outbreak of the COVID-19 virus and the topological extent in which this virus has spread over the course of a few months, this study aims to highlight the effect of time-delay in the progress of such infectious diseases in the meta-population networks rather than individuals or a single population. In this regard, the notions of epidemic network centrality in terms of the underlying interaction graph of the network, structure of the uncertainties, and symptom development duration are investigated to establish a centrality-based analysis of the disease evolution. A traffic volume convex optimization method is then developed to control the outbreak by identifying the sub-populations with the highest centrality and then isolating them while maintaining the same overall traffic volume (motivated by economic considerations) in the meta-population level. The numerical results, along with the theoretical expectations, highlight the impact of time-delay as well as the importance of considering the worst-case scenarios in investigating the most effective methods of epidemic containment.

\section{Introduction}
The large-scale spread of an infectious disease occurs every few years and leads into serious crises before it eventually dies out \cite{1}. The extend in which a high-speed epidemic continues depends mostly on first, the government interventions, and second, the existence of an effective treatment against the disease. In this regard, the study of epidemic propagation by network

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models have been theoretically proven to be useful in determining the most effective methods of epidemic management and control as well as allocating treatment and immunization resources [2], [3].

The macro-modeling (or meta-population) representation of the epidemic networks is studied in [4], where the author introduces the general form of the spread dynamics in a community of sub-populations. Many studies have been using meta-population network of Susceptible-Infected-Susceptible (SIS) [5], Susceptible-Infected-Removed (SIR) [6], [7], and Susceptible-Exposed-Infected-Removed (SEIR) [7] models to develop the dynamics of epidemic in a large scale.

The centrality analysis of a time-delayed consensus network has been studied in [8] and a closed form of the node and link centralities in the presence of different noises in the network is offered. In [9] the authors have studied the convergence analysis of the time-delayed networks with linear dynamics and established a connection between their convergence rate and delay (in addition to the correlation it has with the largest eigenvalue of the graph). The stochastic delayed SIS model has been exploited in [10] to find the threshold behavior of the systems with vaccination and double diseases.

Many works have been done in the area of epidemic elimination, disease spread control, and designing public health control measures against the epidemics [11]–[13]. Some are suggesting that an optimized alternation in the disease network structure could result in hindering the disease effectively. Node or edge removal is the direct way of modifying the network structure which is also called spectral control, as it aims to minimize the largest eigenvalue of the network to locally stabilize it around the disease-free equilibrium [5], [14]. However, the spectral radius minimization has been proven to be an NP-hard problem [15]. Network metric-based node or edge removal is another common approach in spectral control. A removal strategy based on the number of the closed walks and assortativity effect has been proposed in [15].

On the other hand, vaccination and traffic flow restriction modify the epidemic recovery and infection rates, which in turn, affect the disease progress pattern. Various control strategies have been proposed in this area. A geometric programming-based approach has been presented in [5] for the optimal resource allocation in a meta-population network. In this study, the infection and recovery rates are assumed to be the optimization variables. The intercity traffic restriction of an SIS mate-population model has also been studied in [16]. An optimal and a heuristic local solution are proposed to completely eradicate the disease, where the cost of second solution
is shown to be higher than the first one. In terms of social awareness effect on the epidemic evolution, [17] has proposed a semidefinite programming-based optimization to control a network that follows the Susceptible-Alert-Infected-Susceptible (SAIS) epidemic model.

In the real-world cases, node removal happens by social distancing, quarantining, vaccinating individuals, or complete shut down of certain areas in the city [18]. In this regard, [19] has introduced PageRank-based vaccination strategy for realistic social networks. [20] has also proposed a network attack strategy based on degree and betweenness centrality for both passive and online node and edge removal approaches. A Pulse vaccination strategy has been offered in [6] which can eradicate the disease in all the sub-populations.

In 2020, many network scientists have established their study on the modeling and control of COVID-19, as it has turned into a global crisis. In the recent research study, the optimal intervention starting time and duration that will reduce the epidemic peak and length in an SIR epidemic model has been proposed. The effect of a complete suppression, mistimed interventions, and sustained interventions have also been investigated in this study [21].

A more generalized SEIR model of COVID-19 nationwide spread in China including self-protection and quarantine compartments has been established to predict the disease propagation pattern and to find a possible extinction time for some of the provinces [22]. An SIRD model has been developed to estimate the COVID-19 epidemic parameters in Italy, using real-data and a non-convex parameter identification approach [23].

The effect of social distancing start time, period, and duration on the infection peak of COVID-19 in epidemic models has been investigated in [24]. It is also shown that a well-timed intervention improves flattening the infection curve in various epidemic models, such as SIR, feedback-SIR (fSIR), Susceptible-Infected-Quarantined-Susceptible (SIQR), Susceptible-Infected-Diagnosed-Ailing-Recognized-Threatened-Healed-Extinct (SIDARTHE), and 6 compartment SIR. An agent-based model of COVID-19 dynamics is utilized to simulate the multi-wave behavior of the spread while applying different possible government interventions such as testing and tracing, and travel restriction on it [25].

**Our contributions.** Inspired by the recent COVID-19 outbreak, this study is dedicated to the epidemiological investigation of the infectious diseases with the following main contributions:

i. Modeling the time-delayed epidemic dynamics of meta-populations using the network SIS model (Section [1]).
ii. Investigating the effect of time-delay and different sources of uncertainty on the modeled network dynamics (Sections II and III).

iii. Developing the explicit centrality measure of sub-populations with respect to time-delay and transportation network structure (Section III).

iv. Designing optimal and robust methods of traffic restriction based on the steady-state performance of the network (Section IV).

The simulation results are presented in Section V.

II. DELAYED EPIDEMIC NETWORK OF META-POPULATION MODELS

The proportion of pre-symptomatic individuals in the population could play an important rule in the disease propagation pattern. In the case of COVID-19 for instance, studies indicate that a significant proportion of positive tests belongs to the pre-symptomatic individuals who are just the carriers [26] and might not develop any symptoms up to 14 days [27]. The effect of pre-symptomatic individuals in the spread of the disease can be modeled as a time-delay in the network dynamics. In this study, the average time of symptom development for all the individuals is assumed to be identical and equal to a non-negative constant $\tau$.

A. Deterministic meta-population SIS model with time-delay

Let the undirected and weighted graph $G = (\mathcal{V}, \mathcal{E}, w)$ represent a meta-population in the epidemic network. $\mathcal{V} = \{1, 2, \ldots, n\}$ shows the set of nodes in the graph or the group of cities, states, countries, or in general, sub-populations in the epidemic network. $\mathcal{E}$ denotes the edge set which shows the connection between every two member of $\mathcal{V}$ with the corresponding weight of $w_e = a_{ij}$ for all $e = \{i, j\} \in \mathcal{E}$. The counterpart of weighted edges in the epidemic network would be the transportation capacity or traffic volume between every two sub-population in $\mathcal{V}$. The adjacency matrix of the corresponding graph is then defined as $A = [a_{ij}]$ for $i \neq j$.

The state of epidemic network at time $t \geq 0$ is represented by vector $p(t) = [p_1(t), \ldots, p_n(t)]^T$, where $p_i \in [0, 1]$ is the marginal probability of sub-population $i$ being infected at time $t$ such that $p_i(t) = 1$ if the sub-population $i$ is infected and $p_i(t) = 0$ if it is susceptible. Assuming that every sub-population is experiencing a delay $\tau$ due to the reasons explained earlier, the approximated spread dynamics of sub-population $i$ can then be described using the mean-field approximation.
model with time-delay as below,

\[
\dot{p}_i(t) = -\delta_i p_i(t - \tau) + \beta_i \sum_{j=1}^{n} a_{ij} p_j(t - \tau)(1 - p_i(t - \tau)),
\]

\[y_i(t) = p_i(t),\]

where \(\beta_i\) is the infection rate at which sub-population \(j\) will contaminate sub-population \(i\), and \(\delta_i\) is the recovery rate of sub-population \(i\). \(a_{ij} = w_e\) is the \(ij^{th}\) component of the adjacency matrix of the coupling graph which is equivalent to the weight of edge \(e = \{i, j\}\). Here, the desired output \(y_i(t)\) is the infection probability of sub-population \(i\) at time \(t\) which is required to monitor and update the performance measure of network. The compact form of meta-population SIS model can be expressed as below,

\[
\dot{p}(t) = \mathcal{A} p(t) - P(t - \tau) B A p(t - \tau),
\]

\[y(t) = p(t),\]

where \(P(t - \tau) = \text{diag}(p(t - \tau))\), and infection and recovery matrices are

\[B = \text{diag}([\beta_1, \ldots, \beta_n]),\]  

\[\Delta = \text{diag}([\delta_1, \ldots, \delta_n]).\]  

Matrix \(\mathcal{A}\) is composed of off diagonal infection rates and diagonal recovery rates as follows:

\[
\mathcal{A} = BA - \Delta = 
\begin{bmatrix}
-\delta_1 & \beta_1 a_{12} & \cdots & \beta_1 a_{1n} \\
\beta_2 a_{21} & -\delta_2 & \cdots & \beta_2 a_{2n} \\
\vdots & \vdots & \ddots & \vdots \\
\beta_n a_{n1} & \beta_n a_{n2} & \cdots & -\delta_n
\end{bmatrix}.
\]

In Eq. (1), \(p_i(t)\) is the infected proportion of the sub-population \(i\) at time \(t\). For every sub-population in the community, it can be assumed that \(p_i(t)\) is a value close to zero. For instance, this is a valid assumption for the recent COVID-19 pandemic, because even though there were a substantial number of infected individuals from the beginning, the proportion of the infected population between January 19 (The day that first case was reported) through February 28 within
the US was still close to zero\(^1\) It therefore makes sense to linearize the epidemic model around the zero state. Assuming \( p_i(t) \ll 1 \), equation (1) can be linearized as follows:

\[
\dot{p}_i(t) = -\delta_i p_i(t - \tau) + \beta_i \sum_{j=1}^{n} a_{ij} p_j(t - \tau) + b_i^T \xi(t).
\] (5)

The compact version of this network is in the following form.

\[
\begin{align*}
\dot{p}(t) &= \mathcal{A} p(t - \tau) + \mathcal{B} \xi(t), \\
y(t) &= p(t),
\end{align*}
\] (6)

in which \( p = [p_1, \ldots, p_n]^T \) is the state vector, \( y = [y_1, \ldots, y_n]^T \) is the output vector, and \( \xi = [\xi_1, \ldots, \xi_l]^T \) is the effect of an uncertainty in the disease spread behaviour on sub-populations or their interconnection. \( \xi \in \mathbb{R}^{l \times 1} \) is the vector of independent Gaussian white noise with zero mean and matrix \( \mathcal{B} = [b_1, \ldots, b_n]^T \in \mathbb{R}^{n \times l} \) is the noise coefficient matrix.

It should be noted that the following assumptions apply throughout the rest of this paper.

**Assumption 1:** The infection rate of all the sub-populations is equal. Therefore, \( \beta_i = \beta \) for all \( i \in \mathcal{V} \).

**Assumption 2:** The network graph is assumed to be undirected, i.e., \( a_{ij} = a_{ji} \).

**B. Stability analysis**

For the undirected network (2), the initial infection \( p(0) \in [0, 1]^n \) will exponentially die out if

\[
\lambda_{\max}(\mathcal{A}) \leq -\varepsilon.
\] (7)

In other words, an \( \alpha > 0 \) could be found to satisfy \( \|p_i(t)\| \leq \alpha \|p_i(0)\|e^{-\varepsilon t} \) and as a result, the disease-free equilibrium of the system is globally exponentially stable with rate \( \varepsilon \)\(^5\).

The minimum eigenvalue of \( \mathcal{A} \) is also lower-bounded by the time-delay as below,

\[
\lambda_{\min}(\mathcal{A}) \geq -\frac{\pi}{2\tau}
\] (8)

which is the direct result of frequency domain stability analysis of the delayed systems\(^9\). The network is globally asymptotically stable if and only if this condition is satisfied. Therefore, the

\(^1\)https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/cases-in-us.html
stability of the network depends not only on the maximum but also the minimum eigenvalue of \( \mathcal{A} \).

Combining conditions (7) and (8) results in the following matrix inequality with respect to the positive semi-definite cone \( S^n_+ \),

\[
\varepsilon I_n \preceq -\mathcal{A} \preceq \frac{\pi}{2\tau} I_n.
\] (9)

C. Performance analysis

The steady-state performance, \( \rho_{ss} \), of network (6) can be expressed as,

\[
\rho_{ss}(\mathcal{A}; \mathcal{B}; \tau) = \lim_{t \to \infty} \mathbb{E} \left[ y(t)^T y(t) \right]
\] (10)

which is a function of \( \mathcal{A} \); therefore, the bounds on the eigenvalues of \( \mathcal{A} \) determine the range in which the long-run performance of the network will change. More details on the performance measure of a class of consensus networks under the influence of exogenous white noises can be found in the reference papers [28]–[31]. According to [32], the performance of a network can also be found by the frequency domain definition of its \( \mathcal{H}_2 \)-norm as below,

\[
\rho_{ss}(\mathcal{A}; \mathcal{B}; \tau) = \frac{1}{2\pi} \text{Tr} \left[ \int_{-\infty}^{+\infty} G^H(j\omega)G(j\omega)d\omega \right],
\] (11)

where \( G(j\omega) \) is the transfer function of the network.

Lemma 1: For the undirected network (6), the closed form solution of (11) is,

\[
\rho_{ss}(\mathcal{A}; \mathcal{B}; \tau) = \sum_{i=1}^{n} -\Phi_i \frac{\cos (\lambda_i \tau)}{2\lambda_i} \frac{1 + \sin (\lambda_i \tau)}{1 + \sin (\lambda_i \tau)},
\] (12)

in which \( \Phi_i \) is the \( i^{th} \) diagonal element of the matrix \( Q^T \mathcal{B} \mathcal{B}^T Q \), where \( Q = [q_1, \ldots, q_n] \in \mathbb{R}^{n \times n} \) is the orthonormal matrix of eigenvectors of \( \mathcal{A} \).

Proof 1: The transfer matrix of (6) is,

\[
G(s) = (sl_n - e^{-\tau s} \mathcal{A})^{-1} \mathcal{B}
= Q \text{diag} \left( \left[ \frac{1}{s - \lambda_1 e^{-\tau s}}, \ldots, \frac{1}{s - \lambda_n e^{-\tau s}} \right] \right)^T \mathcal{B}^T.
\] (13)
For this transfer function matrix we have,

$\text{Tr} \left[ G^H(j\omega)G(j\omega) \right] =$

$\text{Tr} \left[ Q^T \mathcal{C} \mathcal{C}^T Q \text{diag} \left( \begin{bmatrix} 1 & 1 \\ -\lambda_1 e^{j\tau\omega} - j\omega & -\lambda_n e^{j\tau\omega} - j\omega \end{bmatrix}^T \right) \right]$

and by substituting (14) in (11), the performance will be,

$\rho_{ss}(\mathcal{C}; \mathcal{D}; \tau) = \frac{1}{2\pi} \sum_{i=1}^{n} \int_{-\infty}^{+\infty} \Phi_i d\omega \left( \frac{\Phi_i d\omega}{(j\omega + \lambda_i e^{j\tau\omega})(\lambda_i e^{-j\tau\omega} - j\omega)} \right)$. 

A proof follows by simplifying the above integral.

It should be noted that the smaller values of $\rho_{ss}$ result in a better performance, therefore, a lower value of performance is desired.

III. CENTRALITY INDICES

The importance of every sub-population in the disease spread can be analysed by various indices. In this study, the centrality index, $\eta_i$, is the basis to rank the sub-populations.

For network (6), let $\xi_i(t) \sim \mathcal{N}(0, \sigma_i^2)$ be the noise affecting the $i^{th}$ sub-population’s infection dynamics, which might stem from modeling imperfections, testing error or inaccurate epidemic rates. The centrality of sub-population $i$ is then defined as the rate of performance with respect to disturbance variance,

$\eta_i := \frac{\partial \rho_{ss}}{\partial \sigma_i^2}$. 

The centrality index with respect to two important sources of disturbance will be established in the following sections.

A. Modeling error

Model simplifications implemented on the epidemic dynamics affect the state of the infected sub-populations as below,

$\dot{p}_i(t) = -\delta_i p_i(t - \tau) + \beta_i \sum_{j=1}^{n} a_{ij} p_j(t - \tau) + \xi_i(t)$, 

(17)
where $\xi_i(t) = \sigma_i \hat{\xi}_i$. The compact form of (17) would be,

$$\dot{p}(t) = \mathcal{A} p(t - \tau) + \mathcal{B}_1 \hat{\xi}(t),$$

(18)

in which $\mathcal{B}_1 = \text{diag} ([\sigma_1, \ldots, \sigma_n]) \in \mathbb{R}^{n \times n}$.

**Corollary 1:** For the network (18), the centrality index of the $i^{th}$ sub-population is,

$$\eta_i(\mathcal{A}; \tau) = -\frac{1}{2} \left[ \mathcal{A}^{-1} \cos(\tau \mathcal{A}) (I_n + \sin(\tau \mathcal{A}))^{-1} \right]_{ii},$$

for all $i \in V$.

**Proof 2:** In the case of having modeling noise, the network dynamics is the same as (6) with $\mathcal{B} = \mathcal{B}_1$. Hence, the performance will be in the following matrix operator form,

$$\rho_{\text{ss}}(\mathcal{A}; \mathcal{B}_1; \tau) =$$

$$\frac{1}{2} \text{Tr} \left[ -\text{diag} ([\sigma_1^2, \ldots, \sigma_n^2]^T) \mathcal{A}^{-1} \cos(\tau \mathcal{A}) (I_n + \sin(\tau \mathcal{A}))^{-1} \right].$$

(19)

On the other hand, the value of centrality measure $\rho_{\text{ss}}$ is a linear function of variance of elements of noise input. For the centrality index (16) the performance is defined as below,

$$\rho_{\text{ss}} = \sum_{i \in V} \eta_i \sigma_i^2.$$  

(20)

Substituting equation (19) in the above definition, the centrality will be obtained.

**B. Testing error**

In many cases of epidemic, especially when an infectious disease like COVID-19 first emerges, the testing methods are not completely accurate in terms of identifying the infected individuals. The incorrect results generate inaccurate statistics regarding the population of the confirmed cases which triggers impaired judgment and inappropriate containment methods.

In theory, the testing error affects every sub-population’s state in the following way,

$$\dot{p}_i(t) = -\delta_i (p_i(t - \tau) + \xi_i(t)) + \beta_i \sum_{j=1}^{n} a_{ij} (p_j(t - \tau) + \xi_j(t)),$$

(21)

where $\xi_i \sim \mathcal{N}(0, \sigma_i^2)$ for $i \in V$. The state of the infected population in this case will be the
same as (6) with $B = B_2 = \mathcal{A} \text{diag}([\sigma_1, \ldots, \sigma_n]) \in \mathbb{R}^{n \times n}$ as below,

$$\dot{p}(t) = \mathcal{A} p(t - \tau) + B_2 \hat{\xi}(t).$$  \hspace{1cm} (22)

**Corollary 2:** For the network (22), the centrality index is,

$$\eta_i(\mathcal{A}; \tau) = -\frac{1}{2} \left[ \mathcal{A} \cos(\tau \mathcal{A}) (I_n + \sin(\tau \mathcal{A}))^{-1} \right]_{ii},$$  \hspace{1cm} (23)

for all $i \in V$.

**Proof 3:** With the testing error noise, the network dynamics is the same as (6) with $B = B_2$. Therefore, the compact form of $\rho_{ss}$ is,

$$\rho_{ss}(\mathcal{A}; B_2; \tau) =$$

$$\frac{1}{2} \text{Tr} \left[ -\text{diag} \left( [\sigma_1^2, \ldots, \sigma_n^2]^T \right) \mathcal{A} \cos(\tau \mathcal{A}) (I_n + \sin(\tau \mathcal{A}))^{-1} \right].$$  \hspace{1cm} (24)

A proof follows by using the definition (20) and above equation to find centrality.

**IV. Epidemic Containment by Traffic Volume Optimization at Community Levels**

**A. Optimal traffic restriction**

Monitoring and regulation of the traffic volume is one of the potential government interventions to mitigate the epidemic threat. Regarding the underlying epidemiological network, traffic restriction between two sub-populations directly changes the corresponding edge weight, $w_e$, of those sub-populations in the network. Therefore, the stability around disease-free state could be obtained by monitoring and management of the transportation network and restriction of the traffic volume between the highly infected and highly susceptible candidates. Although the complete isolation of the sub-populations seems to be the easiest and safest prevention method, especially in the case of COVID-19 which has now last for several months, it is not a permanent solution mostly because of the economic considerations. Therefore, a proper balance must be found in the decision-making process. In this study, a convex optimization method is offered to determine the proper volume of the transportation by minimizing the value of corresponding
network performance, and consequently improving the performance, with respect to the graph weights.

This optimal traffic control problem for the network with modeling error noise can be expressed as below,

\[
\begin{align*}
\text{minimize} & \quad \rho_{ss}(\mathcal{A}; \mathcal{B}_1; \tau) \\
\text{subject to:} & \quad w_e \geq 0, \\
& \quad \sum_{e \in \mathcal{E}} w_e = c, \\
& \quad \mathcal{A} = B \sum_{e \in \mathcal{E}} w_e A_e - \Delta, \\
& \quad -\pi 2 I \leq \mathcal{A} \leq -\epsilon I. \\
\end{align*}
\]

(25)

Here, the first constraint forces a limit on the network weights to avoid negative outputs that do not lie in the domain of feasible solutions. The second constraint determines the total weight of the network edges or overall traffic volume, which could acquire any desired value \( c \) depending on the intensity of isolation. The third constraint in which \( A_e \) is the adjacency matrix of link \( e \) gives the definition of \( \mathcal{A} \) with respect to edge weights. The last constraint imposes another limitation on the edge weights to respect the domain of stable solutions. \( B \) and \( \Delta \) are the infection and recovery rate matrices defined in equation (3).

Noting that the optimization problem,

\[
\begin{align*}
\text{minimize} & \quad f_0(x) \\
\text{subject to:} & \quad f_i(x) \leq b_i, \quad i = 1, \ldots, q, \\
& \quad h_i(x) = 0, \quad i = 1, \ldots, r. \\
\end{align*}
\]

(26)

is considered a convex problem if all the functions \( f_0, \ldots, f_q \) are convex and all the equality constraints \( h_1, \ldots, h_r \) are affine [33], then problem (25) does not fall into the category of convex problems. Hence, some modifications need to be implemented on the original optimal problem (26). In this regard, an approximation of the performance has been offered by [31] which converts the product of non-convex trigonometric functions to a linear function of \( \mathcal{A} \) and its inverse. Using
this approximation, the network performance will be,

$$\rho_{ss}(A; B_1; \tau) \approx \frac{1}{2} \text{Tr} \left[ -A_0 A^{-1} + \frac{4\tau}{\pi} A_0 \left( \frac{\pi}{2} I_n + \tau A \right)^{-1} - c_1 \tau^2 A_0 A + \frac{c_0}{2} \tau A_0 \right],$$  \hspace{1cm} (27)

where $A_0 = B_1 B_1^T$ and the constant parameters $c_0 = 0.1873$ and $c_1 = -0.01$ are estimated to minimize the mean squared error of the approximated performance. This approximation is still not a convex function, as it includes the non-convex inverse functions $A^{-1}$ and $\left( \frac{\pi}{2} I_n + \tau A \right)^{-1}$. Substituting the epigraph variables $X_1 = A^{-1}$ and $X_2 = \left( \frac{\pi}{2} I_n + \tau A \right)^{-1}$ will turn a convex function. The optimization problem (25) can now be cast as the following approximate form, which is a convex optimization problem as the objective function as well as all the inequality constraints are convex and the equality constraints are affine with respect to the only optimization variable $w_e$.

$$\begin{align*}
\text{minimize} & \quad \text{Tr} \left[ A_0 X_1 + \frac{4\tau}{\pi} A_0 X_2 - c_1 \tau^2 A_0 A \right] \\
\text{subject to:} & \quad w_e \geq 0, \\
& \quad \sum_{e \in E} w_e = c, \\
& \quad A = B \sum_{e \in E} w_e A_e - \Delta, \\
& \quad A + \frac{\pi}{2\tau} I_n \succeq 0, \\
& \quad -A - \varepsilon I_n \succeq 0, \\
& \quad \begin{bmatrix} X_1 & I_n \\ I_n & -A \end{bmatrix} \succeq 0, \\
& \quad \begin{bmatrix} X_2 & I_n \\ I_n & \frac{\pi}{2} I_n + \tau A \end{bmatrix} \succeq 0.
\end{align*}$$  \hspace{1cm} (28)

B. Robust traffic restriction

While the target of proposed optimal approach is to improve the overall network performance with a uniform uncertainty distribution, i.e. $\sigma_i = 1$ for $i = 1, \ldots, n$, there are cases in which the sub-populations with highest centrality experience higher levels of disturbance. Worst case noise distribution highlights the role of high centrality sub-populations in epidemic growth which requires us to design a more robust containment approach. Therefore, in this section, the traffic
restriction problem is investigated as a robust design optimization where the worst-case scenario is the optimization target. Such a case can be expressed as a min-max problem as below,

\[
\begin{align*}
\text{minimize} & \quad \max_{\sigma_i, \forall i \in \mathcal{V}} \rho_{\text{eq}}(\mathcal{I} \setminus \mathcal{B}_1; \tau) \\
\text{subject to} & \quad \sum_{i \in \mathcal{V}} \sigma_i^2 = n, \\
& \quad w_e \geq 0, \\
& \quad \sum_{e \in \mathcal{E}} w_e = c, \\
& \quad \mathcal{A} = B \sum_{e \in \mathcal{E}} w_e A_e - \Delta, \\
& \quad -\frac{\pi}{2\tau} I_n \preceq \mathcal{A} \preceq -\epsilon I_n. 
\end{align*}
\tag{29}
\]

The first constraint limits the sum of squared noise variances and the rest of the constraints are the same as explained for problem (25). The inner optimization loop is to find the highest performance of the network with respect to the uncertainty \(\sigma_i\) in order to improve the robustness against the disease spread. Using the performance definition in (20), the inner optimization problem in (29) can be rewritten in the following form,

\[
\begin{align*}
\text{maximize} & \quad \sum_{i \in \mathcal{V}} \eta_i(\mathcal{A} \setminus \mathcal{B}_1; \tau) \sigma_i^2 \\
\text{subject to} & \quad \sum_{i \in \mathcal{V}} \sigma_i^2 = n.
\end{align*}
\tag{30}
\]

Here, the cost function and constraint are only linear functions of the optimization variable \(\sigma_i\). Hence, the maximum performance occurs in the boundary, where for one variable we have \(\sigma_i^2 = n\) and \(\eta_i(\mathcal{A} \setminus \mathcal{B}_1; \tau)\) has its maximum value, and for the rest \(\sigma_i^2 = 0\), i.e.,

\[
\sigma_i^2 := \begin{cases} 
  n & \text{if } i = \arg\max_{j \in \mathcal{V}} \eta_j(\mathcal{A} \setminus \mathcal{B}_1; \tau) \\
  0 & \text{otherwise}
\end{cases}
\tag{31}
\]

As a result, the maximum objective function of problem (30) is equal to the maximum value of \(-\frac{\pi}{2} \left[ \mathcal{I}^{-1} \cos(\tau \mathcal{A}) (I_n + \sin(\tau \mathcal{A}))^{-1} \right]_{ii}\) for all \(i \in \mathcal{V}'\). Note that this solution is a non-convex function of \(w_e\); therefore, to use it in the outer minimization loop, its approximated epigraph
version will be used. Problem (29) can now be cast in the following form,

\[
\begin{align*}
\text{minimize} & \quad \ x \\
\text{subject to} & \quad w_e \geq 0, \\
& \quad \sum_{e \in E} w_e = c, \\
& \quad A = B \sum_{e \in E} w_e A_e - \Delta, \\
& \quad \mathcal{A} + \frac{\pi}{2\tau} I_n \succeq 0, \\
& \quad -\mathcal{A} - \epsilon I_n \succeq 0, \\
& \quad x \geq \frac{n}{2} \left[ \sum_{e} \frac{4\tau}{\pi} A_o X_1 + c_1 \tau^2 A_o \mathcal{A} \right], \forall i \in \mathcal{V}, \\
& \quad \begin{bmatrix} X_1 & I_n \\ I_n & -\mathcal{A} \end{bmatrix} \succeq 0, \\
& \quad \begin{bmatrix} X_2 & I_n \\ I_n & \frac{\pi}{2} I_n + \tau \mathcal{A} \end{bmatrix} \succeq 0.
\end{align*}
\]

It is worth mentioning that the same methods in the preceding sections can be implemented on the networks with the described testing error noise as well.

V. RESULTS

To evaluate the performance of the proposed methods, a core-periphery network consisting of three communities has been simulated as the representative of a meta-population. The simulations are based on the dynamics (2) over a tree graph with three connected star graphs consisting of 20 nodes and 19 weighted edges. A combination of multiple star graphs is a good candidate for a meta-population, as in reality some of the sub-populations are considered as traveling hubs while the others connect to the rest of the sub-populations through these hubs. The effect of time-delay on the epidemic evolution of such a meta-population has been illustrated in Fig. 1, where the structure of the modeled network is shown in Fig. 2. Based on Fig. 1 it can be concluded that the higher the delays in identifying infected individuals, the higher the risk of experiencing an extreme epidemic peak with multiple pulses. The time-delay also shows a correlation with the onset of epidemic peak, which is a decisive factor in designing the proper government interventions.
Fig. 1. The average infection size, $\bar{p}(t) = \frac{1}{n} \sum_{i \in V} p_i(t)$, of the meta-population network shown in Fig. 2 over time with different time-delays.

In Fig. 2, the sub-populations are ranked based on their centrality index, $\eta_i$, which is reflected through the size of their indicating circles. The interconnections are ranked by their corresponding traffic volume which is specified by the thickness of edges as well as their color.
Fig. 2. Meta-population network of 20 sub-populations and their normal traffic volume specified by the thickness and color of the links. The sub-populations are ranked based on their centrality index $\eta_i$ which is correlated with their corresponding circle diameter.

Fig. 3. Optimal meta-population network of Fig. 2 designed by the optimal approach (28), where a uniform noise distribution is applied.

Fig. 4. Robust meta-population network of Fig. 2 designed by the robust approach (32), where the worst case of applying the maximum noise input to the sub-population with highest centrality is considered.
Let us assume that the network shown in Fig. 2 is randomly infected by a virus and affected by the modeling uncertainties explained earlier. Using the convex optimization method (28) with $c$ being the overall traffic volume in the absence of traffic restrictions, the network structure changes into Fig. 3. The traffic volume between the hub nodes 1, 2, and 15 is decreased by 68 percent on average, which was expected given the high centrality of node 2. For the nodes with lower centrality connected to node 2, the average volume of traffic flow to or from the hub has decreased by about 10 percent to isolate it as much as possible. The considerable decrease in the traffic volume of connections to the hub node 2 is compensated by increasing the traffic volume of nodes connected to hubs 1 and 15 with lower centralities by 13 and 18 percent, respectively. Note that the overall traffic volume of this meta-population is constrained to be constant.

The result of the robust optimization approach (32), shown in Fig. 4, is mostly close to that of the convex optimization method (28). The robust optimization tends to consider the worst case scenario where the uneven noise distribution amplifies the effect of node 2 with the highest centrality, which makes it a bigger threat requiring it to be even more isolated. Hence, the robust optimizer is more conservative in manipulating the traffic volumes.

To make a better comparison between the resulted networks, the bar diagram of the edge weights, or traffic volumes, is represented in Fig. 5.
Fig. 6. Performance comparison between the original network Fig. 2, the optimal network of Fig. 3 and the robust network of Fig. 4 in the case of uniform noise distribution for both.

Fig. 7. Performance comparison between the original network Fig. 2, the optimal network of Fig. 3 and the robust network of Fig. 4 in the case of extreme noise distribution for all.

Fig. 6 shows the logarithmic scale performance of the original, optimal, and robust networks with respect to the desired traffic volume of the meta-population when all of the sub-populations, regardless of their centrality, experience an equal level of disturbance. As it was mentioned earlier, the smaller the value of performance, the better the obtained performance. Hence, the optimal and robust controllers have been successful in improving the performance of the original network, while the optimal network results in a slightly better performance due to its less conservative weight distribution.

Additionally, a comparison between the highest performance of both optimization methods is presented in Fig. 7 to put more emphasis on the importance of considering worst cases while determining the intensity of traffic restriction during the epidemic. As the overall traffic volume constraint becomes harder in terms of reaching its maximum possible value and increases from 0.1 to 63, the difference between the maximum performance of the two resulting networks increases by 13 times.

The detailed results of the performance improvement through optimal and robust epidemic controls are represented in Table. 1 for Case 1 with uniform noise distribution, i.e. \( \sigma_i = 1 \) for \( i = 1, \ldots, n \), and Case 2 is the worst case explained in (31).

The changes in average infection size of network 2 when the optimal and robust controls are
Fig. 8. Average infection size of the three networks in Figs. 2, 3, and 4 with respect to time.

applied is shown in Fig. 8

TABLE I. THE VALUES AND PERCENTAGES OF PERFORMANCE ENHANCEMENT FOR THE NETWORKS SHOWN IN FIGS. 3 AND 4 COMPARED WITH FIG. 2

| Network Type                   | Case 1   | Case 2   |
|--------------------------------|----------|----------|
| Original network (Fig. 2)      | 19.29 (0%) | 106.21 (0%) |
| Optimal network (Fig. 3)       | 9.53 (+51%) | 13.99 (+87%) |
| Robust network (Fig. 4)        | 9.57 (+50%) | 12.71 (+88%) |

VI. CONCLUSION

In this study, the nonlinear and linear dynamics of an SIS network model within a randomly infected meta-population has been investigated. The modeled meta-population is assumed to be experiencing delays due to the considerable proportion of pre-symptomatic population. The explicit centrality indices in the presence of model simplifications and testing errors have been derived and then used to develop optimal and robust traffic restriction methods for epidemic containment purposes. The simulation results on a core-periphery network indicate that the
unavoidable delays in symptom development and infection identification can result in a significant
difference in epidemic evolution, which requires more attention while designing potential gov-
ernment interventions. The proposed optimal and robust approaches, both based on the convex
control method, shown to be capable of enhancing the delayed network’s performance, and
therefore, decreasing the infection rate considerably. Although adding more compartments to the
network model increases the complexity of deriving explicit centrality indices, it can provide
more realistic results. Implementing the proposed methods on the directed graphs with time-
varying weights is also an interesting direction for improving the results of the current study.

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