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A Convex Framework for Epidemic Control in Networks

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Abstract
With networks becoming pervasive, research attention on dynamics of epidemic models in networked populations has increased. While a number of well understood epidemic spreading models have been developed, little to no attention has been paid to epidemic control strategies; beyond heuristics usually based on network centrality measures. Since epidemic control resources are typically limited, the problem of optimally allocating resources to control an outbreak becomes of interest.

Existing literature considered homogeneous networks, limited the discussion to undirected networks, and largely proposed network centrality-based resource allocation strategies.

In this thesis, we consider the well-known Susceptible-Infected-Susceptible spreading model and study the problem of minimum cost resource allocation to control an epidemic outbreak in a networked population. First, we briefly present a heuristic that outperforms network centrality-based algorithms on a stylized version of the problem previously studied in the literature. We then solve the epidemic control problem via a convex optimization framework on weighted, directed networks comprising heterogeneous nodes. Based on our spreading model, we express the problem of controlling an epidemic outbreak in terms of spectral conditions involving the Perron-Frobenius eigenvalue. This enables formulation of the epidemic control problem as a Geometric Program (GP), for which we derive a convex characterization guaranteeing existence of an optimal solution. We consider two formulations of the epidemic control problem -- the first seeks an optimal vaccine and antidote allocation strategy given a constraint on the rate at which the epidemic comes under control. The second formulation seeks to find an optimal allocation strategy given a budget on the resources. The solution framework for both formulations also allows for control of an epidemic outbreak on networks that are not necessarily strongly connected. The thesis further proposes a fully distributed solution to the epidemic control problem via a Distributed Alternating Direction Method of Multipliers (ADMM) algorithm. Our distributed solution enables each node to locally compute its optimum allocation of vaccines and antidotes needed to collectively globally contain the spread of an outbreak, via local exchange of information with its neighbors. Contrasting previous literature, our problem is a constrained optimization problem associated with a directed network comprising non-identical agents. For the different problem formulations considered, illustrations that validate our solutions are presented. This thesis, in sum, proposes a paradigm shift from heuristics towards a convex framework for contagion control in networked populations.

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A CONVEX FRAMEWORK FOR EPIDEMIC CONTROL IN NETWORKS

To my family

and the intellectually curious

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ABSTRACT

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With networks becoming pervasive, research attention on dynamics of epidemic models in networked populations has increased. While a number of well understood epidemic spreading models have been developed, little to no attention has been paid to epidemic control strategies; beyond heuristics usually based on network centrality measures. Since epidemic control resources are typically limited, the problem of optimally allocating resources to control an outbreak becomes of interest. Existing literature considered homogeneous networks, limited the discussion to undirected networks, and largely proposed network centrality-based resource allocation strategies.

In this thesis, we consider the well-known Susceptible-Infected-Susceptible spreading model and study the problem of minimum cost resource allocation to control an epidemic outbreak in a networked population. First, we briefly present a heuristic that outperforms network centrality-based algorithms on a stylized version of the problem previously studied in the literature. We then solve the epidemic control problem via a convex optimization framework on weighted, directed networks comprising heterogeneous nodes. Based on our spreading model, we express the problem of controlling an epidemic outbreak in terms of spectral conditions involving the Perron-Frobenius eigenvalue. This enables formulation of the epidemic control problem as a Geometric Program (GP), for which we derive a convex characterization guaranteeing existence of an optimal solution. We consider two formulations of the epidemic control problem – the first seeks an optimal vaccine and antidote allocation

strategy given a constraint on the rate at which the epidemic comes under control. The second formulation seeks to find an optimal allocation strategy given a budget on the resources. The solution framework for both formulations also allows for control of an epidemic outbreak on networks that are not necessarily strongly connected. The thesis further proposes a fully distributed solution to the epidemic control problem via a Distributed Alternating Direction Method of Multipliers (ADMM) algorithm. Our distributed solution enables each node to locally compute its optimum allocation of vaccines and antidotes needed to collectively globally contain the spread of an outbreak, via local exchange of information with its neighbors. Contrasting previous literature, our problem is a constrained optimization problem associated with a directed network comprising non-identical agents. For the different problem formulations considered, illustrations that validate our solutions are presented. This thesis, in sum, proposes a paradigm shift from heuristics towards a convex framework for contagion control in networked populations.

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Chapter 1

Introduction

1.1 Motivation and Background

Epidemic outbreaks occur when there are reported cases of a contagious ailment beyond what is traditionally expected within a population. A major epidemic outbreak recorded in the last century is the 1918 global Spanish flu outbreak, which resulted in more deaths than was recorded in World War I, [2]. The last 5 decades has also seen several reported cases of HIV/AIDs, SARS [3] [4] and influenza-like epidemics [5] [6] including the very recent outbreak of the Middle East Respiratory Syndrome (MERS) [7] and Ebola outbreak [8], with several fatal cases reported in parts of the Middle East and West Africa. Reports from the World Health Organization (WHO), in addition, indicate that an estimated 13 million deaths a year (globally) result from infectious diseases [9] [10]. The problem of developing realistic epidemic spreading models and controlling the outbreak and spread of infectious diseases is not only important, but also topical given the current ebola virus epidemic [8]. That said, the mathematical framework of this thesis, fortunately, goes beyond just infectious

diseases as the models and solutions can be applied to other dynamical (epidemic-type) processes in networked populations. An example is the area of advertising and marketing, where the behavior and choice of people in one's immediate neighborhood or community is likely to affect an individual's decision.

In the event of an epidemic outbreak in a population, public health officials are tasked with determining the *cost-optimal* strategy to allocate usually limited resources – vaccines and/or antidotes amongst individuals and segments within the population to rapidly contain the spread of the outbreak.[1]. Contrasting much of the literature on epidemics in networks, as will be highlighted in Section 1.2, the key results of this thesis

- considers an epidemic process on a network comprising non-identical agents,
- assumes a directed contact network with weighted edges,
- presents a convex framework for simultaneously allocating vaccines and antidotes to control an epidemic outbreak, and
- proposes a fully distributed resource allocation strategy to control an epidemic outbreak.

Further, the solution we propose are applicable to epidemic processes occurring on networks that are not necessarily strongly connected without resorting to heuristics. The distinctions highlighted above are critical since many modern real networks are heterogeneous and directed; for example, the air traffic networks.

Resource allocation problems of this flavor are not unique to public health and infection propagation. As noted earlier, in marketing, for instance, where product

[1]Another component of the response plan may be a *speed optimal* approach, where the objective is to contain the spread as quickly as possible, without much focus on the associated cost of control.

advertisement by word-of-mouth and information diffusion is critical, of interest is how to optimally target markets or customers for advertising in a way that enables massive adoption of a product in the population in minimal time [11]. Problems of this sort also arise in telecommunication where optimal placement of base stations to achieve maximal end-to-end information flow rate is of interest. The question of how to optimally and efficiently allocate treatment and immunization resources, via a convex optimization framework, to control the outbreak of an epidemic in networked populations is the nucleus of this thesis. Contrasting existing literature that have focused on heuristics, our convex framework comprises a Semidefinite Programming (SDP) solution for a formulation of the epidemic control in undirected networks; as well as a Geometric Programming (GP) formulation that allows for simultaneous allocation of vaccines to control the infection rates and antidotes to control the recovery rates of individuals in the population.

1.2 Literature Review

Controlling the spread of epidemic outbreaks in populations is an age-old problem in human existence. The quest to understand how best to contain a contagion in populations upped research efforts at understanding dynamics of spreading processes within populations [12] [13] [14]. There have been several studies seeking to develop models that aid the understanding and analyses of the interplay of network structures and its effects on individual decisions/responses to information spreading in connected populations [15] [16] [17]. One of such studies – the earliest known mathematical model of epidemic dynamics, dates back to Daniel Bernoulli in the 18th century when he modelled the spread of small pox and argued for *variolation* as a way of increasing the life expectancy of the French [18].

A significant mathematical contribution to understanding the spread dynamics of epidemics came from the work of William Hamer and Ronald Ross [19]. In the early 1900s, Hamer and Ross developed a spatial model for the spread of malaria through mosquitoes. A key result from their model was that the spread of malaria could be controlled by reducing the population of mosquitoes per human below a *threshold*[2]. Ross' result of a population threshold for mosquitoes is the first known notion of an epidemic threshold in the literature.

Building on Ross' result from [19], Kermack and McKendrik in [20] developed epidemic models involving ordinary differential equations based on a population model. Also called compartmental models, population models partitioned a population of N individuals into 3 groups based on their state - Infected I, Susceptible S, and Recovered R. In Kermack and Kendrick's model, infected individuals can independently infect susceptible individuals with some probability β. They can also recover with probability δ.

[2]An epidemic threshold, τ_c, is a quantity that measures how potent the spread of an infection is; or the effective spread rate. τ_c is typically characterized by the infection and recovery rates of the population. When $\tau_c < 1$, the spread rate of the infection reduces and the infection dies out. On the other hand, $\tau_c > 1$, implies an increase in the spread rate resulting in an epidemic in the population. In the epidemiology literature, the threshold is more commonly known as the basic (infection) reproduction number and denoted by R_0.

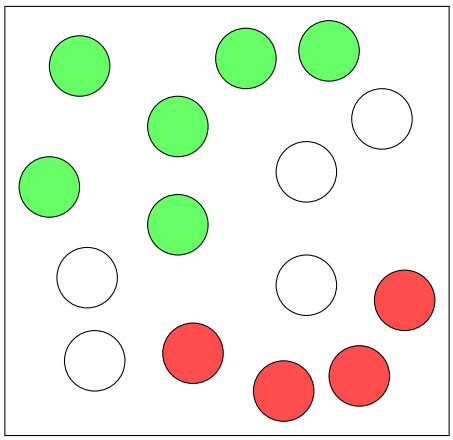

Figure 1.1: A population partitioned into infected (red), recovered (green) and susceptible (white) groups. In population-based models, the structure of inter-agent interactions has no bearing on how the contagion evolves.

If $S(t), I(t)$ and $R(t)$ respectively denote the number of susceptible, infected and recovered individuals in the population at time t, and $s(t) = S(t)/N,\ i(t) = I(t)/N$ and $r(t) = R(t)/N$ respectively represent the fraction of susceptible, infected and recovered individuals, then $s(t) + i(t) + r(t) = 1$, and the population of each group evolves as follows:

$$\frac{ds(t)}{dt} = -\beta s(t)i(t), \tag{1.1}$$

$$\frac{dr(t)}{dt} = \delta i(t), \tag{1.2}$$

$$\frac{di(t)}{dt} = \beta s(t)i(t) - \delta i(t). \tag{1.3}$$

In Kermack and Kendrick, each individual is assumed to have equal susceptibility to the infection with rate β, considered to be the the infection rate of the disease. Assuming each individual can make contact with every other individual in the network, each infected individual is able to transmit the disease with βN others per unit time, and the fraction of contacts by an infected individual with the susceptible population at time-step t is $s(t)$ resulting in (1.1). Assuming a uniform recovery rate

of δ, and an infected population of $i(t)$, the population of the recovered group $r(t)$ evolves according to (1.2), since all infected individuals may recover at the recovery rate of δ. Susceptible individuals become infected and infected infected individuals recover. Concurrent occurrence of these processes results in the evolution of the infected population as captured in (1.3).

A key result from the epidemic process with dynamics in (1.1)-(1.3) is that a significantly large fraction of the population is infected by the epidemic if and only if $\tau_c = \beta/\delta > 1$. The effective spread rate τ_c, highlighted earlier, measures the potency of infection. If $\tau_c > 1$, it implies the rate of infection of agents in the population exceeds the recovery rate, in which case, it is expected that an epidemic outbreak results.

Of interest to public health officials is how to control the potency of the infection relative to τ_c, since it determines whether or not an initial infection will result in an epidemic outbreak and spread through the population. Worth noting is that establishing the existence of an epidemic threshold τ_c, (determinable for different epidemic models), has been identified as one of the most significant contributions of mathematical analysis to the study of infectious disease and epidemiology. More mathematical contributions to epidemiology can be attributed to Bailey's work in [18], where a number of infection models were presented and characterized. In [21], Bailey also analyzed a number of related topics including recurrent epidemics, endemics, multistate spreading models, immunization programs and public health control, amongst others. These seminal works formed the underpinnings for modern literature in computational epidemiology.

While population models enabled description and analyses of epidemic dynamics and the evolution of partitions represented in Figure 1.1, they lacked the richness that networked models could provide. Population models are not, for instance, able

to capture and account for the effects of the structure of interpersonal contacts and interactions within the population on the spreading process. In addition, population models do not account for how the structural relationship of agents in the population can guide an epidemic containment strategy. Network-based models, on the other hand, are capable of these. Network models in epidemiology have been the focus of very recent work in the area of mathematical epidemiology [12] [13] [22]. Network models for the Susceptible-Infected-Susceptible (SIS) spreading models and its variants like the Susceptible-Infected-Recovered (SIR), Susceptible-Exposed-Infected-Susceptible (SEIS), the Susceptible-Alert-Infected-Susceptible (SAIS) have also been developed [14, 23, 24, 25, 26, 27, 28, 29, 30].

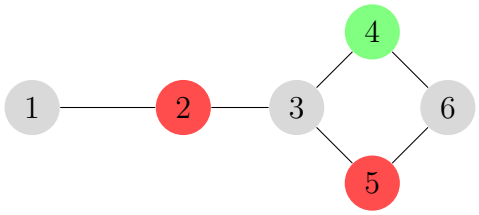

Figure 1.2: A network comprising susceptible (grey), infected (red), and recovered (green) nodes. In networked epidemic models, the effect of the network structure on the evolution of the contagion is accounted for.

One of the earliest network models in epidemiology is credited to Kephart and White [31], [32]. They adapted earlier epidemiological models of biological viruses to study the spread of viruses on a computer network, including the likelihood of an epidemic occurrence, and the dynamics and evolution of the expected number of infected computers as a function of time. In [31], they assumed homogeneity[3] in the graph structure considered and used only the degree of nodes in the network as the network characteristic. Kephart and White modeled connection between computers

[3]Homogeneous graph models assume that each agent in the network has a connection with every other agent in the network and the rate of infection is primarily based on the density of the infected group. Homogeneous models in epidemiology are based on *Regular* graphs.

as a directed graph with each edge between two connected nodes having an infection rate β and each node a recovery rate δ. If $i(t) = I(t)/N$ represents the infected population at time t, then

$$\frac{di(t)}{dt} = \beta[k]i(t)(1 - i(t)) - \delta i(t), \tag{1.4}$$

where the growth rate of the infected population is the product of the infection rate β per link and the number of infected neighbors, $\delta i(t)$ is the cure rate for a fraction of the infected nodes i, and $[k]$ is the average degree of the nodes. Similar to Ross' findings earlier in the 20th century, Kephart and White identified the *epidemic threshold* – the tipping point beyond which an epidemic ensues as $\tau_c = 1/[k]$, for their model. Noted earlier, a key assumption that makes the Kephart and White disease propagation model valid is the assumption that the connection between individuals or computers is sufficiently homogeneous.

Wang et al. in [33] generalized the Kephart and White model to arbitrary networks with more generic network characteristics than just node degrees using a discrete-time model. The model of Wang et al. assumed agents can either be susceptible or infected and considered a linearized SIS spreading model. Agents were assumed to have a common infection rate β and uniform curing rate δ. In their work, they studied the propagation of an outbreak by considering the probability of infection of each individual in a network, describing its evolution by the following dynamical system

$$\mathbf{p}(t + 1) = (\beta A + I - \delta I)\mathbf{p}(t), \tag{1.5}$$

(where I is the identity matrix, and A is an adjacency matrix representing the contacts between individuals in the networked population). The dynamics in (1.5) is

a *mean-field approximation* of a nonlinear SIS spreading model[4] resulting from the transitions between susceptible and infected states and the network effects. A major result of the Wang model was the existence of an epidemic threshold

$$\tau_c = \frac{1}{\lambda_{\max}(A)}, \tag{1.6}$$

where the matrix A is the adjacency matrix of the network and $\lambda_{\max}(A)$ is its largest eigenvalue. While the model of Wang et al. assumed homogeneous infection and recovery rates for agents in the network, the epidemic model studied in this thesis allows for heterogeneity in the disease parameters amongst agents in the network.

The literature on network epidemics in the last decade has grown. Newman in [13] presented solutions to a class of standard epidemic spreading models for different networks even for cases where agents have non-uniform, but correlated transmission rates and times spent in the infected state. Further, Boguna and Pastor-Satorras [34] analyzed the spread of a virus in correlated networks. There, they established that the epidemic threshold on networks with explicit correlations in node connectivities is the inverse of the largest eigenvalue of the connectivity matrix and not dependent on the connectivity distribution as is the case for uncorrelated networks. Their result confirmed a threshold earlier established by Wang et al [33]. The model considered in this thesis is based on [22], which is a heterogeneous, continuous-time version of the model discussed in [33]. Details of the model development follow in Chapter 2.

Problems on immunization strategies, aiming to keep the diseases propagation below epidemic levels, have also received some attention including [35] [36][37], where heuristics are presented and analyzed. Other efforts on epidemic control include [38] where the expected time until extinction of a spreading virus is analyzed in terms of

[4]In Chapter 2, details of the nonlinear spreading model used in in this thesis as well as [33] are presented

the curing resources available. The focus in Drakopoulos et al [38] was on undirected graphs and the proposed methods based on CutWidths of graphs were identified to have significant computational cost. In [39], the cost of an epidemic outbreak was studied using tools from random matrix theory. The authors focused on transient dynamics of the epidemic process and presented an economic viewpoint on the impact and cost of controlling the outbreak. A converse problem was studied in [40], where the authors sought to maximize the spread of influence. There, the authors present provable approximation guarantees for heuristics based on submodular functions.

In this thesis, we take a convex optimization-based approach that outperforms heuristics. The main focus and results of this thesis center around developing optimal resource allocation strategies and control schemes to contain the spread of epidemic outbreaks in connected networks with possibly weighted and directed edges of arbitrary structures. The primary problem addressed is – faced with an epidemic outbreak, how should limited treatment and protective resources be allocated across the network to guarantee that the infection spread dies out at a desired rate?

1.3 Contributions of the thesis

In the previous section, we presented an overview of the status of research on the modeling and control of epidemic processes in networks. A key contribution of this thesis to the literature is the development of a mathematical framework to control epidemic outbreaks on connected networks with arbitrary structure using techniques from convex optimization.

In particular we consider both strongly and not necessarily strongly connected networks comprising heterogeneous nodes and weighted, directed edges. Our solution framework is one that allows for concurrent computation of the optimum resource

allocation strategy to control the infection and recovery rates of agents in the network in a manner that contains the spread of an epidemic outbreak. Results of this thesis also show that though network centrality-based epidemic control strategies are intuitive and widely accepted, their performance is suboptimal. In addition, the key results in this thesis opens up a new area in the literature on control of epidemic processes on directed networks, contrasting most of the current research that focus on undirected networks. We also formulate the epidemic control task as a decentralized resource allocation problem and propose a fully distributed solution. The distributed solution shows that in the absence of a social planner, control of an outbreak in large networks can be effectively achieved without incurring the costs associated with central processing and computation of the optimum resources needed at each node or locality in the network.

In the thesis, we have framed the problem of controlling an epidemic outbreak as one of optimal (treatment and vaccination) resource allocation. As a result, the phrases *epidemic control problem* and *resource allocation problem* may be used interchangeably. Also, in the context of a network, the terms *agent*, *individual* and *node* will be assumed synonymous. The second chapter presents the spreading model studied in the rest of the thesis, assumptions of the model, and a formal statement of the epidemic control problem.

Chapter 3 briefly introduces measures of network centrality and presents a solution to the resource allocation problem when a binary decision (to either allocate or not allocate resources), needs to be made. After presenting a network centrality-based heuristic using a combinatorial formulation of the problem, common in the literature, we present a greedy algorithm and show that our greedy algorithm outperforms the network centrality-based method, using solutions to the Lagrange dual problem as a benchmark. The chapter concludes with a Semidefinite Programming

(SDP) formulation and solution when agents in the network have a uniform, fixed curing rate and the only control cost incurred is the investment on preventive resources. The SDP formulation relaxes the binary constraint in the combinatorial formulation.

In chapter 4, the focus shifts to more general, directed networks. After introducing Geometric Programs (GPs) and their convex characterizations, which guarantee globally optimal solutions, we solve the resource allocation problem on positively weighted, strongly connected networks via a GP formulation. Formulations of the epidemic control problem on positively weighted, directed but not necessarily strongly connected networks are presented as well in Chapter 4. For both cases – strongly connected and not necessarily strongly connected networks, we consider two formulations – one in which the decay rate of the probability of infection is constrained (with no constraint on the budget), and a second in which the available resources (budget) to expend are capped.

Chapter 5 of the thesis presents a fully distributed framework for solving the optimal resource allocation problem via a distributed implementation of the Alternating Direction Method of Multipliers (ADMM) optimization algorithm. The problem considered here is – how can agents in the network locally make optimal investments to adjust their infection and recovery rates by only locally interacting with their neighbors, in a way that collectively controls the spread of an epidemic outbreak? Based on a reformulation of the GP presented in Chapter 4, we briefly discuss convexity of the decentralized formulation allowing us to claim optimality of our solution via a D-ADMM algorithm.

For each solution method presented, we illustrate our results on some *real networks* including a subgraph of the social networking site *Facebook*, as well as a subnetwork of the Global Air Traffic network. For the distributed solution, a synthetic

strongly connected digraph is considered. A summary of the thesis and abridged discussion of open problems follow in Chapter 6, with referenced Appendices following. Though written as a monograph, this thesis is based on some published works including [1], [41],[42],[43],.

Chapter 2

Notation, model and problem

2.1 Preliminaries & Notation

In this chapter, we introduce notation and definitions of building blocks used in the rest of the thesis. In addition we discuss the epidemic spreading model under consideration, as well as relevant assumptions. We respectively denote by $\mathbb{R}^n_+$ and $\mathbb{R}^n_{++}$ the set of n-dimensional vectors with nonnegative and strictly positive entries. Vectors are denoted using boldface letters and matrices using capital letters. The letter I denotes the identity matrix and $\mathbf{1}$ the vector of all ones. $\mathbb{R}(z)$ denotes the real part of $z \in \mathbb{C}$.

2.1.1 Graph Theory

We define a weighted graph as $\mathcal{G} \triangleq (\mathcal{V}, \mathcal{E}, \mathcal{W})$, where $\mathcal{V} \triangleq \{v_1, \ldots, v_n\}$ is a set of n nodes, $\mathcal{E} \subseteq \mathcal{V} \times \mathcal{V}$ is a set of ordered pairs of nodes called edges, and the function $\mathcal{W} : \mathcal{E} \to \mathbb{R}_{++}$ associates *positive* real weights to the edges in $\mathcal{E}$. The node pair (v_j, v_i) form an undirected edge in $\mathcal{G}$. If $\mathcal{G}$ is a directed network, the pair (v_j, v_i) is an oriented edge from node v_j to node v_i. For an undirected graph $\mathcal{G}$, we

define the neighborhood of node v_i as $N_i \triangleq \{j : (v_j, v_i) \in \mathcal{E}\}$. When the graph $\mathcal{G}$, is directed, we define the in-neighborhood of node v_i (that is, the set of nodes with edges pointing towards v_i), as $N_i^{in} \triangleq \{j : (v_j, v_i) \in \mathcal{E}\}$. We respectively define the weighted in-degree and out-degree of node v_i as $\deg_{in}(v_i) \triangleq \sum_{j \in N_i^{in}} \mathcal{W}((v_j, v_i))$ and $\deg_{out}(v_i) \triangleq \sum_{j \in N_i^{out}} \mathcal{W}((v_j, v_i))$. A directed path from v_{i_1} to v_{i_l} in $\mathcal{G}$ is an ordered set of vertices $\left(v_{i_1}, v_{i_2}, \ldots, v_{i_{l+1}}\right)$ such that $\left(v_{i_s}, v_{i_{s+1}}\right) \in \mathcal{E}$ for $s = 1, \ldots, l$. A directed graph $\mathcal{G}$ is *strongly connected* if, for every pair of nodes $v_i, v_j \in \mathcal{V}$, there is a directed path from v_i to v_j.

We denote the adjacency matrix of a weighted, undirected graph $\mathcal{G}$, by $A = [a_{ij}]$, an $n \times n$ matrix defined entry-wise as $a_{ij} = \mathcal{W}(v_j, v_i)$ if edge $(v_j, v_i) \in \mathcal{E}$, and $a_{ij} = 0$ otherwise. When $\mathcal{G}$ is directed, the entry $a_{ij} = \mathcal{W}(v_j, v_i)$ if the edge $(v_j, v_i) \in \mathcal{E}$ points from v_j to v_i, and $a_{ij} = 0$ otherwise. Given an $n \times n$ matrix M, we denote by $\mathbf{v}_1(M), \ldots, \mathbf{v}_n(M)$ and $\lambda_1(M), \ldots, \lambda_n(M)$ the set of eigenvectors and corresponding eigenvalues of M, respectively, where we order them in decreasing order of their real parts, i.e., $\mathbb{R}(\lambda_1) \geq \mathbb{R}(\lambda_2) \geq \ldots \geq \mathbb{R}(\lambda_n)$. We respectively call $\lambda_1(M)$ and $\mathbf{v}_1(M)$ the dominant eigenvalue and eigenvector of M. We denote by $\rho(M)$, the spectral radius of M, which is the maximum modulus across all eigenvalue of M.

A set $\mathcal{S}$ is convex if whenever $\mathbf{x},\ \mathbf{y} \in \mathcal{S}$ and $\lambda \in [0,\ 1]$, the convex combination $(1-\lambda)\mathbf{x}+\lambda\mathbf{y} \in \mathcal{S}$. Further, a function defined on the convex set $\mathcal{S}$, $f : \mathbb{R}^n \to \mathbb{R}$ is said to be convex if $f((1-\lambda)\mathbf{x}+\lambda\mathbf{y}) \leq (1-\lambda)\mathbf{x}+\lambda\mathbf{y}$. For a detailed treatment of convex sets, convex functions and convex optimization problems, readers are referred to [44]. Because the focus is on networks with positively weighted edges, the adjacency matrix of all graphs is always nonnegative. Finally, a nonnegative matrix A is irreducible if and only if its associated graph $\mathcal{G}$ is strongly connected.

2.2 Epidemic spreading model

2.2.1 Spreading Model in Arbitrary Networks

The model studied in this thesis is a heterogeneous networked SIS epidemic spread model and is based on the continuous-time SIS epidemic model, called the N-intertwined SIS model, recently proposed by Van Mieghem et al. in [22]. There, the authors analyzed the effect of the network characteristics on a virus spreading process via a Markov model. A detailed comparison of an Exact 2^N-state Markov Chain and the N-intertwined model that employs a mean field approximation was presented. A discrete-time version of the model was first studied in [33].

We consider a network comprising n agents, where each agent can be in one of two states – *susceptible* to the infectious disease or *infected* by the disease. As time evolves, the state of each agent $v_i \in \mathcal{V}$ changes according to a stochastic process parameterized by its infection rate β_i, and curing rate δ_i. As part of our model, we assume the respective infection and curing rates β_i and δ_i at the different agents v_i can be tuned by injecting vaccine and treatment resources.[1]:

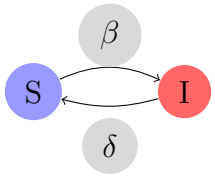

Figure 2.1: Node transitions between infected and susceptible states

The state of node v_i at time $t \geq 0$ is a binary random variable $X_i(t) \in \{0, 1\}$. The state $X_i(t) = 0$ indicates that node v_i is in the Susceptible state, which we denote as S and the state $X_i(t) = 1$ indicates that node v_i is in the Infected state, denoted I. Let the vector of states be defined as $X(t) = (X_1(t), \ldots, X_n(t))^T$. Next,

[1]This subsection of the thesis closely follows the development in [1]

we describe the state evolution of each node –

1. Suppose node v_i is susceptible to the infection at time t, it can transition to infected state during a small time interval $(t, t + \Delta t)$ with a probability that depends on its infection rate $\beta_i > 0$, the strength of its incoming connections from its neighbors $\{a_{ij},\ \text{for } j \in N_i^{in}\}$, as well as the states of its in-neighbors $\{X_j(t),\ \text{for } j \in N_i^{in}\}$. Figure 2.2 provides an illustration of this transition. Formally, the probability of v_i transiting to the infected state can be expressed as

$$\Pr\left(X_i(t+\Delta t) = 1 | X_i(t) = 0, X(t)\right) \ = \ \sum_{j \in N_i^{in}} a_{ij}\beta_i X_j(t)\,\Delta t \ + \ o(\Delta t), \quad (2.1)$$

where $\Delta t > 0$ is an asymptotically small time interval.

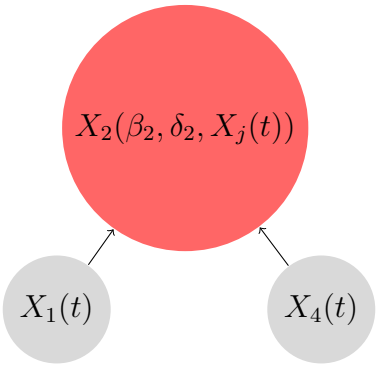

Figure 2.2: State transition of node v_2 due to effects of nodes v_1 and v_4

2. If node v_i is infected, its probability of transitioning to a susceptible state in the time interval $[t, t + \Delta t)$ depends on its recovery rate δ_i and is given by

$$\Pr(X_i(t+\Delta t) = 0 | X_i(t) = 1, X(t)) = \delta_i \Delta t + o(\Delta t). \quad (2.2)$$

At each time instant, the state of the entire network comprising n agents is defined by all possible combinations of states in which the agents can be at that time instance. Thence, the state space of the Markov chain for the continuous-time infection process defined above comprises 2^n states in the limit $\Delta t \to 0^+$. As the network size increases, the exponentially increasing state space makes this networked SIS epidemic model difficult to analyze. However, it is standard to make simplifying approximations that yield a mathematically tractable formulation. In line with that, our model development will adopt the so-called *mean field approximation* first used on SIS epidemic models in [33], and common in the literature [12] [22] [39] [45] [46].

Let the random variable $Q_i(t) \triangleq 1_{\{X_i(t)=1\}}$ be such that $Q_i(t) = 1$ when node v_i is infected and 0 otherwise. Hence, $Q_i(t)$ changes based on the state $X_i(t)$ of node v_i at each time step. From (2.1) and (2.2), the change of $Q_i(t)$ in a sufficiently small time interval Δt is [22]:

$$\frac{Q_i(t+\Delta t) - Q_i(t)}{\Delta t} = (1 - Q_i(t))\beta_i \sum_{j \in N_i^{in}} a_{ij} 1_{\{X_j(t)=1\}} - \delta_i Q_i(t).$$

Suppose we denote the probability of infection at node v_i as $p_i(t)$. Then, $p_i(t) \triangleq \Pr(X_i(t) = 1) = \mathbb{E}(Q_i(t))$ and we have that the probability of infection $p_i(t)$ at node v_i evolves according to

$$\frac{p_i(t+\Delta t) - p_i(t)}{\Delta t} = \beta_i \sum_{j \in N_i^{in}} a_{ij} p_j(t) - \delta_i p_i(t) - \mathbb{E}\left[1_{\{X_i(t)=1\}} \beta_i \sum_{j \in N_i^{in}} a_{ij} 1_{\{X_j(t)=1\}}\right]. \tag{2.3}$$

Since we have assumed the contact networks under consideration have no self loops; that is, the diagonal entries $[a_{ii}],\ i = 1, \ldots, n$ of the adjacency matrix A are zero,

we have that

$$\begin{aligned}\mathbb{E}\left(1_{\{X_i(t)=1\}}1_{\{X_j(t)=1\}}\right) &= \Pr\left(X_i(t)=1, X_j(t)=1\right) \\ &= \Pr\left(X_j(t)=1|X_i(t)=1\right)\Pr\left(X_i(t)=1\right).\end{aligned}$$

As $\Delta t \to 0$, it follows that [22],

$$\frac{dp_i(t)}{dt} = \beta_i \sum_{j\in N_i^{in}} a_{ij}p_j(t) - p_i(t)\left(\beta_i \sum_{j\in N_i^{in}} a_{ij}\Pr\left(X_j(t)=1|X_i(t)=1\right)+\delta_i\right). \quad (2.4)$$

If we make an implicit assumption that[2]

$$\Pr\left(X_j(t)=1, X_i(t)=1\right) = [\Pr\left(X_j(t)=1\right)][\Pr\left(X_i(t)=1\right)], \quad (2.5)$$

and taking into account the fact that $1-p_i(t) = \Pr[X_i(t)=0]$, the Markov differential equation for state $X_i(t)=1$ can be approximated by [22]:

$$\boxed{\frac{dp_i(t)}{dt} = (1-p_i(t))\beta_i \sum_{j=1}^{n} a_{ij}p_j(t) - \delta_i p_i(t),} \quad (2.6)$$

which we can more compactly write as

$$\boxed{\frac{d\boldsymbol{p}\left(t\right)}{dt} = \left(BA-D\right)\boldsymbol{p}\left(t\right) - P\left(t\right)BA\boldsymbol{p}\left(t\right),} \quad (2.7)$$

where $\boldsymbol{p}\left(t\right) \triangleq \left(p_1\left(t\right),\ldots,p_n\left(t\right)\right)^T$, $B \triangleq \mathrm{diag}(\beta_i)$ is a diagonal matrix comprising the infection rates across the nodes, $D \triangleq \mathrm{diag}\left(\delta_i\right)$ is a diagonal matrix comprising the curing rates across the nodes, and $P\left(t\right) \triangleq \mathrm{diag}(p_i\left(t\right))$. The *epidemic control problem*

[2]This assumption of independence is a component at the core of the *mean field approximation* for the SIS dynamics, typically done to derive a tractable representation of the complex spreading dynamics.

(which we mathematically formulate in the next section), is one of determining the optimum investment in vaccination and treatment resources at each agent v_i to control the outbreak of an epidemic. The vaccination and treatment resources applied at the nodes respectively lower and increase the infection and recovery rates to guarantee stability of (2.7). Of interest to us is to derive a sufficient condition, based on the dynamics in (2.7), that guarantee the probability of infections converge to zero exponentially fast across the network. When agents in the network have uniform infection and recovery rates, the next result presents such condition.

Proposition 2.1. *Given the SIS epidemic model with uniform infection and recovery rates across all agents, the probability of infection (from an initial infection), converges to zero exponentially fast if*

$$\frac{\beta}{\delta} < \tau_c = \frac{1}{\lambda_1(A)}, \tag{2.8}$$

where β and δ are respectively the infection and curing rates, and $\lambda_1(A)$ is the maximum eigenvalue of the network adjacency matrix A.

Since our model assumes a network of non-identical agents, Proposition 2.1 though intuitive, does not capture the heterogeneity of agents assumed in our model. We will derive an epidemic threshold based on the spread model in (2.7); that is, conditions under which the probability of an initial infection in the network converges to zero.

First observe that (2.7) has both a Disease-Free Equilibrium (DFE), as well as an Endemic Equilibrium (EE) [47] [48]. The DFE is the equilibrium at which the probability of infection is zero; that is, the expected number of agents in infected state is zero and all agents in the network are only susceptible to the infection. While the EE is the equilibrium at which the infection or disease in question is always present

without any re-introduction being necessary. In [42], the authors showed that we can upper bound the nonlinear system (2.7) via a linearization around its DFE. Hence, to stabilize, (2.7), it is sufficient to stabilize its linearized dynamics, which is a sufficient upper bound as we show in the the following result:

Proposition 2.2. *([42]) The dynamics* $(BA - D - P(t)BA)p(t)$ *is upper bounded by* $(BA - D)p(t)$.

Proof. Recall that (2.7) is the matrix-vector representation of (2.6). Hence,

$$\begin{aligned} \frac{dp_i(t)}{dt} &= \beta_i \sum_{j=1}^{n} a_{ij} p_j(t) - \delta_i p_i(t) - \beta_i p_i(t) \sum_{j=1}^{n} a_{ij} p_j(t) \\ &\leq \beta_i \sum_{j=1}^{n} a_{ij} p_j(t) - \delta_i p_i(t) \end{aligned} \tag{2.9}$$

since as part of the model we had assumed that $\beta_i, \delta_i, p_i(t)$ and $a_{ij} \geq 0$; which implies that

$$\frac{dp_i(t)}{dt} = \beta_i \sum_{j=1}^{n} a_{ij} p_j(t) - \delta_i p_i(t) \tag{2.10}$$

upper bounds (2.9) assuming they have identical initial conditions. □

Having established (2.10) as an upperbound to the original nonlinear dynamics, its matrix-vector form

$$\dot{\mathbf{p}}(t) = (BA - D)\mathbf{p}(t) \tag{2.11}$$

will be the focus of this thesis. Since $\mathbf{p}(t)$ are probabilities of infection of agents across the network, we are interested in conditions on the dynamics in (2.11) that guarantee its stability. We formally state this condition next.

Proposition 2.3. *Consider the heterogeneous networked SIS model in* (2.11)*, with*

$A \geq 0$, B, $D \succ 0$. Then, if the eigenvalue with largest real part of $BA - D$ satisfies

$$\mathbb{R}\left[\lambda_1\left(BA - D\right)\right] \leq -\varepsilon, \tag{2.12}$$

for some $\varepsilon > 0$, the disease-free equilibrium ($\boldsymbol{p}^* = \mathbf{0}$) is globally exponentially stable, i.e., $\|\boldsymbol{p}\left(t\right)\| \leq \|\boldsymbol{p}\left(0\right)\| K \exp\left(-\varepsilon t\right)$, for some $K > 0$.

Remark 2.4. When agents in the network have heterogeneous infection and recovery rates, (2.12) is the epidemic threshold of the linearized model. If the inequality fails to hold for some $\varepsilon > 0$, an epidemic is guaranteed to ensue.

The discrete-time analog of (2.10) is given as [33]:

$$\mathbf{p}(t+1) = \left(BA + I - D\right)\mathbf{p}(t). \tag{2.13}$$

Details of its derivation can be found in Appendix A. The necessary condition for control of an epidemic outbreak considering the spreading dynamics (2.13) is the spectral condition $\rho(BA + I - D) < 1$.

2.3 Epidemic Control Problem

An assumption in our model and problem set up is that the infection rate β_i and recovery rate δ_i for node v_i can be adjusted at a cost. We assume preventive resources (vaccines) at node v_i reduces its infection rate β_i within feasible intervals $0 < \underline{\beta}_i \leq \beta_i \leq \bar{\beta}_i$, with associated cost $f_i(\beta_i)$; and that treatment resources (antidotes) at node v_i ups its recovery rate δ_i within feasible intervals $0 < \underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$ and accrues a cost $g_i(\delta_i)$. Our choices for the associated cost functions is such that $f_i\left(\beta_i\right)$ is monotonically decreasing w.r.t. β_i and $g_i\left(\delta_i\right)$ is monotonically increasing w.r.t. δ_i.

The epidemic control problem considered is the following:

Problem 2.5. *Given a vaccination cost function $f_i(\beta_i)$, for β_i within a feasible interval $0 < \underline{\beta}_i \leq \beta_i \leq \bar{\beta}_i$, and treatment cost function $g_i(\delta_i)$, for δ_i within a feasible interval $0 < \underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$, determine the optimal allocation of vaccines and treatment resources to control the spread of an epidemic outbreak with an asymptotic exponential decaying rate ε for a minimum cost. Mathematically, this problem can be formulated as follows:*

$$\underset{\{\beta_i,\delta_i\}_{i=1}^{n}}{minimize} \sum_{i=1}^{n} f_i(\beta_i) + g_i(\delta_i) \tag{2.14}$$

$$subject\ to\ \mathbb{R}\left[\lambda_1\left(diag\left(\beta_i\right)A - diag\left(\delta_i\right)\right)\right] \leq -\varepsilon, \tag{2.15}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \tag{2.16}$$

$$\underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i,\ i = 1, \ldots, n, \tag{2.17}$$

where $f_i(\beta_i)$ is the vaccination cost incurred at node v_i, $g_i(\delta_i)$ is the treatment cost at node v_i, A is the adjacency matrix associated with the network.

In the problem above, (2.14) represents the cost of allocating vaccine and treatment resources across nodes in a network; and the spectral constraint (2.15) represents the critical point or epidemic threshold. Feasible bounds of the attained infection and recovery rates are represented by (2.16) and (2.17).

In Chapter 3 we will consider a variant of (2.14) - (2.17), and briefly highlight existing approaches based on network centrality measures. Following that, a greedy algorithm that outperforms centrality-based heuristics will be presented. The optimal solution to the dual problem, will be used as a benchmark in comparing the performance of the heuristics. Chapter 3 will then conclude by presenting a Semidefinite Programming approach to solving (2.14) - (2.17). Later, in Chapter 4, we will solve

(2.14) - (2.17) in its most general form, where the infection and recovery rates are simultaneously optimized and the network in question comprises positively weighted, directed edges.

Chapter 3

Optimal Resource Allocation Strategy: From Heuristics to a Convex Framework

The epidemic control problem formulated in the preceding chapter is the following:

Problem 3.1. *Given a vaccination cost function $f_i(\beta_i)$, for β_i within a feasible interval $0 < \underline{\beta}_i \leq \beta_i \leq \bar{\beta}_i$, and treatment cost function $g_i(\delta_i)$, for δ_i within a feasible interval $0 < \underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$, determine the optimal allocation of vaccines and treatment resources to control the spread of an epidemic outbreak with an asymptotic exponential decaying rate ε for a minimum cost. Concisely –*

$$\begin{aligned} &\underset{\{\beta_i,\delta_i\}_{i=1}^n}{minimize} \sum_{i=1}^{n} f_i(\beta_i) + g_i(\delta_i) \\ &subject\ to\ \mathbb{R}\left[\lambda_1\left(diag(\beta_i)\,A - diag(\delta_i)\right)\right] \leq -\varepsilon, \\ &\qquad \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \\ &\qquad \underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i,\ i = 1, \ldots, n. \end{aligned} \tag{3.1}$$

In this chapter, we will consider a combinatorial formulation of (3.1) on an undirected network, to which we apply heuristics.

3.1 Combinatorial Resource Allocation Problem

In considering a combinatorial variant of (3.1), the binary constraint $\beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}$ is imposed on β_i. Further, we assume that the network is undirected, in which case its associated network adjacency matrix is symmetric; that is, $A = A^T$ and that all agents in the network have a uniform curing rate $\delta_i = \delta$. The combinatorial formulation essentially seeks to determine the optimum minimum-cost allocation of vaccination resources to a subset of agents in the network to guarantee an exponentially stable, disease-free equilibrium. A formal statement of the problem is the following:

Problem 3.2. *Given curing rates $\{\delta_i : v_i \in \mathcal{V}\}$ and a vaccination cost function $f_i(\beta_i)$, for $\beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}$, determine the optimal allocation of vaccination resources to control the propagation of an epidemic outbreak with exponential decay rate ε with the least cost.*

Mathematically, this can be stated as:

$$\begin{aligned} &\underset{\{\beta_i\}_{i=1}^n}{\text{minimize}} \sum_{i=1}^{n} f_i\left(\beta_i\right) \\ &\text{subject to } \mathbb{R}\left[\lambda_1\left(\text{diag}(\beta_i)A - D\right)\right] \leq -\varepsilon, \\ &\beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}. \end{aligned} \tag{3.2}$$

Optimization problems with binary constraints as in (3.2) are known to be NP-hard; for instance, see [49] [50] [51]. We will solve (3.2) via heuristics based on

network centrality measures. Following that, we will employ a greedy algorithm and finally construct an upper bound that approximates the optimal solution. We obtain our bound via the dual problem using Lagrange duality theory. This method of approximating combinatorial problems has been employed in other works including [52] [53].

The constraint $\beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}$ in (3.2) indicates that each agent is either going to be vaccinated at a cost or not vaccinated at all. The set of agents $\{v_i\}$ chosen to be vaccinated will have their infection rates β_i set to $\underline{\beta}_i$, since a lower infection rate results in lower probability of infection. And the infection rates of agents not allocated any vaccines will be set to $\overline{\beta}_i$. Let us assume that the cost incurred at node v_i at the peak infection rate is zero; that is, $f_i(\overline{\beta}_i) = 0$, and that the maximum cost of vaccinating node v_i is attained when its infection rate is $\underline{\beta}_i$; that is, $f_i(\underline{\beta}_i) \triangleq c_i$, resulting from the affine function

$$f_i(\beta_i) \triangleq c_i \frac{\beta_i - \overline{\beta}_i}{\underline{\beta}_i - \overline{\beta}_i}. \tag{3.3}$$

We assume that the vaccination cost function $f_i(\beta_i)$ is monotonically decreasing in the interval $\beta_i \in [\underline{\beta}_i, \overline{\beta}_i]$, and that the vaccination cost function $f_i(\beta_i)$ is twice differentiable and satisfies

$$f_i''(\beta_i) \geq -\frac{2}{\beta_i} f_i'(\beta_i), \tag{3.4}$$

for $\beta_i \in [\underline{\beta}_i, \overline{\beta}_i]$. Hence, based on (3.3), the resource distribution strategy that minimizes $\sum_{i=1}^{n} f_i(\beta_i)$ is equivalent to that which maximizes $\sum_{i=1}^{n} c_i \beta_i$. Suppose we

let $\mathbf{c} \triangleq (c_1, \ldots, c_n)^T$ and $\mathbf{b} = (\beta_1, \ldots, \beta_n)^T$, we can reformulate (3.2) as

$$\begin{aligned} &\underset{b}{\text{maximize}} \quad \mathbf{c}^T\mathbf{b} \\ &\text{subject to } \mathbb{R}[\lambda_1(BA - D)] \leq -\varepsilon, \\ &\qquad \beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}, \quad i = 1, \ldots, n, \end{aligned} \tag{3.5}$$

where $B = \text{diag}(\mathbf{b})$. Presented with the resource allocation problem in (3.5), the most intuitive and common approach is to employ heuristics based on network centrality measures. In summary, nodes are chosen to be immunized or vaccinated based on how 'important' they are in the network. We briefly introduce this concept next.

3.2 Network Centrality Measures

Centrality measures are commonly used in the literature to describe influence of nodes in a network [54]. They are useful in, for instance, determining how fast information can spread in a network by exploiting the influence – *how central* – certain individuals are within the network. While there are several well-studied measures of centrality in the literature, we briefly introduce three measures of importance that will be used in the rest of the thesis.

3.2.1 Degree centrality

Degree centrality is the most intuitive of the common centrality measures in networks. It measures relative importance of nodes based on their degrees; that is, the number of neighbors each node has in the network (when the network is undirected). In directed graphs, two degree centrality measures can be defined – in-degree centrality and out-degree centrality. In-degree centrality of a node is the number of

incoming neighbors (edges) it has; while the out-degree of a node is the number of outgoing neighbors (edges) it has. Formally, suppose a directed network is represented by a nonnegative, possibly weighted adjacency matrix A, then

$$\deg_{in}(v_i) = \sum_{j=1}^{n} A_{ji} \tag{3.6}$$

$$\deg_{out}(v_i) = \sum_{j=1}^{n} A_{ij}. \tag{3.7}$$

For instance, in the undirected network in Figure 3.1, node v_3 has the highest degree and node v_1 has the least degree centrality.

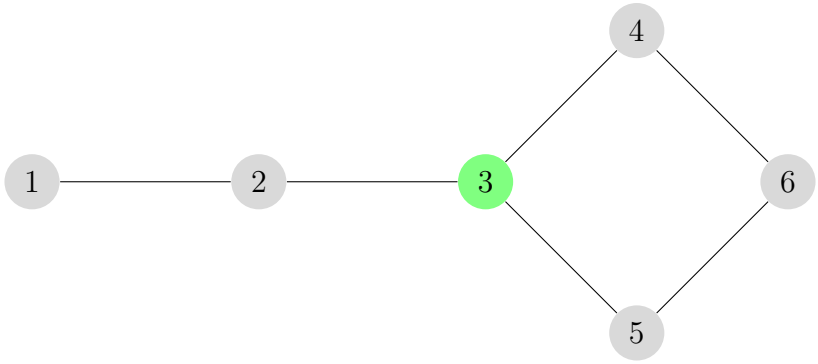

Figure 3.1: A 6−node graph with node v_3 having the highest degree centrality

3.2.2 Eigenvector centrality

Introduced by Bonacich in [54], eigenvector centrality is based on the idea that the centrality of a node in a network is determined by the centrality of its neighbors. It is a centrality measure that reflects the fact that not all connections in a network are equal. The eigenvector centrality measure is derived from the values of the leading eigenvector (associated with the largest eigenvalue) of the network adjacency matrix. Suppose, λ is the leading eigenvalue of the square, stochastic adjacency matrix A,

and $\mathbf{x}$ is its associated eigenvector, the following relation holds $\lambda\mathbf{x} = A\mathbf{x}$, and the eigenvector centralities of the nodes $v_i \in \mathcal{V}$ for $i = 1, \ldots, n$ can be expressed as:

$$\mathbf{x}(v_i) = \sum_{v_j \in \mathcal{V}} A_{ij}\mathbf{x}(v_j), \tag{3.8}$$

and the normalized entries of $\mathbf{x}$ give the centrality weights of the agents in the network. It is easy to verify that node v_3 in Figure 3.1 also has the highest eigenvector centrality in the network.

3.2.3 PageRank centrality

A variant of the eigenvector centrality, the pagerank centrality is a more common measure in directed networks and determines the importance of a node based on how central its incoming neighbors are. The computation of the PageRank centrality measure adds a damping factor α to the adjacency matrix[1]. The added damping factor helps avoid the potential for an infinite series of pageranks; that is, it lightens the effect of incoming pageranks of neighboring nodes to any given node. Mathematically, the PageRank centrality $\text{pr}(v_i)$ of a node v_i in a network is defined as:

$$\text{pr}(v_i) = \alpha \sum_j A_{ji} \frac{\text{pr}(v_j)}{L(j)} + \frac{1-\alpha}{N}, \tag{3.9}$$

where $0 \leq \alpha \leq 1$, $L(j) = \sum_j A_{ij}$ is the number of neighbors of node j (or number of outbound links in a directed graph). Relative to the eigenvector centrality measure, a major difference is the presence of a scaling factor $L(j)$, as well as the PageRank vector being a left eigenvector via the reversed indices A_{ji}.

Because centrality measures, indicate how certain nodes influence the network,

[1] α falls in the range $[0, 1]$, and is typically set to 0.85; see [55] for details.

including speed of information propagation, they have been widely proposed as a strategy in resource allocation and network immunization problems; see for instance, [36], [37], [56], [57], [58] [59].

3.2.4 Resource Allocation via Network Centrality

Given the binary constraint in (3.5), network centrality approach to vaccination is to immunize nodes in decreasing order of their centrality score.[2] In other words, nodes in the immunized group; that is, nodes with $\beta_i = \underline{\beta}$ will be those with high centrality scores, while nodes with $\beta_i = \overline{\beta}$ will be those with low centrality scores.

Let the centrality measures of the nodes be $\mathcal{C} = \{\xi_1, \ldots, \xi_n\}$, where ξ_i is the centrality score of node v_i and let $\mathcal{S}_k$ be the set of nodes that have been allocated vaccination resources at time-step k. At each epoch, the node with the highest centrality score is added to the set $\mathcal{S}_k$ until the spectral constraint in $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k}A - D)) \leq -\varepsilon$ is satisfied, where $B_{\mathcal{S}_k}$ is the diagonal matrix of infection rates comprising the subset $\mathcal{S}_k \subseteq \mathcal{V}$ of nodes in the network that have been vaccinated. For instance, in a 5-node network where nodes v_2 and v_4 have been immunized by time-step k,

$$B_{\mathcal{S}_k} = \begin{pmatrix} \overline{\beta}_1 & 0 & \ldots & & 0 \\ 0 & \underline{\beta}_2 & & & \vdots \\ \vdots & & \overline{\beta}_3 & & \\ & & & \underline{\beta}_4 & \\ 0 & \ldots & & & \overline{\beta}_5 \end{pmatrix} \tag{3.10}$$

A network centrality-based algorithm is presented in Algorithm 1.

[2]The choice of centrality measure is not relevant in the strategy, since the nodes in the network can be ordered in decreasing order of any centrality measure used.

Algorithm 1 Centrality-based Algorithm for solving (3.2)

Given ε
Start with $\mathcal{S}_1 = \{\ \}$
repeat
    $\mathcal{S}_{k+1} = \mathcal{S}_k + v_i$, where $v_i = \max_i\{\xi_1, \ldots, \xi_n\}$ ▷ Node with next highest centrality score is immunized
    Set $k = k + 1$
until $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k}A - D)) \leq -\varepsilon$ ▷ Epidemic control condition satisfied

Observe that at each epoch k, the choice of the node to be added to $\mathcal{S}_k$ is independent of the state dynamics $BA - D$, and dependent solely on the network adjacency matrix A, from which the centrality scores $\xi_1, \ldots, \xi_n$ were derived. This observation will, in part, explain why Algorithm 1 has a lower performance relative to Algorithms 2 and 3.

3.2.5 Resource Allocation via a Greedy Algorithm

Again, let $\mathcal{S}_k$ be the set of nodes that have been vaccinated at iteration k; and let the matrix $B_{\mathcal{S}_k}$ be the diagonal matrix of infection rates where the subset $\mathcal{S}_k \subseteq \mathcal{V}$ of nodes in the network have been vaccinated. The greedy algorithm presented here to solve (3.5), depends not only on the network adjacency matrix A, but also on changes to the spectrum of $B_{\mathcal{S}_k}A - D$ when the set of vaccinated nodes in $\mathcal{S}_k$ change. Since the nodes are assumed to have identical costs c_i, the goal of the algorithm is to vaccinate the fewest nodes that result in satisfaction of the spectral constraint $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k}A - D)) \leq -\varepsilon$. Hence, at each step of the algorithm, to the set $\mathcal{S}_k$, we add the node i_k that provides the most significant benefit per unit cost; that is,

$$i_k = \arg\max_i \ \Delta(i, \mathcal{S}_k), \tag{3.11}$$

where

$$\Delta(i, \mathcal{S}_k) \triangleq \frac{\mathbb{R}(\lambda_1(B_{\mathcal{S}_k} A - D)) - \mathbb{R}(\lambda_1(B_{\mathcal{S}_{k+\{i\}}} A - D))}{c_i}, \tag{3.12}$$

until the spectral constraint $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k} A - D)) \leq -\varepsilon$ is satisfied. The greedy algorithm is formally presented in Algorithm 2.

Algorithm 2 A Greedy Algorithm for solving (3.2)

Given ε
Start with $\mathcal{S}_1 = \{\ \}$
repeat
    $i_k \triangleq \arg\max_i \quad \Delta(i, \mathcal{S}_k)$
    $\mathcal{S}_{k+1} = \mathcal{S}_k + \{i_k\}$ ▷ Node with maximum benefit per unit cost is immunized
    Set $k = k + 1$
until $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k} A - D)) \leq -\varepsilon$ ▷ Epidemic control condition satisfied

3.2.6 Reverse Greedy Algorithm

We present a variant of Algorithm 2, called the *Reverse Greedy* algorithm. The Reverse Greedy algorithm begins with all nodes assumed to be vaccinated; that is, the set $\mathcal{S}_1 = \mathcal{V}$ and $\beta_i = \underline{\beta}$, $\forall\, v_i \in \mathcal{V}$. It then iteratively removes nodes from the set $\mathcal{S}_k$ at each step of the algorithm until the spectral radius constraint $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k} A - D)) > -\varepsilon$ is satisfied. The set of vaccinated nodes is then $\mathcal{S}_{k-1}$. Implementation of the reverse greedy algorithm as presented in Algorithm 3 on (3.5) results in a higher objective, compared to Algorithm 2. The reverse greedy algorithm is summarized in Algorithm 3.

Algorithm 3 Reverse Greedy Algorithm for solving (3.2)

Given ε
Start with $\mathcal{S}_1 = \mathcal{V}$
repeat
    $i_k \triangleq \arg\min_i \quad \Delta(i, \mathcal{S}_k \setminus \{i\})$
    $\mathcal{S}_{k+1} = \mathcal{S}_k - \{i_k\}$
    Set $k = k + 1$
until $\mathbb{R}(\lambda_1(B_{\mathcal{S}_k} A - D)) > -\varepsilon$ ▷ Epidemic control condition is satisfied

3.3 Bounds on performance of the Greedy algorithm

Via Lagrange duality theory we present quality guarantees (a benchmark) for the performance of the Algorithms 1, 2 and 3.

Theorem 3.3. *([42]) Given the following optimization problem*

$$\begin{aligned} T_C^* = \underset{b}{maximize} \quad & \mathbf{c}^T\mathbf{b} \\ subject\ to\ & (D-\varepsilon I)B^{-1} - A \succeq 0, \\ & \beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}, \quad i = 1, \ldots, n, \end{aligned} \tag{3.13}$$

the primal optimal solution T_C^* is upper bounded by D_C^*, which is the solution to the following dual problem

$$\begin{aligned} D_C^* = \underset{Z,\mathbf{u}}{minimize} \quad & \mathbf{1}^T\mathbf{u} - trace(AZ) \\ subject\ to \quad & u_i \geq c_i\overline{\beta}_i + \frac{\delta_i}{\overline{\beta}_i}Z_{ii}\ \forall\ i, \\ & u_i \geq c_i\underline{\beta}_i + \frac{\delta_i}{\underline{\beta}_i}Z_{ii}\ \forall\ i \\ & Z \succeq 0. \end{aligned} \tag{3.14}$$

Proof. To prove this, we first write the matrix $(D-\varepsilon I)B^{-1}-A$ as $\sum_i e_i e_i'(\delta_i-\varepsilon)/\beta_i - A$, where e_i is the unit vector in the standard basis. Using this new expression, the

Lagrangian of 3.13 can be expressed as

$$\mathcal{L}(b, Z) = \mathbf{c}^T \mathbf{b} + \text{trace}\left(Z \left(\sum_i e_i e_i' \frac{\delta_i}{\beta_i} - A \right) \right),$$
$$\text{subject to} \quad \beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}, \tag{3.15}$$
$$Z \succeq 0.$$

The Lagrangian above can be simplifies to

$$\mathcal{L}(b, Z) = \sum_i \left(c_i \beta_i + \frac{\delta_i}{\beta_i} Z_{ii} - \text{trace}(ZA) \right), \tag{3.16}$$

with the binary constraint $\beta_i \in \{\underline{\beta}_i, \overline{\beta}_i\}$ and $Z \succeq 0$ kept implicit as domain constraints. To drive the dual objective function, the Lagrangian is maximized with respect to the primal variable β_i as follows:

$$q(Z) = \sum_i \left(\max \; c_i \beta_i + \frac{\delta_i}{\beta_i} Z_{ii} - \text{trace}(ZA) \right). \tag{3.17}$$

By exploiting the properties of trace, which enabled separation of the Lagrangian per node, and given the binary constraint on β_i at each node, the maximization of the Lagrangian can be locally carried out at each node. First, let

$$u_i = \max \left\{ c_i \overline{\beta}_i + \frac{\delta_i}{\overline{\beta}_i} Z_{ii}, \; c_i \underline{\beta}_i + \frac{\delta_i}{\underline{\beta}_i} Z_{ii} \right\}. \tag{3.18}$$

To construct the dual, we use an epigraph formulation of (3.17), expressing it as

$$q(Z, \mathbf{u}) = \sum_{i=1}^{n} u_i - \text{trace}(ZA), \tag{3.19}$$

$$\text{subject to} \quad u_i \geq c_i \overline{\beta}_i + \frac{\delta_i}{\overline{\beta}_i} Z_{ii} \; \forall \; i, \tag{3.20}$$

$$u_i \geq c_i \underline{\beta}_i + \frac{\delta_i}{\underline{\beta}_i} Z_{ii} \; \forall \; i. \tag{3.21}$$

The dual is a minimization problem and $q(Z, \mathbf{u})$ in (3.17) is strictly increasing in u, then either (3.20) or (3.21) is binding to guarantee that (3.18) holds at optimality. The dual in (3.14) is obtained by minimizing (3.19), subject to the epigraph constraints (3.20), (3.21), as well as domain constraints $Z \succeq 0$. And by weak duality theory, the solution to the dual problem satisfies $D_C^* \geq T_C^*$ [44]. Hence, for a problem of the form (3.13), we can upper bound our solution by

$$T_C^* - \mathbf{c}^T \mathbf{b} \leq D_C^* - \mathbf{c}^T \mathbf{b} \tag{3.22}$$

by solving the dual problem. And since strong duality does not hold, equality of the terms $\mathbf{c}^T \mathbf{b}$ and D_C^* will not hold. □

Later in the chapter, we will illustrate performance of the algorithms above in relation to the Lagrange dual bound just presented.

3.4 Resource Allocation via Semidefinite Programming

In this section, we take a Semidefinite programming approach to solve the epidemic control problem first presented in (3.2), after relaxing the binary constraint, allowing

β_i to take values in the interval $[\underline{\beta}, \overline{\beta}]$. Again, the goal here is to compute the cost optimal vaccine allocation to each node in the network that a DFE at an exponential rate. The specific problem considered is the following:

$$\begin{aligned} &\underset{\{\beta_i\}_{i=1}^n}{\text{minimize}} \ \sum_{i=1}^{n} f_i\left(\beta_i\right) \\ &\text{subject to } \mathbb{R}\left[\lambda_1\left(BA - D\right)\right] \leq -\varepsilon, \\ &\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \end{aligned} \tag{3.23}$$

where $f_i(\beta_i)$ is the immunization cost incurred at node v_i, A is a symmetric adjacency matrix, $B = \text{diag}(\beta_i)$ is a diagonal matrix of the infection rates and $D = \text{diag}(\delta)$ is a diagonal matrix of the recovery rates.

Definition 3.4. Semidefinite Programs (SDPs) are optimization problems that minimize a linear cost function subject to linear matrix inequality constraints, and are of the form:

$$\begin{aligned} &\underset{\mathbf{x}}{\text{minimize}} \ \mathbf{c}^T\mathbf{x} \\ &\text{subject to } F(\mathbf{x}) \geq 0. \end{aligned} \tag{3.24}$$

Where $F(\mathbf{x}) \triangleq F_0 + \sum_{i=1}^{n} \mathbf{x}_i F_i$. The problem data are the vector $\mathbf{c} \in \mathbb{R}^n$ and the symmetric matrices $F_0, \ldots, F_n \in \mathbb{R}^{n\times n}$. The inequality $F(\mathbf{x}) \geq 0$ means that $F(\mathbf{x})$ is positive semidefinite; that is $\mathbf{y}^T F(\mathbf{x})\mathbf{y} \geq 0$, for all $\mathbf{y} \in \mathbb{R}^n$. The inequality $F(\mathbf{x}) \geq 0$ is called Linear Matrix Inequality (LMI) and the problem (3.24), an SDP. SDPs are convex optimization problems since their cost functions as well as constraints are convex.

To derive an SDP formulation for (3.23), we first present a result that enables expression of the spectral constraint in (3.23) as an LMI.

Lemma 3.5. *([42]) Given a symmetric A, $B = diag(\beta_i)$, and $D = diag(\delta)$, then $\mathbb{R}(\lambda_1(BA - D)) \leq -\varepsilon$, if $(D - \varepsilon I)B^{-1} - A \succeq 0$.*

Proof. By a similarity transformation, we can express the matrix $BA - D$ as $B^{1/2}AB^{1/2} - D$ by pre and post-multiplying $BA - D$ by $B^{-1/2}$ and $B^{1/2}$ respectively. The eigenvalues of $B^{1/2}AB^{1/2} - D$ are real, since it is symmetric. Hence, $\lambda_1(BA - D) \leq -\varepsilon$ if

$$\lambda_i((D - \varepsilon I) - BA)) = \lambda_i\left((D - \varepsilon I) - B^{1/2}AB^{1/2}\right) \geq 0,$$

which is equivalent to $((D - \varepsilon I) - B^{1/2}AB^{1/2}) \succeq 0$. If we apply a congruence transformation to $(D - \varepsilon I) - B^{1/2}AB^{1/2}$ by pre and post multiplying it by $B^{-1/2}$, we obtain $\lambda_1(BA - D) \leq -\varepsilon$ if $(D - \varepsilon I)B^{-1} - A \succeq 0$, which proves the result. □

Based on Lemma 3.5, (3.23) can be reformulated as the following (still nonconvex) optimization problem

$$\begin{aligned} \underset{\{\beta_i\}_{i=1}^n}{\text{minimize}} \ & \sum_{i=1}^{n} f_i\left(\beta_i\right) \\ \text{subject to } & (D - \varepsilon I)B^{-1} - A \succeq 0, \\ & \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i. \end{aligned} \tag{3.25}$$

Observe that though the cost function f_i in 3.25 had been restricted to be convex, the LMI constraint is not convex because of the negative exponent on the matrix B. To address this challenge and obtain a convex problem, we carry out the following change of variables; let $\gamma_i = \beta_i^{-1}$, and $\Gamma \triangleq diag(\gamma_i)$. By the earlier stated strong convexity assumption on the cost function f_i, the change of variable from β_i to γ_i still leaves us with a convex objective function. This can be verified via a second-order

convexity conditions as follows:

$$\frac{d^2}{d\gamma_i^2} f_i(\gamma_i^{-1}) = f_i''(\gamma_i^{-1})\frac{1}{\gamma_i^4} + 2f_i'(\gamma_i^{-1})\frac{1}{\gamma_i^3} \geq 0,$$

for each node $v_i \in \mathcal{V}$. Since the sum of convex functions is trivially convex, we have that (3.26) is convex. With the change of variable from β_i to γ_i, the spectral constraint in (3.26) becomes convex in the following optimization problem:

$$\begin{aligned} \underset{\{\gamma_i\}_{i=1}^n}{\text{minimize}} \ & \sum_{i=1}^{n} f_i(\gamma_i) \\ \text{subject to } & (D - \varepsilon I)\Gamma - A \succeq 0, \\ & \frac{1}{\overline{\beta}_i} \leq \ \gamma_i \leq \frac{1}{\underline{\beta}_i}, \end{aligned} \tag{3.26}$$

which can be solved efficiently.

Thus far, in this chapter, we have formulated the problem of resource allocation to control an epidemic outbreak, based on a spread model in which the curing rates of agents are uniform, and the contact network is undirected. We briefly presented widely proposed network centrality-based approach, two greedy algorithms and an SDP-based convex framework (with a relaxed binary constraint on the infection rates) to solve the problem. As will be illustrated in the simulations in section 3.5, the somewhat positive correlation observed between the resources allocated to nodes across the network and their centrality measures indicate that the convex programming formulations and solutions take into account more general (algebraic and structural) network characteristics without resorting to heuristics, thus, encompassing centrality-heuristics.

3.5 Numerical Results

Illustrated in this section are results from the solution methods presented above. We compare the solutions obtained from the network centrality and greedy algorithms, as well as results of the SDP framework. We use a subgraph from the social networking website, Facebook comprising $n = 247$ nodes. We assume a cost function defined in (3.3). We further assume that all agents in the network have a uniform recovery rate of $\delta = 0.1$. The epidemic threshold here is $\tau_c = \delta/\lambda_1(A)$. If the infection rates of the nodes satisfy $\beta > \tau_c$, an epidemic ensues. If however, the infection rates at the node $\beta < \tau_c$, then the outbreak is contained.

3.5.1 Illustration of the SDP Approach

The largest eigenvalue of the network considered is $\lambda_1(A) = 13.52$. By Proposition 2.3, assuming a uniform curing rate δ, the choice of the maximum $\overline{\beta} = 2.4\tau_c$ and minimum $\underline{\beta} = 0.2\overline{\beta}$ infection rates are such that when nodes are node vaccinated; that is, $\beta_i = \overline{\beta}\ \forall i$, the DFE is unstable; and when nodes are vaccinated; that is, $\beta_i = \underline{\beta},\ \forall i$, the DFE is stable. Problem (3.26) is solved using CVX, a package for specifying and solving convex programs [60] [61].

Results of our simulations using the parameters above are presented in Figure 3.2. The plot relates the immunization cost per node relative to its degree centrality measure. Each point (blue circle) in the plot represents an individual in the undirected 247-node network considered. The immunization cost function $f_i(\beta_i)$ used was normalized to take values in the interval $[0, 1]$ (indicated on the horizontal axis). And the plot illustrates the relationship between the degree of each individual (vertical axis), and the cost of immunizing each individual (horizontal axis). We observe that nodes with degree less than approximately 8 accrue no immunization cost. Nodes

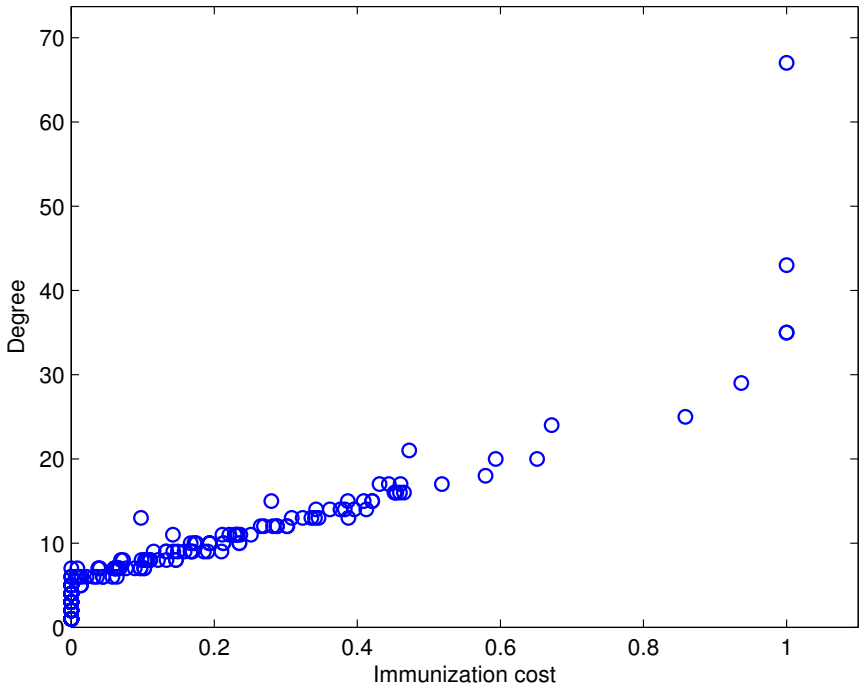

Figure 3.2: The plot above illustrates the relationship between the node degree centralities (on the vertical axis), and the immunization cost per node (on the horizontal axis) using a subgraph of the social networking site Facebook. Each blue circle represents an individual in the 247-node subgraph.

with degree greater than 8 are immunized at a cost.

3.5.2 Solutions to Combinatorial Formulations

In this section, we illustrate results of the network centrality and greedy algorithms 1, 2 and 3 on the same social network comprising $n = 247$ nodes, and compare the outcomes of their solutions. The dual problem is solved using CVX, a package for specifying and solving convex programs [60] [61]. The recovery rate was, again, fixed at $\delta = 0.1$, and upper and lower bounds on the infection rate chosen such that when no nodes are immunized, the DFE of the spread dynamics is unstable; and when nodes are immunized, the DFE of the spread dynamics is stable. In particular, we choose $\overline{\beta} = 3\tau_c$ and $\underline{\beta} = 0.25\overline{\beta}$. Recall, the objective is $\max_{\mathbf{b}} \ \mathbf{c}^T\mathbf{b}$. After solving the problem using the different algorithms discussed earlier, we find that the dual problem yields the optimal solution, and the vaccination based on eigenvector centrality resulted in the least optimal solution. The dual solution obtained was 4.19126; the solution from the reverse greedy algorithm was 3.7977; the greedy algorithm resulted in a solution of 3.7811, and the degree and eigenvector centrality heuristics respectively yielded 3.3486 and 1.6022. As expected, the dual solution serves as a benchmark and outperforms the other algorithms. We see that the greedy and reverse greedy algorithm performs better than the network centrality-based algorithms.

Figure 3.3 illustrates the relationship between the fraction of nodes with a given eigenvector centrality measure and the node eigenvector centrality scores. It shows that all the algorithms favor the vaccination of nodes with higher eigenvector centralities. The horizontal axis of Figure 3.4 represents the node degrees plotted in log scale; and the vertical axis are the fraction of nodes with a certain degree that are immunized. Though a slight correlation between the immunized nodes and cen-

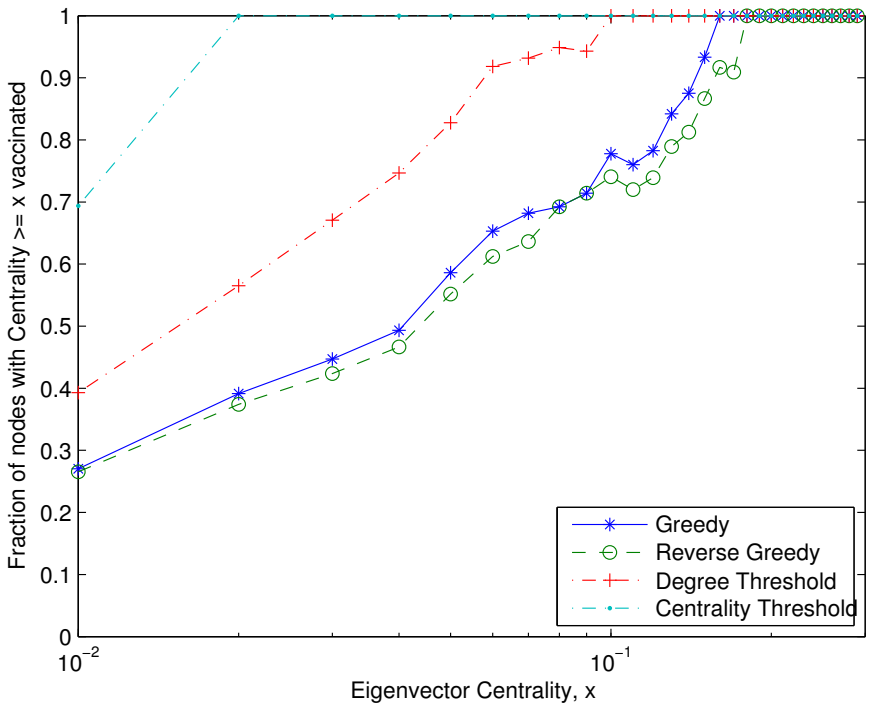

Figure 3.3: For each algorithm considered, this graph illustrates the relationship between the eigenvector centrality of each node and the fraction of nodes exceeding a certain centrality that are immunized.

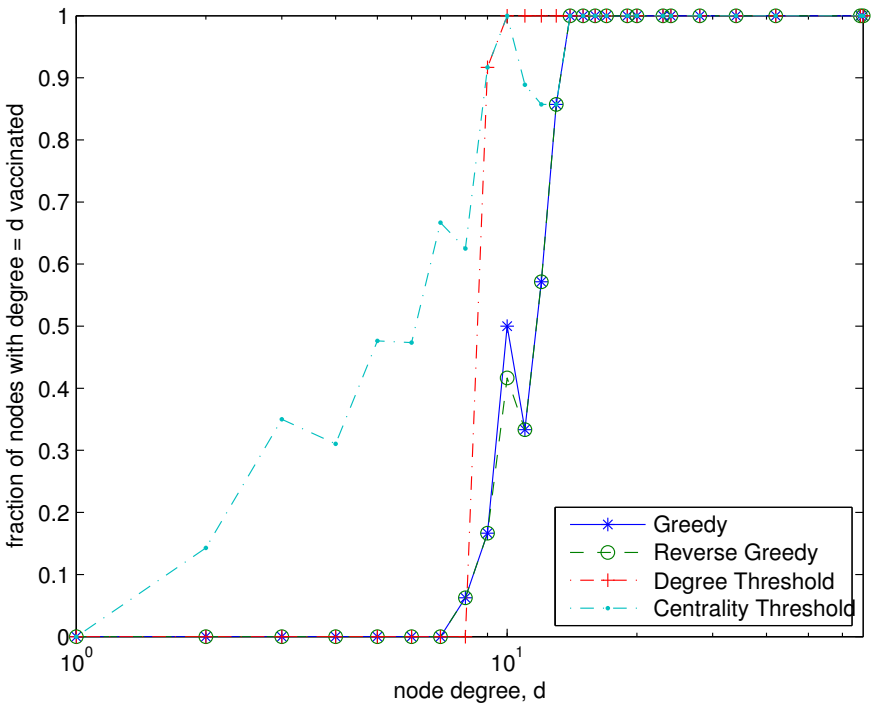

Figure 3.4: This graph illustrates the relationship between the fraction of nodes with a certain degree that are immunized and the node degrees for each of the algorithm employed

trality measures is observed (in both the degree and eigenvector centrality), we find that the curves are not strictly monotone increasing in the fraction of nodes vaccinated, which indicate that vaccinating nodes with higher centrality scores does not always yield an optimal vaccination strategy. Centrality measures depend solely on the graph structure and not on the entirety of the 'state matrix' $BA - D$. As a result, the centrality measures of the network does not change when the set of vaccinated nodes in the matrix $BA - D$ changes. This is the primary reason why network centrality-based heuristics perform poorly relative to the *Greedy* and *Reverse Greedy* algorithms presented.

In this chapter, we have considered a design problem where the objective was to optimally allocate vaccines to control an epidemic outbreak. We considered a combinatorial formulation where agents in the network are either vaccinated or not; we also considered a non-binary scenario where the vaccination investment per node is allowed to vary based on the desired infection rate at that node. We have established that though intuitive, network centrality-based heuristics are suboptimal.

Chapter 4

Resource Allocation for Epidemic Control via Geometric Programming

In the previous chapter, we considered the following optimization problem

$$\begin{aligned} &\underset{\{\beta_i\}_{i=1}^n}{\text{minimize}} \sum_{i=1}^{n} f_i(\beta_i) \\ &\text{subject to } (D-\varepsilon I)B^{-1} - A \succeq 0, \\ &\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i. \end{aligned} \tag{4.1}$$

where the goal was to optimally allocate vaccines to reduce the infection rate β_i across all nodes in the network, to contain the spread of an outbreak. In (4.1), the matrix A was assumed to be symmetric and the decision variable was $B = \text{diag}(\beta_i)$.

In this chapter we consider the resource allocation problem in which both optimum preventive (vaccines) and corrective resources (antidotes) are concurrently

allocated to the nodes to reduce the infection rates β_i and recovery rates δ_i within feasible intervals $0 < \underline{\beta}_i \leq \beta_i \leq \bar{\beta}_i$, and $0 < \underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$ for each node v_i at respective costs $f_i(\beta_i)$ and $g_i(\delta_i)$. While the resource allocation problems in chapter 3 considered undirected networks, the problems discussed in this chapter are for general, possibly weighted, directed, strongly connected networks[1]. Two problems are presented – one in which the exponential decay rate of the probability of infection is constrained, which we term the *rate-constrained* problem; and a second in which the total investment (on preventive and corrective resources) cost across all nodes is constrained – termed the *budget-constrained* problem. These problems are formulated as Geometric Programs, which we introduce after formally stating the resource allocation problems in their natural form.

4.1 Rate-constrained resource allocation problem

In the rate-constrained problem, the objective is to determine the cost-optimal allocation of vaccines and treatment resources that attain a desired exponential decay rate in the probability of infection for all nodes in the network.

Problem 4.1. *Given a positively weighted directed network with associated adjacency matrix A, sets of node cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$ and bounds on the infection and curing rates $0 < \underline{\beta}_i \leq \beta_i \leq \bar{\beta}_i$, and $0 < \underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$ respectively, an exponential decay rate $\varepsilon > 0$, determine the cost-optimal distribution of vaccines and antidotes to attain the desired decay rate in the probability of infection.*

[1]Later in this chapter, we adapt our solution framework to contact networks that are not necessarily strongly connected.

We state this as the following optimization problem:

$$\underset{\{\beta_i,\delta_i\}_{i=1}^n}{\text{minimize}} \sum_{i=1}^{n} f_i(\beta_i) + g_i(\delta_i) \tag{4.2}$$

$$\text{subject to } \mathbb{R}(\lambda_1(\text{diag}(\beta_i)A - \text{diag}(\delta_i))) \leq -\varepsilon, \tag{4.3}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \tag{4.4}$$

$$\underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i,\ i = 1, \ldots, n, \tag{4.5}$$

where (4.2) is the total investment, (4.3) constrains the decay rate to ε, and (4.4)-(4.5) maintain the infection and recovery rates in their feasible limits.

4.2 Budget-constrained resource allocation problem

The objective in the budget-constrained problem is to maximize the exponential decay rate $\hat{\varepsilon}$, given a budget (limit) $\mathcal{M}$ on the investment in protective and corrective resources. The budget-constrained allocation problem is formulated as follows:

Problem 4.2. *Given a positively weighted, directed network $\mathcal{G}$ with adjacency matrix A, a set of cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$, bounds on the infection and recovery rates $0 < \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i$ and $0 < \underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i$, $i = 1, \ldots, n$, and an investment budget $\mathcal{M}$; find the cost-optimal distribution of vaccines and antidotes to maximize the exponential decay rate $\hat{\varepsilon}$.*

Concisely, we have the following optimization program:

$$\underset{\hat{\varepsilon},\{\beta_i,\delta_i\}_{i=1}^n}{\text{maximize}}\ \hat{\varepsilon} \tag{4.6}$$

$$\text{subject to } \mathbb{R}\left[\lambda_1\left(\text{diag}\left(\beta_i\right)A - \text{diag}\left(\delta_i\right)\right)\right] \leq -\hat{\varepsilon}, \tag{4.7}$$

$$\sum_{i=1}^{n} f_i\left(\beta_i\right) + g_i\left(\delta_i\right) \leq \mathcal{M}, \tag{4.8}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \tag{4.9}$$

$$\underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i,\ i = 1, \ldots, n, \tag{4.10}$$

where (4.8) is the budget constraint. Note that if $B = \text{diag}(\beta_i)$ and $D = \text{diag}(\delta_i)$ are decision variables, the spectral constraint $(D - \varepsilon I)B^{-1} - A \succeq 0$ derived in Lemma 3.5 of Chapter 3 is not an LMI in the decision variables, given the product term DB^{-1}. In addition, since A is assumed to be asymmetric in Problems 4.1 and 4.2, the SDP formulation employed in the previous chapter is no longer possible, since SDPs are defined for symmetric matrices and LMI constraints. We will formulate Problems 4.1 and 4.2 as GPs, from which optimal solutions will be derived.

4.3 Geometric Programs

Geometric Programs are a class of nonlinear, nonconvex optimization problems that minimize posynomial functions, subject to posynomial inequality and monomial equality constraints. Though GPs are nonconvex in their natural form, they can be transformed to convex optimization problems, which can be efficiently solved for globally optimal solutions [62]. GP solvers are numerically efficient, with interior point methods having polynomial time complexity [63]. The convexity and duality properties of GPs are well studied and understood [44] [64]. GPs have been applied

to different problems in the past some of which include power optimization problems in communication [65] and electrical circuit design problems [66].

GPs in standard form are constrained optimization of a class of functions called posynomials; while GPs in convex form are obtained from a logarithmic change of variables, and logarithmic transformation of the objective and constraint functions.

Definition 4.3. Monomial Functions: We define a monomial (of the variables $x_1, \ldots, x_n$) as a function $f : \mathbb{R}^n_{++} \to \mathbb{R}$:

$$f(\mathbf{x}) = c\ x_1^{a_1}\ x_2^{a_2} \ldots\ x_n^{a_n},$$

where $c > 0$ is the coefficient of the monomial and $a_i \in \mathbb{R}$ are exponents of the monomial.

Definition 4.4. Posynomial Functions: A sum of monomials; that is, a function of the form

$$f(\mathbf{x}) \ = \ \sum_{k=1}^{K} c_k\ x_1^{a_{1k}}\ x_2^{a_{2k}} \ldots x_n^{a_{nk}},$$

where $c_k > 0$, for $k = 1, 2, \ldots, K$ and $a_{jk} \in \mathbb{R}$ for $j = 1, \ldots, n$ and $k = 1, \ldots, K$ is called a posynomial.

For example, while $x_1 + x_2^4$ and $x_1 x_2^{0.3} \pi$ are posynomials, $x_1 - x_2^2$ is not a posynomial because of the negative coefficient of the second term.[2] A standard form GP is one in which a posynomial function is minimized subject to posynomial upper bound

[2]More general functions with negative coefficient are called *signomials*.

inequality constraints and monomial equality constraints, represented as

$$\begin{aligned} \text{minimize} \quad & f_0(\mathbf{x}) \\ \text{subject to} \quad & f_i(\mathbf{x}) \leq 1, \quad i = 1, \ldots, m, \\ & g_i(\mathbf{x}) = 1, \quad i = 1, \ldots, p. \end{aligned} \tag{4.11}$$

In (4.11), f_i are posynomial functions, g_i are monomials and x_i are the optimization variables, with an implicit domain constraint that $x_i > 0$. GPs in standard form are not convex optimization problems, since posynomials are not convex functions. However, with a logarithmic change of variables and multiplicative constants: $y_i = \log x_i, \quad b_l = \log c_l, \quad b_{ik} = \log c_{ik}$ and a logarithmic change of the functions' values, (4.11) can be transformed to the following equivalent problem in the variable $\mathbf{y}$:

$$\begin{aligned} \text{minimize} \quad & h_0(\mathbf{y}) = \log \sum_{k=1}^{K_0} \exp(\mathbf{a}_{0k}^T \mathbf{y} + b_{0k}) \\ \text{subject to} \quad & h_i(\mathbf{y}) = \log \sum_{k=1}^{K_i} \exp(\mathbf{a}_{ik}^T \mathbf{y} + b_{ik}) \leq 0, \quad \forall\ i \\ & q_i(\mathbf{y}) = \mathbf{a}_l^T \mathbf{y} + b_l = 0, \quad l = 1, \ldots, M. \end{aligned} \tag{4.12}$$

Problem (4.12) is called a GP in convex form and is a convex optimization problem since the Log-sum-exp (LSE) function is convex [62]. The convex characterizations presented here will be important in deriving solutions for the decentralized resource allocation problem considered in Chapter 5.

4.4 Geometric Programming formulation for Resource Allocation

In this section, we formulate the epidemic control problem as a GP in standard form. We start by expressing the spectral radius constraint (4.3) in an equivalent form using the Perron-Frobenius lemma, from the theory of nonnegative matrices. This equivalent form will enable the expression of the spectral constraint as a set of posynomial functions in the decision variables.

Lemma 4.5. *(Perron-Frobenius) Let M be a nonnegative, irreducible matrix. Then, the following statements about its spectral radius, $\rho(M)$, hold:*

1. *$\rho(M) > 0$ is a simple eigenvalue of M,*
2. *$M\mathbf{u} = \rho(M)\,\mathbf{u}$, for some $\mathbf{u} \in \mathbb{R}^n_{++}$, and*
3. *$\rho(M) = \inf\left\{\lambda \in \mathbb{R} : M\mathbf{u} \leq \lambda\mathbf{u} \text{ for } \mathbf{u} \in \mathbb{R}^n_{++}\right\}$.*

Recall that a working assumption made so far is that the directed network on which the contagion is occurring is strongly connected. Further, since irreducibility of a matrix is equivalent to its associated digraph being strongly connected, lemma 4.5 holds for the spectral radius of the adjacency matrix of any positively weighted, strongly connected digraph. A corollary of Lemma 4.5 is the following:

Corollary 4.6. *Let M be a nonnegative, irreducible matrix. Then, its eigenvalue with the largest real part, $\lambda_1(M)$, is real, simple, and equal to the spectral radius $\rho(M) > 0$.*

This next result helps us understand the effect of the variables $\{\beta_i\}_{i=1}^n$ and $\{\delta_i\}_{i=1}^n$ on the real part of the lead eigenvalue of $\mathrm{diag}(\beta_i)A + \mathrm{diag}(\delta_i)I$.

Lemma 4.7. *([1]) Consider the adjacency matrix A of a (positively) weighted, directed, strongly connected graph $\mathcal{G}$, and two sets of positive numbers $\{\beta_i\}_{i=1}^n$ and $\{\delta_i\}_{i=1}^n$. Further, let $B = diag(\beta_i)$ and $D = diag(\delta_i)$. Then, $\mathbb{R}(\lambda_1(BA - D))$ is an increasing function w.r.t. β_i, and a monotonically decreasing function w.r.t. δ_i for $i = 1, \ldots, n$.*

Proof. See Appendix C. □

From Lemma 4.7, it is clear that the infection rates $\{\beta_i\}_{i=1}^n$ and recovery rates $\{\delta_i\}_{i=1}^n$ both have an effect on the leading eigenvalue of $BA - D$, and will need to be jointly controlled to satisfy the epidemic control condition.

Proposition 4.8. *([1]) Consider the $n \times n$ nonnegative, irreducible matrix $M(\mathbf{x})$ with entries being either 0 or posynomials with domain $\mathbf{x} \in \mathcal{S} \subseteq \mathbb{R}_{++}^k$, where $\mathcal{S}$ is defined as $\mathcal{S} = \bigcap_{i=1}^m \left\{\mathbf{x} \in \mathbb{R}_{++}^k : f_i(\mathbf{x}) \leq 1\right\}$, f_i being posynomials. Then, we can minimize $\lambda_1(M(\mathbf{x}))$ for $\mathbf{x} \in \mathcal{S}$ by solving the following GP:*

$$\underset{\lambda, \{u_i\}_{i=1}^n, \mathbf{x}}{minimize}\ \lambda \tag{4.13}$$

$$subject\ to\ \frac{\sum_{j=1}^n M_{ij}(\mathbf{x})\, u_j}{\lambda u_i} \leq 1,\ i = 1, \ldots, n, \tag{4.14}$$

$$f_i(\mathbf{x}) \leq 1,\ i = 1, \ldots, m. \tag{4.15}$$

The problem above is a valid GP in standard form, derived from Corollary 4.6, since it comprises a monomial objective function and posynomial constraint functions. The constraint in (4.14) comes from expanding the constraint $M\mathbf{u} \leq \lambda\mathbf{u}$ in the third equivalent definition of Lemma 4.5 and expressing it as a set of posynomial functions in the variables $\mathbf{x}, u_i$ and λ. Further, note that 4.14 is a posynomial since it is the ratio of posynomial to a monomial function resulting in a posynomial

inequality. Also, note that if $\mathbf{u} = (u_1, \ldots, u_n)^T$ is a solution of 4.14, so is $\alpha\mathbf{u}$ for any $\alpha > 0$. Based on Proposition 4.8, solutions to the rate and budget-constrained resource allocation problems are proposed in the next subsection.

4.4.1 Solution to the Budget-Constrained Allocation Problem for Strongly Connected Digraphs

Before stating the solution within the GP framework, note that the constraint (4.7) cannot be directly expressed as a set of posynomial functions as was done in Proposition 4.8 because of the negative coefficient of the term $\text{diag}(\delta_i)$. To overcome this challenge for Problem 4.2, an equivalent reformulation is derived.

Suppose the objective functions f_i and g_i are posynomial functions and the contact graph $\mathcal{G}$ is strongly connected, the budget-constrained resource allocation problem in 4.2 can be solved as follows:

Theorem 4.9. *([1]) Given a strongly connected graph $\mathcal{G}$ with adjacency matrix A, posynomial cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$, bounds on the infection and recovery rates $0 < \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i$ and $0 < \underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i$, $i = 1, \ldots, n$, and a maximum investment budget $\mathcal{M}$ on protection and correction resources. Then, the optimal investment on vaccines and antidotes for node v_i to solve Problem 4.2 are $f_i(\beta_i^*)$ and $g_i\left(\overline{\Delta} + 1 - \widehat{\delta}_i^*\right)$, where $\overline{\Delta} \triangleq \max\left\{\overline{\delta}_i\right\}_{i=1}^n$ and $\beta_i^*, \widehat{\delta}_i^*$ are the optimal solution for*

β_i and $\widehat{\delta}_i$ in the following GP:

$$\underset{\lambda,\left\{u_i,\beta_i,\widehat{\delta}_i,t_i\right\}_{i=1}^{n}}{\text{minimize}} \quad \lambda \tag{4.16}$$

$$\text{subject to } \frac{\beta_i \sum_{j=1}^{n} A_{ij}u_j + \widehat{\delta}_i u_i}{\lambda u_i} \leq 1, \tag{4.17}$$

$$\sum_{k=1}^{n} f_k\left(\beta_k\right) + g_k\left(t_k\right) \leq \mathcal{M}, \tag{4.18}$$

$$\left(t_i + \widehat{\delta}_i\right) / \left(\overline{\Delta} + 1\right) \leq 1, \tag{4.19}$$

$$\overline{\Delta} + 1 - \overline{\delta}_i \leq \widehat{\delta}_i \leq \overline{\Delta} + 1 - \underline{\delta}_i, \tag{4.20}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \ i = 1, \ldots, n. \tag{4.21}$$

Proof. The proof is culled from [1]. First, from Proposition 4.8, we have that maximizing ε in (4.6) subject to (4.7)-(4.9) is equivalent to minimizing $\lambda_1\left(BA - D\right)$ subject to (4.8) and (4.9), where $B \triangleq \text{diag}\left(\beta_i\right)$ and $D \triangleq \text{diag}\left(\delta_i\right)$. Let us define $\widehat{D} \triangleq \text{diag}\left(\widehat{\delta}_i\right)$, where $\widehat{\delta}_i \triangleq \overline{\Delta} + 1 - \delta_i$ and $\overline{\Delta} \triangleq \max\left\{\overline{\delta}_i\right\}_{i=1}^{n}$. Then, $\lambda_1\left(BA + \widehat{D}\right) = \lambda_1\left(BA_{\mathcal{G}} - D\right) + \overline{\Delta} + 1$. Hence, minimizing $\lambda_1\left(BA - D\right)$ is equivalent to minimizing $\lambda_1\left(BA + \widehat{D}\right)$. The matrix $BA + \widehat{D}$ is nonnegative and irreducible if A is the adjacency matrix of a strongly connected digraph. Therefore, applying Proposition 4.8, we can minimize $\lambda_1\left(BA + \widehat{D}\right)$ by minimizing the cost function in (4.16) under the constraints (4.17)-(4.21). Constraints (4.20) and (4.21) represent bounds on the achievable infection and curing rates. Notice that we also have a constraint associated with the budget available, i.e., $\sum_{k=1}^{n} f_k\left(\beta_k\right) + g_k\left(\overline{\Delta} + 1 - \widehat{\delta}_i\right) \leq \mathcal{M}$. Even though $g_k(\delta_k)$ is a posynomial function in δ_k, $g_k\left(\overline{\Delta} + 1 - \widehat{\delta}_k\right)$ is a signomial function in $\widehat{\delta}_i$. To express it as a posynomial, we can replace the argument of g_k by a new variable t_k, along with the constraint $t_k \leq \overline{\Delta} + 1 - \widehat{\delta}_k$, which can be expressed as the posynomial inequality, $\left(t_k + \widehat{\delta}_k\right) / \left(\overline{\Delta} + 1\right) \leq 1$. As we proved in Lemma

4.7, the largest eigenvalue $\lambda_1(BA - D)$ is a decreasing value of δ_k and the antidote cost function g_k is monotonically increasing w.r.t. δ_k. Thus, adding the inequality $t_k \leq \overline{\Delta} + 1 - \widehat{\delta}_k$ does not change the result of the optimization problem, since at optimality t_k will saturate to its largest possible value $t_k = \overline{\Delta} + 1 - \widehat{\delta}_k$. □

4.4.2 Solution to Rate-Constrained Allocation Problem for Strongly Connected Digraphs

Similar to the budget-constrained problem in 4.2, the spectral constraint in the rate-constrained Problem 4.1 poses a hurdle when formulating it as a GP in standard form. As was done for the budget-constrained problem, an equivalent reformulation is derived for Problem 4.1 and preseneted in the following result:

Theorem 4.10. *([1]) Given a strongly connected graph $\mathcal{G}$ with adjacency matrix A, posynomial cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$, bounds on the infection and recovery rates $0 < \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i$ and $0 < \underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i$, $i = 1, \ldots, n$, and a desired exponential decay rate $\overline{\varepsilon}$. Then, the optimal investment on vaccines and antidotes for node v_i to solve Problem 4.1 are $f_i(\beta_i^*)$ and $g_i\left(\widetilde{\Delta} + 1 - \widetilde{\delta}_i^*\right)$, where $\widetilde{\Delta} \triangleq \max\{\overline{\varepsilon}, \overline{\delta}_i \text{ for } i = 1, \ldots, n\}$ and $\beta_i^*, \widetilde{\delta}_i^*$ are the optimal solution for β_i and $\widetilde{\delta}_i$ in*

the following GP:

$$\underset{\{u_i,\beta_i,\widetilde{\delta}_i,t_i\}_{i=1}^n}{minimize} \sum_{k=1}^{n} f_k(\beta_k) + g_k(t_k) \tag{4.22}$$

$$subject\ to\ \frac{\beta_i \sum_{j=1}^n A_{ij}u_j + \widetilde{\delta}_i u_i}{\left(\widetilde{\Delta} + 1 - \overline{\varepsilon}\right) u_i} \leq 1, \tag{4.23}$$

$$\left(t_i + \widetilde{\delta}_i\right) \Big/ \left(\widetilde{\Delta} + 1\right) \leq 1, \tag{4.24}$$

$$\widetilde{\Delta} + 1 - \overline{\delta}_i \leq \widehat{\delta}_i \leq \widetilde{\Delta} + 1 - \underline{\delta}_i, \tag{4.25}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i,\ i = 1, \ldots, n. \tag{4.26}$$

Proof. The proof of this theorem is similar to the proof of Theorem 4.9. The primary objective is to derive an equivalent expression enabling us to write the spectral constraint as a set of posynomial functions. The key differences are highlighted in Appendix B □

The rate and budget-constrained resource allocation problems for epidemic control presented as standard form GPs above assumed strong connectivity of the contact graph $\mathcal{G}$; where from Perron-Frobenius result in Lemma 4.5, the entries of the eigenvector $\mathbf{u}$ are stricly positive. In the next section, we present formulations of the resource allocation problems for general – in particular, not necessarily strongly connected directed graphs.

4.5 Epidemic Control for General, not necessarily strongly connected directed graphs

The Perron-Frobenius lemma presented in Lemma 4.5 holds for nonnegative, irreducible matrix M; stating that its spectral radius $\rho(M)$ is simple and strictly positive,

and the associated dominant eigenvector has strictly positive components. However, the Perron-Frobenius as presented in Lemma 4.5 does not hold for directed graphs that are not strongly connected, since their associated adjacency matrix is not irreducible. To apply the lemma to weighted digraphs that are not necessarily strongly connected, it is weakened as follows [67]:

Lemma 4.11. *Let M be a nonnegative matrix. Then, the following statements hold:*

1. $\rho(M) \geq 0$ is an eigenvalue of M (not necessarily simple).
2. $M\mathbf{u} = \rho(M)\,\mathbf{u}$, for some $\mathbf{u} \in \mathbb{R}^n_+$.
3. $\rho(M) = \inf\left\{\lambda \in \mathbb{R} : M\mathbf{u} \leq \lambda\mathbf{u} \text{ for } \mathbf{u} \in \mathbb{R}^n_+\right\}$.

Relative to Lemma 4.5, in the third statement of Lemma 4.11, the components of $\mathbf{u}$ are nonnegative as opposed to being strictly positive. This poses a challenge in the use of Proposition 4.8, since the components of $\mathbf{u}$ must be strictly positive to express the spectral constraint as a set of posynomial functions. In particular, the constraint (4.14) may be undefined for certain u_i that take value zero. The next two results present a way to identify where the zeros of $\mathbf{u}$ are, and how to work around them in the GP formulation.

Let us define the function $\mathcal{Z}(\mathbf{u}) \triangleq \{i : u_i = 0\}$, i.e., a function that returns the set of indices indicating the location of the zero entries of a vector $\mathbf{u} = [u_i]$.

Lemma 4.12. *([1]) Consider a square matrix M. The following transformations preserve the location of zeros in the dominant eigenvector:*

(a) $T_\alpha : M \to M + \alpha I$, for any $\alpha \in \mathbb{R}$, and

(b) $T_R : M \to RM$, for $M \geq 0$ and $R = diag(r_i)$, $r_i > 0$.

Proof. In Appendix D. □

Corollary 4.13. *Consider a nonnegative matrix A and two diagonal matrices $B = diag(b_i)$ and $D = diag(d_i)$ with $b_i, d_i > 0$. Then, the location of the zero entries of the dominant eigenvector of $BA-D$ are the same as those of A, i.e., $\mathcal{Z}(\mathbf{v}_1(BA-D)) = \mathcal{Z}(\mathbf{v}_1(A))$.*

Proof. This directly follows by applying the transformations in Lemma 4.12; see details in Appendix E. □

Corollary 4.13 presents a scheme to determine the location of the zeros of $\mathbf{u}_1(BA-D)$ for any given graph with adjacency matrix $A \geq 0$, without the need to have knowledge of the infection and curing rates of the nodes in matrices B and D. The set $\mathcal{Z}(\mathbf{u}_1(A)) \triangleq \mathcal{Z}_{\mathcal{G}}$ determines the location of the zeros of $\mathbf{u}_1(BA-D)$ based on Lemma 4.12, which coincide with the set of nodes of the graph $\mathcal{G}$ with zero eigenvector centrality [54]. Having determined the location of the zeros, the variables u_i for $i \in \mathcal{Z}_{\mathcal{G}}$ can be excluded from the GPs in Theorems 4.9 and 4.10. Given the exclusion of the variables in the set $\{u_i : i \in \mathcal{Z}_{\mathcal{G}}\}$ from the spectral constraints (4.17) and (4.23), in the GP, the resource allocation problem can be divided up into two sets of decision variables – $\{u_i : i \in \mathcal{Z}_{\mathcal{G}}\}$ and $\{u_i : i \notin \mathcal{Z}_{\mathcal{G}}\}$, with the first group having a trivial allocation policy as illustrated in the next section.

4.5.1 Rate-Constrained Allocation Problem for General Digraphs

Suppose the set of decision variables in (4.16) are divided into two sets: $V_z \triangleq \{u_i, \beta_i, \widetilde{\delta}_i\}_{i \in \mathcal{Z}_{\mathcal{G}}}$ and $V_{nz} \triangleq \{u_i, \beta_i, \widetilde{\delta}_i, t_i\}_{i \notin \mathcal{Z}_{\mathcal{G}}}$. For the set V_z of decision variables,

the following optimization problem is formulated:

$$\begin{aligned}
&\underset{\left\{\beta_i,\widehat{\delta}_i\right\}_{i\in\mathcal{Z}_{\mathcal{G}}}}{\text{minimize}} \sum_{k=1}^{n} f_k\left(\beta_k\right)+g_k\left(\widetilde{\Delta}+1-\widetilde{\delta}_i^*\right)\\
&\text{subject to } \widetilde{\Delta}+1-\overline{\delta}_i \leq \widehat{\delta}_i \leq \widetilde{\Delta}+1-\underline{\delta}_i,\\
&\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \text{ for } i\in\mathcal{Z}_{\mathcal{G}},
\end{aligned}$$

where a trivial policy for resource allocation is obvious if the cost functions f_i is decreasing in β_i, and g_i is increasing in δ_i. The optimal infection and recovery rates are $\beta_i^* = \overline{\beta}_i$ and $\delta_i^* = \underline{\delta}_i$ for all $i \in \mathcal{Z}_{\mathcal{G}}$ and correspond to the minimum possible value of investment on those nodes. Essentially, nodes $i \in \mathcal{Z}$ with zero eigenvector centrality [54] incur the least possible cost in preventive and corrective resources.

The decision variables in the set V_{nz}, however, are amenable to a GP formulation in Theorem 4.10, as indicated in the following Theorem.

Theorem 4.14. *([1]) Given a positively weighted digraph with adjacency matrix A, posynomial cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$, bounds on the infection and recovery rates $0 < \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i$ and $0 < \underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i$, $i = 1, \ldots, n$, and a desired exponential decay rate $\overline{\varepsilon}$. Then, the optimal spreading and recovery rate in Problem 4.1 are $\beta_i^* = \overline{\beta}_i$ and $\delta_i^* = \underline{\delta}_i$ for $i \in \mathcal{Z}_{\mathcal{G}}$.* For $i \notin \mathcal{Z}_{\mathcal{G}}$, *the optimal rates can be computed from*

the optimal solution of the following GP:

$$\underset{\left\{u_i,\beta_i,\widetilde{\delta}_i,t_i\right\}_{i\notin\mathcal{Z}_\mathcal{G}}}{minimize} \sum_{k=1}^{n} f_k\left(\beta_k\right)+g_k\left(t_k\right) \tag{4.27}$$

$$subject\ to\ \frac{\beta_i\sum_{j\notin\mathcal{Z}_\mathcal{G}}A_{ij}u_j+\widetilde{\delta}_i u_i}{\left(\widetilde{\Delta}+1-\overline{\varepsilon}\right)u_i}\leq 1, \tag{4.28}$$

$$\left(t_i+\widetilde{\delta}_i\right)\Big/\left(\widetilde{\Delta}+1\right)\leq 1, \tag{4.29}$$

$$\widetilde{\Delta}+1-\overline{\delta}_i\leq\widehat{\delta}_i\leq\widetilde{\Delta}+1-\underline{\delta}_i, \tag{4.30}$$

$$\underline{\beta}_i\leq\beta_i\leq\overline{\beta}_i,\ for\ i\notin\mathcal{Z}_\mathcal{G}. \tag{4.31}$$

The optimal spreading rate β_i^* is directly obtained from the solution, and the recovery rate is $\delta_i^*=\widetilde{\Delta}+1-\widetilde{\delta}_i^*$, where $\widetilde{\Delta}\triangleq\max\left\{\overline{\varepsilon},\overline{\delta}_i \text{ for } i=1,\ldots,n\right\}$.

4.5.2 Budget-Constrained Allocation Problem for General Digraphs

For the budget-constrained problem, the same splitting of the variable demonstrated in section 4.5.1 can also be employed. In particular, for nodes with zero eigenvector centrality; that is, nodes $\{v_i\in\mathcal{V}:i\in\mathcal{Z}_\mathcal{G}\}$, as was the case for the rate-constrained problem, the optimal spreading and recovery rates are $\beta_i^*=\overline{\beta}_i$ and $\delta_i^*=\underline{\delta}_i$, which means the protective and corrective cost incurred per node is $f_i(\overline{\beta}_i)+g_i(\underline{\delta}_i)$. Since the original fixed budget for all nodes in the network is $\mathcal{M}$, nodes $\{v_i\in\mathcal{V}:i\notin\mathcal{Z}_\mathcal{G}\}$ with a non-zero eigenvector centrality measure are collectively allocated up to

$$\overline{\mathcal{M}}=\mathcal{M}-\sum_{i\in\mathcal{Z}_\mathcal{G}}f_i\left(\overline{\beta}_i\right)+g_i\left(\underline{\delta}_i\right) \tag{4.32}$$

in protective and corrective resources. Hence, for general, not necessarily strongly connected digraphs, the budget-constrained resource allocation problem in 4.2 can be formulated as:

Theorem 4.15. *([1]) Given a positively weighted digraph with adjacency matrix A, posynomial cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$, bounds on the infection and recovery rates $0 < \underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i$ and $0 < \underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i$, $i = 1, \ldots, n$, and a maximum budget $\mathcal{M}$ to invest in protection resources. Then, the optimal spreading and recovery rate in Problem 4.2 are $\beta_i^* = \overline{\beta}_i$ and $\delta_i^* = \underline{\delta}_i$ for $i \in \mathcal{Z}_{\mathcal{G}}$. For $i \notin \mathcal{Z}_{\mathcal{G}}$, the optimal rates can be computed from the optimal solution of the following GP:*

$$\underset{\lambda, \left\{u_i, \beta_i, \widehat{\delta}_i, t_i\right\}_{i \notin \mathcal{Z}_{\mathcal{G}}}}{minimize} \quad \lambda \tag{4.33}$$

$$subject\ to\ \frac{\beta_i \sum_{j \notin Z_G} A_{ij} u_j + \widehat{\delta}_i u_i}{\lambda u_i} \leq 1, \tag{4.34}$$

$$\sum_{k \notin \mathcal{Z}_{\mathcal{G}}} f_k(\beta_k) + g_k(t_k) \leq \overline{\mathcal{M}}, \tag{4.35}$$

$$\left(t_i + \widehat{\delta}_i\right) / \left(\overline{\Delta} + 1\right) \leq 1, \tag{4.36}$$

$$\overline{\Delta} + 1 - \overline{\delta}_i \leq \widehat{\delta}_i \leq \overline{\Delta} + 1 - \underline{\delta}_i, \tag{4.37}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i,\ i \notin \mathcal{Z}_{\mathcal{G}}, \tag{4.38}$$

where $\overline{\mathcal{M}}$ is defined in (4.32), the optimal spreading rate β_i^* is directly obtained from the solution, and the recovery rate is $\delta_i^* = \overline{\Delta} + 1 - \widehat{\delta}_i^*$, where $\overline{\Delta} \triangleq \max\left\{\overline{\delta}_i\right\}_{i=1}^n$.

4.6 Numerical Results

Illustrated in this section are optimal resource allocation derived from the GP formulations above for strongly connected networks. We consider an epidemic outbreak

propagating on a subset of a global air transportation network [68]. Based on travel data from the network, we determine the cost-optimal distribution of protective and corrective resources to contain the spread of an epidemic outbreak. We solve this for both the rate-constrained and budget-constrained problems formulated earlier. The simulations presented are limited to airports with inbound passenger traffic greater than 8 million passengers per year (MPPY). From this network data set, there are 74 such airports globally, and there are $2,694$ direct flights linking the airports. Each directed edge in the network is assigned a weight that accounts for the number of passengers that fly that route year round.

The weighted, directed network formed by these airports has a spectral radius $\rho(A) = 10.048$. The epidemic threshold is $\tau_c = \underline{\delta}/\rho(A)$. The choice of lower and upper bounds on the infection and recovery rates across the network are such that when nodes are allocated protective and corrective resources (that is, when $\beta_i = \underline{\beta}$ and $\delta_i = \overline{\delta}$), the DFE of the spreading dynamics is stable. Further, when nodes are not allocated any protective or corrective resources; that is, when $\beta_i = \overline{\beta}$ and $\delta_i = \underline{\delta}$, the DFE is unstable. In particular, we let $\underline{\delta} = 0.5$ and $\overline{\delta} = 0.9$ and $\overline{\beta} = 1.99 \times 10^{-2}$ and $\underline{\beta} = 0.3\overline{\beta}$, both determined from the epidemic threshold , and chosen so that when there are no investments in protection and correction we have that $\mathbb{R}[\lambda_1(\overline{\beta}A + \underline{\delta}I)] = 0.7 > 0$.

The cost functions considered here are such that $f_i(\beta_i)$ is monotonically decreasing in β_i, and $g_i(\delta_i)$ is monotonically non-decreasing in δ_i. In particular, the cost functions are quasiconvex and capture the fact that only marginal benefit is gained by allocating a high amount of resources to any particular node. The following cost

functions are used:

$$f_i(\beta_i) = \frac{\beta_i^{-1} - \bar{\beta}_i^{-1}}{\underline{\beta}_i^{-1} - \bar{\beta}_i^{-1}}, \quad g_i(\delta_i) = \frac{(1-\delta_i)^{-1} - (1-\underline{\delta}_i)^{-1}}{\left(1-\bar{\delta}_i\right)^{-1} - (1-\underline{\delta}_i)^{-1}}. \tag{4.39}$$

The normalization of the cost functions above is to ensure that whenever $\underline{\beta}_i \leq \beta_i \leq$

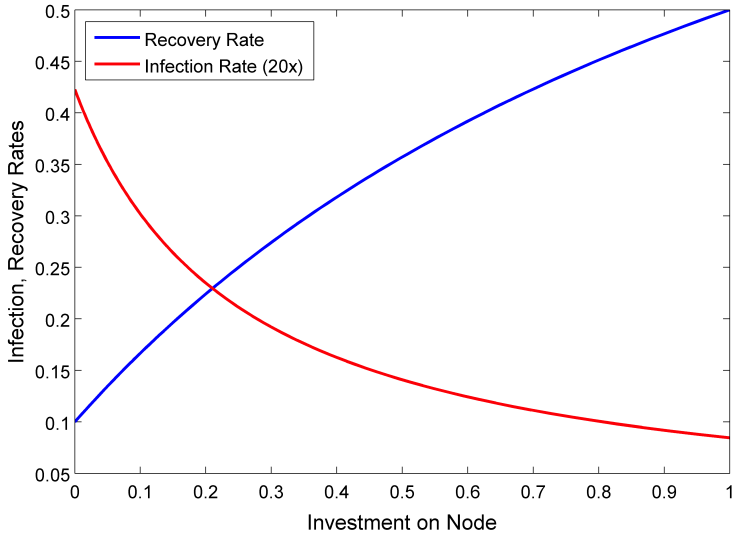

Figure 4.1: Infection rate (in red, and multiplied by 20, to improve visualization) and recovery rate (in blue) achieved at node v_i after an investment on protection (in abscissas) is made on that node [1].

$\bar{\beta}_i$ and $\underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$, the range of the functions lie in the interval $[0, 1]$. As shown in Figure 4.1, horizontal and vertical axes respectively represent the amount invested in protective and corrective resources; and the infection and recovery rates attained by the investment. As the investment on vaccines increase from 0 to 1, the infection rate at the node falls from $\bar{\beta}_i$ to $\underline{\beta}_i$ (red line). Similarly, as the investment in correction resources increases at a node v_i, the recovery rate grows from $\underline{\delta}_i$ to $\bar{\delta}_i$ (blue line). The budget-constrained and rate-constrained resource allocation problems presented in Theorems 4.9 and 4.10 are solved based on the parameters above, using the global air traffic network defined earlier.

4.6.1 Rate-constrained problem

A desired rate of $\bar{\varepsilon} = 10^{-3}$ was specified for the rate-constrained problem. Figure 4.2 captures the interplay between the cost associated with the two decision variables – the infection and recovery rates. The abscissa of figure represents the cost of corrective resources allocated to each airport $g_i(\delta_i^*)$, and the ordinate represents the cost of protective resources allocated to each airport $f_i(\beta_i^*)$. Since there are two decision variables, we can observe that while some airports are not allocated any corrective or protective resources, others are allocated only corrective resources and some others a combination of both corrective and protective resources.

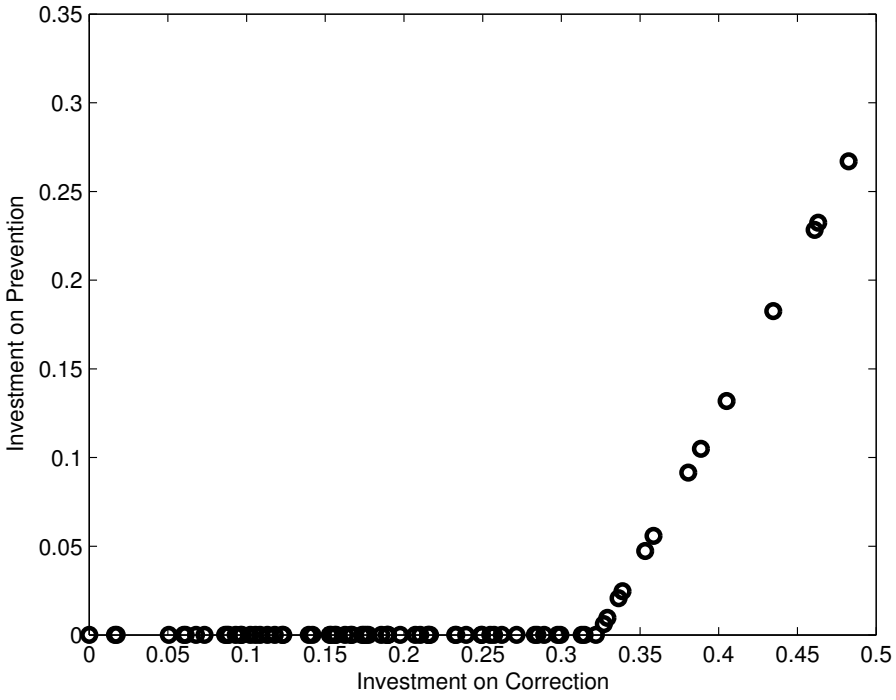

Figure 4.2: A scatter plot of investment in protection against investment in corrective resources per node for a 74-node weighted, directed network.

In comparing the resources allocated to each airport with its centrality measure,

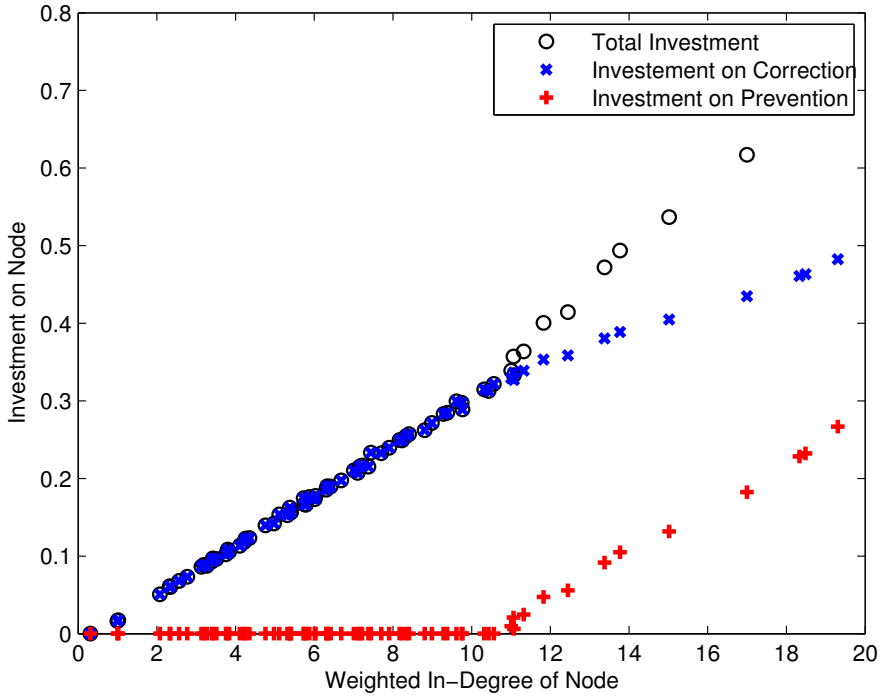

Figure 4.3: A plot of the total investment against the weighted in-degree of each node for a 74-node weighted, directed network.

we find a nontrivial pattern for the different centrality measures considered – *in-degree, eigenvector, and pagerank* centralities. For the in-degree centrality, in Figure 4.3, we observe the existence of a threshold-like behavior. In particular, we observe that almost all airports with less than 1 MPPY are allocated no correction and protection resources, airports with incoming passengers between 1 and 11 MPPY are allocated only correction resources; and airports with incoming traffic exceeding 11 MPPY are allocated both corrective and protective resources.

A similar behavior is observed when the investments per node is compared with the PageRank centrality of each node; see Figure 4.4.

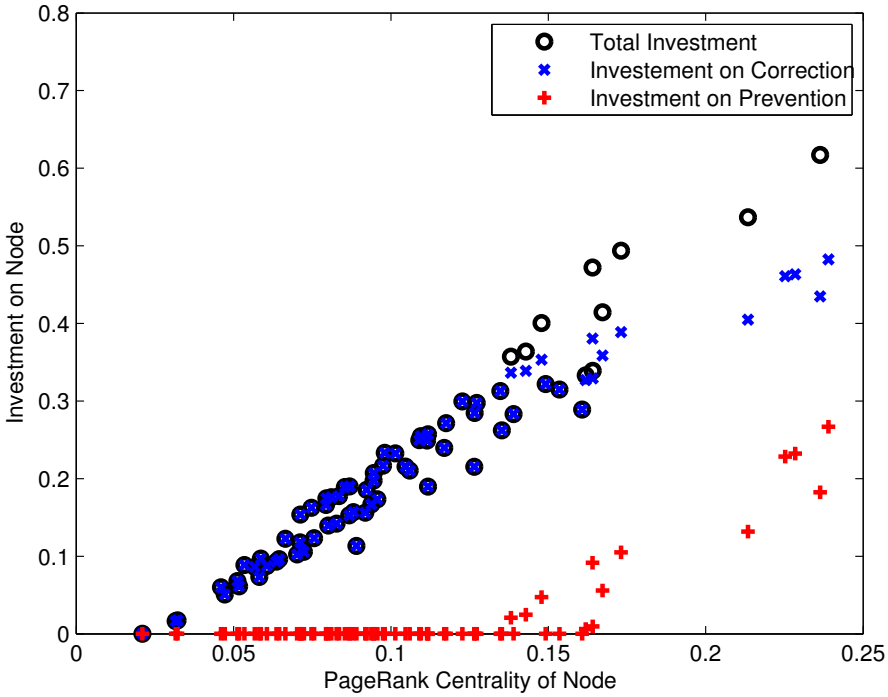

Figure 4.4: A plot of the total investment against the PageRank centralities of each node for a 74-node weighted, directed network.

4.6.2 Budget-constrained problem

Solutions to the budget-constrained problem are summarized in Figures 4.5 and 4.6. The budget here is 45% beyond the optimal investment (or cost) computed in the rate-constrained problem. This slight increase gives the network operator some more resources to achieve a faster decay rate of 0.9469. As expected, achieving a faster decay rate comes at a cost. As can be observed from Figures 4.5 and 4.6, all airports receive the maximum possible investment in correction. When the investment at each node is compared with the incoming passenger traffic, we find that while airports with incoming passenger traffic less than 4 MPPY do not receive any investment in prevention; airports with incoming passenger traffic exceeding 4 MPPY receive investment in prevention.

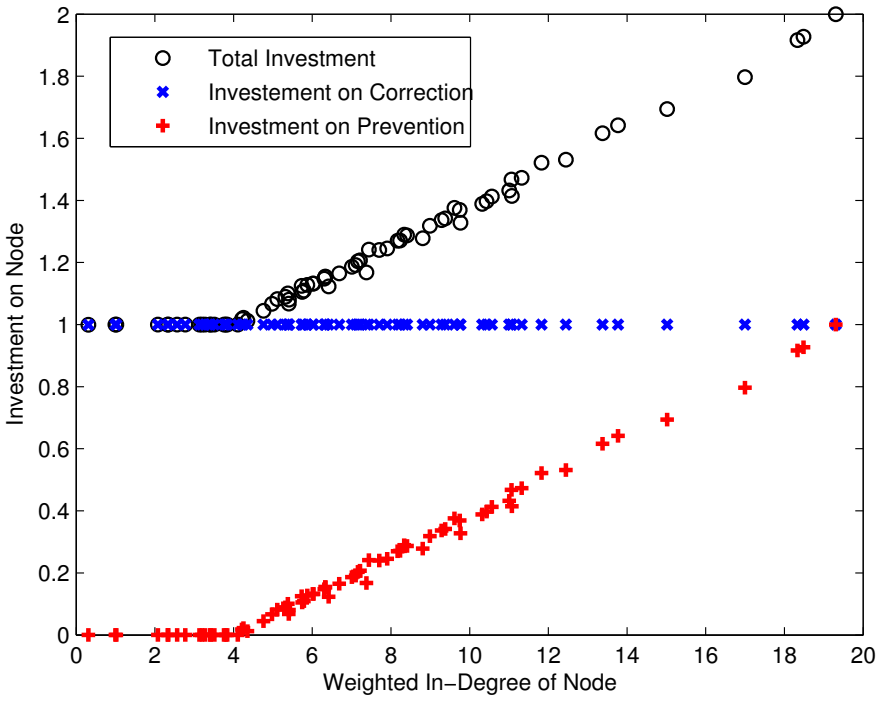

Figure 4.5: A plot of the total investment against the weighted in-degree of each node for a 74-node weighted, directed network.

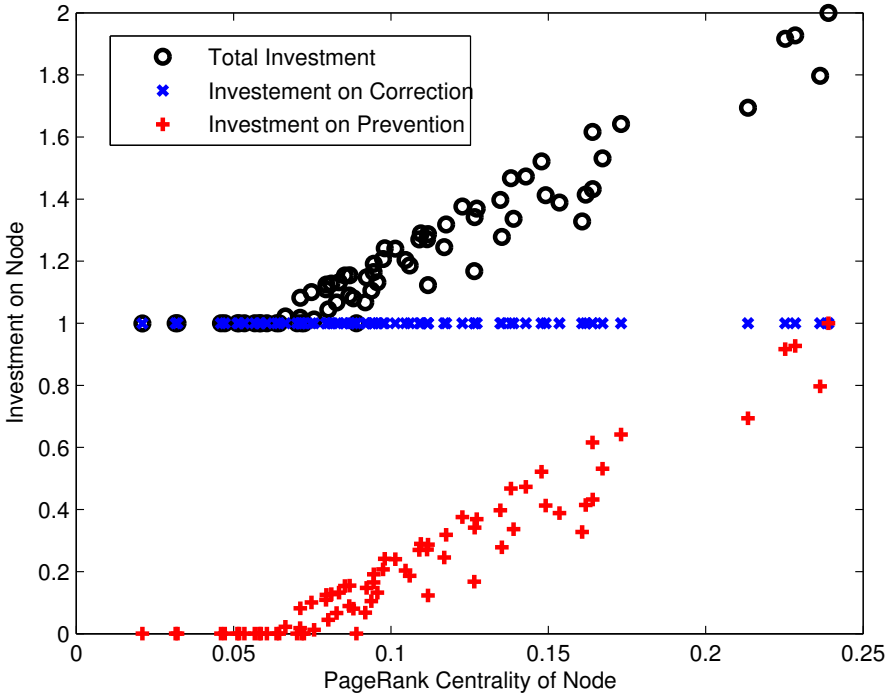

Figure 4.6: A plot of the total investment against the PageRank centralities of each node for a 74-node weighted, directed network.

4.7 Summary

From the numerical results of the preceding section, a concluding fact is that although there is a strong correlation between centrality and investments, there is no trivial protocol to achieve the optimum resource allocation solely based on network centrality measures. With two decision variables – β_i and δ_i per node, where both variables have an effect on epidemic threshold; computing the optimum investment $f_i(\beta_i) + g_i(\delta_i)$ per node via heuristics will be suboptimal since both decision variables need to be simultaneously optimized. Because the solutions presented above are derived from GPs, for which convex representations exist, they are optimal.

Chapter 5

Distributed Resource Allocation via ADMM

The absence of a social planner and computation cost associated with centrally computing and communicating optimal allocation of resources per agent in large scale networks motivate the need for distributed optimization methods for epidemic control. Furthermore, while convex optimization problems can be solved in polynomial time, the size of real problems are too large to be solved in a centralized way, so even if a centralized planner were to solve the large convex optimization, it would need to solve the problem on multiple processors. In this chapter, we present a framework illustrating how the resource allocation problem for epidemic control can be fully distributed and solved amongst agents in a network.

In the distributed resource allocation problem, agents across the network locally optimize their preventive and corrective resource investments based on local interactions, to collectively control the spread of the epidemic outbreak. A decentralized approach to solving the epidemic control problem enables reduction in communication overhead across the network, since each agent's decision is solely based on

information local to it and its neighbors.

We present a fully distributed solution to the cost optimal, *rate-constrained* resource allocation problem for epidemic control first presented in Chapter 4 via a Distributed Alternating Direction Method of Multipliers (D-ADMM) algorithm. The distributed resource allocation problem studied here is analogous to a number of problems in machine learning, statistical inference, social networks as well as problems in wireless sensor networks. Such problems are usually characterized by a system of networked agents, with each agent having a local (and possibly private) cost function, which on aggregate contributes to achieving a system-wide behavior or objective; for instance, see [69] [70] [71], [72] [73] [74]. Problems of this sort stimulated research in the development of distributed methods for solving optimization problems [75] [76] [77] [78] [79] [80]. The conventional approach to distributed optimization is dual decomposition via (sub)gradient methods. This method suffers a number of setbacks. In addition to the fragile adjustment of the step-size, which results in slow convergence particularly for large-scale problems, dual decomposition methods typically require strict technical assumptions on the cost functions and problem structure.

Distributed stability tests for positive systems based on Lyapunov stability theory is studied in [81] [70], which is related to our approach except that we take a fully distributed approach. Other works include [82], [35], [83]. The distributed solution presented in this chapter is similar in spirit to [81] where tests for distributed control of positive systems were presented. A major challenge between the particular problems addressed in [81], [84] and this thesis is that our problem in its natural form is a nonconvex optimization problem. The distributed optimization problem discussed in this chapter will be confined to strongly connected networks, and focus on the rate constrained problem below:

Problem 5.1. *Given a positively weighted directed network with associated adjacency matrix A, node cost functions $\{f_i(\beta_i), g_i(\delta_i)\}_{i=1}^n$ and bounds on the infection and curing rates $0 < \underline{\beta}_i \leq \beta_i \leq \bar{\beta}_i$, and $0 < \underline{\delta}_i \leq \delta_i \leq \bar{\delta}_i$ respectively, and an exponential decay rate $\bar{\varepsilon} > 0$. Locally determine the cost-optimal distribution of vaccines and treatment resources to attain the desired decay rate via local interaction between nodes.*

We state this as the following optimization problem.

$$\underset{\{\beta_i,\delta_i\}_{i=1}^n}{\text{minimize}} \sum_{i=1}^{n} f_i(\beta_i) + g_i(\delta_i) \tag{5.1}$$

$$\text{subject to } \mathbb{R}(\lambda_1(\text{diag}(\beta_i)A - \text{diag}(\delta_i))) \leq -\bar{\varepsilon}, \tag{5.2}$$

$$\underline{\beta}_i \leq \beta_i \leq \overline{\beta}_i, \tag{5.3}$$

$$\underline{\delta}_i \leq \delta_i \leq \overline{\delta}_i, \ i = 1, \ldots, n, \tag{5.4}$$

where (5.1) is the total investment, (5.2) constrains the decay rate to $\bar{\varepsilon}$, and (5.3)-(5.4) maintain the infection and recovery rates in their feasible limits. In Chapter 4, we showed that (5.1) is equivalent to

$$\underset{u_i,\beta_i,\delta_i,}{\text{minimize}} \sum_{i=1}^{n} f_i(\beta_i) + g_i(\delta_i) \tag{5.5}$$

$$\text{subject to } \frac{\beta_i \sum_{j=1}^{n} A_{ij}u_j + \delta_i u_i}{u_i} \leq 1, \tag{5.6}$$

$$\underline{\delta} \leq \delta_i \leq \overline{\delta} \tag{5.7}$$

$$\underline{\beta} \leq \beta_i \leq \overline{\beta}, \quad i = 1, \ldots, n; \tag{5.8}$$

where for simplicity of notation, the auxiliary variables $\widetilde{\Delta}, t_i, \widetilde{\delta}_i$ introduced in Theorem 4.14 to express the spectral constraint as a set of posynomial functions have been factored into the upper and lower bounds on δ_i in (5.7). To implement a distributed solution, first note that the cost function and constraint functions of the GP in (5.5) is separable per agent v_i. Each agent is able to locally solve the following problem:

$$\underset{u_i,\beta_i,\delta_i,}{\text{minimize}} \quad f_i(\beta_i) + g_i(\delta_i) \tag{5.9}$$

$$\text{subject to } \frac{\beta_i \sum_{j=1}^{n} A_{ij} u_j + \delta_i u_i}{u_i} \leq 1, \tag{5.10}$$

$$\underline{\delta} \leq \delta_i \leq \overline{\delta} \tag{5.11}$$

$$\underline{\beta} \leq \beta_i \leq \overline{\beta}. \tag{5.12}$$

Though separable with most of the variables being local, for agent v_i to minimize its cost function it needs the value u_j from nodes in its neighboring nodes. The need for u_j in computing the optimum cost of node v_i comes from the equivalent representation of the spectral constraint described earlier – $(\text{diag}(\beta_i)A + \text{diag}(\delta_i))\mathbf{u} \leq \lambda\mathbf{u}$. In this constraint, each node v_i needs neighboring entries u_j from the global variable $\mathbf{u} \triangleq (u_1, \ldots, u_n)^T$ in (5.9)-(5.12) to compute its optimum cost and satisfy the spectral (global) constraint. To address this problem, we employ the Alternating Direction Method of Multipliers algorithm, which is well suited for such distributed optimization problems.

While (sub)gradient and conjugate ascent or descent algorithms are commonly used for differentiable and unconstrained optimization problems, ADMM can be used for any convex problem. A significant merit of the ADMM algorithm is that it allows for solving large-scale distributed optimization problems, where the composite problems can be easily broken down into subproblems, which can be solved locally.

The ADMM algorithm is also more general than comparable optimization methods since it allows for a loss function that is not necessarily differentiable. The ADMM algorithm works by decomposing the original optimization problem into smaller subproblems that can be sequentially solved in parallel by each agent, allowing for full distributed solutions to large-scale optimization problems.

5.1 Alternating Direction Method of Multipliers

The ADMM falls into the class of dual based methods for solving constrained optimization problems in which an augmented Lagrangian function is minimized with respect to the primal variables, and the dual variables are updated accordingly. Recent surveys on augmented Lagrangian methods, as well as the ADMM algorithm can be found in the monograph by Bertsekas [85] and a survey by Boyd et al. [86], where illustrations and solutions to different optimization problems via the ADMM algorithm are presented. The ADMM algorithm works by decomposing the original optimization problem into subproblems that can be sequentially solved in parallel by each agent, allowing for distributed solutions to large-scale optimization problems. Though results on convergence of the algorithm are still in the early phase of being established, it has been shown to have a convergence rate of $\mathcal{O}(\frac{1}{k})$ [87] [88], [89].

The standard ADMM solves the following problem

$$\begin{aligned} \underset{x,z}{\text{minimize}} \quad & f(x) + g(z) \\ \text{subject to} \quad & Ax + Dz = c, \end{aligned} \tag{5.13}$$

where the variables $x \in \mathbb{R}^n$, $z \in \mathbb{R}^m$, matrices $A \in \mathbb{R}^{p \times n}$, $D \in \mathbb{R}^{p \times m}$, and $c \in \mathbb{R}^p$.

The Augmented Lagrangian function for (5.13) is given by

$$L_\rho(x, z, \lambda) = f(x) + g(z) - \lambda^T(Ax + Dz - c) + \frac{\rho}{2}\|Ax + Dz - c\|^2, \tag{5.14}$$

where λ is the Lagrange multiplier associated with the constraint $Ax + Dz = c$ and ρ is a positive scalar. The update rules for the variables x, z and λ in the ADMM implementation is given by

$$x(k+1) = \arg\min_x L_\rho(x, z(k), \lambda(k)) \tag{5.15}$$

$$z(k+1) = \arg\min_z L_\rho(x(k+1), z, \lambda(k)) \tag{5.16}$$

$$\lambda(k+1) = \lambda(k) - \rho\left((Ax(k+1) + Dz(k+1) - c\right) \tag{5.17}$$

The updates in (5.15)-(5.17) is similar to those of dual descent algorithms (see [79] for instance), except that augmented Lagrangian is used and penalty parameter ρ is used as the step size in the dual updates.

5.2 Distributed Resource Allocation via ADMM

Our goal is to present a distributed solution to the GP in (5.9)-(5.12). To make (5.9)-(5.12) amenable to a distributed solution via the ADMM algorithm, we introduce n-dimensional variables $\mathbf{u}_i \in \mathbb{R}^n$ representing a local copy of the global variable $\mathbf{u} = (u_1, \ldots, u_n)^T$ at each node v_i. In addition, we introduce an auxiliary variable $\mathbf{z}_{ij} \in \mathbb{R}^n$, to enable communication and enforce consensus in the values of $\mathbf{u}_i$ and $\mathbf{u}_j$ for all neighboring nodes $(v_i, v_j) \in \mathcal{E}$. The auxiliary variables z_{ij} can be interpreted as being associated with the edge (v_i, v_j) with the goal of enforcing consensus of the variables $\mathbf{u}_i$ and $\mathbf{u}_j$ of its adjacent nodes v_i and v_j. With these new variables, and

consensus constraint, we can reformulate (5.9)-(5.12) as

$$
\begin{aligned}
&\underset{\mathbf{u}_i,\beta_i,\delta_i,}{\text{minimize}} \quad f_i(\beta_i) + g_i(\delta_i) \\
&\text{subject to } \frac{\beta_i \sum_{j=1}^{n} A_{ij}\mathbf{u}_i^j + \delta_i \mathbf{u}_i^i}{\mathbf{u}_i^i} \leq 1, \\
&\qquad \prod_{j=1}^{n} \mathbf{u}_i^j = 1, \\
&\qquad \underline{\delta} \leq \delta_i \leq \overline{\delta} \\
&\qquad \underline{\beta} \leq \beta_i \leq \overline{\beta}. \\
&\qquad \mathbf{u}_i = \mathbf{z}_{ij} \quad \textit{and} \quad \mathbf{u}_j = \mathbf{z}_{ij}, \quad (v_i, v_j) \in \mathcal{E},
\end{aligned}
\tag{5.18}
$$

where $\mathbf{u}_i^j$ is the jth entry of the local estimate $\mathbf{u}_i$ at node v_i. The constraints $\mathbf{u}_i = \mathbf{z}_{ij}$ and $\mathbf{u}_j = \mathbf{z}_{ij}$ imply that for all pairs of agents (v_i, v_j) that form an edge, the feasible set of (5.18) is such that $\mathbf{u}_i = \mathbf{u}_j$. Assuming a strongly connected contact network, these local consensus constraints imply that feasible solutions must satisfy $\mathbf{u}_i = \mathbf{u}_j$ for all, not necessarily neighboring, pairs of agents v_i and v_j. The formulation presented above differs from existing literature on Distributed ADMM algorithm [87] [90] [91], whose original problem prior to applying D-ADMM have been unconstrained, and only consensus of local estimates is required. The distributed resource allocation problem considered here, evident in (5.18), is a constrained optimization problem (in its original form), in addition to the consensus constraints of local estimates $\mathbf{u}_i$.

Recall that $\mathbf{u}_i$ is an estimate of the eigenvector $\mathbf{u}$ from the inequality $(BA+D)\mathbf{u} \leq \mathbf{u}$ in (5.6), local to node v_i. To compute the augmented Lagrangian of (5.18), we let the dual variables $\alpha_{ij} \in \mathbb{R}^n$ and $\gamma_{ij} \in \mathbb{R}^n$ be associated with equality constraints $\mathbf{u}_i = \mathbf{z}_{ij}$, and $\mathbf{u}_j = \mathbf{z}_{ij}$, $\forall\ (v_i, v_j) \in \mathcal{E}$ in (5.18) respectively. Suppose we define $\Gamma_i(k+1) \triangleq \beta_i(k+1), \delta_i(k+1), \mathbf{u}_i(k+1)$. The augmented Lagrangian at each node

v_i is then:

$$\begin{aligned}\Gamma_i(k+1) = \underset{\mathbf{u}_i,\beta_i,\delta_i,}{\arg\min} \quad & f_i(\beta_i) + g_i(\delta_i) + \sum_{j\in N(i)} \alpha_{ij}^T(\mathbf{u}_i - \mathbf{z}_{ij}(k)) + \gamma_{ij}^T(\mathbf{u}_j(k) - \mathbf{z}_{ij}(k)) \\ & + \frac{\rho}{2}\sum_{j\in N(i)} \|\mathbf{u}_i - \mathbf{z}_{ij}(k)\|_2^2 + \|\mathbf{u}_j(k) - \mathbf{z}_{ij}(k)\|_2^2 \\ \text{subject to } & \frac{\beta_i \sum_{j=1}^n A_{ij}\mathbf{u}_i^j + \delta_i \mathbf{u}_i^i}{\mathbf{u}_i^i} \leq 1, \qquad (5.19) \\ & \prod_{j=1}^n \mathbf{u}_i^j = 1, \\ & \underline{\delta} \leq \delta_i \leq \overline{\delta} \\ & \underline{\beta} \leq \beta_i \leq \overline{\beta}.\end{aligned}$$

And the dual variable updates in the iterations of the D-ADMM algorithm are:

$$\alpha_{ij}(k+1) = \alpha_{ij}(k) + \frac{\rho}{2}(\mathbf{u}_i(k) - \mathbf{u}_j(k)) \;\forall\; j \in N(i) \qquad (5.20)$$

$$\gamma_{ij}(k+1) = \gamma_{ij}(k) + \frac{\rho}{2}(\mathbf{u}_j(k) - \mathbf{u}_i(k)) \;\forall\; j \in N(i). \qquad (5.21)$$

The key primal variables of (5.19) local to node v_i are $\beta_i, \delta_i, \mathbf{u}_i$. Further, suppose we introduce a local dual variable $\phi_i \in \mathbb{R}^n$, such that $\phi_i(k) \triangleq \sum_{j\in N(i)}(\alpha_{ij}(k) + \gamma_{ji}(k)) \;\forall\; v_i \in V$. Given initial variables $\mathbf{u}_i(0) \in \mathbb{R}^n$ and $\phi_i(0) = \mathbf{0}$, the iterative computations and updates of the D-ADMM algorithm are summarized in Algorithm 4: Given the inability to derive closed-form expressions for the primal update, a numerical solver[1] is used for this portion of the algorithm.

[1] We use CVX, a package for specifying and solving convex programs [60] [61].

Algorithm 4 Distributed ADMM for solving (5.18)

1: **Given** initial variables $\beta_i(0), \delta_i(0) \in \mathbb{R}$, $\mathbf{u}_i(0), \phi_i(0) \in \mathbb{R}^n$ for each agent $v_i \in \mathcal{V}$.
2: **Set** $k = 1$
3: **repeat**
4: For all $v_i \in \mathcal{V}$

$$\phi_i(k+1) = \phi_i(k) + \rho \sum_{j \in N(i)} (\mathbf{u}_i(k) - \mathbf{u}_j(k)) \tag{5.22}$$

$$\begin{aligned} \Gamma_i(k+1) = \arg \min_{\beta_i, \delta_i, \mathbf{u}_i} \; & f_i(\beta_i) + g_i(\delta_i) + \phi_i^T(k+1)\mathbf{u}_i \\ & + \rho \sum_{j \in N(i)} \|\mathbf{u}_i - \frac{\mathbf{u}_i(k) + \mathbf{u}_j(k)}{2}\|_2^2 \\ \text{subject to } & \frac{\beta_i \sum_{j=1}^n A_{ij}\mathbf{u}_i^i + \delta_i \mathbf{u}_i^j}{\mathbf{u}_i^i} \leq 1, \\ & \prod_{j=1}^n \mathbf{u}_i^j = 1, \\ & \underline{\delta} \leq \delta_i \leq \overline{\delta} \\ & \underline{\beta} \leq \beta_i \leq \overline{\beta}. \end{aligned}$$

5: **Set** $k = k + 1$
6: **until** $\sum_{i=1}^n \sum_{j \in N(i)} \|\mathbf{u}_i(k) - \mathbf{u}_j(k)\| \leq \eta$, for η arbitrarily small.

5.3 Numerical Simulations

In this section, we illustrate performance of the D-ADMM Algorithm 4 on a strongly connected 8-node directed network, and briefly discuss its convergence. To explicate functional correctness of Algorithm 4, we will show that the investment in vaccines and antidotes for all agents in the network from the distributed solution does, indeed, converge to the solution obtained in the centralized case. We will, in addition, show that the agents do reach consensus in their local estimate of the global variable, Further, we will show convergence of the of the dual variables ϕ_i for all agents v_i.

The D-ADMM algorithm was used to solve (5.9)-(5.12), which is an equivalent formulation of Problem 5.1, on a 8-node network with the following parameters: The epidemic threshold $\tau_c = (1-\overline{\delta})/\rho(A)$ was computed based on the spectral radius and lower bound on the recovery rate. The upper and lower bounds on the infection and recovery rates $\overline{\beta} = 2\tau_c$, $\underline{\beta} = 0.2\overline{\beta}$, $\overline{\delta} = 0.9$, $\underline{\delta} = 0.5$ were chosen in such a way as to ensure the DFE is unstable in the absence of any investment in vaccines and/or antidotes, and stable otherwise. The following quasi-convex functions

$$f_i(\beta_i) = \frac{\beta_i^{-1} - \bar{\beta}_i^{-1}}{\underline{\beta}_i^{-1} - \bar{\beta}_i^{-1}}, \quad g_i(\delta_i) = \frac{(1-\delta_i)^{-1} - (1-\underline{\delta}_i)^{-1}}{\left(1-\overline{\delta}_i\right)^{-1} - (1-\underline{\delta}_i)^{-1}}, \tag{5.23}$$

were used to normalize the investment in vaccines and antidotes. The functions are such that for $\beta_i = \overline{\beta}_i$, $f_i(\overline{\beta}_i) = 0$ and for $\beta_i = \underline{\beta}_i$, $f_i(\underline{\beta}_i) = 1$. Similarly, for $\delta_i = \overline{\delta}_i$, $g_i(\overline{\delta}_i) = 1$ and for $\delta_i = \underline{\delta}_i$, $g_i(\underline{\delta}_i) = 0$. The penalty parameter ρ in the primal and dual updates of Algorithm 4 was chosen to be $\rho = 4$.

For the 8-node example considered, the bounds on the infection and recovery rates were $\underline{\delta} = 0.025$ and $\overline{\delta} = 0.750$ and $\underline{\beta} = 0.1142$ and $\overline{\beta} = 0.4393$. Since a feasibility constraint is $\rho(BA - D) < 1$, the upper and lower bounds of β_i and δ_i are such that[2]

[2]Specifically, the upper and lower bounds of β_i and δ_i were chosen as follows and based on the

when $\beta_i = \underline{\beta}$ and $\delta_i = \underline{\delta}$, across all agents in the network, $\rho(BA - D) = 0.4600$. Further, when $\beta_i = \overline{\beta}$ and $\delta_i = \overline{\delta}$, $\rho(BA - D) = 1.6$. And when $\beta_i = \underline{\beta}$ and $\delta_i = \overline{\delta}$, $\rho(BA - D) = 1.04$. Finally, when $\beta_i = \overline{\beta}$ and $\delta_i = \underline{\delta}$, $\rho(BA - D) = 1.02$. These bounds ensure that all agents in the network are not easily allocated resources to yield the minimum possible infection rate or the maximum possible recovery rates in the network, since the epidemic control criterion will be violated.

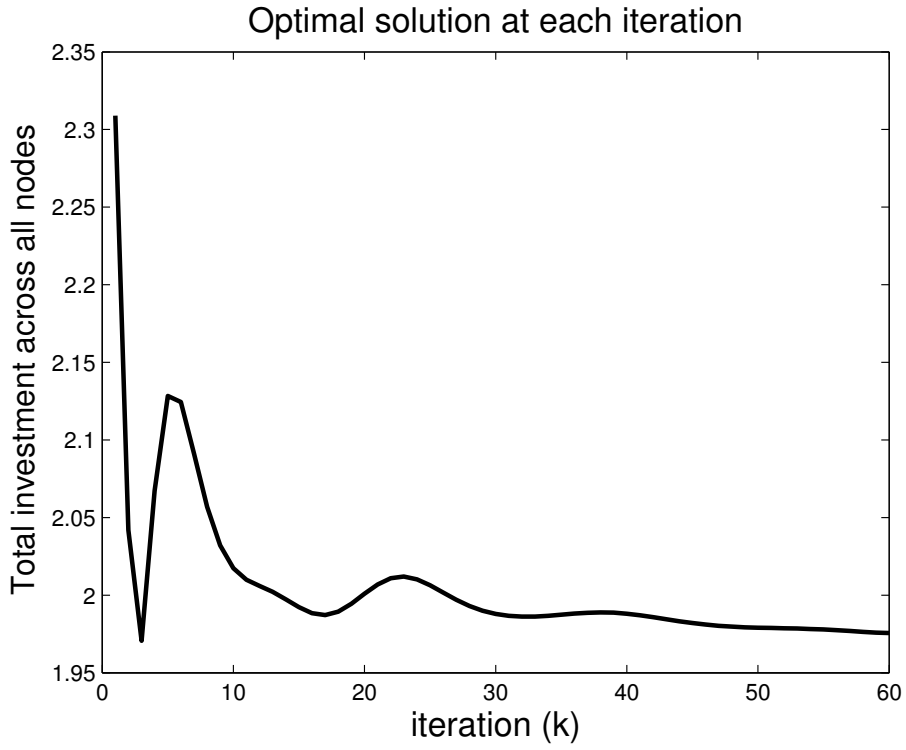

Figure 5.1: Illustrates convergence of the optimal solution. The value approaches 1.9731, which was the solution when solved in a centralized framework for an 8-node network.

Using the cost functions specified in (5.23), the optimal solution (that is, total investment) obtained in the centralized solution was 1.9731. As illustrated in Figure 5.1, where the optimal solution from the distributed problem is presented, we

spectrum of A. We have $\overline{\delta} = 0.8$, $\underline{\delta} = 3.9 \times \frac{2}{10}$, $\tau_c = (2/10)/\lambda_1(A)$, $\overline{\beta} = 4\tau_c$, $\underline{\beta} = 30\%\overline{\beta}$, all chosen in a way that ensures infeasibility of a solution when all agents are assigned the maximum or minimum possible infection or recovery rates.

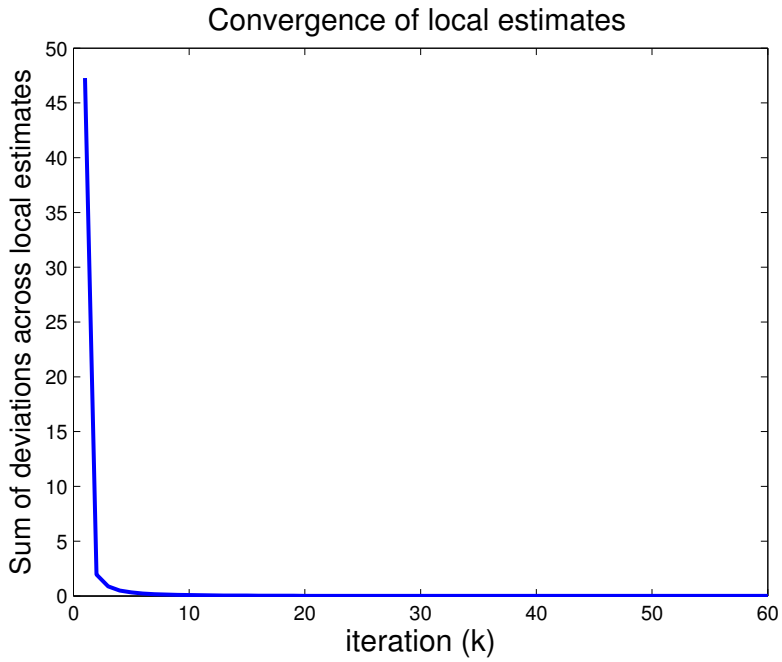

Figure 5.2: The plot above shows convergence to zero, of the errors or deviations in local estimates $\mathbf{u}_i$ at each node. The values on the vertical axis are the aggregate deviations across all agents $\sum_i \sum_{j \in N(i)} \|\mathbf{u}_i - \mathbf{u}_j\|_2$, for an 8-node network.

observe that the optimal solutions of the distributed problem converges to within an ϵ-neighborhood of the centralized problem. The plot in Figure 5.2 shows the convergence of errors in the consensus constraint across all agents. The ordinate in the plot represents the aggregate deviation from consensus across all agents; that is, $\sum_i \sum_{j \in N(i)} \|\mathbf{u}_i - \mathbf{u}_j\|_2$. While the abscissa represents iterations of the algorithm. It shows that the local estimates $\mathbf{u}_i$ of the global variable at node each node v_i converges to those of their neighbors and the agents reach consensus in their estimates. As illustrated in Figure 5.3, the dual variables ϕ_i associated with the D-ADMM consensus constraint for all nodes also converge. We illustrate the solution on a larger strongly connected network comprising 20 nodes. As was done in the case of the 8-node network, the values $\underline{\delta} = 0.025$, $\overline{\delta} = 0.750$, $\underline{\beta} = 0.0641$ and $\overline{\beta} = 0.2464$ were chosen such that when $\beta_i = \underline{\beta}$ and $\delta_i = \underline{\delta}$, across all agents in the network,

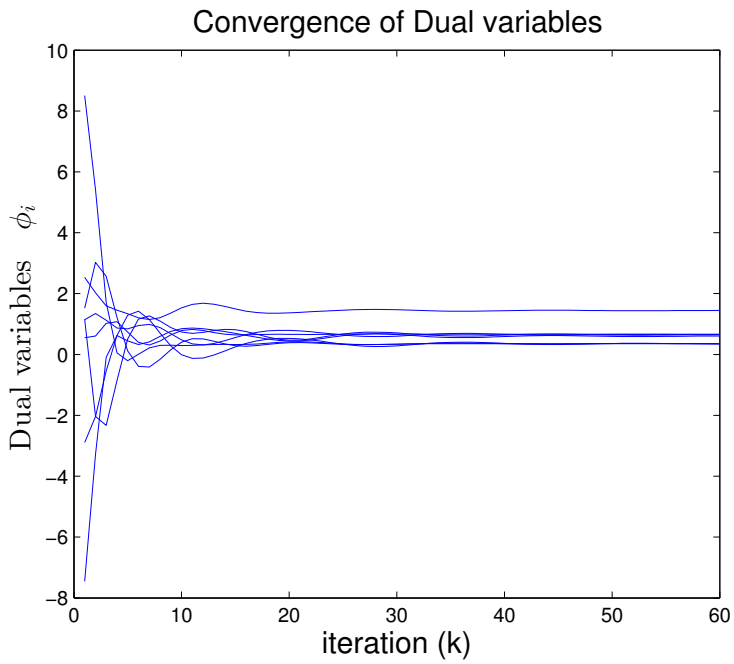

Figure 5.3: This plot shows convergence of dual variable ϕ_i at each iteration of the algorithm for an 8-node network.

$\rho(BA - D) = 0.3500$. Further, when $\beta_i = \overline{\beta}$ and $\delta_i = \overline{\delta}$, $\rho(BA - D) = 2.000$. And when $\beta_i = \underline{\beta}$ and $\delta_i = \overline{\delta}$, $\rho(BA - D) = 1.0750$. Finally, when $\beta_i = \overline{\beta}$ and $\delta_i = \underline{\delta}$, $\rho(BA - D) = 1.2750$.

After solving the program in (5.18) by Algorithm 4, the optimal solution (total investment after normalizing using the quasi-convex functions in (5.23) obtained from the centralized solution for the 20-node network was 4.8868. In Figure 5.4, we find that the total investment obtained from the distributed solution also converges to within an ϵ-neighborhood of the centralized optimal solution. Further, the local estimates $\mathbf{u}_i$ of the nodes reach consensus as illustrated in Figure 5.5. The dual variables associated with the equality constraint in the ADMM formulation also converge as illustrated in Figure 5.6.

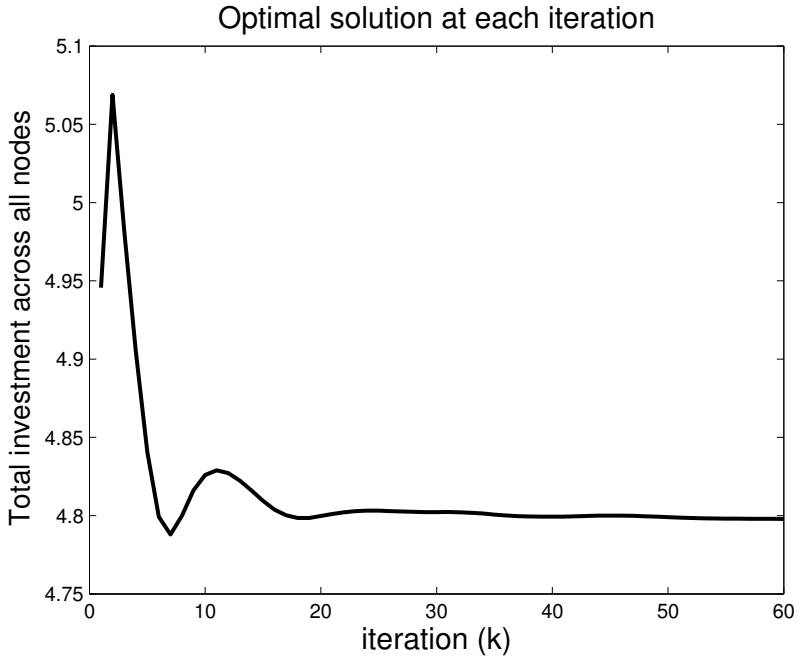

Figure 5.4: Illustrates convergence of the optimal solution. The value approaches 4.8868, which was the solution when solved in a centralized framework for a 20-node network.

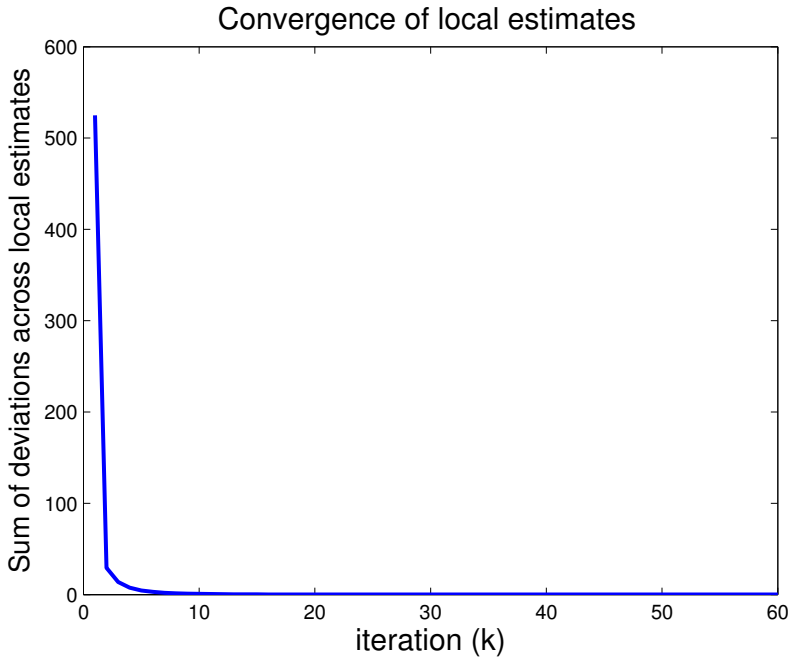

Figure 5.5: The plot above shows convergence to zero, of the errors or deviations in local estimates $\mathbf{u}_i$ at each node. The values on the vertical axis are the aggregate deviations across all agents $\sum_i \sum_{j \in N(i)} \|\mathbf{u}_i - \mathbf{u}_j\|_2$, for a 20-node network.

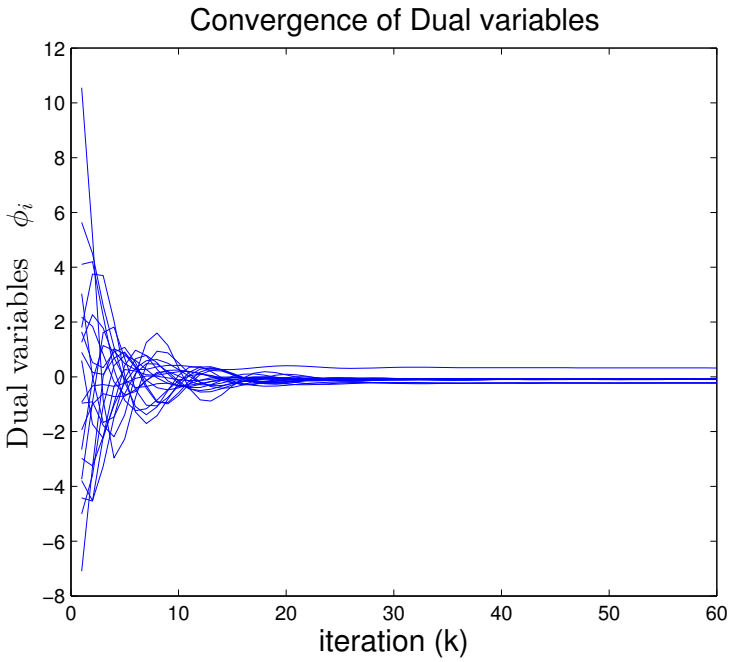

Figure 5.6: This plot shows convergence of dual variable ϕ_i at each iteration of the algorithm for an 20-node network.

5.3.1 On convergence of the D-ADMM Algorithm 4

The ADMM algorithm is known to converge when applied to convex problems [78, 86, 87]. The convex characterization of our resource allocation problem using Geometric Programming presented in Chapter 4 guarantees that the D-ADMM solution in Algorithm 4 converges.

In most existing literature on distributed ADMM, the algorithm is typically applied to unconstrained optimization problems where the only constraint is consensus in the local estimates of the agents; for example [70, 86, 87]. Given such optimization problems comprising a local consensus constraint and smoooth, differentiable convex cost functions, an explicit computation of the local decision variables is possible at each iteration. This then allows for analytical expression of the optimal iterates, which enables convergence rate analysis of the algorithm.

Our original problem, however, is a constrained optimization problem. In addition to the consensus constraint, each agent also has three local constraints to satisfy to attain a feasible solution at each iteration of the algorithm. The use of numerical solvers in computing the local optimal solution at each node (in line 4 of Algorithm 4) poses a challenge on the question of presenting a convergence rate analysis.

5.4 Summary

In this chapter of the thesis, we proposed a fully distributed solution to the problem of optimally allocating vaccine and antidote investment to control an epidemic outbreak in a networked population at a desired rate. Our proposed solution implemented a Distributed-ADMM algorithm, which enables each node to locally compute its optimum investment in vaccine and antidotes needed to globally contain the spread of an outbreak, via local exchange of information with its neighbors. Our problem, unlike most existing literature on Distributed ADMM, is a constrained optimization problem associated with a directed network comprising non-identical agents. Since numerical solvers are used to solve convex subproblems at each node, a convergence rate analysis of the D-ADMM algorithm is not presented. However, illustrations of our fully distributed solution were presented above, and show that the optimal solutions are, indeed, attained; and that the local estimates do converge. Further, it is known that the ADMM algorithm converges when applied to convex problems [86], and since our problem has a convex characterization, we are guaranteed convergence as illustrated numerically in the preceding section. Our proposed fully distributed resource allocation strategy for epidemic control presents a framework to contain an outbreak in the absence of a central social planner.

Chapter 6

Summary and Open Problems

In this thesis, we presented a contagion model and formulated the problem of controlling an outbreak with minimum cost. We formulated the epidemic control objective as a budget-constrained and decay rate-constrained resource allocation problem on a general, positively weighted, directed, strongly connected and not necessarily strongly connected contact network. The problems, formulated as GPs, were efficiently solved, by simultaneously optimizing over the infection and recover rates of each agent, within feasible limits. In addition, we presented a fully distributed solution to the resource allocation problem via a D-ADMM algorithm. An overarching, key contribution of this thesis to the literature on contagion control in networks is the proposed paradigm shift from heuristics to a convex optimization framework on the problem of controlling epidemic outbreaks in networked populations. As we illustrated, the convex framework not only subsumes heuristics, but also takes into account structural and algebraic properties and their effects of the network on the control strategy.

In addition to the proposed paradigm shift from heuristics towards a convex optimization framework, a novelty of this thesis lies in the simultaneous allocation

of vaccines and antidotes to agents in an heterogeneous network comprising weighted and directed edges.

Studying and solving the problems presented above generated other interesting research problems, which we briefly highlight next. Despite the theoretical advances in this problem space, some other related problems remain unsolved. For example, while the solutions presented are exact for the model considered, the Mean-Field approximation (MFA) on the spreading dynamics may need further characterization and remains an open problem. While mean-field techniques have long been applied to epidemic models, especially since they allow for mathematically tractable formulations, more analyses and exposition is needed on, possibly key, lost information resulting from the MFA. Further analyses is needed in understanding how certain classes of cost functions affect the resource allocation strategy; for instance, what would the optimal solution look like if the agents had different cost functions?

In addition, interactions between networked agents are typically not fixed, since agents are mobile and links between agents are established and broken over time. Hence, though challenging, the study of epidemic processes and control strategies on networks with dynamic topologies needs attention. Somewhat related to dynamic networks are multiplex networks – where agents are participants in several layers of networks of interactions simultaneously [92] [93] [94]. Analyzing the interplay of the network layers and its effect on an epidemic process and control strategies spread across multiple network layers is an open problem.

The epidemic problem set up studied in this thesis assumed a fixed, one-time investment in vaccines and antidotes. Formulating the epidemic control objective as an optimal control problem over time, in which the control is a trajectory, as opposed to a fixed input is also a problem that needs attention [95]. Furthermore, the spreading model considered in this thesis lacks spatial dependency. This is crucial

in real networks where administering vaccines and/or antidotes to 'far away' nodes can be costly. How can models that account for temporal and spatial dependencies be developed [96] [97]?

The epidemic control problem considered so far assumes that agents in the network have no specified task and the cost of control does not account for other (possibly conflicting) objectives that the network is performing. In real networks, links in the network serve multiple purposes; for instance, air traffic between airports serving as disease carriers as well as an airlines bottom line. In this scenario, how can an epidemic outbreak be contained or controlled (by reducing activity levels at airports), without excessively hurting passenger flow? This is part of an ongoing work by the author.

Appendices

Appendix A

Delineating the discrete-time SIS epidemic model

Though the The derivation of (2.13) closely follows the development in [39]. Consider a sample path s, and let $p_i^s(t)$ be the probability of infection at node v_i at time-step t. Based on (2.1), the evolution of $p_i^s(t+1)$ is conditioned on the state of of node v_i at time t in s; that is, $X_i^s(t)$. Assuming node v_i is not infected at time t, let the random variable $\mathcal{S}_i^s(t)$ denote the number of infected neighbors of node v_i at time t. If node v_i is infected, $\mathcal{S}_i(t) = 0$.

Hence the probability of infection at time node v_i at time $t+1$, given that it was not infected at time t is $(1-\beta_i)^{\mathcal{S}_i(t)}$. For small β_i, $(1-\beta_i)^{\mathcal{S}_i(t)}$ can be approximated by $1-\beta_i\mathcal{S}_i(t)$. Hence, the evolution of $p_i(t)$ can be described as

$$p_i^s(t+1) = \beta_i\mathcal{S}_i^s(t)(1-p_i^s(t)) + (1-\delta_i)p_i^s(t) \tag{A.1}$$

Taking expectation of (A.1) over all sample paths s, and assuming $p_i(t) = \mathbb{E}_s\left(p_i^s(t)\right)$,

we have that [39]:

$$p_i(t+1) = \beta_i \, \mathbb{E}_s \left[(1 - p_i^s(t)) \cdot \mathcal{S}_i^s(t) \right] + (1 - \delta_i) p_i(t). \tag{A.2}$$

Suppose the neighborhood set of each node to be large, then we can assume that $\mathcal{S}_i^s(t)$ concentrates around its mean by the law of large numbers. This enables us consider the terms $(1 - p_i^s(t))$ and $\mathcal{S}_i^s(t)$ as independent; so that we can approximate $p_i(t+1)$ by

$$p_i(t+1) \approx \beta_i \, (1 - p_i(t)) \cdot \mathbb{E}_s[\mathcal{S}_i^s(t)] + (1 - \delta_i) p_i(t). \tag{A.3}$$

The term $\mathbb{E}_s[\mathcal{S}_i^s(t)]$ is defined as the sum of the probabilities of infection of the neighbors of node v_i, given that node v_i is susceptible at time t in the sample path s. The term $\mathbb{E}_s[\mathcal{S}_i^s(t)]$ can be approximated by leaving off the conditioning on the susceptibility of node v_i at time t. As noted in [39], this approximation is reasonable, if the behavior of the sample path s closely mimics the behavior of the mean of all sample paths. Thence, $\mathbb{E}_s[\mathcal{S}_i^s(t)] \approx \sum_{j \in N_i^i n} p_j(t)$. This yields the nonlinear system [39]:

$$p_i(t+1) = \beta_i (1 - p_i(t)) \sum_{j \in N_i^i n} p_j(t) + (1 - \delta_i) p_i(t). \tag{A.4}$$

Noting that $1 - p_i(t) \leq 1$, we have that

$$p_i(t+1) \leq \beta_i \sum_{j \in N_i^i n} p_j(t) + (1 - \delta_i) p_i(t), \tag{A.5}$$

whose matrix-vector is (2.13), where A in (2.13) is the network adjacency matrix. Readers are referred to [39] for a more detailed exposition of the linear approximation, where simulations illustrating its validity are also presented.

Appendix B

Proof of Theorem 4.10

The proof closely follows the proof for Theorem 4.9 and only the key differences are highlighted here. Define $\widetilde{D} \triangleq \text{diag}\left(\widetilde{\delta}_i\right)$ where $\widetilde{\delta}_i \triangleq \widetilde{\Delta} + 1 - \delta_i$ and $\widetilde{\Delta} \triangleq \max\left\{\overline{\varepsilon}, \overline{\delta}_i \text{ for } i = 1, \ldots, n\right\}$. Since $\lambda_1\left(BA + \widetilde{D}\right) = \lambda_1\left(BA_{\mathcal{G}} - D\right) + \widetilde{\Delta} + 1$, the spectral condition $\lambda_1\left(BA - D\right) \leq -\overline{\varepsilon}$ is equivalent to $\lambda_1\left(BA + \widetilde{D}\right) \leq \widetilde{\Delta} + 1 - \overline{\varepsilon}$. From the definition of $\widetilde{\Delta}$ we have that $\widetilde{\Delta} + 1 - \overline{\varepsilon} > 0$. Also, $BA + \widetilde{D}$ is a nonnegative and irreducible matrix if $\mathcal{G}$ is a strongly connected digraph. From (4.14), we can write the spectral constraint $\lambda_1\left(BA + \widetilde{D}\right) \leq \widetilde{\Delta} + 1 - \overline{\varepsilon}$ as

$$\frac{\beta_i \sum_{j=1}^{n} A_{ij} u_j + \widetilde{\delta}_i u_i}{\left(\widetilde{\Delta} + 1 - \overline{\varepsilon}\right) u_i} \leq 1,$$

for $u_i \in \mathbb{R}_{++}$, $\lambda \in \mathbb{R}$, which results in constraint (**??**). The rest of constraints can be derived following similar derivations as in the Proof of Theorem 4.9.

□

Appendix C

Proof of Lemma 4.7

We define the auxiliary matrix $M \triangleq \text{diag}\,(\beta_i)\,A - \text{diag}\,(\delta_i) + \Delta I$, where $\Delta \triangleq \max\{\delta_i\}$. Thus, $\lambda_1(M) = \lambda_1(\text{diag}\,(\beta_i)\,A - \text{diag}\,(\delta_i)) + \Delta$. Notice that both M and M^T are nonnegative and irreducible if $\mathcal{G}$ is strongly connected. Hence, from Lemma 4.5, there are two positive vectors $\mathbf{v}$ and $\mathbf{w}$ such that

$$M\mathbf{v} = \rho\mathbf{v},$$
$$\mathbf{w}^T M = \rho\mathbf{w}^T,$$

where $\rho = \rho(M) = \lambda_1(M)$, and $\mathbf{v}$, $\mathbf{w}$ are the right and left dominant eigenvectors of M. From eigenvalue perturbation theory, we have that the increment in the spectral radius of M induced by a matrix increment ΔM is [67]

$$\rho(M + \Delta M) - \rho(M) = \mathbf{w}^T \Delta M \mathbf{v} + o(\|\Delta M\|). \tag{C.1}$$

To study the effect of a positive increment in β_k in the spectral radius, we define

$\Delta B = \Delta\beta_k \mathbf{e}_k \mathbf{e}_k^T$, for $\Delta\beta_k > 0$, and apply C.1 with $\Delta M = \Delta B\, A$. Hence,

$$\begin{aligned}\rho\left(M + \Delta M\right) - \rho\left(M\right) &= \Delta\beta_k \mathbf{w}^T \mathbf{e}_k \mathbf{e}_k^T A \mathbf{v} + o\left(\|\Delta\beta_k\|\right) \\ &= \Delta\beta_k w_k \mathbf{a}_k^T \mathbf{v} + o\left(\|\Delta\beta_k\|\right) > 0,\end{aligned}$$

where $\mathbf{a}_k^T = \mathbf{e}_k^T A$ and the last inequality if a consequence of $\Delta\beta_k$, w_k, and $\mathbf{a}_k^T \mathbf{v}$ being all positive. Hence, a positive increment in β_k induces a positive increment in the spectral radius.

Similarly, to study the effect of a positive increment in δ_k in the spectral radius, we define $\Delta D = \Delta\delta_k \mathbf{e}_k \mathbf{e}_k^T$, for $\Delta\delta_k > 0$. Applying C.1 with $\Delta M = -\Delta D$, we obtain

$$\begin{aligned}\rho\left(M + \Delta D\right) - \rho\left(M\right) &= -\Delta\delta_k \mathbf{w}^T \mathbf{e}_k \mathbf{e}_k^T \mathbf{v} + o\left(\|\Delta\delta_k\|\right) \\ &= -\Delta\delta_k w_k v_k + o\left(\|\Delta\delta_k\|\right) < 0.\end{aligned}$$

□

Appendix D

Proof of Lemma 4.12

The proof of (a) is trivial and valid for any square matrix M. To prove (b), we consider the eigenvalue equations for M and RM, i.e., $M\mathbf{u} = \lambda_1(M)\,\mathbf{u}$ and $RM\mathbf{w} = \lambda\mathbf{w}$, where $\mathbf{u} = \mathbf{v}_1(M) = [u_i]$ and $\mathbf{w} = \mathbf{v}_1(RM) = [w_i]$. We expand the eigenvalue equations component-wise as,

$$\sum_{j=1}^{n} m_{ij}u_j = \lambda u_i, \tag{D.1}$$

$$\sum_{j=1}^{n} r_i m_{ij}w_j = \lambda w_i, \tag{D.2}$$

for all $i = 1, \ldots, n$. We now prove statement (b) by proving that $v_i = 0$ if and only if $w_i = 0$.

If $u_i = 0$, then (D.1) gives $\sum_j m_{ij}v_j = 0$. Since $m_{ij}, v_i \geq 0$, the summation $\sum_j m_{ij}v_j = 0$ if and only if the following two statements hold: $(a1)$ $m_{ij} > 0 \implies v_j = 0$ and $(a2)$ $v_j > 0 \implies m_{ij} = 0$. Since $t_i > 0$, these two statements are equivalent to: $(b1)$ $t_i m_{ij} > 0 \implies v_j = 0$ and $(b2)$ $v_j > 0 \implies t_i m_{ij} = 0$. Statements $(b1)$ and $(b2)$ are true if and only if $\sum_j (t_i m_{ij})\, w_j = 0 = w_i$, where the

last equality comes from (D.2). Hence, we have that $v_i = 0 \iff w_i = 0$; hence, $\mathcal{Z}(\mathbf{u}) = \mathcal{Z}(\mathbf{w})$.

□

Appendix E

Proof of Corollary 4.13

Our proof is based on the transformations defined in Lemma 4.12. Starting from a matrix $BA - D$, we then apply the following chain of transformations:

(*i*) $T_\alpha (BA - D) = BA + \Delta$, for $\alpha = \max\{d_i\}$. Hence, $\Delta = \max\{d_i\} I - D$ and $BA + \Delta \geq 0$.

(*ii*) $T_R (BA + \Delta) = \Delta^{-1} BA + I$, for $R = \Delta^{-1}$.

(*iii*) $T_\alpha (\Delta^{-1} BA + I) = \Delta^{-1} BA$, for $\alpha = -1$.

(*iv*) $T_R (\Delta^{-1} BA) = A$, for $R = B^{-1}\Delta$.

From Lemma 4.12, these transformations preserve the of the zeros in the dominant eigenvector. Thus, the input to the first transformation, $BA - D$, and the output to the last transformation, A, satisfy $\mathcal{Z}(\mathbf{v}_1 (BA - D)) = \mathcal{Z}(\mathbf{v}_1 (A))$.

□

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