Control of Generalized Discrete-time SIS Epidemics via Submodular Function Minimization

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Abstract—In this paper, we study a novel control method for a generalized SIS epidemic process. In particular, we use predictive control to design optimal protective resource distribution strategies which balance the need to eliminate the epidemic quickly against the need to limit the rate at which protective resources are used. We expect that such a controller may be useful in mitigating the spread of biological diseases which do not confer immunity to those who have been infected previously, with sexually transmitted infections being a prominent example of such. Technically, this paper provides a novel contribution in demonstrating that the particular combinatorial optimal control problem used to design resource allocations has an objective function which is submodular, and so can be solved in polynomial time despite its combinatorial nature. We test the performance of the proposed controller with numerical simulations, and provide some comments on directions for future work.

I. INTRODUCTION

Due to increased population mobility and a rapidly changing climate, the likelihood of a major biological epidemic occurring in modern times is significant [1]. It is therefore prudent to invest effort into understanding the control of epidemics. Epidemic control is not a new field of research. Indeed, the modern study of epidemic processes dates back to at least the 1920s [2], with work focusing specifically on the optimal control of spreading processes dating back to at least the 1970s [3]–[6]. Notably, nearly all such work focuses on infection statistics, and to use these approximations to equations which gives provable upper- and lower- bounds it is possible to construct a system of ordinary differential equations which protective resources used. We expect that such a controller may be useful in mitigating the spread of biological diseases which do not confer immunity to those who have been infected previously, with sexually transmitted infections being a prominent example of such. Technically, this paper provides a novel contribution in demonstrating that the particular combinatorial optimal control problem used to design resource allocations has an objective function which is submodular, and so can be solved in polynomial time despite its combinatorial nature. We test the performance of the proposed controller with numerical simulations, and provide some comments on directions for future work.

It was shown in [9] that varying the healing rate of an SIS process sufficiently aggressively enables a controller to drive the contagion out of the network quickly. It was shown in [10] that for any priority order strategy, there exists a sufficiently large budget so as to guarantee that the epidemic is driven out of the network quickly. It was shown in [11] that it is possible to construct a system of ordinary differential equations which gives provable upper- and lower- bounds on infection statistics, and to use these approximations to enforce a stability constraint that guarantees fast extinction of the epidemic. However, in all of these works, the optimization problems which fundamentally characterize the controllers are NP-hard. As such, only suboptimal solutions may be obtained, where the performance attained may vary.

It is interesting to consider if there is any way to design predictive controllers for stochastic spreading process models in such a way that the underlying optimization is tractable. Indeed, it has been known for some time that optimizing seed selection under general threshold spreading models is a submodular maximization problem [12], [13], and so can be well approximated by a greedy algorithm. Similar sorts of structural properties have been identified in many variations of problems on threshold-type spreading models, (see, e.g., [14]–[16]), but such models are only appropriate for modeling non-recurrent epidemics. That is, they study processes in which each agent in the network can only actively spread the phenomenon for one interval of time, and afterwards remains inactive. While such a feature seems appropriate in many contexts, e.g. when a disease confers immunity to survivors, this is not always the case. Indeed, many sexually transmitted infections (e.g. chlamydia, gonorrhea) do not confer such immunity (see, e.g., [17]).

The study of discrete-time models with recurrent compartmental memberships seems to have originated in [18], where the stability of a deterministic approximation to a discrete-time Susceptible-Infected-Susceptible (SIS) model was studied. Since then, deterministic approximations to discrete-time models have been studied in a few contexts [19]–[22]. While it is known that in some cases the open-loop stability of the deterministic approximation implies a similar notion of open-loop stability of the stochastic model [19]–[21], it appears that [23] was the first to consider closed-loop control of such processes. Whereas [23] studied the control of the discrete-time SIS process by direct control of the process parameters, here we study the control of a generalized SIS model by way of direct allocation of protective resources.

Statement of Contributions: To the best of the authors’ knowledge, this paper is the first to develop a feedback controller for discrete-time, recurrent epidemic processes using node removal as a means of actuation. With respect to prior work, the present article builds on a preliminary paper [23], which studied the control of a discrete-time SIS process by direct control of the process parameters. Principally, our work here differs in that we focus on control via node removal. This change in focus allows us to
model control actions more realistically: protective devices (e.g., latex gloves, barriers, condoms) are discrete objects by nature, and so should be treated as such in the model.

The primary technical contribution of this work is in demonstrating that a particular combinatorial optimal control problem can be posed as a submodular minimization problem with respect to a ground set that is a subset of the set of nodes, and so can be solved efficiently (i.e., in polynomial time with respect to the size of the graph). Additionally, we investigate the performance of the controller by way of numerical simulations, and provide some comments on directions for future work which we believe to be promising.

II. PROBLEM STATEMENT

In this section, we formally develop the problem studied in the paper. Section II-A details the generalized SIS model studied. Section II-B details the actuation model studied. Section II-C details the optimal control problem studied.

A. Epidemic Model

We study an epidemic process on an $n$ node graph $G$, with node set $V$, and edge set $E$. Epidemic processes are mathematical objects which evolve on graphs, in which each node is considered to be an agent, and each edge a relationship between agents. The current health status of an agent is modeled by a collection of compartments, where at each time, every agent belongs to exactly one compartment. The process we study has one susceptible (i.e. healthy) compartment (denoted by $S$), and $p$ infected spreading compartments (denoted by $\{I_k\}_{k \in [p]}$). A transition from susceptibility to infection comes from contact between a susceptible node and an infected node. Other transitions are due to random events internal to the agent (e.g. progressing through the stages of the disease). Note that the multiplicity of infected compartments here is used to shape the amount of time an agent spends in infection.

We denote by $X^S_i(t)$ an indicator random variable which takes the value 1 if node $i$ is in compartment $S$ at time $t$, and takes the value 0 otherwise. We take compartmental memberships to evolve in a way that generalizes the standard discrete-time SIS process [18]. A node $i$ transitions from susceptible to the first infected compartment (i.e. $I_1$) on the increment from time $t$ to time $t+1$ through contact with nodes which are in an infected compartment at time $t$. Given that a node $i$ is infected at time $t$, it transitions to other model compartments independently of all external phenomena. We then have that the indicator $X^S_i$ evolves as

$$X^S_i(t+1) = X^S_i(t) - Z^S_{i,X} + \sum_{k=1}^{p} X^I_k Z^I_k - \sum_{k=1}^{p} Z^S_{i,k},$$

where the random variable $Z^S_{i,k}$ is an indicator that at least one infection event (indicated by random variables $Y_{ij}$) has occurred on an edge between node $i$ and an infected node $j$ in the set of neighbors of $i$ (denoted $N_i$), and is defined as

$$Z^S_{i,k} \triangleq \min \{1, \sum_{j \in N_i} Y_{ij} \sum_{k=1}^{p} X^I_k\}.$$  

and the random variables $Z^I_k$ are indicators denoting a transition from the $k$'th infected compartment to susceptibility. Likewise, we have that the indicator $X^I_i$ evolves as

$$X^I_i(t+1) = X^I_i(t) + \sum_{k=1}^{p} X^I_k Z^I_k - \sum_{k=1}^{p} X^I_i Z^I_k,$$

and the indicators $X^I_k$ for $k \neq 1$ evolve as

$$X^I_k(t+1) = \sum_{\ell \in L \setminus \{S\}} X^I_k Z^I_{k,\ell} - \sum_{\ell \in L \setminus \{S\}} Z^I_{k,\ell},$$

where $L$ is the set of all compartmental labels. Note that for all nodes to belong to a unique compartment at all times, we must have that $Z^I_{k,\ell}$ each take values on $\{0, 1\}$ and satisfy $\sum_{\ell} Z^I_{k,\ell} = 1$ with probability one.

To make matters concrete, we fix one particular way of enforcing this. We assume all $Y_{ij}$ in (2) are independently distributed Bernoulli random variables with known success probabilities, and that when a node is infected, the compartmental membership random variables evolve independently as a discrete-time Markov chain with $p+1$ states, structured so that $S$ is its unique absorbing state, and that $S$ is reached in finite time with probability one. This level of generality allows us to model the amount of time taken to recover from an infection with more precision than a standard SIS epidemic, in which there is only one infected compartment, and $Z^I_{k}$ is an independent Bernoulli random variable at all times. Such an assumption forces the time taken to recover to be distributed as a geometric random variable. Under the model specified here, the time taken to recover follows a discrete phase-type distribution, and so is quite general [24].

B. Actuation Model

We consider allocating protective barriers in order to preventing the spread of an infection. Formally, our controller actuates the process detailed in Section II-A by selecting a subset of nodes $P$ to protect against infection. Because protective devices (e.g. latex gowns, gloves, condoms) are often single-use, it is appropriate to model the economic cost of protecting the set of nodes $P$ as being the sum over all edges in which one adjacent node is a susceptible node that is protected, and the other adjacent node is infected.

That is, we only pay to protect a particular node if it interacts with an infected person, and we pay in proportion to the extent of interaction between the protected node and its infected neighbors over the discretized time period. For example, if we are allocating condoms to mitigate the spread of chlamydia and we want to protect a particular person $i$, we will provide $i$ with one condom for each contact between it and all infected partners. Mathematically, we have

$$C(P|X) \triangleq \sum_{i \in S(X)} \sum_{j \in N_i \cap P} c_{ij},$$

where $I_{i,j}$ is a $\{0, 1\}$ indicator function, the non-negative constants $c_{ij}$ model the cost of providing a protective barrier for all interactions between $i$ and $j$ over one time period, $S(X)$ denotes the set of susceptible nodes at state $X$, and
and the controlled dynamics for $X^S_i$ follow

$$X^S_i = X^S_i(1 - 1_{(i \in P)}Z^S_{i \rightarrow i}I_i) + \sum_{k=1}^{P} X^I_i \cdot Z^S_{i \rightarrow k},$$

and the controlled dynamics for $X^I_i$ are changed similarly.

### C. Optimal Control Problem

If we were strictly concerned with minimizing the total accumulated cost for our controller, it is feasible to take the action $P = \emptyset$ at all times. Doing so incurs zero cost. However, this may result in the infection persisting in the population for a long time. This is a socially undesirable outcome. In general, we may wish to consider the presence of infection as a sort of soft cost imposed on the controller.

Previous works studying optimal control of epidemics (e.g., [3]–[6]) often do so by posing a problem of the form

$$\min_{\pi \in \Pi} \mathbb{E}_\pi \left[ \sum_{\tau=0}^{\infty} J(X(\tau), \theta(\tau))[X], \right] \quad (7a)$$

$$J(X, \theta) = \mu c(\theta) + (1 - \mu)q(I(X)) \quad (7b)$$

where $\Pi$ is the set of all non-anticipating control policies which map observations of the process as it evolves to controls in the policy class. $\theta, c(\cdot)$ is the instantaneous cost of setting the processes parameters to $\theta$, $I(X)$ is a function which maps the state $X$ to the number of infected nodes in state $X$, and the function $q$ is some non-negative valued function which determines the extent to which we should care about the existence of infection. In our case, if it were so that the process $\{X(t)\}$ was a Markov chain on a small state space and the set of possible control actions were small, (7a) could be solved by treating it as a Markov decision process and applying a standard solution technique, e.g., value iteration. However, even for standard SIS processes, $\{X(t)\}$ evolves as a Markov chain with $2^n$ states, and there are $2^n$ possible choices of the set $P$. That is, the complexity of using a standard Markov decision process algorithm here is exponential, due to the curse of dimensionality. As such, it is an interesting task to construct a controller which allows for the same qualitative tradeoff as (7a), but is computationally tractable to implement.

To accomplish this, we consider applying controls which solve the infinite-horizon optimal control problem

$$\min_{P \subseteq V} h_X(P) \quad (8a)$$

$$h_X(P) = \mu C(P|X) + (1 - \mu)Q(P|X) \quad (8b)$$

where $C(P|X)$ is the cost function (5), and $Q(P|X)$ is the cost of applying the rollout policy which protects all susceptible nodes for all future times, given that the set of nodes $P$ is protected at the current time (see, e.g., [25, Section 6.4] for background on the use of rollout policies in infinite horizon optimal control). Intuitively, our controller designs the protection set $P$ at the current time while anticipating that at all future times, every reasonable action will be implemented to eradicate infection as quickly as possible. Thus, our controller anticipates that a cost $c_{ij}$ occurs at every time where exactly one of the node $i$ or $j$ is susceptible. That is, $Q$ is defined mathematically as

$$Q(P|X) \triangleq \mathbb{E}_{\Theta(P)} \left[ \sum_{\tau=1}^{\infty} \sum_{\{i, j\} \in E} (X^S_i(\tau) \oplus X^S_j(\tau))c_{ij}|X] \right], \quad (9)$$

where $\Theta(P)$ is the measure induced by protecting the set of nodes $P$ at the current time, and by $\oplus$ we denote the exclusive or operator, i.e. for two variables $Y, Z \in \{0, 1\},$

$$Y \oplus Z \triangleq \begin{cases} 0, & Y = 0, Z = 0; \\ 1, & Y = 1, Z = 0; \\ 1, & Y = 0, Z = 1; \\ 0, & Y = 1, Z = 1. \end{cases} \quad (10)$$

Note that because we update our decision $P$ at every time, this total protection strategy is not actually implemented. Rather, $Q$ plays the same role in (8a) as $q(I)$ plays in (7a). It penalizes the existence of infection in the network, and so allows a control designer a means for trading off between an immediate resource expenditure and the rate of decay in the population of infected individuals by appropriately selecting $\mu$. However, it is not immediately clear that actions can be efficiently computed as in (8a), as the set over which the optimum must be computed has $2^n$ elements, and it is not clear that the objective $h_X$ is sufficiently well structured so as to enable efficient optimization.

In the body of this paper, we are concerned with determining whether solutions of (8a) can be computed efficiently (i.e. in polynomial time with respect to the size of the graph), and whether the engendered controller provides reasonable behavior. We address computational efficiency in Section III-B, in which we show that the objective of the minimization in (8a) is submodular, which allows us to minimize $h_X$ using polynomial time algorithms. We assess the controller’s behavior in Section IV with a numerical example.

### III. Predictive Control of Generalized SIS

In this section, we develop the mathematical foundations required to efficiently implement a controller as described in Section II. Section III-A provides some technical preliminaries which are needed in our analysis. Section III-B demonstrates that the optimization problem (8a) has sufficient structure so as to allow for the use of efficient optimization algorithms, and provides some references to software packages that can be used to solve problem.

#### A. Mathematical Preliminaries

The key mathematical concept which allows for the efficient solution of (8a) is submodularity. Submodularity is a mathematical formalization of the concept of diminishing
returns: adding an object to a larger set has less of an impact than adding the same object to a smaller set. Formally, a submodular function satisfies the following definition.

**Definition 1 (Submodular Functions)** Let $\Omega$ be a finite ground set of objects, and suppose $f : 2^\Omega \to \mathbb{R}$, where $2^\Omega$ denotes the power set of $\Omega$, i.e. the set of all subsets of $\Omega$. The function $f$ is said to be submodular if and only if

$$f(Z \cup \{z\}) - f(Z) \leq f(Y \cup \{z\}) - f(Y) \quad (11)$$

holds for all $Y \subset Z \subseteq \Omega$, and $z \in \Omega \setminus Z$.

It is frequently the case that the submodularity of a complicated function is verified by reducing the proof to checking the submodularity of a simpler function. We use such an argument later (Section III-B), using the exclusive-or function as the simple function, which is submodular:

**Lemma 1 (Submodularity of Restricted Exclusive Or)**

Let $\Omega$ be a finite ground set, take $a, b \in \Omega$, $A, B \in \{0, 1\}$, and let $\oplus$ denote the operator defined by (10). The function

$$f(W) = A \mathbb{I}_{\{a \in W\}} \oplus B \mathbb{I}_{\{b \in W\}},$$

is submodular.

**Proof:** Note that if $A = B = 0$, then $f(W) = 0$, and is trivially submodular. Note also that if $A \neq B$, then $f(W) = A \mathbb{I}_{\{a \in W\}} + B \mathbb{I}_{\{b \in W\}}$, which is non-negative sum of submodular functions, so is submodular [26]. It remains to consider $A = B = 1$. In this case, if $a = b$, we have $f(W) = 0$, which is trivially submodular. It remains to consider the case in which $a \neq b$.

Take $Z \supseteq Y, z \in \Omega \setminus Z$, and suppose neither $a$ nor $b$ are in $Z$. By $Z \supseteq Y$, we have that neither $a$ nor $b$ are in $Y$. If $z \in \{a, b\}$, then $f(Z \cup \{z\}) - f(Z) = f(Y \cup \{z\}) - f(Y)$. If $z \notin \{a, b\}$, then $f(Z \cup \{z\}) - f(Z) = f(Y \cup \{z\}) - f(Y)$.

Now, take $Z \supseteq Y, z \in \Omega \setminus Z$, and suppose both $a$ and $b$ are in $Z$. The condition $z \in \Omega \setminus Z$ implies that $z \notin \{a, b\}$, and so $f(Z \cup \{z\}) - f(Z) = 0 = f(Y \cup \{z\}) - f(Y)$.

Finally, take $Z \supseteq Y, z \in \Omega \setminus Z$, and suppose exactly one of $a$ or $b$ is in $Z$. For concreteness, suppose $a \in Z$. There are two cases to consider. First, suppose $a \notin Y$. Then, if $z \notin \{b\}$, we have $f(Z \cup \{z\}) - f(Z) = 0 = f(Y \cup \{z\}) - f(Y)$. If $z \in \{b\}$, then $f(Z \cup \{z\}) - f(Z) = 1 \leq f(Y \cup \{z\}) - f(Y)$. Now, suppose $a \in Y$. If $z \notin \{b\}$, then $f(Z \cup \{z\}) - f(Z) = 0 = f(Y \cup \{z\}) - f(Y)$. If $z \in \{b\}$, then $f(Z \cup \{z\}) - f(Z) = 1 = f(Y \cup \{z\}) - f(Y)$.

**B. Efficiently Computing Optimal Sets of Protected Nodes**

A principal reason that submodularity is an important concept is that it allows for a variety of combinatorial optimization problems to be solved (or approximately solved) in polynomial time. In this subsection, we demonstrate that (8a) is one such problem. As it is well-known that the minimum of a submodular function over a finite ground set can be computed in time which grows polynomially with respect to the size of the ground set (see, e.g., [27], [28]), it suffices for us to demonstrate that the objective of (8a) is a submodular function. We accomplish this in the following result:

**Theorem 1 (Submodularity of Objective Function)**

Fix a particular generalized SIS process (as detailed in Section II-A), a number $\mu \in [0, 1]$, and a state $X$. Let $C$ be defined as in (5), and $Q$ as in (9). Then, the function $h_X(P)$ as defined by (8b) is a submodular set function, where the ground set is taken to be the set of susceptible nodes at state $X, S(X)$.

**Proof:** Since a function defined by a non-negative weighted sum of submodular functions is itself a submodular function [26], it suffices to show that $C$ and $Q$ are both submodular functions. We handle each separately.

**Submodularity of $C$:** Because the constants $c_{ij}$ are non-negative, it suffices to note that indicators of the form $f(W) = \mathbb{1}_{\{r \in W\}}$ are submodular. This is straightforward if $x \in \Omega \setminus W$, then $f(W \cup \{x\}) - f(W) = 1$ when $x = r$, and 0 otherwise, regardless of the choice of $W$. This implies that (11) is met with equality, satisfying Definition 1.

**Submodularity of $Q$:** Since the generalized SIS process defined in Section II-A is defined on a finite state space in finite time, we may construct a finite sample space for it. Call this sample space $\Xi$. Because having exactly one node in a pair of nodes $i$ and $j$ be susceptible is equivalent to having exactly one node in a pair of nodes belong to some infected compartment, we have

$$Q(P | X) = \mathbb{E}_{\xi \in \mathcal{P}} \sum_{\tau = 1}^{\infty} \sum_{(i,j) \in \mathcal{E}} (X^i_\tau(\tau) \oplus X^j_\tau(\tau)) c_{ij} |X|,$$

where we denote the dependence on the choice of protected nodes and the underlying sample $\xi$ explicitly, use the notation $Pr(\xi | X)$ for the probability that the sample $\xi$ is drawn from $\Xi$, given that the process is currently in state $X$, and define the shorthand notation $X^i_\tau = \sum_{k=1}^{\infty} X^i_{\tau+k}$. Since the terms $c_{ij}$ and $Pr(\xi | X)$ are nonnegative, it suffices to show that the terms $(X^i_\tau(\tau) ; P,\xi) \oplus X^j_\tau(\tau) ; P,\xi) c_{ij} Pr(\xi | X)$ are submodular functions.

Because the rollout policy removes all susceptible nodes which are adjacent to an infected node, we have that all nodes which are infected at some time $\tau \geq t + 1$ were either infected at time $t$, or became infected on the transition from time $t$ to time $t + 1$. In the case where the node $i$ was infected at time $t$, the definition of the process has that all indicators $X^i_\tau(\tau) ; P,\xi)$ are independent of the choice $P$, and are determined only by the particular choice of $\xi$. In particular, we have that

$$X^i_\tau(\tau) ; P,\xi) = \begin{cases} \mathbb{1}_{\{r \leq \mathcal{H}_\xi(\xi, X_i(l))\}}, & X^i_\tau = 1; \\ \mathbb{1}_{\{E \in \mathcal{P}\}} \mathbb{1}_{\{V_{j \in N_i, Y_j(\xi)}X^j_\tau\}} \mathbb{1}_{\{r \leq \mathcal{H}_\xi(\xi, X_i(l+1))\}}, & X^i_\tau = 1; \end{cases}$$

where $\mathcal{H}_\xi(\xi, X_i(l))$ is a random variable denoting the next time at which an event which transitions the node $i$ from infected to susceptible happens, given that the node is in its current compartment, and $\vee$ denotes the logical or operation, used here because all infection events have the same effect on an unprotected susceptible node.
the choice
immediately from noting that with
optimum, as it is the sum of a monotone increasing modular
the development of faster algorithms for computing the exact
objective function with respect to each sample path used can
simulate the evolution of the process with the sampled state
parameters, one can first sample from this, and then forward-
simulate the evolution of the process with the sampled state
and parameter set fixed. Verifying the submodularity of the
objective function with respect to each sample path used can
be done with the same argument as in the proof of Theorem
1, and so merits no further discussion here.

Remark 2 (Tuning Controller Behavior) Selecting \( \mu \)
appropriately is important. For small values of \( \mu \), the
objective of (8a) is dominated by the effect of \( Q(A|X) \). In
the extreme case where \( \mu = 0 \), the controller takes an action
which minimizes the future cost associated to guaranteeing
the infection spreads no further. For large values of \( \mu \), the
objective of (8a) is dominated by the effect of \( C(A|X) \). In
the extreme case where \( \mu = 1 \), it is simple to show that
the optimal action will be to protect no nodes, under any
circumstance. Intuitively, intermediate values of \( \mu \) balance
the immediate cost of resource expenditure against its long-
term consequences. This is seen in Figure 1.

IV. A Numerical Example

We consider a graph with 200 nodes. We assume that for
each \( i, j \in \mathcal{V} \) such that \( i \neq j \), the edge \( \{i, j\} \) is in the set of
graphs \( \mathcal{E} \) independently with probability 0.01. We assume
that an unprotected contact between two nodes results in an
infection spreading from the infected node to the susceptible
node with a probability randomly generated from the unit
interval. We assume that nodes come into contact with each
other a maximum of three times per day, and so edge costs
take values in the set \( \{0, 1, 2, 3\} \), where the value 0 is taken
if the edge \( \{i, j\} \notin \mathcal{E} \), and the other values are chosen
uniformly at random if \( \{i, j\} \in \mathcal{E} \). Because treatments
for sexually transmitted infections (e.g. chlamydia) often take in
excess of a week to be effective [34], the Markov chain used
to model the infectious compartments was chosen so as to
make the distribution of times from infection to susceptibility
uniformly distributed on the set \( \{7, 8, 9, 10\} \).

A plot demonstrating the effect of varying \( \mu \) is given
in Figure 1, where the lightest shaded region contains the
middle 98% of the 100 sample paths generated, the darker
shade of blue contains the middle 80% of the sample paths
generated, and the central blue line gives the sample average.
As anticipated in Remark 2, there is a continuous range
of values \( \mu \) over which the controller’s behavior markedly
changes. For this particular example, this region was found
to be the interval \([.8, .9]\). Note that in general, appropriate
values of \( \mu \) must be determined for each fixed model. It
may well be possible to automate a procedure for finding
appropriate values of \( \mu \) online. However, developing and
applying any such method would likely be quite technically
involved, and is best saved as a task for future work.

The behavior of the controller with \( \mu = 0.85 \) is given
in Figure 2, where the lightest shaded region contains the
middle 98% of the 100 generated samples, the darker shaded
region contains the middle 80% of the generated samples,
and the central blue line gives the sample average of the
process. We see that the controller drives the epidemic out
of the network quickly, while using resources at a lesser rate
than one which protects all susceptible nodes at all times.

V. Conclusions and Future Work

In this paper, we have developed a controller which
allocates discrete protective devices in order to mitigate the
spread of an epidemic process. Such a controller could be
useful in fighting many forms of biological disease, which
often do not confer immunity to people that have survived
infections, and so are well-modeled by \( SIS \)-type processes.

As topics for future work, one may consider the task of
developing efficient observers and estimators for the process’
state and parameters. While Remark 1 suggests how the

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{The effect of varying \( \mu \) to tune controller. As expected, the
controller’s behavior experiences a phase transition from a domain
in which future cost is prioritized (here, below \( \mu = 0.8 \), and a
domain in which instantaneous spending is prioritized (here, above
\( \mu = 0.88 \)). Note: the simulation parameters reported in the example
of Section IV were used to generate this figure.}
\end{figure}
controller can be used in the case where the state and spreading parameters are not perfectly known, developing numerically efficient methods for providing the required distributions remains an important open task. We believe the work presented here provides clear motivation for future researchers to engage in such work, and so is valuable itself.

REFERENCES

[1] C. A. Arias and B. E. Murray, “Antibiotic-Resistant Bugs in the 21st Century: A Clinical Super-Challenge,” The New England Journal of Medicine, pp. 439–443, 2009.

[2] W. O. Kermack and A. G. McKendrick, “A Contribution to the Mathematical Theory of Epidemics,” Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences, vol. 115, no. 772, pp. 700–721, 1927.

[3] K. H. Wickwire, “Optimal Isolation Policies for Deterministic and Stochastic Epidemics,” Mathematical Biosciences, vol. 346, no. 3, pp. 325–346, 1975.

[4] S. P. Sethi, “Optimal Quaranitme Programmes for Controlling an Epidemic Spread,” The Journal of the Operational Research Society, vol. 29, no. 3, pp. 265–268, 1978.

[5] H. Behncke, “Optimal control of deterministic epidemics,” Optimal Control Applications and Methods, vol. 21, no. 6, pp. 269–285, 2000.

[6] E. Gubarr and Q. Zhu, “Optimal control of influenza epidemic model with virus mutations,” 2013 European Control Conference, pp. 3125–3130, 2013.

[7] C. Nowzari, V. M. Preciado, and G. J. Pappas, “Analysis and Control of Epidemics,” IEEE Control Systems Magazine, vol. 36, no. 1, pp. 26–46, 2016.

[8] N. Gast, B. Gaujal, and J. Le Boudec, “Mean Field for Markov Decision Processes: From Discrete to Continuous Optimization,” IEEE Transactions on Automatic Control, vol. 57, no. 9, pp. 2266–2280, 2012.

[9] K. Drakopoulos, A. Ozdaglar, and J. Tsitsiklis, “An Efficient Curing Policy for Epidemics on Graphs,” IEEE Transactions on Network Science and Engineering, vol. 1, no. 2, pp. 67–75, 2015.

[10] K. Scaman, A. Kalogeratos, and N. Vayatis, “Suppressing Epidemics in Networks Using Priority Planning,” IEEE Transactions on Network Science and Engineering, vol. 3, no. 4, pp. 271–285, 2016.

[11] N. J. Watkins, C. Nowzari, and G. J. Pappas, “Robust Economic Model Predictive Control of Continuous-time Epidemic Processes,” arXiv preprint, no. arXiv:1707.00742, 2017.

[12] D. Kempe, J. Kleinberg, and E. Tardos, “Maximizing the spread of influence through a social network,” in Proceedings of the Ninth ACM SIGKDD International Conference on Knowledge Discovery and Data Mining, pp. 137–146, 2003.

[13] E. Mossel and S. Roch, “On the Submodularity of Influence in Social Networks,” in Proceedings of the Thirty-Ninth Annual ACM Symposium on Theory of Computing, pp. 128–134, 2007.

[14] X. He and D. Kempe, “Robust Influence Maximization,” in Proceedings of the ACM SIGKDD International Conference on Knowledge Discovery and Data Mining, pp. 795–804, 2016.

[15] C. J. Kuhlman, G. Tuli, S. Swarup, M. V. Marathe, and S. S. Ravi, “Blocking Simple and Complex Contagion By Edge Removal,” in Proceedings - IEEE International Conference on Data Mining, ICDM, pp. 399–408, 2013.

[16] I. Bogunovic, “Robust protection of networks against cascading phenomena,” PhD thesis, ETH Zürich, 2012.

[17] M. Kretzschmar, Y. T. H. P. van Duynhoven, and A. J. Severijnen, “Modelling prevention strategies for Gonorrhea and Chlamydia using stochastic network simulations,” American Journal of Epidemiology, vol. 144, 3, no. 3, pp. 306–317, 1996.

[18] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, and Y. Moreno, “Discrete-time Markov chain approach to contact-based disease spreading in complex networks,” EPL, vol. 89, 2010.

[19] H. J. Ahn and B. Hassibi, “On the Mixing Time of the SIS Markov Chain Model for Epidemic Spread,” in Proceedings of the 2014 IEEE Conference on Decision and Control, pp. 6221–6227, 2014.

[20] N. A. Ruhi and B. Hassibi, “SIRS epidemics on complex networks: Concurrence of exact Markov chain and approximated models,” in Proceedings of the IEEE Conference on Decision and Control, pp. 2919–2926, 2015.

[21] N. A. Ruhi, C. Thrampoulidis, and B. Hassibi, “Improved Bounds on the Epidemic Threshold of Exact SIS Models on Complex Networks,” in Proceedings of the IEEE Conference on Decision and Control, 2016.

[22] C. Eksin, J. S. Shamma, and J. S. Weitz, “Disease dynamics in a stochastic network game: a little empathy goes a long way in averting outbreaks,” Scientific Reports, vol. 7, no. July 2016, p. 44122, 2017.

[23] N. J. Watkins, C. Nowzari, and G. J. Pappas, “Inference, Prediction, and Control of Networked Epidemics,” in IEEE American Control Conference, (Seattle, WA, USA), pp. 5611 – 5616, 2017.

[24] A. Bobbio, A. Horvth, M. Scarpa, and M. Telek, “Acyclic discrete phase type distributions: Properties and a parameter estimation algorithm,” Performance Evaluation, vol. 54, no. 1, pp. 1–32, 2003.

[25] D. P. Bertsekas, Dynamic Programming and Optimal Control: Volume 1, third ed., 2005.

[26] S. T. McCormick, “Discrete Optimization,” Handbooks in Operations Research and Management Science, vol. 12, pp. 321–391, 2005.

[27] S. T. McCormick, “Submodular Function Minimization,” Handbooks in operations research and management science, vol. 12, pp. 321–391, 2005.

[28] S. Iwata, “Submodular function minimization,” Mathematical Programming, vol. 112, no. 1, pp. 45–64, 2007.

[29] D. Krause, “SFO: A Toolbox for Submodular Function Optimization,” Journal of Machine Learning Research, vol. 11, pp. 1141 – 1144, 2010.

[30] S. Fujishige, T. Hayashi, and S. Isotani, “The Minimum-Norm-Point Algorithm Applied to Submodular Function Minimization and Linear Programming,” RIMS, pp. 1–19, 2006.

[31] D. Chakrabarty, P. Jain, and P. Kothari, “Provable Submodular Minimization using Wolfe’s Algorithm,” in Advances in Neural Information Processing Systems, pp. 802–809, 2014.

[32] S. Jegelka, H. Lin, and J. Bilmes, “On fast approximate submodular minimization,” in Advances in Neural Information Processing Systems, pp. 460–468, 2011.

[33] D. Chakrabarty, Y. T. Lee, A. Sidford, and S. C.-w. Wong, “Subquadratic Submodular Function Minimization,” arXiv, vol. arXiv:1610, pp. 0–21, 2016.

[34] “Chlamydia - CDC Fact Sheet (Detailed).”