Optimal Control of Epidemic Information Dissemination Over Networks

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***Abstract*—Information dissemination control is of crucial importance to facilitate reliable and efficient data delivery, especially in networks consisting of time-varying links or heterogeneous links. Since the abstraction of information dissemination much resembles the spread of epidemics, epidemic models are utilized to characterize the collective dynamics of information dissemination over networks. From a systematic point of view, we aim to explore the optimal control policy for information dissemination given that the control capability is a function of its distribution time, which is a more realistic model in many applications. The main contributions of this paper are to provide an analytically tractable model for information dissemination over networks, to solve the optimal control signal distribution time for minimizing the accumulated network cost via dynamic programming, and to establish a parametric plug-in model for information dissemination control. In particular, we evaluate its performance in mobile and generalized social networks as typical examples.**

***Index Terms*—Epidemic model, information dissemination control, information dynamics, malware propagation, message delivery, mobile and social networks.**

I. Introduction

With the advance of modern technologies and the intricate connections between communication devices, attaining information dissemination control in such complicated networks has received tremendous attention to facilitate reliable and efficient communications [1], [2], e.g., content delivery and online advertisement, to name a few. Modern communication networks are composed of several interdependent networks, including social networks and physical communication networks, where the nodes are connected by intermittent links (e.g., vehicular communication), delocalized links (e.g., email from your friends) and localized links (e.g., message from proximity). Although modern technology benefits from diverse links, in the meanwhile these time-varying and heterogeneous links unintentionally nurture the breeding of malware and incur data deluge. Without adequate control, these redundant or undesired information may consume tremendous network resources resulting in disastrous damage to network operations and incurring erroneous message delivery. For instance, when disseminating emergent weather reports such as news of hurricanes and typhoons, one may want to come up with an information dissemination control scheme that is capable of updating imminent messages and deleting outdated messages in an timely manner. Since these messages are disseminated through various interfaces, e.g., televisions, Internet, and instant messages, a reasonable model for describing information dissemination dynamics and an effective control policy must be employed to evaluate and improve the system performance.

Investigating information dissemination control has twofold purposes. On one hand, studying information dissemination dynamics provides useful insights on devising efficient message delivery protocols to enhance system performance. On the other hand, we can mitigate the damage caused by malware and enhance system reliability. Despite their purposes, these information share common attributes in terms of spreading patterns. Since the abstraction of information dissemination much resembles the spread of epidemics [3]–[6], epidemic models [7]–[9] are used to specify information dissemination dynamics. Based on the epidemic models, we establish a framework for determining optimal control signal distribution time to minimize the accumulated network cost. In particular, the control signal may be the time-to-live packet information for data delivery or the security patch distribution strategy for malware propagation.

Inspired by epidemiology, we relate information dissemination to an epidemic by categorizing the status of a node into three states. Analogously, a node is in the infected state if it receives the information and becomes an infectious node. A node is in the recovered state if it is immune to the information (i.e., it refuses to receive the information). A node is in the susceptible state if it is neither in the infected state nor in the recovered state (i.e., it is still vulnerable to the information). This epidemic model is known as the susceptible-infected-recovered (SIR) model [7]–[9]. Traditionally, most research implicitly assume that the control capability (i.e., the ability to recover from infection) takes effect immediately right after the information dissemination. However, this assumption

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cannot be viable in communication networks, especially for the execution of real-time applications such as antivirus process [10], [11], since the control signals (e.g., security patches or system updates) are usually not available when a new malware emerges. Alternatively, we consider a more realistic scenario that the control capability is a function of its distribution time, and determining the optimal control signal distribution time becomes challenging in the sense that the overall network cost is associated with the distribution time.

In addition, we point out that the immunity mechanisms in epidemiology also have direct mapping to information dissemination control. Following the notions from epidemiology, self healing scheme refers to the recovery of an infected node, and vaccine spreading scheme refers to the instance that a recovered node participates in vaccinating the susceptible nodes against the epidemic. Throughout this paper, we will investigate the engineering interpretations and the effects of these two immunity schemes on information dissemination control. To the best of authors' knowledge, the tradeoffs between the time-dependent control capability and the resulting information dissemination dynamics still remain open [11], and the task is further complicated in networks of time-varying links (e.g., mobile networks), networks of random links (e.g., opportunistic networks) and networks of heterogeneous links (e.g., generalized social networks [12]).

To solve the optimal control signal distribution time, we first formulate the problem via optimal control theory [13] with an aim to minimize the accumulated cost, which relates not only to the damage caused by malware but also the number of replicated data packets in relay-assisted networks. However, optimal control theory assumes full manipulation of the control function and therefore its solution is inadequate for determining the optimal control signal distribution time. Consider time-dependent control capability, dynamic programming [14] is proposed to obtain the optimal control signal distribution time in real time with respect to the information dissemination process. We also provide early-stage analysis [15] to obtain closed-form expressions of such SIR model. Using the proposed techniques, we show that the accumulated cost for information dissemination in mobile networks and generalized social networks can be greatly reduced via the proposed approach. Furthermore, the controllability of a network is illustrated by the phase diagram to study the relations between control capability and infection rate.

In summary, our main contributions are to provide a theoretic framework for evaluating information dissemination dynamics and to establish a parametric plug-in model for information dissemination control in different networks. Applying the proposed method to mobile and generalized social networks, our method is shown to capture the information dissemination dynamics and attain reliable information dissemination control. The applications include but are not limited to security patch distribution in computer networks, vaccination strategy in contact networks, broadcast protocol design in communication systems, content delivery and online advertisement, to name a few.

The rest of this paper is organized as follows. Section II summarizes the related works. We provide preliminary knowledge and problem formulation for control of epidemic information dissemination in Section III. We investigate the performance of information dissemination and the optimal control signal distribution time in mobile networks and generalized social networks in Section IV and Section V, respectively. Finally, Section VI concludes this paper.

II. Related Works

Information dissemination is influenced by network topology and it has been extensively investigated in the contexts of synchronization and consensus in static or dynamic multiagent systems [16]–[21]. Synchronization and consensus can be regarded as performing a specific collective networked task by exchanging or updating information with a subset of nodes (usually neighboring nodes) in the network, whereas information dissemination refers to an information diffusion process over a network [6]. In other words, research on synchronization and consensus are more concerned with the criterions to guarantee convergence and the ways to improve convergence rate, whereas information dissemination focuses on the spreading pattern and the associated cost, and on how to attain adequate control for information dissemination. In particular, the aforementioned aspects establish a link between information dissemination and epidemiology.

In recent years, epidemic models and their applications have been widely investigated in communication systems [3], [10]. Malicious codes such as Internet worms may leverage the inherently fixed topology to sabotage network operations [22], [23] due to complicated interactions and immense size of communication networks. In [24] and [25], the authors find that the spread of Internet worms is similar to the spreading patterns of epidemics and it poses severe threats on system security. Castellano and Pastor-Satorras [26] showed that the epidemic will break out if the infection rate exceeds a certain threshold in a network with fixed topology, and the threshold tends to vanish when the network has a skewed degree distribution [27], such as the Internet [28]. Chen and Carley [29] proposed countermeasure competing strategies based on the idea that computer viruses and countermeasures spread through two separate but interlinked complex networks.

Investigations toward dynamics of Internet worm propagation show that the damage caused by Internet worms can be greatly mitigated with efficacious detection techniques or defense at the imminent stages [4], [15], [30]–[34]. Hu *et al.* [35] also showed that a tightly interconnected proximity network can be exploited as a substrate for spreading of malware to launch massive fraudulent attacks. Moreover, in case of mobile environments, malware can still propagate in such intermittently connected networks by taking advantage of opportunistic encounters [36]. Wang *et al.* [37] studied spreading patterns of mobile phone viruses which may traverse through multimedia messaging services (MMS) or Bluetooth by simulations. Cheng *et al.* [12] further modeled malware propagation in generalized social networks consisting of delocalized and localized links. Ramachandran and Sikdar [38] pointed out the vulnerabilities of smart phone networks, where a malware is able to transfer between wired and wireless

networks. The results show that the contamination of malware speeds up drastically if a malware is able to propagate through heterogenous links.

Optimal control theory [13] has gained its popularity across different fields. Khouzani *et al.* [39] investigated the tradeoffs between energy consumption and malware attack capability in battery-constrained mobile wireless networks. Altman *et al.* [40] formulated the energy tradeoffs between activation and transmission in delay tolerant networks as an optimal control problem. Jung *et al.* [41] used optimal control theory to devise optimal strategy for preventing avian influenza pandemic. Khouzani *et al.* [42] derived the structural characteristics of the optimal communication range based on optimal control theory for optimal quarantine of malware in wireless networks. Note that the aforementioned works presume full manipulation of the control function, whereas we move one step further to consider a more realistic scenario that the control capability is associated with its distribution time. Our previous work [43] focuses on optimal control of information dissemination in mobile ad hoc networks. In this paper, we enlarge the scope to embrace different networking paradigms and provide a dynamic programming approach to determine the optimal control signal distribution time.

In addition to malware propagation modeling, epidemic models can also be utilized to evaluate the performance of data delivery in different networks of interest. Moreno *et al.* [44] analyzed rumor spreading dynamics in social networks. Shah and Zaman [45] used epidemic model to estimate the source of a rumor in a network. De *et al.* [46] evaluated the vulnerability of broadcast protocols in wireless sensor networks. In cases where networks are intermittently connected, such as delay tolerant networks [47], a message is delivered in a store-and-forward manner and the transportation much resembles the spread of epidemics [5], [48], which is known as epidemic routing [49]. Intuitively, epidemic routing incurs tremendous system loads due to its inherently spreading nature, and thus adequate control is required to enhance the system performance [50]. It is worth mentioning that despite the variants of these research topics, ordinary differential equations (ODEs) have been shown to be a good fit for analyzing information dissemination dynamics from the approximation of Markov chains [51].

III. Problem Formulation

A. Epidemic Information Dissemination and SIR model

Analog to epidemiology, a node is in the infected state if it receives the information and turns itself into an infectious node. A node that recovers from the epidemic or becomes a vaccinee against the epidemic is said to be in the recovered (immune) state. Please note that a node transits from the infected state to the recovered state for the former case while a node transits from the susceptible state to the recovered state for the latter case. Only susceptible nodes are vulnerable to the epidemic and the recovered nodes are immune to the epidemic for good. Throughout this paper, such state transitions are referred to as the SIR model, where $S(t)$, $I(t)$ and $R(t)$ are the normalized susceptible, infected and recovered population at time t, respectively, i.e., $S(t)+I(t)+R(t) = 1$. Furthermore, we define a network to be *p-controllable* if there are p fraction of nodes left unharmed (i.e., they remain in the susceptible state) when the epidemic comes to a halt. The p-controllability of a system serves as the performance metric suggesting that we are able to control the information dissemination in a network to a certain extent without perturbing too many nodes.

B. Self Healing and Vaccine Spreading

We consider a more realistic scenario that it takes time to strengthen the control capability when the epidemic emerges, and the cost of reproduction can be high so that the control capability is determined after the control signal is distributed, such as system renewals or hardware updates. We assume the control capability to be a positive and nondecreasing function $f(T_D)$, where T_D is the time instance for the control signal distribution. That is, upon the reception of the healing signal, a node in the infected state is able to recover from the epidemic with probability $f(T_D)$. Note that if $f(T_D)$ is a positive constant, then immediate control signal distribution is preferred to minimize the network cost. However, if $f(T_D)$ is an increasing function of distribution time, it remains open to determine the optimal control signal distribution time as, intuitively, early control signal distribution leads to slight cure of the infected population while late control signal distribution fails to prevent the epidemic from outbreak. Moreover, if the nodes in the recovered state participate in distributing the vaccine signals to the susceptible nodes against the epidemic, a susceptible node becomes a vaccinee and is therefore immune to the epidemic. The probability that a susceptible node becomes a vaccinee is denoted by κ. Such cooperative immunity scheme may further mitigate the dissemination of epidemic.

C. Fluid Analysis of SIR model

Consider the immunity schemes and the time-dependent control capability, let $u(t)$ be the recover probability of the self healing scheme, where

$$u(t) = \begin{cases} 0, & t < T_D, \\ f(T_D), & t \geq T_D. \end{cases} \tag{1}$$

By substituting the equation $S(t) = 1-I(t)-R(t)$ and relaxing the states to be continuous and nonnegative valued, we have, for a small interval Δt

$$I(t+\Delta t) = I(t) + \Upsilon_{S\to I}(t)\Delta t - \Upsilon_{I\to R}(t)\Delta t \tag{2}$$

where $\Upsilon_{X\to Y}(t)$ is the expected population transition rate from state X to state Y at time t. We obtain the first-order ODE (state equation)

$$\begin{aligned} \dot{I}(t) &= \lim_{\Delta t\to 0} \frac{I(t+\Delta t) - I(t)}{\Delta t} = \Upsilon_{S\to I}(t) - \Upsilon_{I\to R}(t) \\ &\triangleq G_I(I(t), R(t), u(t)). \end{aligned} \tag{3}$$

Similarly, let $\phi(t)$ be the recover probability of the vaccine spreading scheme, the ODE of recovered population is

$$\begin{aligned} \dot{R}(t) &= \Upsilon_{I\to R}(t) + \Upsilon_{S\to R}(t) \\ &\triangleq G_R(I(t), R(t), u(t), \phi(t)) \end{aligned} \tag{4}$$

where

$$\phi(t) = \begin{cases} 0, & t < T_D, \\ \kappa, & t \geq T_D. \end{cases} \tag{5}$$

When $\kappa = 0$, the fluid model degenerates to a noncooperative network where no nodes participate in vaccine spreading. Without loss of generality, we use the state equations of vaccine spreading to obtain the optimal control signal distribution time T_D^* since self healing is a special case of vaccine spreading when there is no cooperation (i.e., $\kappa = 0$).

D. Optimal Control

The ultimate goal of this paper is to determine the optimal distribution time T_D^* such that the accumulated cost caused by the epidemic is minimized. Via optimal control theory [13], we aim to solve the optimization problem

$$\begin{aligned} \text{Minimize} \quad & J = \int_{T_0}^{T_f} [NI(t)]^\beta + \nu \cdot u^2(t)\, dt \\ \text{Subject to} \quad & \dot{I}(t) = G_I(I(t), R(t), u(t)) \\ & \dot{R}(t) = G_R(I(t), R(t), u(t), \phi(t)) \\ & S(t) + I(t) + R(t) = 1, \\ & S(t) \geq 0, \ I(t) \geq 0, \ R(t) \geq 0 \end{aligned} \tag{6}$$

where $\beta > 0$ represents the severeness of the epidemic, T_0 is the initial time which is set to be 0 and T_f is the completion time which is assumed to be free. ν is the coefficient representing the cost of control signal distribution with respect to the information dissemination process and for simplicity it is normalized to $\nu = \frac{1}{2}$. If $\nu = 0$, then the cost of control signal distribution is irrelevant of the information dissemination process. The performance measure J represents the accumulated cost caused by the epidemic and it takes its quadratic form for the control function $u(t)$ such that it is jointly convex in $I(t)$ and $u(t)$. The physical interpretation of J is that it is proportional to the accumulated infected population, which relates to the number of nodes which have received the information over time. Moreover, when $\beta = 1$, it accounts for the accumulated infected population from T_0 to T_f, which coincides with the performance measure in various networks of our interest [3], [42], [52].

With (6), we aim to find the optimal control signal distribution time T_D^* such that $T_D^* = \arg\min_{T_D} J$. By Pontryagin's minimum principle [53], if $G_I(I(t), R(t), u(t))$ and $G_R(I(t), R(t), u(t), \phi(t))$ are jointly concave in $I(t)$, $R(t)$, $u(t)$ and $\phi(t)$, the optimal control function $u^*(t)$ can be obtained by minimizing the Hamiltonian (Lagrangian dual function) with costate variables $\Lambda_I(t)$ and $\Lambda_R(t)$, where

$$\begin{aligned} &\mathcal{H}(I(t), R(t), u(t), \phi(t), \Lambda_I(t), \Lambda_R(t)) \\ &= J(I(t), u(t)) + \Lambda_I(t) G_I(I(t), R(t), u(t)) \\ &\quad + \Lambda_R(t) G_R(I(t), R(t), u(t), \phi(t)). \end{aligned} \tag{7}$$

The costate variables are updated by the costate equations

$$\dot{\Lambda}_I(t) = -\frac{\partial \mathcal{H}}{\partial I} \ \ \dot{\Lambda}_R(t) = -\frac{\partial \mathcal{H}}{\partial R} \tag{8}$$

where $\dot{\Lambda}_I(t) \geq 0$ and $\dot{\Lambda}_R(t) \geq 0$ with boundary conditions $\Lambda_I(T_f) = \Lambda_R(T_f) = 0$. Note that during the update process, the negative state values are truncated to zero such that the nonnegativity state constraints $(S(t), I(t), R(t) \geq 0)$ are satisfied.

The solution of optimal control theory resides in the fact that there is no inherent restriction on the control function $u(t)$. However, it is worth noting that when the control capability is associated with T_D, the solution of optimal control theory only provides the trends of the system outputs and may fail to be a feasible operation for control signal distribution. Despite its impracticality, the results obtained from Pontryagin's minimum principle provide performance comparisons to our proposed approach. To compensate the insufficiency of optimal control theory, we adopt dynamic programming [14] to solve the optimal control signal distribution time. By discretizing the time into M intervals with length $\Delta t = \frac{T_f}{M}$, we define the cost C_m as a function of the infected population at the mth period and the newly infected population between the mth and $m+1$th stage, $0 \leq m \leq M-1$, where

$$\begin{aligned} C_m &= [NI(m\Delta t) + NG_I(I(m\Delta t), R(m\Delta t), u(m\Delta t)) \cdot \Delta t]^\beta \\ &= [NI((m+1)\Delta t)]^\beta . \end{aligned} \tag{9}$$

Let $V_m(I(m\Delta t), R((m\Delta t)), u(m\Delta t))$ denote the accumulated cost from the mth stage with terminal condition $V_M(I(M\Delta t), R(M\Delta t), u(M\Delta t)) = 0$ (i.e., the entire system is in its stable stage), the optimal distribution time can be obtained by solving the optimality equation

$$V_m = \min_{a_m \in \{0,1\}} \{C_m + V_{m+1}\}, \ 0 \leq m \leq M-1 \tag{10}$$

where $a_m = 1$ means that the control signal is distributed and the immunity mechanisms take effect from the mth stage. That is, $T_D^* = m\Delta t$ and $f(m\Delta t) = f(n\Delta t), \ \forall\, n \geq m$. V_0 represents the minimum accumulated cost which is equivalent to the performance measure J in (6). Equation (10) is equivalent to finding an optimal one-time switch from 0 to 1 among all possible one-time switch paths of the M stages to minimize the accumulated cost, and it can be solved via Bellman-Ford algorithm [14] with $O(2^M)$ complexity. In other words, incorporating the information dissemination process and the time-dependent control capability, the optimal control signal distribution time can be obtained via dynamic programming in (10) in real time to minimize the accumulated network cost.

E. Early-Stage Analysis

In addition to dynamic programming approach, early-stage analysis provides an analytically tractable model and serves as a quick reference to system monitoring and defense at the imminent phase [15]. At early stages before the outbreak of epidemic (e.g., malware proliferation or data deluge), most of the nodes remain in the susceptible state, and the state equations can be approximated as a coupling regulator problem with $S(t) \approx 1$. The advantage of early-stage analysis is that it relaxes the need for solving the simultaneous ODEs for optimal control as discussed in Section III-D, but it may overestimate the spread of epidemic and control signal

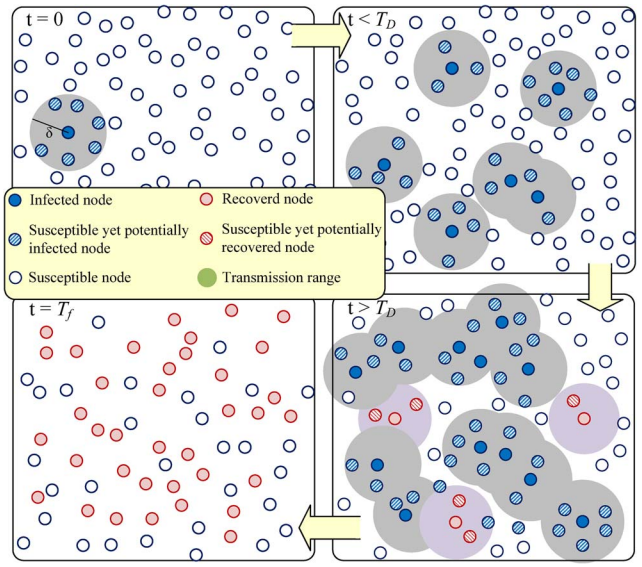

Fig. 1. Illustration of information dissemination and control signal distribution in mobile networks. A node is infected at $t = 0$. Susceptible nodes may be infected (vaccinated) by the nearby infected (recovered) nodes. T_D denotes the control signal distribution time and T_f denotes the time instance for the eradication of epidemic.

distribution with the assumption that $S(t) \approx 1$ at early stages. Throughout this paper, we use early-stage analysis as the performance benchmark to dynamic programming approach.

IV. Information Dissemination in Mobile Networks

Consider a mobile network where N nodes move around in a $L \times L$ square area with identical transmission range δ. The dynamics of information dissemination and control signal distribution are illustrated in Fig. 1. Let λ be the pairwise infection rate, η be the average number of neighboring nodes and $\hat{X}(t) = N \cdot X(t)$ be the subpopulation of state X at time t. From fluid analysis [1], [8], [9], we have

$$\Upsilon_{S\to I}(t) = \frac{1}{N}\lambda\eta\hat{I}(t)S(t) = \lambda\eta I(t)S(t) \tag{11}$$

$$\Upsilon_{I\to R}(t) = \frac{1}{N}u(t)\hat{I}(t) = u(t)I(t) \tag{12}$$

where $S(t)$ accounts for the fraction of susceptible nodes to be compromised and $\eta = \pi\delta^2/L^2$ is the average number of neighboring nodes. The state equation of $I(t)$ becomes

$$\dot{I}(t) = \lambda\eta I(t)S(t) - u(t)I(t). \tag{13}$$

Similarly, with $\Upsilon_{S\to R}(t) = \phi(t)\eta S(t)R(t)$, we have

$$\dot{R}(t) = u(t)I(t) + \phi(t)\eta S(t)R(t). \tag{14}$$

A. Optimal Control

To solve the optimization problem in (6), with (13) and (14) we write the Hamiltonian in (7) as

$$\mathcal{H} = [NI(t)]^\beta + \frac{1}{2}u^2(t) + \Lambda_I(t)\left[\lambda\eta S(t)I(t) - u(t)I(t)\right] + \Lambda_R(t)\left[u(t)I(t) + \phi(t)\eta S(t)R(t)\right] \tag{15}$$

from which the costate equations are $\dot{\Lambda}_I(t) = -\frac{\partial\mathcal{H}}{\partial I}$ and $\dot{\Lambda}_R(t) = -\frac{\partial\mathcal{H}}{\partial R}$. By Pontryagin's minimum principle, since the state equations $G_I(\cdot)$ and $G_R(\cdot)$ are jointly concave in the corresponding states and control function, optimal values of state and costate variables exist according to the optimal control function $u^*(t)$. We rewire the Hamiltonian in (15) with the switching function $\theta^*(t) \triangleq \left[\Lambda_I^*(t) - \Lambda_R^*(t)\right]I^*(t)$ as

$$\mathcal{H} = [NI^*(t)]^\beta + \frac{1}{2}u^2(t) - \theta^*(t)u(t) + \eta S^*(t)\left[\Lambda_I^*(t)\lambda I^*(t) + \Lambda_R^*(t)\phi(t)R^*(t)\right]. \tag{16}$$

The unconstrained optimal control function $U^*(t)$ that minimizes J is the solution of the equation $\frac{\partial\mathcal{H}}{\partial u} = 0$, and $U^*(t) = \theta^*(t)$, i.e, $U^*(t)$ can be obtained by solving the state and costate equations. Moreover, given that $u(t)$ is a probability which is confined in $[0, 1]$, we have the admissible (constrained) optimal control function

$$u^*(t) = \begin{cases} 0, & \theta^*(t) \le 0, \\ \theta^*(t), & \theta^*(t) \in (0, 1) \\ 1, & \theta^*(t) \ge 1 \end{cases} \tag{17}$$

where $u^*(t)$ is a saturation function of $U^*(t)$. Some more advanced methods can be used to obtain a constrained optimal control function [54], [55]. Note that the admissible optimal control function $u^*(t)$ is obtained by assuming that immediate reaction is permissible from the initial time, whereas in (1) we consider a more realistic scenario that the state transition from the infected state to the recovered state takes place only after the control signal distribution time T_D^*, which can be obtained via dynamic programming in (10). Therefore, information dissemination control using (17) is an unattainable lower bound with respect to the time-dependent control capability.

B. Early-Stage Analysis

At some early stage t', we assume $I(t') \approx I_0$ and $S(t') \approx 1$, where I_0 is the initially normalized infected population. The state equation of the recovered population in (14) can be simplified as

$$\dot{R}(t') = I_0u(t) + \phi(t)\eta R(t'). \tag{18}$$

With the initial condition $R(0) = 0$, we have

$$R(t') = \frac{I_0u(t)}{\phi(t)\eta}\exp\left\{\phi(t)\eta t'\right\} - \frac{I_0u(t)}{\phi(t)\eta} \approx I_0u(t)\left[t' + \frac{\phi(t)\eta}{2}t'^2\right]. \tag{19}$$

The approximation is based on the second-order approximation that $\exp\{x\} \approx 1 + x + \frac{x^2}{2}$ when x is quite small. Therefore, we obtain

$$I(t') = 1 - I_0 - I_0u(t)\left[t' + \frac{\phi(t)\eta}{2}t'^2\right]. \tag{20}$$

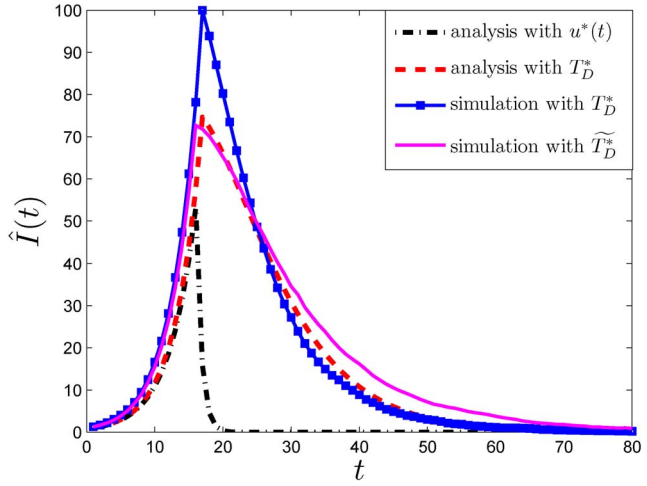

Fig. 2. Infected population under self healing scheme in mobile networks. $N = 1000$, $L = 1000$, $I_0 = 1/N$, $\delta = 2$, $\lambda = 0.25$, $\alpha = 2$, $\beta = 2$, $\kappa = 0$, $T_f = 80$, $M = 1000$, $\Lambda_I(0) = 15$, $\Lambda_R(0) = 10$, $t' = 1$, and $c = 10^{-3}$ over 300 simulations.

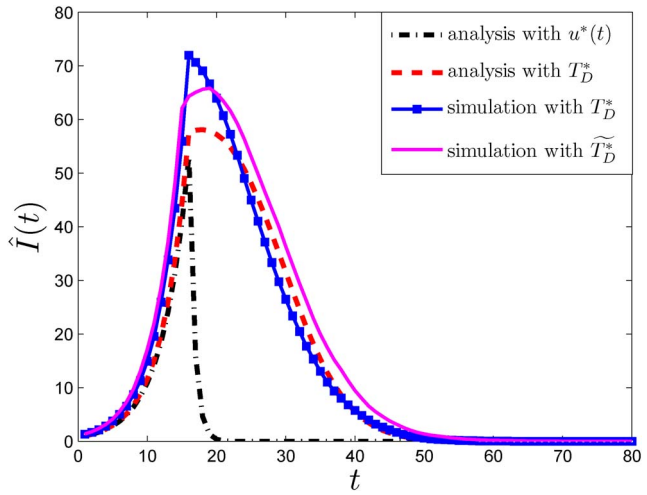

Fig. 3. Infected population under vaccine spreading scheme in mobile networks. $N = 1000$, $L = 1000$, $I_0 = 1/N$, $\delta = 2$, $\lambda = 0.25$, $\alpha = 2$, $\beta = 2$, $\kappa = 0.1$, $T_f = 80$, $M = 1000$, $\Lambda_I(0) = 15$, $\Lambda_R(0) = 10$, $t' = 1$ and $c = 10^{-3}$ over 300 simulations.

The state equation of the infected population in (13) becomes

$$\dot{I}(t) = [\lambda\eta S(t') - u(t)]I(t). \tag{21}$$

Therefore by (1) and (5), we obtain

$$I(t) = \begin{cases} I_0 \exp\left\{\lambda\eta S(t')t\right\}, & t < T_D \\ I_0 \exp\left\{[\lambda\eta S(t') - f(T_D)]t\right\}, & t \geq T_D. \end{cases} \tag{22}$$

Note that if we choose $t' = 0$, early-stage analysis does not reveal the effects of vaccine spreading. The closed-form expression of performance measure J in (6) becomes

$$\begin{aligned} J &= \int_0^{T_f} [NI(t)]^\beta + \frac{1}{2}u^2(t)\, dt \\ &= \int_0^{T_D} [NI(t)]^\beta dt + \int_{T_D}^{T_f} [NI(t)]^\beta + \frac{1}{2}f^2(T_D)\, dt \end{aligned} \tag{23}$$

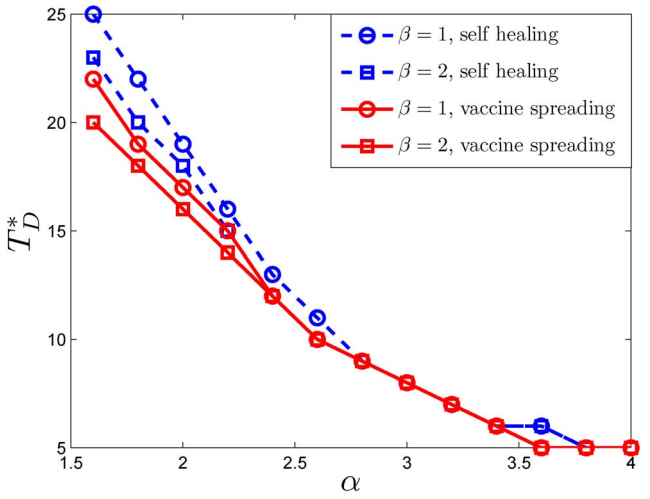

Fig. 4. Optimal control signal distribution time via dynamic programming under different (α,β) configurations in mobile networks. $N = 1000$. $L = 1000$. $I_0 = 1/N$. $\delta = 2$. $\lambda = 0.25$. $\kappa = 0.2$. $T_f = 200$. $M = 1000$. $t' = 1$. $c = 10^{-3}$.

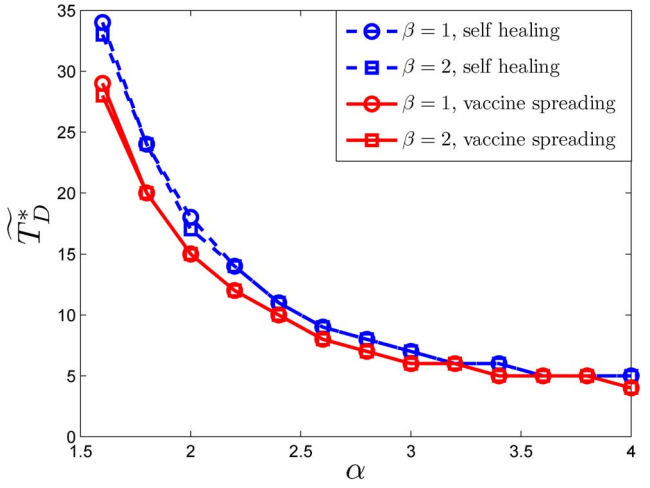

Fig. 5. Optimal control signal distribution time via early-stage analysis under different (α,β) configurations in mobile networks. $N = 1000$. $L = 1000$. $I_0 = 1/N$. $\delta = 2$. $\lambda = 0.25$. $\kappa = 0.2$. $T_f = 200$. $t' = 1$. $c = 10^{-3}$.

$$\begin{aligned} &= \frac{(NI_0)^\beta}{\lambda\eta S(t')\beta}\left(\exp\{\lambda\eta S(t')\beta T_D\} - 1\right) + \frac{(NI_0)^\beta}{[\lambda\eta S(t') - f(T_D)]\beta} \\ &\quad \times \Big(\exp\{[\lambda\eta S(t') - f(T_D)]\beta T_f\} \\ &\quad - \exp\{[\lambda\eta S(t') - f(T_D)]\beta T_D\}\Big) + \frac{1}{2}f^2(T_D)(T_f - T_D). \end{aligned}$$

The optimal control signal distribution time when adopting early-stage analysis can be obtained by $\widetilde{T}_D^* = \arg\min_{T_D} J$.

C. Performance Evaluation

To demonstrate the tradeoffs between control signal distribution and the resulting impacts on information dissemination, we set the function $f(T_D)$ in (1) to be $f(T_D) = \min\left\{1, c \cdot T_D^\alpha\right\}$, where α is a nonnegative value which accounts for the effectiveness of the control signal and c is a positive constant. The effect of control signal has a power-law growth with respect to the control signal distribution time. This power-law growth model is a general parametric model and it can be used to

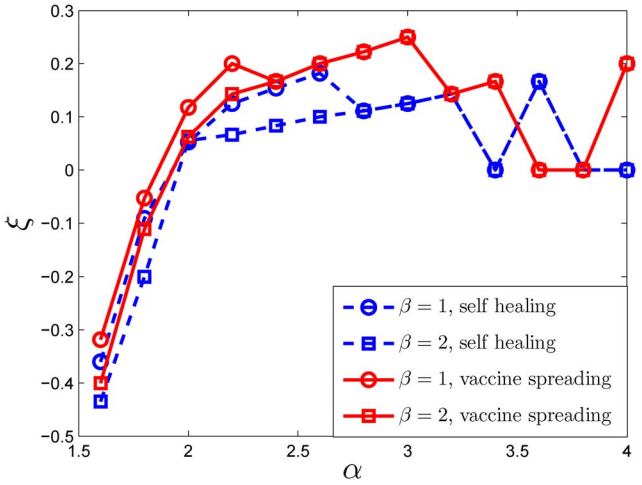

Fig. 6. Relative difference of optimal control signal distribution time under different (α,β) configurations in mobile networks. $N = 1000$. $L = 1000$. $I_0 = 1/N$. $\delta = 2$. $\lambda = 0.25$. $\kappa = 0.2$. $T_f = 200$. $M = 1000$. $t' = 1$. $c = 10^{-3}$.

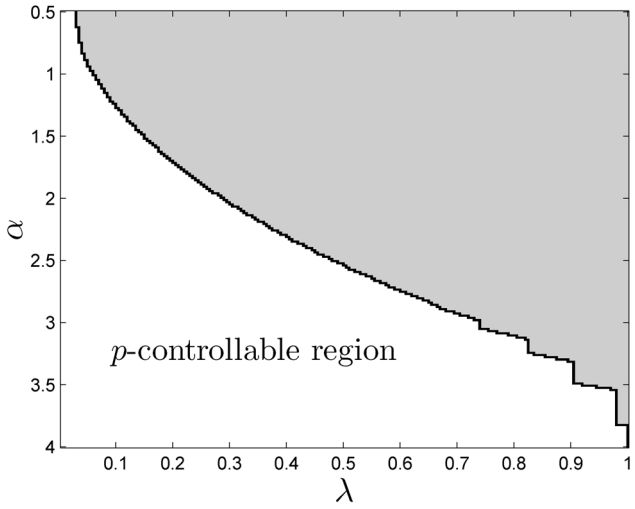

Fig. 7. Phase diagram of self healing scheme in mobile networks. $N = 1000$. $L = 1000$. $I_0 = 1/N$. $\delta = 2$. $\kappa = 0$. $p = 0.5$. $T_f = 200$. $M = 1000$. $c = 10^{-3}$.

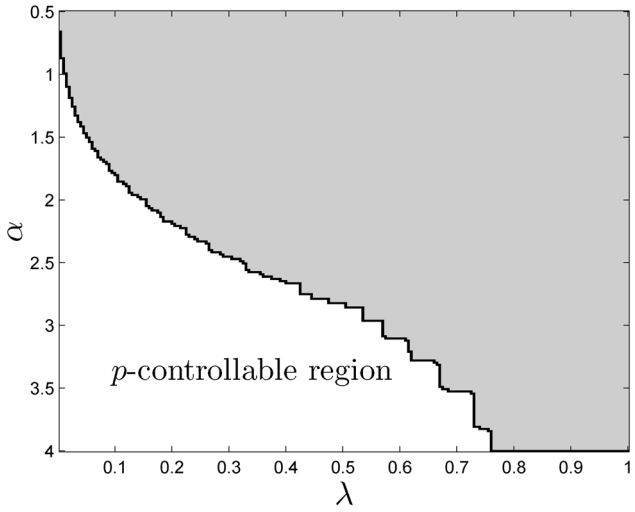

Fig. 8. Phase diagram of vaccine spreading scheme in mobile networks. $N = 1000$. $L = 1000$. $I_0 = 1/N$. $\delta = 2$. $\kappa = 0.2$. $p = 0.5$. $T_f = 200$. $M = 1000$. $c = 10^{-3}$.

investigate the tradeoffs between control capability and control signal distribution timeliness. The exponent α is associated with the effectiveness of the control capability. $\alpha = 0$ degenerates to the scenario that the control capability is irrelevant of its distribution time. For the simulation setup, N nodes are traversing in the square area in wrap-around condition via the Lèvy walk mobility model [56], where the step length and the pause time follow a power-law distribution with negative exponent, respectively. We set the length exponent $l = 1.5$ and the pause time exponent $\varphi = 1.38$, which fit the trace-based data of human mobility patterns collected in UCSD and Dartmouth [57]. Moreover, our previous research [5] has shown that the information dissemination dynamics in such mobile networks can be captured via epidemic model.

The infected population under self healing scheme is shown in Fig. 2. The differences between the infected population via optimal control function $u^*(t)$ and the simulations via optimal control signal distribution time T_D^* reside in the fact that in the simulation we take the effect of control signaling $f(T_D)$ into consideration, whereas $u^*(t)$ assumes full manipulation of the control signal and neglects the effect of $f(T_D)$. Therefore, information dissemination control via $u^*(t)$ is an unattainable lower bound of the dynamic programming approach. The solution of T_D^* via dynamic programming in (10) is shown to be efficacious to eradicate the epidemic since it minimizes the performance measure J which is proportional to the area of the infected population up to time T_f. Moreover, the solution $\widetilde{T}_D^*$ from early-stage analysis has a similar impact on the eradication of epidemic but with a slower decaying infected population compared with T_D^* due to early-stage approximation. In addition, the infected population under vaccine spreading can be further suppressed with the aid of vaccine spreading as shown in Fig. 3, where the growth of vaccinees effectively decelerates the spread of epidemic, even for small κ ($\kappa = 0.1$).

In Fig. 4, the optimal control signal distribution time decreases with the increase of α and β, since early distribution is intuitively more efficacious if the effectiveness of the signal (α) is stronger, or if we are facing a severe epidemic (larger β). Moreover, we observe that vaccine spreading also results in early distribution by launching the cooperative immunity mechanism to create more vaccinees against the epidemic. Similar results can be found for early-stage analysis as shown in Fig. 5. When α is small, the assumption that $S(t) \approx 1$ at early stages may not hold due to slight cure, leading to the pessimistic outcome of late distribution. When α is large, the overestimation of infection speed leads to the optimistic outcome of early distribution. We plot the relative difference $\xi = (T_D^* - \widetilde{T}_D^*)/T_D^*$ in Fig. 6. Compared with optimal control approaches, $\xi < 0$ refers to late distribution while $\xi > 0$ refers to early distribution for early-stage analysis.

The phase diagram of self healing and vaccine spreading schemes are shown in Figs. 7 and 8, respectively. The p-controllable region consists of the points where p fraction of nodes remain in the susceptible state when the epidemic comes

to a halt. For the self healing scheme, as the effectiveness of control signaling (α) decreases or the pairwise infection rate (λ) increases, the network is out of the p-controllable region since more than p fraction of nodes have been infected by the epidemic. For the vaccine spreading scheme, the p-controllable region shrinks due to the fact that not only the epidemic but also the spread of vaccines stimulate the state transitions of nodes so that less nodes can remain in the susceptible state.

V. Information Dissemination in Generalized Social Networks

In a generalized social network, information may disseminate through various access links, which includes localized links via short-range wireless communication technology such as WiFi or Bluetooth, or delocalized links via social relations, such as MMS [12], [37]. The interdependency of these two infection schemes tend to accelerate the infection speed and thereby increases the system loads. We assume that N nodes are uniformly distributed in a $L \times L$ square area with population density $\rho = \frac{N}{L^2}$ and identical transmission range δ. The average number of localized contacts is denoted by $\eta_\ell = \rho\pi\delta^2$. We further assume that every node randomly selects η_d nodes as its social contacts to form a generalized social network, which distinguishes from localized contacts. Note that the results are still valid if we treat η_d and η_ℓ as random variables and apply their means to our model. The dynamics of information dissemination and control signal distribution are illustrated in Fig. 9. Information is disseminated through localized and delocalized links. The control signal distribution resembles the information dissemination in the sense that it is distributed through these heterogeneous links to alleviate network cost. The overall normalized infected population is

$$I(t) = I_d(t) + I_\ell(t) \tag{24}$$

where $I_d(t)$ represents the normalized infected subpopulation via delocalized infection and $I_\ell(t)$ represents the normalized infected subpopulation via localized infection.

Following similar arguments in (13), the state equation of delocalized infection is

$$\dot{I}_d(t) = \lambda_d(\eta_d - 1)S(t)I(t) - u(t)I_d(t) \tag{25}$$

where λ_d is the pairwise infection rate on a delocalized link and $\eta_d - 1$ accounts for the fact that a node is infected implies that at least one of its neighbors is infected [5]. On the other hand, due to the interdependency of localized and delocalized infections, the localized infection stretches out from the infected source nodes generated by delocalized infections as shown in Fig. 9. The localized infection spreads out like a ripple centered at the infected source node and grows with time. For a single ripple with radius $r(t)$, $\rho\pi r^2(t) = N \cdot I_\ell(t)$ and the infected population in the peripheral circular strip of width δ are $\rho\pi r^2(t) - \rho\pi(r(t) - \delta)^2$. We have

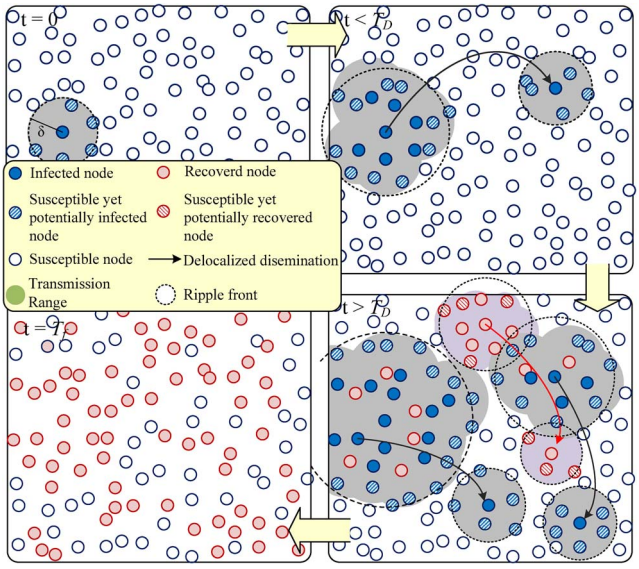

Fig. 9. Illustration of information dissemination and control signal distribution in generalized social networks. Localized and delocalized links are exploited for information dissemination. A node is infected at $t = 0$. T_D denotes the control signal distribution time and T_f denotes the time instance for the eradication of epidemic. Information is disseminated through localized and delocalized links. The control signal distribution resembles the information dissemination in the sense that it is distributed through these heterogeneous links to alleviate the information dissemination.

$$\begin{aligned}\Upsilon_{S\to I_\ell}(t) &= \frac{1}{N}\lambda_\ell\frac{1}{2}\eta_\ell S(t)\left[\rho\pi r^2(t) - \rho\pi(r(t)-\delta)^2\right] \\ &= \frac{1}{N}\lambda_\ell\frac{1}{2}\eta_\ell S(t)\left[2\rho\pi\delta r(t) - \rho\pi\delta^2\right] \\ &= \frac{1}{N}\lambda_\ell\eta_\ell S(t)\left[\delta\sqrt{\rho\pi N I_\ell(t)} - \frac{1}{2}\rho\pi\delta^2\right] \\ &\cong \frac{1}{N}\sigma\lambda_\ell\eta_\ell S(t)\sqrt{N I_\ell(t)}\end{aligned} \tag{26}$$

where $\sigma = \delta\sqrt{\rho\pi}$ and $\frac{1}{2}\rho\pi\delta^2$ is usually negligible compared with N [46]. $\frac{1}{2}\eta_\ell$ accounts for the average number of localized contacts which is located outside of the peripheral circular strip. Since delocalized infection creates multiple infected source nodes over time, we denote the incremental spatial infected population of a ripple which is generated at time z and keeps stretching for s time units by

$$\dot{W}(z,s) \triangleq \frac{dW(z,s)}{ds} = \sigma\lambda_\ell\eta_\ell S(z+s)\sqrt{W(z,s)} \tag{27}$$

where $W(z,0) = 1$. The state equation of the aggregated localized infection can be characterized as

$$\dot{I}_\ell(t) = \frac{1}{N}\int_0^t \dot{I}_d(\tau)\dot{W}(\tau, t-\tau)d\tau - u(t)I_\ell(t). \tag{28}$$

It means that $\dot{I}_d(t)d\tau$ infected source nodes are generated at time τ and each contributes to $\dot{W}(\tau, t-\tau)$ incremental spatial infection at time t. The overall state equation of $I(t)$ becomes

$$\begin{aligned}\dot{I}(t) &= \left[\lambda_d(\eta_d - 1)S(t) - u(t)\right]I(t) \\ &\quad + \frac{1}{N}\int_0^t \dot{I}_d(\tau)\dot{W}(\tau, t-\tau)d\tau.\end{aligned} \tag{29}$$

Similarly, the immunity scheme can also leverage the localized and delocalized links to eradicate the epidemic. The state equation of delocalized recovery is

$$\dot{R}_d(t) = u(t)I_d(t) + \phi(t)(\eta_d - 1)S(t)R(t). \tag{30}$$

The incremental spatial recovery process is characterized by

$$\dot{Q}(z, s) = \sigma\phi(t)\eta_\ell S(z + s)\sqrt{Q(z, s)} \tag{31}$$

with $Q(z, 0) = 1$. The state equation of localized recovery is

$$\dot{R}_\ell(t) = \frac{1}{N}\int_0^t \dot{R}_d(\tau)\dot{Q}(\tau, t - \tau)d\tau + u(t)I_\ell(t). \tag{32}$$

The overall state equation of $R(t)$ becomes

$$\begin{aligned}\dot{R}(t) = u(t)I(t) + \frac{1}{N}\int_0^t \dot{R}_d(\tau)\dot{Q}(\tau, t - \tau)d\tau \\ + \phi(t)(\eta_d - 1)S(t)R(t).\end{aligned} \tag{33}$$

Consistent with (6), regarding generalized social networks, we are still interested in minimizing the accumulated cost subject to the state equations (29), (33), and the population constraint $S(t) + I(t) + R(t) = 1$.

A. Optimal Control

With the state equations, the corresponding Hamiltonian is obtain be plugging the parameters (1), (5), (6), (29), and (33) into (7)

$$\begin{aligned}\mathcal{H} = [NI(t)]^\beta + \frac{1}{2}u^2(t) + \Lambda_I(t)\Bigg[\lambda_d(\eta_d - 1)S(t)I(t) \\ + \frac{1}{N}\int_0^t \dot{I}_d(\tau)\dot{W}(\tau, t - \tau)d\tau - u(t)I(t)\Bigg] \\ + \Lambda_R(t)\Bigg[u(t)I(t) + \frac{1}{N}\int_0^t \dot{R}_d(\tau)\dot{Q}(\tau, t - \tau)d\tau \\ + \phi(t)(\eta_d - 1)S(t)R(t)\Bigg]\end{aligned} \tag{34}$$

from which the costate equations are $\dot{\Lambda}_I(t) = -\frac{\partial\mathcal{H}}{\partial I}$ and $\dot{\Lambda}_R(t) = -\frac{\partial\mathcal{H}}{\partial R}$. With the switching function $\theta^*(t) = \left[\Lambda_I^*(t) - \Lambda_R^*(t)\right]I^*(t)$, the constrained optimal control function $u^*(t)$ that minimizes J is the saturation function

$$u^*(t) = \begin{cases} 0, & \theta^*(t) \le 0 \\ \theta^*(t), & \theta^*(t) \in (0, 1) \\ 1, & \theta^*(t) \ge 1. \end{cases} \tag{35}$$

Consider the time-dependent control capability, the optimal control signal distribution time T_D^* can be obtained by solving the dynamic programming in (10). Similarly, the saturation function in (35) only provides an attainable lower bound on information dissemination control with time-dependant control capability.

B. Early-stage Analysis

With the approximation that $S(t) \approx 1$ at early stages and the initial condition $W(z, 0) = 1$, from (27) we have the approximation of incremental spatial infection

$$W(z, s) = \left(\frac{\sigma\lambda_\ell\eta_\ell}{2}s + 1\right)^2. \tag{36}$$

Moreover, we also have the approximation that $I(t) \approx I_d(t)$ since at early stages $I_d(t) \propto I(t)$ while $I_\ell(t) \propto \sqrt{I(t)}$. That is, the information disseminates at a faster speed through delocalized links than localized links [12], [37]. At some early stage t', $S(t') = 1 - I_0 - I_0u(t)\left[t' + \frac{\phi(t)(\eta_d - 1)}{2}t'^2\right]$, and we have the first-order ODE

$$\begin{aligned}\dot{I}(t) = \left[\lambda_d(\eta_d - 1)S(t') - u(t)\right]I(t) \\ + \frac{1}{N}\int_0^t \dot{I}(\tau)\dot{W}(\tau, t - \tau)d\tau.\end{aligned} \tag{37}$$

Using the subgradient of $u(t)$ at $t = T_D$ to define the subderivative $\dot{u}(T_D) = 0$ and differentiating (37) with respect to t at both sides, we have the second-order ODE (neglecting the second-order term of $W(z, s)$)

$$\begin{aligned}\ddot{I}(t) &= [\lambda_d(\eta_d - 1)S(t') + \sigma\lambda_\ell\eta_\ell N^{-1} - u(t)]\dot{I}(t) \\ &\triangleq [K_1 - K_2\phi(t) - u(t)]\dot{I}(t)\end{aligned} \tag{38}$$

where $K_1 = \lambda_d(\eta_d - 1)[1 - I_0 - I_0u(t)t'] + \sigma\lambda_\ell\eta_\ell N^{-1}$ and $K_2 = I_0u(t)\frac{\eta_d - 1}{2}t'^2$. With the initial values $I(0) = I_0$ and $\dot{I}(0) = \lambda_d(\eta_d - 1)(1 - I_0)I_0 \triangleq K_3$, we obtain

$$\begin{aligned}I(t) &= \frac{K_3}{K_1 - K_2\phi(t) - u(t)}\exp\{[K_1 - K_2\phi(t) - u(t)]t\} \\ &\quad + I_0 - \frac{K_3}{K_1 - K_2\phi(t) - u(t)} \\ &= \begin{cases} \frac{K_3}{K_1}\exp\{K_1t\} + I_0 - \frac{K_3}{K_1}, & t < T_D, \\ \frac{K_3}{K_1 - K_2\kappa - f(T_D)}\exp\{[K_1 - K_2\kappa - f(T_D)]t\} \\ \quad + I_0 - \frac{K_3}{K_1 - K_2\kappa - f(T_D)}, & t \ge T_D. \end{cases}\end{aligned} \tag{39}$$

The performance measure J in (6) can be evaluated as

$$\begin{aligned}J &= \int_0^{T_D}[NI(t)]^\beta dt + \int_{T_D}^{T_f}[NI(t)]^\beta + \frac{1}{2}f^2(T_D)\,dt \\ &= \frac{\left(\frac{NK_3}{K_1}\right)^\beta}{K_1\beta}(\exp\{K_1\beta T_D\} - 1) + \left(I_0 - \frac{K_3}{K_1}\right)T_D \\ &\quad + \frac{\left(\frac{NK_3}{K_1 - K_2\kappa - f(T_D)}\right)^\beta}{[K_1 - K_2\kappa - f(T_D)]\beta} \times \Big(\exp\{[K_1 - K_2\kappa - f(T_D)]\beta T_f\} \\ &\quad - \exp\{[K_1 - K_2\kappa - f(T_D)]\beta T_D\}\Big) \\ &\quad + \left(I_0 - \frac{K_3}{K_1 - K_2\kappa - f(T_D)} + \frac{1}{2}f^2(T_D)\right)(T_f - T_D).\end{aligned} \tag{40}$$

For early-stage analysis, the optimal control signal distribution time can be obtained by $\widetilde{T_D^*} = \arg\min_{T_D} J$.

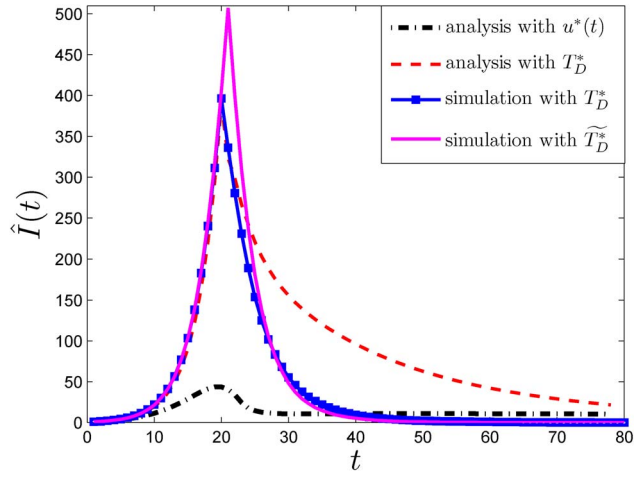

Fig. 10. Infected population under self healing scheme in generalized social networks. $N = 2000$, $L = 50$, $I_0 = 1/N$, $\delta = 1.1$, $\lambda_d = \lambda_\ell = 0.05$, $\eta_d = 6$, $\eta_\ell = 3$, $\alpha = 2$, $\beta = 1$, $\kappa = 0$, $T_f = 200$, $M = 1000$, $\Lambda_I(0) = 200$, $\Lambda_R(0) = 100$, $t' = 1$, and $c = 10^{-3}$ over 300 simulations.

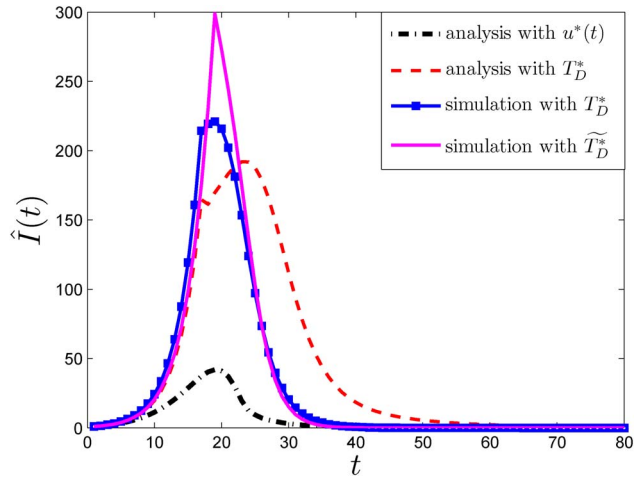

Fig. 11. Infected population under vaccine spreading scheme in generalized social networks. $N = 2000$, $L = 50$, $I_0 = 1/N$, $\delta = 1.1$, $\lambda_d = \lambda_\ell = 0.05$, $\eta_d = 6$, $\eta_\ell = 3$, $\alpha = 2$, $\beta = 1$, $\kappa = 0.1$, $T_f = 200$, $M = 1000$, $\Lambda_I(0) = 200$, $\Lambda_R(0) = 100$, $t' = 1$, and $c = 10^{-3}$ over 300 simulations.

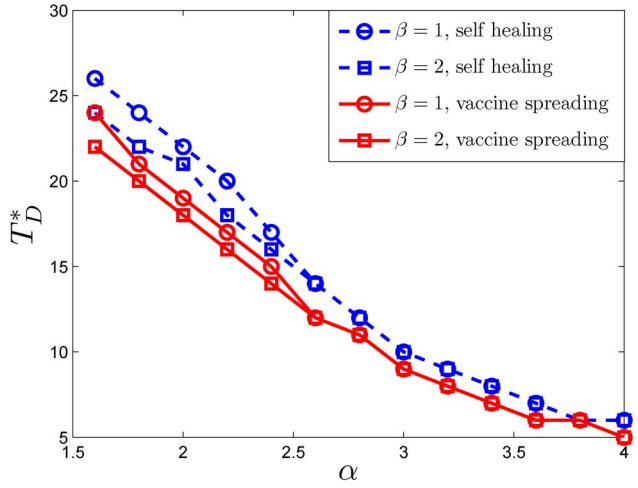

Fig. 12. Optimal control signal distribution time via dynamic programming under different (α,β) configurations in generalized social networks. $N = 2000$. $L = 50$. $I_0 = 1/N$. $\delta = 1.1$. $\lambda_d = \lambda_\ell = 0.05$. $\eta_d = 6$. $\eta_\ell = 3$. $\kappa = 0.1$. $T_f = 200$. $M = 1000$. $t' = 1$. $c = 10^{-3}$.

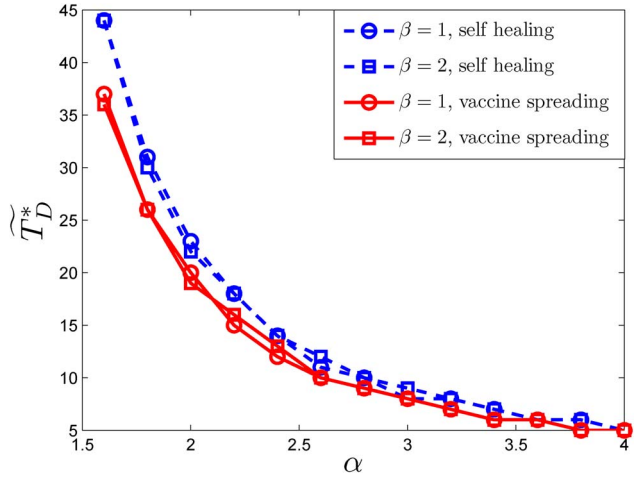

Fig. 13. Optimal control signal distribution time via early-stage analysis under different (α,β) configurations in generalized social networks. $N = 2000$. $L = 50$. $I_0 = 1/N$. $\delta = 1.1$. $\lambda_d = \lambda_\ell = 0.05$. $\eta_d = 6$. $\eta_\ell = 3$. $\kappa = 0.1$. $T_f = 200$. $t' = 1$. $c = 10^{-3}$.

C. Performance Evaluation

Consistent with the performance evaluation of mobile networks in Section IV-C, the control capability is set to be $f(T_D) = \min\{1, c \cdot T_D^\alpha\}$. For the simulation setup, 2000 nodes are placed uniformly in a square area with density $\rho = 0.8$. Following the data sheet in [37], we set the average number of delocalized contacts to be $\eta_d = 6$, and we also set the average number of localized contacts to be $\eta_\ell = 3$ ($\delta \approx 1.1$) since in general the local transmission range is limited.

The infected population under the self healing scheme is shown in Fig. 10. Prior to the control signal distribution, our SIR model captures the simulation results of information dissemination in generalized social networks. Although both delocalized and localized pairwise infection rates are quite low ($\lambda_d = \lambda_\ell = 0.05$), the infection spreads out drastically since the information dissemination benefits from these heterogeneous links. After control signal distribution, the analytical infected population decreases at a slower speed compared with the simulation results due to the fact that recovery actually disrupts the stretch of local infection, and the ripples are likely to coincide with other ripples as time evolves, which leads to overestimation of information dissemination. In addition, early-stage analysis suggests early distribution and hence its infection has a slow decaying curve. The infection curve via optimal control theory also implies that we can have better control of the information dissemination if we can have full manipulation of the control capability.

Similar results can be found in Fig. 11 for information dissemination under the vaccine spreading scheme. With the help of vaccine spreading through delocalized and localized links, we can further mitigate the infection compared with that of the self healing scheme. Since susceptible nodes are likely to become vaccinees under the vaccine spreading scheme, the immune nodes may hinder the growth of the local infection

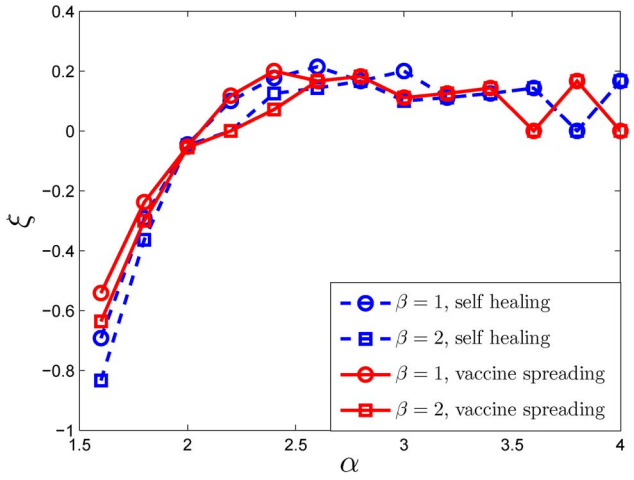

Fig. 14. Relative difference of optimal control signal distribution time under different (α,β) configurations in generalized social networks. $N = 2000$. $L = 50$. $I_0 = 1/N$. $\delta = 1.1$. $\lambda_d = \lambda_\ell = 0.05$. $\eta_d = 6$. $\eta_\ell = 3$. $\kappa = 0.1$. $T_f = 200$. $M = 1000$. $t' = 1$. $c = 10^{-3}$.

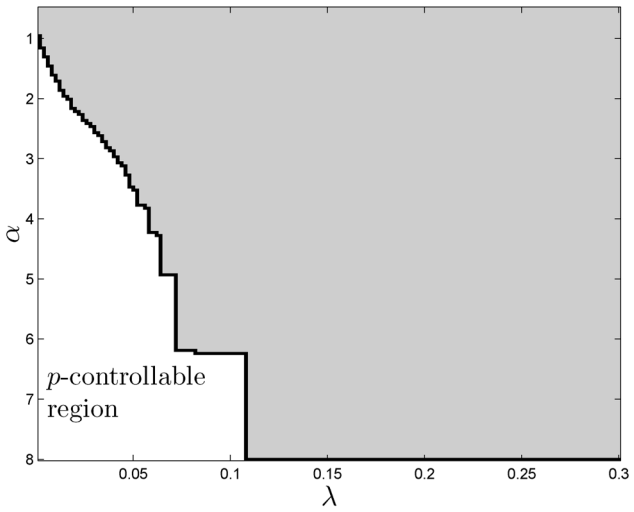

Fig. 15. Phase diagram of self healing scheme in generalize social networks. $N = 2000$. $L = 50$. $I_0 = 1/N$. $\delta = 1.1$. $\lambda_d = \lambda_\ell = \lambda$. $\eta_d = 6$. $\eta_\ell = 3$. $\kappa = 0$. $p = 0.5$. $T_f = 200$. $c = 10^{-3}$.

ripple and thereby decelerate the infection, which again leads to overestimation of the SIR model after the control signal distribution. In Figs. 10 and 11, $u^*(t)$ from optimal control theory elucidates the discrepancy of taking the time-dependent control capability $f(T_D)$ into consideration. Time-dependent control capability inevitably incur more network cost compared with that of optimal control function.

When dynamic programming is applied to determine the optimal distribution time, severe epidemics (large β) contribute to early distribution to minimize the accumulated cost as shown in Fig. 12. Moreover, both optimal control and early-stage analysis suggest early distribution as the effectiveness of signal (α) increases as shown in Fig. 13. The relative difference of these two approaches are plotted in Fig. 14. Comparing with the early-stage analysis, optimal control via dynamic programming prefers early distribution when α is small, while it prefers late distribution as α increases, which is consistent with the case of information dissemination in mobile networks.

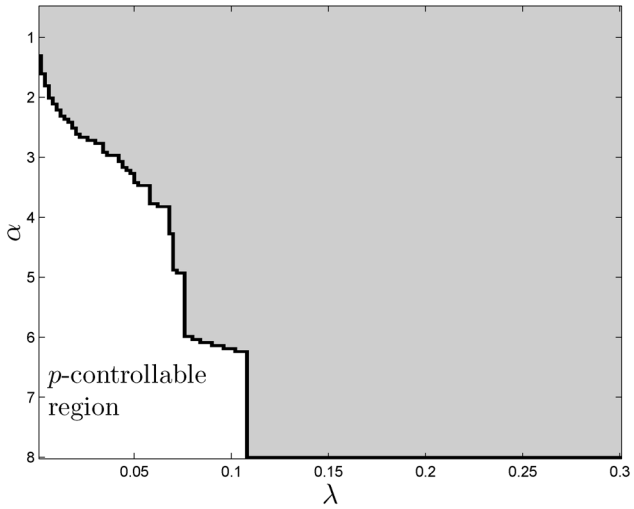

Fig. 16. Phase diagram of vaccine spreading scheme in generalize social networks. $N = 2000$. $L = 50$. $I_0 = 1/N$. $\delta = 1.1$. $\lambda_d = \lambda_\ell = \lambda$. $\eta_d = 6$. $\eta_\ell = 3$. $\kappa = 0.8$. $p = 0.5$. $T_f = 200$. $c = 10^{-3}$.

The phase diagram of the self healing and vaccine spreading schemes are shown in Figs. 15 and 16, respectively. Compared with the phase diagram in mobile networks, the p-controllable region in generalized social networks covers a smaller area, indicating that the heterogeneous links indeed strengthen the spread of epidemics. Since control signals can also traverse through the heterogeneous links, the vaccine spreading scheme contributes to worse controllability than the self healing scheme when α is small while it may improve the controllability when α is large, suggesting that the vaccine spreading scheme can achieve better control of information dissemination in networks with heterogeneous links provided that the initial control capability is high.

VI. Conclusion

The contributions of this paper are twofold. First, with the aid of epidemic modeling, we provide an analytically tractable parametric plug-in model for information dissemination control regarding the time-dependent control capability, with an aim to determine the optimal control signal distribution time to minimize the accumulated network cost in real time via dynamic programming. Second, we demonstrate how to use our developed tools to control information dissemination in mobile and generalized social networks. Optimal control signal distribution time is solved via dynamic programming with respect to the associated information dissemination process and time-dependent control capability, and the results show that the time-dependent control capability inevitably incurs additional network cost relative to optimal control function. Compared with the self healing scheme, we show that the vaccine spreading further mitigates the accumulated cost when the immune nodes participate in forwarding control signal. The controllability of a network is characterized by the phase diagram, where severe infection rate and proactive vaccine spreading scheme tend to devor the population of susceptible nodes, contributing to the shrinkage of controllable region.

Consequently, this paper provides novel mathematical tools for information dissemination control over networks, and this framework can be applied to many applications in computer network, communication systems, and social networks.

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