Optimal Control of Epidemic Evolution

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***Abstract*—Epidemic models based on nonlinear differential equations have been extensively applied in a variety of systems as diverse as infectious outbreaks, marketing, diffusion of beliefs, etc., to the dissemination of messages in MANET or p2p networks. Control of such systems is achieved at the cost of consuming the resources. We construct a unifying framework that models the interactions of the control and the elements in systems with epidemic behavior. Specifically, we consider *non-replicative* and *replicative* dissemination of messages in a network: a pre-determined set of disseminators distribute the messages in the former, whereas the disseminator set continually grows in the latter as the nodes that receive the patch become disseminators themselves. In both cases, the desired trade-offs can be attained by activating at any given time only fractions of disseminators and selecting their dissemination rates. We formulate the above trade-offs as optimal control problems that seek to minimize a general aggregate cost function which cogently depends on both the states and the overall resource consumption. We prove that the dynamic control strategies have simple structures: (1) it is never optimal to activate a partial fraction of the disseminators (all or none) (2) when the resource consumption cost is concave, the distribution rate of the activated nodes are bang-bang with at most one jump from the maximum to the minimum value. When the resource consumption cost is convex, the above transition is strict but continuous. We compare the efficacy and robustness of different dispatch models and also those of the optimum dynamic and static controls using numerical computations.**

I. Introduction

a) Motivation: Epidemic behavior emerges whenever interactions among a large number of individual entities affect the overall evolution of the encompassing system. Mathematical models based on nonlinear differential equations have been developed and applied in a variety of systems as diverse as infectious outbreaks [1] and information diffusion in a human society [2], to the dissemination of messages in MANET [3] or p2p networks [4]. What a resource manager of such systems is interested in is to control the evolutions of the states. Most often, exertion of a control incurs a cost, either directly as the control may consume restricted resources, or indirectly as it may introduce adverse side effects. Much work has been done in modeling and validating the epidemic models, relatively less, however, is known about optimal control of such systems. This constitutes the focus of this paper.

Dynamic optimal control is of paramount importance in the networking context. One important example is in countering the spread of a malware in a MANET, a wireline p2p, or a client-server network. Worms spread from *infective* nodes to vulnerable but not yet infected, i.e. *susceptible* nodes, when such a pair communicates, or as we will refer to, when they contact. Hence, spread of malware behaves as per an epidemic. Note that a contact may entail physical proximity, as in the case of MANETs, or may represent an opportunity of infiltration, as in the case of server-client networks. Worms, as malicious self-replicating codes, can disrupt the normal functionalities of the hosts, steal their private information, and/or use them to eavesdrop on other nodes. The worm can also render the host dysfunctional, e.g. by deliberately draining its battery as in the case of Cabir worm [5] in a cellular network, or by executing a pernicious code that incurs irretrievable critical hardware or software damage say by re-fleshing the BIOS corrupting the bootstrap program required to initialize the OS [6]. Such dysfunctional nodes are referred to as *dead*. Software patches can *immunize* susceptible nodes against future attacks, by rectifying their underlying vulnerability, or *heal* the infectives and render them robust against future attacks. Nodes which have been immunized or healed are denoted as *recovered*. Such patches can be distributed by mobile agents and/or downloaded from designated servers, but patch distribution consumes both energy and bandwidth (critical in wireless networks), and thereby incurs a cost that depends on both the number of active agents/servers and the transmission rates they use. The incident of Welchia [7], which was designed as a counter-worm to defeat Blaster, demonstrated how unrestrained spread of security patches can indeed create substantial network traffic and rapidly destabilize even the well-provisioned network of Internet. This adverse effect of application of countermeasures is likely to be aggravated in wireless networks, where due to inherent properties such as interference, intermittent links, limited battery, *etc.*, the resource limitations are more stringent.

The security patches can be distributed in a *non-replicative* or *replicative* manner (fig.1). In the former, a number of (mobile or stationary) agents, referred to as *disseminators*, are pre-loaded with the patch, and other nodes receive it from them. In the replicative model, the receptors, i.e., the recovered nodes, in addition, become disseminators of the security patch themselves - hence the disseminators *replicate.* The replicative method immunizes nodes more rapidly, as it has a growing number of disseminators, but at the expense of consuming increasingly larger amounts of limited underlying resources. Thus, the choice between the two, and the differences in their controls are not a priori clear. The overall system cost depends on (i) the number of infectives and dead nodes, and (ii) the resource consumption in distribution of countermeasures. In both scenarios, dynamic optimal control of the fraction of activated disseminators and the distribution rates of activated nodes can minimize the overall cost and thereby attain desired trade-offs between network security and resource consumption.

A special case of the epidemic evolution in fig.1 also

The contributions of MHR. Khouzani and S. Sarkar are supported through grants NSF-CNS-0914955, NSF-CNS-0915203, NSF-CNS-0915697.

captures propagation of messages in Delay Tolerant Networks (DTNs). A server may seek to broadcast a message to as many nodes as possible, before a deadline, by employing minimal resources such as energy and bandwidth. In this case, susceptibles are the nodes that are yet to receive the message, and the recovered are the ones which have received it. Dissemination of the message may either be performed in non-replicative or replicative manner. Infectives and dead nodes are absent in this problem. The overall 'cost' is (i) decreasing in the number of recovered (i.e., recipient) nodes, and is (ii) increasing in the transmission rates of the activated disseminators. Again, dynamic optimal control can be utilized to resolve a problem of practical importance in the context of networking.

The epidemiological evolution has natural analogues in the spread of a contagious disease in a human society, with the caveat that the inoculation and healing processes are non-replicative. The cost is aggregation of infective and dead individuals and the overall human-hour of trained staff [8]. Application of the optimal control of epidemiological evolution in social context is, however, not restricted to the containment of contagious diseases. Another noteworthy problem is dynamic management of advertising resources in adoption of a new technology. We discuss two practical examples which we refer to as *Reclamation* and *Rivalry* cases, respectively. First, consider a simple scenario where (at least initially) most individuals in a society are subscribed to a specific technology through incumbent company A (e.g., Comcast for cable TV in Philadelphia) - they are the susceptibles. A new technology/company B (e.g., DTV) aims to capture the market. They win over some customers, who constitute the converts (infectives). Social exchanges (contacts) between infectives and susceptibles (converts and subscribers) may convert the latter. Company A seeks to regain the share of the market, by recapturing (healing) the infectives and re-confirming (immunizing) the susceptibles, say via offering lucrative long-term contracts (patches) - the resulting pledged subscribers constitute the recovered. New contracts are long-term and thus the recovered are immune to further change in subscription. The reclamation occurs through the efforts of advertising agents (disseminators) who communicate to the infectives and susceptibles through tele-marketing, e-marketing and/or word of mouth. The disseminators may either be from an initial pool (non-replicative dispatch), or may include the recovered nodes as well, e.g. by offering pledged subscribers additional service discounts through referral rewards, *etc.* (replicative dispatch). There is however no "death" in this setting. The overall 'cost' for company A is (i) increasing in the number of infectives, as they are the only lost subscribers to A, and is (ii) increasing in the number of active agents and the amount of discounts they offer in order to make the contracts appealing. Thus, optimal (dynamic) control of activating agents and selecting discount rates maximizes the net profit for company A, where profit equals the income generated through subscription minus the cost incurred in marketing/advertising over time.

For the second case (Rivalry), suppose that both competing companies enter the market for a new technology at around the same time. Now, susceptibles are those who are yet to choose either, infectives encompass those who have chosen B (the rival), and recovered are those who have chosen A. Both infectives and recovered may convert susceptibles (the undecided) to their respective groups whenever the respective pairs contact, e.g., through social communications - the dispatch is therefore replicative. It is also possible that some infectives can not be healed as both companies may offer long-term contracts. The overall cost for company A is similar, except that it is now decreasing (hence the revenue is increasing) in the number of recovered, as only recovered are subscribed to company A in this case.

b) Contributions and Road-map: First, we formulate the minimization of the aggregate cost associated with epidemic state evolution as an optimal control problem. The cost represents a trade-off between desirability/harmfulness of the state and the cost of consuming resources in order to manage the state. We demonstrate the extent of generality of our model through different examples. We consider both replicative and non-replicative dispatch scenarios and minimize the overall costs by dynamically selecting the activation of the disseminators and their distribution rates. We develop a framework for solving this non-linear optimal control problem using Pontryagin's Maximum Principle [9], [10].

Next, in both non-replicative and replicative settings, we *prove* that the optimal policies have the following simple structure: when the resource consumption cost is concave, until a certain time, all disseminators are activated and they distribute patches at the maximum possible rate, and subsequently no disseminator is activated until the end of the system operation period (§§III-A and IV-A). Optimality of a bang-bang control (that is the property that it assumes only either its minimum or maximum possible values at any given time) and quantifying the maximum possible number of jumps to be *one* are despite the facts that the network state evolutions do not constitute monotonic functions of time, involve non-linear dynamics, the cost functions are not assumed to be linear and the control (activation fraction, transmission rate) is a two-dimensional function. When the resource consumption cost is convex, the optimal activation fraction function has the same structure. The optimal transmission rate function has similar behavior except that its potential transition from the maximum to minimum values is strict, but continuous rather than abrupt. The generality of the model allows for a unified theoretical framework for optimizing a sundry of problems of practical importance in networking. Moreover, the simplicity of the structure of the optimal controls makes them amenable to implementation in practice.

Finally, using numerical evaluation, we assess the relative efficacy of the replicative and non-replicative dispatch and static and dynamic optimal controls (§V). We demonstrate that in general, optimal dynamic controls incur significantly lower aggregate costs than optimum static controls in both replicative and non-replicative settings. Also, in presence of dynamic optimal control, replicative dispatch of security patches incurs substantially lower aggregate costs than non-replicative dispatch.

c) Related Literature: Optimal control has been extensively used to find the best deployment of resources in treating infectious epidemics [11], [12], advertising and marketing [10], [13], [14] and recently in securing communication networks against malware outbreaks [15]–[17]. An extensive

overview of the existing work is beyond the scope of this article. In what follows, we mention and differentiate from some of the most related works.

Optimal control in treatment of infectious epidemics is mostly applied to systems where only vaccination or healing/quarantining is present, the cost is linear in the treatment rate and there is no mortality among infectives [11], [12]. In contrast, our system integrates both vaccination and healing/quarantining, the cost of treatment is any general concave or convex function, and it depends on both infective and the deceased as well. Moreover, there is no equivalent of replicative immunity in the case of infectious diseases.

Also, our work generalizes the existing treatment of models is advertising and marketing [10], [13], [14] which mostly consider only either public advertisement or word-of-mouth advertisement with linear benefits, and optimizations are mostly with respect to the steady state behaviour of the market, rather than the transitional patterns, which is the salient feature of the diffusion of new technologies.

In the context of security in communication networks, [17] investigates a different counter-measure: that of reduction of reception gain of wireless nodes for slowing down the spread of malware in wireless networks. Our work differs from [16] in that we consider (i) both replicative and non-replicative patching, (ii) more general network state evolution dynamics in that the counter-measure involves both immunization and healing, moreover the worm may cause mortality, and (iii) cost functions which are only assumed to be either concave or convex and therefore more general than quadratic functions in [16]. Also unlike [16] we do not use any linearization of the system which can be very poor in the context of epidemic behaviour. Investigation of optimal solutions in our context thus require different analytical arguments. [15] considers only a one-dimensional control of bandwidth. That model is thus not suitable for capturing the cost related to the total consumed energy, which is more critical than bandwidth in DTN networks. Moreover, the cost function does not include the benefit of recovered, which is essential for application in marketing or DTN settings.

Optimal forwarding of packets emanating from a single source in a delay tolerant energy-constrained wireless network is studied in [18], [19] and it is shown that optimal strategy follows a threshold-based structure. [18], [19] analytically rely on some simplifying assumptions that will make them as special cases in our context. Specifically, [18] considers only networks that use two-hop routing, and therefore, the resulting dynamics of the number of *recovered* (i.e. nodes that have received the packet) follows our non-replicative model with no infective or dead. Also, [19] investigates a *monotonic* epidemic model, which arises when none of the nodes that have received a desired packet lose it, which is mapped to a special case of our replicative case with no infective or dead. Our model, unlike those two works, considers a general cost function that involves a general reward for number of recipient nodes and any (concave-linear-or convex) power function, and is therefore, a generalization of works in [18], [19].

II. System model

We first present the state evolution and formulate the cost minimization goal as an optimal control problem at an abstract level. In particular, we use terms such as *infectives, susceptibles, recovered, dead* and *disseminating, immunization and healing*. Later, in §II-C, we motivate the model by instantiating each of these terms in the different settings discussed in the introduction (§I-A).

A. Dynamics of Non-Replicative Dispatch

A system consists of N entities, and at time t, a number of $n_S(t), n_I(t), n_R(t)$ and $n_D(t)$ of them are respectively in **infective**, **susceptible**, **recovered** and **dead** state. Let the corresponding fractions be $S(t) = n_S(t)/N$, $I(t) = n_I(t)/N$, $R(t) = n_R(t)/N$, and $D(t) = n_D(t)/N$. Thus, for all t, $S(t)+I(t)+R(t)+D(t) = 1$.A pre-determined set of entities, referred to as disseminators are pre-loaded with the patches that immunize and/or heal. These disseminators constitute an R_0 fraction of the the total population N, that is, their number is NR_0 where $0 < R_0 < 1$. We assume that the disseminators can not be infected and hence they are recovered right from the beginning. At time $t = 0$, let $0 \leq S(0) < 1$, $0 \leq I(0) = I_0 \leq 1$, $0 < R(0) = R_0 \leq 1$, $D(0) = 0$. Thus, $S(0) = 1 - I_0 - R_0$. When infectives do not exist, $I_0 = 0$. No entity is aware of the state of other entities, except that they know who the disseminators are.

A susceptible is infected whenever it is in contact with an infective. We assume homogeneous mixing, that is, an infective is equally likely to contact with any other entity and with the same inter-meeting delay distribution. Hence an infective meets with each susceptible at the same rate, say $\hat{\beta}$. We later partially relax this assumption using simulations (§V). At any given t, there are $n_S(t)n_I(t)$ infective-susceptible potential pairs. Susceptibles are therefore transformed to infectives at rate $\hat{\beta}n_S(t)n_I(t)$.

The system manager controls the resources consumed in distribution of the *patches* by dynamically activating a fraction of the disseminators, as well as determining the distribution rates of the activated disseminators. Let the fraction of activated disseminators at time t be $\varepsilon(t)$, and each transmits at rate $u(t)$. The disseminators distribute their patches to infectives and susceptibles upon contact, which has similar connotations as for the spread of infection. The patches immunize the susceptibles, and thus susceptibles recover at rate $\tilde{\beta}\varepsilon(t)NR_0n_S(t)u(t)$ at each t. Clearly,

$$0 \leq \varepsilon(t) \leq 1,\ 0 \leq u(t) \leq 1 \ \text{ at each } t. \tag{1}$$

The last upper bound follows by normalization of $\tilde{\beta}$.

The efficacy of the patch may be lower for infectives than for susceptibles. We capture the above possibility by introducing a coefficient $0 \leq \pi \leq 1$: $\pi = 0$ occurs when the patch is completely unable to heal the infectives and only immunizes the susceptibles, whereas $\pi = 1$ represents the other extreme scenario where a patch can equally well immunize and heal susceptibles and infectives. If the patch heals an infective, its state changes to recovered, otherwise, it continues to remain an infective. Thus, the infectives recover at rate $\pi\tilde{\beta}NR_0\varepsilon(t)u(t)n_I(t)$ at each t.

Each infective *dies* at rate δ, where $\delta \geq 0$, and the overall death rate is $\delta n_I(t)$ at each t. Note that $\delta = 0$ corresponds to systems without death. Let $\beta_0 := \lim_{N\to\infty} N\hat{\beta}$ and $\beta_1 :=$

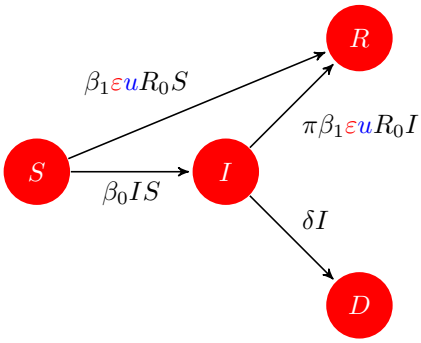


Fig. 1: State transitions for non-replicative case. The only difference in the replicative case is that transition rates from S to R is at rate $\beta_1\varepsilon u R S$ and from I to R at rate $\pi\beta_1\varepsilon u R I$ instead.

$\lim_{N\to\infty} N\tilde{\beta}$ are limits of the respective R.H.S.[1] If the total number of entities (N) is large, then [21, p.1]), $S(t)$, $I(t)$ and $D(t)$ converge to the solution of the following system of differential equations:[2]

$$\dot{S}(t) = -\beta_0 I(t)S(t) - \beta_1 R_0 \varepsilon(t)u(t)S(t) \quad (2a)$$

$$\dot{I}(t) = \beta_0 I(t)S(t) - \pi\beta_1 R_0 \varepsilon(t)u(t)I(t) - \delta I(t) \quad (2b)$$

$$\dot{D}(t) = \delta I(t) \quad (2c)$$

with initial constraints: $I(0) = \lim_{N\to\infty} n_I(0)/N = I_0$, $0 < S(0) < 1 - I_0$, $D(0) = 0$, and which also satisfy the following constraints at all t: $0 \le S(t), I(t), D(t)$ and $S(t) + I(t) + D(t) \le 1$. Thus, $(S(.), I(.), D(.))$ constitutes the system state function and $(\varepsilon(.), u(.))$ constitutes the (2-dimensional) control function.[3] Henceforth, wherever not ambiguous, we drop the dependence on t and make it implicit. Fig.1 illustrates the transitions between different states of nodes and the notations used.

B. Dynamics of Replicative Dispatch

In the replicative model, all recovered nodes become disseminators, and hence the fraction of disseminators grows to $R(t)$ at time t, whereas in the non-replicative model, the fraction of disseminators continue to be R_0 at all times. The dynamics in (2) hence needs to be modified. First, since $S(t) + I(t) + R(t) + D(t) = 1$ at any given time, we can represent the system using any three of the above states. In the non-replicative case we chose $(S(t), I(t), D(t))$, whereas in the replicative case we adopt $(S(t), I(t), R(t))$ instead. The specific choices make the analyses more convenient in each case.

$$\dot{S}(t) = -\beta_0 I(t)S(t) - \beta_1 \varepsilon(t)u(t)R(t)S(t) \quad (3a)$$

$$\dot{I}(t) = \beta_0 I(t)S(t) - \pi\beta_1 \varepsilon(t)u(t)R(t)I(t) - \delta I(t) \quad (3b)$$

$$\dot{R}(t) = \beta_1 \varepsilon(t)u(t)R(t)S(t) + \pi\beta_1 \varepsilon(t)u(t)R(t)I(t) \quad (3c)$$

[1]It can be shown that these limits exist as long as the node density $\lim_{N\to\infty} N/A$ exists for large N [20].

[2]Throughout the paper, variables with dot marks (e.g., $\dot{S}(t)$) will represent their time derivatives (e.g., time derivative of $S(t)$).

[3]Formally, if we further assume the inter-meeting times are exponentially distributed, then from mean field approximations [21, p.1], $\forall\ \epsilon > 0\ \forall\ t > 0$, $\lim_{N\to\infty} \Pr\{\sup_{\tau\le t} |\frac{n_S(\tau)}{N} - S(\tau)| > \epsilon\} = 0$, and likewise for $I(t)$ and $D(t)$.

with initial constraints: $I(0) = I_0$, $R(0) = R_0$, $S(0) = 1 - I_0 - R_0$, and as before, $0 \le I_0 \le 1, 0 < R_0 < 1$, $I_0 + R_0 < 1$. Also similarly, $0 \le S(t), I(t), R(t)$ and $S(t)+I(t)+R(t) \le 1$.

If $\delta = 0$, the latter holds as an equality.

The following lemma, which we prove in [20], shows that the state constraints in both non-replicative and replicative models hold for any control-pair that satisfies (1) - thus, these constraints can be ignored henceforth, i.e., we can deal with optimal control problems with no state constraints.

lemma 1: (A) In non-replicative case, for any control function pair $(\varepsilon(.), u(.))$ that satisfies (1), $((S(t), I(t), D(t)))$, satisfies the state constraints for the non-replicative case in the $[0, T]$ interval, i.e., $0 \le S(t), I(t), D(t)$ and $S(t) + I(t) + D(t) \le 1$. Moreover, (i) $S(t) > 0$ for all $t \in [0, T]$, (ii) if $I_0 > 0$, $I(t) > 0$ for all $t \in [0, T]$, and (iii) if $\delta > 0$, $D(t) > 0$ for all $t \in [0, T]$.
(B) Similarly, in the replicative case, for any control function pair $(\varepsilon(.), u(.))$ that satisfies (1), $((S(t), I(t), R(t)))$, satisfies the state constraints for this case, i.e., $0 \le S(t), I(t), R(t)$ and $S(t) + I(t) + R(t) \le 1$ in the $[0, T]$ interval. Moreover, (i) $R(t), S(t) > 0$ for all $t \in [0, T]$, (ii) if $I_0 > 0$, $I(t) > 0$ for all $t \in [0, T]$, and (iii) if $\delta = 0$, $S(t) + I(t) + R(t) = 1$.

C. Motivation of the models and Instantiation

In the introduction section (§I), we described the motivations for the models presented in previous section through different examples from which interpretation of each of the corresponding states is straightforward. Here, and we add more comments on the nature of interactions in each example. First thing to point out is that, except for the case of infectious disease, replicative and non-replicative scenarios are conceivable.

Network Security: In a client-server based, p2p or cellular network, node A contacts (i.e., communicates with) node B if A knows the (ID or) address of B, and have the right permissions or infiltrates it. The homogeneous mixing assumption can represent worm propagation in 3G and 4G cellular networks (peer-to-peer, resp.) where infective mobiles (peers, resp.) try to infect randomly and uniformly generated (IDs or) addresses. Note that in any such mobile to mobile communication, irrespective of the locations of the mobiles, there are two wireless communications between access points and mobiles and the rest of the communications are through the backbone network where the delays and congestions are relatively limited. Similarly, peers communicate through the backbone network where delays are limited. Thus, in both p2p and cellular networks, the inter-meeting times have the same distribution irrespective of the location of the pairs. In a MANET, a contact occurs only when two nodes move into communication range of each other. Under mobility models such as random waypoint or random direction model (explained in [22]), Groenevelt *et al.* [23] has established the homogeneous mixing property for such contact processes in a highly mobile network. Security patches are distributed by mobile or stationary agents (in MANETs) or base stations (in AP and cellular networks) or a set of central servers (in wired networks). In replicative case, each recipient also forwards the security patch to nodes it contacts in future. The rates of contacts are determined by system specific parameters such as address scanning rates of infectives, communication

rates, mobility, communication ranges *etc.* The worm may completely prevent the download or installation of the patch in an infective node. This case corresponds to $\pi = 0$.

Delay Tolerant Networks (DTNs): Contact occurs when two nodes roam into communication range of each other. There is no infective or dead nodes. This can be modeled by setting $I_0 = D_0 = 0$ in our system dynamics equations.

Marketing-Reclamation/Rivalry: There is no dead state in these cases. Here, contacts constitute social interactions such as meetings, phone communications or email exchanges. The non-replicative case arises when only agents of the incumbent/rival attempt to persuade the customers, while in the replicative mode, each convert/subscriber advertises for the service through word of mouth as is incentivised by referral-based rewards/discounts. $\pi = 0$ represents the case in which customers are also pledged to the competitor and cannot be claimed by the incumbent/rival. Intermediate values of π corresponds to different resistance (inertia) of customers to switch.

D. The Objective Function

We seek to minimize the overall cost in a time window $[0, T]$, where T is a parameter of choice. At any given time t, the system incurs costs at the rates of $f(I(t))$, $g(D(t))$ and benefit at the rate of $L(R(t))$ where $f(.), g(.), L(.)$ are non-decreasing and differentiable functions such that (WLoG) $f(0) = g(0) = L(0) = 0$. In addition, each activated disseminator charges, or consumes resources at the rate $h(u(t))$ at time t since it uses a distribution rate of $u(t)$, and $\varepsilon(t)R_0$ fraction of the nodes are the activated disseminators at time t. Here, $h(x)$ is a twice-differentiable and increasing function in x such that $h(0) = 0$ and $h(x) > 0$ when $x > 0$. Note that the assumptions on $f(.), g(.), h(.)$ are mild and natural, and a large class of functions satisfy them. The aggregate system cost therefore is

$$J = \int_0^T f(I(t)) + g(D(t)) - L(R(t)) + \varepsilon(t)R_0 h(u(t))\, dt + \kappa_I I(T) + \kappa_D D(T) - \kappa_R R(T). \tag{4}$$

Replacing R_0 with $R(t)$ in (4) gives the overall cost for the replicative case, as here, activated disseminators at time t constitute $\varepsilon(t)R(t)$ (instead of $\varepsilon(t)R_0$) fraction of the total nodes. For both cases, at least one of the function f, g or L is not the null function, and h is either a concave, linear or a convex function of u.

Problem Statement: The system seeks to minimize the aggregate cost in (4) by appropriately regulating $\varepsilon(.), u(.)$ at all t subject to (1), when the states evolve **(A)** as per (2) for non-replicative, and (**B**) as per (3) for replicative dispatch, and satisfy the respective initial state conditions.

Here we briefly motivate the cost model for each of our different settings. Our cost model in (4) (and its replicative counterpart) is general enough to capture all of the cases.

Network Security: In communication networks, *each activated* disseminator consumes power and/or bandwidth at rate $h(u(t))$ at time t for transmission of patches. The total number of activated disseminators at time t is respectively $N\varepsilon(t)R_0$ and $N\varepsilon(t)R(t)$ for non-replicative and replicative dispatch. Infective and dead (dysfunctional) nodes incur accumulative costs to the network as well (represented by f and g functions respectively). Also κ_I and κ_D respectively represent the (scaled) cost per infective and dead node at the end of the network operation (i.e., time T). In this case, $L(R) \equiv 0$ and $\kappa_R = 0$.

Delay Tolerant Networks (DTNs): Similarly, activation and transmission of disseminators consume power, which is especially critical in energy constrained DTNs. Here, there are no infective ot dead nodes and hence, $f = g \equiv 0$ (also $\kappa_I = \kappa_D = 0$). There is reward associated with increasing the total number of nodes which have received a copy of the disseminated message. Also, the sooner the message is disseminated, the better, hence the integration of $L(R(t))$ over time (note that the negative sign convert the minimization problem to a maximization one). [24, appendix-A] directly relates the integral over time of the fraction of recovered nodes to the probability that a message is delivered to a sink before deadline T. Hence the minimum delay problem is transferred to maximization of $\int_0^T R(t)\, dt$, which corresponds to the special case of linear $L(x) = -x$ in our setting (also ref. [18], [19]). If T, as in [18], [19], [24], represents the deadline before the disseminated message reaches a (set of) destination(s), then $\kappa_R = 0$. If however, the objective is broadcasting a message by time T to many nodes, then κ_R represents the scaled benefit per node which has received the message at time T.

Marketing-Reclamation: The optimizer in this case is the incumbent who incurs a cost of J. Here, $g, L \equiv 0$, as infectives are the only group of customers who are not subscribed to the incumbent. That is, the incumbent incurs a cost only through infectives, since their converting away results in reduction of revenue (cessation of their subscription fee) over time. This loss is captured by integration of $f(I)$ over time. Among the individuals who are contacted, only those who are persuaded by the offers will switch back. The cost for advertisement, captured by integration of the term involving $h(.)$, is associated with the amount of discount offers and rewards provided to lure the customers back. The incumbent seeks to minimize its overall loss due to the entrance of the competitor, by dynamically determining the fraction of the individuals who should be selected for a special offer and how much discount should be provided, which in turn determines the efficacy u of the switch to the incumbent. Here, $\kappa_D = \kappa_R = \kappa_I = 0$.

Marketing-Rivalry: The optimization here, is from the viewpoint of one of the rivals. There is no dead state in this model, hence, similar to the reclamation case, $g \equiv 0$. However, $f \equiv 0$ instead of L, since only recovered are those customers who subscribe to the company of the optimizer (susceptibles are not subscribed to either). The revenue comes from the subscription fee of the recovered nodes, and is represented through integration of the $L(R)$ function over time. The cost for advertisement is similar to the Reclamation case. Here, $\kappa_I = \kappa_D = \kappa_R = 0$.

III. Optimal Non-replicative Dispatch

We apply Pontryagin's Maximum Principle to obtain a framework for solving the optimal control problem as posed in Problem Statements **(A)** and **(B)**. Let $((S, I, D), (\varepsilon, u))$ be an optimal solution to the problem posed in problem state-

ment (**A**) in the previous section, consider the *Hamiltonian* H, and corresponding *co-state* or *adjoint* functions $\lambda_S(t)$, $\lambda_I(t)$ and $\lambda_D(t)$, defined as follows:

$$H = f(I) + g(D) - L(R) + \varepsilon R_0 h(u) + (\lambda_I - \lambda_S)\beta_0 IS \\ -\beta_1 R_0 \varepsilon u \lambda_S S - \pi\beta_1 R_0 \varepsilon u \lambda_I I + (\lambda_D - \lambda_I)\delta I. \tag{5}$$

where $R = 1 - S - I - D$.

$$\dot{\lambda}_S = -\frac{\partial H}{\partial S} = -L'(R) - (\lambda_I - \lambda_S)\beta_0 I + \beta_1 R_0 \varepsilon u \lambda_S$$

$$\dot{\lambda}_I = -\frac{\partial H}{\partial I} = -L'(R) - f'(I) - (\lambda_I - \lambda_S)\beta_0 S + \pi\beta_1 R_0 \varepsilon u \lambda_I \\ - (\lambda_D - \lambda_I)\delta$$

$$\dot{\lambda}_D = -\frac{\partial H}{\partial D} = -L'(R) - g'(D). \tag{6}$$

along with the *transversality* conditions:

$$\lambda_S(T) = \kappa_R, \quad \lambda_I(T) = \kappa_I + \kappa_R, \quad \lambda_D(T) = \kappa_D + \kappa_R. \tag{7}$$

Then according to Pontryagin's Maximum Principle (e.g., [9, P. 109, Theorem 3.14]), there exist continuous and piecewise continuously differentiable co-state functions λ_1, λ_2 and λ_3, that at every point $t \in [0 \dots T]$ where ε and u is continuous, satisfy (6) and (7). Also,

$$(\varepsilon, u) \in \arg \min_{\underline{\varepsilon}, \underline{u} \text{ admissible}} H(\vec{\lambda}, (S, I, D), (\underline{\varepsilon}, \underline{u})). \tag{8}$$

A. Structure of the Optimal Non-replicative Dispatch

We establish that the two-dimensional optimal controls of patching in the non-replicative case have simple structures:

Theorem 1: In the problem statement (**A**), for either one of the following two cases: (i) $L \equiv 0$ and $f(.)$ is convex, (ii) $\delta = 0$, an optimal control $(\varepsilon(.), u(.))$ has the following simple structure:

1) When $h(.)$ is concave, $\exists\ t_1 \in [0 \dots T]$ such that (a) $u(t) = 1$ for $0 < t < t_1$, and (b) $u(t) = 0$ for $t_1 < t < T$.
2) When $h(.)$ is strictly convex, $\exists\ t_0, t_1$, $0 \le t_0 \le t_1 \le T$ such that (a) $u(t) = 1$ on $0 < t \le t_0$, (b) $u(t)$ strictly and continually decreases on $t_0 < t < t_1$, and (c) $u(t) = 0$ on $t_1 \le t \le T$.

In both cases, for all $t \in (0, T)$, except possibly for $t = t_1$ when $h(.)$ is strictly concave, $\varepsilon(t) = 1$ if and only if $u(t) > 0$, and $\varepsilon(t) = 0$ otherwise.

Proof: Let function $\varphi(t)$ be defined as follows:

$$\varphi := \beta_1(\lambda_S S + \pi\lambda_I I) \tag{9}$$

$\varphi(.)$ is thus a continuous function of time, which according to (7) has the following final value:

$$\varphi(T) = \beta_1(\kappa_R S(T) + \kappa_R I(T) + \kappa_I I(T)). \tag{10}$$

Also, as we prove in §III-B:

lemma 2: $\varphi(t)$ is a strictly decreasing function of t for $t \in [0, T)$.

We can rewrite the Hamiltonian in (5) as:

$$H = f(I) + g(D) - L(R) + (\lambda_I - \lambda_S)\beta_0 IS \\ +(\lambda_D - \lambda_I)\delta I + \varepsilon R_0(h(u) - \varphi u). \tag{11}$$

From (8), for each admissible control $(\underline{\varepsilon}, \underline{u})$ and for all $t \in [0, T]$,

$$\varepsilon(t)\left(h\left(u(t)\right) - \varphi(t)u(t)\right) \le \underline{\varepsilon}(t)\left(h\left(\underline{u}(t)\right) - \varphi(t)\underline{u}(t)\right)$$

$$\implies (\varepsilon(t), u(t)) \in \arg \min_{\substack{x \in [0,1] \\ y \in [0,1]}} x\left(h\left(y\right) - \varphi(t)y\right). \tag{12}$$

Since $(\varepsilon, u) \equiv (0, 0)$ is an admissible control, we have for all $0 \le t \le T$:

$$\varepsilon(h(u) - \varphi u) \le 0. \tag{13}$$

Note that whenever either u or ε is zero, irrespective of the other, $\varepsilon u = 0$, and since $h(0) = 0$, $\varepsilon h(u) = 0$. Thus, the state dynamics and the instantaneous cost incurred do not depend on the value of the other control function at these epochs. Thus, whenever one control function assumes a zero value, we can, WLoG, choose zero value for the other.

Next, consider a t at which the minimizer of $h\left(y\right) - \varphi y$ in $y \in [0, 1]$ is unique. If this unique minimizer is 0, then $\varepsilon = u = 0$ at t. In order to show this, we only need to show that $u = 0$ at t. Otherwise, if at t, $u > 0$, then $\varepsilon > 0$ at t, and $h(u) - \varphi u > h(0) - \varphi 0 = 0$. This contradicts (22). If this unique minimizer is positive, then at t, $\min_{y \in [0,1]}\left(h\left(y\right) - \varphi y\right) < 0$, and thus from (12), $\varepsilon = 1$ and u equals this unique minimizer. Thus, at any t at which the minimizer of $h\left(y\right) - \varphi y$ in $y \in [0, 1]$ is unique, $\varepsilon = 1$ if and only if $u > 0$, and $\varepsilon = 0$, otherwise.

For establishing the structure of optimal u, we separately consider the cases of concave and strictly convex $h(.)$.

1) $h(.)$ concave: When $h(.)$ is concave (i.e., $h'' \le 0$), at each time t, $h(x) - \varphi(t)x$ is a concave function of x, and thus, for any time t such that $\varphi(t) \ne h(1)$, the unique minimum is either at $x = 0$ or $x = 1$. Then,

$$\varepsilon(t)u(t) = \begin{cases} 0, & \varphi(t) < h(1) \\ 1, & \varphi(t) > h(1). \end{cases} \tag{14}$$

Following lemma 2, there can be at most one t at which $\varphi(t) = h(1)$ in $[0, T]$. Moreover, lemma 2 implies that if such t exists, say t_1, then $\varphi(t) > h(1)$ for $t \in [0, t_1)$, and $\varphi(t) < h(1)$ for $t \in (t_1, T]$. The theorem follows from (14).

2) $h(.)$ strictly convex: Since $h(.)$ is strictly convex (i.e., $h'' > 0$), the minimizer of $h\left(y\right) - \varphi(t)y$ in $y \in [0, 1]$ is unique irrespective of t. Thus, $\varepsilon(t) = 1$ if and only if $u(t) > 0$, and $\varepsilon(t) = 0$, otherwise. When $h(.)$ is strictly convex (i.e., $h'' > 0$), (12) implies that, if $\frac{\partial}{\partial x}\left(R_0 h\left(x\right) - \varphi(t)x\right)|_{x=y} = 0$ at a $y \in [0, 1]$, then $u(t) = y$, else $u(t) \in \{0, 1\}$. Then,

$$u = \begin{cases} 0, & \varphi \le R_0 h'(0) \\ h'^{-1}(\frac{\varphi}{R_0}), & R_0 h'(0) < \varphi \le R_0 h'(1) \\ 1, & R_0 h'(1) < \varphi. \end{cases} \tag{15}$$

Thus, from continuity of φ and h', u is continuous at all $t \in [0, T]$. Since $h(.)$ is strictly convex, $h'(.)$ is a strictly increasing function - hence, $h'(0) < h'(1)$. Thus, following lemma 2, there exist t_0, t_1, $0 \le t_0 \le t_1 \le T$, such that $\varphi > h'(1)$ on $0 < t \le t_0$, $h'(0) < \varphi \le h'(1)$ on $t_0 < t < t_1$, and $\varphi \le h'(0)$ on $t_1 \le t \le T$. The theorem follows from (15). ■

B. Proof of lemma 2

Proof: The state and co-state functions, and hence the φ function, are differentiable at each time $t \in [0,T)$ at which the (ε, u) function is continuous. Since (ε, u) is piecewise continuous, the lemma follows if we can show that $\dot{\varphi}$ is negative at each such t. Noting that $\beta_1 > 0$, at each such $t \in [0,T)$ we have:

$$\begin{aligned}\frac{\dot{\varphi}}{\beta_1} = \frac{1}{\beta_1}\frac{d}{dt}\varphi &= \dot{\lambda}_S S + \lambda_S \dot{S} + \pi\dot{\lambda}_I I + \pi\lambda_I \dot{I} \\ &= -\lambda_I\beta_0 IS + \pi\lambda_S\beta_0 IS - \pi f'(I)I - \pi\lambda_D\delta I \\ -L'(R)(S+\pi I) &= -(\lambda_I - \lambda_S)\pi\beta_0 IS - (1-\pi)\lambda_I\beta_0 IS \\ &\quad -\pi\lambda_D\delta I - \pi f'(I)I - L'(R)(S+\pi I)\end{aligned} \tag{16}$$

The right hand side is negative at each $t \in [0,T)$ since $I, S > 0$ at all $t \in [0,T]$ (lemma 1-A), $\beta_0 > 0$, $\delta \geq 0, 0 \leq \pi \leq 1$ and $f'(x), L'(x) \geq 0$ for all x (since $f(.)$ and $L(.)$ are non-decreasing functions), and because:

lemma 3: For all $0 \leq t < T$, we have $\lambda_D \geq 0$, $\lambda_I > 0$, and $(\lambda_I - \lambda_S) > 0$.

We prove lemma 3 in our tech. report [20].

■

IV. Optimal Replicative Dispatch

Similar to the non-replicative case, we define the Hamiltonian as:

$$\begin{aligned}H = f(I) + g(D) - L(R) + \varepsilon Rh(u) + (\lambda_I - \lambda_S)\beta_0 IS \\ -(\lambda_S - \lambda_R)\beta_1\varepsilon uRS - (\lambda_I - \lambda_R)\pi\beta_1\varepsilon uRI - \lambda_I\delta I.\end{aligned} \tag{17}$$

where $D = 1-(S+I+R)$. The system of co-state differential equations is as:

$$\begin{aligned}\dot{\lambda}_S &= -\frac{\partial H}{\partial S} = -(\lambda_I - \lambda_S)\beta_0 I + (\lambda_S - \lambda_R)\beta_1\varepsilon uR + g'(D) \\ \dot{\lambda}_I &= -\frac{\partial H}{\partial I} = -f'(I) - (\lambda_I - \lambda_S)\beta_0 S + (\lambda_I - \lambda_R)\pi\beta_1\varepsilon uR \\ &\quad + \lambda_I\delta + g'(D) \\ \dot{\lambda}_R &= -\frac{\partial H}{\partial R} = (\lambda_S - \lambda_R)\beta_1\varepsilon uS + (\lambda_I - \lambda_R)\pi\beta_1\varepsilon uI - \varepsilon h(u) \\ &\quad + g'(D) + L'(R).\end{aligned} \tag{18}$$

and the transversality conditions as:

$$\lambda_S(T) = 0, \lambda_I(T) = \kappa_I, \lambda_R(T) = -\kappa_R. \tag{19}$$

Then, according to Pontryagin's Maximum Principle ([9, P. 109, theorem 3.14]), there exist continuous and piece-wise continuous functions $\lambda_S(t)$ to $\lambda_R(t)$ that satisfy (18) and (19) at any t at which $(\varepsilon(t), u(t))$ is continuous, and the optimal (ε, u) satisfies:

$$(\varepsilon, u) \in \arg \min_{(\underline{\varepsilon},\underline{u}) \text{ admissible}} H(\vec{\lambda}, (S,I,D), (\underline{\varepsilon},\underline{u})). \tag{20}$$

The above framework can be used for numerically computing the optimum control and the minimum aggregate cost.

A. Structure of the Optimal Replicative Dispatch

Theorem 2: Consider an optimal control $(\varepsilon(.), u(.))$ to the problem posed in problem statement **B**.The same structural properties as in Theorem 1 (i.e., for the non-replicative case) also holds here.

The above results are somewhat surprising in that the activation fraction $\varepsilon(.)$ is completely specified by $u(.)$, and hence the two-dimensional control is reduced to a one-dimensional solution. The practical implication is that the activation scheme is *all* or *none*, and it is not optimal to activate a portion of the dispatchers. When $h(.)$ is strictly concave, the optimum transmission range, and hence the entire solution, is bang-bang and has at most one jump from 1 down to 0, and it is optimal to patch as aggressively as possible early on (as soon as the infection is detected and the patch is produced) and halt the patching after a certain time. When $h(.)$ is strictly convex, $\varepsilon(.)$ continues to be bang-bang and has at most one jump from 1 down to 0, but $u(.)$ has a strict but continuous descent to 0.

In the rest of the subsection, we prove Theorem 2.

Proof: Consider φ as defined in the following:

$$\varphi := (\lambda_S - \lambda_R)\beta_1 RS - (\lambda_I - \lambda_R)\pi\beta_1 RI$$

Now from (20) and referring to (17), for each admissible control $(\underline{\varepsilon}, \underline{u})$, and for all $t \in [0,T]$,

$$\begin{aligned}&\varepsilon(t)\left(R(t)h\left(u(t)\right) - \varphi(t)u(t)\right) \leq \underline{\varepsilon}(t)\left(R(t)h\left(\underline{u}(t)\right) - \underline{u}(t)\varphi(t)\right) \\ &\implies (\varepsilon(t), u(t)) \in \arg \min_{\substack{x\in[0,1] \\ y\in[0,1]}} x\left(R(t)h\left(y\right) - \varphi(t)y\right).\end{aligned} \tag{21}$$

Since $(\varepsilon, u) \equiv (0,0)$ is an admissible control, we have for all $0 \leq t \leq T$:

$$\varepsilon(Rh(u) - \varphi u) \leq 0. \tag{22}$$

The optimality of the $\varepsilon(t)$ as stated in Theorem 2 follows by similar argument following (13). We prove the structure of u separately for the cases of strictly concave and strictly convex $h(.)$, using the following lemma, which we prove in §IV-B.

lemma 4: Let $\psi(t) = \frac{\varphi(t)}{R(t)}$. Then, $\psi(t)$ is a strictly decreasing function of t for $t \in [0,T)$.

1) $h(.)$ concave: Since $h(.)$ is concave (i.e., $h'' < 0$) and $R > 0$ by lemma 1-B, no $y \in (0,1)$ attains $\min_{y\in[0,1]}\left(Rh\left(y\right) - \varphi y\right)$. Thus, if at time t, $\varphi - Rh(1) < 0$, then $y = 0$ is the unique minimizer of $Rh\left(y\right) - \varphi y$ in $y \in [0,1]$. Thus, $\varepsilon = u = 0$ at any such time. If $\varphi - Rh(1) > 0$, $y = 1$ is this unique minimizer. Thus, $\varepsilon = u = 1$ at any such time. Thus:

$$(\varepsilon, u) = \begin{cases}(0,0) & \varphi - Rh(1) < 0 \\ (1,1), & \varphi - Rh(1) > 0\end{cases} \tag{23}$$

Using lemma 4, we conclude that $\varphi/R = h(1)$ at at most one time epoch in $(0,T)$, say t_1, and $\varphi/R > h(1)$ in $(0,t_1)$ and, if such t_1 exists, then $\varphi/R < h(1)$ in (t_1,T). The theorem follows from (23).

2) $h(.)$ strictly convex: Since $h(.)$ is strictly convex (i.e., $h'' > 0$), the minimizer of $R(t)h(y) - \varphi(t)y$ in $y \in [0,1]$ is unique irrespective of t. Thus, $\varepsilon(t) = 1$ if and only if $u(t) > 0$, and $\varepsilon(t) = 0$, otherwise. Thus, we only need to prove the requisite properties of u. This minimizer, and hence u, is:

$$\begin{cases} 0, & \frac{\varphi}{R} \le h'(0) \\ h'^{-1}(\frac{\varphi}{R}), & h'(0) < \frac{\varphi}{R} \le h'(1) \\ 1, & h'(1) < \frac{\varphi}{R}. \end{cases} \tag{24}$$

Now, since φ, R, h' are continuous, h' is strictly increasing, $R > 0$ at all $t \in [0,T]$, u is continuous at all $t \in [0,T]$. $R(t) > 0$ at all $t \in [0,T]$ by lemma 1-B, and $h'(x) \ge 0$ for all x. The proof follows if we can show that $\dot{u} < 0$, when $h'(0) < \frac{\varphi}{R} \le h'(R)$. Now, for $h'(0) < \frac{\varphi}{R} \le h'(R)$, we have

$$u = h'^{-1}(\frac{\varphi}{R}) \Rightarrow \dot{u} = \frac{\frac{d}{dt}(\frac{\varphi}{R})}{h''(u)}$$

According to lemma 4, this is negative. ■

B. Proof of lemma 4

Proof: We prove this lemma using lemma 5 which we state next, but prove in our tech. report [20].

lemma 5: For all $0 \le t < T$, we have $(\lambda_I - \lambda_S) > 0$, $(\lambda_S - \lambda_R) > 0$ and $\lambda_R \le 0$.

Since (ε, u) is piecewise continuous and $h(.)$ is continuous, $\varepsilon u, \varepsilon h(u)$ are piecewise continuous as well. Thus, from continuity of φ, R, we need to show that $\dot{\psi} < 0$ at any $t \in [0,T)$ at which (ε, u) is piecewise continuous. Now, at such a t,

$$\begin{aligned} \dot{\varphi} &= (\dot{\lambda}_S - \dot{\lambda}_R)\beta_1 RS + (\dot{\lambda}_I - \dot{\lambda}_R)\pi\beta_1 RI \\ &+(\lambda_S - \lambda_R)\beta_1 \dot{R}S + (\lambda_I - \lambda_R)\pi\beta_1 \dot{R}I \\ &+(\lambda_S - \lambda_R)\beta_1 R\dot{S} + (\lambda_I - \lambda_R)\pi\beta_1 R\dot{I} \\ &= -\pi\beta_1\beta_0 RIS\lambda_R - \beta_1\beta_0 RIS\lambda_I \\ &+\beta_1\beta_0 RIS\lambda_R + \pi\beta_1\beta_0 RIS\lambda_S - \pi\beta_1 f'(I)RI + \pi\beta_1 RI\delta\lambda_R \\ &-L'(R)R\beta_1(S + \pi I) + \varepsilon Rh(u)\beta_1(S + \pi I) \\ &\to \pm\beta_0\beta_1 RIS\lambda_S \text{ and re-arrangement} \to \\ &= -\beta_0\beta_1(1-\pi)RIS(\lambda_S - \lambda_R) - \beta_0\beta_1 RIS(\lambda_I - \lambda_S) \\ &-\pi\beta_1 f'(I)RI + \pi\beta_1 RI\delta\lambda_R \\ &-L'(R)R\beta_1(S + \pi I) + \varepsilon Rh(u)\beta_1(S + \pi I) \\ &= \{\text{negative term}\} + \varepsilon Rh(u)\beta_1(S + \pi I). \end{aligned} \tag{25}$$

The expressions denoted as $\{\text{negative term}\}$ is negative at each $t \in [0,T)$ owing to lemma 5 and since $\beta_0, \beta_1 > 0, \delta \ge 0, 0 \le \pi \le 1$ by assumption and $S, I, R > 0$ by lemma 1-B. At any such t,

$$\dot{\psi}(t) = \frac{d}{dt}(\frac{\varphi}{R}) = \frac{\dot{\varphi} - \frac{\varphi}{R}\dot{R}}{R} \tag{26}$$

$$= \frac{\{\text{negative term}\} + \varepsilon Rh(u)\beta_1(S + \pi I) - \dot{R}\frac{\varphi}{R}}{R}$$

$$= \frac{\{\text{negative term}\} + \varepsilon\left(Rh(u) - \varphi u\right)\beta_1(S + \pi I)}{R}$$

$$\le \frac{\text{negative term}}{R} \tag{27}$$

The last inequality follows from (22), lemma 1-B and since $\beta_1 > 0, \pi \ge 0$. The lemma follows since the right hand side is negative at each $t \in [0,T)$. ■

V. Numerical Computations

First, with the intention of illustrating the theorems, we depict the optimal controls for the general case, i.e., when all of the states exist, and the cost is in the general form. The parameters used are stated in the caption of the fig.2. The figures on the right side are related to a concave $h(u)$ function and and the ones on the right figures are according to a convex $h(u)$ (for both replicative and non-replicative cases).

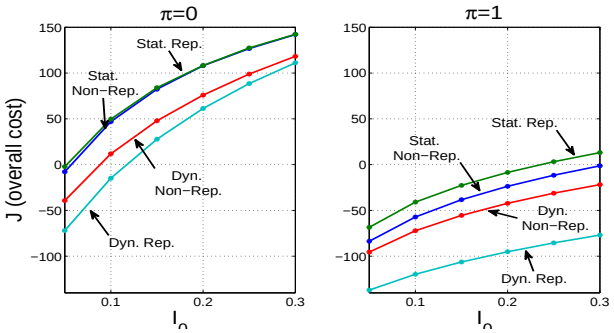
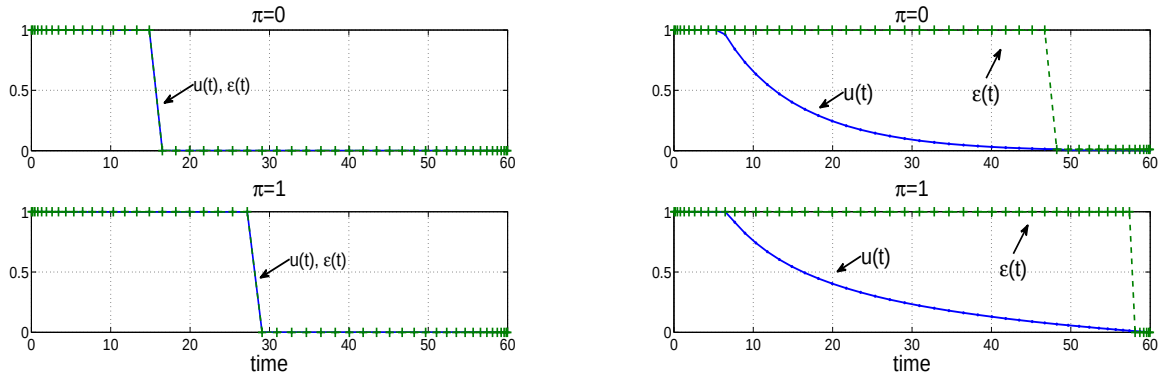


Fig. 3: Comparison of costs for four policies for various I_0. Dynamic replicative policy achieves the best performance amongst the four. The parameters used (except for the parameter in the horizontal axis) are the same as in fig.2

Next, we have depicted a comparison of the aggregate costs that is incurred as a result of applying each of these four different policies: optimal replicative dispatch, optimal non-replicative dispatch, best static replicative dispatch, best static non-replicative dispatch. The aim is to explore the efficacy of the replicative dispatch over the non-replicative dispatch and dynamic control over static control. For the static policies, the control assumes a fixed value throughout the interval of $[0 \ldots T]$. We have then varied this fixed value and selected the one that leads to the least cost (hence, the 'best static'). For different values of I_0, as we can see in fig.3, under each dispatch model, the optimal dynamic control will incur lower aggregate cost than the best static control. This is because the set of feasible solutions for a dynamic control is a strict superset of that for a static control - the former can always choose the immunization rate function as a constant, whereas the latter can never vary the immunization rate as a function of time. The difference is more emphasized for the case of replicative dispatch where optimal dynamic policy achieves 50 to 100% better cost values compared to the best static policies. Also, the optimal dynamic replicative dispatch incurs lower aggregate cost than its non-replicative counterpart, since the replicative dispatch can emulate non-replicative: one can always activate only a fraction of the dispatchers in the replicative setting so that it equals the number of active dispatchers in non-replicative case.

In the end, we illustrate the robustness of dynamic policies. A practical issue in implementing the dynamic polices in this paper is that the parameters of the system are not always accurately known, and only rough estimate is available. Therefore, it is important to investigate the sensitivity of the



(a) Concave $h(u)$, Non-replicative

(b) Convex $h(u)$, Replicative

Fig. 2: Illustration of the theorems. The common parameters are $\delta = 0.01$, $\beta = 0.15$, $I_0 = 0.2$, $R_0 = 0.25$, $D_0 = 0$, $T = 60$, $f(I) = 5I$, $g(D) = 10D$, $L(R) = 5R$. For concave $h(u)$ (fig.2(a)) we have used $h(u) = 10u$, and for convex $h(u)$ (fig.2(b)) we have used $h(u) = 10u^2$.

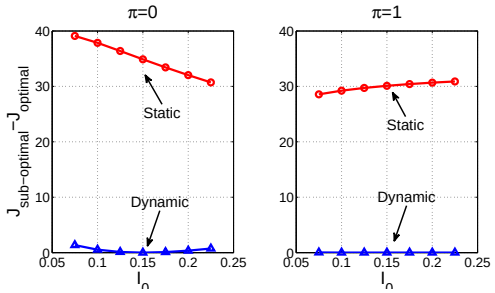


Fig. 4: Inaccurate I_0, Non-replicative.

Fig. 5: Robustness of dynamic policy. The parameters used are the same as in the caption of fig.2 (except for the variable in the horizontal axis, and that $R_0 = 0.2$). The increase in the overall cost, as a result of 50% inaccuracy in the estimation of the value of I_0 and β is less than 5%.

efficacy of the defense to these inaccuracies. Let's say that the initial fraction of the infective nodes is estimated to be $I_0 = 0.15$, however with potential inaccuracy of 50%. We apply the dynamic and static policies that are calculated based on this estimation to systems in which the actual values were off from this estimate (up to 50%), assuming other parameters are fixed. Then we depict the increase in the total cost due to applying these sub-optimal policies, that is, the cost when the sub-optimal policy (the dynamic and static optimal control calculated based on the inaccurate estimate $I_0 = 0.15$) minus the cost when the actual optimal dynamic policy for the accurate value of I_0 is applied. As fig.4 shows, the increase in the total damage for the optimal dynamic policy due to inaccurate estimation of I_0 is significantly low, showing the robustness of the non-replicative dynamic policies with respect to erroneous estimation of I_0. Similar behaviour is observed for estimation of β and replicative policy [20].

REFERENCES

[1] O. Diekmann and J. Heesterbeek, *Mathematical epidemiology of infectious diseases: Model building, analysis, and interpretation*. Wiley, 2000.

[2] P. Dodds and D. Watts, "A generalized model of social and biological contagion," *Journal of Theoretical Biology*, vol. 232, no. 4, 2005.

[3] X. Zhang, G. Neglia, J. Kurose, and D. Towsley, "Performance modeling of epidemic routing," *Computer Networks*, vol. 51, no. 10, 2007.

[4] S. Shakkottai and R. Srikant, "Peer to peer networks for defense against internet worms," in *Proc. of ACM Interperf'06*, ACM, 2006.

[5] A. Bose, X. Hu, K. Shin, and T. Park, "Behavioral detection of malware on mobile handsets," in *Proc. of the ACM Mobisys'08*, ACM, 2008.

[6] N. Weaver, V. Paxson, and S. Staniford, "A worst-case worm," in *Proc. of WEIS'04*, 2004.

[7] F. Castaneda, E. Sezer, and J. Xu, "WORM vs. WORM: preliminary study of an active counter-attack mechanism," in *Proc. of the 2004 ACM workshop on Rapid malcode*, ACM, 2004.

[8] C. Fraser, S. Riley, R. Anderson, and N. Ferguson, "Factors that make an infectious disease outbreak controllable," *Proc. of the National Academy of Sciences of the United States of America*, vol. 101, no. 16, 2004.

[9] D. Grass, J. Caulkins, G. Feichtinger, G. Tragler, and D. Behrens, *Optimal control of nonlinear processes: with applications in drugs, corruption, and terror*. Springer Verlag, 2008.

[10] S. Sethi and G. Thompson, *Optimal control theory: applications to management science and economics*. Springer Netherlands, 2000.

[11] H. Behncke, "Optimal control of deterministic epidemics," *Optimal control applications and methods*, vol. 21, no. 6, 2000.

[12] K. Wickwire, "Mathematical models for the control of pests and infectious diseases: a survey," *Theoretical Population Biology*, vol. 11, no. 2, 1977.

[13] G. Feichtinger, R. Hartl, and S. Sethi, "Dynamic optimal control models in advertising: recent developments," *Management Science*, vol. 40, no. 2, pp. 195–226, 1994.

[14] S. Sethi, "Dynamic optimal control models in advertising: a survey," *SIAM review*, vol. 19, no. 4, pp. 685–725, 1977.

[15] M. Khouzani, S. Sarkar, and E. Altman, "Dispatch then Stop: Optimal Dissemination of Security Patches in Mobile Wireless Networks," in *49th IEEE CDC*, 2010.

[16] M. Bloem, T. Alpcan, and T. Başar, "Optimal and robust epidemic response for multiple networks," *Control Engineering Practice*, vol. 17, no. 5, pp. 525–533, 2009.

[17] M. Khouzani, E. Altman, and S. Sarkar, "Optimal Quarantining of Wireless Malware Through Power Control," in *ITA'09*, 2009.

[18] E. Altman, A. Azad, T. Basar, and F. De Pellegrini, "Optimal activation and transmission control in delay tolerant networks," *Infocom 2010*.

[19] E. Altman, T. Basar, and F. Pellegrini, "Optimal monotone forwarding policies in delay tolerant mobile ad-hoc networks," *ACM Inter-Perf*, 2008.

[20] M. Khouzani, S. Sarkar, and E. Altman, "Optimal Control of Epidemic Evolution (Detailed Version)," in *webpage http://www.seas.upenn.edu/ swati/publication.htm*.

[21] T. Kurtz, "Solutions of ordinary differential equations as limits of pure jump Markov processes," *Journal of Applied Probability*, vol. 7, no. 1, 1970.

[22] C. Bettstetter, "Mobility modeling in wireless networks: categorization, smooth movement, and border effects," *ACM SIGMOBILE Mobile Computing and Communications Review*, vol. 5, no. 3, 2001.

[23] R. Groenevelt, P. Nain, and G. Koole, "The message delay in mobile ad hoc networks," *Performance Evaluation*, vol. 62, no. 1-4, 2005.

[24] T. Small and Z. Haas, "The shared wireless infostation model: a new ad hoc networking paradigm (or where there is a whale, there is a way)," in *MobiHoc'03*, ACM, 2003.