

# Information Disclosure about Booster Efficacy in a Non-Stationary Environment

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**Abstract**—This paper investigates the dynamic disclosure of information in non-stationary environments. In particular, a planner iteratively discloses information about the efficacy of an immunizing booster shot that stochastically evolves over time amid the long-run spread of an infectious disease whose severity also varies over time. Each time period, a heterogeneous population of agents uses the disclosed information to determine whether they should obtain the booster shot, and then whether to remain isolated or active. The central planner’s objective is to ensure that the active population remains above a minimum threshold each period. We characterize a Markov decision process over the state of beliefs and how signalling mechanisms act on them. We highlight the “greedy” disclosure rule which provides the least amount of information possible subject to the planner maximizing the likelihood of achieving the active population threshold in the current period. Our results demonstrate that the greedy disclosure rule becomes optimal in finite time. We show this for settings where the population’s belief over the booster’s efficacy becomes more pessimistic than the belief required in the long-run.

## I. INTRODUCTION

### A. Motivation

Repetitively administering multiple vaccine doses or “boosters” is a potent tool in mitigating long-run disease spread in order to enable in-person activities. However, as diseases mutate, boosters may require dynamic updates and could become unsafe or less effective [1]. This makes it difficult to persuade individuals to make use of boosters, especially if the previous information they have collected suggests that boosters are flawed. The COVID-19 outbreak renewed interest in how information affects serial immunization and social activity [2], [3]. This paper investigates the use of *strategic dynamic information disclosure* to shape public beliefs and maintain desired activity levels over time. Each time period, a central planner carefully designs stochastic “experiments” that depend on the true uncertain booster efficacy at that time. The experiments’ outcomes (i.e. signals) are used by the population to update their existing beliefs over the true booster efficacy in that time period. Planners can thereby influence the population towards more desirable beliefs and consequently desirable outcomes. Examples of these experiments may include choosing to fund specific types of studies that measure vaccine effectiveness or establishing rules that censor particular data sources.

### B. Our work

In this paper, we consider a model where, each period, a unit mass of strategic agents has the choice to be active or remain

isolated amid disease spread. The active agents can choose to take a booster shot at a cost to receive an additional level of protection against the disease. The planner aims to ensure the active population size remains above a specified threshold. The disease’s *infectiousness* changes over time according to finite Markov chain. Independent of this, the booster shot’s efficacy also evolves according to a finite Markov chain. The planner can provide information about the booster’s efficacy in each period, but disclosing precise information in one period limits the planner’s ability to shape agents’ beliefs in future periods. Each period in our model practically could correspond to the time length between the emergence of new disease variants or booster technologies. We investigate the optimal dynamic disclosure rule and identify which signalling mechanisms the planner should optimally choose in each period by solving a non-stationary Markov decision process. Our work contributes to the literature by providing new insights into the optimal design of dynamic information for heterogeneous agents in non-stationary environments such as long-run disease transmission. Our technical contribution includes a new strategy to identify the optimal dynamic disclosure rule when the set of beliefs the planner seeks to induce is non-stationary.

### C. Literature Review

Information disclosure is the key tool we motivate as an effective disease mitigation tool [4], [5]. Several works have demonstrated the value of information disclosure as a means to disrupt disease spread [6], [7]. The authors of [8] consider a model of hybrid workers that we adapt by making dynamic and by allowing agents to immunize with boosters.

Critically, in contrast to existing work on information disclosure to mitigate disease spread, our model discloses information about booster efficacy each period. Our work falls under the subdomain of *dynamic information disclosure* which requires planners to account for both the latent evolution in uncertainty and the implications of information disclosed in the present on future beliefs.

Several works provide insightful models into dynamic information disclosure [9], [10], [11]. Most notably, [12] solves for the optimal dynamic information disclosure rule for forward-looking agents that choose when to halt a stochastic process to match a switch in the uncertain binary state. By contrast, agents in our setting continuously make decisions and we consider Markovian dynamics on the uncertain parameter which has direct modelling applications for disease evolution. Their results suggest that planners can delay information transmission to entice the agent to wait longer. We

recover a similar insight about keeping agents less informed in our model as we show that planners defer information disclosure when the current period reward is maximized.

Most closely related to our work, [13] presents a model with a myopic receiver where the uncertain parameter also evolves according to a Markov chain. Our work specifically improves upon their results and this subdomain by considering multiple heterogeneous agents and non-stationary objectives. Our model has two sources of non-stationary uncertainty – booster efficacy and disease infectiousness. Since beliefs on both uncertain quantities impact agents’ actions, the time-varying disease infectiousness creates a non-stationary environment that cannot be captured in their setting. Using distinct technical tools, our results analogously show the robustness and optimality of the “greedy” mechanism that *minimizes* information disclosure subject to maximizing the current period reward.

More practically, various works studying disease severity over time have shown that diseases mutate in favor of *continued survival* which leads diseases to become easier to transmit, but less deadly with time [14], [15]. Moreover, the dynamic mutation of disease has empirically resulted in changes to the efficacy of pharmaceuticals such as boosters and vaccines [16]. Our model captures these features by allowing the uncertain parameters and beliefs to drift latently according to the Markov chain. Furthermore, the utility of information disclosure has been practically motivated as [17] and [18] have shown that communicating information about the efficacy or safety of vaccines significantly influences the adaptation of boosters and vaccines.

#### D. Outline

We outline the technical contributions of our work. In Section II, we describe the model of our agents and the evolution of both the disease infectiousness and the uncertain booster efficacy. We formalize the signalling mechanisms that the planner uses to disclose information to the agents and describe the planner’s utility model.

In Section III, we compute a cutoff for the private benefit from activity. At equilibrium, agents with values below the cutoff isolate and agents with values above the cutoff are active – either all with a booster or all without a booster (Prop. 1). This enables us to compute the isolated mass in terms of the beliefs over the effectiveness of the booster and the infectiousness in the current period. We show that the map between beliefs and the isolated mass and vice versa are monotone and continuous (Lemmas 1, 2, 3).

Next, in Section IV, we characterize the value functions mapping current beliefs to the total discounted sum of future rewards. We show that the functions are concave (Prop. 2) and that identifying the segments where the value function is linear is sufficient to determine the optimal disclosure rule (Lemma 4). We define the “greedy” disclosure rule which discloses the minimal amount of information subject to maximizing the current period reward. We show that, after finite time, this disclosure rule becomes optimal for perpetuity (Theorem 1 and Prop. 7).

Finally, in Section V, we provide a brief numerical compar-

ison of the greedy disclosure rule against fully-informative and non-informative disclosure rules.

Due to space limitation, proofs are deferred to [19].

## II. PROBLEM FORMULATION

### A. Agent Model and Disease Evolution

We consider a dynamic model of agents’ decision-making subject to information disclosure in the presence of an infectious disease. The time horizon is discrete, indexed by  $t = 1, \dots, \infty$ . In each period  $t$ , a unit-mass of myopic, risk-neutral individuals (agents) face a choice between remaining isolated ( $\ell_R$ ) or participating in a public activity with ( $\ell_B$ ) or without ( $\ell_S$ ) taking a period-specific booster shot that provides an *uncertain* level of added immunity to the disease for that period only. The public location and activity are general to a variety of practical settings such as working in office spaces or stadium attendance for larger events. The agents are supervised by a strategic, long-run *central planner* that aims to manage the public activity level over the entire time horizon. The myopicity of agents is justified by the renewal of the population as in the case of stadium attendance or the agents’ having shorter-term objectives than that of the planner as in the case of work.

As is common in the literature, we model the infectiousness of the disease  $\theta^t \in \{\theta_L, \theta_H\}$  using a two-state, irreducible, homogeneous Markov chain with a commonly-known transition matrix  $\mathcal{M}_\Theta$ . In practice, new disease variants periodically emerge with stochastically generated mutations. The corresponding dynamics of the disease’s infectiousness have been well-approximated by Markov chains [20]. In an abuse of notation, denote the probability of transitioning from  $\theta^t = \theta_j$  to  $\theta^{t+1} = \theta_k$  by  $(\mathcal{M}_\Theta)_{jk} := \mathbb{P}[\theta^{t+1} = \theta_k | \theta^t = \theta_j]$ . The initial distribution  $m_\Theta^\circ = [\mathbb{P}[\theta^1 = \theta_L], \mathbb{P}[\theta^1 = \theta_H]]$  is common knowledge at the start of period  $t = 1$ . We denote  $\zeta_\Theta := (\mathcal{M}_\Theta)_{HL}$ ,  $\nu_\Theta := \frac{(\mathcal{M}_\Theta)_{LH}}{(\mathcal{M}_\Theta)_{HL}}$ , and the stationary distribution of  $\{\theta^t\}_{t \geq 1}$  by  $m_\Theta$ . We assume neither the agents nor the planner can directly observe the initial value  $\theta^1$  or any future states  $\theta^t$ , so the infectiousness is *uncertain* across time.

Agents receive a private benefit  $v$  from participating in the activity ( $\ell_B$  or  $\ell_S$ ), which is drawn from a commonly-known distribution denoted by  $G$  supported on  $[0, M]$  which we assume for convenience is continuous with bounded, non-zero density  $0 < g \leq \frac{dG}{dv} \leq \bar{g}$ . Agents that participate in the activity incur a stochastic cost that depends on the infectiousness of the disease in that time period  $\theta^t$  and the size of the isolated population  $y^t$  (i.e. active population is  $1 - y^t$ ). We model the infectious risk as being independent of the fraction of the active population that is boosted, as boosted agents may be partially immunized but are still capable of disease transmission which poses a risk to others. This stochastic cost incurred without a booster is denoted by  $\beta(y^t; \theta^t)$ .

Agents that receive the booster have their cost subsequently dampened, in proportion to the efficacy of the booster in that period, denoted by  $\gamma^t$ . The booster administered in this period only provides the agent with protection in the current

period, as immunity is not carried over across periods. Specifically, the cost incurred by agents who receive the booster is  $(1-\gamma^t)\beta(y^t; \theta^t)$ , however, they incur an additional cost  $\kappa$  to acquire the booster. The utility of agents who choose to remain isolated ( $\ell_R$ ) is zero. When indifferent between any two choices, we assume that agents choose  $\ell_B$  over  $\ell_S$  and  $\ell_R$ , and choose  $\ell_S$  over  $\ell_B$ . Consequently, the utilities of agents can be expressed as follows.

$$u_v(\ell_R, y^t; \theta^t, \gamma^t) = 0 \quad (1)$$

$$u_v(\ell_S, y^t; \theta^t, \gamma^t) = v - \beta(y^t; \theta^t) \quad (2)$$

$$u_v(\ell_B, y^t; \theta^t, \gamma^t) = v - \kappa - (1 - \gamma^t)\beta(y^t; \theta^t) \quad (3)$$

Motivated by a simple epidemiological model of community transmission (see [8]), we assume that this cost is linear in the true state  $\theta^t$  and decreasing in the mass of agents choosing to remain isolated  $y^t$ , and can be expressed as:

$$\beta(y^t; \theta^t) := \theta^t c_1(y^t) + c_2(y^t), \quad (4)$$

where  $c_1, c_2 : [0, 1] \rightarrow \mathbb{R}$  are publicly known functions with following properties: (i)  $c_1(1) = c_2(1) = 0$ ,  $c_1(0) = C_1$ ,  $c_2(0) = C_2$ ; (ii)  $c_1$  is strictly decreasing and continuous; and (iii)  $c_2$  is weakly decreasing and continuous.

We assume that the booster's current efficacy  $\gamma^t \in \{0, E\}$  for some commonly-known scalar  $0 < E < 1$  and that the efficacy evolves over time according to a Markov chain parameterized by a known stochastic transition matrix  $\mathcal{M}_\Gamma$ .

We denote the initial distribution by  $m_\Gamma^\circ$  and the stationary distribution by  $m_\Gamma$ . Moreover, denote  $\zeta_\Gamma := (\mathcal{M}_\Gamma)_{E0}$  and  $\nu_\Gamma = \frac{(\mathcal{M}_\Gamma)_{0E}}{(\mathcal{M}_\Gamma)_{E0}}$ .

In our model, the two stochastic processes  $\{\gamma^t\}_{t \geq 1}$  and  $\{\theta^t\}_{t \geq 1}$  are independent, i.e.  $\{\gamma^t\}_{t \geq 1} \perp \{\theta^t\}_{t \geq 1}$ . We believe this to be a practical assumption when considering settings where the technology used to develop boosters itself changes across time periods or the genetic characteristics of the disease change significantly from period to period.

### B. Signalling

The central planner is a strategic entity and each period implements a *signaling mechanism* to publicly disclose information *only* about the current efficacy of the booster  $\gamma^t$  through a mechanism  $\pi^t = \langle \mathcal{I}^t, \{z_\gamma^t\}_{\gamma \in \{0, E\}} \rangle$ . The set  $\mathcal{I}^t$  is an alphabet of signals, and  $z_\gamma^t$  are probability distributions over  $\mathcal{I}^t$ . Denote the set of all such signalling mechanisms  $\pi^t$  by the set  $\Pi$ . The disclosure of information in period  $t$  occurs as follows. First, the planner commits to and discloses a signaling mechanism  $\pi^t \in \Pi$ . Next, the true state  $\gamma^t$  is realized using the Markovian dynamics from the previous state  $\gamma^{t-1}$  with both states not directly observed by the agents or planner. The corresponding probability distribution  $z_{\gamma^t}^t$  is used to disclose a signal to all the agents; that is,  $i^t \in \mathcal{I}^t$  is publicly signaled with probability  $z_{\gamma^t}^t(i^t)$ . Finally, agents use the received signal to update their belief over the state  $\gamma^t$  and make simultaneous choices about their choice of action in  $\{\ell_R, \ell_B, \ell_S\}$ .

We believe it practical for the planner to only convey information about the booster efficacy and not the infectiousness

of the disease in any given period due to the comparative cost in estimating both quantities dynamically. The infectiousness of the disease is a parameter that both encapsulates the cost of illness and the likelihood of transmission – both of these quantities have high variance and are costly to estimate via experiment. However, randomized control trials and studies requiring limited samples can provide accurate estimates of the efficacy of pharmaceutical interventions like boosters [21].

Notationally, we distinguish between the public belief just before and after the information  $i^t$  is disclosed in period  $t$ . We express the agents' beliefs prior to the revelation of  $i^t$  by  $\underline{p}^t := \mathbb{P}[\gamma^t = E \mid \{i^j\}_{j \leq t-1}]$ . Then, on receiving signal  $i^t \in \mathcal{I}^t$ , the agents update their belief  $\bar{p}^t := \mathbb{P}[\gamma^t = E \mid \{i^j\}_{j \leq t}]$  according to Bayes' rule:

$$\begin{aligned} \bar{p}^t &= \mathbb{P}[\gamma^t = E \mid \{i^j\}_{j \leq t}] \\ &= \frac{\underline{p}^t z_E^t(i^t)}{\underline{p}^t z_E^t(i^t) + (1 - \underline{p}^t) z_0^t(i^t)} \end{aligned} \quad (5)$$

To keep quantities notationally consistent, let  $p^{eq} := (m_\Gamma)_E$ . Between periods, observe that the states evolve according to known *linear* Markovian dynamics, so in each period  $t$ :

$$\begin{aligned} \underline{p}^t &:= \phi_\Gamma(\bar{p}^{t-1}) \\ &= \bar{p}^{t-1} (\mathcal{M}_\Gamma)_{EE} + (1 - \bar{p}^{t-1}) (\mathcal{M}_\Gamma)_{0E} \\ &= p^{eq} + (1 - \nu_\Gamma \zeta_\Gamma - \zeta_\Gamma) (\bar{p}^{t-1} - p^{eq}) \end{aligned} \quad (6)$$

Analogously, we can express the beliefs over  $\theta^t$  at any given time by  $r^t = \mathbb{P}[\theta^t = \theta_H]$  with  $r^{eq} := (m_\Theta)_H$ . Since the manipulation of beliefs with respect to the disclosed information does not affect  $r^t$ , this is a known sequence over time with  $r^t \rightarrow r^{eq}$ . Precisely, independent of the chosen  $\{\pi^t\}$ :

$$\begin{aligned} r^t &:= \phi_\Theta(r^{t-1}) \\ &= r^{eq} + (1 - \nu_\Theta \zeta_\Theta - \zeta_\Theta) (r^{t-1} - r^{eq}) \end{aligned} \quad (7)$$

In this paper, we restrict to settings where  $0 < \nu_\Theta \zeta_\Theta + \zeta_\Theta < 1$  and  $0 < \nu_\Gamma \zeta_\Gamma + \zeta_\Gamma < 1$  to study scenarios where the Markov chains update beliefs in infectiousness and booster efficacy monotonically. Here,  $r^1 > r^{eq}$  (resp.  $r^1 < r^{eq}$ ) corresponds to settings when the infectiousness is believed to be progressively getting weaker (resp. stronger) on average over time. That is,  $r^t$  is either strictly decreasing or increasing in  $t$ .

The mechanism  $\pi^t$  establishes a set of posterior distributions for each signal with each posterior distribution incident with a particular probability. We thus can equivalently represent  $\pi^t$  by a set of tuples  $\{(q_i^t, \mu_i^t)_{i \in \mathcal{I}^t}\}$  of the probability that each signal  $i \in \mathcal{I}^t$  is realized ( $q_i^t$ ) and the posterior belief on observing that signal ( $\mu_i^t$ ). Formally, for all  $i \in \mathcal{I}^t$ :

$$q_i^t := \underline{p}^t(0) z_0^t(i) + \underline{p}^t(E) z_E^t(i) \quad [\text{signal probability}] \quad (8)$$

$$\mu_i^t := \frac{\underline{p}^t(E) z_E^t(i)}{\underline{p}^t(0) z_0^t(i) + \underline{p}^t(E) z_E^t(i)} \quad [\text{posterior belief}] \quad (9)$$

The intuition of the representation is that the mechanism

equivalently sets  $\bar{p}^t = \mu_i^t$  with probability  $q_i^t$ . Since,  $\mu_i^t$  establishes a probability over a binary set, appealing to the results of [22], all mechanisms in  $\Pi$  can be bijectively mapped to the set of all tuples  $\{(q_i, \mu_i)_{i \in \mathcal{I}}\}$  such that  $q_i, \mu_i \in [0, 1]$ ,  $\sum_{i \in \mathcal{I}} q_i \mu_i = \underline{p}^t$  and  $\sum_{i \in \mathcal{I}} q_i = 1$ . As is customary in the literature, we will refer to these as *splittings* of the prior belief  $\underline{p}^t$  into posterior beliefs  $\bar{p}^t = \mu_i$  with weights  $q_i$  [22].

### C. Planner Objective

The planner's aim is to manage the mass of the active population each period over the entire time horizon. Precisely, the planner receives unit reward each period if and only if the active population mass ( $\ell_B$  and  $\ell_S$ ) lies in a compact interval  $\mathcal{Y} := [0, x]$  where  $0 < x < 1$ .<sup>1</sup> Practically, this can be considered a capacity floor on the active population that is externally mandated or estimated based on the needs of the public location and activity. If the isolated population mass lies outside this interval, the planner receives zero reward. The planner discounts future periods by a factor  $\delta$  so their total utility is a discounted sum of all future rewards. This utility is a function of the masses  $y^t$  which represent the aggregate choices agents make in response to the induced beliefs  $\bar{p}^t$  in each period. These are generated as a function of the planner's choices  $\{\pi^t\}_{t \geq 1}$  and the randomness in the Markovian dynamics of  $\{\gamma^t\}_{t \geq 1}$ . The planner's utility is evaluated on expectation over both sources of randomness. For any initial belief  $r^1$  and  $\underline{p}^1$ , the planner utility equals:

$$V_{\delta, \mathcal{Y}}(\{\pi^t\}_{t \geq 1}; r^1, \underline{p}^1) = \mathbb{E} \left[ \sum_{t=1}^{\infty} (1 - \delta) \delta^{t-1} \mathbb{I}\{y^t \in \mathcal{Y}\} \right]$$

To determine the planner's optimal signalling mechanism  $\pi^t$  in period  $t$ , we recursively define value functions for the forward utility starting from period  $t$ . We express the current period reward using  $v_{\mathcal{Y}}(\bar{p}^t) := \mathbb{I}\{y^t \in \mathcal{Y}\}$  since the posterior belief in period  $t$  is used by agents to determine their actions and the isolated mass in aggregate. The posterior belief is a function of the planner's mechanism  $\pi^t$  since the distribution over the beliefs  $\bar{p}^t$  that can be induced from  $\underline{p}^t$  is specified by  $\pi^t$  as in (9). Thus, in each period  $t$ , given the current belief  $\underline{p}^t$ , the planner's design problem is to maximize value and determine the optimal signalling mechanism for the given belief. We refer to the optimal mapping of beliefs  $\underline{p}^t$  to signalling mechanisms as the optimal *disclosure* rule  $\pi_*^t \in \Pi$ .<sup>2</sup>

$$V_{\delta, \mathcal{Y}}^t(\underline{p}^t) = \max_{\pi^t \in \Pi} \mathbb{E}[(1 - \delta)v_{\mathcal{Y}}(\bar{p}^t) + \delta V_{\delta, \mathcal{Y}}^{t+1}(\underline{p}^{t+1})] \quad (10)$$

$$\pi_*^t(\underline{p}^t) \in \arg \max_{\pi^t \in \Pi} \mathbb{E}[(1 - \delta)v_{\mathcal{Y}}(\bar{p}^t) + \delta V_{\delta, \mathcal{Y}}^{t+1}(\underline{p}^{t+1})] \quad (11)$$

Observe that the setting we have described is a non-stationary

<sup>1</sup>Note that our design approach can be extended to more general sets of desired isolated masses. We can, with mild adjustment, address unions of intervals but for the sake of exposition we restrict to this family of preferences.

<sup>2</sup>We can use max because  $V_{\delta, \mathcal{Y}}^t$  is Lipschitz over  $\underline{p}^t$ , the expression in braces of (10) is upper hemi-continuous w.r.t.  $\underline{p}^t$  in the weak-\* topology on  $\Pi$ , and  $\Pi$  is compact in that topology. We omit the details as they are standard [13], [5].

Markov decision process where the planner's state is represented by the tuple  $(t, \underline{p}^t)$ . The action set can be represented by the set of mechanisms  $\Pi$ , with rewards  $v_{\mathcal{Y}}(\mu_i^t)$  with probability  $q_i^t$  as dictated by the chosen mechanism  $\pi^t \in \Pi$ , and transitions from states  $(t, \underline{p}^t)$  to states  $(t + 1, \phi_{\Gamma}(\mu_i^t))$  with probability  $q_i^t$ .

## III. EQUILIBRIUM CHARACTERIZATION

We use the solution concept of Bayes-Nash equilibrium to characterize the outcome of agents' choices in period  $t$  upon receiving signal  $i^t$  as generated by the signaling mechanism  $\pi^t = \{(q_i^t, \mu_i^t)_{i \in \mathcal{I}^t}\}$ . We represent the equilibrium mass of isolated agents in period  $t$  by  $y_{\pi^t}^*(i^t)$  which results from all the *myopic* agents simultaneously making their choices under the posterior belief  $\bar{p}^t = \mu_{i^t}^t$  corresponding to  $i^t$ . We can represent the expected infectious cost faced by agents choosing  $\ell_S$  with the posterior belief  $r^t$  and an active population of  $1 - w$  by  $\tilde{\beta}(w; r^t) = c_1(w)(r^t(\theta_H - \theta_L) + \theta_L) + c_2(w)$ . The following result shows that we can exactly characterize the equilibrium in terms of these two quantities. At equilibrium, the agents that choose to be active ( $\ell_S$  and  $\ell_B$ ) will be those that have the largest private benefits from  $G$ . Moreover, those that will choose to be active will either all choose to take booster ( $\ell_B$ ) or all elect to not do so ( $\ell_S$ ).

*Proposition 1:* For any signal  $i^t \in \mathcal{I}^t$  realized by mechanism  $\pi^t$  setting  $\bar{p}^t = \mu_{i^t}^t$ , the equilibrium mass of isolated agents  $y^*(i^t) = m(\bar{p}^t; r^t)$  is given by the unique solution  $w^* \in [0, 1]$  to the following equation:

$$G^{-1}(w) = \min\{\tilde{\beta}(w; r^t), \kappa + (1 - \bar{p}^t E)\tilde{\beta}(w; r^t)\} \quad (12)$$

Furthermore, at equilibrium, agents with private value from being active  $v$  choose  $\ell_B$  if  $v \geq G^{-1}(m(\bar{p}^t; r^t))$  and  $\tilde{\beta}(m(\bar{p}^t; r^t); r^t) \geq \kappa + (1 - \bar{p}^t E)\tilde{\beta}(m(\bar{p}^t; r^t); r^t)$ , choose  $\ell_S$  if  $v \geq G^{-1}(m(\bar{p}^t; r^t))$  and  $\tilde{\beta}(m(\bar{p}^t; r^t); r^t) < \kappa + (1 - \bar{p}^t E)\tilde{\beta}(m(\bar{p}^t; r^t); r^t)$ , and choose  $\ell_R$  otherwise. ■

Leveraging Proposition 1, we can further identify the functional relationship of how the parameters and beliefs governing the model affect the outcome. The following lemmas show that the isolated mass in equilibrium is well-behaved in response to the beliefs over the booster efficacy and the infectiousness of the disease.

*Lemma 1:* For any  $r^t \in [0, 1]$ , the map  $m(\bar{p}^t; r^t)$  is continuous, bounded and weakly decreasing in  $\bar{p}^t$ . Moreover,  $m(\bar{p}^t; r^t)$  is Lipschitz continuous in  $\bar{p}^t$ .

*Lemma 2:* For any  $\bar{p}^t \in [0, 1]$ , the map  $m(\bar{p}^t; r^t)$  is continuous, bounded and weakly increasing in  $r^t$ . Moreover,  $m(\bar{p}^t; r^t)$  is Lipschitz continuous in  $r^t$ .

These lemmas establish that the mapping between the belief on the  $\theta^t$  and  $\gamma^t$  and the isolated mass are continuous, monotone mappings. We depict an example of such a function  $m$  in Figure 1. The continuity of  $m$  implies that we can invert from isolated agent masses  $y_i^*$  to beliefs. Namely, observe that we can define  $\mathcal{W}(r^t) = \{p : m(p, r^t) \in \mathcal{Y}\}$ . Since  $\mathcal{Y} = [0, x]$  and  $m$  is weakly decreasing in  $p$ , either  $m(1, r^t) > x$  and  $\mathcal{W}(r^t) = \emptyset$ , or there exists some  $W(r^t) := \inf\{\mathcal{W}(r^t)\}$  in  $[0, 1]$  such that  $\mathcal{W}(r^t) = [W(r^t), 1]$ . For convenience, we let

$W(r^t) = \infty$  if  $\mathcal{W}(r^t) = \emptyset$ . The following lemma establishes that  $W(\cdot)$  also is weakly increasing.

*Lemma 3:*  $W(r)$  is weakly increasing in  $r$  and locally Lipschitz continuous over the interior domain  $\text{dom}(W) := \{r : 0 < W(r) < 1\}$ .

The previous lemma shows that the relationship between the beliefs about the infectiousness of a disease and the set of beliefs we need to induce on the booster efficacy for the planner to achieve reward is monotone.

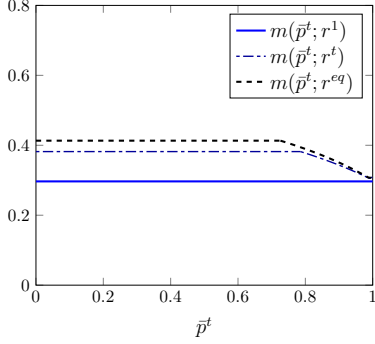


Fig. 1:  $m(\bar{p}^t; r)$  for different values of  $r^t$ .  $G = \text{Unif}[0, 10]$ ,  $E = \frac{99}{100}$ ,  $\theta_L = 4$ ,  $\theta_H = 16$ ,  $r^1 = \frac{1}{6}$ ,  $r^t = \frac{1}{2}$ ,  $r^{eq} = \frac{2}{3}$ ,  $\beta(y^t; \theta^t) = \theta^t (y^t)^2$ .

Moreover, Lemma 3 ensures that as the severity of the disease increases, our ability to induce beliefs on the booster to achieve a desirable outcome becomes more limited since  $\mathcal{W}(r)$  shrinks as  $r$  increases. This is evident in Figure 1, as the domain of  $\bar{p}^t$  that map to any isolated mass  $m(\bar{p}^t; r^t)$  in  $[0, x]$  contracts as  $r^t$  increases.

The local Lipschitz continuity of  $W(r)$  guarantees that the set of beliefs in period  $t$ ,  $\mathcal{W}(r^t) = [W(r^t), 1]$  that we need to induce in the booster effectiveness  $\bar{p}^t$  to achieve an isolated mass  $y_{\pi^t}^*(i^t)$  in  $\mathcal{Y}$  converges over time. If  $\mathcal{W}(r^t)$  becomes  $\emptyset$  eventually, we will not have this closure and hence the planner can never induce beliefs that map to the required active population in the long-run. Likewise, if  $W(r^t)$  converges to 0 or 1, trivial mechanisms become necessary in the long-run and  $W$  will not be locally Lipschitz at these limit points. Hence, in this paper we will ignore these edge cases where  $W(r^{eq}) = 0, 1$ , or  $\infty$ . With this assumption, we can show that  $W$  preserves the convergence we know we will observe in the belief  $r^t$ .

*Assumption 1:*  $0 < W(r^{eq}) < 1$

*Corollary 1:* Under Assumption 1,  $\lim_{t \rightarrow \infty} W(r^t) \rightarrow W(r^{eq})$ .

## IV. OPTIMAL DISCLOSURE RULE

### A. Structure of Value Functions

In order to characterize the optimal mechanism, we first identify structural properties of the value functions specified in (10) that are maximized when using  $\pi_*^t$ . In particular, the shape of the value function  $V_{\delta, \mathcal{Y}}^t$  provides insight into the structure of  $\pi_*^t$ .

We first show the following proposition which establishes the concavity of the value function of the planner with respect to the population's belief over the booster's efficacy.

*Proposition 2:* For all  $t \geq 1$ ,  $V_{\delta, \mathcal{Y}}^t(p^t)$  is concave in  $p^t$ .

This result establishes a critical trade-off between the planner disclosing information (i.e. splitting across multiple posterior beliefs  $\bar{p}^t$ ) to enhance their current stage reward  $v_{\mathcal{Y}}(\bar{p}^t)$  and the cost of this information disclosure on the future value functions  $V_{\delta, \mathcal{Y}}^{t+1}$ . It suggests that if the planner is already maximizing their current stage reward with the population's current belief  $p^t$ , there is no need to divulge additional information since it could lead to worse continuation payoffs in future periods due to the concavity of future value functions. To this end, we motivate the non-informative mechanism,  $\pi_{NI}(p^t) = \{(1, p^t)\}$ , which conveys no information and thus does not affect the belief.

We consequently show in the following proposition that  $\pi_*^t(p^t) = \pi_{NI}(p^t)$  whenever  $v_{\mathcal{Y}}(p^t) = 1$ . The non-informative mechanism  $\pi_{NI}(p^t)$  can be constructed by choosing  $\mathcal{I}^t = 1$  as no information is conveyed on seeing the only possible signal and on employing this mechanism in period  $t$ ,  $\bar{p}^t = p^t$  with probability one. Similarly, if the set of beliefs the planner seeks to induce with the signalling mechanism  $\mathcal{W}(r^t) = \emptyset$  (i.e.  $W(r^t) = \infty$ ), then regardless of the initial belief  $p^t \in [0, 1]$ , the non-informative mechanism maximizes the stage reward as no reward is possible. Therefore,  $\pi_*^t(p^t) = \pi_{NI}(p^t)$  as the non-informative mechanism is optimal.

*Proposition 3:* For all  $t \geq 1$ , if  $p^t \geq W(r^t)$  or if  $W(r^t) = \infty$ , then  $\pi_*^t(p^t) = \pi_{NI}(p^t)$ .

Next, we further extend this insight by highlighting that the current stage reward  $v_{\mathcal{Y}}(\bar{p}^t)$  is binary and consequently necessitates only two signals at optimality. Namely, the optimal signalling mechanism should generate two posterior beliefs by using only two signals  $\mathcal{I} = \{a, b\}$  – one that will yield an outcome that maximizes the current period reward (i.e.  $\mu_a^t \geq W(r^t)$ ), and one that yields an outcome that does not achieve a current period reward ( $\mu_b^t < W(r^t)$ ). Any additional information revelation is suboptimal by Proposition 4. We standardize the posteriors so that for all  $t$ ,  $\mathcal{I}^t = \{a, b\}$  and  $\mu_b^t < W(r^t)$ ,  $\mu_a^t \geq W(r^t)$ .

*Proposition 4:* For all  $t \geq 1$ , if  $p^t < W(r^t)$ ,  $\pi_*^t(p^t)$  is based on  $\mathcal{I}^t = \{a, b\}$  with  $\mu_b^t \leq p^t < W(r^t)$  and  $\mu_a^t \geq W(r^t)$ .

Since the optimal disclosure rule specifies that a mechanism need split into only two posteriors when  $p^t < W(r^t)$ , we need only specify the two posterior means  $\mu_b^t$  and  $\mu_a^t$  in any period  $t$  for any given  $p^t$ . Observe that  $q_b^t$  and  $q_a^t$  are implicitly defined as a result since  $q_b^t + q_a^t = 1$  and  $q_b^t \mu_b^t + q_a^t \mu_a^t = p^t$ .

We next analyze the recursive relationship between value functions. We define the *posterior value function* in period  $t$  to be  $\bar{V}_{\delta, \mathcal{Y}}^t(\bar{p}^t) := (1 - \delta)v_{\mathcal{Y}}(\bar{p}^t) + \delta V_{\delta, \mathcal{Y}}^{t+1}(\phi_{\Gamma}(\bar{p}^t))$ . Observe that these functions represent the value-to-go once the signal  $i^t$  has already been realized. By the known Markovian update between  $p^{t+1} = \phi_{\Gamma}(\bar{p}^t)$ :

$$\begin{aligned} V_{\delta, \mathcal{Y}}^t(p^t) &= \max_{\pi_t \in \Pi} \mathbb{E}[(1 - \delta)v_{\mathcal{Y}}(\bar{p}^t) + \delta V_{\delta, \mathcal{Y}}^{t+1}(\phi_{\Gamma}(\bar{p}^t))] \\ &= \max_{\pi_t \in \Pi} \mathbb{E}[\bar{V}_{\delta, \mathcal{Y}}^t(\bar{p}^t)] \end{aligned} \quad (13)$$

Thus, each point on the value function  $V_{\delta,y}^t(p^t)$  is equal to largest possible linear average of points on the posterior value function. Hence,  $V_{\delta,y}^t(p^t)$  must be the *concave envelope* of the *posterior value function*  $\bar{V}_{\delta,y}^t$ . Following [23], we denote  $k(p)$  as the concave envelope of  $V_{\delta,y}^t(p)$  if  $k(p) \geq V_{\delta,y}^t(p)$  for all  $p \in [0, 1]$  satisfying the condition that  $\max \{ \bar{V}_{\delta,y}^t(p) - k(p), k''(p) \} = 0$  for all  $p$ .

*Proposition 5:*  $V_{\delta,y}^t = \text{conc}(\bar{V}_{\delta,y}^t)$  where  $\text{conc}(\cdot)$  denotes the concave envelope.

The previous proposition provides us with another formulation to compute  $V_{\delta,y}^t$ . We observe that this additional formulation allows us to characterize the optimal disclosure rule based on the shape of the value function  $V_{\delta,y}^t$ , as on intervals where  $V_{\delta,y}^t$  is linear, the optimal disclosure rule prescribes a mechanism  $\pi_*^t$  that splits over the two posterior beliefs at the endpoints of this linear interval.

*Lemma 4:* For all  $t \geq 1$ ,  $p < W(r^t)$ , suppose that  $\pi_*^t(p)$  splits on  $\mu_b^t \leq p < W(r^t)$  and  $\mu_a^t \geq W(r^t)$ , then  $V_{\delta,y}^t(p^t)$  is linear on  $[\mu_b^t, \mu_a^t]$ . Furthermore, for all  $\mu_a^t \leq p \leq \mu_b^t$ ,  $\pi_*^t(p)$  splits on  $\mu_b^t$  and  $\mu_a^t$ .

This lemma provides us with a powerful insight into the optimal disclosure rule. Under the optimal disclosure rule  $\pi_*^t(p^t)$ , the posteriors  $\mu_b^t$  and  $\mu_a^t$  are fixed across different values of  $p^t < W(r^t)$  since  $V_{\delta,y}^t$  will have intervals where  $V_{\delta,y}^t$  is concave and coincides with  $\bar{V}_{\delta,y}^t$  – here, the non-informative mechanism is optimal. Otherwise,  $V_{\delta,y}^t$  will have intervals where it is linear and the posteriors chosen by the optimal mechanism  $\pi_*^t$  ( $\mu_b^t$  and  $\mu_a^t$ ) are identical across all  $p^t$  in that interval. There can only be one such linear segment since to the left of and to the right of  $W(r^t)$ ,  $\bar{V}_{\delta,y}^t$  is already concave so no linear segment is added wholly within  $[0, W(r^t))$  or  $[W(r^t), 1]$ . Two such segments cannot exist since they would necessarily coincide which leads to a contradiction. Hence, the one set of posteriors  $\mu_b^t$  and  $\mu_a^t$  is sufficient to describe  $\pi_*^t$ .

To see this more concretely, observe that for the non-informative mechanism must be optimal for all  $p^t \geq W(r^t)$ ,  $\text{conc}(\bar{V}_{\delta,y}^t)$  must coincide with  $\bar{V}_{\delta,y}^t$  on that interval. However, as depicted in Fig. 2 and in Equation (13), there is a sudden jump in the posterior value function  $\bar{V}_{\delta,y}^t$  at  $p^t = W(r^t)$ . Therefore, by definition of the concave envelope  $V_{\delta,y}^t = \text{conc}(\bar{V}_{\delta,y}^t)$ , it must incorporate a non-trivial, *maximal* interval where it is linear and *strictly* above the posterior value function  $\bar{V}_{\delta,y}^t$  – in fact, by Proposition 4 and Lemma 4, one end of the linear segment must lie in  $\mathcal{W}(r^t)$  and the other end below it in order to cross this discontinuity jump and concavify  $\bar{V}_{\delta,y}^t$ . Thus, in order for the posterior value function to coincide with its concave envelope for  $p^t \geq W(r^t)$ , the linear segment of  $\text{conc}(\bar{V}_{\delta,y}^t)$  must end precisely at the posterior belief  $W(r^t)$ . However, by Lemma 4, this right endpoint of the interval where  $V_{\delta,y}^t$  is linear is the second posterior  $\mu_a^t$ .

*Proposition 6:* If  $W(r^t) \leq 1$  and  $p^t < W(r^t)$ , there exists an optimal mechanism  $\pi_*^t(p^t)$  with  $\mu_a^t = W(r^t)$  for all  $t$ . To summarize the insights on the optimal disclosure rule thus far: (i) if  $p^t \geq W(r^t)$  or  $W(r^t) = \infty$ , we can choose the optimal signalling mechanism  $\pi_*^t(p^t) = \pi_{NI}(p^t)$ ; (ii)

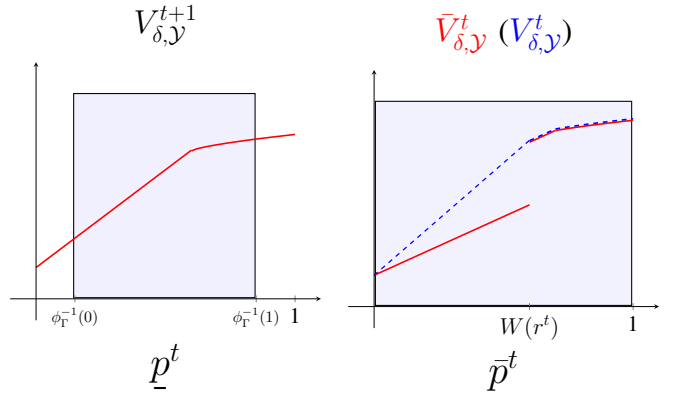


Fig. 2:  $V_{\delta,y}^{t+1}$  (red, on left),  $\bar{V}_{\delta,y}^t$  (red, on right), and  $V_{\delta,y}^t = \text{conc}(\bar{V}_{\delta,y}^t)$  (blue, on right).

if  $p^t < W(r^t)$ , we need to only specify the posterior belief  $\mu_b^t < p^t$  as an optimal signalling mechanism exists that has at most two posteriors with one of them being  $\mu_a^t = W(r^t)$ . In fact,  $\mu_b^t$  is fixed across the disclosure rule  $\pi_*^t$  since by Lemma 4, if  $p^t \leq \mu_b^t$  or if  $p^t \geq W(r^t)$ , then  $\pi_*^t(p^t) = \pi_{NI}$ , and otherwise if  $\mu_b^t < p^t < W(r^t)$ , the disclosure policy chooses  $\pi_*^t(p^t) = \{(\frac{W(r^t)-p^t}{W(r^t)-\mu_b^t}, \mu_b^t), (\frac{p^t-\mu_b^t}{W(r^t)-\mu_b^t}, W(r^t))\}$ . Hence, all that remains to determine the optimal disclosure rule across time is to solve for the optimal sequence  $\{\mu_b^t\}_{t \geq 1}$ .

#### B. Optimality of Greedy Disclosure Rule

Our results in Section IV-A establish that the planner seeks to disclose as little information as they can due to the concavity of their value functions. We now formalize the *greedy* disclosure rule at time  $t$  which simultaneously provides as little information as possible and maximizes the current period reward. Observe that this is achieved by splitting into posterior beliefs  $\mu_b^t = 0$  and  $\mu_a^t = W(r^t)$ , as intuitively  $W(r^t)$  is the *closest* belief we can induce to achieve the current period reward. Moreover, by pushing  $\mu_b^t$  to 0, we maximize the probability  $q_a^t$  that we induce a posterior belief leading to a reward.<sup>3</sup>

*Definition 1:* The greedy disclosure rule  $\pi_{\dagger}^t(p^t)$  is such that if  $p^t \geq W(r^t)$  or  $W(r^t) = \infty$  then  $\pi_{\dagger}^t(p^t) = \pi_{NI}(p^t)$ . If  $p^t < W(r^t)$ ,  $\pi_{\dagger}^t(p^t) = \{(\frac{p^t}{W(r^t)}, W(r^t)), (\frac{W(r^t)-p^t}{W(r^t)}, 0)\}$ .

We seek to show that the greedy disclosure rule will become optimal after some finite amount of time. We can directly comment on the quality of the greedy disclosure rule against the optimal disclosure rule as specified by the optimal sequence  $\{\mu_b^t\}_{t \geq 1}$ . Observe the greedy disclosure rule chooses posterior beliefs 0 and  $W(r^t)$  whenever  $p^t < W(r^t) \leq 1$ .

Here, we only focus on the most interesting case where  $p^{eq} < W(r^{eq})$ . Observe that, if  $p^{eq} > W(r^{eq})$ , the Markovian drift on the beliefs under no information will translate  $p^1$  to  $p^t = \phi_{\Gamma}^{(n-1)}(p^1)$  and, that for some finite  $n$ ,  $p^{t'} \geq W(r^{eq})$  for all  $t' \geq n$ . Hence, without any

<sup>3</sup>This mechanism is unique and exists strictly because the state space of the uncertainty set is of size two.

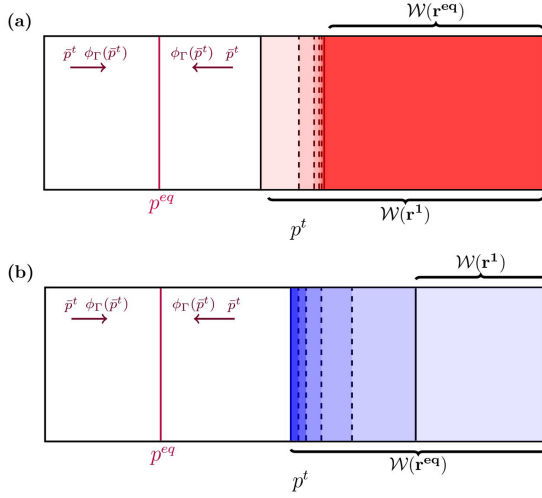


Fig. 3: Diagram of the two cases described in Theorem 1.

intervention, the planner will eventually begin using no information and collecting the period rewards for perpetuity. The planner's problem when  $p^{eq} < W(r^{eq})$  is consequently of more direct interest, and we show that the greedy disclosure rule eventually becomes optimal in finite time. In fact, we can show that the greedy disclosure rule eventually becomes optimal once the planner cannot guarantee a reward in the next period by providing no information when he does not earn a reward in the current period by doing so (i.e. we require  $\phi_\Gamma(W(r^t)) \leq W(r^{t+1})$ ).

*Theorem 1:* If  $p^{eq} < W(r^{eq})$ , subject to Assumption 1, the following holds:

- (a) If  $r^t$  is increasing, then  $\pi_*^t = \pi_\dagger^t$  for all  $t > t_\dagger$  where  $t_\dagger = \sup \{ t : W(r^t) < p^{eq} \} < \infty$ .
- (b) If  $r^t$  is decreasing, then  $\pi_*^t = \pi_\dagger^t$  for all  $t > t_\dagger$  where  $t_\dagger = \sup \{ t : \phi_\Gamma(W(r^t)) > W(r^{t+1}) \} < \infty$ .

We depict both cases graphically in Fig. 3. The theorem affirms that the optimal disclosure rule for a planner eventually becomes the greedy disclosure rule, which seeks to minimize information sharing while maximizing the current period reward. Specifically, this property holds when the beliefs  $p^t$  move away from the equilibrium belief set  $W(r^{eq})$ . In practice, when individuals grow more pessimistic about the booster's efficacy over time and converge to a belief that is more pessimistic than required for the desired outcome, the planner best maximizes their utility by greedily inducing the belief with maximum probability that achieves the *easiest* outcome in  $\mathcal{Y}$  (i.e. achieving the threshold  $y_t^* = x$ ).

The intuition of the optimality of the greedy mechanism is as follows. If a posterior  $\mu_b^t$  slightly larger than 0 is chosen, the advantage is that if signal  $b$  is drawn by the mechanism, the planner starts the next period at  $p^{t+1} = \phi_\Gamma(\mu_b^t)$  where  $\phi_\Gamma(0) < \phi_\Gamma(\mu_b^t) < W(r^t)$ . However, because beliefs further away from  $p^{eq}$  converge to  $p^{eq}$  faster under Markovian dynamics,  $|\phi_\Gamma(\mu_b^t) - \phi_\Gamma(0)| \leq |\mu_b^t - 0|$ , so the positive effect is counteracted by the more rapid mixing of the Markov chain. The key disadvantage of choosing  $\mu_b^t > 0$  is that we are limited to smaller weights  $q_a^t$  on beliefs  $\mu_a^t$  that induce current period rewards. Therefore, intuition suggests that we

should maximize the current period reward since not doing so comes at a minimal cost.

Observe that the greedy disclosure rule will only provide informative signals when  $p^t < W(r^t)$  and since  $p^{eq} \leq W(r^{eq})$ , eventually the beliefs  $\phi_\Gamma(W(r^t))$  will always shift out of the beliefs necessary in the next period  $\mathcal{W}(r^{t+1})$ . If  $p^t < W(r^t)$ , the greedy disclosure rule will optimistically generate posterior beliefs  $\bar{p}^t = W(r^t)$  in any period  $t$ , and then the Markov chain updates the next period's initial belief to  $p^{t+1} = \phi_\Gamma(W(r^t)) \notin \mathcal{W}(r^{t+1})$ . Thus, every period for perpetuity, the planner must provide informative signalling. This mimics something closer to practical settings where planners cannot abstain from providing information for perpetuity.

The previous theorem does not provide guidance on the optimal strategy  $\pi_*^t$  in the interim when  $t \leq t_\dagger$ . Generally, as we discuss in more detail in the proof of Theorem 1, since  $t_\dagger$  is finite, this can be solved efficiently using dynamic programming since the value functions  $V_{\delta, \mathcal{Y}}^{t_\dagger}$  can be easily computed and backtracking at each point in time  $t \leq t_\dagger$ , we need only consider a finite number of possible  $\mu_b^t$ .

The main technical challenge preventing stronger guarantees on the optimality of greedy disclosure is that  $W$  is only locally Lipschitz without added structure imposed on  $G$  (which simplifies  $m$  and consequently  $W$ ). Therefore, the relationship between the beliefs required in the next period compared to the current period is monotone, but lacking further insight. However, the following proposition shows that under some mild regularity on  $\delta$  and the mixing rate imposed by  $\phi_\Gamma$ , we can guarantee that the greedy disclosure rule is optimal even in the interim periods  $t \leq t_\dagger$ .

*Proposition 7:* If  $\delta \leq \min_{j \leq t \leq t_\dagger} \frac{\phi_\Gamma^{-1}(W(r^{t+1}))}{W(r^t)}$ , then  $\pi_*^t = \pi_\dagger^t$  for all  $t \geq j$ .

Specifically, this proposition states that if the planner is sufficiently impatient and discounts significantly, then the greedy disclosure rule is optimal. This is intuitive as the planner should be more willing to take a gamble of only securing a current period reward with the maximal probability  $q_a^t$  rather than ensuring a reward next period by choosing non-informative signalling. Likewise, suppose that  $r^t$  is decreasing, then observe that if  $\phi_\Gamma$  mixes faster (i.e.  $1 - \nu_\Gamma \zeta_\Gamma - \zeta_\Gamma$  is small) then  $\phi_\Gamma^{-1}(W(r^{t+1}))$  becomes larger. Therefore, when  $\phi_\Gamma$  mixes fast, the possibility of the planner guaranteeing rewards in the next period dissipates since the population belief on the booster quickly moves close to  $p^{eq}$  which is outside  $\mathcal{W}(r^t)$ . Therefore, in practice, the viability of the greedy disclosure rule is most robust when the population beliefs quickly converge to stationary beliefs and planners are also operating on shorter time horizons.

## V. NUMERICAL STUDY

We present a brief numerical comparison of the total discounted reward  $\sum_{t=1}^{\infty} \delta^{t-1} (1 - \delta) v_{\mathcal{Y}}(\bar{p}^t)$  that the planner accrues under the greedy disclosure rule  $\pi = \pi_\dagger$  as opposed to under the non-informative disclosure rule ( $\pi^t = \pi_{NI}$  for all  $t$ ) and under the fully informative disclosure rule ( $\pi^t = \pi_{FI}$  for all  $t$  where

$\pi_{FI} = \{(p^t, 1), (1 - p^t, 0)\}$ . Fully-informative disclosure directly reveals the state of  $\gamma^t$  in each period  $t$ . In Figure 4, we plot the numerical estimates of  $\tilde{V}(p^1; \pi)$  under each of the three disclosure rules. Appealing to Proposition 7, the optimal disclosure rule coincides with the greedy disclosure rule in both these settings for  $t \geq 1$ , so  $\tilde{V}(p^1; \pi_{\dagger})$  is equal to the value function  $V^1(p^1)$ . In both settings presented  $p^{eq} < W(r^{eq})$ , but in case (i)  $r^t$  is increasing and in case (ii)  $r^t$  is decreasing. The greedy disclosure rule strictly dominates both benchmarks for all initial beliefs  $p^1$ . Moreover, we can demonstrate that the greedy disclosure rule not only outperforms the benchmarks on expectation but can also do so on an instance level [19]. By design, the greedy disclosure rule is minimizing information sharing *subject to maximizing the current period reward*, so the greedy disclosure rule also outperforms these two mechanisms by achieving weakly higher period rewards in every period. Observe that the greedy value function is piecewise linear in the initial belief  $p^1$ , as the strategy involves splitting across  $\mu_b^1 = 0$  and  $\mu_a^1 = W(r^1)$ . The dominance of the greedy disclosure rule across both settings in Fig. 4 affirm the value of greedy information disclosure.

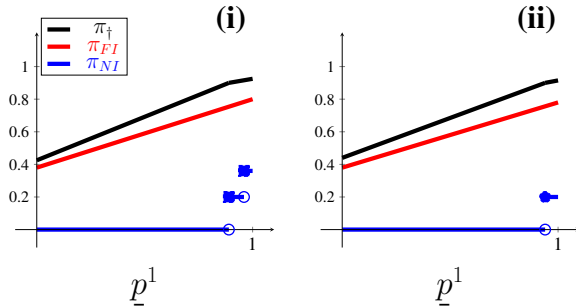


Fig. 4: Discounted sum of rewards,  $\tilde{V}(p^1; \pi)$ , under the same parameters as Fig. 1 with  $\delta = 0.8$ ,  $p^{eq} = 0.2$ ,  $x = 0.35$  and  $(1 - \nu_{\Theta}\zeta_{\Theta} - \zeta_{\Theta}) = (1 - \nu_{\Gamma}\zeta_{\Gamma} - \zeta_{\Gamma}) = 0.8$ . For case (i),  $r^1 = 0.5$ ; and for case (ii),  $r^1 = 0.8$ .

## VI. CONCLUDING REMARKS

In this paper, we introduced a model to study optimal dynamic information disclosure over booster efficacy amid non-stationary disease infectiousness. Our model captures two novel features: (a) heterogeneous agents making strategic decisions to trade-off activity against the infectious risks with or without a booster; (b) a non-stationary environment where the planner's value from inducing certain beliefs over booster efficacy varies as the infectiousness changes over time. We provided a complete description of the strategic equilibrium as a function of beliefs over booster efficacy. We also show that greedy disclosure eventually coincides with the optimal disclosure rule. Future work should examine how the optimal disclosure rule changes when the changes in disease infectiousness and booster efficacy are correlated.

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