Path-Dependent Equilibrium and Polarization in Public Responses to a Pandemic*

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Abstract

A model of dynamic game among heterogeneous agents in response to a pandemic is developed, in which the agents' communicable-activity decisions collectively affect the *transmission risk* of the environment and individually affect each agent's utility flows along with their *probability of exposure* to the transmission risk. Agents' equilibrium actions exhibit (1) path-dependent dynamics when their probability of asymptomatic infection is positive and (2) *polarization* when the hazard rate of exposure probability exceeds their degree of risk aversion. Comparative statics analyses show that the levels of agents' equilibrium communicable activity are nonincreasing in their discount factor, their probability of showing symptoms upon infection, and their expectation of suffering upon showing symptoms. If the virus persists, then the model predicts that the only long-run equilibrium outcome is herd immunity.

Keywords: heterogeneous agents; path-dependent equilibrium; hazard rate; asymptomatic infection; equilibrium polarization.

JEL Classification: C73; D01; D82

"It is not about the virus. It is about us."
— EURONEWS.

1 Introduction

Understanding the collective behavior of the public regarding issues that impact the entire society is a primary area of interest to social scientists. The COVID-19 pandemic is one such issue—it has imposed on every economic agent a decision-making problem: To what extent should one voluntarily take preventive measures, such as wearing a face mask in public places, social distancing, avoiding unnecessary travels, and so on to reduce the risk of infection? Evidence from the extensive YouGov survey, comprising 189,955 observations across 44 metropolitan areas in 23 countries, reveals a widespread puzzling phenomenon: most agents, whenever possible, either take maximal measures or do not take any preventive measure at all. The polarized pattern has been documented in various dimensions including social distancing, reduced social activities, and avoidance of physical contact and crowded places. [1,2] Despite these empirical regularities, the existing literature has been unable to offer a rational reason for the polarized public responses to the pandemic.

To improve our understanding of the polarized behavior,[3] in this study we take

[1]Empirically, we construe "polarization" as a phenomenon that the distribution of people's preferences/actions for disease prevention exhibits two peaks: one centered close to the lower end (maximal prevention) and the other to the upper end (life as usual).

[2]Data from Citizens' Attitudes Under Covid-19 Project, a 4-wave tracking survey conducted in 11 countries, reveals that, on average, approximately half of the population opted for the extreme ends of the preventive measures spectrum. Over time, individual choices exhibit a discontinuous jump from strict protective measures to minimum protection instead of gradual transition, as the transitional probabilities towards minimum protection are significantly higher than those towards its adjacent choices (Han et al., 2023b). Counterfactual analysis also supports the existence of polarized protective behaviors based on changes in face-to-face socializing time in the U.S. (Han et al., 2023a).

[3]Formally, we say that a dynamic equilibrium exhibits polarization if in every period, the agents' optimal actions belong to two disjoint sets connected to the lower and upper end of the feasible action set, respectively, and no equilibrium action exists in the interval separating the two sets.

a serious look at individuals' rational choices against the backdrop of a pandemic. We develop a new dynamic public response model that incorporates transmission uncertainty, multi-dimensional heterogeneity in agents' types and preferences, and the possibility of asymptomatic infection. We model the economic agents as playing a dynamic noncooperative game, optimally trading off their levels of communicable activity (or actions) for reductions of the probability of being exposed to the transmission risk of the environment—while updating information rationally and anticipating other agents to do so as well. A key assumption in our theoretical investigation is that the exposure probability, being a function of the levels of communicable activity, exhibits memoryless dynamics in that the instantaneous probability of getting exposed to transmission risk is the same regardless of the level of one's past activities—conditional on them having not been exposed to risk. This assumption marks a significant departure from the existing studies, most of which assume that the infection probability is linear in agents' actions (see Section 2).

Our analysis reveals that the hazard rate and the shape of the agents' utility function jointly affect equilibrium behavior. We find that whenever the hazard rate exceeds some agents' Arrow-Pratt degree of absolute risk aversion, a polarized equilibrium arises (see Figure 2 in Section 4.2). The degree of polarization, when measured by the distance between the two disjoint sets of optimal actions, increases as the hazard rate increases. These results do not rely on behavioral biases or bounded rationality, nor require polarization of underlying preferences or beliefs. Instead, we find that polarization can be rooted in individual rationality and occur under any atomless distribution of agents' types.

In our dynamic model, engaging in more communicable activities today increases the probability of asymptomatic infection and subsequent immunity in the future. Technically, the possibility of being unknowingly infected in the past leads to a sequence of path-dependent beliefs about one's probability of being immune for the active agents, which significantly complicates our analysis (see Theorem 1). By transforming the problem into a mathematically equivalent one, we find a way to simplify the problem and obtain sharp characterizations of the dynamic equilibrium (see Theorem 2). We show that each active agent's marginal period-t expected payoff can be expressed succinctly as

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - g'(a_t)M_t \tag{1}$$

where $a_t \in [0,1]$ denotes an agent's action, $v(a_t)$ the agent's period-t utility, $g(a_t)$ the agent's exposure probability to the transmission risk, and M_t (> 0) a function(al) whose value depends on the underlying stochastic process and several endogenous state variables, such as the agent's type, continuation payoff, and equilibrium future actions. Thanks to the principle of optimality for stochastic dynamic programming, a crucial property of M_t is that it can be analyzed as though it is independent of a_t. Thus the marginal cost and benefit of action today on the continuation payoff is proportional to its marginal effect on the infection probability today; the dynamic effects will not distort the shape of the overall expected payoff function.

An important insight emerges then, from (1), that the type of polarization as presented in Theorem 2, and pictured in Figure 2 in Section 4.2, is a consequence of the ratio $v'(a)/g'(a)$ being increasing in a, and that this condition holds as long as the hazard rate λ is sufficiently large, i.e., $\lambda = -\frac{g''}{g'} > -\frac{v''}{v'}$. When $v'(a)/g'(a)$ is increasing, g' declines faster than v' as a increases. Consequently, whenever $\frac{\partial V_t}{\partial a_t} \geq 0$ at some action $c \in [0,1)$, the marginal benefit of increasing a_t at all action levels $a_t > c$ exceeds the expected marginal cost and therefore no equilibrium actions can be found in the interval $[c,1)$. In Corollary 2, we show that the above insight does not rely on g being memoryless and v being DARA or CARA. The conclusions of Theorem 2 hold as long as $\frac{v''}{v'} - \frac{g''}{g'}$ has a single-crossing property that it crosses 0 at $c \in [0,1)$ from below. The type of problems involving marginal expected payoffs of the form in (1) need not be confined to a pandemic context. The insight obtained from this study can be useful for understanding polarization of equilibrium decisions in other situations.

Social polarization, in general, has been known to cause unrest and instability.[4] Understanding the causes of such polarized behavior is important not only for comprehending the far-reaching implications of the COVID-19 crisis in its aftermath, but also sheds light on other observed polarized behavior. Our paper contributes to this line of discussion by offering an explanation for polarized responses that does not rest on polarization of underlying preferences or the action space.

In a nutshell, the present study has four novel aspects of contributions:

[4] According to the Armed Conflict Location & Event Data Project (ACLED), as of March 4, 2022, there had been 61,830 pandemic-fueled (violent) demonstrations, public protests, or riots around the globe.

i showing new insights into polarized public behavior by leveraging on the hazard rate of the exposure probability in an epidemiological context;

ii offering a novel approach, a dynamic game with Bayesian updating of information, to the analysis of epidemiological models;

iii modelling explicitly path-dependent beliefs, with a tractable analysis of path-dependent equilibria;

iv featuring three-dimensional heterogeneous agents together with novel comparative statics results.

In Section 2, we discuss related literature along these dimensions. Section 3 presents the pandemic-response model. Section 4 presents the main results of the analysis and Section 5 concludes. The Appendix contains all proofs.

2 Related Literature and New Results

Strategic individual responses. The pandemic literature mostly adopts the SIR (susceptible, infected, recovered or removed) approach, building upon the seminal work by Kermack and McKendrick (1927). Recent studies have extended this framework by incorporating individual preventive behaviors (Carnehl et al., 2023; Dasaratha, 2022; Baril-Tremblay et al., 2021; Toxvaerd, 2020; McAdams et al., 2023; Farboodi et al., 2021; Egorov et al., 2021; Keppo et al., 2021; Javadi et al., 2021). Their common theme is to study how endogenous preventive actions impact contagion dynamics, such as the trajectories of infection rate and prevalence, and suggest different policy interventions.[5] We instead aim to enrich our understanding of the vast difference in public choices by relaxing various key assumptions in previous literature. A notable distinction here is that our model may entail sharp polarization in public responses, regardless of the population composition. This finding sets

[5]See also Alvarez et al. (2020); Atkeson (2020); Bethune and Korinek (2020); Kremer (1996); Boucekkine and Laffargue (2010); Chakraborty et al. (2010); Chen (2012); Fenichel (2013); Toxvaerd (2019). Another strain of literature studies the macroeconomic consequences of the social distancing incentive (Eichenbaum et al., 2021; Jones et al., 2021; Bhattacharya et al., 2021; Forero-Alvarado et al., 2021).

our study apart from previous research, which predominantly focuses on interior solutions.[6]

Infection probability. In the behavioral SIR literature, the infection probability is often modeled as a product of three components: a constant transmission rate, individual activity, and the aggregate activity of the infected population. As such, individual actions contribute linearly to the infection probability. Keppo et al. (2021) and Javadi et al. (2021) propose a more general filter function $f(v) = (1+v)^{-\gamma}$ to capture a nonlinear relationship between individual avoidance effort v and the infection rate, in which γ represents a constant filter elasticity indicating that a one percent increase in the avoidance effort reduces γ percent of the infection rate.[7] Acemoglu et al. (2023) also derives an infection probability function that is concave in the individual action as a result of an endogenous contact network.[8] However, both papers assume the agents are myopic, which prevents them from considering aspects i and iv as studied in our model. In addition, different types of dynamic equilibrium are distinguished in our model based on a more natural concept, the hazard rate of the exposure-probability function, rather than elasticity. Our public-response model further generalizes the SIR model in two additional aspects. First, the transmission rate follows a Markov process, which can fluctuate due to unknown factors such as virus mutations while depending endogenously on the equilibrium actions of the active agents. Second, we allow symptomatic and asymptomatic infections to affect

[6]While many empirical studies have examined the differences in public responses to the pandemic, few dig into the distribution of these behaviors. According to Han et al. (2023b) and Han et al. (2023a), the polarization in public response distribution is observed in a good number of countries that vary dramatically in the level of political polarization. As such, our explanation, based on natural conditions on the exposure-probability function, could be more plausible than the alternative that polarization of responses is a mere reflection of polarization in underlying preferences or beliefs (e.g., political views as in Allcott et al. (2020)).

[7]These authors model the relationship in a way that each agent minimizes a convex objective function, leading to a unique interior solution.

[8]There are other conceivable ways to extend the canonical SIR models in this dimension. For example, Acemoglu et al. (2020) generalizes this "matching technology" by allowing for a more flexible degree of "increasing returns to scale" in the last two components. Dasaratha (2022) adds group-specific transmission rate in a binary group environment. Network properties are also at play in Bouveret and Mandel (2021) and Karaivanov (2020).

the transmission risk of the environment and the infection probability differently.

Asymptomatically infected agents. While the possibility of asymptomatic infections is well recognized in the epidemiological-economic literature (Kremer, 1996; Gersovitz and Hammer, 2003; Brauer et al., 2008; Acemoglu et al., 2021; Farboodi et al., 2021; Baril-Tremblay et al., 2021; Dasaratha, 2022; Keppo et al., 2021; Javadi et al., 2021), most studies do not track a path-dependent belief over asymptomatic infection and subsequent immunity. A notable exception is the work of Baril-Tremblay et al. (2021) where the population is divided into two types: a fixed proportion of agents do not show symptoms after infection (asymptomatic type) while the rest do (severe type). The authors explicitly characterize the evolution of agents' beliefs and examine its role in determining the effectiveness of different policy interventions. But their paper does not take aspects i and iv into consideration as in our dynamic public response model. Hence, the discussion is limited in terms of how heterogeneous agents interact and behave dynamically in the presence of asymptomatic infection.

Heterogeneous agents with private or partially observable types. Individual heterogeneity is a fundamental premise of the emergence of a polarized equilibrium in our model. One way to model heterogeneity is to partition the population into a finite number of groups based on certain primitives, e.g., infection costs and the flow payoffs of social activities as in Dasaratha (2022), or their observable traits, as in Acemoglu et al. (2021) and Gollier (2020). The latter two develop a multi-group SIR model and demonstrate the superiority of targeting different groups with different policies compared to a uniform lockdown policy. Our model differs in that each agent is endowed with an atomlessly distributed type, which allows for a more nuanced representation of individual differences. Javadi et al. (2021) makes a similar assumption of type distribution as ours, except that agents only differ in their loss from infection. We consider a three-dimensional type distribution allowing agents to differ in their discount factor, the probability of developing symptoms after infection, and their expected suffering after symptomatic infection.[9]

Outside the pandemic literature, our paper also contributes to the dynamic

[9]The type can be private information or imperfectly observable. We report some comparative statistics results in Proposition 1 which highlights the influence of these personal characteristics on an agent's equilibrium response to the pandemic.

modelling of decisions under varying patterns of the hazard rate of success or failure probabilities. Previous research by Khan and Stinchcombe (2015) highlights how the hazard rate pattern determines the optimal timing to take a costly action in a semi-Markovian environment. Our model relates to one scenario of this paper in which the delay in a precautionary measure towards future changes has a sure benefit but a stochastic cost. In this case, a decreasing hazard rate of future changes predicts either an immediate action or never taking action. While Khan and Stinchcombe (2015) focuses on an individual decision problem, we look at a public response game and particularly the effect of the hazard rate on the aggregate behavioral patterns in equilibrium. Different hazard rate patterns have also been studied in the experimentation literature (Boyarchenko, 2020; Thomas, 2021), media economics (Oliver, 2022), and financial economics (Boyarchenko, 2021; Klishchuk, 2022). Most of the discussion, however, centers around the types of situations leading to a unique interior solution.

3 Dynamic Public Response Model

Consider an environment with a countable number of periods $t = 0, 1, 2, ...$ and a continuum of agents with a population size (or measure) equal to 1. A pandemic occurs at $t = 0$ and evolves over time $t \in \{1, 2, ..., T\}$ with $T \in \mathbb{N} \bigcup \{\infty\}$.[10] In any period t, agents can be partitioned into three subpopulations: the *infectious* with size ϑ_t, the *innocuous* (including those who passed away) with size ρ_t, and the *susceptible* with size $1 - \vartheta_t - \rho_t$.

The *infectious* population consists of those who were infected in the previous period $t - 1$, which can be further divided into two groups: the *symptomatic* and the *asymptomatic*. The former has shown symptoms of the disease by the end of $t - 1$, and the latter will never show symptoms. The symptomatic agents may either die or, like the asymptomatic agents, recover and acquire immunity by the end of period t. To focus on the main behavioral issues, we sidestep the possibilities

[10] To ease exposition, we treat T as a finite number in the modelling and subsequent analyses. Owing to uniformly bounded utility, marginal utility flows, and discounting, the definitions and results with a finite time horizon T extend to $T \to \infty$ straightforwardly.

of testing and vaccination and assume recovered agents are no longer infectious.[11] Consequently, the *innocuous* population in period t consists of all those who had been infected before the end of $t-2$, including the symptomatic ones who know they are now immune and the asymptomatic ones who do not know the fact with certainty. Regarding the *susceptible* population, it consists of agents who have not been infected by the end of $t-1$. Again, the possibility of asymptomatic infection in the past prevents the apparently healthy agents from telling with certainty whether they are infectious, innocuous, or susceptible.

We say that an agent is *active* in period t if so far they have not shown symptoms (see Figure 1). Thus, the susceptible and the asymptomatically infected agents are all active. The main factors influencing the development of the pandemic are the levels of *communicable activity* (or *action* for short) of the active agents. To ease exposition, we say that an agent is *passive* if they are not active. Thus a passive agent can be passed away, recovered, or symptomatically infected in period $t-1$. By assumption, activities of the innocuous agents no longer matter. As for the symptomatically infected agents in $t-1$, there are numerous possible consequences of being sick, ranging from hospitalization, (self-) quarantine, through various degrees of observable symptoms. Consequently, we model the overall infectiousness of this group, rather than individual activities, in each period in Section 3.2.

The state variable $\mu_t \in [0,1]$ is the *transmission-intensity rate* of the pandemic. We will model μ_t as a Markov process in Section 3.3 given the exogenous state $\mu_0 \in (0,1)$. The realization of μ_t is publicly known at the start of each period t.

3.1 Heterogeneous agents and communicable activity

Each active agent is endowed with a three-dimensional type $x \in X \subset \mathbb{R}^3_+$, written as $x = (\delta_x, \gamma_x, D_x)$. According to (δ, γ, D), without the pandemic, each type-x agent would live a normal life and enjoy a utility flow of $u \in (0, \infty)$ per period. With $\delta_x \in (0, \bar{\delta}]$ ($\bar{\delta} < 1$) being their discount factor, a type-x agent would have a life-time

[11] Incorporating the possibility that some people may be infected several times, recovered people may still be infectious, or that costly and imperfect antigen tests or vaccination are available, can be done but is not expected to change any qualitative conclusions of this study.

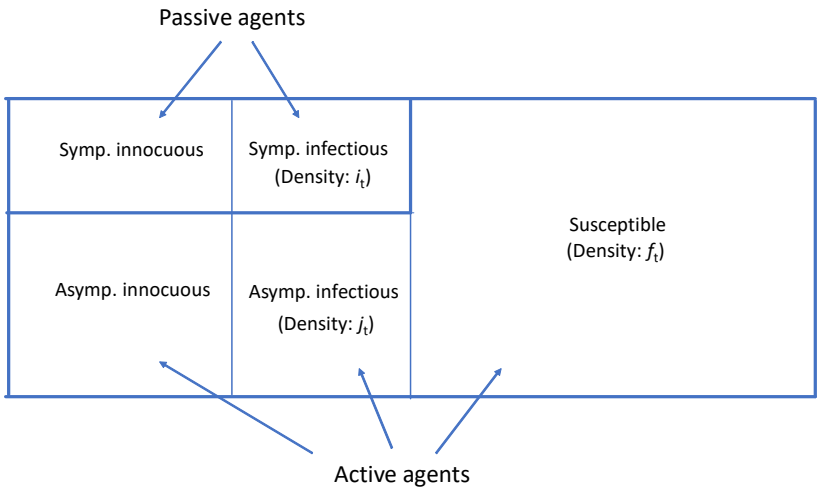

Figure 1: Partition of the population at the start of each period t. Infectious agents are those infected in period $t-1$. Innocuous agents are those infected in period $t-2$ or earlier. (The densities i_t, j_t, and f_t will be derived in Section 3.2.)

discounted utility equal to

$$U_x = \sum\nolimits_{t=0}^{\infty} \delta_x^t u = \frac{u}{1-\delta_x} \tag{2}$$

Agents observe—and start reacting to—the pandemic in period 1. When a type-x agent is infected during any period t, they will show symptoms with probability $\gamma_x \in (0,1]$ by the end of period t and no symptoms with probability $1-\gamma_x$. If the agent is symptomatic of the disease, they have an expected utility of $D_x \in [0, \frac{u_0}{1-\delta_x}]$, where $0 < u_0 < u$.[12] Initially, in period 0, the types of population are distributed according to probability measure F on the Borel subsets of X. To focus on pure strategies, we assume that the distribution F is atomless, with a measurable associated density f.[13]

[12] Our analysis focuses on agents' *expected* utility D_x in the state of being symptomatically sick, which includes the possibility of death, without specifying how D_x is calculated.

[13] A probability measure F over X is atomless if every $B \subseteq X$ with $F(B) > 0$ has a subset $C \subset B$ for which $F(B) > F(C) > 0$. Milgrom and Weber (1985). In special cases, F may have an associated density function. In general, since X is multi-dimensional, an atomless F also permits some of the variables, δ, γ, or D to have discrete marginal distributions.

Starting from period 1, each active agent can choose a level of communicable activity (or action) $a_t \in [0,1]$ in each period $t \geq 1$ that restricts their normal way of living—as long as the agent remains active. The risk of being infected increases in a_t, with $a_t = 0$ being a "safety first" action and $a_t = 1$ being a "life as usual" action. We assume that there is a utility function of communicable activity $v : [0,1] \to [u_0, u]$ that is twice differentiable on $(0,1)$, satisfying $v(0) = u_0$, $v(1) = u$, and $v' > 0$. Thus, u_0 indicates the agent's utility in a period without any communicable activity. The reduction of normal-life utility, $u - v(a_t)$, can be seen as a deadweight utility loss. We perceive communicable activity to be directly related to one's utility or wellbeing, not consumption. For instance, reducing communicable activity by wearing a mask, frequently washing hands, or practicing social-distancing does not incur a high monetary cost. Nonetheless, these protective measures can reduce people's *sense* of freedom and wellbeing.

Now let μ_0 be given and let $\mu^t := (\mu_1, ..., \mu_t) \in [0,1]^t$ denote the history of the transmission-intensity rates in periods 1 through t.

Definition 1 *Given $\mu_0 \in (0,1)$, for all $t \in \{1, 2, ..., T\}$ and $\mu^t \in [0,1]^t$, a* public-response function *in period-t is a measurable function $\alpha_t(\cdot, \mu^t) : X \to [0,1]$. Given information μ^t in period t, $\alpha_t(x, \mu^t)$ is the level of action chosen by the type-x active agents, in period t.*

Definition 2 *A* public-response plan *is a sequence of public-response functions $\alpha = (\alpha_1, ..., \alpha_T)$ with $\alpha_t : X \times [0,1]^t \to [0,1]$, given $\mu_0 \in (0,1)$ and $\alpha_0 \equiv 1$. Therefore, for every $x \in X$, the type-x active agents will choose action $\alpha_1(x, \mu^1)$ in period 1 and plan to choose $\alpha_t(x, \mu^t)$ for every future period $t \in \{2, 3, ..., T\}$, contingent on $\mu^t \in [0,1]^t$, as long as they are active until T. As soon as an agent shows symptoms, they will drop out and no longer take action in the subsequent periods.*

Remark. We find no need to consider each agent's private state of being "active" or "passive" in a period t. As the population has a continuum of agents, every point of density $x \in X$ involves a continuum of agents even when the set of type-x has a zero measure. A plan can be viewed as invariably feasible because after some type-x agents drop out when they are symptomatic, there are other active type-x agents to execute the remainder of the plan.

3.2 Endogenous state variables

We now define and characterize several endogenous state variables in a pandemic, assuming that (almost) all active agents follow the public-response plan α in choosing their actions. Since different types may be exposed to different probabilities of disease, we define the *type densities* of the following population groups as follows (see Figure 1): for $t \in \{1, 2, ..., T\}$,

$$\begin{array}{rcl} i_t & : & \text{density of symptomatically infectious types over } X \text{ in period } t \\ j_t & : & \text{density of asymptomatically infectious types over } X \text{ in period } t \\ f_t & : & \text{density of susceptible types over } X \text{ in period } t \end{array}$$

For $t = 0$, we define $f_0 = f$ as all agents were susceptible at the start of period 0. We assume that the initial outbreak of the pandemic in period 0 was due to an exogenous shock by Nature, causing I_0 of the agents infected. For $t \geq 1$, we will derive i_t, j_t, and f_t jointly with other endogenous state variables.

To model the quantitative effects of communicable activity, observe that an active agent increasing action a_t in a period t has dual effects: it increases the probability that they may get infected *and* the probability that they may infect the others. Although each agent may neglect the effect of their action on others, the collective actions do matter for the overall transmission environment. We now introduce an important variable, considered as a numerical representation of the *transmission risk* of the environment in period t.

Definition 3 *Under a public-response plan α, the transmission risk of the environment in period $t \in \{1, 2, ..., T\}$ is defined by*

$$\begin{array}{rcl} \chi_t & : & = \displaystyle\int_X \theta(x) i_t(x) dx + \int_X h(\alpha_t(x, \mu^t)) j_t(x) dx, \\ t & \in & \{1, ..., T\} \end{array} \tag{3}$$

where the function $\theta : X \to [0, 1]$ is measurable, and $h : [0, 1] \to [0, 1]$ is continuously differentiable such that $h(0) = 0$, $h(1) = 1$, and $h' > 0$ on $(0, 1)$.

The first term on the right side of (3) captures the infectiousness of the symptomatic agents in the preceding period, where $\theta \equiv 0$ corresponds to the case in

which all sick agents were immediately (self-) quarantined and no infected agent would deliberately infect others. As θ increases, sick agents play an increasing role in transmitting the disease. The last term in (3) captures the infectiousness from the asymptomatically infected agents. Without symptoms, these agents continue to follow the public-response plan in choosing their actions in period t. The function h measures the contribution of different levels of actions to the overall infectiousness of this group. For the special case where $\theta \equiv 1$ and $h \equiv 1$, the transmission risk χ_t reaches its upper bound ϑ_t, the size of the infectious population in period t. We do not assume specific functional forms for the primitives θ and h so as to keep the model more amenable to empirical studies.

Given μ_t, we assume that when a susceptible agent is *exposed* to the transmission risk of the environment in period t, they will be infected with probability $\mu_t\chi_t$. Associated with χ_t are a pair of related functions, the *infection probability* p_t, and the *disease probability* q_t.

Definition 4 *The* exposure function *is a cumulative probability function* $g : [0, \infty) \to [0, 1)$ *satisfying* $g(0) = 0$, $\lim_{a\to\infty} g(a) = 1$, *and* $g' > 0$ *on* $(0, 1)$ *such that in any period* t, *a susceptible agent choosing activity* a_t *will be exposed to the transmission risk of the environment with probability* $g(a_t)$.

Since there is no reason to assume that taking action 1 will expose the agent to the transmission risk with certainty, we allow $g(1) \leq 1$.[14] We will be interested in the behavior of the hazard rate of the exposure function $\lambda := \frac{g'}{1-g}$. The hazard rate $\lambda(a_t)$ indicates the marginal probability of an agent being exposed to the transmission risk by a marginal increase of a_t, conditional on them not being exposed to such a risk at action level a_t.

Given the exposure function g, the *infection probability* p_t is given by

$$p_t(a_t, \mu_t, \chi_t) = g(a_t)\mu_t\chi_t \tag{4}$$

for any susceptible agent choosing activity a_t in period t.

Our modeling is complicated by the consideration that the active agents do not know with certainty whether they are susceptible, infectious, or immune in any

[14] Since $a_t \in [0, 1]$ is merely a normalization, defining g on $[0, \infty)$ maintains model generality and allows g to be taken from a larger family of distribution functions.

given period. They may have been unknowingly infected in the past without showing symptoms. Thus, we allow rational agents to infer their disease probability, given their past actions. Consider an active agent with type x who has chosen activities $a_0, a_1..., a_{t-1}$ in periods 0 through $t-1$. Suppose these activities have exposed the agent to infection probabilities p_s, with the associated disease probabilities q_s, over periods $s = 0, 1, ..t-1$. Let $\Pr(\text{susceptible}_t|\text{active}_t)$ denote the conditional probability that the agent is susceptible in period t. Since being susceptible is equivalent to being uninfected in the past, and since being active implies that the agent has not been sick, we obtain

$$\Pr(\text{susceptible}_t|\text{active}_t) = \frac{(1-p_0)(1-p_1)...(1-p_{t-1})}{(1-q_0)(1-q_1)...(1-q_{t-1})} := S_{t-1} \tag{5}$$

Thus, when (almost) all other agents follow the plan α, an active type-x agent with a history of actions $a_0 = 1$ and $a^t := (a_1, ..., a_t)$ has an expected *disease probability* in any period $t = 0, 1, ...$ equal to (defining $S_{-1} = 1$)

$$q_t(x, S_{t-1}, p_t) = \gamma_x S_{t-1} p_t \tag{6}$$

Summarizing, we may perceive the sequence of events that leads to sickness of an agent so far without symptoms at the start of period t as follows.

Being susceptible & exposed to risk $\rightarrow$	Infected $\rightarrow$	Symptomatic
Prob $= S_{t-1}g(a_t)$	Prob $= \mu_t\chi_t$	Prob $= \gamma_x$

(7)

The sequence has the following interpretation. At the start of period t, the agent commits to action $a_t \in [0, 1]$. Through this period, the agent runs the risk of being susceptible and exposed to the transmission risk with probability $S_{t-1}g(a_t)$. Conditional on being susceptible and exposed to the risk, the agent will be infected with probability $\mu_t\chi_t$. If the agent is infected, they will show symptoms with conditional probability γ_x.

The *infection rate* of the population in a period t, which we denote by I_t, can be now calculated by integration, using density $f_t(x)$ of the susceptible types:

$$I_t = \mu_t\phi_t\chi_t \text{ where } \phi_t = \int_X g(\alpha_t(x, \mu^t))f_t(x)dx \tag{8}$$

Thus, I_t can be seen as μ_t multiplied by a generalized matching function $M_t = \phi_t\chi_t$ where the first part ϕ_t is directly related to the density and actions

of the susceptible population, and the second part χ_t the density and actions of the infectious population. The formula includes the basic SIR model of infection rate as a special case when there is no asymptomatic transmission ($\gamma \equiv 1$), full symptomatic infectiousness ($\theta \equiv 1$), and no prevention measures ($g \equiv 1$ and $h \equiv 1$). Then, ϕ_t reduces to the size of the susceptible population and χ_t the size of the infectious population, as is assumed in the basic SIR models.[15]

Lemma 1 *Let $\mu_0 \in (0,1)$ and $I_0 \in (0,1)$ be given in $t = 0$. Suppose (almost) all active agents follow public-response plan $(\alpha_t)_{t=1}^T$ with $\alpha_0 \equiv 1$. The law of motion for $(i_t, j_t, f_t, \chi_t, p_t, q_t)$ is jointly given by*[16]

$$
\begin{aligned}
i_t(x) &= \gamma_x p_{t-1}(x, \mu^{t-1}) f_{t-1}(x) && (9)\\
j_t(x) &= (1-\gamma_x) p_{t-1}(x, \mu^{t-1}) f_{t-1}(x) && (10)\\
f_t(x) &= (1 - p_{t-1}(x, \mu^{t-1})) f_{t-1}(x) && (11)\\
\chi_t(\mu^t) &= \int_X \theta(x) i_t(x) dx + \int_X h(\alpha_t(x, \mu^t)) j_t(x) dx && (12)\\
p_t(x, \mu^t) &= g(\alpha_t(x, \mu^t)) \mu_t \chi_t(\mu^t) && (13)\\
q_t(x, \mu^t) &= \gamma_x S_{t-1}(x, \mu^{t-1}) p_t(x, \mu^t) \text{ where } S_{t-1} = S_{t-2} \frac{1 - p_{t-1}(x, \mu^{t-1})}{1 - q_{t-1}(x, \mu^{t-1})} && (14)
\end{aligned}
$$

with $f_0 = f$, $p_0 = I_0$ and $q_0(x, \mu^0) = \gamma_x I_0$.

From the above analysis it follows that the sizes of the infectious and innocuous populations in period t are given by

$$
\begin{aligned}
\vartheta_t(\mu^t) &= \int_X p_{t-1}(x, \mu^{t-1}) f_{t-1}(x) dx \\
\rho_t(\mu^t) &= \sum_{s=0}^{t-1} \vartheta_s(\mu^s)
\end{aligned}
$$

3.3 Dynamic game and equilibrium

We assume that in any period $t \in \{0, 1, 2, ..., T\}$, the distribution of μ_{t+1} is governed by transition probability measure $\hat{\Phi}_t : [0,1] \times [0, \vartheta_t] \to \Delta([0,1])$, such that given $(\mu_t, \chi_t) \in [0,1] \times [0, \vartheta_t]$, the state μ_{t+1} is a random variable distributed according to

[15] Since time is continuous in the SIR models, the infected agents are simultaneously infectious.

[16] To ease exposition, $p_t(x, \mu^t)$ denotes $p_t(\alpha_t(x, \mu^t), \mu_t, \chi_t)$, etc.

$\hat{\Phi}_t(\mu_t, \chi_t) \in \Delta([0,1])$ (with $\hat{\Phi}_T(\mu_T, \chi_T)$ assigning probability 1 to the event $\{\mu_{T+1} = 0\}$ for $T < \infty$).[17,18]

Given $\hat{\Phi}$ $(= (\hat{\Phi}_0, \hat{\Phi}_1, ..., \hat{\Phi}_T))$, and the initial states $\mu_0 \in (0,1)$ and $\chi_0 = 1$, by the Tulcea Extension Theorem each public-response plan α $(= (\alpha_1, ..., \alpha_T))$ uniquely defines a probability measure over the sequence of states $(\mu_t)_{t=1}^T \in [0,1]^T$ for $T \leq \infty$. Therefore, a unique stochastic process is defined for $T \leq \infty$. Subsequently, we fix $\hat{\Phi}$ and let $\mathbb{E}_t^\alpha[\cdot|\mu_t, \chi_t]$ denote the conditional expectation operator for μ_{t+1} under the public-response plan α, given (μ_t, χ_t). The expected payoff V_t^* of each active type-x agent in period t, given μ^t, can be described recursively:[19]

$$V_t^*(x, \mu^t) = v(\alpha_t(x, \mu^t)) + \delta_x \left\{(1 - q_t(x, \mu^t))\mathbb{E}_t^\alpha \left[V_{t+1}^*(x, \mu^{t+1})|\mu_t, \chi_t\right] + q_t(x, \mu^t)D_x\right\} \tag{15}$$

for $t \in \{1, 2, ..., T\}$, with $V_{T+1}^*(x, 0) = U_x$ if $T < \infty$.

The interpretation of (15) is as follows. At the start of period t given μ^t, for all $x \in X$, the active type-x agents choose action $\alpha_t(x, \mu^t)$ and enjoy utility equal to $v(\alpha_t(x, \mu^t))$ over period t. By the end of the period, with probability $q_t(x, \mu^t)$, each type-x agent will be symptomatically ill and drop out, in which case the agent expects a life-time utility D_x $(< U_x)$; and with probability $1 - q_t(x, \mu^t)$, each type-x agent will remain active and choose $\alpha_{t+1}(x, \mu^{t+1})$ over period $t+1$, according to the then realized history μ^{t+1}. This leads to the continuation payoff $V_{t+1}(x, \mu^{t+1})$. For $T < \infty$, from period $T+1$ onwards, all the then active agents will enjoy life-time utility U_x, according to their type $x \in X$.

The active agents play a dynamic noncooperative game in choosing their actions, each attempting to maximize their expected payoff. The game will end after T, if T is finite.

Definition 5 *The public-response plan* $\alpha = (\alpha_1, ..., \alpha_T)$ *forms a* sequential public-

[17] $\Delta([0,1])$ denotes the set of probability measures over $[0,1]$.

[18] To focus on individual behaviors, we choose this Markov formulation solely for simplicity. There is also no need to assume any knowledge of how the state variables are serially correlated, except a condition proposed in Assumption 1 and Proposition 1. Extension to the more general processes could follow the treatment and discussion in Pavan et al. (2014) and Athey and Segal (2013).

[19] The existence of an optimal plan $(\alpha_t)_{t=1}^T$ for each type of active agents is guaranteed because V_t is a continuous function of actions defined on the closed interval $[0,1]$ (see (16)).

response equilibrium *(SPRE) if and only if the following conditions hold:*

(i) Sequential rationality. *Given* $\mu_0 \in (0,1)$ *and any realized history* $\mu^t \in [0,1]^t$, $t \in \{1,2,...T\}$, *for almost all* $x \in X$, *if the agent of type* x *is active and has followed the plan* α *in the past, they will optimally choose action* $\alpha_t(x, \mu^t)$ *in the current period and plan to follow* $\alpha_{t+1}(x,\cdot), \alpha_{t+2}(x,\cdot)...$*until* T. *Supporting this plan is the agent's belief that almost all other active agents have followed the plan in the past, and will continue to follow the plan in the current and future periods.*

(ii) Consistent updating of information. *All active agents who have followed the plan up to* t *update their beliefs using Bayes rule regarding the density of susceptible types* f_t, *infection-probability function* p_t, *and disease-probability function* q_t, *according to Lemma 1.*

In an SPRE, active agents are not affected by any other individual agent's type or action, and the information set regarding other active agents' types remains the same X for all periods. However, agents are affected by the transmission risk χ_t of the environment, which is important for their assessment of the infection and disease probabilities p_t and q_t, respectively. The equations (9)–(14) for updating $(i_t, j_t, f_t, \chi_t, p_t, q_t)$ over time can be seen as related to the Bayesian updating of beliefs in a Perfect Bayesian Equilibrium for games with incomplete information. Since the agents' disease probabilities depend on their past actions, as in (6), agents who deviate from the plan α may find it optimal to continue deviating from it. However, under an SPRE, the set of deviating agents has a zero measure across all periods so that the law of motion for $(i_t, j_t, f_t, \chi_t, p_t, q_t)$ is unaffected.

4 Equilibrium Analysis

This section contains the main theoretical results. We first establish the existence of SPRE in Theorem 1 under general conditions. We then characterize polarized equilibrium in Theorem 2 for DARA or CARA agents under constant hazard rate of the exposure function, and show that the obtained SPRE is unique. The equilibrium behavior and comparative statics results are then presented in Proposition 1. Finally, we present in Proposition 2 the long term behavior of SPRE as t tends to infinity.

4.1 Path dependent equilibrium

We start with a public-response plan α and consider the individual decisions of an arbitrary active agent in period $t \geq 1$. To ease notation, we suppress the expression of variables unless it is needed for clarity.

Let $a^{t-1} = (a_1, ..., a_{t-1})$ denote the agent's past actions, such that $a^t = (a^{t-1}, a_t)$. By the principle of optimality for stochastic dynamic programming,[20] the agent's optimal expected payoff V_t^* in any period t can be described as (suppressing variables x, μ^t, χ_t), recalling that $q_t = \gamma_x S_{t-1} p_t$ where $p_t = g(a_t)\mu_t\chi_t$,

$$\begin{aligned} V_t^*(a^{t-1}) &= \max_{a_t \in [0,1]} \left\{ v(a_t) + \delta(1 - q_t(a^t)) \mathbb{E}_t^\alpha \left[V_{t+1}^*(a^t) | \mu_t, \chi_t \right] + \delta q_t(a^t) D \right\} \quad (16) \\ t &\in \{1, 2, ..., T\} \end{aligned}$$

with $V_{T+1}^* = U$ if $T < \infty$. Let V_t $(= V_t(x, a_t, a^{t-1}, \mu^t, \chi_t))$ denote the term in curly brackets in (16):

$$V_t = v(a_t) + \delta \left\{ (1 - q_t) \mathbb{E}_t^\alpha \left[V_{t+1}^* | \mu_t, \chi_t \right] + q_t D \right\} \quad (17)$$

To establish equilibrium existence, we invoke the following assumption.

Assumption 1 *The cumulative distribution function $\Phi_t(\cdot|\mu_t, \chi_t)$ that corresponds to the transition probability measure $\hat{\Phi}_t(\mu^t, \chi_t)$ is twice differentiable in all arguments, with the density function φ_t $(= \Phi_t'(\mu_{t+1}|\mu_t, \chi_t))$ satisfying, for any $t \in \{1, ..., T\}$,*

$$\frac{\partial \varphi_t / \partial \chi_t}{\varphi_t} \geq -1 \quad (18)$$

at all points where $\varphi_t > 0$.

Roughly, Assumption 1 requires that a marginal change in χ_t should not change the density function of μ_{t+1} "by too much" in the sense of (18). This technical assumption implies the following behavioral assumption: in every period t, each active agent's expected payoff is submodular in (a_t, χ_t), i.e., $\frac{\partial^2 V_t}{\partial a_t \partial \chi_t} \leq 0$ (see Lemma 4). According to this behavioral assumption, agents play a submodular game by responding to a marginal increase in χ_t with a marginal decrease in their action

[20] See, e.g., Chapter 9, Stokey et al. (1989). Although our model is different from those treated in their book, the analysis follows similar lines.

in any period t. The reason is that a higher level of χ_t increases the probability of infection to all susceptible agents, and thus decreases each agent's incentive to choose a higher a_t. Therefore, the role of Assumption 1 is to provide a sufficient condition for agents to play a submodular game that is based only on the primitives φ_t in the model.

Theorem 1 *Consider the dynamic public response model of Section 3. Suppose Assumption 1 holds. Then there exists a sequential public-response equilibrium (SPRE)* $\alpha = (\alpha_t)_{t=1}^T$ *characterized by (i)*

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - \delta \frac{\partial q_t}{\partial a_t} \mathbb{E}_t^\alpha \left[V_{t+1}^* - D | \mu_t, \chi_t \right] + \delta(1 - q_t) \mathbb{E}_t^\alpha \left[\left. \frac{\partial V_{t+1}^*}{\partial a_t} \right| \mu_t, \chi_t \right] = 0 \quad (19)$$

at $a_t = \alpha_t(x, \mu^t)$ *for which (16) has an interior solution, otherwise* $\alpha_t(x, \mu^t) = 0$ *or* 1*, depending on whether* $V_t(x, a_t, \alpha^{t-1}(x, \mu^{t-1}), \mu^t, \chi_t)$ *is the greatest at* $a_t = 0$ *or* 1 *through the entire interval* $[0, 1]$*, for all* $x \in X$ *and* $t \in \{1, 2, ..., T\}$*, where* $\frac{\partial q_t}{\partial a_t} = g'(a_t)\gamma_x S_{t-1}\mu_t\chi_t$ *and* $\frac{\partial V_{t+1}^*}{\partial a_t}$ *is recursively defined via*

$$\begin{aligned} & \mathbb{E}_t^\alpha \left[\left. \frac{\partial V_{t+s}^*}{\partial a_t} \right| \mu_t, \chi_t \right] \\ = \; & -\delta \mathbb{E}_t^\alpha \left[\left(V_{t+s+1}^* - D \right) \frac{\partial q_{t+s}}{\partial a_t} - (1 - q_{t+s}) \frac{\partial V_{t+s+1}^*}{\partial a_t} \middle| \mu_t, \chi_t \right] \\ s \; = \; & 1, ..., T - t \end{aligned}$$

and (ii)

$$\chi_t = \int_X \theta(y) i_t(y) dy + \int_X h(\alpha_t(y, \mu^t)) j_t(y) dy \quad (20)$$

Here, (19) is the first-order condition and (20) is an additional equilibrium condition. In (19), we interpret $v'(a_t)$ as a direct marginal benefit and the term involving $V_{t+1}^* - D$ as the marginal cost, of increasing action at a_t. This cost is related to the risk of being symptomatically ill, in which case the agent drops out by the end of the period and suffers an expected loss of payoff $V_{t+1}^* - D$. Thus, if the third term involving $\frac{\partial V_{t+1}^*}{\partial a_t}$ doesn't exist, an interior solution will be characterized by equating the marginal cost with the marginal benefit. However, this third term is a result of asymptomatic infection and can play an important role in influencing agents' optimal actions. By backward induction, it can be easily shown (with the proof omitted) that increasing a_t has a positive marginal effect on the continuation payoff V_{t+1}^*, as given in Corollary 1.

The proof of Theorem 1 is lengthy, complicated by path dependency of V_t. In Appendix 1, we prove Theorem 1 by transforming the problem into a mathematically equivalent one that removes the path dependency of the expected continuation payoffs. Note that the equilibrium in Theorem 1 may not be unique under the general structure of the problem.

Corollary 1 *Suppose Theorem 1 holds. Then, in an SPRE,* $\frac{\partial V_{t+1}^*}{\partial a_t} \geq 0$ *for all* $x \in X$ *and* $t \in \{1, 2, , ..., T\}$.

This corollary suggests that if the agent remains active by the end of the period, having taken a higher level of action would be more desirable.

4.2 Equilibrium polarization

In this subsection, we are interested in an SPRE that exhibits polarization of the following property: there exists an interval $(c, 1)$ with $c \in [0, 1)$ that partitions the agents into two camps: in equilibrium, in any period $t \in \{1, 2, ..., T\}$, part of the agents choose actions in $[0, c]$ and the rest of agents choose actions in $\{1\}$. The extreme case is where $c = 0$, in which a sharply polarized equilibrium arises, with the entire active population either preferring action 0 or 1.

To ease the analysis and sharpen the equilibrium characterization, we invoke two more assumptions.

Assumption 2 *Agents' periodic utility function* v *satisfies* $v'' < 0$[21] *and exhibits decreasing or constant absolute risk aversion (DARA or CARA), i.e.,* $-\frac{v''(a)}{v'(a)}$ *is nonincreasing in action* a *on* $[0, 1]$.

Assumption 3 *The exposure function* g *has the memoryless property such that* $g(a) = 1 - \exp(-\lambda a)$ *for some* $\lambda > 0$.

The intuition behind Assumption 2 is similar to that of the DARA assumption on utility functions for money. For instance, consider the choice between a small increase of communicable activity Δa and an uncertain increase of activity $\Delta\tilde{a}$. If an agent currently enjoying activity level a prefers the sure increase of Δa, then it

[21] For $v'' \geq 0$, the conclusion of Case (i) in Theorem 2 holds as a straightforward corollary.

seems reasonable to posit that the agent would also prefer the sure increase of Δa when their current activity level is lower than a.

In Assumption 3, the memoryless hazard rate assumption may be construed as follows. The hazard rate $\lambda(a_t)$ indicates the marginal probability of an agent being exposed to the transmission risk by a marginal increase of a_t to $a_t + \Delta a$, conditional on them not being exposed to such a risk at action level a_t. Now let us consider a thought experiment. Suppose there are some people shopping in a grocery store without wearing a mask. Let Δa denote their shopping activities and ask this question: Take any two of these people who have not been exposed to the transmission risk when they entered the store, should their probability of risk exposure, due to shopping at the store, differ? Although the two may have taken very different levels of communicable activity before shopping, we have no reason to surmise that their probabilities of risk exposure differ in the same shop. Therefore, it seems a reasonable starting point to assume that under a given transmissible environment, the hazard rate of risk exposure is a constant.

Theorem 2 *(Part 1) Under Assumption 1, the SPRE $\alpha = (\alpha_t)_{t=1}^T$ characterized in Theorem 1 can be equivalently characterized by a positive function H_t defined by*

$$H_t = v(\alpha_t) - (1-\delta)\, D + \delta(1-p_t)H_{t+1} \tag{21}$$

for $t \in \{1, 2, ..., T\}$, with $H_{T+1} = U - D$ if $T < \infty$, replacing (19) with

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - \delta \frac{\partial q_t}{\partial a_t} \mathbb{E}_t^{\alpha} \left[H_{t+1} | \mu_t, \chi_t \right] = 0. \tag{22}$$

(Part 2) Under Assumptions 1-3, there exists a threshold point c such that

$$c = \begin{cases} \min\{a \in [0,1] : -\frac{v''(a)}{v'(a)} \leq \lambda\} & \text{if } -\frac{v''(1)}{v'(1)} < \lambda \\ 1 & \text{if } -\frac{v''(1)}{v'(1)} \geq \lambda \end{cases} \tag{23}$$

Given $\mu_0 \in (0,1)$ and $\alpha_0 \equiv 1$, the SPRE $\alpha = (\alpha_t)_{t=1}^T$ is uniquely[22] characterized by:

(i) if $c = 0$, then $\alpha_t(x, \mu^t) \in \{0, 1\}$ for all $x \in X$;

(ii) if $c \in (0,1)$, then $\alpha_t(x, \mu^t) \in [0, c) \cup \{1\}$, satisfying (22) for $\alpha_t(x, \mu^t) \in (0, c)$ or else $\alpha_t(x, \mu^t) \in \{0, 1\}$;

[22] In the present context, uniqueness of equilibrium α means that any other equilibrium may differ from α, only in a set of types with a zero F_t-measure in each period $t \in \{1, 2, ..., T\}$.

(iii) if $c = 1$, *then* $\alpha_t(x, \mu^t) \in [0, 1]$, *satisfying (22) for* $\alpha_t(x, \mu^t) \in (0, 1)$ *or else* $\alpha_t(x, \mu^t) \in \{0, 1\}$.

Here, H_t serves as an auxiliary function: the expected value of H_{t+1} in (22), when multiplied with $\frac{\partial q_t}{\partial a_t}$, captures the overall marginal cost given in square brackets in (19). The most useful property of H_{t+1} is that it does not depend anymore on the path-dependent disease probabilities $q_t, q_{t+1}, ...$ We see from Theorem 2 that the equilibrium behavior critically depends on the level of the hazard rate λ of the exposure function g. For λ sufficiently high such that $\lambda > -\frac{v''}{v'}$ on $[0, 1]$, Case (i) holds as $c = 0$. And the theorem predicts a sharply polarized equilibrium that partitions the agents into those who choose "safety first ($\alpha_t = 0$)" and those who choose "life as usual ($\alpha = 1$)." In Case (ii) where $\lambda > -\frac{v''(a)}{v'(a)}$ on $(c, 1] \subset [0, 1]$, a milder polarized equilibrium holds in which some agents maximize by choosing actions in $[0, c)$ and the others choose 1. A common feature of Cases (i) and (ii) is that the equilibrium exhibits polarization: a threshold c partitions the agents' equilibrium actions into two disjoint sets, with no one choosing actions in $(c, 1)$.

The key insight into the polarized behavior derives from the fact that

$$-\frac{g''(a)}{g'(a)} > -\frac{v''(a)}{v'(a)} \text{ if and only if } \frac{v'(a)}{g'(a)} \text{ increases in } a$$

The ratio $\frac{v'(a)}{g'(a)}$ being an increasing function suggests that g' declines faster than v' as a goes up. In the present context, $v'(a)$ is the marginal benefit of increasing action a and $g'(a)$ the marginal probability of getting exposed to the transmission risk of the environment—both measured at level a. In other words, when the marginal benefit of increasing a equals the expected marginal cost due to increased probability of exposure at level c, then the marginal benefit of increasing a exceeds the expected marginal cost at all action levels $a > c$. To be more precise, let M_t denote the expected cost conditional on being exposed to the transmission risk so that $\frac{\partial V_t}{\partial a_t} = v'(a_t) - g'(a_t)M_t$. Since $-\frac{g''(a)}{g'(a)} = \lambda$, for Cases (i)-(ii) of Theorem 2 we have $\frac{v'(a)}{g'(a)}$ increasing in a on $(c, 1]$ for $c \in [0, 1)$. Thus $\frac{\partial V_t}{\partial a_t} \geq 0$ (or equivalently, $\frac{v'(a)}{g'(a)} \geq M_t$) at any level $a \geq c$ implies $\frac{\partial V_t}{\partial a_t} > 0$ at all levels $a_t > a$. Consequently, all actions $a_t \in (c, 1)$ are inferior to $a_t = 1$. It follows then, for Cases (i)-(ii), that the equilibrium actions must be equal to $\max\{\arg\max_{a \in [0,c]} V_t(a), 1\}$, for all types of agents and all periods.

What seems to be counter intuitive is that polarization is more likely to occur when the hazard rate of the exposure function g is high. It is true that given any $a > 0$, the probability $g(a)$ is higher and therefore equilibrium actions lower for a higher λ. But the conclusions of Theorem 2 does not rely on the absolute values of g. Instead, polarization is a consequence of the relative marginal values of v and g.

A Two-period Example

To gain some visual insights into polarized behavior, consider a two-period example. In period 0, a pandemic took place and infected I_0 agents. Agents react by choosing communicable activities in period 1 knowing that effective vaccinations will be available for everyone by the end of the period. Assume $v(a) = u_0 + a^\theta(u - u_0)$ $(0 < \theta < 1)$ and $g(a) = 1 - \exp(-\lambda a)$. Then, we have $c = \frac{1-\theta}{\lambda}$ and polarization will occur for $\lambda > 1 - \theta$. Further assume that the agents' type set is of single dimension such that $\delta_x \equiv \delta$, $\gamma_x \equiv \gamma$, so that agents differ only in their expected utility upon symptomatic illness, D_x. Thus, for type x we have $\mathbb{E}_1^\alpha [H_2|\mu_1, \chi_1] = U - D_x$. Suppressing the subscripts, an active type-D agent maximizes expected payoff

$$\begin{aligned} V &= v(a) + \delta U - \delta q(a)(U - D) \\ &= u_0 + a^\theta(u - u_0) + \delta U - \delta \left(1 - \exp(-\lambda a)\right) \gamma\mu\chi(U - D) \end{aligned}$$

Figure 2 depicts the function $V(a)$ for different types D, and for $\lambda = 1$ and 2. Here, the computation assumes $\theta = 0.5$ so that $c = 0.5$ for $\lambda = 1$ and $c = 0.25$ for $\lambda = 2$. Other parameter values are $u = 1$, $u_0 = 0.5$, $\delta = 0.9$, $\gamma = 0.5$, $\mu = 0.6$. It is assumed that in equilibrium, $\chi = 0.33$ for $\lambda = 1$ and $\chi = 0.3$ for $\lambda = 2$, taking into account that equilibrium actions are (weakly) decreasing in λ. (These equilibrium values of χ can be justified given our degrees of freedom in choosing parameter I_0 and functions θ, h, and F). We find that $D = 2.82$ (resp. 3.83) is the type that is indifferent between the optimal action from $[0, c)$ and the maximal action 1 for $\lambda = 1$ (resp. 2). The figures illustrate that V is concave on $(0, c)$ and convex on $(c, 1)$, so that no agent would choose actions from the set $[c, 1)$. Polarization is more prominent for the higher λ $(= 2)$ case, as the two sets of equilibrium actions are more separated apart.

We conclude this subsection by a corollary of Theorem 2, the proof of which follows the proof of Theorem 2 straightforwardly and is hence omitted.

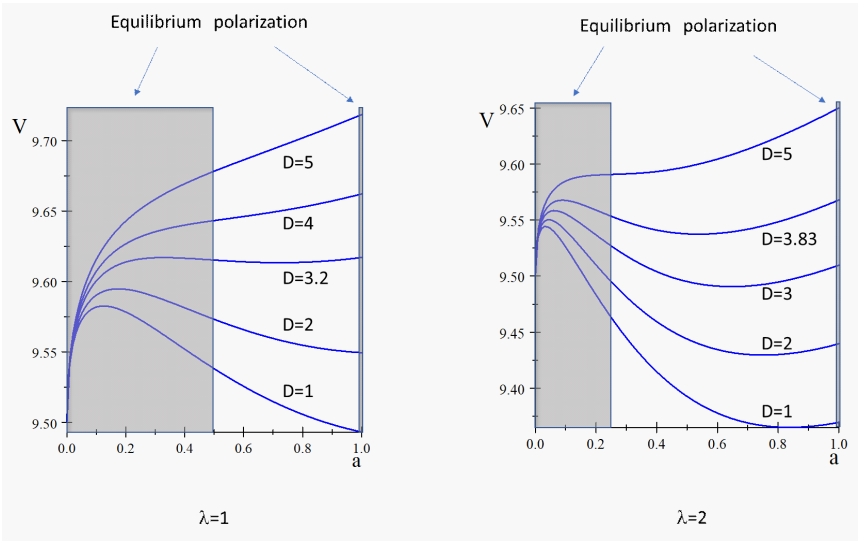

Figure 2: *Illustration of equilibrium polarization.* Actions maximizing V are either in the lower shaded set or in $\{1\}$. The types $D = 3.2$ and $D = 3.83$ are indifferent between the two choice sets for $\lambda = 1$ and $\lambda = 2$, respectively. Polarization is more prominent in that the lower action set shrinks to the left as λ increases.

Corollary 2 *Suppose Theorem 1 holds. Further assume that functions v and g satisfy the following single-crossing property: there exists $c \in [0, 1)$ such that*

$$\frac{v''(a)}{v'(a)} - \frac{g''(a)}{g'(a)} \begin{cases} \leq 0 \text{ for } a \leq c \\ > 0 \text{ for } a > c \end{cases}$$

Then the conclusions of Theorem 2 hold.

This corollary shows that Assumptions 2 and 3 are sufficient, but not necessary, for Theorem 2 and the equilibrium polarization can arise under more general circumstances.

4.3 Comparative statics and long-run equilibrium behavior

We now consider the comparative equilibrium behavior among agents endowed with different types. As the types are three-dimensional, it is impossible to have a complete ranking of the preferences and behavior based on agents' types. Nevertheless, each dimension of the types, δ, γ, and D has unambiguous implications for the equilibrium behavior.

Proposition 1 *The equilibrium public-response functions α_t has the following properties: for all $t \in \{1, 2, ..., T\}$,*

(i) α_t is nonincreasing in δ;

(ii) α_t is nonincreasing in γ;

(iii) α_t is nondecreasing in D.

Prediction (i) of this proposition might appear controversial. On one hand, it seems reasonable to predict higher levels of communicable activity among people who subscribe to *carpe diem* (pluck the day), or *yolo* (you live only once), as something close to their philosophy of life. A lower discount factor would be then consistent with their penchant to make the most of the present time and give little thought to the future. On the other hand, it would be misleading to predict higher levels of communicable activity among older people, as they might exhibit lower δ but at the same time higher γ and lower D—given that they are more likely to be sick or die upon infection. Therefore, it is worth emphasizing that all personal traits jointly influence agents' behavior. The main intuition why part (i) of the proposition holds, can be seen from the fact that once infected symptomatically, an agent's loss of utility ($U - D = \frac{u}{1-\delta} - D$) is positively related to their δ. Therefore, given two agents with the same γ and D, the agent with a higher δ has less incentive to take risks. Likewise, the disease probability when infected, γ, has a similar effect as δ because a higher γ means that the agent is more likely to suffer illness than agents with lower γ. The level of expected utility when diseased, D, is positively related to an agent's action because a higher D means a lower loss of utility ($U - D$).

By Proposition 1, we may call agents choosing actions below c in Theorem 2 "the more vulnerable" and the agents choosing action 1 "the less vulnerable." Notably, the prediction that active agents may choose polarized actions amidst a pandemic is based on the primitives v and g only, and is derived under common information and beliefs. For instance, the equilibrium predictions in Cases (i) and (ii) do not require agents to have polarized distribution for types or personal characteristics. Under any atomless distribution of the individual characteristics (δ, γ, D), Theorem 2(i)-(ii) hold for sufficiently high λ, even if the agents have infinitesimal type differences and are observationally "nearly" homogeneous.

Our second proposition shows the long-run behavior of the equilibrium and infection trends under an SPRE.

Proposition 2 *Then the following holds for any SPRE.*

(i) If $\mu_\tau \vartheta_\tau = 0$ *for some* $\tau \in \{1, 2, ..., T\}$, *then* $\alpha_t \equiv 1$ *and* $\vartheta_t = 0$ *for all* $t \geq \tau$.

(ii) Suppose $T = \infty$. *Then, the sequence of probabilities* $\Pr(\alpha_t(x, \mu^t) = 1) \to 1$ *uniformly over* X, *and* $\vartheta_t \to 0$, *as* $t \to \infty$.

Based on the COVID-19 experience, the random behavior of the coronavirus is highly unpredictable. We therefore focus on two broad scenarios, making no assumption about the dynamic behavior of the state variable μ_t. In scenario (i), either the transmission-intensity rate hits zero by a fluke or the infectious population shrinks to zero. While $\mu_\tau = 0$ is a chancy event, $\vartheta_\tau = 0$ can occur for various reasons. For instance, consider the extreme case when $\gamma \equiv 1$, i.e., all infected agents will be sick so that the last term in (12) equals zero, and $\theta \equiv 0$, i.e., the infected agents will be so sick as to have no capacity to transmit the disease to others. Then, the transmission risk defined in (12) vanishes, implying no new infection in the current period and therefore no infectious agents in the subsequent period. The severe Ebola virus disease, which could cause up to 90% of death but never became a pandemic, might be considered an example for this case. Another possibility for scenario (i) is that an extremely high rate of infection occurred in a period, causing a large population of infectious agents, or a large transmission risk in the subsequent period such that all the remaining active agents find it optimal to choose action 0. If a strict lockdown policy were feasible for the whole population, then, by Proposition 2(i), the population would be able to get rid of the virus quickly. (Of course, this is a big 'if' given the virtual impossibility of a worldwide, coordinated lockdown.) In scenario (ii) of the proposition, the virus persists. Then, the proposition predicts herd immunity as the only long-run equilibrium outcome.

5 Concluding Remarks

We have studied a dynamic game model with heterogeneous agents responding to the transmission risks of a widespread disease, and obtained three main results. The first result provides a rational explanation for the puzzling phenomenon of polarization in people's views and behaviors during a pandemic. We show that polarization crucially depends on the hazard rate of the exposure probability function: the higher

is the hazard rate the more prominent will be the polarization of actions in equilibrium. This result improves our understanding of human behavior in times of a pandemic, highlighting that the polarization phenomenon is a combined consequence of individual rationality, personal conditions, and the nature of disease transmission.

The second result stems from the fact that infected people can be asymptomatic and acquire immunity without noticing. With this possibility in mind, the agents in our model are enabled to rationally update their probability of disease based on their past actions, using the Bayes rule. The result, therefore, predicts an effect of past actions: the higher levels of the past actions, the more incentives an active agent has to take further high-risk actions. As time goes by, the result predicts that people will increasingly neglect the risk of disease and live their life as usual during a pandemic so that the long-run equilibrium moves toward herd immunity.

The third result shows that in equilibrium, the agents' actions are intimately related to their personal traits. Specifically, an agent's communicable activity is inversely related to their (1) discount factor, (2) probability of contracting the disease upon infection, and (3) expected loss of utility in the event of disease. These personal characteristics may be used as indications of an agent's vulnerability to transmission risk and help us better understand individual reactions to the pandemic.

In terms of policy considerations, our results corroborate the view of Acemoglu et al. (2021) and Gollier (2020) regarding the validity of targeting different types of agents with different policies. For instance, instead of full-fledged lockdowns, a government can consider playing a more constructive role by facilitating more vulnerable people to choose safety-first while allowing the less vulnerable to conduct life as usual. Since the lesser vulnerable population would not be a heavy social healthcare burden and will mostly acquire immunity through (asymptomatic) infections among themselves, the potential cost of facilitating such a policy could be conceivably much lesser than implementing a (partial) lockdown for everyone. The general framework and analysis presented here could be useful for policy-makers, organizations, and individuals to mitigate the potential damages in the future if such a crisis arises again.

6 Appendix 1: Proofs of Theorems

Consider a mathematically equivalent problem to (16) of maximizing

$$\begin{aligned} V_t - D &= v(a_t) - (1-\delta)D + \delta(1-q_t)\mathbb{E}_t^{\alpha}\left[V_{t+1}^* - D|\mu_t, \chi_t\right] \\ \forall t &\in \{1, ..., T\} \end{aligned}$$

Thus, $V_t - D$ can be expanded *as though* it was the expected sum of a sequence of discounted payoffs, with an associated probability of receiving the payoff in each period:

$$\left|\begin{array}{llll} \text{Period} & \text{Probability} & \text{Discounting} & \text{Payoff flow} \\ t & 1 & 1 & v(a_t) - (1-\delta)D \\ t+1 & 1-q_t & \delta & v(\alpha_{t+1}) - (1-\delta)D \\ \dots & \dots & \dots & \dots \\ t+s & (1-q_t)...(1-q_{t+s-1}) & \delta^s & v(\alpha_{t+s}) - (1-\delta)D \\ \dots & \dots & \dots & \dots \\ T+1 & (1-q_t)...(1-q_T) & \delta^{T-t+1} & U - D \end{array}\right| \tag{24}$$

where the last row vanishes as $T \to \infty$. Defining $\Pi_{r=1}^{0}\left(1 - q_{t+r}\right) = 1$ and taking expectation of the sum, we can write

$$V_t - D = \hat{\mathbb{E}}_t^{\alpha}\left[\begin{array}{c} v(a_t) - (1-\delta)D \\ + \sum_{s=1}^{T-t} \delta^s \left[\left(\prod_{r=1}^{s}(1-q_{t+r-1})\right)\left(v(\alpha_{t+s}) - (1-\delta)\,D\right)\right] \\ + \delta^{T-t+1}\left(\prod_{r=1}^{T-t+1}\left(1-q_{t+r-1}\right)\right)(U-D) \end{array}\right] \tag{25}$$

where $\hat{\mathbb{E}}_t^{\alpha}$ denotes the expectation operator over the random variables $(\mu_{t+1}, ..., \mu_T)$, conditional on the information at t. Again, due to discounting and bounded payoff, the last term in (25) vanishes as $T \to \infty$. The expression of $V_t - D$ in the above sequence effectively simplifies the problem, as shown in the following lemma.

Lemma 2 *The partial derivative in (19) satisfies, for all* $t \in \{1, 2, ..., T\}$,

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - \delta\frac{\partial q_t}{\partial a_t}\mathbb{E}_t^{\alpha}\left[H_{t+1}|\mu_t, \chi_t\right] \tag{26}$$

where H_{t+1} is a positive function defined recursively by

$$H_{t+s} = v(\alpha_{t+s}) - (1-\delta)\, D + \delta(1-p_{t+s})H_{t+s+1}$$

for $s \in \{1, ..., T-t\}$, with $H_{T+1} = U - D$ if $T < \infty$.

The proof of Theorems 1 and 2, and the subsequent propositions, also rely on a number of general properties of the cross-partial derivatives of V_t, as presented in Lemmas 3-5.

Lemma 3 *The function V_t as defined in (17) has the cross-partial derivative $\frac{\partial^2 V_t}{\partial a_t \partial a_s} \geq 0$ for all $x \in X$, and all $s, t \in \{1, 2, ..., T\}$ such that $s < t$.*

This lemma shows that V_t is supermodular in (a_s, a_t) for each active agent and for all $s < t$. This property implies a "risk-taking fosters risk-taking" effect in that higher levels of communicable activity in the past encourage the active agents to take higher levels of activity today. This result will be also useful for the analysis of comparative statics and equilibrium trends in Section 4.3.

Lemma 4 *Suppose Assumption 1 holds. The function V_t as defined in (17) has the cross-partial derivative $\frac{\partial^2 V_t}{\partial a_t \partial \chi_t} \leq 0$ for all $t \in \{1, 2, ..., T\}$.*

Lemma 5 *For all $t \in \{1, 2, ..., T\}$, given any (a^{t-1}, μ^t, χ_t), the function V_t as defined in (17) has the following properties:*

(i) $\frac{\partial^2 V_t}{\partial a_t \partial \delta} < 0$
(ii) $\frac{\partial^2 V_t}{\partial a_t \partial \gamma} < 0$
(iii) $\frac{\partial^2 V_t}{\partial a_t \partial D} > 0$

The above two lemmas show that V_t is submodular in (a_t, χ_t), (a_t, δ), (a_t, γ), and supermodular in (a_t, D). These properties have monotone comparative statics implications (Topkis, 1978; Milgrom and Shannon, 1994; Athey, 2002), as will be presented in the next section.

We are ready now to complete the proofs of Theorems 1 and 2.

Proof of Theorem 1. Step 1 (individual optimality). By Lemma 2, the problem of maximizing V_t as defined in (17) can be expressed by the program (suppressing other variables):

$$\max_{a_t \in [0,1]} V_t(a_t) = v(a_t) - \delta q_t(a_t) \mathbb{E}_t^{\alpha} \left[H_{t+1} | \mu_t, \chi_t \right] + V_t(0)$$

Since V_t is continuous in a_t and this decision variable is chosen in each period from a closed interval, the existence of an individual plan $(a_t^*)_{t=0}^T$ for each type of the active agents, taking a public plan α and the process of the transmission risk $(\chi_t)_{t=0}^T$ (yet to be established) as given, is guaranteed.

Starting with an arbitrary $T < \infty$. By backward induction on t, it is easily seen that given any past actions a^{t-1}, history μ^t, and current transmission risk χ_t of the environment, for each type x there exists an optimal solution

$$a_t^*(x, a^{t-1}, \mu^t, \chi_t) = \max\{\arg\max_{a_t \in [0,1]} V_t(x, a_t, a^{t-1}, \mu^t, \chi_t)\}$$

where, for multiple solutions, we choose the highest action for equilibrium. Equation (19) with the described corner solutions are therefore both necessary and sufficient for an individual optimal plan. Because $\delta_x \leq \bar{\delta} < 1$ and $U_x - D_x \leq \frac{u}{1-\bar{\delta}} < \infty$ for all $x \in X$, the last term in (25) converges uniformly to 0 as $T \to \infty$. Thus, the characterization of a_t^* in (19) with the described corner solutions extends to the infinite horizon case as $T \to \infty$.

Step 2 (equilibrium). Now we pick any $t \in \{1, 2, ..., T-1\}$ and define a mapping $\Gamma_t : [\theta_t, \vartheta_t] \to [\theta_t, \vartheta_t]$ by

$$\Gamma_t(\chi) = \theta_t + \int_X h(a_t^*(x, a^{t-1}, \mu^t, \chi)) j_t(x) dx, \quad \forall \chi \in [\theta_t, \vartheta_t]$$

where $\theta_t := \int_X \theta(x) i_t(x) dx$. By Lemma 4, $a_t^*(x, a^{t-1}, \mu^t, \chi)$ is nonincreasing in χ. This implies, given that $h(a_t^*(\cdot))$ is bounded between 0 and 1, that the mapping Γ_t is continuous. We now show that Γ_t is also continuous. Brouwer's fixed point theorem, combined with Γ_t being nonincreasing, implies a unique fixed point $\chi_t = \Gamma_t(\chi_t)$ that satisfies (20). The proof of the proposition is thus completed by (forward) induction on $t \in \{1, 2, ..., T\}$ for arbitrary $T \leq \infty$. ■

Proof of Theorem 2. Part 1 of the theorem is a straightforward corollary of Theorem 1 and Lemma 2. For Part 2, Case (iii) is covered by Theorem 1 and the proof of Case (i) follows the same lines of the proof of Case (ii). We therefore focus on proving Case (ii) with $c \in (0, 1)$. It is easily seen that if v exhibits CARA, then $c = 0$ or 1 by definition, implying Case (i) or Case (iii). So we assume that v exhibits DARA so that c is uniquely defined by $-\frac{v''(c)}{v'(c)} = \lambda \ (\equiv -\frac{g''}{g'})$ such that

$$-\frac{v''(a_t)}{v'(a_t)} \begin{cases} > \lambda & \text{if } a_t \in [0, c) \\ < \lambda & \text{if } a_t \in (c, 1] \end{cases} \tag{27}$$

$-\frac{v''}{v'} > (<)\lambda$ on $[0,c)$ $((c,1])$. Then, for the characterizations of equilibrium, it suffices to show that no action a_t in $[c,1)$ can be optimal.

As given in the proof of Theorem 1, the mathematical program of concern is

$$\max_{a_t\in[0,1]} V_t(a_t) = v(a_t) - \delta q_t(a_t)\mathbb{E}_t^{\alpha}\left[H_{t+1}|\mu_t,\chi_t\right] + V_t(0) \tag{28}$$

Since $q_t(a_t) = g(a_t)\gamma_x S_{t-1}\mu_t\chi_t$, differentiating V_t yields

$$\begin{aligned} V_t'(a_t) &= v'(a_t) - g'(a_t)M_t \\ \text{and } V_t''(a_t) &= v''(a_t) - g''(a_t)M_t \end{aligned}$$

with $M_t := \delta\gamma_x S_{t-1}\mu_t\chi_t\mathbb{E}_t^{\alpha}\left[H_{t+1}|\mu_t,\chi_t\right] > 0$. If at some point $a_t \in [0,1]$ the derivative $V_t'(a_t) = 0$ so that $v'(a_t) = g'(a_t)M_t$, then by (27) and $v' > 0$

$$V_t''(a_t) = v'(a_t)\left(\frac{v''(a_t)}{v'(a_t)} + \lambda\right)\begin{cases} < 0 & \text{if } a_t \in [0,c) \\ = 0 & \text{if } a_t = c \\ > 0 & \text{if } a_t \in (c,1] \end{cases}$$

Consequently, any interior solution to the program in (28) must lie in the interval $(0,c)$ and characterized by (22);[23] otherwise $\alpha_t(x,\mu^t) = 0$ or 1. We have thus completed the proof of equilibrium characterization for Case (ii).

What remains is to show that the equilibrium is unique. This amounts to showing that *almost all* types of active agents should find their equilibrium action $\alpha_t(x,\mu^t)$ uniquely optimal in all period t. From the above analysis, the equilibrium actions $\alpha_t(x,\mu^t) \in [0,c)\cup\{1\}$ are unique unless

$$\max_{a_t\in[0,c)} V_t(a_t,x) = V_t(1,x). \tag{29}$$

Let B_t denote the set of types $x = (\delta,\gamma,D)$ satisfying the condition in (29). We show that the type distribution being atomless implies that B_t has a zero measure in any period $t \geq 1$.

First, consider types that differ only in one dimension of the type vector, e.g., D. Denote by $a^t = (a_1,...,a_t)$ and $\hat{a}^t = (\hat{a}_1,...,\hat{a}_t)$ the optimal history of actions up to t for the types (δ,γ,D) and $(\delta,\gamma,\hat{D})$, respectively, such that (29) holds and

[23] Note that $a_t = c$ cannot be optimal because $V_t'(c) = 0$ implies $V_t(a_t) > V_t(c)$ for all $a_t \in (c,1]$.

w.l.o.g. suppose $\hat{D} > D$. Then, by Lemmas 3 and 5, $\hat{a}^t \geq a^t$, and V_t is strictly supermodular in (a^t, D). Thus, we have

$$V_t(\hat{a}^{t-1}, 1, \hat{D}) - V_t(\hat{a}^{t-1}, \hat{a}_t, \hat{D}) > V_t(a^{t-1}, 1, D) - V_t(a^{t-1}, \hat{a}_t, D)$$

But a_t being optimal for type D in period t implies that the right side of the inequality is greater than $V_t(a^{t-1}, 1, D) - V_t(a^{t-1}, a_t, D) = 0$. It follows that when any type D is indifferent between an interior optimal action a_t and 1, no other type $\hat{D} \neq D$ can be indifferent between an interior optimal action $\hat{a}_t$ and 1.

Extending the above arguments, it can be shown by Lemma 5 that for any two types $x, y \in X$ such that $\delta_y \leq \delta_x, \gamma_y \leq \gamma_x, D_y \geq D_x$ with at least one inequality strict, if type x is indifferent between an interior optimal action $\alpha_t(x, \mu^t)$ and 1, then type y will strictly prefer action 1.

Now, by contradiction, suppose $F_t(B_t) > 0$. Then, given $X \subset \mathbb{R}^3$, we can partition B_t into 2^3 subsets with positive measures and find out two of these:

$$\begin{aligned} B_t^+ &= \{x \in B_t : \delta_x \leq \delta_0, \gamma_x \leq \gamma_0, D_x \geq D_0)\} \\ B_t^- &= \{x \in B_t : \delta_x \geq \delta_0, \gamma_x \geq \gamma_0, D_x \leq D_0)\} \end{aligned}$$

by properly choosing the vector $x_0 = (\delta_0, \gamma_0, D_0) \in X$. Since x_0 has a zero measure, removing x_0 from these sets preserves the measures of these subsets while causing at least one of the inequalities to hold strictly for $B_t^+ \backslash \{x_0\}$ and $B_t^- \backslash \{x_0\}$. This contradicts the definition of B_t so that we must have $F_t(B_t) = 0$. ■

7 Appendix 2: Proofs of Lemmas 2-5

Proof of Lemma 1. We derive $(i_t, j_t, f_t, \chi_t, p_t, q_t)$ by induction. For $t = 0$, we are given $\mu_0 \in (0, 1)$ and $I_0 \in (0, 1)$. Define $f_0 = f$, $p_0 \equiv I_0$ and $S_{-1} \equiv 1$, so that

$q_0(x,\mu^0)=\gamma_x I_0$. Subsequently, for $t=1$ and given any $\mu_1\in(0,1]$, we derive

$$\begin{aligned}
f_1(x) &= (1-I_0)f_0(x)\\
i_1(x) &= \gamma_x I_0 f_0(x)\\
j_1(x) &= (1-\gamma_x)I_0 f_0(x)\\
\chi_1(\mu^1) &= \int_X \theta(x)i_1(x)dx+\int_X h(\alpha_1(x,\mu^1))j_1(x)dx,\\
p_1(x,\mu^1) &= \mu_1 g(\alpha_1(x,\mu^1))\chi_1(\mu^1)\\
q_1(x,\mu^1) &= \gamma_x S_0 p_1(x,\mu^1) \text{ where } S_0=S_{-1}\frac{1-p_0}{1-q_0}
\end{aligned}$$

By induction, suppose for $t\in\{1,2,...T-1\}$, the states $(i_s,j_s,f_s,\chi_s,p_s,q_s)$ are well defined for $s=0,1,...,t-1$. Consider now period t. Since for each type $x\in X$, a fraction $p_{t-1}(x,\mu^{t-1})$ of the agents were infected in period $t-1$, we have

$$\begin{aligned}
i_t(x) &= \gamma_x p_{t-1}(x,\mu^{t-1})f_{t-1}(x)\\
j_t(x) &= (1-\gamma_x)p_{t-1}(x,\mu^{t-1})f_{t-1}(x)
\end{aligned}$$

and the density of susceptible types shrinks to

$$f_t(x)=(1-p_{t-1}(x,\mu^{t-1}))f_{t-1}(x)$$

It follows that

$$\chi_t(\mu^t)=\int_X \theta(x)i_t(x)dx+\int_X h(\alpha_t(x,\mu^t))j_t(x)dx$$

and for all $x\in X$,

$$\begin{aligned}
p_t(x,\mu^t) &= \mu_t g(\alpha_t(x,\mu^t))\chi_t(\mu^t)\\
q_t(x,\mu^t) &= \gamma_x S_{t-1}(x,\mu^{t-1})p_t(x,\mu^t) \text{ where } S_{t-1}=S_{t-2}\frac{1-p_{t-1}(x,\mu^{t-1})}{1-q_{t-1}(x,\mu^{t-1})}
\end{aligned}$$

Note that since f and α_t are measurable functions of x, so are $(i_t,j_t,f_t,\chi_t,p_t,q_t)$ for all $t\in\{1,2,...,T\}$. ■

Proof of Lemma 2. Fix any $t\in\{1,2,...,T\}$. We show by induction on $s=1,...,T-t$ that the product term in (24) has a derivative

$$\frac{\partial}{\partial a_t}\prod_{r=1}^{s}(1-q_{t+r-1})=-\gamma S_{t-1}p_t'(a_t)\prod_{r=1}^{s-1}(1-p_{t+r}) \tag{30}$$

where $\Pi_{r=1}^{0}\,(1-p_{t+r})$ is defined as unity. For $s=1$, from (6) we have

$$1-q_t = 1-\gamma S_{t-1}p_t(a_t) \tag{31}$$

so that $\frac{\partial(1-q_t)}{\partial a_t} = -\gamma S_{t-1}p_t'(a_t)$, conforming (30). Now, supposing (30) holds for arbitrary $s \geq 1$, let us consider the case with $s+1$. Noting from (5) and (6) that

$$q_{t+s} = \gamma S_{t+s-1}p_{t+s} = \gamma S_{t-1}p_{t+s} \times \frac{\prod_{r=1}^{s}(1-p_{t+r-1})}{\prod_{r=1}^{s}(1-q_{t+r-1})}$$

Therefore

$$\begin{aligned}
\prod_{r=1}^{s+1}(1-q_{t+r-1}) &= \prod_{r=1}^{s}(1-q_{t+r-1}) - q_{t+s}\prod_{r=1}^{s}(1-q_{t+r-1}) \\
&= \prod_{r=1}^{s}(1-q_{t+r-1}) - \gamma S_{t-1}p_{t+s}\prod_{r=1}^{s}(1-p_{t+r-1}) \\
&= \prod_{r=1}^{s}(1-q_{t+r-1}) - \gamma S_{t-1}p_{t+s}(1-p_t)\prod_{r=1}^{s-1}(1-p_{t+r})
\end{aligned}$$

Differentiating w.r.t. a_t and invoking the induction hypothesis, the first term on the right side of the third equation satisfies (30). The second term depends on a_t only through p_t. Thus,

$$\begin{aligned}
&\frac{\partial}{\partial a_t}\prod_{r=1}^{s+1}(1-q_{t+r-1}) \\
= \;& -\gamma S_{t-1}p_t'(a_t)\prod_{r=1}^{s-1}(1-p_{t+r}) + \gamma S_{t-1}p_t'(a_t)\prod_{r=1}^{s-1}(1-p_{t+r})p_{t+s} \\
= \;& -\gamma S_{t-1}p_t'(a_t)\prod_{r=1}^{s}(1-p_{t+r})
\end{aligned}$$

This shows that (30) holds for all $s \geq 1$. Now, differentiating (25) with respect to a_t using (30), and defining H_{t+1} by

$$H_{t+1} = \sum_{s=1}^{T-t}\prod_{r=1}^{s-1}(1-p_{t+r})\delta^s\left(v(\alpha_{t+s}) - (1-\delta)\,D\right) \tag{32}$$

$$+\left(\prod_{r=1}^{T-t+1}(1-p_{t+r-1})\right)\delta^{T-t}\,(U-D) \tag{33}$$

one can readily verify that (22) holds, where the equivalent recursive expression of H_{t+1} derives from (32)–(33).

Finally, to determine whether H_{t+1} is positive, we have $H_{T+1} = U - D > 0$. Since $0 \leq p_{t+s} \leq 1 - \rho_{t+s} < 1$ and $v(a) \geq u_0 \geq (1-\delta)D$ For all $a \in [0,1]$, by backward induction, we derive

$$\begin{aligned} H_{t+s} &= v(\alpha_{t+s}) - (1-\delta)\, D + \delta(1-p_{t+s})H_{t+s+1} \\ &\geq \delta(1-p_{t+s})H_{t+s+1} > 0 \\ \text{for all } t &\in \{1,2,...,T\},\ s \in \{1,...,T-t\} \end{aligned}$$

■

Proof of Lemma 3. Pick any $t \in \{2,...,T\}$ and $s \in \{1,...,t-1\}$. Recall that $q_t = \gamma S_{t-1} p_t$, so that the cross-partial derivative

$$\frac{\partial^2 q_t}{\partial a_t \partial S_{t-1}} = \gamma p_t' > 0$$

Notice further that S_{t-1} can be written as (define $\prod_{r=1}^{0}(1-p_{s+r}) = 1$)

$$S_{t-1} = A \frac{(1-p_s)}{\prod_{r=1}^{t-s}(1-q_{s+r-1})}$$

where $A := S_{s-1} \prod_{r=1}^{t-s-1}(1-p_{s+r}) > 0$ is independent of a_s. Therefore,

$$\begin{aligned} \frac{\partial S_{t-1}}{\partial a_s} &= A \frac{\partial}{\partial a_s} \frac{(1-p_s)}{\prod_{r=1}^{t-s}(1-q_{s+r-1})} \\ &= A \frac{-p_s' \prod_{r=1}^{t-s}(1-q_{s+r-1}) - (1-p_s)\frac{\partial}{\partial a_s}\prod_{r=1}^{t-s}(1-q_{s+r-1})}{\left(\prod_{r=1}^{t-s}(1-q_{s+r-1})\right)^2} \end{aligned} \tag{34}$$

By (30) in the proof of Lemma 2, we derive

$$\frac{\partial}{\partial a_s}\prod_{r=1}^{t-s}(1-q_{s+r-1}) = -p_s' S_{s-1}\gamma \prod_{r=1}^{t-s-1}(1-p_{s+r})$$

It follows that

$$\begin{aligned}
& -p'_s \prod_{r=1}^{t-s}(1-q_{s+r-1}) - (1-p_s)\frac{\partial}{\partial a_s}\prod_{r=1}^{t-s}(1-q_{s+r-1}) \\
= \; & -p'_s \prod_{r=1}^{t-s}(1-q_{s+r-1}) + (1-p_s)p'_s S_{s-1}\gamma \prod_{r=1}^{t-s-1}(1-p_{s+r}) \\
= \; & \left(-p'_s + (1-p_s)p'_s S_{s-1}\gamma \frac{\prod_{r=1}^{t-s-1}(1-p_{s+r})}{\prod_{r=1}^{t-s}(1-q_{s+r-1})} \right) \prod_{r=1}^{t-s}(1-q_{s+r-1}) \\
= \; & -(1-\gamma S_{t-1})\, p'_s \prod_{r=1}^{t-s}(1-q_{s+r-1}) \qquad (35)
\end{aligned}$$

Now, substituting A and (35) into (34) yields

$$\frac{\partial S_{t-1}}{\partial a_s} = -S_{t-1}\frac{(1-\gamma S_{t-1})\, p'_s}{(1-p_s)} < 0$$

because $0 < \gamma, S_{t-1}, p_s < 1$. Thus, from (26) we derive

$$\frac{\partial^2 V_t}{\partial a_t \partial a_s} = -\delta \frac{\partial^2 q_t}{\partial a_t \partial S_{t-1}} \frac{\partial S_{t-1}}{\partial a_s} \int_0^1 H_{t+1} d\Phi_t > 0$$

■

Proof of Lemma 4. For any $t \in \{1, ..., T\}$, we can write

$$\begin{aligned}
\frac{\partial V_t}{\partial a_t} &= v'(a_t) - \gamma S_{t-1}\mu_t g'(a_t)\chi_t \int H_{t+1}\varphi_t(\mu_{t+1}|\mu_t, \chi_t) d\mu_{t+1} \\
&= v'(a_t) - \gamma S_{t-1}\mu_t g'(a_t)\chi_t \int_{\varphi_t > 0} H_{t+1}\varphi_t(\mu_{t+1}|\mu_t, \chi_t) d\mu_{t+1}
\end{aligned}$$

It follows that

$$\begin{aligned}
\frac{\partial^2 V_t}{\partial a_t \partial \chi_t} &= -\gamma S_{t-1}\mu_t g'(a_t) \int_{\varphi_t > 0} H_{t+1}\varphi_t d\mu_{t+1} - \gamma S_{t-1}\mu_t g'(a_t)\chi_t \int_{\varphi_t > 0} H_{t+1}\frac{\partial \varphi}{\partial \chi_t} d\mu_{t+1} \\
&= -\gamma S_{t-1}\mu_t g'(a_t) \int_{\varphi_t > 0} \left(H_{t+1}\varphi_t \times \left(1 + \chi_t \frac{\partial \varphi / \partial \chi_t}{\varphi} \right) \right) d\mu_{t+1}
\end{aligned}$$

Because $H_{t+1} > 0$ and $0 < \chi_t < 1$, Assumption 1 implies $1 + \chi_t \frac{\partial \varphi / \partial \chi_t}{\varphi} > 0$ and therefore $\frac{\partial^2 V_t}{\partial a_t \partial \chi_t} \leq 0$ for all $t \in \{1, 2, ..., T\}$ and $T \leq \infty$. ■

Proof of Lemma 5. We fix the equilibrium response functions $\alpha_1, ..., \alpha_T$ and check the sign of the partial derivatives of $\frac{\partial V_t}{\partial a_t}$ w.r.t. (δ, γ, D) by backward induction. Write $\frac{\partial V_t}{\partial a_t}$ as

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - \delta \frac{\partial q_t}{\partial a_t} \int_0^1 H_{t+1} d\Phi_t.$$

(i) Differentiating $\frac{\partial V_t}{\partial a_t}$ w.r.t. δ yields

$$\frac{\partial^2 V_t}{\partial a_t \partial \delta} = -\frac{\partial q_t}{\partial a_t} \int_0^1 H_{t+1} d\Phi_t - \delta \frac{\partial q_t}{\partial a_t} \int_0^1 \frac{\partial}{\partial \delta} H_{t+1} d\Phi_t$$

Because $H_{t+1} > 0$ by Lemma 2, it suffices to show $\frac{\partial H_{t+1}}{\partial \delta} \geq 0$. As shown in Lemma 2, the functions H_{t+s} are recursively defined by, for $s = 1, ..., T - t$,

$$H_{t+s} = v(\alpha_{t+s}) - (1 - \delta) D + \delta(1 - p_{t+s}) H_{t+s+1}$$

Differentiating w.r.t. δ yields

$$\frac{\partial}{\partial \delta} H_{t+s} = D + (1 - p_{t+s}) H_{t+s+1} + \delta(1 - p_{t+s}) \frac{\partial}{\partial \delta} H_{t+s+1}$$

Since $D \geq 0$ and $\frac{\partial H_{T+1}}{\partial \delta} = 0$, backward induction implies

$$\frac{\partial^2 V_t}{\partial a_t \partial \delta} < 0, \ t \in \{1, 2, ..., T\}$$

(ii) Differentiating $\frac{\partial V_t}{\partial a_t}$ w.r.t. γ yields

$$\frac{\partial^2 V_t}{\partial a_t \partial \gamma} = -\delta \frac{\partial^2 q_t}{\partial a_t \partial \gamma} \int_0^1 H_{t+1} d\Phi_t$$

From $q_t = \gamma S_{t-1} p_t$, we derive

$$\frac{\partial^2 q_t}{\partial a_t \partial \gamma} = \left(S_{t-1} + \gamma \frac{\partial}{\partial \gamma} S_{t-1} \right) p_t'$$

Because p_s is independent of γ and q_s increasing in γ,

$$\frac{\partial S_{t-1}}{\partial \gamma} = \frac{\partial}{\partial \gamma} \frac{(1 - p_0)(1 - p_1)...(1 - p_{t-1})}{(1 - q_0)(1 - q_1)...(1 - q_{t-1})} > 0$$

Consequently, we have

$$\frac{\partial^2 V_t}{\partial a_t \partial \gamma} < 0, \ t \in \{1, 2, ..., T\}$$

(iii) Differentiating $\frac{\partial V_t}{\partial a_t}$ w.r.t. D yields

$$\frac{\partial^2 V_t}{\partial a_t \partial D} = -\delta \frac{\partial q_t}{\partial a_t} \int_0^1 \frac{\partial}{\partial D} H_{t+1} d\Phi_t.$$

We have $\frac{\partial H_{T+1}}{\partial D} = \frac{\partial (U-D)}{\partial D} = -1$, implying

$$\frac{\partial}{\partial D} H_{t+s} = -(1-\delta) + \delta(1-p_{t+s}) \frac{\partial}{\partial D} H_{t+s+1} < 0$$

and therefore

$$\frac{\partial^2 V_t}{\partial a_t \partial D} > 0, \ t \in \{1, 2, ..., T\}$$

■

8 Appendix 3: Proofs of the Propositions

Proof of Proposition 1. We prove the proposition by induction, using the results of Lemma 5.

(i) Starting from $t = 1$. Suppose the two types x and y differ only in $\delta_x > \delta_y$. Notice that y can always choose to mimic the plan of x. However, y can be better. For instance, by Lemma 5, $\frac{\partial^2 V_1}{\partial a_t \partial \delta} < 0$ implies that y would be better off by switching from the plan of x to a plan that differs in period 1, with $\alpha_1(y, \mu^1) \geq \alpha_1(x, \mu^1)$. Therefore, we let y choose $\alpha_1(y, \mu^1)$.

According to Lemma 3, in period $t = 2$, the type-y agents who remain active have even less incentives to mimic the plan of x because $\alpha_1(y, \mu^1) \geq \alpha_1(x, \mu^1)$ implies $S_1(y) \leq S_1(x)$. This advantage is reflected in $q_2(y, a_2, \mu^2) \leq q_2(y, a_2, \mu^2)$, which, together with $\frac{\partial^2 V_1}{\partial a_1 \partial \delta} < 0$, suggests that y can, again, do better by switching from the plan of x in period 2 to a higher level of action $\alpha_2(y, \mu^1) \geq \alpha_2(x, \mu^1)$.

Repeating the same argument for $t = 3, ..., T$, we can show that in all periods, y can do better by choosing a (weakly) higher action level than x. Note that the derived actions for y are not necessarily equilibrium actions. These are used only to indicate the direction of change as a result of increasing δ.

For conclusions (ii) and (iii), similar arguments apply, and hence, they are omitted. ■

Proof of Proposition 2. (i) Suppose $\mu_\tau \vartheta_\tau = 0$ for some $0 < \tau < \infty$. Then from (3) $\chi_\tau(\mu^\tau) = 0$, which implies $\alpha_\tau \equiv 1$ and no new infection in period τ and therefore $\vartheta_{\tau+1} = 0$. The conclusion thus holds by induction.

(ii) By contradiction, suppose $\vartheta_t \to 0$ were false. Then, $\exists \varepsilon > 0$ such that for all $\tau > 0$, there exists $t(\tau) \geq \tau$ such that $\vartheta_{t(\tau)} > \varepsilon$.

Consider now the process $(\rho_t)_{t=0}^{\infty}$ of the size of the innocuous population. It is nondecreasing and bounded from above by 1. So the process has a limit $\bar{\rho} \leq 1$ as $t \to \infty$. It implies that $\forall \hat{\varepsilon} > 0$, there exists $\hat{\tau} > 0$ such that

$$\bar{\rho} - \hat{\varepsilon} \leq \rho_t \leq \bar{\rho} \text{ for all } t \geq \hat{\tau}.$$

But, choosing $\hat{\varepsilon} < \varepsilon$ and $\tau > \hat{\tau}$, we derive

$$\rho_{t(\tau)} \geq \rho_\tau + \varepsilon \geq \bar{\rho} - \hat{\varepsilon} + \varepsilon > \bar{\rho}$$

This contradiction shows that $\vartheta_t \to 0$ as $t \to \infty$.

Now to show $\Pr(\alpha_t(x, \mu^t) = 1) \to 1$, we need to show that $\forall \varepsilon > 0$, $\exists \tau > 0$ such that for all $t \geq \tau$ and $\mu^t \in (0,1]^t$, $\Pr(\alpha_t(x, \mu^t) = 1) > 1 - \varepsilon$, or, equivalently, $1 - \Pr(\alpha_t(x, \mu^t) = 1) = \Pr(\alpha_t(x, \mu^t) < 1) < \varepsilon$.

$$\begin{aligned}
\frac{\partial V_t}{\partial a_t} &= v'(a_t) - \delta \frac{\partial q_t}{\partial a_t} \mathbb{E}_t^\alpha \left[H_{t+1} | \mu_t, \chi_t \right] \\
&\quad \text{Integrating over } (a, 1) \text{ yields} \\
&\quad V_t(1) - V_t(a) \\
&= v(1) - v(a) - \left[g(1) - g(a) \right] \delta \gamma S_{t-1} \mu_t \chi_t(\mu^t) \mathbb{E}_t^\alpha \left[H_{t+1} | \mu_t, \chi_t \right] \\
&\geq v(1) - v(a) - \vartheta_t \left[g(1) - g(a) \right] \delta \gamma S_{t-1} \mu_t \mathbb{E}_t^\alpha \left[H_{t+1} | \mu_t, \chi_t \right] \quad (\chi_t(\mu^t) \leq \vartheta_t) \\
&\geq v(1) - v(a) - \vartheta_t \left[g(1) - g(a) \right] \sup_{x \in X} (U_x - D_x) \\
&> 0 \text{ for } t \text{ sufficiently large, because } \vartheta_t \to 0
\end{aligned}$$

Consequently, for all ε such that

$$\frac{v(1) - v(a_t)}{\left[g(1) - g(a) \right] \sup_{x \in X} (U_x - D_x)} > \varepsilon > 0,$$

there exists $\tau > 0$ such that for all $t \geq \tau$, $\vartheta_t < \varepsilon$ and therefore $V_t(1) - V_t(a) > 0$ for all $x \in X$. Since a can be chosen arbitrarily close to 1, we conclude that $\Pr(\alpha_t(x, \mu^t) < 1)$ converges to 0 uniformly on X as $t \to \infty$. ■

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