Path-dependent Preferences and Polarized Public Response to Pandemics*

Audrey Hu
City University of Hong Kong

Xu Han
City University of Hong Kong

Joyce Liu
Peking University

August 10, 2022

*We are much indebted to Jim Bergin, Piotr Dworczak, Steve Matthews, Andy Skrzypacz, Wenji Xu, Yating Yuan and Liang Zou for their invaluable comments, inputs, and advice, without which the paper would not have come to fruition. Our gratitude also extends to Te Bao, Jan Boone, Dongkyu Chang, Yi-Chun Chen, Francesc Dilme, Christian Gollier, Ilwoo Hwang, Shenzhe Jiang, Yang Jiao, Jinwoo Kim, Nenad Kos, Stephan Lauermann, Jiangtao Li, Jingfeng Lu, Theo Offerman, Masao Ogaki, Marco Ottaviani, Yangbo Song, Dezsö Szalay, Zaifu Yang, Andy Zapechelnyuk,Wenbo Zhao, Yu Zheng, Junjie Zhou, the seminars participants of Chinese University of Hong Kong, National University of Singapore, Osaka University, Seoul National University, University of Bonn and York University, and conference participants at Asian meeting of the Econometrics society (Tokyo) and Conference on Mechanism and Institution Design (Singapore), for their helpful comments and feedbacks.

Abstract

People with liberal traditions exhibit polar opposite views and behavior toward COVID-19. We analyze this phenomenon by employing a dynamic game model involving stochastic transmission-intensity rates, asymptomatic infections, and heterogeneous agents making communicable-activity decisions in each period under disease uncertainty. Active agents freely choose communicable activities that increase their utility flows along with infection probabilities. Our analysis reveals the following: (1) The polarized public response to the pandemic arises when the disease-probability function is more concave than the agents' utility function for communicable activities, which suggests that polarization can be rooted in individual rationality. (2) Asymptomatic infection implies a path-dependent disease probability that declines with agents' past activities, which makes sense of a gradually relaxing lockdown policy even when the transmission intensity remains the same. (3) Monotone comparative statics results suggest that agents with lower discount factors, lower probability of being sick upon infection, or lower expectation of suffering upon being sick tend to choose higher equilibrium activities. (4) If the virus persists, then the only long-run equilibrium outcome without government intervention is herd immunity.

Keywords: Stochastic dynamic programming; probability function of disease; asymptomatic infection; polarized public responses

JEL Classification: C73; D01; D82; C25

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

— EURONEWS.

1 Introduction

The COVID-19 pandemic has imposed on every economic agent a decision-making problem: To what extent should one 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 catching the disease? Casual observation and survey data suggest a stark polarized phenomenon: most agents, whenever possible, either take maximal measures or do not take any preventive measure at all.

In this paper, we study a new model of rational decision-making during a pandemic and show that it provides a rationale for the observed polarization of people's responses to the pandemic. In our dynamic public-response model, heterogeneous agents choose varying degrees of communicable, or transmissible, activities that increase their utility along with the probability of being exposed to the transmission risk. We show natural conditions on the exposure-probability function that lead to a polarized equilibrium in which everyone chooses either the minimum or the maximum activities in every period. Our results do not rely on behavioral biases or boundedly rational agents, and do not require polarization of underlying preferences or beliefs. Instead, we show that any atomless distribution of agents' types can lead to polarized equilibrium responses to the pandemic.

A typical example demonstrating the polarized attitudes towards COVID-19 is the controversy concerning whether or not face masks should be worn in public. Figure 1 shows the mask-wearing behavior of residents in four leading European capitals: Berlin, London, Madrid, and Paris, during the early stage of the pandemic in April 2020.[1] A striking commonality is observed across the four cities, as over 70

[1]During this period, mask-wearing was not mandated in London, Madrid, or Paris and people could take protective actions according to their own decisions. The results are obtained using Yougov's survey data described in the Online Appendix, where we show similar results in 12 cities

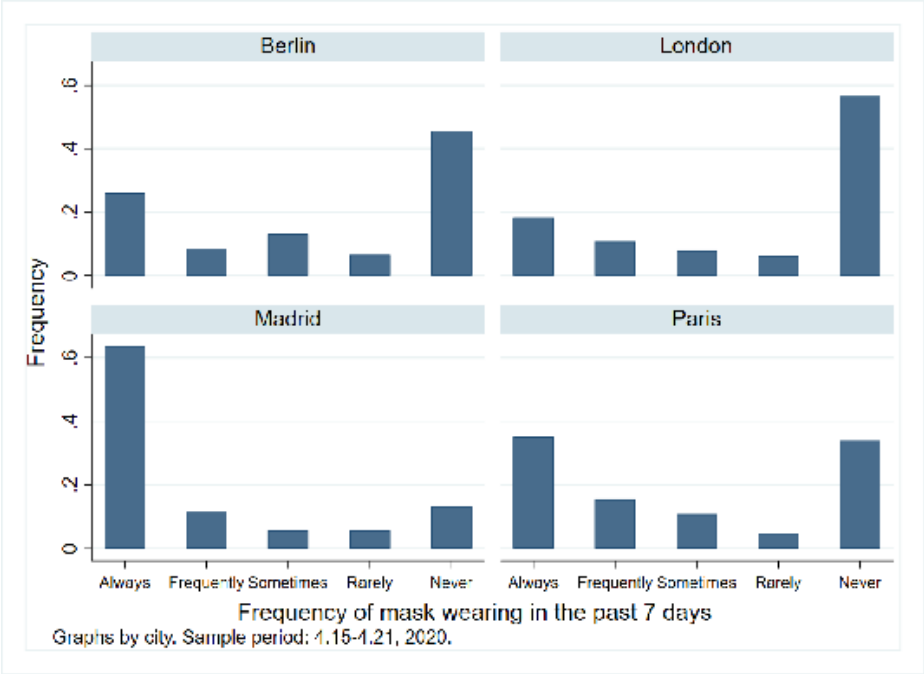

Figure 1: People's mask-wearing frequency in four cities

percent of residents choose the extreme option of either "always wearing a mask in public" or "never wearing a mask in public." This example is by no means unique. Our empirical investigation will show that such extreme choices were prevalent also in people's choices of social activities, out dining, and exercising in public indoor spaces in the U.S. during the pandemic.

Polarization in the face of a pandemic has been known to cause social unrest and instability.[2,3] Understanding the causes that may underlie such polarized

with vast differences in cultural background, political structure and ethnicity.

[2]Anecdotal evidence suggests that wars, revolts, and social unrest frequently occurred during and after a severe plague or epidemic, influencing the course of human history (Hays 2005). The highly referenced Antonine plague (165–180 AD), Justinian plague (541–549 AD), the bubonic plague of the 14th Century, and the 1918 influenza pandemic are but a few examples. It has been widely observed that epidemics sow seeds of conflict or act as incubators of severe social disorders or institutional change (e.g., Snowden 2020a, 2020b; Censolo and Morelli 2020; Jedwab et al. 2021).

[3]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

behavior is important not only for their potential policy implications but also for improving our perception of the far-reaching implications of the COVID-19 crisis in its aftermath. To reduce the unrest and hence the costs of the pandemic, policy makers need to understand the root causes so they can properly evaluate different policies. Our paper contributes to this discussion by offering an explanation for polarized responses without polarization of underlying preferences.

Another contribution of this paper is a formal analysis of the effects of asymptomatic infections, i.e., that agents can be unknowingly infected without symptoms.[4] These agents, disguised by their good health while engaging in communicable activities, could conceivably become a major source of disease transmission. Although asymptomatic transmission of the coronavirus disease is a well observed fact, its effects have received no formal analysis in the existing literature. We incorporate this fact in our model by allowing each infected agent to have a type-dependent probability of being asymptomatic. As a result, in every period, the infectious population consists of previously infected agents who were either symptomatic or asymptomatic. In the absence of continuous and effective testing, a crucial distinction emerges between these two groups of infectious agents: the former knows the fact that they are infectious and the latter does not. Consequently, while the infectiousness of the symptomatic group can be limited by exogenous possibilities such as quarantine, hospitalization, or death, the infectiousness of the asymptomatic group depends endogenously on the agents' communicable activities.

To capture the varying degrees of infectiousness among the infected agents, we

around the globe. About two-thirds of these were protests against the authorities' anti-coronavirus measures, one-third demanded greater protection and attention, and a few noteworthy instances included public protests calling for solidarity. "While COVID-19 had its biggest impact on civil unrest and political instability, it also significantly invoked feelings of unsafety and interpersonal violence. Some of these negative effects are likely to last for years to come." (VISION OF HUMANITY report *https://www.visionofhumanity.org/the-impact-of-the-covid-19-pan)demic-on-peace/*)

[4]For instance, China reports daily symptomatic and asymptomatic infections under compulsory testing. Recent data from the Shanghai lockdown suggest that asymptomatic infections are about 10 times more than symptomatic infections.

introduce a general function(al) χ_t as a numerical representation of the transmission risk of the environment, defined by the sum of the infectiousness of the symptomatically and asymptomatically infected population in the preceding period. In our model, each active agent can decide their probability of exposure to the transmission risk in any period by choosing a level of communicable activities in that period, taking into account the possibility of being unknowingly infected in the past without a symptom. Thus, for all agents who have not been sick and who have been exposed to the transmission risk with positive probabilities in the past, the Bayes rule implies that they are more likely to be asymptomatic and therefore safe. This fact leads to a sequence of path-dependent expected payoffs for each active agent and significantly complicates our analysis. However, by transforming the problem into a mathematically equivalent one, we find a way to simplify the problem and obtain sharper characterizations of equilibrium.

In Section 2, we discuss related literature along with the new aspects of this study and results. Section 3 presents the pandemic-response model. Section 4 presents the main results of the analysis, Propositions 1-4 and Corollaries 1-2. Section 5 investigates people's behavior empirically. And Section 6 concludes. All proofs of the lemmas and propositions are contained in the Appendix.

2 Related literature and New Results

The polarization in equilibrium responses to the pandemic, one of the main results from this study (Proposition 1 and Corollary 1), is new. Some previous studies document the differences from aggregate data in the Americans' responses to the pandemic due to partisan polarization, as seen in the Trump-Biden transition era in the United States.[5] We are not aware of any previous work that is directly related

[5]For instance, see Allcott et al. (2020); Grossman et al. (2020). Allcott et al. (2020) find significant partisan differences in Americans' response to the COVID-19 pandemic. To explain such differences, they assume that party members have limited rationality and can be influenced by their political leaders.

to our polarization result, except the frequent discussions on social media relating polarization in attitudes toward the pandemic to the partisan polarization in US. Such political explanation is limited, however, by the fact that US is the only OECD country that sees sharp partisan polarization (Boxell et. al. 2022). Public opinions and behaviors in response to the pandemic were not less polarized in Europe than in America.

Our modelling and analysis of the asymptomatic infection and path-dependent disease probabilities are also new. Given the abundant evidence proving that asymptomatic infection plays an important role in spreading an epidemic, we present a formal analysis by allowing the population to consist of agents who have been infected but show no symptoms. The possibility of asymptomatic infections is well recognized in a number of studies, but there has been no formal analysis of their effects. For instance, in his study of the HIV epidemics, Kremer (1996, footnote 7) noted that older people who had more previous sexual partners were more likely to be infected; therefore, being healthy makes them more likely to increase their risk-taking behavior. Although Kremer (1996) did not explicitly model asymptomatic infection, his idea is perfectly in line with our Bayes updating of the disease probability for each active agent.

The way our study is related to the existing literature is better discussed with each specific aspect of the study.

Stochastic transmission-intensity rate and matching function. There are two mainstream approaches to modelling the spread of epidemic diseases, the SIS (susceptible-infected-susceptible) approach and the SIR (susceptible, infected, recovered) approach. The former was popular around the turn of this century, which is well suited for estimating and analyzing the spreading of computer viruses. The more recent literature on issues related to the COVID-19 pandemic almost ubiquitously adopt the SIR approach, extending the framework laid by Kermack, McKendrick, and Walker in 1927. Since the original SIR model was proposed almost a century ago, by modern standards it has several limitations. For instance, most SIR models are deterministic and predict a single peak of newly infected cases. This is incon-

sistent with the fact that, by far, COVID-19 has produced multiple peaks of newly infected cases. To overcome this limitation, we consider a generalized SIR model in which the transmission-intensity rate is an exogenous state variable that may be affected by the unknown factors such as mutations of the virus. The sequence of transmission-intensity rates $(\mu_t)_{t=0}^{\infty}$ is thus a stochastic process, which may be influenced by the current transmission risk of the environment determined by agents' aggregate communicable activities. Our model is thus more amenable to policy discussions and empirical work.

The key assumption of all the SIR models is that the infection rate at any time involves a matching function of two components, the infectious component and the susceptible component. The matching function returns the value of the product of the two components, measured by the density of the infected and the susceptible populations, respectively. Acemoglu et al. (2020) discuss the effects of generalizing this matching function by allowing for a more flexible degree of "increasing returns to scale" of the two components. Our pandemic-response model generalizes the matching function of SIR in different ways. We formulate in (2) the infectious component as a time-t state variable that incorporates symptomatic and asymptomatic infections, and the heterogeneous types and reactions of the agents. The infection rate is then formulated in (7), also incorporating the agents' heterogeneous types and actions. The development of these measures is detailed in Section 3.

Heterogeneous agents with private information. Acemoglu et al. (2021) and Gollier (2020) develop a multi-group SIR model, in which agents are partitioned into a finite number of groups according to their observable traits, focusing on age. They demonstrate the superiority of targeting different groups with different policies compared to a uniform lockdown policy (see also Brotherhood et al. 2020, Favero et al. 2020, Fischer 2020, Glover et al. 2020, and Wilder et al. 2020). In this study, we present a general three-dimensional type set X, and assume that each agent is endowed with a type $x = (\delta_x, \gamma_x, D_x) \in X$, drawn ex ante according to an atomless probability measure F over X. Consequently, the multi-group model is extended to a

personal-characteristics model, in which agents' types can be private information or imperfectly observable. Here, δ_x indicates the discount factor a type-x agent uses for evaluating their life-time utility flows, γ_x indicates the agent's probability of getting symptomatically ill when infected, and D_x indicates the agent's expected utility when diseased. Our comparative statistics analysis in Proposition 3 showing how these personal characteristics affect an agent's equilibrium response to the pandemic has not been explored by the existing literature.

Endogenous individual actions and infection probability. Several recent studies incorporate agents' actions into the SIR model and propose policy recommendations in response to the COVID-19 pandemic.[6] For instance, Eichenbaum et al. (2021) develop an SIR-based macroeconomics model focusing on balancing the severities of the epidemic and the potential recession. Jones et al. (2021) study the dynamics of offering mitigation incentives to the agents working from home at the cost of lower productivity. Farboodi et al. (2021) develop a model that trades off agents' utility benefits of social activities against the probability of infection. Garibaldi et al. (2020) incorporate optimal behavior into the SIR model. The common theme across these studies is that when agents make decisions, they do not consider their decision's negative externality imposed on others. Thus, the resulting distortion from agents' actions from social optimality justifies government intervention.

Our study differs from previous studies in three aspects. First, most previous work focuses on interior solutions, assuming a homogeneous agent population. In contrast, our model derives its equilibrium outcomes from a heterogeneous population and a set of flexible primitive assumptions. Polarized actions subsequently emerge as a plausible equilibrium under natural conditions. Second, we include asymptomatic infection in our model. To our best knowledge, no other research distinguishes the asymptomatic cases from the symptomatic ones when agents' responses to an epidemic are endogenous. The third unique feature of our study is more fundamental: While the existing research largely focuses on optimal policies

[6]Earlier work related to this study includes Kremer (1996) and Toxvaerd (2019).

during the pandemic, we focus on understanding individual behavior and the potential sources of social unrest and political instability, especially in the aftermath of the pandemic (see Censolo and Morelli 2020).

Dynamic game with sequential rationality and Bayes updating. In our model, agents play a dynamic, noncooperative game in choosing or planning their contingent communicable activities. Although we do not assume the role of a centralized decision-maker, our approach is related to the literature on dynamic mechanism design with incomplete information, for example, the discrete-time, infinite horizon models of Athey and Segal (2013) and Pavan et al. (2014). We formulate a solution concept of equilibrium called sequential public-response equilibrium (SPRE), which requires sequential rationality and Bayes updating akin to perfect Bayesian equilibrium. Owing to the continuum of agents, a set of measure zero of agents can be faulty—either in the past or in the planning of future activities—in an SPRE.

Empirical observation. A large empirical literature has emerged since the outbreak of COVID-19. Related work includes those that document people's choices regarding mask-wearing and social distancing based on their distinctive features. For instance, people may differ in their partisan differences and political beliefs in the U.S. (Allcott et al. 2020, Barrios 2020, Gadarian et al. 2021, Grossman et al. 2020, Milosh et al. 2021, Painter and Qiu 2021); in their media consumption and misinformation in the U.S. (Andersen 2020, Ananyev et al. 2021, Gupta et al. 2021); in their ethnic diversity in Russia (Egorov et al. 2021), economic conditions in the U.S. (Wright et al. 2020), scientific beliefs in the U.S. (Brzezinski et al. 2021); degrees of individualism and trust in Europe (Bargain and Aminjonov 2020) and the U.S. (Bazzi et al. 2021), civic capital in Italy (Durante et al. 2021) and the U.S. (Barrios et al. 2021), and in their degrees of risk-tolerance in the U.S. (Fan et al. 2020). Other demographic characteristics considered in the empirical literature include age and gender, amongst others.

While the existing literature examines the differences among people's communicable behaviors, this study goes further and emphasizes the polarized distributions of people's socializing and mask-wearing behaviors. Our empirical evidence suggests

that the polarization of people's COVID-19 responses is observed in a good number of countries that vary dramatically in the level of political polarization. The evidence suggests that 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 political views.

3 Pandemic-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 $T \in \mathbb{N} \bigcup \{\infty\}$.[7] 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 of testing and vaccination and assume recovered agents are no longer infectious.[8] 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

[7] 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.

[8] 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.

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 2). Thus, the susceptible and the asymptomatically infected agents are all active. The main factors influencing the development of the pandemic are *communicable, or transmissible, activities* (henceforth *actions*) 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.

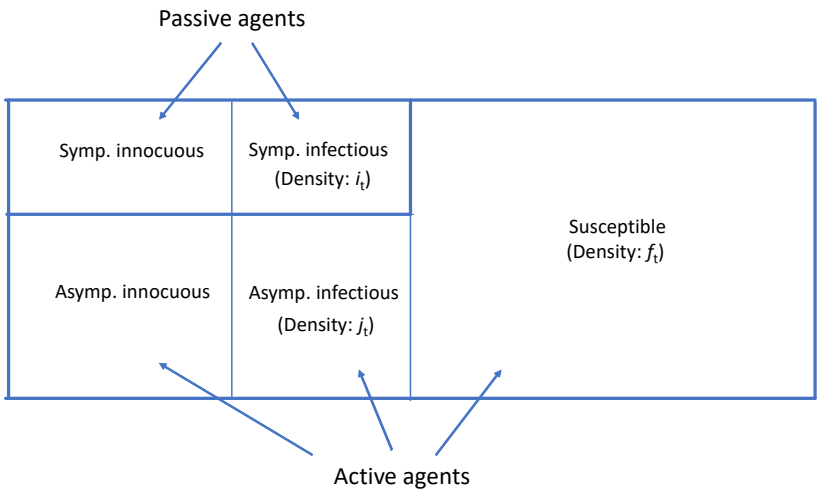

Figure 2: The partition of the population at the start of each period t. The densities i_t, j_t, and f_t are defined in Section 3.2.

The exogenous random state variable is the *transmission-intensity rate* $\mu_t \in$

$[0,1]$ of the pandemic, which determines the probability of a susceptible agent getting infected in period t when they are exposed to the *transmission risk* of the environment that will be defined and quantified later. We will model μ_t as a Markov process influenced by μ_{t-1} and the transmission risk in period $t-1$ in Section 3.3. The realization of μ_t is publicly known at the start of each period t.

3.1 Heterogeneous agents and communicable activities

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 discounted utility equal to

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

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 $0 < u_0 < u$.[9] 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.[10]

Starting from period 1, each active agent can choose a level of communicable activities (or actions) $a_t \in [0,1]$ in each period $t \geq 1$ that restricts their normal way of

[9] 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.

[10] 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.

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 for communicable activities $v : [0,1] \to [u_0, u]$ 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 activities to be directly related to one's utility or wellbeing, not consumption. For instance, reducing communicable activities 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. Thus, unlike the traditional utility models for money or consumption, we do not require v to be a concave function.

Now let μ_0 be given and let $\mu^t := (\mu_1, ..., \mu_t) \in [0,1]^t$ denote the history of the exogenous states 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

When active agents follow a public-response plan in choosing their actions, different types may be exposed to different probabilities of disease. We need to track the type densities of the following population groups (see Figure 2): for $t \in \{1, 2, ..., T\}$,

i_t : density of symptomatically infectious types over X in period t

j_t : density of asymptomatically infectious types over X in period t

f_t : density of susceptible types over X in period t

We have $f_0 = f$, 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 activities, 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, defined by

$$\begin{aligned} \chi_t &: = \int_X \theta(x) i_t(x) dx + \int_X h(\alpha_t(x, \mu^t)) j_t(x) dx, \\ t &\in \{1, ..., T\} \end{aligned} \tag{2}$$

where $\theta : X \to [0, 1]$ is a measurable, nonnegative function and $h : [0, 1] \to [0, 1]$ is continuously differentiable, satisfying $h(0) = 0$, $h(1) = 1$, and $h' > 0$ on $(0, 1)$. The first term on the right side of (2) 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 (2) 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 reduces to $\chi_t = \vartheta_t$, the population size of the infectious.

Now we assume that when a susceptible agent is *exposed* to the transmission risk of the environment, they will be infected with probability $\mu_t \chi_t$. Associated with χ_t are a pair of related state variables, the *infection-probability function* p_t, and the *disease-probability function* q_t. We assume that given μ_t, and given that all other active agents follow the response function $\alpha_t(\cdot, \mu^t)$ in choosing their actions, a susceptible agent choosing activity a_t in period t has the following infection probability

$$p_t(a_t, \mu_t, \chi_t) = g(a_t)\mu_t \chi_t \tag{3}$$

where $g : [0, \infty) \to [0, 1)$ is twice continuously differentiable, satisfying $g(0) = 0$, $\lim_{a\to\infty} g(a) = 1$, and $g' > 0$ on $(0, 1)$. We interpret g as the *cumulative probability of transmission risk exposure (henceforth, exposure function).* Since there is no reason to assume that taking action 1 will expose the agent to the transmission risk with certainty, we allow $g(1) \leq 1$.[11] We will be interested in the behavior of the hazard rate of exposure function $\eta := \frac{g'}{1-g}$. The hazard rate $\eta(a_t)$ 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. A special case of interest is where η is memoryless, i.e., a constant. We then have $g(a) = 1 - \exp\{-\eta a\}$.

Our modeling is complicated by the consideration that the active agents do

[11] 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.

not know whether they are susceptible, infectious, or immune in any 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

$$\Pr(\text{susceptible}_t|\text{active}_t) = \frac{(1-p_0)(1-p_1)...(1-p_{t-1})}{(1-q_0)(1-q_1)...(1-q_{t-1})} := S_{t-1} \tag{4}$$

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 $S_{-1} = 1$)

$$q_t(x, S_{t-1}, p_t) = \gamma_x S_{t-1} p_t \tag{5}$$

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 $= \gamma_x$

(6)

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, denoted by I_t, can be

calculated by integration over the susceptible types:

$$I_t = \mu_t \phi_t \chi_t \text{ where } \phi_t = \int_X g(\alpha_t(x, \mu^t)) f_t(x) dx \tag{7}$$

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 size and actions of the susceptible population, and the second part χ_t the size 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 active prevention from the population ($g \equiv 1$ 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 in the basic assumption of the SIR models. (Since time is continuous in the SIR models, the infected agents are simultaneously infectious.)

Now, fix public-response plan α and suppose all active agents follow the plan. The law of motion for $(i_t, j_t, f_t, \chi_t, p_t, q_t)$ can be derived recursively as follows.[12]

For $t = 0$, suppose we are are given exogenously $\mu_0 \in (0, 1)$ and an infection rate $I_0 \in (0, 1)$. Then, given $f_0 = f$ and $\alpha_0 \equiv 1$, we have $p_0 \equiv I_0$. Defining $S_{-1} \equiv 1$ gives $q_0(x, \mu^0) = \gamma_x I_0$. Consequently,

$$\begin{aligned} f_1(x) &= (1 - I_0) f_0(x) \\ i_1(x) &= \gamma_x I_0 f_0(x) \\ j_1(x) &= (1 - \gamma_x) I_0 f_0(x) \\ \text{and } \chi_1 &= \int_X \theta(x) i_1(x) dx + \int_X h(\alpha_1(x, \mu^1)) j_1(x) dx, \end{aligned}$$

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

$$i_t(x) = \gamma_x p_{t-1}(x, \mu^{t-1}) f_{t-1}(x) \tag{8}$$

$$j_t(x) = (1 - \gamma_x) p_{t-1}(x, \mu^{t-1}) f_{t-1}(x) \tag{9}$$

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

and the density of susceptible types shrinks to

$$f_t(x) = (1 - p_{t-1}(x, \mu^{t-1}))f_{t-1}(x) \tag{10}$$

It follows that

$$\chi_t(\mu^t) = \int_X \theta(x) i_t(x) dx + \int_X h(\alpha_t(x, \mu^t)) j_t(x) dx \tag{11}$$

and for all $x \in X$,

$$\begin{aligned} p_t(x, \mu^t) &= \mu_t g(\alpha_t(x, \mu^t))\chi_t(\mu^t) & (12) \\ q_t(x, \mu^t) &= \gamma_x S_{t-1}(x, \mu^{t-1}) p_t(x, \mu^t) \text{ where } S_{t-1} = S_{t-2}\frac{1 - p_{t-1}(x, \mu^{t-1})}{1 - q_{t-1}(x, \mu^{t-1})} & (13) \end{aligned}$$

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

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 $\hat{\Phi}_t : [0, 1] \times [0, 1 - \rho_t] \to \Delta([0, 1])$, such that given $(\mu_t, \chi_t) \in [0, 1] \times [0, 1 - \rho_t]$, the state μ_{t+1} is a random variable distributed according to $\hat{\Phi}_t(\mu_t, \chi_t) \in \Delta([0, 1])$ (with $\hat{\Phi}_T(\mu_T, \chi_T)$ assigning probability 1 to the event $\{\mu_{T+1} = 0\}$ for $T < \infty$).[13,14]

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

[14] 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 3. Extension to the more general processes could follow the treatment and discussion in Pavan et al. (2014) and Athey and Segal (2013, Appendix A).

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:[15]

$$V_t^*(x, \mu^t) = v(\alpha_t(x, \mu^t)) + \delta_x \left\{ (1 - q_t(x, \mu^t)) \mathbb{E}_t^{\alpha} \left[V_{t+1}^*(x, \mu^{t+1}) | \mu_t, \chi_t \right] + q_t(x, \mu^t) D_x \right\} \tag{14}$$

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

The interpretation of (14) 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 communicable activities, each attempting to maximize their expected payoff. The game will end after T, if T is finite.

Definition 3 *The public-response plan $\alpha = (\alpha_1, ..., \alpha_T)$ forms a* sequential public-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*

[15] 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 (15)).

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 active-population measure* F_t*, infection-probability function* p_t*, and disease-probability function* q_t*, according to (10)– (13).*

In an SPRE, active agents are not affected by any other individual agent's type or actions, 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 (8)–(13) 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 (5), 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 and uniqueness of SPRE in propositions 1 and 2. We then derive the equilibrium behavior and comparative statics results in propositions 4 and 3.

4.1 Path-dependent preferences

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,[16] the agent's optimal expected payoff V_t^* in any period t can be described as (suppressing variables x, μ^t, χ_t)

$$\begin{aligned} V_t^*(a^{t-1}) &= \max_{a_t \in [0,1]} \left\{ v(a_t) + \delta(1 - q_t(a^t)) \mathbb{E}_t^\alpha \left[V_{t+1}^*(a^t) | \mu_t, \chi_t \right] + \delta q_t(a^t) D \right\} \quad (15) \\ t &\in \{1, 2, ..., T\} \end{aligned}$$

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 (15):

$$V_t = v(a_t) + \delta \left\{ (1 - q_t) \mathbb{E}_t^\alpha \left[V_{t+1}^* | \mu_t, \chi_t \right] + q_t D \right\} \quad (16)$$

Maximization of V_t yields the following necessary condition for all optimal actions a_t^*, for which (15) has an interior solution

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - \delta \mathbb{E}_t^\alpha \left[\left(V_{t+1}^* - D \right) \frac{\partial q_t}{\partial a_t} - (1 - q_t) \frac{\partial V_{t+1}^*}{\partial a_t} \middle| \mu_t, \chi_t \right] = 0, \quad (17)$$

otherwise $a_t^* = 0$ or 1, depending on whether $V_t(x, 0, a^{t-1}, \mu^t, \chi_t)$ or $V_t(x, 1, a^{t-1}, \mu^t, \chi_t)$ is greater than $V_t(x, a_t, a^{t-1}, \mu^t, \chi_t)$ through the entire interval $[0, 1]$.

We interpret the expected term in (17) as the *total* marginal cost, and the term $v'(a_t)$ the marginal benefit, for a marginal increase of a_t. The first term in square brackets in (17) is related to the risk of being symptomatically ill, in which case the agent drops out by the end of the period. The second term in square brackets in (17) is related to the agent's continuation payoff when no symptom appears. We will show that increasing a_t has a positive marginal effect on the continuation payoff, i.e., $\frac{\partial V_{t+1}^*}{\partial a_t} \geq 0$. Consequently, the second term in square brackets offsets the effect of the first term to an extent. It suggests that if the agent remains active by the end of the period, having taken a higher level of action would be more desirable.

A complication caused by path-dependency is reflected in the calculation of $\frac{\partial V_{t+1}^*}{\partial a_t}$ in (17). Replacing subscript t with $t + s$ in (15) and differentiating each V_{t+s}^*

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

w.r.t. a_t shows that $\frac{\partial V^*_{t+1}}{\partial a_t}$ is recursively defined via

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

To obtain sharper characterizations, we need to find a more efficient approach. The idea is to examine a mathematically equivalent problem of maximizing

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

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:

$$\left|\begin{array}{llll}\text{Period} & \text{Probability} & \text{Discounting} & \text{Payoff flow}\\ t & 1 & 1 & v(a_t)-(1-\delta)D\\ t+1 & 1-q_t & \delta & v(\alpha_{t+1})-(1-\delta)D\\ \cdots & \cdots & \cdots & \cdots\\ t+s & (1-q_t)...(1-q_{t+s-1}) & \delta^s & v(\alpha_{t+s})-(1-\delta)D\\ \cdots & \cdots & \cdots & \cdots\\ T+1 & (1-q_t)...(1-q_T) & \delta^{T-t+1} & U-D\end{array}\right| \tag{18}$$

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

$$V_t - D = \hat{\mathbb{E}}^\alpha_t\left[\begin{array}{c} v(a_t)-(1-\delta)D\\ +\sum_{s=1}^{T-t}\delta^s\left[\left(\prod_{r=1}^{s}(1-q_{t+r-1})\right)(v(\alpha_{t+s})-(1-\delta)D)\right]\\ +\delta^{T-t+1}\left(\prod_{r=1}^{T-t+1}(1-q_{t+r-1})\right)(U-D)\end{array}\right] \tag{19}$$

where $\hat{\mathbb{E}}^\alpha_t$ 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 (19) 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 1 *The partial derivative in (17) satisfies, for all* $t \in \{1, 2, ..., T\}$,

$$\frac{\partial V_t}{\partial a_t} = v'(a_t) - \delta \frac{\partial q_t}{\partial a_t} \mathbb{E}_t^{\alpha} \left[H_{t+1} | \mu_t, \chi_t \right] \tag{20}$$

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

$$H_{t+s} = v(\alpha_{t+s}) - (1 - \delta) D + \delta (1 - p_{t+s}) H_{t+s+1} \tag{21}$$

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

The function H_{t+1}, when multiplied with $\frac{\partial q_t}{\partial a_t}$, captures the overall marginal cost given in square brackets in (17). The most useful property of H_{t+1} is that it does not depend anymore on the path-dependent disease probability functions $q_t, q_{t+1}, ...$ This lemma significantly simplifies our problem.

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

Lemma 2 *The function* V_t *as defined in (16) has the cross-partial derivative* $\frac{\partial^2 V_t}{\partial a_t \partial a_s} \geq 0$ *for all* $s, t \in \{1, 2, ..., T\}$ *such that* $s < t$.

4.2 Polarized Equilibrium Behavior

To establish equilibrium existence and uniqueness, 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* $t \in \{1, ..., T\}$,

$$\frac{\partial \varphi_t / \partial \chi_t}{\varphi_t} \geq -1 \tag{22}$$

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 (22). This technical assumption implies the following behavioral assumption: in every period t, the active agents' actions are submodular in (a_t, χ_t), i.e., $\frac{\partial^2 V_t}{\partial a_t \partial \chi_t} \leq 0$ (see Lemma 3). 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 in any period t. Since a higher level of χ_t increases the chances of infection, it naturally lowers each individual agent's incentive to choose higher a_t. Therefore, the submodularity assumption seems fairly reasonable. Thus, 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.

Lemma 3 *Suppose Assumption 1 holds. The function V_t as defined in (16) 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 4 *For all $t \in \{1, 2, ..., T\}$, given any (a^{t-1}, μ^t, χ_t), the function V_t as defined in (16) 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) (Topkis 1978). These properties have monotone comparative statics implications (Milgrom and Shannon 1994, Theorem 6), as to be specified in Proposition 3. The lemmas are also useful for establishing our first proposition below, one of the main results of this study.

By Lemma 1, integrate (20) over $[0, 1]$ and obtain, for any (x, μ^t, χ_t),

$$\begin{aligned} K_t(x, \mu^t, \chi_t) &: = V_t(x, 1, \mu^t, \chi_t) - V_t(x, 0, \mu^t, \chi_t) \\ &= u - u_0 - \delta_x q_t(x, 1, \mu^t, \chi_t) \int_0^1 H_{t+1}(x, \mu^{t+1}) d\Phi(\mu_{t+1} | \mu_t, \chi_t) \quad (23) \end{aligned}$$

where $q_t(x, 1, \mu^t, \chi_t) = g(1)\gamma_x S_{t-1}(x, \mu^{t-1})\mu_t\chi_t$. Further define

$$B(\mu^t, \chi_t) = \left\{x \in X : K_t(x, \mu^t, \chi_t) \geq 0\right\}$$

and

$$J_t(B) = \int_B j_t(x)dx \text{ for all } B \subseteq X$$

Proposition 1 *Suppose Assumption 1 holds, and further assume* $\frac{v''}{v'} \geq \frac{g''}{g'}$ *on* $[0, 1]$. *Then, given* $\mu_0 \in (0, 1)$ *and* $\alpha_0 \equiv 1$, *there exists a unique*[17] *sequential public-response equilibrium* $\alpha = (\alpha_t)_{t=1}^T$ *characterized by polarized actions*

$$\alpha_t(x, \mu^t) = \begin{cases} 1 & \text{if } K_t(x, \mu^t, \chi_t) \geq 0 \\ 0 & \text{if } K_t(x, \mu^t, \chi_t) < 0 \end{cases} \tag{24}$$

where

$$\chi_t = \int_X \theta(y)i_t(y)dy + J_t(B(\mu^t, \chi_t)) \tag{25}$$

for all active type $x \in X$ *and* $t \in \{1, 2, ..., T\}$.

Here, (24) characterizes the polarized actions of the active agents, and (25) is an additional equilibrium condition. For types with $K_t(x, \mu^t, \chi_t) = 0$, the agents are indifferent between choosing 1 or 0, and can choose a randomized strategy. Since the set of indifferent types has a zero measure, as shown in the proof of the proposition, we assume that these agents would choose 1 for ease of exposition.

The intuition for the polarized equilibrium in Proposition 1 is that the condition $\frac{v''}{v'} \geq \frac{g''}{g'}$ implies that each agent's expected payoff is a convex function of their actions. To develop some insights into this condition, we take a look at the hazard rate of the exposure function $\eta = \frac{g'}{1-g}$.

Differentiating η yields

$$\eta' = \eta\left(\eta + \frac{g''}{g'}\right)$$

[17]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 $t \in \{1, 2, ..., T\}$.

Thus, if $\eta' \leq 0$ and $\eta \geq -\frac{v''}{v'}$, then

$$-\frac{g''}{g'} \geq \eta \geq -\frac{v''}{v'} \tag{26}$$

Corollary 1 *Suppose Assumption 1 holds and that the hazard ratio* η $(= \frac{g'}{1-g})$ *is nonincreasing, satisfying* $\eta \geq -\frac{v''}{v'}$ *on* $(0,1)$. *Then the conclusion of Proposition 1 holds.*

The nonincreasing hazard rate assumption in this corollary may be construed as follows. The hazard rate $\eta(a_t)$ 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 consider a mind 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 activities 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 in any subenvironment, the hazard rate of risk exposure is a constant. Since the above example concerns only one of numerous possible communicable activities taken place in different subenvironments, we may assume that g is an expected probability function involving exponential functions of different hazard rates, given by

$$g(a) = \int_0^\infty \left(1 - e^{-\lambda a}\right) d\psi(\lambda)$$

where ψ is any arbitrarily given probability measure over $[0, \infty)$. By the fact that any probability mixture of nonincreasing hazard-rate probability functions has the nonincreasing hazard-rate property (e.g., Barlow et al. 1963, Theorem 3.4), the so defined g above satisfies the assumption of Corollary 1.

Regarding the assumption $\eta \geq -\frac{v''}{v'}$, since $\eta > 0$, the assumption holds invariably for all functions v that are linear or convex. More generally, the polarized

equilibrium in Proposition 1 holds under all circumstances in which the hazard rate η of the exposure function g is nonincreasing and is sufficiently large.

Notably, the prediction that all active agents would choose polarized actions amidst a pandemic is based on the primitives v and g only. For instance, the equilibrium prediction in Proposition 1 does not require agents to have polarized distribution for types or personal characteristics. Under any atomless distribution of the individual characteristics (δ, γ, D), Proposition 1 holds, even if the agents have infinitesimal type differences and are observationally "nearly" homogeneous.

To make our analysis more complete, consider next the complementing case of Proposition 1, when $\frac{v''}{v'} < \frac{g''}{g'}$. Then, each agent's expected payoff is a concave function of their actions. In this case, the equilibrium is characterized by the more standard first-order conditions, as presented in the next proposition.

Proposition 2 *Suppose Assumption 1 holds and further assume* $\frac{v''}{v'} < \frac{g''}{g'}$ *on* $(0,1)$. *Then there exists a unique sequential public-response equilibrium* $\alpha = (\alpha_t)_{t=1}^T$ *characterized by*

$$v'(\alpha_t(x,\mu^t)) = \delta_x \gamma_x S_{t-1}(x,\mu^{t-1}) \mu_t g'(\alpha_t(x,\mu^t)) \chi_t\left(\mu^t\right) \int_0^1 H_{t+1}(x,\mu^{t+1}) d\Phi(\mu_{t+1}|\mu_t,\chi_t) \tag{27}$$

for $\alpha_t(x,\mu^t) \in (0,1)$, *or else* $\alpha_t(x,\mu^t) = 0$ *or* 1, *depending on whether* $V_t(x,0,\mu^t,\chi_t)$ *or* $V_t(x,1,\mu^t,\chi_t)$ *is greater than* $V_t(x,a_t,\mu^t,\chi_t)$ *through* $[0,1]$, *for all* $x \in X$ *and* $t \in \{1,2,...,T\}$, *where*

$$\chi_t\left(\mu^t\right) = \int_X \theta(y) i_t(y) dy + \int_X h(\alpha_t(y,\mu^t)) j_t(y) dy \tag{28}$$

Proof. Omitted, as the main arguments are similar to the proof of Proposition 1, noting that (27) is merely an alternative expression of (17). ■

In this proposition, equation (27) provides the standard first-order conditions for the dynamic maximization problem. The term v' is the marginal benefit of increasing a_t in period t, and the term on the right side is the marginal cost. Equation (28) provides an additional equilibrium condition.

As mentioned earlier, we interpret $u- v$ as a deadweight utility loss caused by restricting one's communicable activities. Therefore, a plausible, simpler modelling is to assume a linear function v, in which case the conditions in Propositions 1-2 simplify to $g'' \leq 0$ and $g'' > 0$, respectively. Consider the following corollary of the two cases.

Corollary 2 *Suppose* $v(a) = u_0 + a(u - u_0)$ *and* $g(a) = a^\zeta$ *with* $\zeta > 0$. *Then, for* $\zeta \leq 1$, *the results of Proposition 1 hold and for* $\zeta > 1$, *the results of Proposition 2 hold. In addition, for the case with* $\zeta > 1$, *we have* $g'(0) = 0$. *This implies, by (27), that* $\frac{\partial V_t}{\partial a_t} = v'(a_t) = u - u_0 > 0$ *at* $a_t = 0$. *Proposition 2 would then predict that* $\alpha_t(x, \mu^t) > 0$ *for all* $x \in X$, $\mu^t \in [0,1]^t$ *and* $t \in \{1, ..., T\}$.

This corollary illustrates that the nonincreasing hazard rate assumption of g in Corollary 2 is not necessary, as $g(a) = a^\zeta$ does not exhibit this property on $[0, 1]$. The condition $\zeta \leq 1$ in this Corollary 2, see formula (7), is consistent with the common assumption in matching theory that the matching function is concave (Petrongolo and Pissarides 2001). For $\zeta > 1$, Corollary 2 reveals an additional prediction of Proposition 2 that no single active agent would choose the safety-first action $a_t = 0$ in any period—regardless of their individual characteristics and the virus transmission intensity. Notably, this additional prediction is inconsistent with our data, as presented in Section 5.

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 3 *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 activities 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 activities 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 ($U - D$).

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

Proposition 4 *The 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 (11) 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 (11) 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 large transmission risk in the subsequent period such that all the remaining active agents find it optimal to choose action 0. Notably, this may not be a possible equilibrium under the assumption of Proposition 2 (see Corollary 2), but is well possible under the assumption of Proposition 1. If there were an effective government that could implement a strict lockdown policy among the whole population, then, by Proposition 4(i), the society 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 Empirical Evidence

In this section, we set out to investigate empirically people's responses to COVID-19, focusing on the testing of the competing predictions of Propositions 1 and 2. We use data from the American Time Use Survey (ATUS) to study how people change their time spent on face-to-face socializing during the pandemic in the absence of stay-at-home orders. Our analysis found that compared to 2019, the proportion of people who did not socialize significantly increased during the pandemic, while the

distribution of social time among those who did socialize remained unchanged. This finding means that some people chose "safety first" and stopped socializing, while others chose to "life as usual" without reducing their social time. This is consistent with the polarization equilibrium predicted by Proposition 1, and it clearly negates the interior equilibrium prediction of Proposition 2, which implies that socially active people will continue their socializing activities, but their social time will be shortened during the pandemic.

We focus on face-to-face social activities in our baseline analysis and include more types of activities for robustness checks. Face-to-face social activities are an appropriate counterpart to behavior in our theoretical model for several reasons. First, they significantly increase the risk of infection because the virus is transmitted by exposure to infectious respiratory fluids including inhalation of fine respiratory droplets and aerosol particles[18]. Second, a reduction in face-to-face socializing decreases an individual's utility. Third, modern communication technologies make face-to-face social interaction relatively dispensable compared to behaviors necessary for survival, such as buying food at a supermarket. Thus, it is possible for a person to (at least temporarily) stop face-to-face socializing, which corresponds to $a_t = 0$ in our theoretical model.

Our analysis employs the data from American Time Use Survey (ATUS) that measures the amount of time people spend doing various activities. Daily time spent socializing is collected from 2 subcategories under category 12 of ATUS data: (1201) socializing and communicating and (1202) attending or hosting social events. These subcategories contain face-to-face social activities with a high risk of infection. For example, conversing with people dining at a restaurant is coded as 1201; attending a meeting or festival party is coded as 1202. Note that communications by telephone or Internet, which have no risk of infection, are excluded from these subcategories and are therefore not defined as social activity in our analysis. The ATUS data also provide information on location and "with whom" during the activity, as well

[18] https://www.cdc.gov/coronavirus/2019-ncov/science/science-briefs/sars-cov-2-transmission.html

as the characteristics of the respondents, including age, family income, education, employment status, number of children, child's age, etc. A descriptive summary of the data is included in the Online Appendix.

The COVID-19 outbreak divided the 2020 ATUS data into two periods: a pre-pandemic period including January and February 2020 and a period during the pandemic from July to December 2020. The data from March to May 2020 are not included because data collection was suspended in 2020 from mid-March to mid-May. Data for June 2020 is excluded because the stay-at-home policy, which restricts people's freedom to socialize, had not been lifted in more than 10% of U.S. counties[19]. Moreover, data for the same months of 2019 are used as a reference for behaviors under normal circumstances. Comparing data from January and February 2019 to 2020, we do not find any annual trend or systematic change in people's social behavior in the absence of the pandemic. If this situation persists for the remainder of 2020, then changes in social time from July 2020 to December 2020 relative to the same period in 2019 can be considered as a result of the pandemic. Lastly, we exclude data beyond 2020 to eliminate the effect of vaccination[20] so that behavioral changes relative to 2019 were purely a result of the pandemic.

The key implication of Proposition 1 is that people will choose oppositely polarized corner solutions. It is worth noting that the corner solution $a_t = 0$ can be directly mapped to zero social time, while the other corner solution $a_t = 1$ does not correspond to a specific amount of social time. Instead, $a_t = 1$ means that people lived a normal life, i.e., they spent a similar amount of time socializing during the pandemic compared to the same period in 2019. Thus, these two corner solutions lead to different forms of testable hypotheses, which are tested separately in the analysis below.

First, we consider the zero corner solution in Proposition 1. Since a respondent might not have any social activities on the day of interview, the dataset has

[19] Our hypothesis testing results are robust to the inclusion of the data for June 2020

[20] According to data from ourworldindata.org, less than 1% of the population were vaccinated in the U.S. by the end of 2020.

observations with zero social time even before the pandemic. Proposition 1 predicts that some people who used to socialize would stop socializing during the pandemic, resulting an increase in the proportion of people choosing zero social time during the pandemic compared to the same period in 2019. In contrast, Proposition 2 shows that all agents would choose an interior solution $a_t > 0$ (see Corollary 2) during the pandemic. This implies that while people reduce their social time, relative to the same month in 2019, the proportion of people choosing zero social time should remain approximately the same. Thus, we test the following hypotheses

$$\begin{aligned} H_0^1 : \ N_1^t &= N_2^t \\ H_1^1 : \ N_1^t &< N_2^t, \end{aligned} \tag{29}$$

where N_1^t and N_2^t denote the proportions of individuals with nonzero daily social time during month t in 2019 and 2020, respectively. Figure 3 plots N_1^t and N_2^t in different months and reports the p-value for the null hypothesis H_0^1. The data show that the proportion of observations with zero social time in January and February 2020 did not change significantly relative to 2019. In contrast, the proportion of observations with zero social time between July and December 2020 was significantly higher than during the same period in 2019, which is consistent with the prediction by Proposition 1.

Next, we consider the other corner solution "life as usual", i.,e., $a_t = 1$, which represents a relative rather than an absolute measure, indicating that people do not change their behavior during the pandemic. In order to test this hypothesis, we conduct a counterfactual analysis of how people who socialized during the pandemic changed their daily social time compared to what they would have done in 2019.

Let X_1 and X_2 denote the personal characteristics of the respondents in 2019 and 2020, respectively, and let Y_1 and Y_2 denote the time they spent socializing each day in 2019 and 2020, respectively. Let $F_{X_2}(x|Y_2 > 0)$ denote the distribution of X_2 conditional on $Y_2 > 0$. Following Chernozhukov et al. (2013), we consider the following distributions

$$F_Y(y)_{t,2} \equiv \int F_{Y_t|X_t,Y_t>0}(y|x)\, dF_{X_2}(x|Y_2 > 0) \tag{30}$$

for $t = 1, 2$, where $F_{Y_t|X_t,Y_t>0}(y|x)$ denotes the conditional distribution of Y_t given X_t and $Y_t > 0$, which captures the dependence of daily social time on people's characteristics in year t given that they socialized. $F_Y(y)_{2,2}$ denotes the fitted distribution of Y conditional on $Y > 0$ in 2020 and $F_Y(y)_{1,2}$ denotes the counterfactual distribution of Y, which integrates the conditional distribution of Y given X and $Y > 0$ in 2019 with respect to the distribution of characteristics X given $Y > 0$ in 2020. Thus, the counterfactual distribution $F_Y(y)_{1,2}$ represents the distribution of time that people who socialized in 2020 would have spent socializing, if they followed their behavioral mode in 2019 without the pandemic.

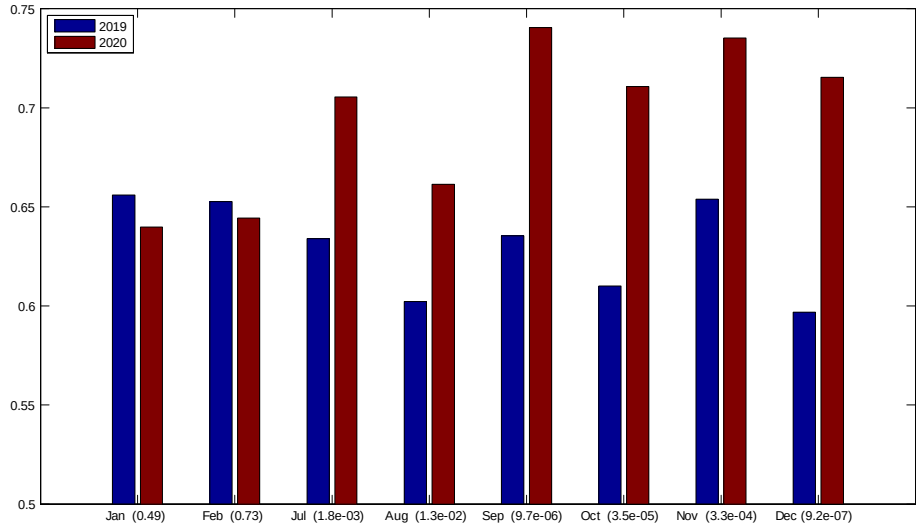

Figure 3: The proportions of people with zero social time in 2019 and 2020

The numbers in paretheses are the p-values for the null hypothesis that the proportion in 2020 remains the same compared to the same month in 2019.

The corner solution $a_t = 1$ in Proposition 1 indicates that some people would choose "life as usual" during the pandemic. This implies that people who continued to socialize during the pandemic should spend a similar amount time socializing in 2019. In contrast, the interior solution in Proposition 2 implies that people who

continued to socialize during the pandemic should have spent more time socializing if there were no pandemic, so $F_Y(y)_{1,2}$ is expected to be smaller than $F_Y(y)_{2,2}$ for a given y (i.e., $F_Y(y)_{1,2}$ has larger quantile values than $F_Y(y)_{2,2}$). In order to test these two competing implications, we compare the difference

$$\delta(y) = F_Y(y)_{2,2} - F_Y(y)_{1,2}.$$

Under Proposition 1, $\delta(y)$ is expected to be close to zero for all $y \in \mathcal{Y}$ with $\mathcal{Y} \subseteq \mathbb{R}^+$. Hence, we test the following hypotheses

$$\begin{aligned} H_0^2 : \sup_{y \in \mathcal{Y}} |\delta(y)| &= 0 \\ H_1^2 : \sup_{y \in \mathcal{Y}} |\delta(y)| &\neq 0. \end{aligned} \tag{31}$$

The null hypothesis in (31) is restrictive in the sense that it requires the empirical distance between $F_Y(y)_{1,2}$ and $F_Y(y)_{2,2}$ close to zero for all y values.

We estimate $F_Y(y)_{t,2}$ using the estimator developed by Chernozhukov et al. (2013) and compute the deciles of $F_Y(y)_{1,2}$ and $F_Y(y)_{2,2}$ with bootstrap uniform confidence bands. Since the social time has mass points at rounded minute values, we follow Chernozhukov et al. (2013) and estimate the conditional distribution $F_{Y_t|X_t,Y_t>0}(y|x)$ using distribution regression (Foresi and Peracchi, 1995; Han and Hausman, 1990) with a probit link function, where the covariates X include the following variables in our baseline specification: age, squared age, family income, employed or not, having kids or not, the number of children, the age of the youngest child, and dummy variables for Monday through Saturday. Adding additional controls (eg., dummy variables for months) did not qualitatively change the results. The sample consists of 542 (521) observations from January and Feburary 2020 (2019) and 1417 (1634) observations from July to December 2020 (2019).

Figure 4 plots the deciles of $F_Y(y)_{2,2}$ and $F_Y(y)_{1,2}$ with the 95% level uniform confidence band of $F_Y(y)_{1,2}$ for the period before the pandemic (January and February 2020, upper left panel) and period during the pandemic (July to December 2020, lower left panel). The right upper and right lower panels plot the difference between the deciles of $F_Y(y)_{1,2}$ and $F_Y(y)_{2,2}$ for the periods before and during the

pandemic, respectively. The fitted distribution is always within the 95% confidence band of the counterfactual distribution before and during the pandemic. Also, the horizontal axis is always within the 95% confidence band of the difference between the deciles of $F_Y(y)_{1,2}$ and $F_Y(y)_{2,2}$ in both periods. Moreover, the p-values of the Kolmogorov-Smirnov statistics for testing H_0^2 are 0.75 and 0.9 before and during the pandemic, respectively, implying that the difference between $F_Y(y)_{1,2}$ and $F_Y(y)_{2,2}$ is not significantly different from zero for all y. Based on these results, we accept the null hypothesis in (31), meaning that people socializing during the 2020 pandemic would spend a similar amount of time socializing if they behaved the same way they did in 2019 in the absence of pandemic. This indicates that these people choose the "life as usual" option, which is in line with the prediction by Proposition 1.

In order to check the robustness of our results, we further analyzed the total time respondents spend per day on a wider range of behaviors. In addition to socializing, we also incorporate dining in restaurants and bars and exercising in public indoor spaces in the absence of orders for closing restaurants, bars, and gyms.[21]. These additional behaviors are apparently associated with high risks of infection and can be avoided at the cost of utility loss during the pandemic (i.e., it is possible to choose $a_t = 0$ for these behaviors). Adding these types of behaviors to our empirical analysis benefits us for at least two reasons. First, it increases the sample size and thus makes the test more powerful. Second, if a person avoids some risky behaviors (e.g., socializing) but continues some other risky behaviors (e.g., exercising in a gym), then the total time spent would be nonzero but less than if there were no pandemic. This means that interior solutions are more likely to be observed when we consider the total time spent on multiple activities. These two

[21] To be specific, we consider time spent eating in public places including restaurants, bars, malls, and grocery stores. Our analysis excluded the purchase of takeout, as it is a relatively low-risk activity if people wear masks and the dwell time is short. For sport, we only consider excercises in indoor public spaces because exercising in the open air or in a private place (such as at home) is much less risky. These two additional types of activities are highly risky because it is difficult or even impossible for people to wear masks during these activities.

reasons will render the null hypothesis H_0^2 more likely to be rejected if it is false. In other words, if we still cannot reject H_0^2 using the augmented data, then it provides a stronger support for the corner solution in our Proposition 1.

The estimated distributions using the augmented data are plotted in Figures A2 in the Online Appendix. The results are similar to those shown in Figure 4. The p-values of the Kolmogorov-Smirnov statistics for testing H_0^2 are 0.87 (Jan-Feb) and 0.79 (Jul-Dec) during the two subperiods in 2020. Thus, the fitted distribution is not significantly different from the counterfactual distribution, which is again consistent with the prediction of Proposition 1.

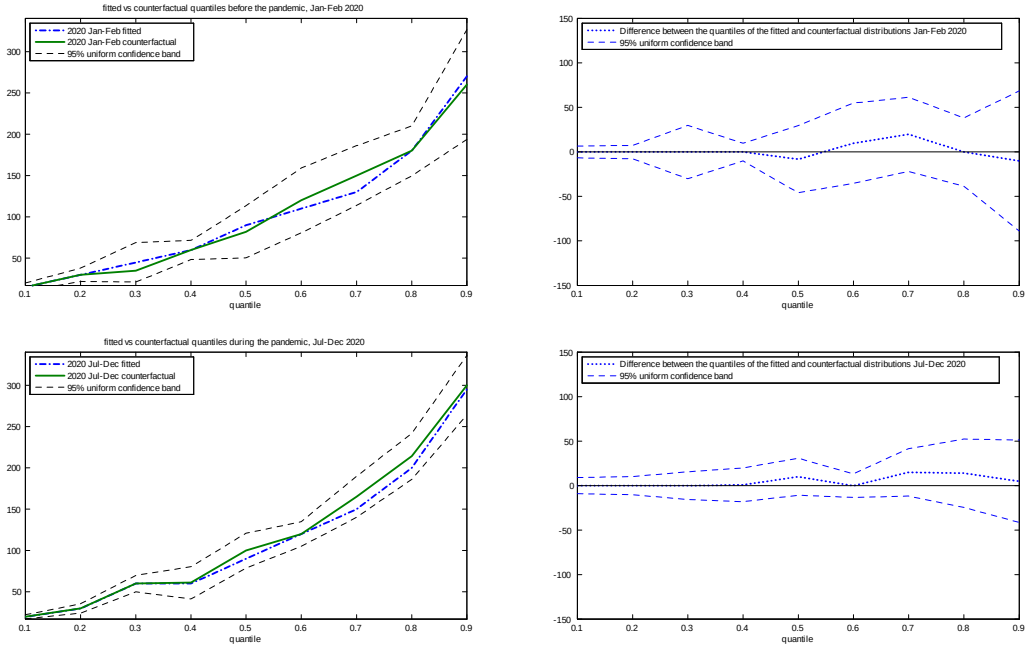

Figure 4: Fitted versus counterfactual quantiles of time spent socializing for people who socialized in 2020

The p-values of the Kolmogorov-Smirnov statistic for testing H_0^2 are 0.75 and 0.9 before and during the pandemic, respectively.

6 Conclusion

We have studied a dynamic game model with heterogeneous agents under pandemic risk and obtained three main results. The first result provides a rational explanation for the puzzling phenomenon of polarization and how people can be deeply divided in their thinking and behaviors during a pandemic. We show that polarized responses during a pandemic do not need to be derived from any assumption of polarized distribution among agents' types. They can, instead, be rationalized under various relative conditions between agents' probability function of exposure to the transmission risk and their utility function for communicable activities. This result improves our understanding of human behavior in times of a pandemic. It highlights that the polarization phenomenon is a combined consequence of individual rationality, personal conditions, and the nature of disease transmission. For a society concerned about peace and community solidarity, a viable way to overcome the conflict in interests is to promote awareness and "accept the rights of others" (*The eight Pillars of Positive Peace* - Vision of Humanity). For instance, people can learn that opposing preferences are unrelated to being "right" or "wrong," and appearing to be "extreme" is irrelevant to irrationality. These phenomena are the natural consequences of individual rationality: chacun à son goût.

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. The resulting path-dependent expected payoffs suggest then that as time goes by, people will increasingly neglect the risk of disease and live their life as usual during a pandemic. Our results regarding the long-run equilibrium towards herd immunity, and our empirical investigations, both support all these propositions.

The third result shows that in equilibrium, the agents' actions are akinly re-

lated to their personal traits. We show that an agent's communicable activities are 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 the 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. However, although such policies seem more plausible in theory, a serious discussion of optimal policies is beyond the scope of this study.

Akin to many historic plagues and epidemics, COVID-19 had a significant impact on the level of conflict and violence worldwide. While the path to recovery is full of uncertainties, the crisis will have far-reaching implications across various dimensions of our society. In a post-pandemic world, studying the patterns observed during the outbreak is crucial to understand the reasons behind them and their implications. This can help governments, policy-makers, organizations, and individuals mitigate the potential damages in the future if such a crisis arises again. We hope that the general framework and analysis presented here will be useful for future research.

7 Appendix

7.1 Proofs of the lemmas

Proof of Lemma 1. Fix any $t \in \{1, 2, ..., T\}$. We show by induction on $s = 1, ..., T - t$ that the product term in (18) has a derivative

$$\frac{\partial}{\partial a_t} \prod_{r=1}^{s} (1 - q_{t+r-1}) = -\gamma S_{t-1} p_t'(a_t) \prod_{r=1}^{s-1} (1 - p_{t+r}) \tag{32}$$

where $\Pi_{r=1}^{0} (1 - p_{t+r})$ is defined as unity. For $s = 1$, from (5) we have

$$1 - q_t = 1 - \gamma S_{t-1} p_t(a_t) \tag{33}$$

so that $\frac{\partial(1-q_t)}{\partial a_t} = -\gamma S_{t-1} p_t'(a_t)$, conforming (32). Now, supposing (32) holds for arbitrary $s \geq 1$, let us consider the case with $s + 1$. Noting from (4) and (5) 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 (32). 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 (32) holds for all $s \geq 1$. Now, differentiating (19) with respect to a_t using (32), and defining H_{t+1} by

$$H_{t+1} = \sum_{s=1}^{T-t} \prod_{r=1}^{s-1} (1 - p_{t+r}) \delta^s \left(v(\alpha_{t+s}) - (1-\delta) D \right) \tag{34}$$

$$+ \left(\prod_{r=1}^{T-t+1} (1 - p_{t+r-1}) \right) \delta^{T-t} (U - D) \tag{35}$$

one can readily verify that (20) holds, where the equivalent recursive expression of H_{t+1} derives from (34)–(35).

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 2. 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,

$$\begin{aligned}\frac{\partial S_{t-1}}{\partial a_s} &= A\frac{\partial}{\partial a_s}\frac{(1-p_s)}{\prod_{r=1}^{t-s}(1-q_{s+r-1})} \\ &= A\frac{-p_s'\prod_{r=1}^{t-s}(1-q_{s+r-1}) - (1-p_s)\frac{\partial}{\partial a_s}\prod_{r=1}^{t-s}(1-q_{s+r-1})}{\left(\prod_{r=1}^{t-s}(1-q_{s+r-1})\right)^2} \qquad (36)\end{aligned}$$

By (32) in the proof of Lemma 1, 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

$$\begin{aligned}&-p_s'\prod_{r=1}^{t-s}(1-q_{s+r-1}) - (1-p_s)\frac{\partial}{\partial a_s}\prod_{r=1}^{t-s}(1-q_{s+r-1}) \\ =\ & -p_s'\prod_{r=1}^{t-s}(1-q_{s+r-1}) + (1-p_s)p_s' S_{s-1}\gamma\prod_{r=1}^{t-s-1}(1-p_{s+r}) \\ =\ & \left(-p_s' + (1-p_s)p_s' S_{s-1}\gamma\frac{\prod_{r=1}^{t-s-1}(1-p_{s+r})}{\prod_{r=1}^{t-s}(1-q_{s+r-1})}\right)\prod_{r=1}^{t-s}(1-q_{s+r-1}) \\ =\ & -(1-\gamma S_{t-1})\,p_s'\prod_{r=1}^{t-s}(1-q_{s+r-1}) \qquad (37)\end{aligned}$$

Now, substituting A and (37) into (36) 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 (27) 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 3. 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

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

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 4. 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 1, it suffices to show $\frac{\partial H_{t+1}}{\partial \delta} \geq 0$. As shown in Lemma 1, 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

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

■

7.2 Proofs of the propositions

Proof of Proposition 1. *Step 1.* We show first $\frac{v''}{v'} \geq \frac{g''}{g'}$ implies that any SPRE necessarily involves only the corner solutions such that $\alpha_t \in \{0, 1\}$. By Lemma 1, the problem of maximizin V_t as defined in (16) can be expressed by the program (suppressing other variables):

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

Since $v' > 0$, we can define $v_t = v(a_t)$ and $a_t = v^{-1}(v_t)$ so that the above program is isomorphic to the program

$$\max_{v_t \in [u_0, u]} V_t(v^{-1}(v_t)) = v_t - \delta\gamma S_{t-1}\mu_t g(v^{-1}(v_t))\chi_t \mathbb{E}_t^{\alpha}\left[H_{t+1}|\mu_t, \chi_t\right] + V_t(0)$$

By Pratt (1964, Theorem 1(d)), $\frac{v''}{v'} \geq \frac{g''}{g'}$ on $[0,1]$ is equivalent to $g(v^{-1}(v_t))$ being a concave function of v_t on $[u_0, u]$, which implies that $V_t(v^{-1}(v_t))$ is a convex function of v_t. Therefore, the optimal solution to the above program must be either $v_t = u_0$ or $v_t = u$, or, equivalently, $a_t = 0$ or $a_t = 1$.

Step 2. We next show the existence and characterization 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.

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

$$\begin{aligned} a_t^*(x, a^{t-1}, \mu^t, \chi_t) &= \arg\max_{a_t \in [0,1]} V_t(x, a_t, a^{t-1}, \mu^t, \chi_t) \\ \text{for all } \left(x, a^{t-1}, \mu^t, \chi_t\right) &\in X \times \{0,1\}^{t-1} \times [0,1]^t \times [\theta_t, \vartheta_t],\ t \in \{1, ..., T\} \end{aligned}$$

where $\theta_t := \int_X \theta(x) i_t(x) dx$. For K_t defined in (23), we modify it for now by

$$K_t(x, a^{t-1}, \mu^t, \chi_t) := V_t(x, 1, a^{t-1}, \mu^t, \chi_t) - V_t(x, 0, a^{t-1}, \mu^t, \chi_t)$$

The conclusion in Step 1 then implies that for any $(x, a^{t-1}, \mu^t, \chi_t)$, the optimal individual plan $(a_t^*)_{t=1}^T$ must satisfy, for all $t \in \{1, ..., T\}$,

$$a_t^*(x, a^{t-1}, \mu^t, \chi_t) = \begin{cases} 1 & \text{if } K_t(x, a^{t-1}, \mu^t, \chi_t) > 0 \\ 0 & \text{if } K_t(x, a^{t-1}, \mu^t, \chi_t) < 0 \end{cases} \tag{38}$$

Types in the set $B_0(a^{t-1}, \mu^t, \chi_t) := \{x \in X : K_t(x, a^{t-1}, \mu^t, \chi_t) = 0\}$ may randomize between 0 and 1.

Now, 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 (19) converges uniformly to 0 as $T \to \infty$. Thus, the characterization of a_t^* in (38) extends to the infinite horizon case as $T \to \infty$.

Step 3. For equilibrium uniqueness, we show next that the set $B_0(a^{t-1}, \mu^t, \chi_t)$ has a zero J_t-measure for all (a^{t-1}, μ^t, χ_t).

Since F is atomless, so is J_t, and therefore, the set of any singleton $\{x\} \subset X$ has a zero measure (see footnote 10). By contradiction, suppose for some (a^{t-1}, μ^t, χ_t) we have $J_t(B_0(a^{t-1}, \mu^t, \chi_t)) > 0$. Then, given $X \subset \mathbb{R}^3$, we can partition B_0 into 2^3 subsets with positive measures and find out two of these:

$$\begin{aligned} B_0^+ &= \{x \in B_0 : \delta_x \leq \delta_0, \gamma_x \leq \gamma_0, D_x \geq D_0)\} \\ B_0^- &= \{x \in B_0 : \delta_x \geq \delta_0, \gamma_x \geq \gamma_0, D_x \leq D_0)\} \end{aligned}$$

by properly choosing the vector $x_0 = (\delta_0, \gamma_0, D_0) \in X$. As x_0 is atomless, removing x_0 from these sets preserves their measures as B_0^+ and B_0^- respectively, while causing at least one of the inequalities to hold strictly for $B_0^+ \backslash \{x_0\}$ and $B_0^- \backslash \{x_0\}$. However, Lemma 4 implies that the types in $B_0^+ \backslash \{x_0\}$ would prefer strictly higher actions than the types in $B_0^- \backslash \{x_0\}$, which contradicts the definition of $B_0(\mu^t, \chi_t)$. Therefore, we must have $J_t(B_0(\mu^t, \chi_t)) = 0$ for all (μ^t, χ_t).

Step 4. Finally, we establish the SPRE by forward induction on t. The argument goes as follows. In period 1, define $\hat{\alpha}_1(x, \mu^1, \chi_1) = a_1^*(x, 1, \mu^1, \chi_1)$ as all agents chose action 1 in period 0. Then we use $\hat{\alpha}_1$ to find an equilibrium level of transmission risk $\chi_1(\mu^1)$ satisfying (25). Substituting into $\hat{\alpha}_1$, we obtain period-1 equilibrium plan $\alpha_1(x, \mu^1) = \hat{\alpha}_1(x, \mu^1, \chi_1(\mu^1))$ for all $(x, \mu^1) \in X \times [0, 1]$. For period $t \geq 2$, suppose we have established the equilibrium plan up to $t-1 : \alpha^{t-1}(x, \mu^{t-1}) = (\alpha_1(x, \mu^1), ..., \alpha_{t-1}(x, \mu^{t-1}))$. Then, defining $\hat{\alpha}_t(x, \mu^t, \chi_t) = a_t^*(x, \alpha^{t-1}(x, \mu^{t-1}), \mu^t, \chi_t)$, we go on to find the equilibrium transmission risk level $\chi_t(\mu^t)$ that satisfies (25), and so on.

To ease notation, we suppress variable μ^t in the remainder of the proof. Recall that we have defined

$$B(\chi) = \{x \in X : K_t(x, \chi) \geq 0\}$$

and

$$J_t(B) = \int_B j_t(x)dx \text{ for all } B \subseteq X$$

Pick any $t \in \{1, 2, ..., T-1\}$ and suppose $\hat{\alpha}_t(x, \mu^t, \chi_t)$ is given. Define mapping $\Gamma_t : [\theta_t, \vartheta_t] \to [\theta_t, \vartheta_t]$ by

$$\Gamma_t(\chi) = \theta_t + \int_X h(\hat{\alpha}_t(x, \chi)) j_t(x) dx = \theta_t + J_t(B(\chi)), \quad \forall \chi \in [\theta_t, \vartheta_t]$$

By Lemma 3, Γ_t is nonincreasing. We now show that Γ_t is also continuous. By Assumption 1, $\mathbb{E}_t^{\alpha}[H_{t+1}|\mu_t, \chi_t]$ is continuous in χ_t, so that $K_t(x, \chi_t)$ is a continuous function of χ_t. It follows that $\forall \varepsilon > 0$,

$$\begin{aligned} & \lim_{\varepsilon \to 0} B(\chi - \varepsilon) \backslash B(\chi + \varepsilon) \\ = & \lim_{\varepsilon \to 0} \{x \in X : K_t(x, \chi_t - \varepsilon) \geq 0, K_t(x, \chi_t + \varepsilon) < 0\} \\ = & \{x \in X : K_t(x, \chi_t) = 0\} = B_0(\chi_t) \end{aligned}$$

which implies

$$\begin{aligned} & \lim_{\varepsilon \to 0} [J_t(B(\chi - \varepsilon)) - J_t(B(\chi + \varepsilon))] \\ = & \lim_{\varepsilon \to 0} J_t(B(\chi - \varepsilon) \backslash B(\chi + \varepsilon)) \\ = & \lim_{\varepsilon \to 0} J_t(B_0(\chi)) = 0 \end{aligned}$$

where the last equation is due to Step 3. Having established the continuity of Γ_t, Brouwer's fixed point theorem, combined with Γ_t being nonincreasing, implies a unique fixed point $\chi_t = \Gamma_t(\chi_t)$ that satisfies (25). The proof of the proposition is thus completed by induction, for arbitrary $T \leq \infty$. ■

Proof of Proposition 3. We prove the proposition by induction, using the results of Lemma 4.

(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 4, $\frac{\partial^2 V_1}{\partial a_t \partial \delta} < 0$ 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 2, 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 4. (i) Suppose $\mu_\tau \vartheta_\tau = 0$ for some $0 < \tau < \infty$. Then from (2) $\chi_\tau(\mu^\tau) = 0$, 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 $t \to \infty$. ■

8 References

Acemoglu, D., Chernozhukov, V., Werning, I., & Whinston, M. D. (2020). Optimal targeted lockdowns in a multigroup SIR model. *National Bureau of Economic Research.* No. w27102.

Acemoglu, D., Chernozhukov, V., Werning, I., & Whinston, M. D. (2021). Optimal targeted lockdowns in a multigroup SIR model. *American Economic Review: Insights*, 3(4), 487- 502.

Allcott, H., Boxell, L., Conway, J., Gentzkow, M., Thaler, M., & Yang, D. (2020). Polarization and public health: Partisan differences in social distancing during the coronavirus pandemic.*Journal of Public Economics*, 191, 104254.

Ananyev, M., Poyker, M., & Tian, Y. (2021). The safest time to fly: pandemic response in the era of Fox News. *Journal of Population Economics*, 34(3), 775-802.

Andersen, M. (2020). Early evidence on social distancing in response to COVID-19 in the United States. Available at SSRN 3569368.

Athey, S., & Segal, I. (2013). An efficient dynamic mechanism. *Econometrica*, 81(6), 2463-2485.

Bargain, O., & Aminjonov, U. (2020). Trust and compliance to public health policies in times of COVID-19. *Journal of Public Economics*, 192, 104316.

Barlow, R. E., Marshall, A. W., & Proschan, F. (1963). Properties of probability distributions with monotone hazard rate. *Annals of Mathematical Statistics*, 375-389.

Barrios, J. M., Benmelech, E., Hochberg, Y. V., Sapienza, P., & Zingales, L. (2021). Civic capital and social distancing during the Covid-19 pandemic. *Journal of Public Economics*, 193, 104310.

Barrios, J. M., & Hochberg, Y. (2020). Risk perception through the lens of politics in the time of the COVID-19 pandemic. *National Bureau of Economic Research.* No. w27008.

Bazzi, S., Fiszbein, M., & Gebresilasse, M. (2021). Rugged individualism" and collective (in) action during the COVID-19 pandemic. *Journal of Public Economics*, 195, 104357.

Bian , B., Li, J., Xu, T., & Foutz, N. Z. (2020). Individualism during crises. *Review of Economics and Statistics*, 1-45.

Boxell, L., Gentzkow, M., & Shapiro, J. M. (2022). Cross-country trends in affective polarization. *Review of Economics and Statistics*, 1-60.

Brotherhood, L., Kircher, P., Santos, C., & Tertilt, M. (2020). An economic model of the Covid-19 pandemic with young and old agents: Behavior, testing and policies. CESifo Working Paper No. 8316, Available at SSRN 3618840.

Brzezinski, A., Kecht, V., Van Dijcke, D., & Wright, A. L. (2021). Science skepticism reduced compliance with COVID-19 shelter-in-place policies in the United States. *Nature Human Behaviour*, 5(11), 1519-1527.

Censolo, R., & Morelli, M. (2020). COVID-19 and the potential consequences for social stability. *Peace Economics, Peace Science and Public Policy*, 26(3).

Chernozhukov, V., Fernndez-Val, I. and Melly, B. (2013). Inference on Counterfactual Distributions. *Econometrica*, 81: 2205-2268.

Durante, R., Guiso, L., & Gulino, G. (2021). Asocial capital: Civic culture and social distancing during COVID-19. *Journal of Public Economics*, 194, 104342.

Egorov, G., Enikolopov, R., Makarin, A., & Petrova, M. (2021). Divided we stay home: Social distancing and ethnic diversity. *Journal of Public Economics*, 194, 104328.

Eichenbaum, M. S., Rebelo, S., & Trabandt, M. (2021). The macroeconomics of epidemics. *Review of Financial Studies*, 34(11), 5149-5187.

Fan, Y., Orhun, A. Y., & Turjeman, D. (2020). Heterogeneous actions, beliefs, constraints and risk tolerance during the COVID-19 pandemic. *National Bureau of Economic Research*. No.w27211

Farboodi, M., Jarosch, G., & Shimer, R. (2021). Internal and external effects of social distancing in a pandemic. *Journal of Economic Theory*, 196, 105293.

Favero, C. A., Ichino, A., & Rustichini, A. (2020). Restarting the economy while saving lives under Covid-19. Available at SSRN 3580626.

Fischer, C., (2020), External costs and benefits of policies to address COVID-19, mimeo.

Foresi, S., & F. Peracchi (1995). The Conditional Distribution of Excess Returns: An Empirical Analysis. *Journal of the American Statistical Association*, 90, 451–466.

Gadarian, S. K., Goodman, S. W., & Pepinsky, T. B. (2021). Partisanship, health behavior, and policy attitudes in the early stages of the COVID-19 pandemic. *Plos one*, 16(4), e0249596.

Garibaldi, P., Moen, E. R., & Pissarides, C. (2020). Modelling contacts and transitions in the SIR epidemics model. *Covid Economics*, 5, 1-20.

Glover, A., Heathcote, J., Krueger, D., & Rios-Rull, J. V. (2020). Health versus wealth: On the distributional effects of controlling a pandemic. *National Bureau of Economic Research.* No. w27046.

Gollier, C. (2020). Cost–benefit analysis of age-specific deconfinement strate-

gies. *Journal of Public Economic Theory*, 22(6), 1746-1771.

Grossman, G., Kim, S., Rexer, J. M., & Thirumurthy, H. (2020). Political partisanship influences behavioral responses to governors' recommendations for COVID-19 prevention in the United States. *Proceedings of the National Academy of Sciences*, 117(39), 24144-24153.

Gupta, S., Nguyen, T., Raman, S., Lee, B., Lozano-Rojas, F., Bento, A., Simon, K., & Wing, C. (2021). Tracking public and private responses to the COVID-19 epidemic: evidence from state and local government actions. *American Journal of Health Economics*, 7(4), 361-404.

Han, A., & J. A. Hausman. (1990). Flexible Parametric Estimation of Duration and Competing Risk Models. *Journal of Applied Econometrics*, 5, 1–28.

Hays, J. N. (2005). *Epidemics and Pandemics: Their Impacts on Human History*. Calif: ABC-CLIO.

Jedwab, R., Khan, A. M., Russ, J., & Zaveri, E. D. (2021). Epidemics, pandemics, and social conflict: Lessons from the past and possible scenarios for COVID-19. *World Development*, 147, 105629.

Jones, C., Philippon, T., & Venkateswaran, V. (2021). Optimal mitigation policies in a pandemic: Social distancing and working from home. *Review of Financial Studies*, 34(11), 5188-5223.

Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character*, 115(772), 700-721.

Kremer, M. (1996). Integrating behavioral choice into epidemiological models of AIDS. *Quarterly Journal of Economics*, 111(2), 549-573.

Milgrom, P. R., & Shannon, C. (1994). Monotone comparative statics. *Econometrica: Journal of the Econometric Society*, 62 (1), 157-180.

Milgrom, P. R., & Weber, R. J. (1985). Distributional strategies for games with incomplete information. *Mathematics of Operations Research*, 10(4), 619-632.

Milosh, M., Painter, M., Sonin, K., Van Dijcke, D., & Wright, A. L. (2021). Unmasking partisanship: Polarization undermines public response to collective risk.

Journal of Public Economics, 204, 104538.

Painter, M., & Qiu, T. (2021). Political beliefs affect compliance with government mandates. *Journal of Economic Behavior & Organization*, 185, 688-701.

Pavan, A., Segal, I., & Toikka, J. (2014). Dynamic mechanism design: A myersonian approach. *Econometrica*, 82(2), 601-653.

Petrongolo, B., & Pissarides, C. A. (2001). Looking into the black box: A survey of the matching function. *Journal of Economic Literature*, 39(2), 390-431.

Pratt, J. W. (1964). Risk Aversion in the Small and in the Large. *Econometrica*, 32(1/2), 122–136.

Snowden, F. (2020a). Uprisings after Pandemics Have Happened Before: Just Look at the English Peasant Revolt of 1381. *Conversation*, June, 5.

Snowden, F. (2020b). *Epidemics and Society: From the Black Death to the Present.* New Haven: Yale University Press.

Stokey, N. L., Lucas, R. E., & Prescott, E. C. (1989). *Recursive Methods in Economic Dynamics*. Harvard University Press.

Topkis, D. M. (1978). Minimizing a submodular function on a lattice. *Operations Research*, 26(2), 305-321.

Toxvaerd, F. (2019). Rational disinhibition and externalities in prevention. *International Economic Review*, 60(4), 1737-1755.

Wilder, B., M. Charpignon, J. Killian, O. Han-Ching, A. Mate, S. Jabbari, A. Perrault, A. Desai, M. Tambe and M. Majumder, (2020). The role of age distribution and family structure on COVID-19 dynamics: A preliminary modeling assessment for hubei and lombardy. Available at SSRN, 3564800.

1 Online Appendix of Supplementary Material (not for publication)

1.1 Patterns of Mask-wearing Frequencies

Figure 1 in the paper shows the mask-wearing frequency in four leading European capitals. This section introduces how the results are obtained and how the frequency of mask-wearing is distributed in more cities during the pandemic. Mask-wearing is an appropriate counterpart to the action in our theoretical model because it is an effective and low-cost intervention to limit the spread of the virus[1]. In addition, facial covers are not worn by ordinary people in the absence of a pandemic and have traditionally been considered essential only for healthcare workers (Taylor et al. 2021), so we assume that a zero mask-wearing frequency corresponds to a choice of "life as usual". Since the frequency is a continuous random variable between zero and one, it can be easily mapped into the behavior variable in the theoretical model, i.e., $a_t = 1-$ frequency of mask-wearing.

Our analysis employs the YouGov COVID-19 behavior tracker data collected by Imperial College London[2], which interviewed around 29,000 people every week since April 2020. The data contain information about people's self-reported choices of normal preventative measures and perceptions of COVID-19. Regarding their

[1]Recent medical research has identified face-covering as one of the primary non-pharmaceutical interventions that can substantially limit the spread of infection during the pandemic (Schunemann et al. 2020, Abaluck et al. 2022), especially when COVID-19 is highly transmissible from presymptomatic and asymptomatic individuals (Howard et al. 2020). Compared to other preventative measures like observing social distancing, compliance with shelter-in-place directives, and working from home, face-covering has a limited negative impact on economic activity (Milosh et al., 2021; Goolsbee and Syverson, 2020, Chetty et al., 2020, Coibion et al., 2020). Although wearing a face cover may cause a reduction in utility due to uncomfortable breathing and difficulty in communication, it comes at a low cost, and is thus less likely to be affected by an individuals financial situation (Wright et al., 2020).

[2]See https://github.com/YouGov-Data/covid-19-tracker for more details of the survey.

mask-wearing frequency, respondents are asked to choose one of the five options, "always", "frequently", "sometimes", "rarely", and "not at all". We refer to "always" and "not at all" as extreme options and refer to the other three as moderate options. Our analysis covers data from April 1st to Dec 31st, 2020, when vaccinated people in each country were still a negligible fraction of the population[3]. Figure A1 shows the proportions of people with extreme and moderate options during April 8 to April 14, 2020[4]. The proportion of extreme options outweighs the moderate options in every country and territory[5].

The polarized pattern of mask-wearing frequency in Figure A1 may, however, be driven by within-country variations in pandemic severity (Allcott et al. 2020). Moreover, individual behaviors can be shifted by face mask usage recommendations from the government (Feng et al. 2020). A mandatory mask-wearing order from the government may compel people to choose to always wear a mask, leading to an ad hoc cluster of peoples choices at extreme options. To address these concerns and achieve more rigorous results, we further conduct analysis using local area data where there is no mandatory policy on mask-wearing.

[3]According to data from ourworldindata.org, less than 1% of the population were vaccinated in the U.S. by the end of 2020.

[4]Due to data limitations, data from April 1-7, 2020 is used for Canada and Mexico. We did not include data from Mainland China because stringent mask-wearing orders have been implemented ever since the outbreak in early 2020.

[5]The percentage of respondents choosing extreme options is 0.50 in Singapore, the lowest among all countries and territories in Figure A1. One reason to the large cross-country variation in Figure A1 could be people's perceptions about the utility reduction caused by mask-wearing. For example, a question included in the YouGov survey during May 2020 asked respondents about their mask-wearing experience outside their homes. The proportion of people who chose "general discomfort" ranges from 8.7% in Mexico to 49.6% in the United Kingdom, while the proportion of people who chose "hard to communicate" ranges from 8.9% in Saudi Arabia to 41.0% in the United Kingdom. Other factors like country-specific exogenous features, such as population demographics, population density, and availability of healthcare resources may influence people's choices by affecting the transmission rate of COVID-19. Nevertheless, Figure A1 shows that the majority of people choosing moderate options is not observed in almost every country and territory.

Local area data: We choose metropolitan areas to strike a balance between adequate number of observations and sufficiently small regions[6]. Our sample covers 18 metropolitan areas worldwide, including Abu Dhabi (United Arab Emirates), Akershus (Norway), Bangkok Metropolitan Region (Thailand), Community of Madrid (Spain), Delhi (India), the Federal District (Mexico), Helsinki-Uusimaa (Finland), Hovedstaden (Denmark), Jakarta (Indonesia), Jeddah (Saudi Arabia), Kuala Lumpur (Malaysia), London (UK), Paris Area (France), Riyadh (Saudi Arabia), Sao Paulo (Brazil), Seoul special city (South Korea), Singapore, and Stockholm (Sweden). These cities are significantly heterogeneous in cultural background, political structure, and ethnicity. In order to reduce the impacts of regional heterogeneity of pandemic severity, we study the distribution of mask-wearing frequency within each city, assuming respondents from the same city are exposed to roughly the same level of transmission-intensity rate. To control for the temporal variation in pandemic severity, we conduct our analysis using observations collected during the same week[7]. This leads to a sample that consists of respondents from 18 cities over 39 weeks (unbalanced), with a total of 404 city-week combinations.

No mandatory policy on mask-wearing: A daily time-series data of each country's facial-covering policy indicator in response to the pandemic is collected from the Oxford COVID-19 Government Response Tracker (OxCGRT).[8]. The policy indica-

[6]The YouGov data provides information about the region of each respondent within a country. However, most of those regions (e.g., a state in the U.S. or a first-level NUTS region in a European country) are too big to rule out the within-region heterogeneity in pandemic severity. Some countries have regional data at a lower administrative level (e.g., Saudi Arabia's data are at the city-level), but the number of respondents is too few to ensure reliable statistical tests.

[7]Respondents are asked how often they wore face masks outside their home in the last seven days. Therefore, the underlying assumption of this setup is that the pandemic severity remains roughly on the same level within a biweekly time window. Since the survey data are mostly collected in two or three consecutive days in a week, the effective length of the time window is mostly 9-10 days. Our main findings still hold if we restrict our analysis using observations interviewed on the same day, with some test results being less significant due to the smaller sample size.

[8]See https://github.com/OxCGRT/covid-policy-tracker for more details of the data.

tor takes integer values ranging from 0 to 4 to stand for different levels of facial covering regulation: no policy, recommended, required in some specified shared/public space with other people present, required in all shared/public space with other people present, and required outside the home at all times regardless of presence of other people. We focus on the periods when the policy indicator is less than or equal to one, and with no policy change within one week, which yields a subsample with 125 city-week pairs from 12 cities.

Test for Polarized Behaviors:

After controlling for policy intervention and regional and temporal heterogeneity in pandemic severity, we now study whether the distribution of mask-wearing frequency is consistent with the polarized pattern predicted by Proposition 1. If the type of respondents follows a smooth distribution with no clustered probability mass at extreme points[9], Proposition 2 predicts that the mask-wearing frequency should also follow a smooth distribution between zero and one. In contrast, Proposition 1 implies that mask-wearing frequency should follow a binary distribution, with only point masses at zero and one. These two distinct predictions imply a testable hypothesis: whether the probability mass of the distribution is concentrated at its boundaries (Proposition 1) or at some interior point (Proposition 2).

Due to the data limitation, we only have quinary instead of continuous observations on the frequency of mask wearing. Hence, in order to test if there are more observations near two boundaries, we consider the following hypotheses

$$\begin{aligned} H_0: \ & \text{Prob(choosing extreme options)} \ \leq \ \mathcal{T} \\ \text{versus} \quad H_1: \ & \text{Prob(choosing extreme options)} \ > \ \mathcal{T}, \end{aligned} \tag{1}$$

where $\mathcal{T}$ is a pre-specified threshold and we set $\mathcal{T} \in \{0.5, 0.6, 0.7\}$. Apparently, H_1 with $\mathcal{T} = 0.5$ means that more than half of the probability mass is concentrated at the extreme options. Hence, a rejection of H_0 in favour of H_1 suggests that the

[9]This is a reasonable assumption in our dataset. The appendix provides the histograms of a number of characteristics of respondents, none of which exhibits a distribution with large probability mass at the extreme points.

data are more likely to be generated by a model that satisfies Proposition 1. The larger $\mathcal{T}$ is, the stronger the evidence to support the polarization pattern predicted by Proposition 2 if H_0 is rejected. The hypotheses in (1) can be examined using a binomial test for each given $\mathcal{T}$. Although the test is valid even in small samples, we focus on city-weeks with at least 50 or 100 respondents to reduce the sampling uncertainty and generate more robust results.

Results: Table A1 summarizes the findings. Panel A shows the results for city-weeks with at least 50 respondents. When the policy index takes a value of 0 or 1 and the threshold $\mathcal{T}$ takes a value of 0.5, 91(85) out of 100 tests reject the null hypothesis at the 5% (1%) significance level. 77/54 (72/51) out of 100 tests reject the null hypothesis at the 5% (1%) significance level when the testing threshold value takes a value of 0.6/0.7. We obtain similar results for city-weeks with at least 100 respondents in Panel B. Roughly 90% (50%) of the city-week pairs reject the null hypothesis at 5% significance level at a threshold $\mathcal{T}$ of 0.5 (0.7). Columns (4) to (6) further show the results when there is no policy recommendation from the government (the policy index takes a value of 0). The results are qualitatively similar. The robust pattern we show in Table A1 demonstrates that people's choices regarding mask-wearing frequency are more inclined to be polarized, which is consistent with Proposition 1 in the theoretical model.

Using the YouGov survey data, we can further test the implications of Proposition 4 by examining how individual characteristics affect mask-wearing behavior. An ordered logit model is estimated for the choice regarding mask-wearing frequency using data collected in the same metropolitan areas[10], and in periods when the policy index takes a value of 0 or 1. Table A2 presents the summary statistics of key variables used in our empirical investigation. We include four sets of variables from the survey to proxy for individual traits in the theoretical model. Firstly, we include each individual's age, gender and underlying medical condition that could be related to their discount factor (δ), probability of contracting the disease upon infection (γ),

[10] We also estimate an ordered logit model using respondents from each country in the data. The results are qualitatively similar.

and expectation of suffering when ill from the disease (D). The underlying medical condition is measured by the number of illnesses the respondent has. Since 97.2% of respondents report no more than 3 underlying disease, we set the number of diseases as discrete values from 0 and 3. Secondly, family status, including fatality status and family size, are also related to δ and D. The fertility status is measured by the number of children a respondent has (truncated at 5 by the survey). The size of the family is denoted by the number of family members (truncated at 8 by the survey). Thirdly, we include each individual's employment status to measure the economic factors that may affect δ and D. The respondents are classified into 7 types based on their employment status: full-time employed, part-time employed, student, retired, unemployed, not working and others. Lastly, we include a self-reported indicator about fear of contracting COVID-19, which is classified as very / fairly / not very / not at all scared of COVID-19 (denoted as integer values 1 to 4). The survey question of "feelings towards contracting COVID-19" was only asked from April to September 2020, which therefore overlaps the sample periods of our analysis. Table A3 shows the results. We find that respondents with more children, a larger family, underlying medical conditions, or who are more scared of the virus are more willing to always wear masks instead of never wearing them. This is consistent with the implication of Proposition 4.

1.2 Asymptomatic Infections and Communicable Behaviors

This section shows empirical observations consistent with Proposition 4. We investigate whether people's belief of having been asymptomatically infected is linked with more communicable activities during the pandemic. Our analysis also uses the YouGov survey data. We find that (1) the proportion of survey respondents who believe they have been infected since March 2020 has a higher growth rate than the officially announced cumulative cases in each country, indicating the existence of asymptomatic (and not tested) infections; (2) people who believe that they have been infected are significantly more risk-taking in each of the 24 communicable activ-

ities surveyed in the data (as listed in Column (1) in Table A4), after we control for individual characteristics. The results are consistent with Lemma 2 and Proposition 4.

Apart from mask-wearing frequency, the YouGov survey interviews respondents about their self-reported frequencies to apply other preventive measures, for instance, washing hands, avoiding taking public transport, avoiding attending public events, etc. To each of the 24 questions regarding these behaviors, respondents are asked to choose one of the five options, "always", "frequently", "sometimes", "rarely", and "not at all". During 1/24-2/1, 2022, the survey introduced one question of "have you tested positive for COVID-19 since March 2020" in 11 countries (as listed in Column (1) in Table A6). Later, during 2/2-3/29, 2022, the question was replaced by "do you believe you have had COVID-19 since March 2020". Using answers to the two questions and self-reported communicable behaviors, our analysis focuses on two questions, (1) are (at least a subset of) the respondents who "believe" they have had Covid-19 have not been tested (either asymptomatic or with mild symptoms); and (2) how is the belief of having been infected linked with people's communicable behaviors.

Regarding the first question, we find that the growth rate of the proportion of respondents who believe they have been infected is higher than that of the officially reported cumulative cases. Firstly, we compare the proportion of respondents who have been tested positive (surveyed during 1/24-2/1, 2022, henceforth, period 1) with that of respondents who believe that they have been infected (surveyed during 2/2-3/29, 2022, henceforth period 2). Column (1) in Table A4 shows the proportion of respondents (surveyed in period 1) who have been tested positive since March 2020. Column (2) is the proportion of respondents (surveyed in period 2) who believe that they have been infected since March 2020. Column (3) shows the growth rate of Column (1) relative to Column (2). In Column (4), (5) and (6), we list the average share of cumulative cases in total population in period 1 and 2, and the growth rate. Column (7) is the difference between Column (3) and (6). It shows that in each of the 11 countries, the value of Column (7) is positive, indicating a

substantial probability that a subset of respondents with the belief of having been infected have not been tested positive.

Furthermore, we look into period 2. For each day in the survey, we compute the proportion of respondents with belief of being infected (believe-ratio) as well as the officially share of reported cumulative cases in population(official-ratio). We conduct a linear regression of the two ratios on daily trend, respectively. The results are shown in Table A5. In Column (1)-(2), we use the OLS, and country fixed effects are included in Column (3) to (6). In Column (5) and (6), we exclude the country-week pairs with less than 20 respondents as robustness checks. The results suggest that the believe-ratio increases faster with time than the cumulative cases, consistent with the finding in Table A4.

Secondly, we compare the communicable behaviors of respondents who answer "yes" and "no" to the question of "do you believe you have had COVID-19 since March 2020". We firstly look into people's mask-wearing behaviors. Figure A2 shows that the proportion of respondents choosing *always* wear a mask outside home is larger for the "yes" group in each country.

Next, we measure communicable behaviors with integer values of 1 to 5 where 1 indicates "always" and 5 indicates "not at all". Table A6 compares the unconditional and conditional mean value of 24 behaviors covered by the survey. Column (4) and (5) shows the difference of the unconditional mean (Column (2) and (3)) of two groups of respondents and t statistics. For all 24 behaviors, the "yes" group has significant higher value than the "no" group. Column (6) and (7) shows the estimated coefficient and t statistics on the "yes" dummy in a country fixed-effects regression of behaviors, controlling for individual characteristics including age, gender, size of household, number of children, employment status, number of underlying diseases. For all of the behaviors (except for avoiding working outside home and letting children to school), the coefficient on the "yes" dummy is significantly positive, indicating that people who believe they have been infected have more risk-taking behaviors, ceteris paribus. The results are qualitatively similar with Figure A2.

1.3 Supplementary results for counterfactual analysis

Table A7 shows a descriptive summary of the duration data we use for counterfactual analysis in Section 5. Panel A and B show that the proportions of respondents with zero socializing time during July to December reduce substantially from 33.8% in 2019 to 26.7% in 2020, while that during January to February only reduced slightly from 31.5% in 2019 to 30.1% in 2020. Moreover, the average durations of non-zero socializing from July to December are similar between 2019 (123.62) and 2020 (123.91). Similar results are obtained in Panel C and D for more communicable behaviors.

Table A8 reports the descriptive statistics for the control variables we use in the distributional regression, including age, employment status (dummy for employed or not), family income (classified into 16 categories from "less than $5,000" to "$150,000 and over"), number of children under 18, age of youngest child under 18, and dummy for not having children under 18 of each respondents, as well as day of the week of the diary day (1-7 for Monday to Sunday). We report the mean and standard deviation (in parenthesis) of each variable for respondents with zero / non-zero socializing time, during Jan to February (pre) and July to December (post), for 2019 and 2020, respectively. It shows that respondents with non-zero socializing time during the pandemic are slightly younger and with higher family income than the other groups.

Figure A3 reports the results for testing (29) using the augmented data for three types of daily activities: socializing, dining in restaurants, and exercising in public indoor spaces. The null hypothesis H_0^1 cannot be rejected when we compare the pre-pandemic period of 2020 to the same months in 2019 (i.e., January and February), but it is strongly rejected in favor of H_1^1 when comparing the second half of 2020 to the same period in 2019. This result consolidates the findings in Section 5.

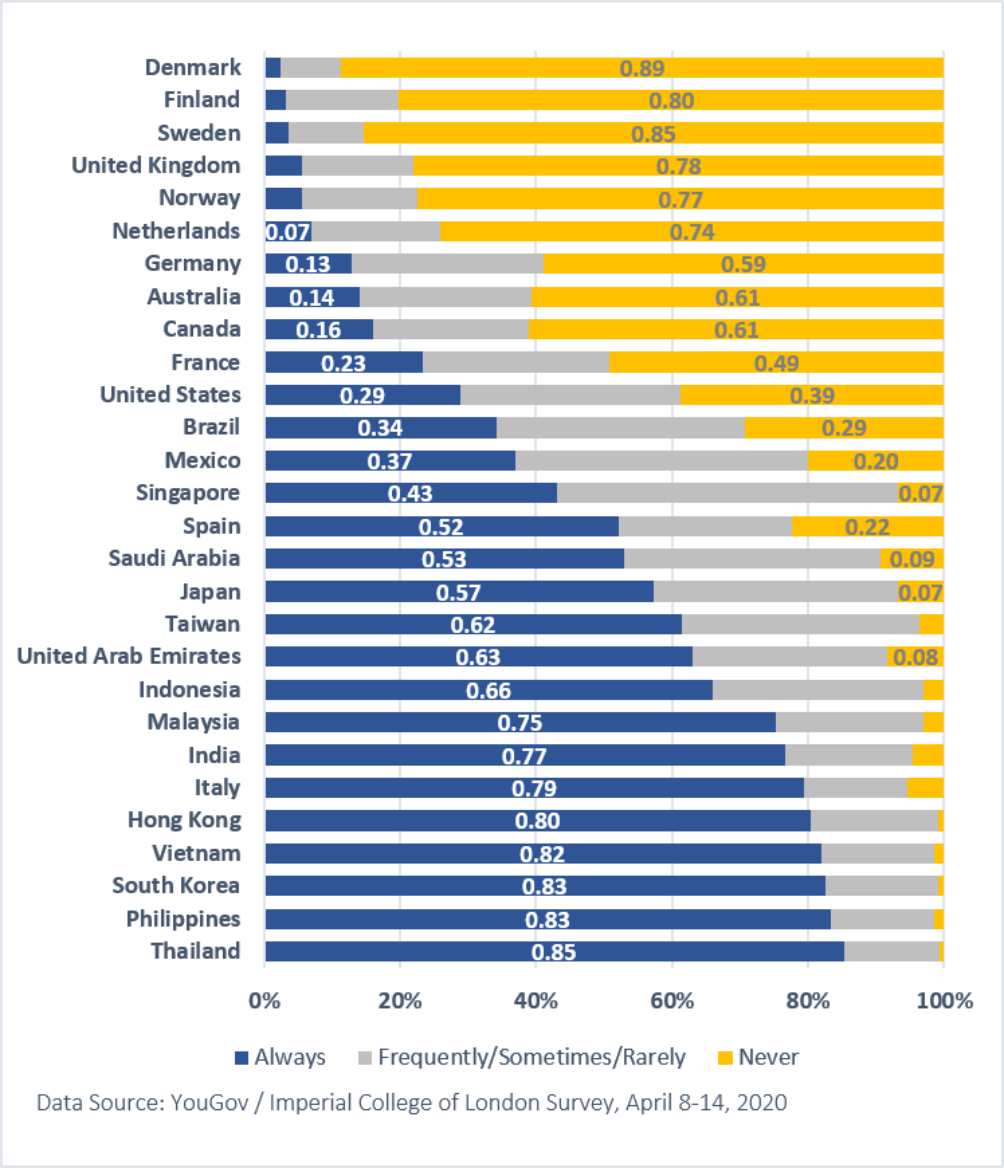

Figure A1. Mask-wearing Frequencies in Different Countries and Territories

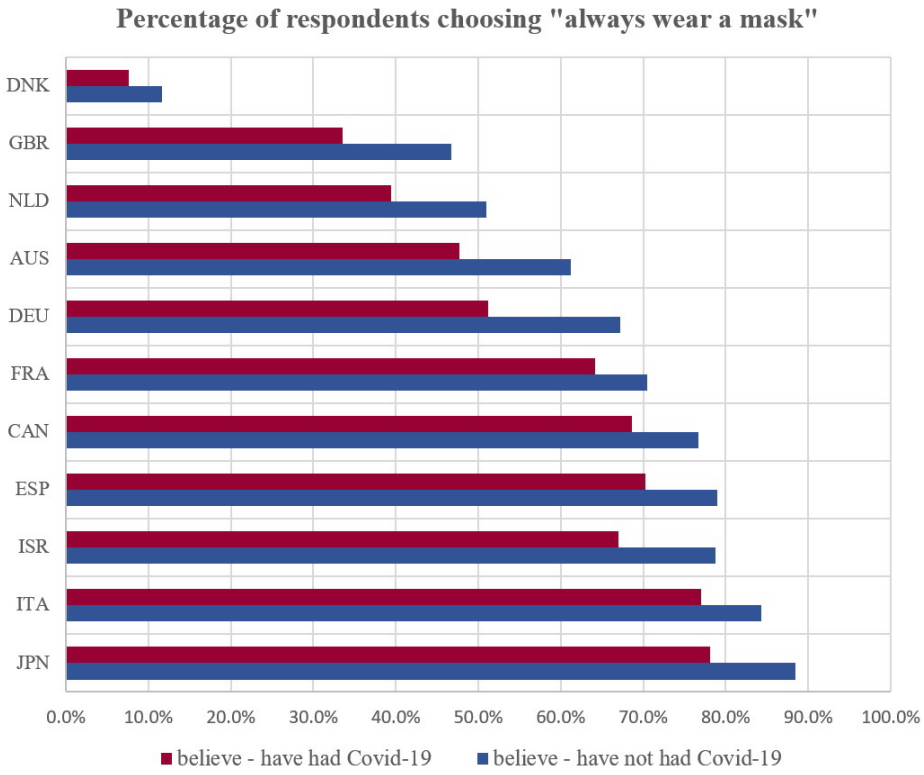

Figure A2. Mask-wearing frequency and people's belief about having been infected

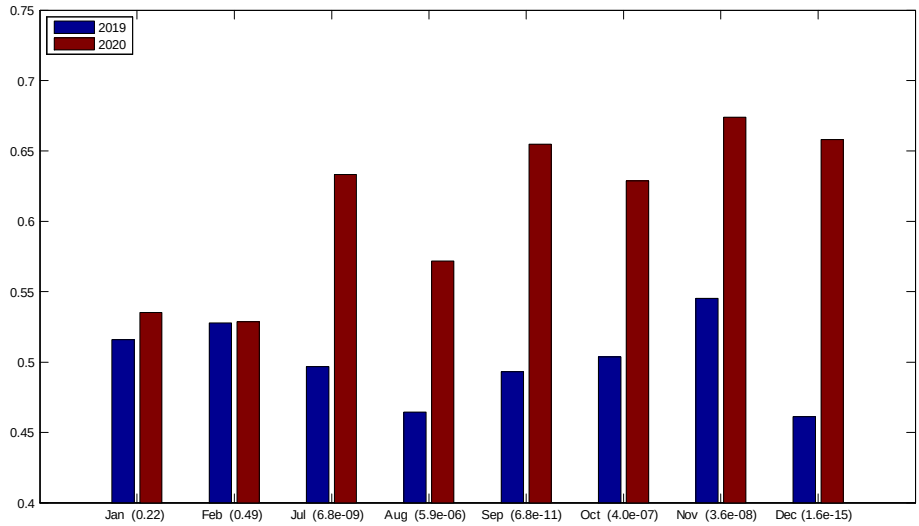

Figure A3. The proportions of people spending no time on socializing, dining in restaurants, and exercising in public indoor spaces in 2019 and 2020

The numbers in paretheses are the p-values for the null hypothesis that the proportion in 2020 remains the same compared to the same month in 2019.

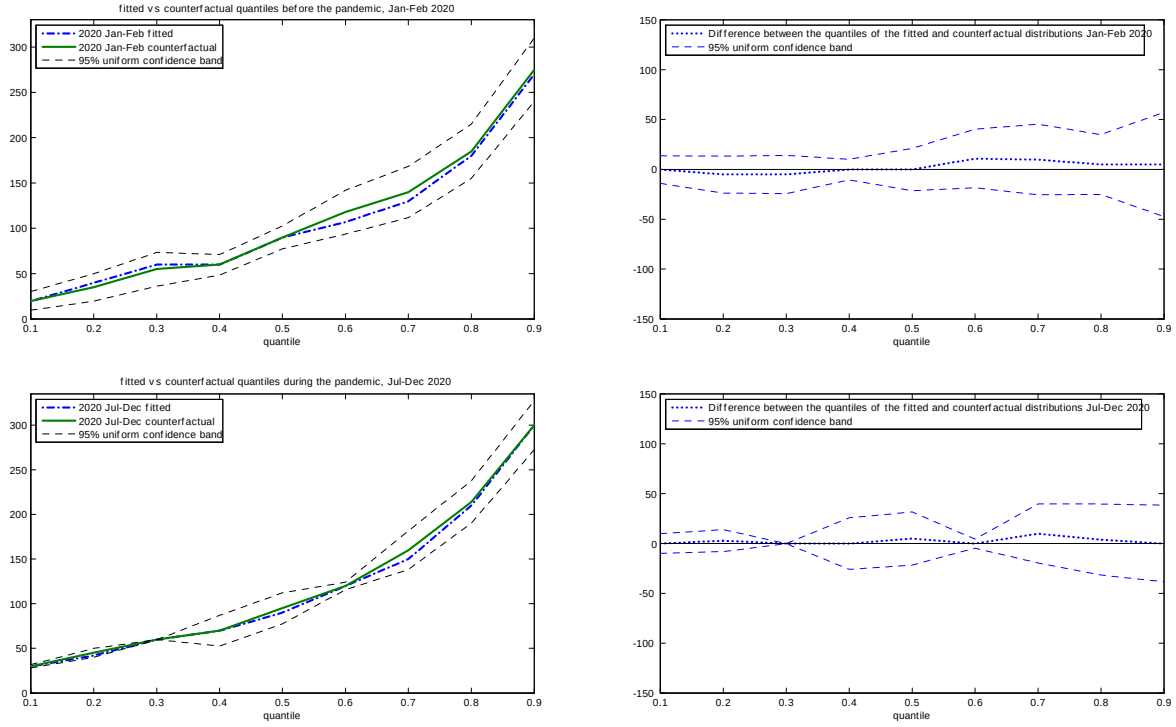

Figure A4. Fitted versus counterfactual quantiles of daily time spent socializing, dining in restaurants, and exercising in public indoor spaces for people who engaged in these activities in 2020

The p-values of the Kolmogorov-Smirnov statistic for testing H■?are 0.87 and 0.79 before and during the pandemic, respectively.

Table A1. Results of tests for polarized behaviors

	(1)	(2)	(3)	(4)	(5)	(6)
policy index	0 or 1			0		
value of threshold $\mathcal{T}$	0.5	0.6	0.7	0.5	0.6	0.7
Panel A: Number of respondents ≥ 50						
total number of city-week pairs	100			74		
Rejecting $H0$ at 5% significance level	91	77	54	71	68	58
Rejecting $H0$ at 1% significance level	85	72	51	71	68	55
Panel B: Number of respondents ≥ 100						
total number of city-week pairs	91			66		
Rejecting $H0$ at 5% significance level	89	72	44	65	63	50
Rejecting $H0$ at 1% significance level	83	68	42	65	63	48

Table A2. Descriptive Statistics of YouGov survey data

Variable	Obs.	Mean	Std. Dev	Min	Max
Mask-wearing frequency	12,852	4.074	1.437	1	5
fear	12,852	2.587	0.843	1	4
disease (#D)	12,852	0.708	0.950	0	3
household size (hh)	12,852	2.434	1.340	1	8
number of children (children)	12,852	0.806	1.113	0	5
female	12,852	0.502	0.500	0	1
age	12,852	46.00	17.08	18	90
Employment status:					
full-time employed	12,852	0.487	0.500	0	1
part-time employed	12,852	0.102	0.302	0	1
student	12,852	0.917	0.289	0	1
retired	12,852	0.211	0.408	0	1
unemployed	12,852	0.059	0.235	0	1
not-working	12,852	0.045	0.207	0	1

Table A3: Individual characteristics and mask-wearing frequency

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Variables	whole	Madrid	Helsinki	Hovedstaden	London	Oslo	Paris	Riyadh	Stockholm
female	0.107***	-0.295	-0.0716	0.830***	0.0328	0.430***	-0.218	-1.202***	0.491***
	(0.0373)	(0.292)	(0.0879)	(0.111)	(0.135)	(0.102)	(0.175)	(0.239)	(0.103)
logage	1.997*	0.361	4.969*	2.951	7.090*	-7.807***	1.046	4.328	4.902*
	(1.170)	(12.60)	(2.599)	(3.338)	(3.896)	(2.980)	(7.130)	(7.579)	(2.909)
logage2	-0.203	-0.125	-0.692*	-0.152	-0.972*	1.259***	-0.141	-0.622	-0.507
	(0.161)	(1.698)	(0.360)	(0.462)	(0.545)	(0.415)	(0.972)	(1.063)	(0.405)
#D.1	-0.0773*	0.308	-0.331***	0.0143	-0.149	-0.126	0.193	0.268	-0.374***
	(0.0454)	(0.309)	(0.102)	(0.140)	(0.159)	(0.114)	(0.212)	(0.235)	(0.116)
#D.2	-0.176**	-0.191	-0.340**	-0.0769	-0.446	-0.627***	-0.322	-0.127	-0.405**
	(0.0689)	(0.464)	(0.141)	(0.206)	(0.300)	(0.168)	(0.359)	(0.380)	(0.171)
#D.3	-0.322***	-0.882	-0.485***	-0.770***	-0.249	-0.405**	-0.182	0.544	-0.806***
	(0.0795)	(0.652)	(0.161)	(0.206)	(0.349)	(0.186)	(0.428)	(0.378)	(0.198)
children	-0.0678***	0.229	-0.0996	-0.152**	-0.0730	-0.173***	-0.122	0.0233	-0.196***
	(0.0235)	(0.170)	(0.0648)	(0.0766)	(0.0828)	(0.0584)	(0.0877)	(0.0544)	(0.0727)
hh	-0.0730***	-0.0328	-0.0530	-0.182***	0.00253	-0.128***	-0.0620	0.0112	-0.0378
	(0.0170)	(0.127)	(0.0462)	(0.0562)	(0.0603)	(0.0445)	(0.0735)	(0.0556)	(0.0554)

Table A3: Individual characteristics and mask-wearing frequency (continued)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Variables	whole	Madrid	Helsinki	Hovedstaden	London	Oslo	Paris	Riyadh	Stockholm
full	0.0879	0.723	-0.411*	-0.310	-0.164	0.0684	-0.399	-0.836	0.472*
	(0.243)	(0.707)	(0.249)	(0.436)	(0.395)	(0.237)	(0.616)	(0.867)	(0.276)
part	-0.241	0.858	-0.557**	-0.453	-0.237	-0.395	-0.689	-0.668	-0.175
	(0.248)	(0.871)	(0.264)	(0.457)	(0.439)	(0.262)	(0.715)	(0.887)	(0.303)
student	-0.121	0.714	-0.560**	0.0738	-0.621	-0.438	-0.345	-0.113	0.0597
	(0.255)	(1.043)	(0.283)	(0.466)	(0.476)	(0.284)	(0.788)	(0.947)	(0.315)
retired	-0.0908	1.087	-0.193	-0.811*	-0.0751	-0.633**	-0.858	-0.352	-0.245
	(0.252)	(0.849)	(0.272)	(0.489)	(0.476)	(0.294)	(0.692)	(1.012)	(0.323)
unemployed	0.261	0.643	0.303	0.461	0.0316	0.155	-0.0436	-0.316	-0.323
	(0.255)	(0.803)	(0.302)	(0.511)	(0.470)	(0.328)	(0.686)	(0.960)	(0.351)
notworking	0.233	1.729**			0.114		-0.480	0.814	
	(0.259)	(0.809)			(0.467)		(0.948)	(0.954)	
fear	0.416***	0.564***	0.594***	0.140**	0.407***	0.577***	0.378***	0.172*	0.552***
	(0.0232)	(0.164)	(0.0602)	(0.0695)	(0.0884)	(0.0600)	(0.0995)	(0.104)	(0.0669)
N	16,367	275	2,787	2,595	865	2,429	478	598	2,494
r2_p	0.301	0.0559	0.0597	0.100	0.0315	0.0953	0.0281	0.0369	0.0706

Note: Ordered logit regression of mask-wearing frequency on individual characteristics. All regressions include week dummies. Standard errors are reported in parentheses, * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A4. Trend in the proportion of respondents who believe they have been infected: Period 1 v.s. Period 2

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Tested positive (period 1)	Believe have (period 2)	Growth rate (survey)	Cum. Cases / population (period 1)	Cum. Cases / population (period 2)	Growth rate (official)	Difference: (3) - (6)
Australia	0.100	0.152	52.37%	0.097	0.135	39.11%	13.26%
Canada	0.114	0.224	96.03%	0.081	0.088	9.17%	86.85%
Denmark	0.209	0.39	87.03%	0.268	0.462	72.28%	14.75%
France	0.189	0.314	66.25%	0.256	0.330	29.13%	37.12%
Germany	0.115	0.225	95.22%	0.108	0.184	69.20%	26.02%
Israel	0.234	0.491	109.68%	0.292	0.400	37.15%	72.53%
Italy	0.168	0.246	46.39%	0.169	0.213	25.95%	20.45%
Japan	0.016	0.036	128.58%	0.018	0.039	115.25%	13.33%
Netherlands	0.233	0.406	74.16%	0.240	0.379	58.38%	15.78%
Spain	0.265	0.353	33.00%	0.201	0.233	15.87%	17.13%
United Kingdom	0.214	0.344	60.42%	0.239	0.286	19.39%	41.03%

Table A5. Trend in the proportion of respondents who believe they have been infected: Period 2

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	official ratio	believe ratio	official ratio	believe ratio	official ratio	believe ratio
time	0.00177***	0.00278***	0.00144***	0.00224***	0.00142***	0.00164***
	(3.584)	(3.559)	(20.80)	(3.741)	(17.15)	(6.185)
country FE	NO	NO	YES	YES	YES	YES
Observations	268	268	268	268	208	208
R-squared	0.046	0.045	0.982	0.468	0.981	0.773

t-statistics in parentheses. *** p<0.01, ** p<0.05, * p<0.1. drop if observations<20 in Column (5) and (6).

Table A6. Communicable behaviors and people's belief about having been infected

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
			unconditional			conditional	
Behavior	obs	Mean-Yes	Mean-No	diff	t stat	beta	t-stat
wearing masks outside	16,474	2.191	1.814	0.377	30.94	0.117***	5.842
wash hands	16,474	1.675	1.514	0.161	16.223	0.0830***	5.038
use hand sanitiser	16,474	2.268	2.033	0.235	23.117	0.159***	7.244
cover nose and mouth when sneezing	16,474	1.538	1.398	0.139	16.511	0.0417**	2.566
avoid contact with people with symptoms	16,474	2.250	1.835	0.415	35.224	0.209***	8.928
avoid going out in general	16,474	3.650	3.304	0.346	33.327	0.149***	5.932
avoid going to hospital	16,474	3.124	2.795	0.329	26.691	0.133***	4.567
avoid taking public transport	16,474	2.941	2.518	0.423	32.547	0.170***	5.864
avoid working outside home	9,151	3.788	3.619	0.169	13.966	0.0395	1.095
avoid letting children to school	5,184	3.894	3.864	0.030	2.459	-0.0363	-0.822
avoid guests to home	16,474	3.217	2.793	0.424	30.676	0.208***	7.720
avoid small gatherings	16,474	3.467	3.014	0.452	41.153	0.201***	7.428
avoid median gatherings	16,474	3.213	2.667	0.546	46.796	0.253***	9.379
avoid large gatherings	16,474	2.885	2.291	0.594	46.709	0.274***	10.230
avoid crowded areas	16,474	2.823	2.272	0.550	46.879	0.266***	10.960
avoid going to shops	16,474	3.724	3.481	0.244	23.767	0.130***	5.305
wearing mask in grocery store	16,474	2.054	1.659	0.395	30.634	0.108***	5.782
wearing mask in clothing store	16,474	2.028	1.727	0.300	19.917	0.117***	5.361
wearing mask in workplace	9,151	2.616	2.232	0.385	26.527	0.103***	3.297
wearing mask on public transport	16,474	2.174	1.842	0.332	24.734	0.141***	5.786
avoid public events	16,474	2.905	2.339	0.567	44.037	0.263***	9.377
avoid mixing with other households	16,474	3.292	2.795	0.497	44.356	0.238***	9.217
avoid staying overnight outside home	16,474	2.871	2.377	0.494	37.285	0.224***	7.658
avoid travelling outside local area	16,474	3.241	2.803	0.438	36.658	0.179***	6.462

Table A7. Descriptive statistics of durations of socializing, outdining and exercising

month	variable	obs.	mean	s.d.	min	max
	A. socializing, 2019					
1-2	duration	1,580	34.67	78.51	0	625
	duration (>0) (share:31.5%)	497	109.58	106.66	1	625
7-12	duration	4,561	41.82	90.67	0	861
	duration (>0) (share:33.8%)	1,543	123.62	119.14	1	861
	B. socializing, 2020					
1-2	duration	1,609	34.88	88.46	0	990
	duration (>0) (share:30.1%)	498	112.70	128.57	2	990
7-12	duration	5,002	33.15	81.13	0	720
	duration (>0) (share:26.7%)	1,338	123.91	115.60	1	720
	C. socializing, outdining, exercising in public spaces, 2019					
1-2	duration	1,580	52.52	93.74	0	720
	duration (>0) (share:44.4%)	702	118.20	109.63	1	720
7-12	duration	4,561	62.62	105.95	0	861
	duration (>0) (share:47.7%)	2,176	131.25	120.52	1	861
	D. socializing, outdining, exercising in public spaces, 2020					
1-2	duration	1,609	50.41	97.66	0	990
	duration (>0) (share:43.0%)	693	117.04	119.79	2	990
7-12	duration	5,002	44.55	93.19	0	750
	duration (>0) (share:34.0%)	1,701	130.99	119.23	1	750

Table A8. Descriptive statistics of control variables

	2019				2020			
variable	0 pre	0 post	>0 pre	>0 post	0 pre	0 post	>0 pre	>0 post
	(obs:824)	(obs:2,253)	(obs:756)	(obs:2308)	(obs:856)	(obs:3,175)	(obs:753)	(obs:1,827)
age	51.49	51.49	50.35	51.04	51.50	52.22	50.61	49.75
	(17.91)	(17.77)	(18.08)	(18.33)	(17.72)	(18.35)	(18.25)	(18.03)
emp	0.58	0.58	0.57	0.57	0.58	0.53	0.56	0.56
	(0.49)	(0.49)	(0.50)	(0.50)	(0.49)	(0.50)	(0.50)	(0.50)
faminc	11.16	11.03	11.66	11.91	11.14	11.71	12.16	12.27
	(4.04)	(4.09)	(3.85)	(3.81)	(4.01)	(3.79)	(3.78)	(3.53)
childnum	0.68	0.68	0.72	0.69	0.64	0.62	0.72	0.68
	(1.10)	(1.10)	(1.06)	(1.05)	(1.04)	(1.02)	(1.12)	(1.07)
kidage	3.72	3.73	4.06	3.95	3.71	3.52	3.99	3.85
	(5.90)	(5.90)	(6.04)	(6.01)	(5.93)	(5.78)	(6.08)	(5.99)
nokid	0.65	0.65	0.61	0.62	0.65	0.66	0.63	0.64
	(0.48)	(0.48)	(0.49)	(0.49)	(0.48)	(0.47)	(0.48)	(0.48)
diaryday	3.75	3.84	3.75	3.99	3.99	3.90	4.08	4.05
	(2.25)	(2.30)	(2.36)	(2.39)	(2.29)	(2.32)	(2.37)	(2.43)

1.4 References

Abaluck, J., Kwong, L. H., Styczynski, A., Haque, A., Kabir, M. A., Bates-Jefferys, E., ... & Mobarak, A. M. (2021). Impact of community masking on COVID-19: A cluster-randomized trial in Bangladesh. *Science* , eabi9069.

Allcott, H., Boxell, L., Conway, J., Gentzkow, M., Thaler, M., & Yang, D. (2020). Polarization and public health: Partisan differences in social distancing during the coronavirus pandemic.*Journal of Public Economics*, 191, 104254.

Andersen, M. (2020). Early evidence on social distancing in response to COVID-19 in the United States. Available at SSRN 3569368.

Chetty, R., Friedman, J. N., Hendren, N., Stepner, M., & The Opportunity Insights Team. (2020). How did COVID-19 and stabilization policies affect spending and employment? A new real-time economic tracker based on private sector data. Cambridge, MA: *National Bureau of Economic Research*, No. w27431.

Coibion, O., Gorodnichenko, Y., & Weber, M. (2020). The cost of the covid-19 crisis: Lockdowns, macroeconomic expectations, and consumer spending. *National Bureau of Economic Research*, No. w27141.

Feng, S., Shen, C., Xia, N., Song, W., Fan, M., & Cowling, B. J. (2020). Rational use of face masks in the COVID-19 pandemic. *Lancet Respiratory Medicine*, 8(5), 434-436.

Goolsbee, A., & Syverson, C. (2021). Fear, lockdown, and diversion: Comparing drivers of pandemic economic decline 2020. *Journal of Public Economics*, 193, 104311.

Howard, J., Huang, A., Li, Z., Tufekci, Z., Zdimal, V., van der Westhuizen, H. M., ... & Rimoin, A. W. (2021). An evidence review of face masks against COVID-19. *Proceedings of the National Academy of Sciences*, 118(4).

Milosh, M., Painter, M., Sonin, K., Van Dijcke, D., & Wright, A. L. (2021). Unmasking partisanship: Polarization undermines public response to collective risk. *Journal of Public Economics*, 204, 104538.

Schunemann, H. J., Akl, E. A., Chou, R., Chu, D. K., Loeb, M., Lotfi, T., ... & Mertz, D. (2020). Use of facemasks during the COVID-19 pandemic. *Lancet Respiratory Medicine*, 8(10), 954-955.

Taylor, S., & Asmundson, G. J. (2021). Negative attitudes about facemasks

during the COVID-19 pandemic: The dual importance of perceived ineffectiveness and psychological reactance. *PLoS One*, 16(2), e0246317.

Wright, A. L., Sonin, K., Driscoll, J., & Wilson, J. (2020). Poverty and economic dislocation reduce compliance with COVID-19 shelter-in-place protocols. *Journal of Economic Behavior & Organization*, 180, 544-554.