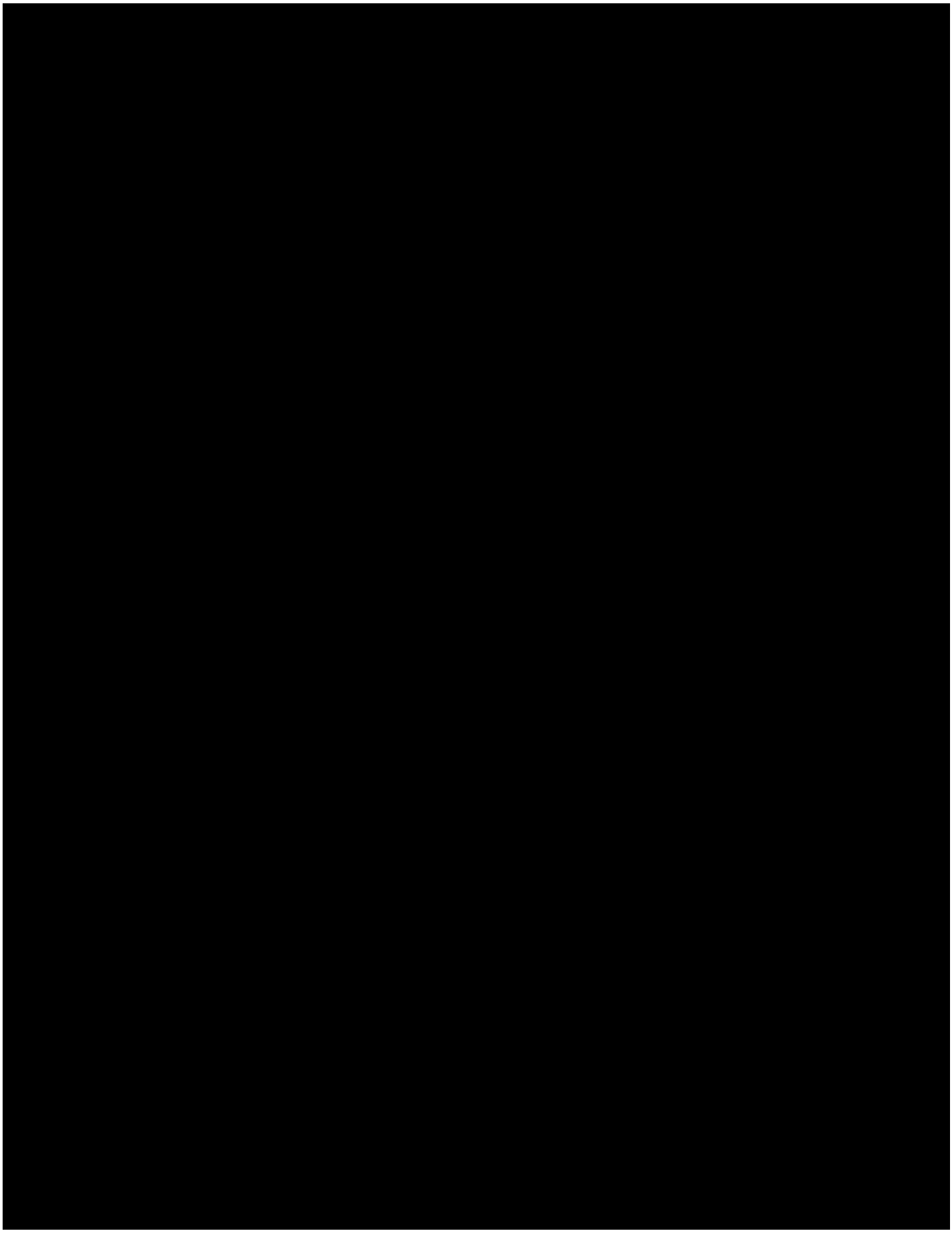

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Information and Inequality in the Time of a Pandemic*

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Abstract

We introduce two types of agent heterogeneity in a calibrated epidemiological search model. First, some agents cannot afford staying home to minimize their virus exposure, while others can. Our results show that these poor agents bear most of the epidemic's health costs. Moreover, we show that having more agents who do not change their behavior during the pandemic could lead to a deeper recession. Second, agents are heterogeneous in developing symptoms. We show that diseases with higher share of asymptomatic cases, even if less lethal, lead to worse health and economic outcomes. Public policies such as testing, quarantining, and lockdowns are particularly beneficial in economies with a larger share of poor agents. However, lockdowns lose effectiveness when part of the agents take precautions to minimize virus exposure independent of government actions.

Keywords: COVID-19, testing, asymptomaticity, time allocation, inequality.
JEL Codes: E17, D62, I12, I14, J22

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I. Introduction

COVID-19 infections present a key feature that make them challenging to control. Different from previous SARS outbreaks, many SARS-COV-2 patients take longer to develop symptoms, and some are completely asymptomatic. Consequently, it is much harder to isolate infected individuals before they transmit the virus to others. Hence, outbreaks can happen rather quickly, many times triggering strict policies such as lockdowns and social distancing.

Moreover, the impact of the pandemic and the measures implemented to try to control it have varied across income levels. Poor neighborhoods, in particular those with heavy minority populations, have seen a significantly larger number of cases and deaths (see Table 1).[1] There are a few reasons for this pattern. First, many of the services deemed essential which have continued to be supplied during the pandemic are done by low wage workers (supermarket personnel, delivery services, etc.). Second, these communities are likely to have denser populations, which facilitates the spread of the disease. Finally, individuals in these communities are usually in occupations that do not allow for remote working (see Dingel and Neiman (2020) and Mongey, Pilossoph, and Weinberg (2020)). They also tend to have very little in fallback savings and wealth. Consequently, they cannot adjust their labor supply optimally in response to a higher likelihood of infection. Similarly, measures to contain the spread of the virus are particularly harsh on poor communities.

Meanwhile, higher income individuals are better able to minimize their exposure to the virus. Even without stay- at-home orders, unconstrained individuals who have the means to adjust their behavior may optimally reduce their exposure to infection. For example, in Figure 1 we show the year-over-year change in the number of seated diners in Texas and the state of Washington using the online, phone, and walk-in reservations from OpenTable.com.[2] The vertical lines indicate the dates in which the states respectively enacted and lifted their stay-at-home orders. Reservations fell significantly well before stay-at-home orders were enacted and remained far below their previous year's levels long after the restrictions were lifted. Similar patterns are observed in air travel and hotel occupancy.

In this paper, we propose and calibrate a search and matching model that allows us to introduce some of these stylized facts in an epidemiological setting. In particular, we consider how

[1] Neighborhood poverty is the percent of a ZIP code's population living below the federal poverty level, in the 2013-2017 American Community Survey. Low poverty: under 10 percent; Medium poverty: 10 percent to 19.9 percent; High poverty: 20 percent to 29.9 percent; Very high poverty: 30 percent and over.

[2] We present Texas and Washington to show a comparison between states that imposed different restrictions and with different timing. All states in Open Table's database show quite similar patterns.

the presence of asymptomatic and pre-symptomatic infected agents as well as the presence of agents who can and cannot optimally adjust their time allocation (henceforth time-constrained agents) impact the evolution of the pandemic. Moreover, we consider how this agents' heterogeneity interact with public policy. Specifically, we discuss how the presence of asymptomatic and pre-symptomatic infected agents as well as time-constrained agents interacts with the effectiveness of tests (both viral and antibody) and lockdowns in reining in the spread of the disease. Furthermore, we consider how these features affect the economic outcomes of policies designed to mitigate GDP losses cased by the disease.

Our results show that introducing asymptomatic and pre-symptomatic agents as well as time-constrained agents is vital to evaluating these disease control policies. Given the same level of pre-infection expected mortality, if the incidence of asymptomatic agents is larger, then the numbers of expected infected agents and expected deaths are larger as well. The reason is because asymptomatic and pre-symptomatic agents spend more time outside infecting healthy but susceptible agents. In turn, the economy will suffer as unconstrained agents are compelled to even more drastically reduce their time spent outdoors as it is riskier to engage in outside activities. Consequently, the impact of testing is significantly larger in an economy with more asymptomatic agents. Viral tests allow us to quarantine infected asymptomatic and pre-symptomatic agents, reducing the infection rate. This not only has the benefit of lowering the expected number of casualties due to the disease, but also allows unconstrained agents to optimally increase their labor supply, improving economic activity. Meanwhile, antibody tests reveal to unconstrained agents who had an asymptomatic infection that they might have acquired immunity, allowing them to optimally adjust their labor supply. As a result, the economic impact of testing is quite substantial in the case in which the incidence of asymptomatic infections is high. In particular, if we consider the case in which 80 percent of the infections are asymptomatic, increasing testing from 0 to 10 percent represents a cumulative gain of around 6.3 percent of GDP after two years. As for the shape of the recession, the harder a country is hit by the epidemic, the quickest its recovery. However, cumulative GDP losses during the pandemic are not recovered.

Similar patterns are observed when the incidence of time-constrained agents is larger. The peak of the infected population is reached earlier and the numbers of infected and dead are significantly higher. Time-constrained agents are particularly affected. Following a calibration that represents an emerging market economy, without considering the possibility of a health system collapse, our results show that 85 percent of time-constrained agents would be infected at some point and 0.18 percent would die. In contrast, only 21 percent of unconstrained agents would get infected and less than 0.05 percent would die. Since the likelihood

of infection is significantly higher in an economy with lots of time-constrained agents, unconstrained agents dramatically reduce their labor supply, with significantly negative impacts on GDP. In our quantitative exercise, an increase of 20 percentage points in the share of time-constrained agents increases GDP losses by 3.7 percentage points.

The impact of viral testing in an economy with more time-constrained agents is again two-fold. First, it reduces the infection rate, by quarantining asymptomatic and pre-symptomatic agents. Notice that the effect of testing is particularly important in the case of time-constrained agents, once these agents do not adjust their time allocation to activities as the infection rate goes up. Consequently, they are not only more likely to contract the virus, but they are also more likely to spread it. Second, as fewer infected agents are allowed to circulate, infection rates go down and unconstrained agents optimally increase their labor supply. In quantitative terms, in an economy with a 50 percent share of time-constrained agents, moving from a 0 percent to a 10 percent incidence of tests implies a reduction of 90,000 in the number of dead from the disease. Similarly, compared to an economy with no testing, a 10 percent testing rate increases GDP by 2.8 percentage points.

In terms of the effect of lockdowns, we show that while lockdowns are able to slow down the virus spread, they are ineffective in reducing economic and human cost of the epidemic if implemented without further measures. In particular, in our benchmark calibration, the introduction of lockdowns reduces the number of deaths due to COVID-19 by 11,500 in the first year and fewer than 5,000 in the second year. In terms of the economic impact, GDP growth falls by 5 percentage points in the first year compared to the no-lockdown benchmark, while there is no difference in the second year. Given the sharp recession in the first year, our quantitative results imply a high cost per life saved. Depending on the type of lockdown – comprehensive and short or mild and long – the cost per life is $26 million and $54 million, respectively. The main reason our costs are significantly higher than the ones featured in other papers in the literature, such as Greenstone and Nigam (2020), is that in our benchmark model, unconstrained agents already optimally adjust their time allocation in response to the possibility of infection. Consequently, the benchmark case already factors in most of the potential benefits of the lockdown at least in a developed country with few time-constrained agents.

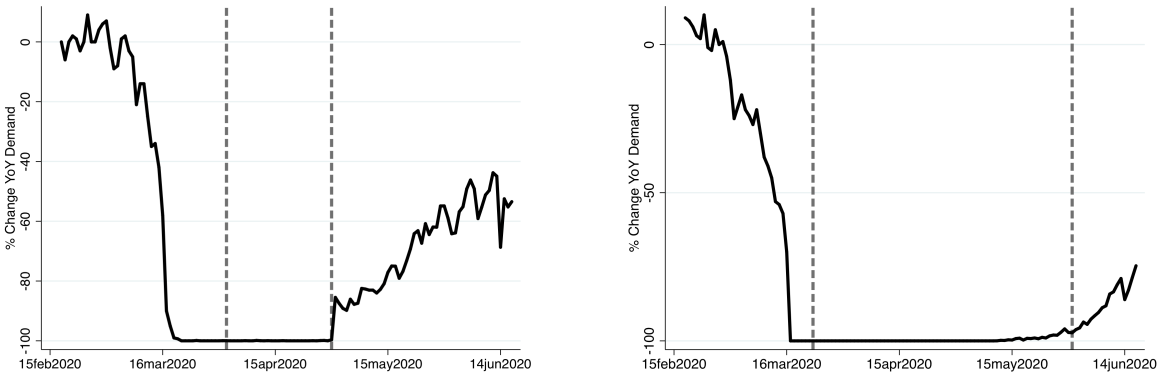
The comparison between the benchmark case and the case with lockdowns highlights that lockdowns are probably more useful in stopping the spread of the disease in developing economies, with high shares of time-constrained agents. In fact, considering the extreme case in which all agents are time-constrained, a mild and long lockdown would be able to save 325,000 lives at a cost of $2.5 million per life, well below our benchmark. However, our

calibration which is closer to an average emerging economy, with around 50 percent time-constrained agents, still shows a cost per life of around $14 million. Moreover, this figure neither factors in the significant costs of preventing time-constrained agents from going to work nor the transfers needed to be made to this population in order to make such a lockdown viable.

In summary, our results show that both viral and antibody testing are necessary to reduce the spread of the disease and the economic cost inflicted by the epidemic. Moreover, we show that the higher the share of the infected population that is asymptomatic and the higher the share of the population who cannot optimally adjust their time allocation, the more important testing becomes. In contrast, lockdowns delay the progression of the virus, but, without further measures, are not an effective way of controlling the human and economic impacts of the epidemic, even in the case of an emerging economy.

Our results are certainly relevant from a policy perspective. First, they highlight the importance of clearly identifying the likelihood that asymptomatic and pre-symptomatic infected agents will spread the virus. While contagion by pre-symptomatic agents has been well-established (He and others (2020)), there are still some questions about the likelihood of asymptomatic agents transmitting the disease (Bai and others (2020)). Second, they show that viral testing, while a very important tool to control the spread of the disease to all countries, is particularly important for developing economies and poor neighborhoods. Unfortunately, as we can see in Figure 2, low-income economies have lagged significantly behind advanced economies in testing their populations.

The paper is divided into 6 sections. Section 2 discusses the literature on COVID-19. Section 3 presents our theoretical model. Section 4 describes the functional forms used in our quantitative results and the calibration of the parameters. Section 5 shows our main quantitative results, while Section 6 concludes the paper.



(a) Texas **(b) Washington**

Figure 1. Change in the Number of Dine-in Customers: Open Table.com

Table 1. COVID-19 Cases in NYC
Differences by Neighborhood Poverty Level

	Cases	Hospitalizations	Deaths
Low poverty	1,806.52	351.38	110.14
Medium poverty	2,189.35	540.92	176.14
High poverty	2,334.32	684.04	221.06
Very High poverty	2,709.08	771.87	525.17

Note: Rate per 100,000 people (age-adjusted)
Source: https://www1.nyc.gov/site/doh/covid/covid-19-data.page

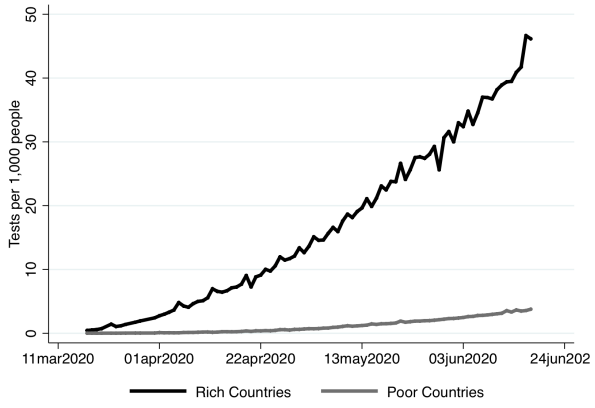


Figure 2. Viral Testing: Rich vs. low-income economies

II. Related Literature

The literature incorporating epidemiology dynamics into economic models is a fast-growing field amid developments from the COVID-19 epidemic. We will focus on summarizing the branch of this literature that is related to the direct contributions of this paper. Few papers discuss the role of testing as it pertains to the epidemic's dynamics and economic outcomes. **?** is one of the first papers to incorporate optimizing behavior and search and matching modeling techniques in an SIR model. They discuss the shape of matching functions in epidemics and make the case for a matching technology with increasing returns to scale. Our choice of the matching function is inspired by the search and matching literature and some of our results follow the intuition of **?**. However, our paper introduces agents' heterogeneity with time-constrained and asymptomatic individuals. Consequently, we are able to discuss the role of testing in an environment with asymptomatic infected agents as well as agents whose economic activities are either essential or time-constrained by economic need.

In the context of known health outcomes and a social planner's optimal policy, Piguillem and Shi (2020), Alvarez, Argente, and Lippi (2020) and Holtemöller (2020) study optimal lockdown, testing, and quarantine policies in a variant of the SIR model. These papers differ from ours because we study a competitive equilibrium with heterogeneous agents. We also allow for an interaction between peoples economic decisions, testing, and the dynamics of the epidemic.

A few recent papers consider models in which health status is not fully known by economic agents when making decisions, which is also a key point in our model. Piguillem and Shi (2020) solve an optimal control problem to find the best testing policy. In their paper, only the central planner's problem is studied and behavioral reactions to the epidemic are absent. Farboodi, Jarosch, and Shimer (2020) analyze a case where health status is never known; that is, they do not allow for the possibility of testing. Agents choose their level of social activity, affecting the contagion rate. Differently from our paper, they do not consider the effects of the epidemic on production decisions. Eichenbaum, Rebelo, and Trabandt (2020b) incorporate economic choices into a time-varying SIR model. In their model, as in ours, people do not know their health status and testing allows a better trade-off between declines in economic activity and health outcomes. In their paper, however, asymptomatic people are not discussed, and the matching function is similar to a standard SIR model. The role of testing is limited to informing agents about their health status, and only forced quarantine can lower transmission. In our paper, however, testing affects economic outcomes through two sources. First, it affects the virus' dynamics by preventing infected persons from contacting susceptible individuals.

Second, the antibody test does not significantly affect the virus' dynamics but does boost the economy by providing information to people who recovered without developing symptoms. Finally, Cherif and Hasanov (2020) also made the case for universal testing and isolation being the most viable way to contain the pandemic. Differently from our paper, they abstract of behavioral responses to the pandemic and do not access the role of heterogeneity.

Similar to our paper, Brotherhood and others (2020) also investigate the role of testing in an amplified SIR epidemiological model with heterogeneous agents and behavioral choices. In their paper, there are only symptomatic people and testing shortens the time of uncertainty. They analyze the efficacy of testing, quarantine, and lockdowns where agents are partially altruistic. Unlike in our paper, their heterogeneity is based on age. Younger people, who are less likely to die, impose externalities on the old. In our case, those externalities come from the time-constrained consumers. They also do not analyze the case of asymptomatic people and the positive impact of that information on health status has on the economy. In terms of agents' heterogeneity, Glover and others (2020) have different age groups in their model and they solve for the optimal containment policy in this scenario. Similarly, Acemoglu and others (2020) also feature age heterogeneity in a multi-group SIR model where optimal containment measures are calculated in a control problem. Kaplan, Moll, and Violante (2020) introduce agents heterogeneity with respect to their occupation. They document that people with lower labor income work in vulnerable occupations. Krueger, Uhlig, and Xie (2020) extend the framework in Eichenbaum, Rebelo, and Trabandt (2020a) to consider sectoral heterogeneity when assessing the epidemic's dynamics. They consider how the substitution of consumption across sectors can help to lower the spread of the virus even without government restrictions on economic activity. Different from our work, they employ a SIR matching function, focus on sectoral effects, and do not discuss the role of asymptomatic agents and testing. Favero, Ichino, and Rustichini (2020) allow for age and sector heterogeneity and analyze the effect of stylized policies to end the lockdown in Italy. They do not consider the feedback loop of individual behavior and transmission, nor do they consider the uncertainty about infection status or testing.

Finally, in contrast to all of the papers cited above, we consider heterogeneous agents based on time allocation constraints.

III. MODEL

A. Environment

Time is discrete. There is a measure n of agents who discount the future at rate $\beta > 0$. We consider two types of agents, distinguished by their ability to optimally adjust their time allocation. Unconstrained agents can optimally allocate their weekly time endowment $\overline{L}$ across work (L^w), leisure (L^l), and time at home (L^h), while time-constrained agents[3] have very limited control over their time allocation. In particular, we assume that they either allocate their time as if there were no pandemic or spend all their time at home if they are sick or in quarantine. We assume that the fraction of time-constrained agents is given by ϑ. Both types of agents can be in the following states: healthy but susceptible (h), infected (i), sick (s), recovered(r), or dead (d). However, transitions across states are not necessarily observed by agents. In particular, infection and recovery, as we describe in detail below.

Healthy but susceptible agents become infected if they are exposed to the virus. They are exposed if they meet infected agents while working or participating in leisure activities. In particular, we assume that the arrival rate of infection $\lambda_t(L_t^w, L_t^l)$ in time t is:

$$\lambda_t(L_t^w, L_t^l) = \frac{m^w(TH_t^w, TI_t^w)}{TH_t^w} L_t^w + \frac{m^l(TH_t^w, TI^w)}{TH_t^l} L_t^l. \tag{1}$$

TH_t^w and TH_t^l are the aggregate times in period t where agents healthy but susceptible are at work and in leisure activities respectively. TI_t^w and TI_t^l are the aggregated times where infected agents are at work and in leisure activities in period t, respectively. Note that these aggregated numbers are the weighted sums of the total hours of time-constrained and unconstrained agents. Moreover, $m^w(TH_t^w, TI_t^w)$ and $m^l(TH_t^l, TI_t^l)$ are the infection matching functions for labor markets and leisure activities, respectively. They present the total number of infectious meetings between healthy but susceptible agents and infected agents. Consequently, we have that $\lambda_t(L_t^w, L_t^l)$ is strictly increasing in both arguments. This is the channel that links time-constrained and unconstrained agents. That is, these two groups can meet at work or at leisure time. The more time-constrained agents there are, the riskier it is for an unconstrained agent to spend another hour outside.

[3]The motivation for these agents comes from the fact that some agents work in services deemed essential which have continued to be supplied during the pandemic. Another fact is that some individuals are in occupations that do not allow for remote working and have very little savings and wealth to rely on. These agents are similar to hand-to-mouth agents, except that we assume that they can be quarantined and not work for some periods.

Infected agents can either become sick or recover without ever developing symptoms. Infected agents who eventually recover without developing symptoms are classified as asymptomatic. Infected agents who eventually develop symptoms are classified as symptomatic, even though they may be pre-symptomatic at the moment. In order to simplify presentation – once infection is no longer observed – we assume that at the time of infection the agent may have received an unobserved shock that made him asymptomatic. The arrival rate of the asymptomatic shock is $\gamma > 0$. We assume that until infected agents are either tested or develop symptoms, they do not know that they have been infected (although they may have a belief about it based on their time of exposure). Similarly, agents shown by test to be infected will not know if they are symptomatic until they either develop symptoms or recover. We assume that tested agents are followed by health professionals, so they observe their transition to recovered, even if they are asymptomatic.

Infected agents who are not asymptomatic become sick with probability $\overline{\mu} > 0$. Sick agents face a flow disutility of $c < 0$ and die at rate δ_t, which depends on the hospital capacity utilization. Death is seen as a one-time negative shock equal to $c_d < 0$. Agents leave the environment once they are dead. We assume that $\delta_t = \delta(\frac{S_t}{ICU_t})$ with $\delta'(\frac{S_t}{ICU_t}) \geq 0$, i.e., we model δ_t as a time-varying Poisson arrival rate that is weakly increasing in the ratio of sick agents to ICU units, indicating that a busy hospital sector is less effective in keeping agents alive. At this point, it is not clear that prior COVID-19 infection confers immunity. For simplification, we assume that is the case and once the agent recovers, she develops immunity and cannot be infected again.

Asymptomatic infected agents initially do not know that they have been infected. Infected agents are tested with probability $\tau_t^I > 0$, which depends on the country's testing capabilities. We allow testing capabilities to vary over time. Asymptomatic infected agents recover at rate $\tilde{\theta}^A > 0$. Asymptomatic agents who recover without getting tested do not know that they have acquired immunity. Consequently, they realize that they have developed immunity only if they are tested for antibodies, which happens with probability $\tau_t^R > 0$. Similarly to the virus test, the antibody test arrival rate depends on the country's testing capabilities, which may vary over time. A graphical representation of the model is presented in Figure 3.

Notice that virus and antibody tests are important because they affect the time allocation decisions of agents. In particular, we assume that infected agents who receive positive viral tests are forced to stay at home, reducing the infection rate. On the other hand, agents who receive a positive antibody test learn that they have developed immunity. Consequently, unconstrained agents optimally adjust their time allocation, boosting their labor supply.

B. Unconstrained agent's problem

We start by considering agents who can optimally adjust their time allocation in order to maximize their utility while taking into account their infection risk. We assume that both infected agents who tested positive in a viral test and sick agents are forced to stay home. Consequently, we focus on the unconstrained agents' decisions in the other states.

1. Known recovered unconstrained agent's problem

We start by evaluating the optimal time allocation of recovered agents who know they have developed immunity. Notice that this knowledge can be acquired in different ways: a.) agents can recover from sickness; b.) agents can recover after testing positive in a viral test while asymptomatic or; c.) agents can test positive in an antibody test. Regardless of the way in which the knowledge about immunity is acquired, the agent is now free to optimally adjust her time allocation without fear of infection. Since the only time-varying features of the model are related to the pandemic's progress, a known recovered agent's value function is stationary.

An agent's flow utility is given by $U(c,L^l,L^h) = u(c) + h(L^l) + \mathtt{u_h} \times L^h$, where c is the amount consumed of the numeraire good, L^l is the time spent in leisure activities, and L^h is the time spent at home. We assume that $u(\cdot)$ and $h(\cdot)$ are both continuous, twice differentiable strictly increasing concave functions. $\mathtt{u_h} > 0$ is a positive parameter that indicates the flow benefit of staying home. It can also be seen as a parameter for home production.

Agents have no savings and no wealth endowments. Labor markets are perfectly competitive. In particular, we follow Eichenbaum, Rebelo, and Trabandt (2020b), assuming a continuum of competitive representative firms of unit measure that produce consumption goods using a linear production function with hours worked as the sole input. As a result, $c = wL^w$, where w is the equilibrium wage rate and L^w the amount of time allocated to work. Similarly, we assume that agents have a total time endowment of $\overline{L}$ hours per period. Consequently, we must have that $L^w + L^l + L^h = \overline{L}$ and $L^i \geq 0$ for all i in $\{l,w,h\}$. Consequently, a known recovered agent's value function is given by:

$$R^K = \frac{u(w \times L^{w_K}) + h(L^{l_K}) + \mathtt{u_h} \times (\overline{L} - L^{w_K} - L^{l_K})}{1-\beta} \tag{2}$$

From the optimal allocation of time, we have the F.O.C.s:

$$\begin{aligned}(L^{w_K}): &\quad wu'(w\times L^{w_K})-\mathtt{u_h}=0\\ (L^{l_K}): &\quad h'(L^{l_K})-\mathtt{u_h}=0\end{aligned}$$

Since $u(\cdot)$ and $h(\cdot)$ are invertible functions, assuming the parameter satisfy an interior solution, we have:

$$L^{w_K}=\frac{1}{w}u'^{-1}\left(\frac{\mathtt{u_h}}{w}\right)\quad L^{l_K}=h'^{-1}(\mathtt{u_h})\quad L^{h_K}=\overline{L}-\frac{1}{w}u'^{-1}\left(\frac{\mathtt{u_h}}{w}\right)-h'^{-1}(\mathtt{u_h}) \tag{3}$$

where we use the subscript K for a known recovered agent.

2. Potentially susceptible unconstrained agent's problem

We now focus on the problem of an agent who is potentially susceptible. In particular, once infection is not observable, an agent cannot distinguish whether she is healthy but susceptible, infected with no positive viral test, or recovered with unknown immunity. Consequently, in all of these states, an unconstrained agent's time allocation must be the same. In order to properly set up the agent's time allocation problem, let us first present the agent's Bellman equations for each state. The Bellman equation for a healthy but susceptible agent at time t is:

$$H_t(L_t^w,L_t^l,L_t^h)=u(wL_t^w)+h(L_t^l)+\mathtt{u_h}\times L_t^h+\beta\left\{\begin{array}{c}\lambda_t(L_t^s,L_t^l)\left[\begin{array}{c}\gamma I_{t+1}^{A_U}(L_{t+1}^w,L_{t+1}^l,L_{t+1}^h)\\ +(1-\gamma)I_{t+1}^{S_U}(L_{t+1}^w,L_{t+1}^l,L_{t+1}^h)\end{array}\right]\\ +[1-\lambda_t(L_t^s,L_t^l)]H_{t+1}(L_{t+1}^w,L_{t+1}^l,L_{t+1}^h)\end{array}\right\} \tag{4}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, $\lambda_t(L_t^s,L_t^l)$ is the arrival rate of infection, γ is the probability of an asymptomatic infection, and $I_{t+1}^{A_U}$ and $I_{t+1}^{S_U}$ are the Bellman equations for the infected asymptomatic and eventually symptomatic agents, respectively. Since at this point the agent has neither received a viral test nor developed symptoms, the infection is not yet known, which is represented by the subscript U. Notice that all of the Bellman equations in equation (4) are non-stationary because transition rates change as the pandemic evolves.

We make a simplifying assumption that susceptible agents are not being tested for viral genetic material or antibodies. Without this assumption, testing and the belief system could interact in ways that would complicate the problem significantly. Moreover, studies point

toward a significant rate of false negative results for both antibody and viral material tests.[4] Consequently, for simplicity we assume that the arrival rate of test results is zero for healthy but susceptible agents.

The Bellman equation for an infected asymptomatic agent without a viral test at time t is:

$$I_t^{A_U}(L_t^w, L_t^l, L_t^h) = u(wL_t^w) + h(L_t^l) + \mathtt{u_h} \times L_t^h + \beta \left\{ \begin{array}{c} \tau_t^I I_{t+1}^{A_K} + \tilde{\theta}^{A_U} R_{t+1}^U(L_{t+1}^w, L_{t+1}^l, L_{t+1}^h) \\ +[1 - \tau_t^I - \tilde{\theta}^{A_U}] I_{t+1}^{A_U}(L_{t+1}^w, L_{t+1}^l, L_{t+1}^h) \end{array} \right\} \tag{5}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, τ_t^I is the arrival rate of viral tests, $I_{t+1}^{A_K}$ the Bellman equation for an asymptomatic infected agent with a positive viral test, $\tilde{\theta}^{A_U}$ is the arrival rate of recovery, and R_{t+1}^U is the Bellman function for a recovered agent with unknown immunity. Notice that $I_{t+1}^{A_K}$ is not a function of the time allocation, since agents with a positive viral test are required to stay home. So the Bellman equation for an asymptomatic infected agent with a positive viral test at time t is given by:

$$I_t^{A_K} = \mathtt{u_h} \times \overline{L} + \beta \left\{ \tilde{\theta}^{A_K} R^K(L^{w_K}, L^{l_K}, L^{h_K}) + (1 - \tilde{\theta}^{A_K}) I_{t+1}^{A_K} \right\} \tag{6}$$

where R^K is the Bellman equation for a recovered agent with known immunity presented in equation (2) and $\tilde{\theta}^{A_K}$ is the arrival rate of recovery. Notice that this Bellman equation is stationary. Therefore $I_t^{A_K} = I^{A_K}$ for every t.

The Bellman equation for an eventually symptomatic agent without a viral test at time t is:

$$I_t^{S_U}(L_t^w, L_t^l, L_t^h) = u(wL_t^w) + h(L_t^l) + \mathtt{u_h} \times L_t^h + \beta \left\{ \begin{array}{c} \tau_t^I I_{t+1}^{S_K} + \overline{\mu}_U S_{t+1} \\ +[1 - \tau_t^I - \overline{\mu}] I_{t+1}^{S_U}(L_{t+1}^w, L_{t+1}^l, L_{t+1}^h) \end{array} \right\} \tag{7}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, τ_t^I is the arrival rate of viral tests, $I_{t+1}^{S_K}$ the Bellman equation for an eventually symptomatic infected agent with a positive viral test, $\overline{\mu}_U$ is the arrival rate of symptoms, and S_{t+1} is the Bellman function. Similarly to $I_{t+1}^{A_K}$, $I_{t+1}^{S_K}$ and S_{t+1} are not functions of time allocation, since agents with either a positive viral test or symptoms are required to stay at home. Let's present these Bellman equations next. The Bellman equation for an eventually symptomatic agent with a positive viral test at time t is:

$$I_t^{S_K} = \mathtt{u_h} \times \overline{L} + \beta \left\{ \overline{\mu}_K S_{t+1} + (1 - \overline{\mu}_K) I_{t+1}^{S_K} \right\} \tag{8}$$

[4] https://www.npr.org/sections/health-shots/2020/04/21/838794281/study-raises-questions-about-false-negatives-from-quick-covid-19-test and https://www.scientificamerican.com/article/what-covid-19-antibody-tests-can-and-cannot-tell-us/

where $\overline{\mu}_K$ is the arrival rate of symptoms. Differently from $I_t^{A_K}$, $I_t^{S_K}$ is non-stationary, since as we see below, S_{t+1} depends on time. In particular, one's outcome as a sick patient depends on how overcrowded the health system is.

The Bellman equation for a sick agent in period t is given by:

$$S_t = \mathtt{u_h} \times \overline{L} - c + \beta \left\{ \delta_t(-c_D) + \theta_t^S R^K(L^{w_K}, L^{l_K}, L^{h_K}) + \left[1 - \delta_t - \theta_t^S\right] S_{t+1} \right\} \tag{9}$$

where c is the flow cost of sickness, δ_t is the death arrival rate, c_D is the one-time cost of death before exiting the environment, θ_t^S is the arrival rate of recovery, and R^K is the Bellman equation for a recovered agent with known immunity presented in equation (2). Because δ_t and θ_t^S vary over time due to over-utilization of health services, S_t is non-stationary.

Finally, let's consider the Bellman function for recovered agents with unknown immunity. In this case, we have:

$$R_t^U(L_t^w, L_t^l, L_t^h) = u(wL_t^w) + h(L_t^l) + \mathtt{u_h} \times L_t^h + \beta \left\{ \begin{array}{c} \tau_t^R R^K(L^{w_K}, L^{l_K}, L^{h_K}) \\ + \left[1 - \tau_t^R\right] R_{t+1}^U(L_{t+1}^w, L_{t+1}^l, L_{t+1}^h) \end{array} \right\} \tag{10}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, τ_t^R is the arrival rate of antibody tests, and R^K is the Bellman equation for a recovered agent with known immunity presented in equation (2).

Finally, we need to consider the optimal time allocation for potentially susceptible unconstrained agents. Notice that agents in the states healthy but susceptible, infected unknown (both asymptomatic and eventually symptomatic), and recovered unknown do not know in which state they actually are. Consequently, we have that agents in the states H_t, $I_t^{A_U}$, $I_t^{S_U}$ and R_t^U solve the same time allocation problem:

$$\max_{L_t^w, L_t^l, L_t^h} \pi_t^H H_t + \pi_t^{I^A} I_t^{A_U} + \pi_t^{I^S} I_t^{S_U} + \pi_t^R R_t^U \tag{11}$$

subject to:

$$L_t^w + L_t^l + L_t^h = \overline{L} \tag{12}$$

where π_t^H, $\pi_t^{I^A}$, $\pi_t^{I^S}$, and π_t^R are the beliefs that a given agent is in one of these states. We assume that, based on rational expectations and the focus on homogeneous solutions, these beliefs are based on an aggregate measure of workers in each one of these states. Consequently, agents' time allocation decisions take beliefs as given. Based on the solution of the potentially susceptible agents presented in equations (11) and (12), we pin down optimal time allocation

$L_t^{w^*}$, $L_t^{l^*}$, and $L_t^{h^*}$. In order to simplify notation, define $\lambda_t = \lambda_t(L_t^{w^*}, L_t^{l^*})$, i.e., λ_t is the rate of infection, taking into account a possibly susceptible agent's optimal time allocation.

Finally, we describe the laws of motion for unconstrained agents across the different states. Define h_t as the measure of unconstrained agents who are healthy but susceptible in period t. Then, the change in the measure of healthy and susceptible agents between periods t and $t+1$ is given by:

$$h_{t+1} - h_t = -\lambda_t h_t \tag{LM_1}$$

Notice that, in this case, since all recovered agents obtain immunity, the share of healthy but susceptible agents only declines at infection rate λ_t. Similarly, define $i_t^{A_U}$ as the measure of unconstrained agents who are asymptomatic infected without a positive viral test. This measure varies between periods t and $t+1$ according to the following law of motion:

$$i_{t+1}^{A_U} - i_t^{A_U} = \lambda_t \gamma h_t - \left(\tilde{\theta}^{A_U} + \tau_t^I\right) i_t^{A_U} \tag{LM_2}$$

where λ_t is the infection rate, γ is the share of infections that will stay asymptomatic, $\tilde{\theta}^{A_U}$ is the rate at which untested asymptomatic agents recover, and τ_t^I is the viral testing rate in period t. Define $i_t^{A_K}$ as the measure of unconstrained agents who are asymptomatic infected with a positive viral test. This measure varies between periods t and $t+1$ according to the following law of motion:

$$i_{t+1}^{A_K} - i_t^{A_K} = \tau_t^I i_t^{A_U} - \tilde{\theta}^{A_U} i_t^{A_U} \tag{LM_3}$$

where τ_t^I is the viral testing rate in period t and $\tilde{\theta}^{A_K}$ is the rate at which asymptomatic tested agents recover. Moreover, define $i_t^{S_U}$ as the measure of eventually symptomatic infected unconstrained agents, that is the pre-symptomatic, without a positive viral test. This measure varies between periods t and $t+1$ according to the law of motion:

$$i_{t+1}^{S_U} - i_t^{S_U} = \lambda_t(1-\gamma)h_t - \left(\overline{\mu}_U + \tau_t^I\right) i_t^{S_U} \tag{LM_4}$$

where λ_t is the infection rate, $1-\gamma$ is the share of infections that will eventually become symptomatic, τ_t^I is the viral testing rate in period t, and $\overline{\mu}_U$ is the rate at which untested eventually symptomatic agents become sick. Similarly, define $i_t^{S_K}$ as the measure of infected pre-symptomatic unconstrained agents with a positive viral test. This measure varies between periods t and $t+1$ according to the following law of motion:

$$i_{t+1}^{S_K} - i_t^{S_K} = \tau_t^I i_t^{S_U} - \overline{\mu}_K i_t^{S_K} \tag{LM_5}$$

where $\overline{\mu}_K$ is the rate at which tested eventually symptomatic agents become sick. Then, define s_t as the measure of sick unconstrained agents in period t. This measure varies between

periods t and $t+1$ according to the following law of motion:

$$s_{t+1} - s_t = \overline{\mu}_U i_t^{S_U} + \overline{\mu}_K i_t^{S_K} - \left(\delta_t + \theta_t^S\right) s_t \qquad (LM_6)$$

where $\overline{\mu}_K$ and $\overline{\mu}_U$ are the rates at which tested and untested infected eventually symptomatic unconstrained agents become sick, δ_t is the rate at which sick agents die, and θ_t^S is the rate at which sick patients recover.

Define r_t^U as the measure of recovered unconstrained agents with unknown immunity in period t. This measure varies between periods t and $t+1$ according to the following law of motion:

$$r_{t+1}^U - r_t^U = \tilde{\theta}^{A_U} i_t^{A_U} - \tau_t^R r_t^U \qquad (LM_7)$$

where $\tilde{\theta}^{A_U}$ is the rate at which asymptomatic untested agents recover and τ_t^R is the antibody testing rate in period t.We now consider the evolution of the measures of agents in the two absorbing states. Define d_t as the measure of dead unconstrained agents in period t. This measure evolves between periods t and $t+1$ according to the law of motion:

$$d_{t+1} - d_t = \delta_t s_t \qquad (LM_8)$$

where δ_t is the rate at which sick agents die and s_t is the measure of sick unconstrained agents in period t. Finally, define r_t^K as the measure of unconstrained recovered agents with known immunity. This measure evolves between periods t and $t+1$ according to the law of motion:

$$r_{t+1}^K - r_t^K = \theta_t^S s_t + \tau_t^R r_t^U \qquad (LM_9)$$

where θ_t^S is the rate at which sick patients recover, s_t is the measure of sick agents in period t, τ_t^R is the antibody testing rate in period t, and r_t^U is the measure of recovered unconstrained agents with unknown immunity in period t.

C. Time-constrained agents

We now consider the case of agents who cannot optimally adjust their time allocation based on the risk of infection. We assume that these agents cannot avoid participating in economic activities either because they perform essential services or because of financial constraints. We call these agents time-constrained and identify them by a subscript c. In order to simplify our calculations, we assume that, whenever these agents are not quarantined due to known infection or sickness, their time allocation follows the optimal choices presented by agents

who knowingly recovered from COVID-19, as presented in Section III.B.1. Consequently, from equation (3), we have that:

$$L^w_{C,t} = \frac{1}{w}u'^{-1}\left(\frac{\mathtt{u_h}}{w}\right) \quad L^l_{C,t} = h'^{-1}(\mathtt{u_h}) \quad L^h_{C,t} = \overline{L} - \frac{1}{w}u'^{-1}\left(\frac{\mathtt{u_h}}{w}\right) - h'^{-1}(\mathtt{u_h}) \tag{13}$$

Notice that, differently from the case of known recovered agents, we keep these measures of time allocation time dependent. The reason for that is that susceptible and infected time-constrained agents can still become sick or test positive in a viral test, thereby being forced to quarantine. In these cases, $L^h_{C,t} = \overline{L}$.

We define λ^C_t as the time-constrained agents' infection rate in period t, its functional form is similar to that of the unconstrained agents, but it is either zero, when the time-constrained agents are sick or quarantined at home, or:

$$\lambda^C_t(TH^w_t, TI^w_t) = \frac{m^w(TH^w_t, TI^w_t)}{TH^w_t}L^w_{C,t} + \frac{m^l(TH^w_t, TI^w)}{TH^l_t}L^l_{C,t} \tag{14}$$

where TH^w_t, TI^w_t, TH^l_t, and TI^l_t are the aggregated total amount of time healthy but susceptible and infected agents spend working and in leisure activities outside in period t, respectively. Notice that $\lambda^C_t \geq \lambda_t$, since time-constrained agents' time allocation does not take into account the risk of infection.

Since time-constrained agents make no decisions, we are now able to present their Bellman equations. The Bellman equation for healthy but susceptible time-constrained agents is:

$$H_{C,t} = u(w \times L^w_{C,t}) + h(L^l_{C,t}) + \mathtt{u_h} \times L^h_{C,t} + \beta \left\{ \begin{array}{c} \lambda^C_t \left[\gamma I^{A_U}_{C,t+1} + (1-\gamma) I^{S_U}_{C,t+1}\right] \\ + \left(1 - \lambda^C_t\right) H_{C,t+1} \end{array} \right\} \tag{15}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, λ^C_t is the arrival rate of infection, γ is the probability of an asymptomatic infection, and $I^{A_U}_{C,t+1}$ and $I^{S_U}_{C,t+1}$ are the Bellman equations for the untested infected asymptomatic and eventually symptomatic time-constrained agents, respectively.

The Bellman equation for an infected asymptomatic time-constrained agent without a viral test at time t is:

$$I^{A_U}_{C,t} = u(w \times L^w_{C,t}) + h(L^l_{C,t}) + \mathtt{u_h} \times L^h_{C,t} + \beta \left\{ \tau^I_t I^{A_K}_{C,t+1} + \tilde{\theta}^{A_U} R^U_{C,t+1} + [1 - \tau^I_t - \tilde{\theta}^{A_U}] I^{A_U}_C \right\} \tag{16}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, τ_t^I is the arrival rate of viral tests, $I_{C,t+1}^{A_K}$ is the Bellman equation for an asymptomatic infected time-constrained agent with a positive viral test, $\tilde{\theta}^{A_U}$ is the arrival rate of recovery, and R_C^U is the Bellman function for a recovered time-constrained agent with unknown immunity. Notice that R_C^U is not time dependent, since recovered agents will never become infected again and time-constrained agents do not adjust their time allocation based on the knowledge of immunity. Consequently, $R_C^U = R_C^K = R^K$. Similarly, notice that $I_C^{A_K}$ is not a function of the time allocation as well, since agents with a positive viral test are required to stay home. So the Bellman equation for an asymptomatic infected time-constrained agent with a positive viral test at time t is given by:

$$I_C^{A_K} = u(b) + \mathtt{u_h} \times \overline{L} + \beta \left\{ \tilde{\theta}^{A_K} R^K + (1 - \tilde{\theta}^{A_K}) I_C^{A_K} \right\} \tag{17}$$

where R^K is the Bellman equation for a recovered agent with known immunity presented in equation (2) and $\tilde{\theta}^{A_K}$ is the arrival rate of recovery. Notice that this Bellman equation is stationary.

The Bellman equation for an eventually symptomatic time-constrained agent without a viral test at time t is:

$$I_{C,t}^{S_U} = u(w \times L_{C,t}^w) + h(L_{C,t}^l) + \mathtt{u_h} \times L_{C,t}^h + \beta \left\{ \tau_t^I I_{C,t+1}^{S_K} + \overline{\mu}_U S_{C,t+1} + [1 - \tau_t^I - \overline{\mu}_U] I_{C,t+1}^{S_U} \right\} \tag{18}$$

where the flow utility is the same as the one described in Section III.B.1, β is the discount rate, τ_t^I is the arrival rate of viral tests, $I_{C,t+1}^{S_K}$ is the Bellman equation for an eventually symptomatic infected time-constrained agent with a positive viral test, $\overline{\mu}_U$ is the arrival rate of symptoms, and $S_{C,t+1}$ is the Bellman function of a sick time-constrained agent. Similarly to $I_C^{A_K}$, $I_{C,t+1}^{S_K}$ and $S_{C,t+1}$ are not functions of the time allocation, since agents with either a positive viral test or symptoms are required to stay at home. Let us present these Bellman equations next. The Bellman equation for an eventually symptomatic time-constrained agent with a positive viral test at time t is:

$$I_{C,t}^{S_K} = u(b) + \mathtt{u_h} \times \overline{L} + \beta \left\{ \overline{\mu}_K S_{C,t+1} + (1 - \overline{\mu}_K) I_{C,t+1}^{S_K} \right\} \tag{19}$$

where $\overline{\mu}_K$ is the arrival rate of symptoms. Differently from $I_C^{A_K}$, $I_t^{S_K}$ is non-stationary, since as we see below, $S_{C,t+1}$ depends on time. In particular, one's outcome as a sick patient depends on how overcrowded the health system is.

The Bellman equation for a sick agent in period t is given by:

$$S_{C,t} = u(b) + \mathtt{u_h} \times \overline{L} - c_S + \beta \left\{ \delta_t(-c_D) + \theta_t^S R_{C,t}^K + [1 - \delta_t - \theta_t^S] S_{C,t+1} \right\} \tag{20}$$

where c is the flow cost of sickness, δ_t is the death arrival rate, c_D is the one-time cost of death before exiting the environment, θ_t^S is the arrival rate of recovery, and R^K is the Bellman equation for a recovered agent with known immunity presented in equation (2). Because δ_t and θ_t^S vary over time due to over-utilization of health services, S_t is non-stationary.

As mentioned before, once time-constrained agents do not adjust their hours based on the risk of infection, we have that $R_{C,t}^U = R_{C,t}^K = R_C^K = R^K$, where R^K is presented in equation (2), i.e.:

$$R_{C,t}^U = R_{C,t}^K = R_C^K = R^K = \frac{u(w \times L_{C,t}^w) + h(L_{C,t}^l) + \mathtt{u_h} \times L_{C,t}^h}{1 - \beta} \tag{21}$$

Finally, the laws of motion for time-constrained agents follow a pattern very similar to that of the unconstrained agents, but replacing the infection rate λ_t with λ_t^C. Consequently, equations (LM_1) – (LM_9) can be rewritten for the case of time-constrained agents as:

$$\begin{aligned}
&h_{C,t+1} - h_{C,t} = -\lambda_t^C h_{C,t} && (LM_1^C)\\
&i_{C,t+1}^{A_U} - i_{C,t}^{A_U} = \lambda_t^C \gamma h_{C,t} - \left(\tilde{\theta}^{A_U} + \tau_t^I\right) i_{C,t}^{A_U} && (LM_2^C)\\
&i_{C,t+1}^{A_K} - i_{C,t}^{A_K} = \tau_t^I i_{C,t}^{A_U} - \tilde{\theta}^{A_U} i_{C,t}^{A_U} && (LM_3^C)\\
&i_{C,t+1}^{S_U} - i_{C,t}^{S_U} = \lambda_t^C (1-\gamma) h_{C,t} - \left(\overline{\mu}_U + \tau_t^I\right) i_{C,t}^{S_U} && (LM_4^C)\\
&i_{C,t+1}^{S_K} - i_{C,t}^{S_K} = \tau_t^I i_{C,t}^{S_U} - \overline{\mu}_K i_{C,t}^{S_K} && (LM_5^C)\\
&s_{C,t+1} - s_{C,t} = \overline{\mu}_U i_{C,t}^{S_U} + \overline{\mu}_K i_{C,t}^{S_K} - \left(\delta_t + \theta_t^S\right) s_{C,t} && (LM_6^C)\\
&r_{C,t+1}^U - r_{C,t}^U = \tilde{\theta}^{A_U} i_{C,t}^{A_U} - \tau_t^R r_{C,t}^U && (LM_7^C)\\
&d_{C,t+1} - d_{C,t} = \delta_t s_{C,t} && (LM_8^C)\\
&r_{C,t+1}^K - r_{C,t}^K = \theta_t^S s_{C,t} + \tau_t^R r_{C,t}^U && (LM_9^C)
\end{aligned}$$

D. Equilibrium

A rational-expectations equilibrium in this economy with an initial measure of agents n consists of a sequence of infection rates for unconstrained agents $\{\lambda_t\}_{t\geq 0}$ such that at every period:

1. Potentially susceptible unconstrained agents optimally solve their time allocation problem presented in equations (11) and (12);

2. Known recovered unconstrained agents optimally solve their time allocation problem presented in equation (2)

3. Resulting laws of motion (LM_1) – (LM_9) and (LM_1^C) – (LM_9^C) give rise to the sequence of infection rates $\{\lambda_t\}_{t\geq 0}$.

Given rational expectations, the beliefs of time-constrained agents and the objective probabilities of different health states coincide:

$$\frac{h_t}{h_t + i_t^{A_U} + i_t^{S_U} + r_t^U} = \pi_t^H, \tag{22}$$

$$\frac{i_t^{A_U}}{h_t + i_t^{A_U} + i_t^{S_U} + r_t^U} = \pi_t^{I^A}, \tag{23}$$

$$\frac{i_t^{S_U}}{h_t + i_t^{A_U} + i_t^{S_U} + r_t^U} = \pi_t^{I^S}, \tag{24}$$

$$\frac{r_t^U}{h_t + i_t^{A_U} + i_t^{S_U} + r_t^U} = \pi_t^R. \tag{25}$$

We use a time-stacking algorithm using the sparsity of the Jacobian blocks in a gradient-based method to solve for the equilibrium paths of all endogenous variables for $t = 0, ..., 500$.

IV. Quantitative Analysis

A. Taking the model to the data

We now must impose functional forms in order to calibrate the model presented in Section III. In particular, in the calibrated model, we assume the following separable utility function:

$$U(c, L^l, L^h) = \alpha \ln(1+c) + (1-\alpha)\ln(1+L^l) + u_h \times L^h \tag{26}$$

while the restrictions on time allocation are the same as the ones presented in Section III ($c = wL^w$, $L^w + L^l + L^h = \overline{L}$, and $L^i \geq 0$ for all i in $\{l, w, h\}$).

Moreover, borrowing from the labor economics literature (see Petrongolo and Pissarides (2001)), we consider Cobb-Douglas matching functions. Therefore, the number of infections due to workplace interactions is given by:

$$m^w(TH_t^w, TI_t^{U_w}) = \mathbb{A}_w (TH_t^w)^{\zeta} \left(TI_t^{U_w}\right)^{1-\zeta} \tag{27}$$

where $TH_t^w = h_{C,t}L_{C,t}^w + h_t L_t^w$ is the total number of hours spent at work by healthy but susceptible agents. Notice that this measure depends on the measure of time-constrained and unconstrained susceptible agents – $h_{C,t}$ and h_t, respectively – and the number of hours spent at work by each group ($L_{C,t}^w$ and L_t^w). Similarly, $TI_t^{U_w} = (i_t^{A_U} + i_t^{S_U})L_t^w + (i_{C,t}^{A_U} + i_{C,t}^{S_U})L_{C,t}^w$ is the total number of hours spent at work by infected agents who have neither been tested nor developed symptoms. $\mathbb{A}_w$ and *zeta* are parameters to be calibrated.

Similarly, the number of infections due to leisure activities is given by:

$$m^l(TH_t^l, TI_t^{U_l}) = \mathbb{A}_l \left(TH_t^l\right)^{\chi} \left(TI_t^{U_l}\right)^{1-\chi} \tag{28}$$

where $TH_t^l = h_{C,t}L_{C,t}^l + h_t L_t^l$ is the total number of hours spent at leisure activities by healthy but susceptible agents. Notice that this measure depends on the measure of time-constrained and unconstrained susceptible agents – $h_{C,t}$ and h_t, respectively – and the number of hours at leisure activities by each group ($L_{C,t}^l$ and L_t^l). Similarly, $TI_t^{U_l} = (i_t^{A_U} + i_t^{S_U})L_t^l + (i_{C,t}^{A_U} + i_{C,t}^{S_U})L_{C,t}^l$ is the total number of hours spent at leisure by infected agents who have neither been tested nor developed symptoms. $\mathbb{A}_l$ and χ are parameters to be calibrated. They are allowed to be different for different agents.

Finally, given the constant returns to scale in the matching functions, we can calculate the rate of infection per unit of time exposed at work and in leisure activities respectively as:

$$\frac{m^s(TH_t^{U_s}, TI_t^{U_s})}{TH_t^s} = \mathbb{A}_s \left(\frac{TI_t^{U_s}}{TH_t^s}\right)^{1-\zeta} = \mathbb{A}_s \left(\frac{i_t^U}{h_t}\right)^{1-\zeta}$$

and

$$\frac{m^l(TH_t^{U_l}, TI_t^{U_l})}{TH_t^l} = \mathbb{A}_l \left(\frac{TI_t^{U_l}}{TH_t^l}\right)^{1-\chi} = \mathbb{A}_l \left(\frac{i_t^U}{h_t}\right)^{1-\chi}$$

B. Parameter values

Our choice of parameters broadly follows the recent literature modeling the transmission of the COVID-19 virus. As discussed in other papers, there is large uncertainty on the true value of these parameters, but the analysis showed in the quantitative results section is qualitatively very similar for a range of different parameter choices.

The model is calibrated so that a time period is one week. We assume that it takes an average 14 days to either recover or die from the infection – that is $\theta^S + \delta = 7/14$. The parameter

μ is calculated so that the incubation period is around 10 days. The death rate comes from a version of Fernández-Villaverde and Jones (2020) and matches early serology tests in Heinsberg, Germany and Iceland. Since recent evidence suggests that the mortality rate might be higher, we will report robustness results using death rates of 1 percent. We also consider the case in which death rates may depend on the ICU occupancy rate in Section V.H. The fraction of asymptomatic agents, 50 percent, also comes from Iceland in a study of voluntary screening.[5] The value of death, c_d, was calculated as the present value of the loss of future income discounted by β as chosen in Eichenbaum, Rebelo, and Trabandt (2020b). The number of people who are initially infected, e, is set to 0.001.

The initial population and the wage rate are normalized to one. The fraction of time-constrained agents, 30 percent, is initially calibrated as in Laxton and others (2010),[6] but different values are also used in the simulations to mimic developing economies. We set the parameters in the utility function, u_h and α, so that the steady-state time spent at home, at work and in outside leisure activities matches the data in the American Time Use Survey, compiled by the Census Bureau. Finally, the parameters in the matching function are calibrated so that the number of infected agents approaches zero after two years.

V. Quantitative Results

A. Testing and its effects on the epidemic and the economy

Our model predicts large health and economic benefits from increasing testing. The model simulation results for an economy without testing and with testing are depicted in Figure 4. The solid line is for the economy without testing, the dashed line represents the economy when 10 percent of infected persons and the same percentage of persons who recovered without ever knowing they were infected are tested per week (so we adjust both τ_I and τ_R)[7]. As also reported in Eichenbaum, Rebelo, and Trabandt (2020b), the economic results with and without testing are qualitatively similar. However, with the level of testing set as in our benchmark model calibration, the US would be able to reduce the number of deaths by more than 60,000 compared to a situation where no tests were conducted by the end of the second year

[5] https://nordiclifescience.org/COVID-19-first-results-of-the-voluntary-screening-in-iceland/

[6] Note that Laxton and others (2010) call this the OLG households.

[7] The 10 percent figure was picked based on evidence from serology tests which show that only 1 in 10 cases of COVID-19 is confirmed by testing in many countries

(Figure 4d). Additionally, by the end of the second year, the cumulative gain with this level of testing would be around 3.4 percent of GDP (Figure 4a).

There are two channels through which these proposed tests affect the economy in our model. The first, which we call the stop spreading channel, operates by changing the epidemics' dynamics. Early testing removes infected agents from contact with susceptible people. The more infected people are removed from contact, the less infection occurs (see Figure 4b), reducing the number of sick and dead people as shown in Figures 4c and 4d, respectively.

This channel affects the economy through two mechanisms. First, infected people are required to quarantine after receiving a positive test, even if they are asymptomatic. This has a negative effect on the labor supply. On the other hand, unconstrained agents who are unaware of their health status choose a higher labor supply when tests are available, because there is less risk of getting infected once more infected people are removed from the labor force. The second effect is larger than the first effect, so tests are net positive to GDP. This channel is also found in Eichenbaum, Rebelo, and Trabandt (2020b) and Brotherhood and others (2020).

The second channel, which we call the information channel, operates purely by providing better information to agents. Some agents do not behave optimally due to a lack of information about their health status. For example, agents who recovered without ever knowing they were infected behave as if they were still possibly susceptible when deciding their labor supply. Consequently, their labor supply choices are identical to those of healthy but susceptible agents, as well as untested asymptomatic and pre-symptomatic infected agents. Testing for antibodies in unknowingly recovered agents boosts economic outcomes without affecting the epidemics' dynamics, a result that allows us to separate this channel from the previously studied ones.

We can partially isolate the effects of this information channel to measure its importance. Figure 5 compares the model in the absence of testing with a model in which antibody tests are substantially scaled-up (the τ_R is increased from 0 percent to about 5 percent). This is the policy being floated for the post-lockdown period in many countries. As mentioned, this policy has minimal effects on the virus dynamics (see Figures 5b, 5c, and 5d), but it has a positive impact on GDP through the higher labor supply from the agents who recovered without ever knowing they were infected (Figure 5a). The cumulative output gain from this policy after two years is worth 0.1 percent of GDP. Note, however, that this policy does not fully capture the information channel in our model, as the viral test also reveals the virus to infected people, allowing them to behave optimally after they have recovered.

B. What if the proportion of asymptomatic cases was different?

The fraction of asymptomatic cases remains uncertain, and robustness in this parameter, the γ in the model description, is warranted. We picked our benchmark calibration of the share of asymptomatic people based on mass testing results conducted in Iceland. However, there is large uncertainty about how many asymptomatic cases there are and about the role of the asymptomatic in transmitting the virus. Thus, it is interesting to check how the epidemic's dynamics change as we move between the extremes in incidences of asymptomatic cases, from 80 percent to just 20 percent of all cases. The exercise is conducted so that the expected death from the virus for a susceptible agent is the same. That is, we adjust the mortality rate, the δ in the model, upward (downward) for the case of more (fewer) asymptomatic cases. If we do not adjust for the δ, the mortality rate would be lower for the instance with more asymptomatic cases. We want to focus here on the role of asymptomatic cases given the same expected mortality. In Section V.F, we discuss the role of different mortality levels.[8]

There are multiple channels through which asymptomatic cases can change an epidemic. On the one hand, having more asymptomatic cases means that people will get less sick, which increases the utility of infected agents. Thus, agents would be less afraid of contracting the virus and could spend more time outside. This channel is therefore positive for the economy but it could lead to more deaths. On the other hand, asymptomatic individuals remain in contact with susceptible ones for longer, since symptomatic cases are assumed to stay home while sick, and it takes less time to get sick than it takes to recover from the virus. This further exposure increases the speed and reach of the epidemic. As the virus reaches a higher fraction of the population, unconstrained agents cut their time spent outside, which is bad for the economy but could reduce overall deaths. Finally, more individuals recover without ever knowing they were infected, and behave as if they were still susceptible. This latter effect would increase and prolong the economic costs of the virus.

These multiple channels act in opposite directions, but our results indicate that the virus is more widespread and more people die when the number of asymptomatic cases is larger. Figure 6 depicts these two more extreme calibrations for asymptomatic cases. The peak of infections and the total number of people ever infected are higher when there is a higher fraction of asymptomatic cases (Figure 6b). The overall death count ends up being higher because a lot more people get infected (Figure 6d).

[8]Note that it has been discussed that the mortality rate from COVID-19 could be lower if there are many asymptomatic cases that have not captured by tests. This is a different point from what we propose here. In our case, we set the mortality rate by calibration, which can be chosen independently from the level of asymptomatic cases, which is another calibration choice.

With respect to the economic effects, the fraction of asymptomatic cases is very important to the current discussion about the depth and shape of the recovery (see Figure 6a). The economic effects at the beginning of the epidemic are somewhat similar for both calibrations, but after 10 weeks the recession is substantially deeper when there are more asymptomatic cases and the virus is more widespread. Time unconstrained agents restrict their labor supply by a lot more in response to the swift transmission of the virus. That is, even though there are fewer people getting sick, becoming infected is is so much more likely that these agents choose to stay home for a higher share of their time. This large response of households to riskier environments was also seen in **?**, where the response also peaks concomitantly with the number of infected agents. Note that this result depends on the assumption that the virus is equally infectious for symptomatic and asymptomatic individuals and on the assumption that sick people stay at home. However, after reaching the bottom of economic activity around 40 weeks, the economy starts to rebound and form more of a V shaped recovery when the number of asymptomatic cases is higher. That is, the recession is milder but also more prolonged if the epidemic features fewer asymptomatic cases. The prolonged recession is a byproduct of the slower transmission of the virus per period.

Given the difference in importance of information in the two calibrations, it is interesting to measure how much more valuable testing is for these different situations. Figure 7 replicates the exercise of the previous section, comparing an economy with no testing to one in which 10 percent of infected patients get tested, but with these two different calibrations for the asymptomatic. After two years, the cumulative GDP gain is of 1.8 percentage points when there are just a few asymptomatic cases compared to around 6.3 percentage points when there are more asymptomatic individuals.

C. Effects of the epidemic for different agents

So far, we have analyzed the epidemic using only the aggregated numbers, but the heterogeneity between the two groups of households is striking and helps to explain the overall dynamic.

It is expected that the virus would impact time-constrained agents more than unconstrained ones. Time-constrained agents cannot afford to miss work and therefore are not able to reduce their time spent outside the house to lower their exposure to the virus. This is what is seen in Figure 8, which presents the same set of variables as shown before for the benchmark calibration, but it breaks down the dynamics for the different groups of households.

However, the difference in the incidence of infection by the end of the epidemic is remarkable. While less than 13 percent of the time unconstrained agents ever get infected by the virus, almost 60 percent of the time-constrained agents get infected at some point in the two years (Figure 8a). This is also reflected in the number of deaths, where only 0.03 percent of the unconstrained agents die compared to 0.12 percent of the time-constrained agents (Figure 8b). These numbers suggest that the greatest burden of the epidemic will be borne by the time-constrained agents. The unconstrained agents, knowing that the time-constrained agents will not change their behavior, choose to lower their outside exposure even more and minimize their health losses.

D. An economy with a larger fraction of time-constrained agents

A natural extension is to ask what would happen in an economy where the fraction of time-constrained agents is larger, which can be a better approximation of many emerging and poor economies. Figure 9 shows the same variables but when 50 percent of the agents are time-constrained (in contrast to the 30 percent of the benchmark calibration), a calibration chosen in Laxton and others (2010) for emerging markets.

The effects of the epidemic in this context are dire, as could be expected from the previous discussion. Results are presented in Figure 9. The peak of the epidemic is reached sooner, at 34 weeks, compared to 42 weeks in the benchmark calibration. The epidemic quickly passes: almost all infections take place in the first year. The total infected peak is at 2.9 percent of the total population, compared to less than 1 percent in the benchmark (Figure 9b). Around 85 percent of time-constrained agents get infected at some point and 0.18 percent die (Figures 10a and 10b). Meanwhile, a little over 21 percent of the unconstrained agents get ever infected and less than 0.05 percent of them die.

Even if health systems were as good in emerging markets as they are in advanced economies, poorer economies would be more severely affected by this epidemic according to our model predictions. This result is just a consequence of the existence of a larger share of agents who are less able to take precautions to minimize exposure to the virus.

The economic effects in a poorer economy are also massive. At first, one might think that the effects would be milder as there are more individuals behaving as they would in normal times during the epidemic. However, since the virus is so much more widespread, the risk of staying outside is substantially larger for the unconstrained agents. In turn, these agents reduce

their time spent outside and their labor supply to an even larger degree, which more than offsets the higher labor supply from time-constrained workers. Consequently, GDP effects are significantly larger in economies with a larger share of time-constrained workers (see Figure 9a).

The poorer economy faces a deeper recession and substantially larger cumulative output losses. While GDP bottoms out at about -8.7 percent in the benchmark calibration, it reaches over -19 percent in this alternative calibration. While the economic effects are much more severe, the recovery is also much faster and V-shaped. After one year, the level of GDP per capita in the economy with a larger share of time-constrained workers is higher than in the benchmark calibration, which experiences a shallower but more prolonged recession. Finally, even with the faster recovery, the present value of cumulative losses is about 3.7 percent of GDP for the economy with more time-constrained agents. Not only is the health situation dire, but also the economic losses are larger (Figure 9a).

E. Testing when the fraction of time-constrained agents is larger

Given the larger cost for economies with a higher fraction of time-constrained agents, it is interesting to study how the testing policy affects epidemiologic and economic outcomes compared to in our benchmark model. Figure 11 plots the same variables we have been plotting, but when the fraction of time-constrained agents is 50 percent of the population. We do the same exercise as before and adjust both τ_I and τ_R.

Testing is more effective in changing the speed of health outcomes when there are more time-constrained agents. In terms of infections, they peak about the same time, but testing reduces the peak value by almost 1.5 percentage points, compared to less than 0.5 percentage points in the benchmark model (Figure 11b). When more testing is available, fewer people are infected at the beginning of the epidemic, but at the end of the epidemic, fewer people ever get infected in the benchmark calibration. In terms of deaths, testing saves substantially more lives in the first year of the epidemic, around 30,000, in the higher time-constrained calibration, but by the end of the epidemic, testing saves around 14,000 more lives in the benchmark calibration (Figure 11d). In sum, testing affects the speed, hence the lower death count in the first year, but not the overall final health outcomes when the fraction of time-constrained agents is larger. By the second year, most of the time-constrained workers are already immune in the alternative calibration, which lowers testing effectiveness in influencing the epidemic's dynamics.

By lowering the speed of the epidemic, testing is also very effective in lowering the economic costs in the economy with more time-constrained agents. While the economy bottoms out with a drop in output of about 19 percentage points below the steady state without testing, it falls by 13 percentage points with testing (Figure 11a). The reason for this is similar to what we discussed before. Testing reduces the speed and in turn the number of interactions between infected and susceptible persons. With less risk of infection, unconstrained people are willing to spend more time outside and supply more labor. Output starts to diminish more in the economy without testing at around 20 weeks, but after 60 weeks, GDP per capita is about the same. On the other hand, the improvement in GDP with testing is milder in the benchmark calibration, but output is above the no-testing case for much longer. By the end of the first year, testing improves GDP by 2.8 percentage points in the calibration with a higher fraction of time-constrained agents compared with an improvement of 1.9 percentage points in the benchmark calibration. This shows that testing pays off even more in the context of poorer economies at the onset of the epidemic.

F. What if mortality is higher?

There is a large uncertainty about the case mortality rate of COVID-19. Different countries will also eventually have different mortality rates because of variation in demographic structures and hospitals' technical and physical capacities. All these reasons motivate the discussion of the sensitivity of our model results to this parameter.

Case mortality rate was initially thought to be as high as 3.4 percent. Some serology tests suggested that it could be as low as 0.3 percent and Fernández-Villaverde and Jones (2020) think that it could be between 0.8 percent and 1.2 percent. Figure 12 presents the results when the case mortality rate is 1 percent and compares it with the benchmark δ calibration.

The epidemiologic dynamic is qualitatively similar, but the peak of infections is lower when mortality is higher (Figure 12a). The case mortality rate per se should not impact the number of infections, since in our model it does not affect the time during which one is infectious. However, when the case mortality rate is higher, unconstrained agents would be even more careful in their activities outside the home, which lowers the possibility of a match between susceptible and infected agents. While high mortality rate, by definition, implies that more infected people would die, more careful agents would lower the number of infections. In our calibration, the first effect is larger, and about 300,000 more people would die in the US after two years (Figure 12d). It is interesting to note that the initial phase of the epidemic is iden-

tical regardless of the assumed mortality, a result also discussed in Fernández-Villaverde and Jones (2020). The epidemic's paths only start to diverge around week 37 and peak at exactly the same time at week 42. However, the peak is lower when the mortality rate is higher. At the end of the second year, slightly fewer people get ever infected when mortality is higher, as expected.

The economic impact of higher mortality is dramatic and the maximum difference in output coincides with the peak of the epidemic. Similar to the case when the fraction of time-constrained agents is larger, a higher mortality rate implies that it is riskier to engage in outside activities. For the first 20 weeks of the epidemic, labor supply by unconstrained agents is about the same. As the number of infected workers increases, engaging in outside activities is riskier, and unconstrained agents cut their labor supply by more when mortality is higher. At the trough, labor supply drops by more than 35 percent, compared to less than 15 percent in the benchmark calibration. Overall, the economic difference is massive (Figure 12a). In the first year, GDP per capita falls by 12.5 percentage points when mortality is higher compared to 4.8 percentage points in the benchmark calibration. Similar to the case with more time-constrained agents, testing is more effective in smoothing the GDP contraction when mortality is higher. The reason is similar to before: testing reduces the speed of the epidemic and encourages unconstrained people to work more.

G. Government-enforced lockdown

Governments around the world used lockdowns as the first line of response to the epidemic. With very few exceptions, lockdowns and mass testing were the chosen tools to delay the speed of the epidemic. This sub-section analyzes the impact of two different lockdown strategies, varying in intensity and duration.

A lockdown could be comprehensive and short, (lockdown A) or mild and long (lockdown B). We have seen countries adopt these two strategies which motivates our policy design scenarios. Following Brotherhood and others (2020), we propose a short and overarching lockdown in which all agents are forced to shelter at home for no less than an extra 25 percent of their time for one month, and another in which all agents are mandated to shelter at home for no less than an extra 10 percent of their time, but for 26 weeks.[9]

[9]We assume that the lockdown starts in the 11th week of the epidemic.

Lockdowns slow the epidemic in the first year but have almost no effect by the end of the second year. This dynamic can be seen in Figure 14b and is similar to the findings in Brotherhood and others (2020). The peak in infections is quite similar, but it is delayed by a few weeks with the lockdowns. In the first year 11,400 and 9,500 lives are saved by lockdowns A and B, respectively. By the end of the second year, the difference is less than 5,000 (Figure 14d). The number of lives saved might small, but it is important to highlight that the counterfactual is our benchmark calibration model, in which agents independently reduce their time spent outside to avoid infection.

Without further measures, the extra recession generated by the lockdowns also disappears in the second year (Figure 14a). By design, all agents are forced to stay home during the lockdown, which explains the extra recession. But the effects on the economy are reduced because when agents are allowed to make decisions optimally, they choose to spend more time outside than they would have chosen without the lockdown (Figure 16). From the moment that the lockdown is lifted until the end of the second year, unconstrained agents always choose to be spend more time outside than they would have in a situation in which no lockdown was ever imposed. The time spent during the lockdown becomes irrelevant for levels of GDP per capita by the end of the second year, but accumulated losses are never recovered. Overall, GDP growth in the first year falls by 5.5 percentage points and 6.6 percentage points below the pre-epidemic steady-state values in lockdown A and B, respectively. This compares with a contraction of around 4 percentage points in the benchmark model. That is, the cost per life is around $26 million and $54 million in lockdowns A and B, respectively. Once again, the counterfactual used to calculate the cost per life is our benchmark model and not a scenario in which people are not careful at all. For example, if we use as a counterfactual our calibrated model, but assume that unconstrained agents do not change their behavior at all, then lockdown B would be responsible for saving 325,000 lives at a cost of $ 2.5 million.

A scenario in which people are less careful can be approximated by an economy with a larger share of time-constrained agents. Our model predicts that the cost per life is more favorable for this type of economy. The explanation for this result has to do again with the counterfactual we are comparing it to. We saw that an economy with more time-constrained agents has worse GDP and death outcomes in the first year. When a lockdown is imposed in this economy, the environment becomes relatively safer for the unconstrained agents, who respond with a significantly less negative labor supply after the lockdown is lifted (Figure 17).

A long government-mandated lockdown becomes ineffective for unconstrained agents. In the economy with a larger share of time-constrained agents, lockdown B becomes irrelevant

for unconstrained agents after week 24, as they voluntarily choose to stay home for longer hours than mandated by the government (Figure 17). By becoming ineffective in changing the choices of part of the population, this lockdown is also less damaging to the economy.

The lockdown strategy is more cost effective in poorer economies because it is marginally less damaging to the economy and more efficient in slowing the speed of the epidemic. The number of lives saved at the beginning of the epidemic with the lockdown more than offsets the higher deaths after the lockdown is lifted, resulting in more lives saved after one year. Overall, almost 17,000 lives are saved in lockdown B after the first year. Meanwhile, average GDP falls by around 10 percentage points in the first year compared to a contraction of 8.6 percentage points if no lockdown is imposed. (Figure 16a). More lives are saved at a lower marginal cost and the cost per life in lockdown B is around $14 million, which is less than half of the cost per life of the same lockdown imposed in an economy with more unconstrained agents.

H. Effectiveness of lockdowns when health systems may collapse

One important argument used in the defense of lockdowns is that the health systems would collapse absent strong government-enforced lockdowns. The idea is that the virus is so contagious that the health systems would quickly become flooded with new hospitalizations and more people would die due to inadequate lack of medical attention. This last model extension tries to incorporate the benefits of "flattening the curve" by assuming that the death rate is a time-varying Poisson arrival rate that is increasing in the ratio of sick agents per ICU unit.

In particular, we assume that $\delta_t = \overline{\delta} + (\frac{Ho_t}{ICU_t})^2 \frac{1}{K}$, where $\overline{\delta}$ is the mortality rate in the case where no physical constraint is considered, Ho_t is the number of hospitalizations, ICU_t is the number of ICU beds, and K is a constant used for calibration. For $\overline{\delta}$, we use our benchmark calibration. For the number of hospitalizations, we assume that 20 percent of sick people need to be hospitalized and 5 percent of them would require ICU treatment. For the number of ICU beds, we follow McCarthy (2020) and assume 34.7 beds per 100,000 inhabitants.[10] Finally, we calibrate K so that the probability of dying is four times larger than in the benchmark at the peak of the epidemic. For this calibration, we use information from Lombardy in Italy, a region highly affected by the epidemic. The mortality rate there was 70 percent higher than

[10]Source is National Center for Biology Information, Intensive Care Medicine, https://www.statista.com/chart/21105/number-of-critical-care-beds-per-100000-inhabitants/

the rest of Italy and 2.85 times larger than in Veneto, a region in Italy that controlled the epidemic early on Odone and others (2020).

Strains on the health system improve the cost-per-life of lockdowns, but also affect the counterfactual of no policy action. When accounting for the higher mortality, lockdowns A and B would be able to reduce first-year deaths by 15,000 and 16,000, respectively (Figure 18d). This number is not substantially higher than in the previous section because the additional strains on the health system also affect the behavior of unconstrained agents. If agents know that the probability of dying depends on the level of ICU hospitalizations, they would be more careful in engaging in outside activities even without a government-mandated lockdown, especially at the peak of the epidemic (Figure 19). The additional deaths from hospital constraints and this endogenous more careful response almost offset each other, and the number of lives saved by the lockdowns is not substantially larger than if those physical constraints were not considered.

On the other hand, this more prudent behavior also leads to more economic costs both in the economies with and without lockdowns. With strained health systems, the economy contracts by 5.6 percentage points without the lockdown and by 6.5 percentage points and 7.7 percentage points in lockdowns A and B (Figure 18a). Overall, the cost per life is indeed lower in this case, totaling $13.4 million and $27.7 million in lockdowns A and B, respectively, which is still somewhat larger than the ones obtained in the literature.

VI. Conclusion

The invisible threats that any epidemics pose are amplified when people do not know their health status. Information is always valuable, but even more so in a context where a large fraction of infections are asymptomatic.

Moreover, not everyone has the choice of foregoing income to avoid a viral infection in which the probability of survival is high. This heterogeneity of choices has deep implications for both the speed and reach of the epidemic, and the incidence of the disease.

In this paper, we proposed a framework to study these issues that have been largely ignored by the literature. We enriched a standard epidemiology model with optimal and heterogeneous economic choices and calibrated it to investigate stylized policy interventions such as a lockdowns and testing. We also discussed how these policies perform when the economic context or the characteristics of the disease and the dissemination of the virus are different.

While the magnitude of our results relies on modelling choices, qualitative results and new intuition and channels would remain valid for a wider range of models. Of course, the magnitude of our results depends on calibration inputs and some of our model assumptions. We have shown that the mortality rate as well as the levels of time-constrained and asymptomatic agents all have important implications for the quantitative results. We showed the importance of testing as a vehicle to change the epidemic's dynamics or to improve the economy by providing information. We showed that this second channel is even more important in the context of an epidemic with a large number of asymptomatic cases. We showed that the economy could suffer more when the fraction of asymptomatic cases is larger. However, we postulated a speedier recovery that would not offset all accumulated losses. We showed that an economy with more time-constrained agents would have a worse economic recession, even though a larger fraction of the population would not change their behavior in response to the epidemic. Our direct model prediction is that the pandemic will eventually have a larger impact on poorer countries' health outcomes, even without considering lower physical and technical hospital capacity.

We showed that the assessment of containment measures has to be done against a realistic counterfactual. That is, we should expect agents to take precautions against the virus even if they are not mandated by the government to do so. Ignoring this fact would overestimate the number of saved lives directly linked to a lockdown. In this context, our model suggests that the cost per life of a lockdown could be smaller when more time-constrained agents are present.

Finally, we showed that the virus is more likely to affect time-constrained agents, but that most of the economic recession would come because unconstrained agents decide to cut back their time spent outside home. While the poor bear most of the health cost, the rich are responsible for the economic slowdown, as they take precautions and stay at home.

Figure 3. Movements across state spaces – Calibrated Model

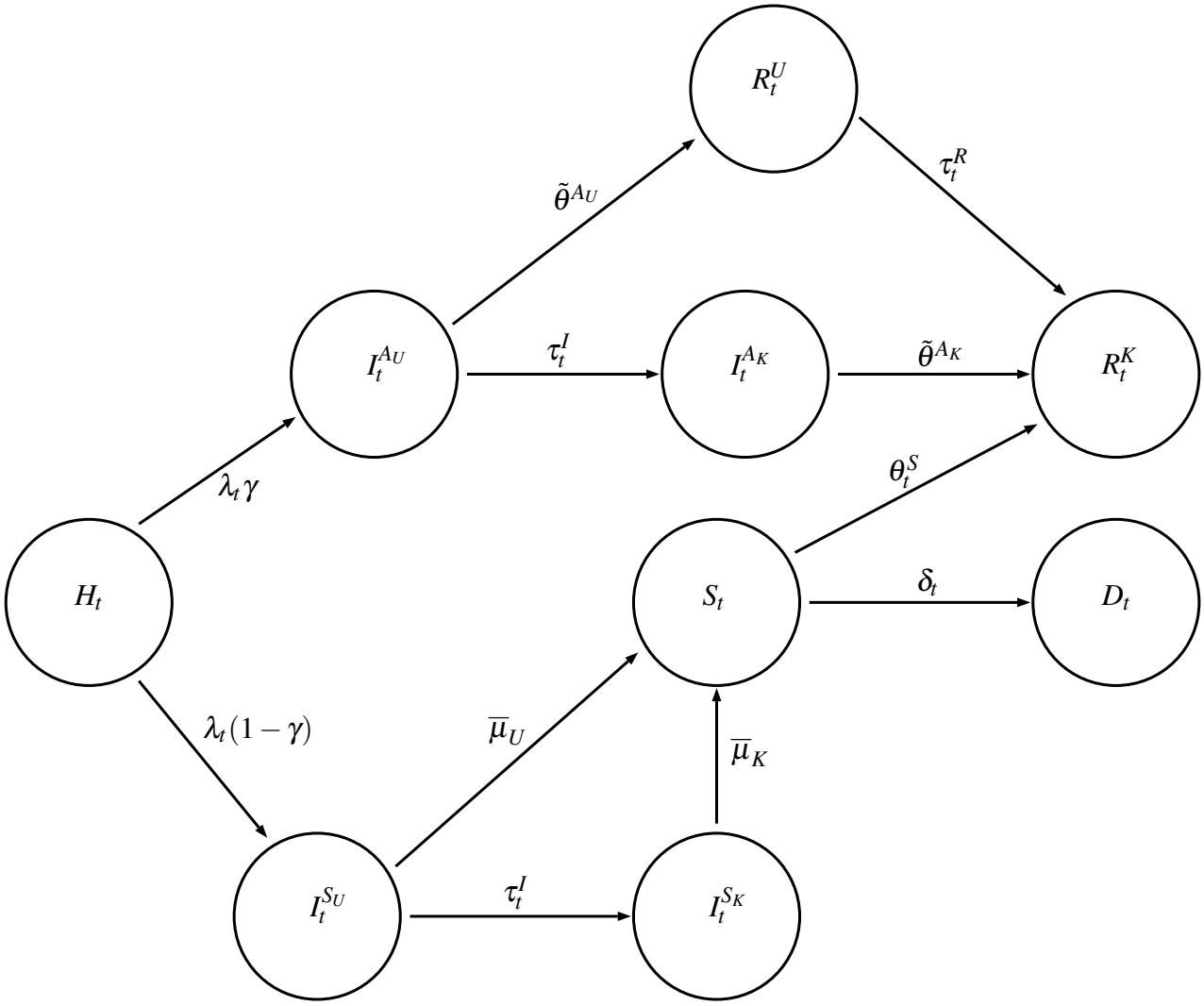
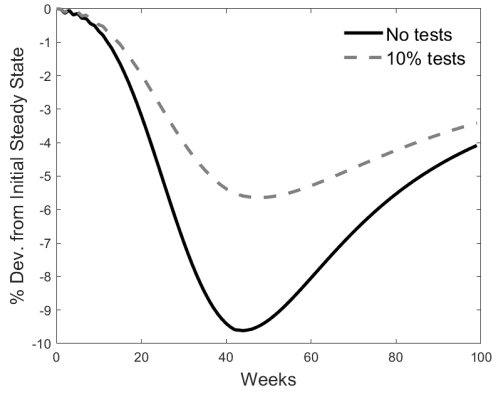
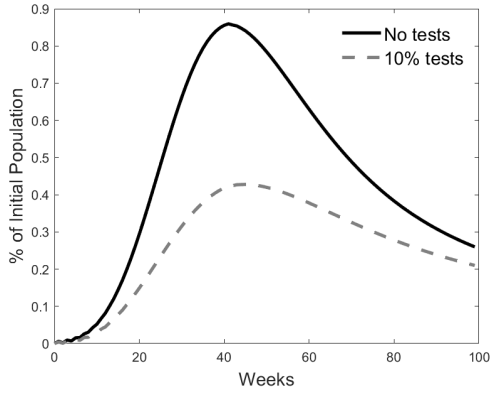


Table 2. Calibrated Parameters

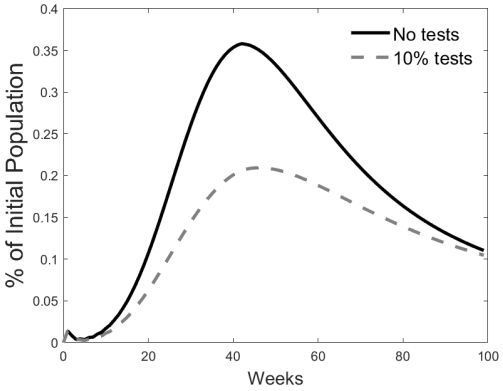
Parameter	Description	Value	Target or Source
$\tilde{\theta}^{A_U}$	Recovery rate - untested asymptomatic	0.5	Avg. time to recover
$\tilde{\theta}^{A_S}$	Recovery rate - tested asymptomatic	0.6	Avg. time to recover - avg. time to get tested
$\overline{\mu}_U$	Arrival rate of symptoms for untested		Avg. time to get sick
$\overline{\mu}_K$	Arrival rate of symptoms for tested		Avg. time to get sick - avg. time to get tested
δ	Death rate	$\frac{0.014}{4}$	From Eichenbaum et al. (2020b)
γ	Fraction asymptomatic infected	0.5	Iceland's incidence of asymptomatic
w	Wage rate	1	Normalization
$\overline{L}$	Agent's total time endowment	1	Normalization
c	Flow cost of sickness	0.2	Utility loss from leisure
c_d	Loss due to death	1,274	Value of Statistical Life.
ζ	Infection matching - labor	0.15	
χ	Infection matching - leisure	0.15	
β	Discount rate	$0.96^{(1/52)}$	From Eichenbaum et al. (2020b)
b	Consumption flow while out of work	0.2	20 percent replacement rate
$\mathbb{A}_s$	Matching function scale - leisure	0.3	
$\mathbb{A}_l$	Matching function scale - labor market	0.3	
u_h	Utility - leisure at home	0.3846	Match ACS avg. time spent home, working, and leisure.
α	Utility - consumption vs. outside leisure	0.51921	Match ACS avg. time spent home, working, and leisure.



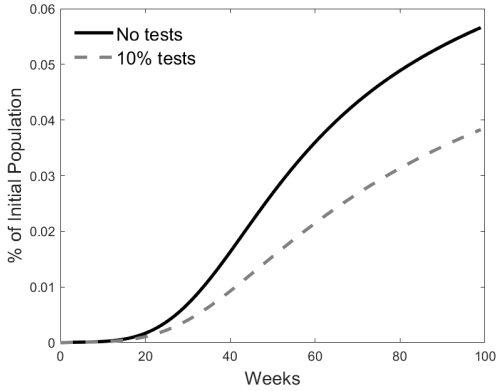
(a) GDP Effects



(b) Infected

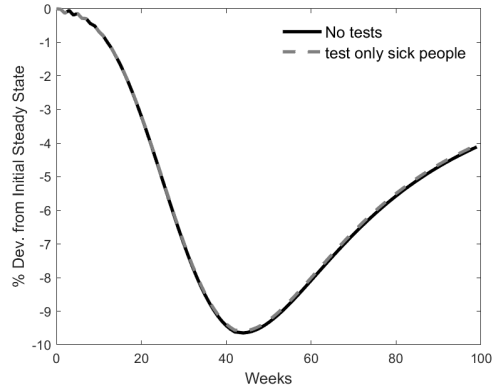


(c) Sick



(d) Dead

Figure 4. Pandemic's pattern: With and without testing



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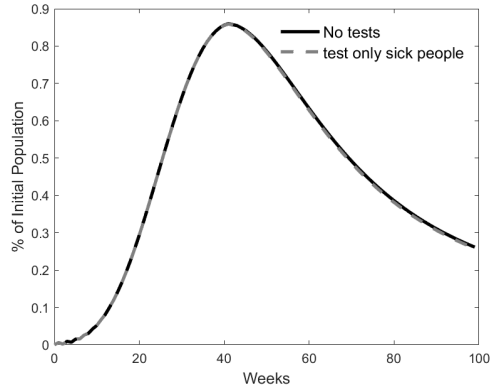

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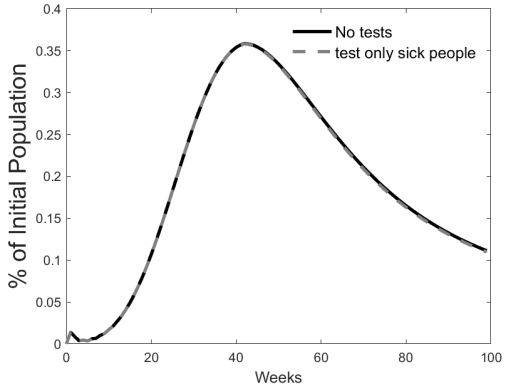

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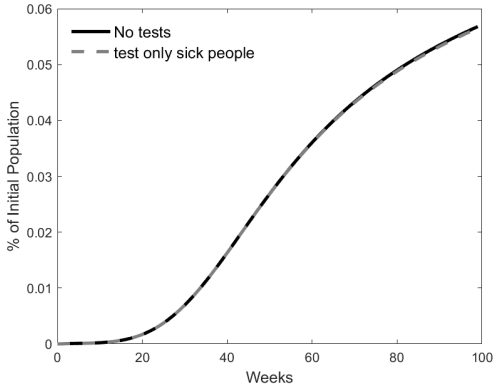
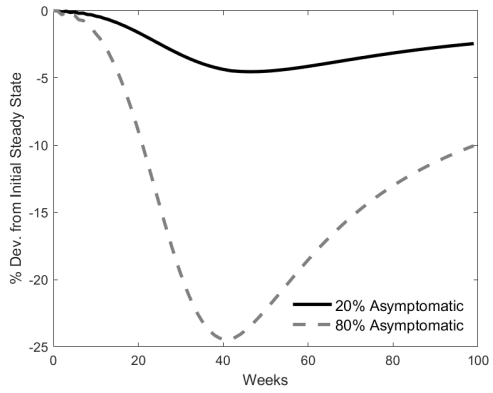
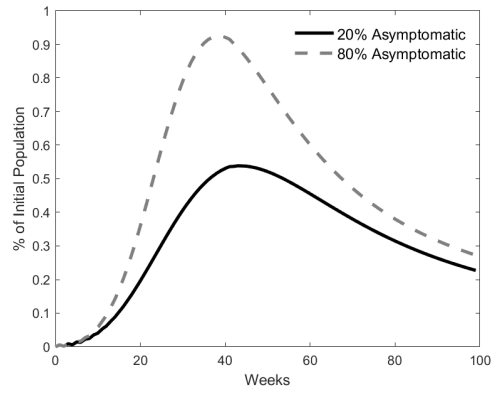


(d) Dead

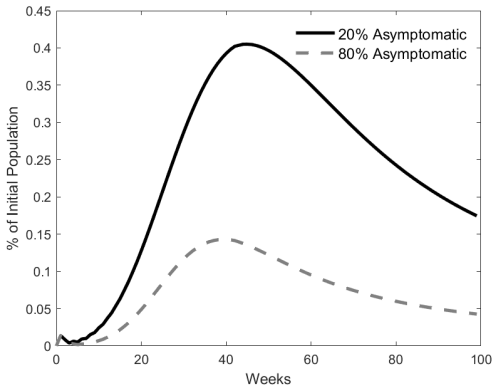
Figure 5. Pandemic's pattern: Testing only sick agents



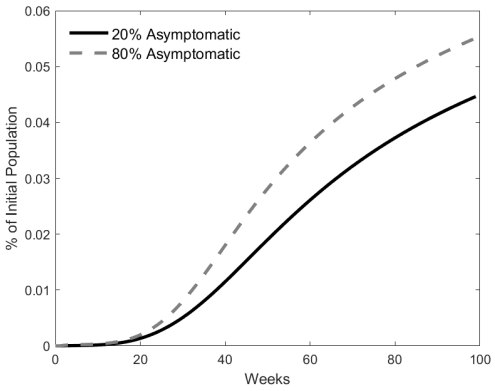
(a) GDP Effects



(b) Infected



(c) Sick



(d) Dead

Figure 6. Pandemic's pattern: Different levels of asymptomatic

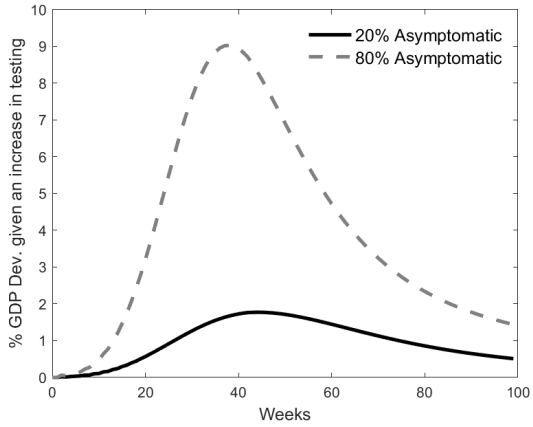
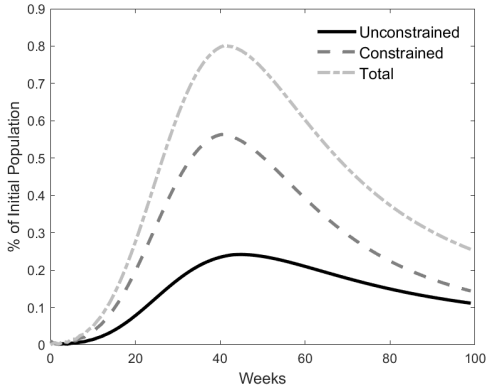
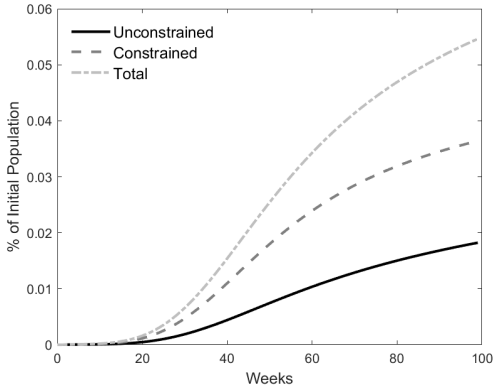


Figure 7. Impact of testing given levels of asymptomatic

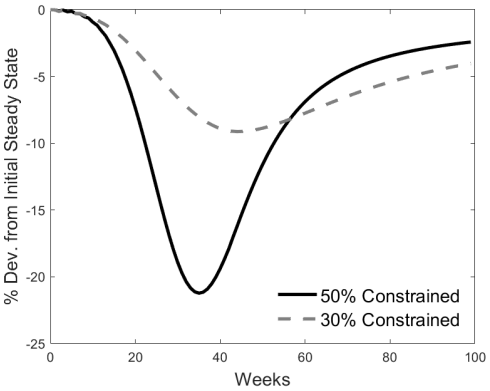


(a) Infected

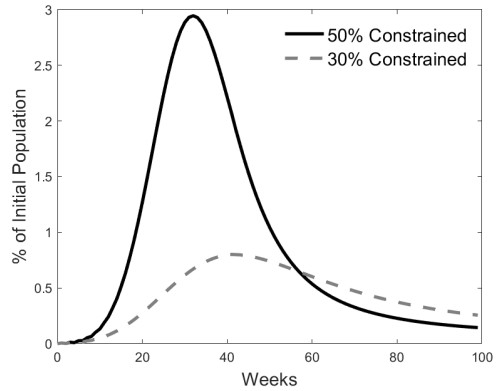


(b) Dead

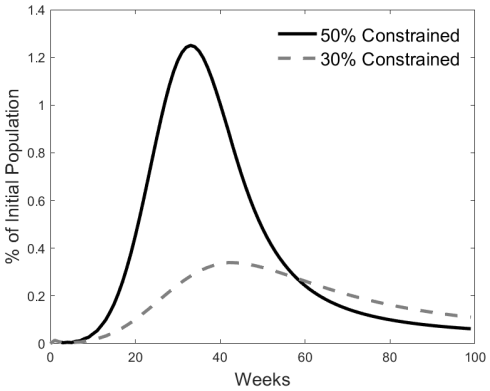
Figure 8. Pandemic's pattern: Unconstrained vs. time-constrained agents - Benchmark



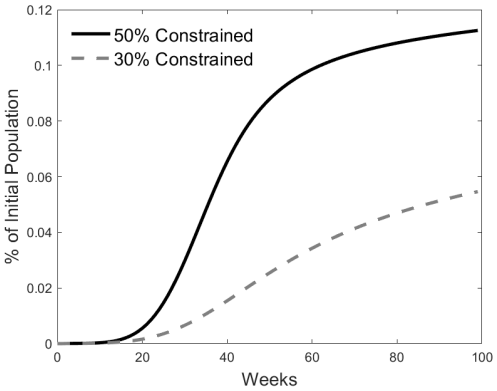
(a) GDP Effects



(b) Infected

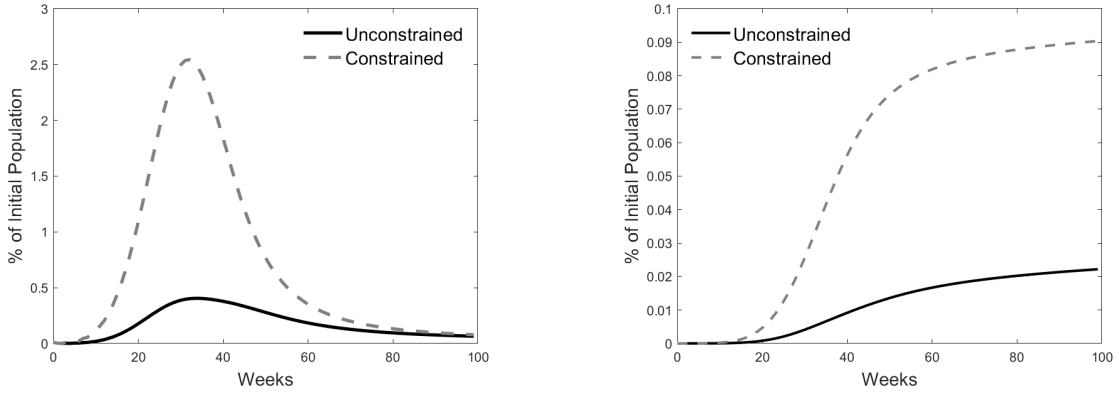


(c) Sick



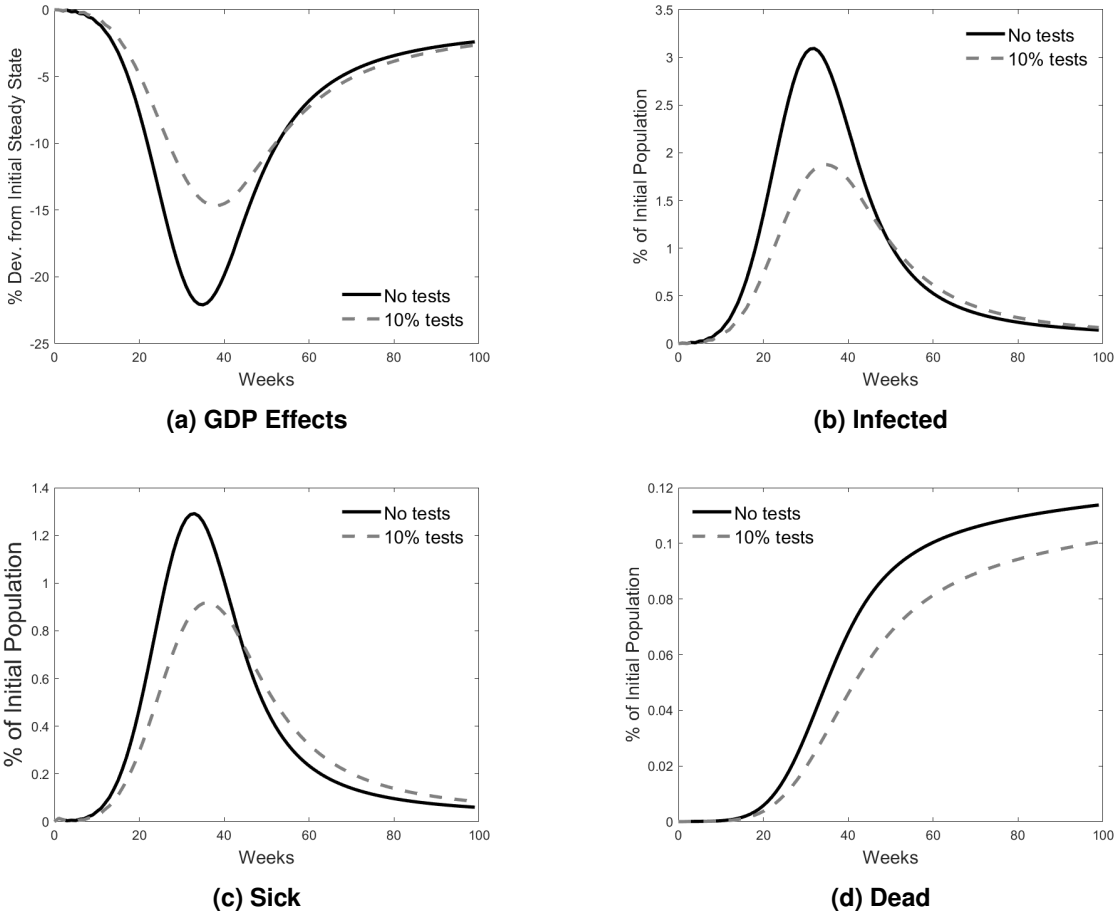
(d) Dead

Figure 9. Pandemic's pattern: Different levels of time-constrained agents



(a) Infected **(b) Dead**

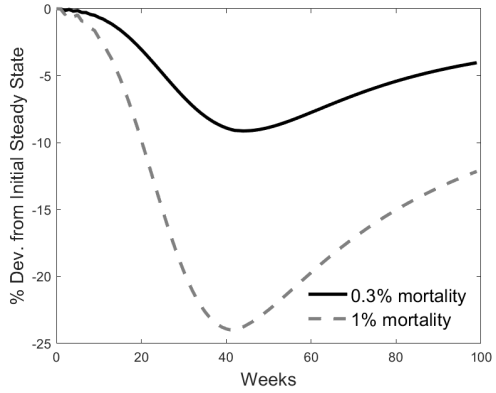
Figure 10. Pandemic's pattern: Unconstrained vs. time-constrained agents - 50 percent constrained



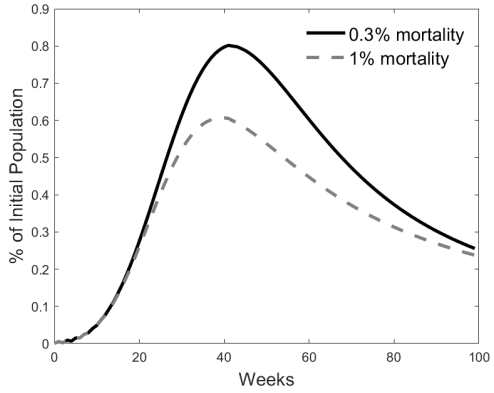
(a) GDP Effects **(b) Infected**

(c) Sick **(d) Dead**

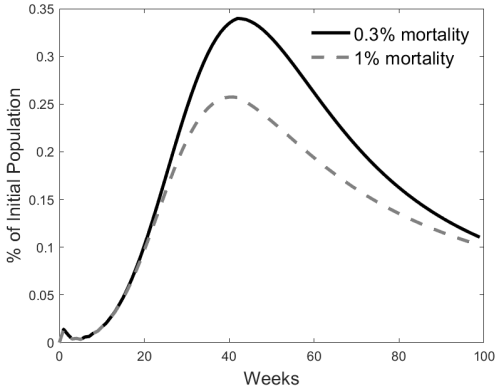
Figure 11. Pandemic's pattern: With and without testing - 50 percent time-constrained



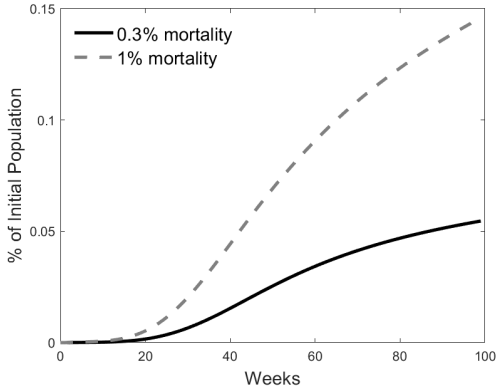
(a) GDP Effects



(b) Infected



(c) Sick



(d) Dead

Figure 12. Pandemic's pattern: High vs. low mortality rates

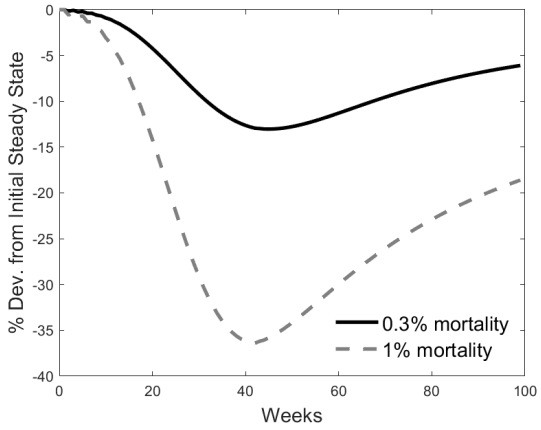
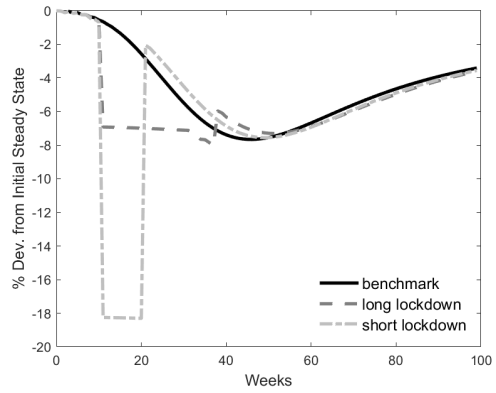
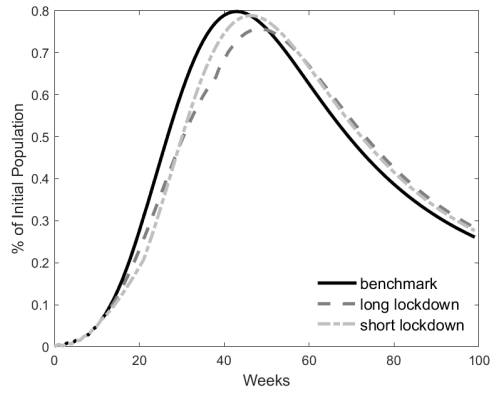


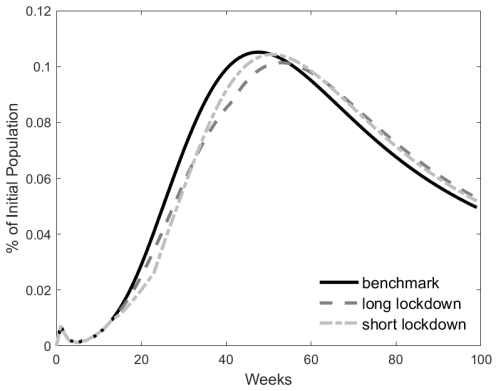
Figure 13. Hours worked by susceptible unconstrained workers



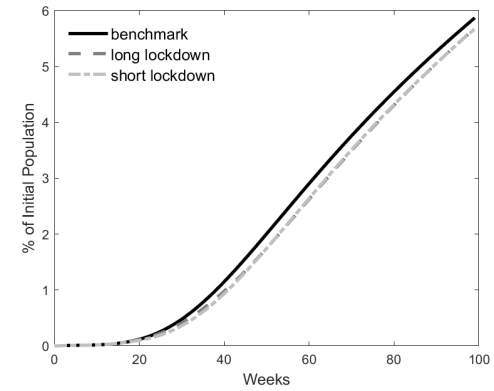
(a) GDP Effects



(b) Infected



(c) Sick



(d) Dead

Figure 14. Pandemic's pattern: Effect of lockdowns

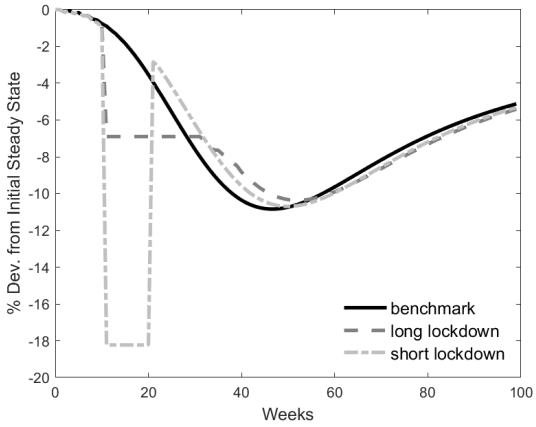


Figure 15. Hours worked by susceptible unconstrained workers

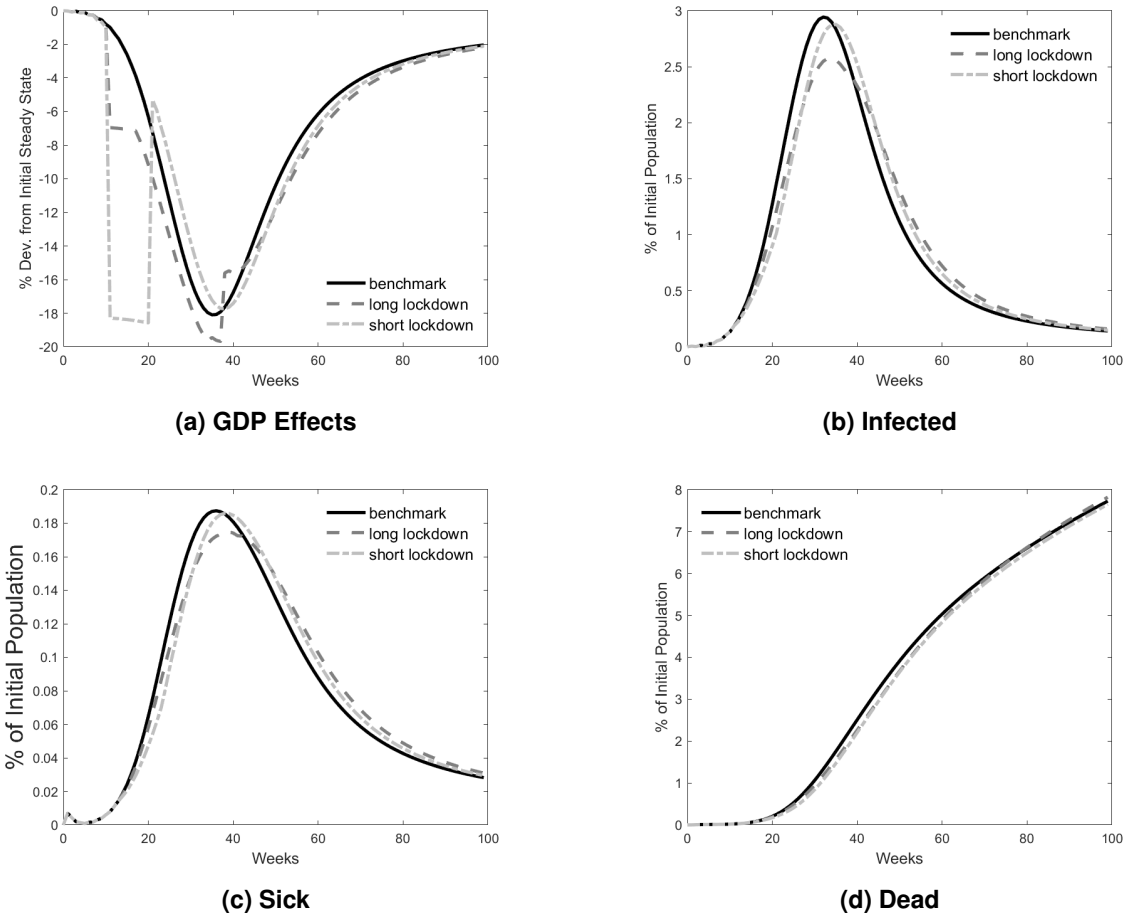


Figure 16. Pandemic's pattern: Effect of lockdowns - 50 percent time-constrained

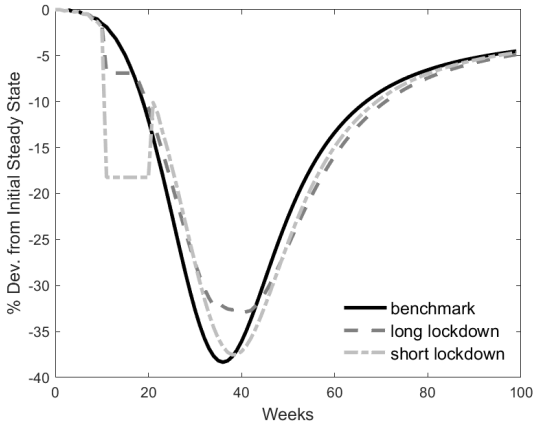
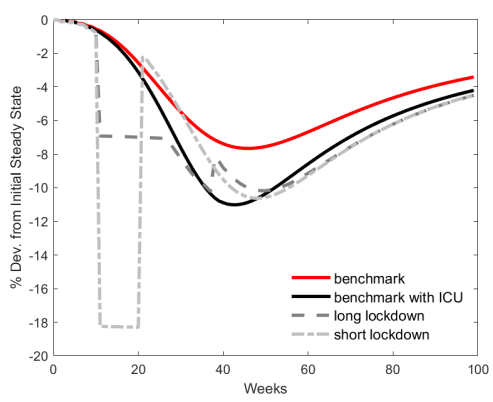
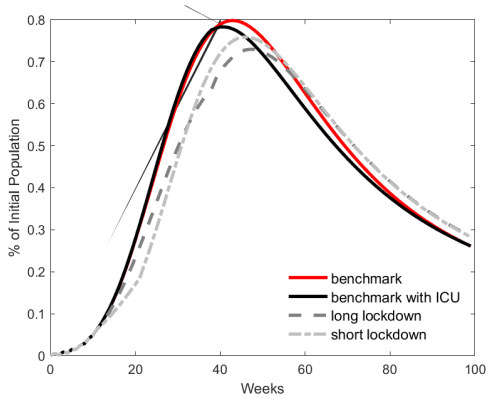


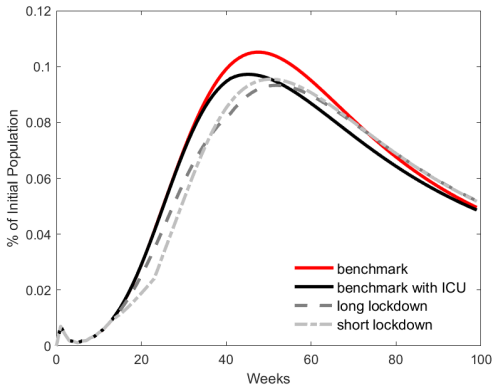
Figure 17. Hours worked by susceptible unconstrained workers



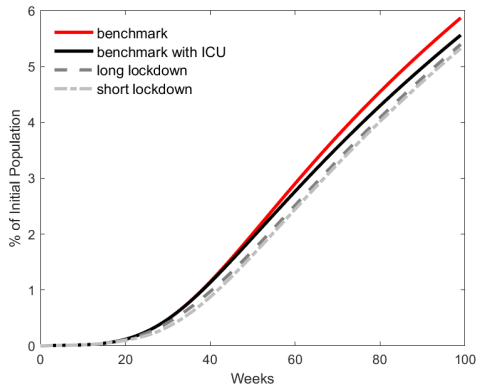
(a) GDP Effects



(b) Infected



(c) Sick



(d) Dead

Figure 18. Pandemic's pattern: Effect of lockdowns – exhaustible ICU beds

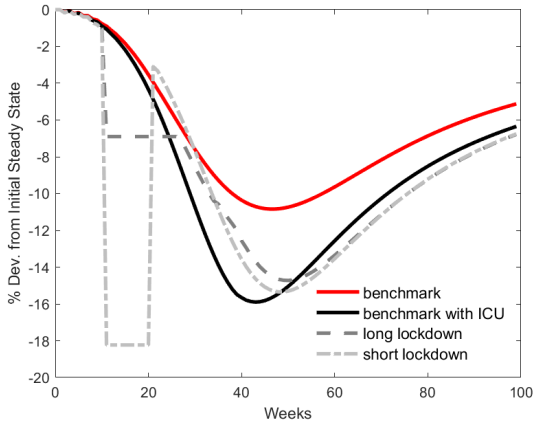


Figure 19. Hours worked by susceptible unconstrained workers

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