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Pandemic Recessions and Contact Tracing

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Abstract

We study contact tracing in a new macro-epidemiological model in which infected agents may not show any symptoms of the disease and the availability of tests to detect these asymptomatic spreaders of the virus is limited. Contact tracing is a testing strategy aiming at reconstructing the infection chain of newly symptomatic agents. A coordination failure arises as agents fail to internalize that their individual consumption and labor decisions raise the number of traceable contacts to be tested, threatening the viability of the tracing system. The collapse of the tracing system considerably aggravates the pandemic's toll on the economy and mortality. A timely, limited lockdown solves the coordination failure allowing policymakers to buy time to expand the testing scale and to preserve the tracing system. We provide theoretical underpinnings to the risk of becoming infected in macro-epidemiological models. Our solution method is not affected by curse-of-dimensionality problems.

JEL Classification: E10, I10, D62.

Keywords: Contact tracing, testing, COVID-19, infection chain, pandemic, lockdown, SIR macro model, heterogeneous agent model

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1 Introduction

The outbreak of COVID-19 led governments of all countries to confront a terrible trade-off between saving people's lives and protecting the economy from the worst recession of modern times. To mitigate this trade-off, quickly expanding the capacity for testing, isolation, and contact tracing has been suggested to be a crucial step by several experts. For instance, Dr. Anthony Fauci, the director of the National Institute of Allergy and Infectious Diseases, said in an interview with Dr. Howard Bauchner, the editor of the Journal of the American Medical Association in April 2020 that: "The keys are to make sure that we have in place the things that were not in place in January, that we have the capability of mobilizing identification – testing – identification, isolation, contact tracing." Contact tracing was indicated by WHO Director General Tedros Adhanom Ghebreyesus as one of the key reasons behind the success of South Korea in containing the spread of the coronavirus without economically damaging lockdowns.[1]

We develop a macro-epidemiological model to study the efficacy of contact tracing and show that even a limited tracing technology goes a long way in improving the solution to the pandemic trade-off. In the model, agents who become infected do not have any symptoms at first. While they remain asymptomatic, they do not know that they are infected and, thereby, keep consuming and working exactly as when they were not infected. In doing so, they create a network of contacts with other agents through which they silently spread the virus. When they turn symptomatic or when they get tested, these spreaders are detected and quarantined by the health authorities so that they cannot infect anyone else.

Contact tracing is a testing strategy that aims to reconstruct as much as possible of the newly symptomatic cases' *infection chain* – i.e., the network of interactions that led the newly symptomatic case to become infected or to infect other agents. This reconstruction forms the basis to decide who to test. How much of the infection chain can be reconstructed by health officials defines the efficiency of the contact tracing technology. The objective of testing is to detect as many asymptomatic spreaders as possible and quarantine them.

Agents' consumption and labor decisions have externalities on the number of subjects that health authorities have to trace and test. Since agents fail to realize the existence of these externalities *ex-ante*, their consumption and labor decisions may end up overburdening the testing system to the point of making it insufficient to contain the spreading of the virus. The heightened risk of becoming infected causes agents to want less economic interactions, which they achieve by reducing their consumption and labor. This *ex-post* adjustment in

[1]WHO Director-General's opening remarks at the media briefing on COVID-19 - 18 March 2020. https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19—18-march-2020.

their consumption and labor schedules sparks a severe pandemic recession.

A timely, limited lockdown solves the coordination failure allowing the health authorities to buy time to ramp up their testing capacity. By averting the collapse of the testing system, the lockdown greatly mitigates the pandemic recession. But this is not the only way to shore up the testing system against agents' coordination failure in the model. Improving the efficiency of contact tracing also makes the testing system more resilient and reduces the extent of lockdown or, in some cases, eliminates the need of a lockdown.

When the epidemiological parameters of the model and the availability of tests are calibrated to match the U.S. data during the pandemic, we find several interesting results. Contact tracing is more successful than random testing because it exploits the existence of an infection chain connecting the newly symptomatic agents with the subjects they have infected in the current period. Therefore, the probability of finding an asymptomatic spreader by testing one contact of a newly symptomatic person is much higher than the probability of catching an asymptomatic spreader by randomly testing one subject in the population. We find that a strategy based on randomly testing the population requires an unrealistically large testing capacity to effectively contain the spread of the virus.

If the contact tracing technology had allowed health officials to trace interactions for a period of one week (*basic contact tracing technology*), the pace at which the U.S. built up testing capacity at the beginning of the pandemic would have not been fast enough to stop the rapid spread of the virus. As we emphasized above, agents consume and work too much as they fail to realize that their individual consumption and labor decisions have negative externalities on the viability of the testing system. However, the tracing and testing system can be preserved by imposing a very mild lockdown.

The lockdown actually mitigates the pandemic recession and reduces its death toll. By ensuring the correct functioning of the testing system, the lockdown prevents the surge in the infection rate and the ensuing drop in consumption and employment. This result underscores the existence of exploitable complementarities between lockdowns and testing and the critical importance of preserving the viability of the testing system for a successful management of the pandemic.

Lockdowns are often enacted in response to an acceleration in the number of infections as a last resort to flatten the curve and to prevent the health system from becoming overburdened. In this paper, we suggest a quite different strategy that envisions lockdowns as a preemptive tool to keep the tracing and testing system viable while policymakers ramp up their testing capacity. Unlike the more common lockdowns, the type of lockdowns studied in this paper are generally less stringent and are used preemptively with the objective of moving ahead of the (infection) curve. Indeed, in our model, a surge in the number of infections is

the unequivocal sign that the tracing system is already not working properly.

Improving the efficiency of the contact tracing technology mitigates the pandemic trade-off considerably. We show that the ability of tracing contacts that occurred as far back as the previous week (*comprehensive contact tracing technology*) would have improved the economic outcomes considerably and saved several thousands of human lives compared to the basic tracing technology. The more comprehensive tracing technology would have allowed a more efficient use of the limited U.S. testing capacity at the beginning of the pandemic. While social welfare maximization requires the government to restrict economic activities somewhat, imposing no restriction has only tiny negative effects on welfare.

The reason behind this result is that the comprehensive contact tracing technology makes the testing system more resilient to the excess consumption and labor owing to agents' coordination failure. The comprehensive tracing technology gives health authorities a second chance to test asymptomatic spreaders who were not detected by testing in previous periods. Under the basic tracing technology, these undetected spreaders will not be traceable via their infection chain in any future period.[2] It is actually worse than that, since the entire infection chain, which each of these undetected spreaders will create going forward, becomes very hard for the health authorities to uncover.[3]

In our simulation, the comprehensive technology allows the government to effectively keep the number of asymptomatic spreaders lower in the very early stages of the pandemic crisis notwithstanding the extremely limited U.S. testing scale. A few periods later the government can cash in on the lower number of asymptomatic spreaders, which, by reducing the number of newly symptomatic subjects, diminishes the number of tests that need to be performed. A less constrained testing system will be able to support more elevated levels of consumption and labor, and consequently, the severity of the required lockdown is reduced.

Modeling contact tracing is extremely challenging as the number of contacts established by a newly symptomatic subject quickly explodes as the number of past periods considered increases. We solve this dimensionality problem by assuming that the probability of a subject entertaining a number of economic interactions with a set of subjects is modeled as a binomial trial. The number of trials depends on how much the agents consume (work) in the period and the probability of success (i.e., meeting with an asymptomatic infected subject). This probability is assumed to depend on the consumption (work) of asymptomatic infected people relative to the overall consumption (labor) in the economy. These binomial distributions allow us to parsimoniously keep track of the fraction of agents of a given type (susceptible,

[2]They can only be detected if they will randomly bump into one of the subjects who will develop the symptoms of the disease. But, as explained already in the text, this is a relatively low-probability event.

[3]This is because it will take at least one period for newly infected people to show symptoms.

asymptomatic, etc.) who have entertained a finite number of contacts with a given subset of agents. This statistical object is enough to solve the model under contact tracing as it contains all the necessary information to reconstruct all the traceable infection chains in our model. We show that this approach of modeling interactions as binomial trials makes the epidemiological part of our model isomorphic to the canonical SIR model proposed by Kermack and McKendrick (1927) and macro-epidemiological models (e.g., Eichenbaum, Rebelo and Trabandt, 2020*a*).

Our model is related to the recent and quickly growing literature that studies the effects of COVID-19 on the economy and how policymakers can mitigate these effects by imposing lockdowns or conducting tests. This literature is quickly growing and includes already quite many path-breaking contributions, including Acemoglu et al. (2020), Akbarpour et al. (2020), Alvarez, Argente and Lippi (2020), Atkeson et al. (2020), Aum, Lee and Shin (2020), Azzimonti et al. (2020), Baqaee et al. (2020), Berger et al. (2020), Bethune and Korinek (2020), Brotherhood et al. (2020), Chari, Kirpalani and Phelan (2020), Eichenbaum, Rebelo and Trabandt (2020*a*,*b*), Galeotti, Steiner and Surico (2020), Glover et al. (2020), and Hornstein (2020).[4]

Our contribution differs from these papers in one or more of the following aspects. First and most importantly, we study a novel type of coordination failure that can disrupt the functioning of the tracing and testing system and we provide an optimal resolution to this problem. Second, we model contact tracing by formally characterizing the network of agents' interactions, which, to our knowledge, has not been done before. This approach makes the set of agents that are traced and need to be tested in every period endogenous. This is because the number of agents exposed to the newly symptomatic subjects in a given period depends on how much agents have consumed and worked in the past. This feature of our model is key for the coordination failure to arise. Third, we use binomial distributions to summarize the network of contacts parsimoniously. Fourth, in our model, the objective of tracing and testing is not to quarantine a fixed fraction of the infected. The objective of testing is to detect asymptomatic spreaders, whose health status is assumed to be non-observable in the model. Fifth, our model is a macro model in which agents consume and work and, in doing so, interact with each other and can become infected.

The paper is organized as follows. In Section 2, we present the model. In Section 3, we formally introduce the basic and the comprehensive technologies of contact tracing. In Section 4, we discuss the solution method and the calibration of the model. In Section 5, we show simulations of the model to illustrate the main lessons we learned in this paper. In Section 6, we conclude.

[4]Atkeson (2020) provides a review of SIR models from an economist's perspective.

2 The Model

The model economy is populated by agents who consume and work, firms which hire labor N_t from agents in a competitive market and produce output according to a linear production function in labor and productivity parameter A. The government which levies taxes on consumption and remits transfers to agents. Labor and output are traded in competitive markets. Health authorities conduct contact tracing, administer tests, and can quarantine agents. Agents become infected through interactions with other agents. Following Eichenbaum, Rebelo and Trabandt (2020a), we assume there are three types of interactions through which the virus spreads out: consumption interactions, work interactions, other interactions.

The time protocol goes as follows: First, agents consume, work, and engage in other interactions at the beginning of the period. Second, agents' health status can change: agent can get infected or infected agents can recover or die. Third, health officials can test agents if a tracing technology is in place and tests are available. We will defer a detailed discussion about tracing and testing to Section 3.

There are six types of agents, which differ in their health status. The first type includes *susceptible agents* who have not contracted the disease, are not carrier, and are not immune. Infected agents can be divided into three types: *Untested asymptomatic agents* if they have not shown symptoms and have not been tested positive, *positive-tested agents* if they are asymptomatic but they have tested positive, and *symptomatic infected agents* if they have shown symptoms regardless of whether they have been previously tested positive. The remaining two types are the recovered agents, which have developed immunity. They are the *observed recovered agents*, which have shown symptoms or have been tested positive and the *unobserved recovered agents* who have recovered without having ever showed any symptoms of the disease or having ever been tested positive.

Types' Transitions. When a *susceptible agents* becomes infected, it first becomes infected asymptomatic. If it does not get tested at the end of the period, it will enter the next period as untested asymptomatic. If it gets tested at the end of the period, it will enter the next period as *positive-tested agents*.[5] An untested asymptomatic can develop symptoms with probability π_{IS}, becoming an infected symptomatic agents, can recover with probability π_R, becoming an unobserved recovered, or can remain untested asymptomatic if it is not tested at the end of the period, or can become ested-positive agent if tested positive at the end of the period. A positive-tested agent can develop symptoms with probability π_{IS}, becoming a symptomatic agent, or can recover with probability π_R, becoming an observed

[5]We allow for false-negative test outcomes. If a false-negative outcome occurs, the infected agent remains untested asymptomatic.

recovered agent, or else remains positive-tested agent. A symptomatic agent at the end of the period can recover with probability π_R, becoming an observed recovered agent, or can die with probability π_D, or can remain symptomatic infected. Both unobserved and observed recovered do not change their health status over time as they have developed immunity.

Observability of Types' Health Status. Since the untested asymptomatic individuals are assumed not to show any symptoms of the disease, *their health status is not observed* by anyone in the model. The health status of untested asymptomatic individuals is publicly revealed if they get tested or they show symptoms. The health status of unobserved recovered individuals is also unobserved. The health status of observed recovered individuals is publicly observed as these individuals have either stopped showing the symptoms of the disease or -if they have never shown symptoms- they no longer test positive.

Susceptibles, untested asymptomatic, and unobserved recovered individuals do not know their type and are assumed to make consumption and labor decision under the assumption that they have not been infected. While this assumption entails a deviation from rationality, this deviation has minimal implications for our results insofar as the fraction of infected people relative to the population remains fairly small. Solving the full-fledged imperfect information problem for these two types of agents is very cumbersome and hinders intuition.

Quarantine. The positive-tested and the symptomatic subjects have their health status revealed and the health authorities immediately quarantines them. Being quarantined means two things. First, in quarantine consumption and labor decisions are subject to restrictions, which are modeled as a consumption tax.[6] Second, quarantined agents are isolated from other subjects and cannot infect anyone. In addition to be quarantined, symptomatic infected subjects are subject to a penalty on their labor productivity. Since positive-tested agents do not show any symptoms, they are not subject to this labor productivity penalty.

Untested asymptomatic individuals cannot be quarantined because the health authorities cannot distinguish them from the susceptible agents. Consequently, these individuals are not subject to any restrictions and can infect other people.

Note that we use the word quarantine to mean a containment policy targeting a single subject or a subset of subjects who have been uncovered by the government as potentially capable to spread the virus (i.e., the tested-positive subjects and the symptomatic infected subjects.) Therefore, quarantine is different from lockdown, which refers to an economy-wide containment measure, which hence affects all the subjects regardless of their health status.

[6]This tax reduces both consumption and labor of quarantined individuals.

Lockdowns are also modeled as a consumption tax.[7]

2.1 Meeting Probabilities

The virus in our model spreads out because susceptible agents may meet with untested asymptomatic agents while consuming, working, or engaging in other non-economic activities.[8] So it is particularly important to characterize the probability that a susceptible individual meets with untested asymptomatic subjects. We make the following assumption to characterize this probability.

Assumption 1. *Every random interaction of an agent with a set of agents of a specified type is modeled as a Bernoulli trial.*

It then follows that the probability that an individual, who randomly meets $n > 0$ other agents in a period, meet k-times with agents of a certain type is given by the binomial distribution

$$\mathcal{B}(k, n, p) = \binom{n}{k} p^k (1-p)^{(n-k)} \tag{1}$$

where p is the probability of meeting with agents of a certain type in one random meeting. In the Bernoullian jargon, there will be n random trials and in each of these trials the individual meets (success) or does not meet (failure) with a specified group of people. We make the following assumption about the probability of meeting with a specified group.

Assumption 2. *The probability for an individual to meet with individuals of a certain type*

a) in one random consumption interaction is given by the share of consumption of the agents of that type relative to the total consumption of non-quarantined agents.

b) in one random working interaction is given by the share of hours worked by the agents of that type relative to the total of hours worked by non-quarantined agents.

c) in one random interaction not associated with either consumption or work is given by the share of agents of that type relative to the population of non-quarantined agents.

For instance, the probability to meet an untested asymptomatic subject in one consumption interaction is given by the size of the consumption of untested asymptomatic people relative to aggregate consumption. In symbols, C_t^A/C_t, where C_t^A denotes total consumption of the untested asymptomatic and C_t stands for the aggregate consumption of non-quarantined

[7] Quarantined agents may or may not be affected by lockdwon. As we shall show, we can encompass both specifications.

[8] Other infected people – positive-tested and the symptomatic individuals – are quarantined and cannot infect anyone.

agents. Analogously, the probability for a worker to meet an untested asymptomatic worker in one unit of work is assumed to be N_t^A/N_t, where N_t^A denotes total labor worked by the untested asymptomatic group and N_t stands for aggregate labor of non-quarantined agents. The probability for an individual to meet with an untested asymptomatic in one non-consumption, non-labor interaction is assumed to be equal to the share of population who is untested asymptomatic. In symbols, I_t^A/Pop_t, where I_t^A denotes the size of the group of individuals who are untested asymptomatic and Pop_t stands for the size of population of non-quarantined agents.

The next assumption is about how much interactions are necessary to consume a given amount of goods, to work a given amount of hours, or to engage in other forms of interaction.

Assumption 3. *An individual of health status i who consumes c_t^i units of good, works n_t^i number of hours at time t makes $\varphi_C : c_t^i \mapsto \mathbb{N} \cup \{0\}$ and $\varphi_N : n_t^i \mapsto \mathbb{N} \cup \{0\}$, respectively, number of interactions, where $\mathbb{N} \cup \{0\}$ denotes the set of natural numbers including zero. The same individual also makes a constant number of φ_O interactions when engaging in activities other than consumption and labor.*

It follows that we can denote the total number of interactions a susceptible individual needs to entertain to consume, work, and enjoy other activities, is given by $\varphi_C(c_t^S)+\varphi_N(n_t^S)+\varphi_O$. This gives us the number of Bernoulli trials due to these three activities in the time unit. Note that the number of trials is endogenous and depends on agents' decisions of how much to consume and work. We can think of the mappings φ_C and φ_N as monotonically increasing step function who takes value of zero when consumption and hours work of the individual i is zero.

Combining all these assumptions allows us to write the probability for a susceptible individual to meet k-times with the set of asymptomatic subjects while consuming an amount c_t^S of goods as follows:

$$f_{c,t}\left(k\right) \equiv \mathcal{B}\left(k, \varphi_C(c_t^S), \frac{C_t^A}{C_t}\right) = \binom{\varphi_C(c_t^S)}{k} \left(\frac{C_t^A}{C_t}\right)^k \left(1 - \frac{C_t^A}{C_t}\right)^{\varphi_C(c_t^S)-k}, \quad k \leq \varphi_C(c_t^S) \tag{2}$$

We can analogously characterize the probability for a susceptible individual to meet k-times with the set of asymptomatic subjects while working an amount n_t^S of hours

$$f_{n,t}\left(k\right) \equiv \mathcal{B}\left(k, \varphi_N(n_t^S), \frac{N_t^A}{N_t}\right) = \binom{\varphi_N(n_t^S)}{k} \left(\frac{N_t^A}{N_t}\right)^k \left(1 - \frac{N_t^A}{N_t}\right)^{\varphi_N(n_t^S)-k}, \quad k < \varphi_N(n_t^S) \tag{3}$$

Finally, the probability for any person to meet with people in the asymptomatic group k times while engaging in other types of interactions is given by

$$f_{o,t}(k) \equiv \mathcal{B}\left(k, \varphi_O, \frac{I_t^A}{Pop_t}\right) = \binom{\varphi_O}{k}\left(\frac{I_t^A}{Pop_t}\right)^k \left(1 - \frac{I_t^A}{Pop_t}\right)^{\varphi_O - k}, \quad k < \varphi \tag{4}$$

Let us denote the number of random interactions due to consumption, work, and other activities is k_c,k_n, and k_o, respectively. The joint probability for a susceptible individual to have a triplet of random meetings (k_c, k_n, k_o) with untested asymptomatic people is defined

$$f_t(k_c, k_n, k_o) \equiv f_{c,t}(k_c) \cdot f_{n,t}(k_n) \cdot f_{o,t}(k_o) \tag{5}$$

2.2 Probability of Getting Infected

We make the following assumption about the probability that a susceptible individual becomes infected conditional on meeting with an untested asymptomatic individual.

Assumption 4. *Conditional on meeting with an untested asymptomatic individual, a susceptible agent will become infected with probability τ.*

Since this probability of getting infected τ is assumed to be the same across the three different types of interactions (consumption, work, or others), a susceptible individual entertaining $k_c + k_n + k_o$ interactions with asymptomatic individuals will become infected with probability $1 - (1-\tau)^{k_c+k_n+k_o}$; that is, one minus the probability that none of these interactions turns out to be infectious, which is given by $(1-\tau)^{k_c+k_n+k_o}$.

We can characterize the average probability for a susceptible individual to get infected conditional on consuming c_t^S and working n_t^S as follows:

$$\tau_t \equiv \sum_{k_c=0}^{\varphi_C(c_t^S)} \sum_{k_n=0}^{\varphi_N(n_t^S)} \sum_{k_o=0}^{\varphi_O} \left[1 - (1-\tau)^{k_c+k_n+k_o}\right] f_t(k_c, k_n, k_o), \tag{6}$$

where $f_t(k_c, k_n, k_o)$ denotes the joint binomial distribution defined in equation (5).

2.3 Susceptible Agents

Susceptible agents choose consumption c_t^s, and labor n_t^s so as to maximize

$$V_t^S = \max_{c_t^S, n_t^S} u\left(c_t^S, n_t^S\right) + \beta\left[\left(1-\tau_t\right) V_{t+1}^S + \tau_t\left\{\pi_{P,t}^T V_{t+1}^P + \left(1 - \pi_{P,t}^T\right) V_{t+1}^A\right\}\right], \tag{7}$$

where agents' period utility is standard: $u\left(c_t, n_t\right) = \ln c_t - \theta/2 n_t^{1/\eta}$ and β denotes agents' discount factor. The variable τ_t is the probability for a susceptible to get infected which is

defined in equation (6). The variable $\pi^T_{P,t}$ denotes the probability that an infected subject will test positive at the end of period t. The probability for an infected agent to test positive depends on the contact tracing technology, the availability of tests, and the probability of false negative outcomes. The characterization of this probability is the topic of Section 3.

With probability $(1-\tau_t)$, susceptible agents will not be infected in period t and hence there will be no change in their health status. However, with probability τ_t the susceptible agent becomes infected and her health status changes. The agent may become positive-tested in the next period $t+1$ if she is tested and the test does not give a false negative outcome at the end of period t. Otherwise, the agent will become an untested asymptomatic subject in the next period $t+1$. In the former case, the agent will receive the utility of the positive-tested agents in the next period, V^P_{t+1}, which will be defined in Section 2.5. In the latter case, the agent will receive the utility of an untested asymptomatic in the next period, V^A_{t+1}, which, for the period t, is given by

$$V^A_t = u(\tilde{c}^s_t, \tilde{n}^s_t) + \beta \left[\pi_{IS} V^{IS}_{t+1} + \pi_R V^{UR}_{t+1} + (1-\pi_{IS}-\pi_R)\left(\pi^T_{P,t} V^P_{t+1} + (1-\pi^T_{P,t}) V^A_{t+1}\right)\right] \tag{8}$$

where $\tilde{c}^s_t$ and $\tilde{n}^s_t$ denote the optimal solution to the susceptible agent's problem in equation (7). This is because the untested asymptomatic agent does not know her health status and keeps on consuming and working as if she were not infected. The probability π_{IS} denotes the fixed probability that the untested asymptomatic turns to symptomatic in period t and will receive utility V^{IS}_{t+1}. In this case, the agent will finally learn her health status and will solve the optimization problem shown in Section 2.6. The untested asymptomatic subject may also recover with fixed probability π_R and her health status turned to unobserved recovered, receiving utility V^{UR}_{t+1}, which is given by

$$V^{UR}_t = u(\tilde{c}^s_t, \tilde{n}^s_t) + \beta V^{UR}_{t+1} \tag{9}$$

Since the unobserved recovered agent is unaware that she has been recovered, she will keep on consuming and working as if she were still susceptible ($\tilde{c}^s_t$ and $\tilde{n}^s_t$).

Finally, the value function in equation (8) shows that if the untested asymptomatic individual does not develop the symptoms of the disease or does not recover, she can either test positive with probability $\pi^A_{P,t}$ or not. In the first case, the agent will become a positive-tested agent in the next period and will receive the value V^P_{t+1}. In the second case, the agent will remain untested asymptomatic and will receive the value V^A_{t+1} in the next period $t+1$. The characterization of the probability of testing positive for an asymptomatic agent, $\pi^A_{P,t}$, will be characterized in Section 3. Note that the probability of testing positive for an untested asymptomatic is different from the probability of testing positive for a newly

infected subject, $\pi^T_{P,t}$, in equation (7).

Budget constraint for non-quarantined agents. The problem solved by the susceptible agent is subject to the budget constraint for non-quarantined agents.

$$(1+\mu^L_{c,t})c^S_t = w^S_t n^S_t + \Gamma^L_t \tag{10}$$

where $\mu^L_{c,t}$ denotes a tax on consumption proxying the effects of a lockdown on consumption and labor. By reducing consumption and labor, the lockdown curtails agents' economic interactions. In doing so, lockdowns reduce the probability for susceptible individuals to become infected (τ_t) and, as we shall show, the number of traceable contacts health authorities have to test at the end of the period. The consumption tax revenue is rebated to the agents the tax is levied on, Γ^L_t. The equilibrium wage w^S_t equals the agent's labor marginal productivity.

2.4 Untested Asymptomatic and Unobserved Recovered Agents

The health status of untested asymptomatic and that of the unobserved recovered are not observed by agents. We assume that agents with those health status think they have never been infected and hence behave as susceptible agents, whose decision problems was explained in the previous section.

2.5 Tested-Positive Agents

Tested-positive agents are individuals who know they are infected even though they do not have symptoms. The choose consumption, c^P_t and labor n^P_t so as to maximize

$$V^P_t = \max_{c^P_t, n^P_t} u\left(c^P_t, n^P_t\right) + \beta\left[\pi_{IS}V^{IS}_{t+1} + \pi_R V^{OR}_{t+1} + (1-\pi_{IS}-\pi_R)V^P_{t+1}\right] \tag{11}$$

where the tested-positive individual can develop symptoms with probability π_{IS} and, in this case, the individual will receive the utility V^{IS}_{t+1} in the next period. The health status of the tested-positive individual can also change to observed recovered with probability π_R and, in this case, the individual will receive the utility V^{OR}_{t+1} in the next period. If the tested-positive individual neither develops symptoms nor recovers, she will remain in her current status of positively-tested in the next period.

Budget constraint for quarantined agents. Tested-positive agents are subject to quarantine until they recover. Thus, the maximization problem for these agents is subject to the

budget constraint

$$\left(1+\mu_c^Q+\alpha\mu_{c,t}^L\right)c_t^P = w_t^P n_t^P + \Gamma_t^Q \tag{12}$$

where μ_c^Q proxies the effects of individual-specific containment measures (aka quarantine) on consumption. Lockdowns are assumed to affect consumption of quarantined subjects as well. The parameter $\alpha \in (0,1)$ controls the additional effects of lockdown measures on quarantined agents's consumption. The tax paid by quarantined agents is rebated to them, Γ_t^Q.

2.6 Infected Symptomatic Agents

As the symptoms of the disease are developed, agents observe their health status, which becomes infected symptomatic. An infected symptomatic subject chooses consumption c_t^{IS} and n_t^{IS} so as to maximize

$$V_t^{IS} = \max_{c_t^{IS}, n_t^{IS}} u\left(c_t^{IS}, n_t^{IS}\right) + \beta\left[\pi_R V_{t+1}^{OR} + \left(1-\pi_R-\pi_D\right)V_{t+1}^{IS}\right] \tag{13}$$

subject to the budget constraint for quarantined subjects, which is shown for the tested-positive agents in equation (12). The probability π_R denotes the (fixed) probability that the health status of the infected symptomatic individual changes to observed recovered and the individual will receive V_{t+1}^{OR} in the next period. The probability π_D denotes the probability that the infected symptomatic individual dies and, in this case, she will get zero utility forever. If neither events happen, the infected symptomatic individual will not change her health status in the next period.

The equilibrium wage paid to the agents is determined by the agent's marginal productivity of labor, which is assumed to be lowered when the symptoms of the disease are developed.[9] This penalty on labor productivity is given by $\phi < 1$.

2.7 Observed Recovered Agents

Observed recovered agents are agents who know they have been infected at some point in the past either because they tested positive or they showed the symptoms of the disease. Since they have become immune to the virus, their health status will never change again and their decision problem reads:

$$V_t^{OR} = \max_{c_t^{OR}, n_t^{OR}} u\left(c_t^{OR}, n_t^{OR}\right) + \beta V_{t+1}^{OR}, \tag{14}$$

[9] Eichenbaum, Rebelo and Trabandt (2020*a*) make a similar assumption for the infected people.

subject to the budget constraint for non-quarantined subjects in equation (10). This assumption implies that lockdown is imposed also on agents that are known by the government to have recovered. While this assumption can be relaxed with virtually no effect on our main results, we make it to emphasize that a lockdown is a containment measure that hits *everyone* with no room for exceptions.

2.8 The Government Budget Constraint

The government balances its budget in every period by satisfying the conditions

$$\mu_{c,t}^{L}\left[C_t+\alpha\left(C_t^{IS}+C_t^{P}\right)\right]=\Gamma_t^{L}\left(S_t+I_t^{A}+R_t^{U}+R_t^{O}+(1-\alpha)\left(I_t^{S}+P_t\right)\right), \tag{15}$$

$$\mu_{c}^{Q}\cdot C_t^{IS}=\Gamma_t^{Q}\cdot I_t^{S}, \tag{16}$$

$$\mu_{c}^{Q}\cdot C_t^{P}=\Gamma_t^{Q}\cdot P_t, \tag{17}$$

where we denote the share of susceptible individuals with S_t, the share of untested asymptomatic individuals with I_t^A, the share of symptomatic infected individuals I_t^S, the share of positive-tested individuals with P_t, the share of unobserved recovered with R_t^U, and the share of observed recovered individuals with R_t^O. $C_t^{IS}\equiv c_t^{IS}I_t^S$ and $C_t^P\equiv c_t^P P_t$ stand for total consumption of the infected symptomatic agents and that of the tested-positive agents, respectively. There is no fiscal redistribution. The revenue of the lockdown and quarantined taxes are rebated to those agents these taxes are levied on.

2.9 Dynamics of Agents' Types

We have six types of agents. In this section, we describe the evolution of the measure of each of these types. The law of motion for the share susceptible agents reads

$$S_{t+1}=S_t-T_t \tag{18}$$

where T_t denotes the share of newly infected subject in period t. This share is defined using the law of large number as follows:

$$T_t=\tau_t\cdot S_t \tag{19}$$

where τ_t is the expected probability for susceptible individuals to become infected – defined equation (6).

The size of untested asymptomatic agents evolves according to the law of motion

$$I_{t+1}^{A}=(1-\pi_{P,t}^{T})T_t+(1-\pi_{P,t}^{A})(1-\pi_{IS}-\pi_R)I_t^{A} \tag{20}$$

where $\pi^A_{P,t+1}$ denotes the probability of testing positive conditional on being untested asymptomatic in the previous period. This set of agents are given by those who were untested asymptomatic I^A_t at the end of the previous period and have not developed symptoms, recovered, or tested positive at the end of the current period. Moreover, subjects who have become infected in this period, T_t and have not been tested positive will also join the set of the untested asymptomatic subjects in the next period.

The pool of tested positive subjects is given by

$$P_{t+1} = (1 - \pi_{IS} - \pi_R)P_t + \pi^T_{P,t}T_t + \pi^A_{P,t}(1 - \pi_{IS} - \pi_R)I^A_t \tag{21}$$

Tested-positive subjects in the current period are people who had this health status at the end of the previous period and have neither developed symptoms nor recovered. The infected agents who have just tested positive also join the positive tested pool.

The pool of infected symptomatic people evolves as follows:

$$I^S_{t+1} = (1 - \pi_R - \pi_D)I^S_t + \pi_{IS}(I^A_t + P_t) \tag{22}$$

A fraction of infected symptomatic agents recovers or dies in the period, the remainder remain infected symptomatic. Untested asymptomatic and positively tested agents can develop symptoms and become symptomatic infected subjects.

The share of unobserved recovered evolves as follows:

$$R^U_{t+1} = R^U_t + \pi_R I^A_t \tag{23}$$

This health status is an absorbing state and the magnitude of this set of agents is increased by untested asymptomatic agents who recover in every period.

The share of observed recovered evolves as follows:

$$R^O_{t+1} = R^O_t + \pi_R(P_t + I^S_t) \tag{24}$$

This health status is also an absorbing state and the magnitude of this set of agents increases as tested-positive and infected symptomatic agents recover.

The measure of population is given by the sum of these six groups. Note that the population size may varies because infected people die. The share of agents who has died by period $t + 1$ is given by

$$D_{t+1} = D_t + \pi_D I^S_t \tag{25}$$

The only two variables we have not yet defined are the probability of testing positive for newly infected agents, $\pi^T_{P,t}$, and untested asymptomatic agents, $\pi^T_{P,t}$. The characterization

of these probabilities is the object of the next section.

3 Contact Tracing and Testing

Health officials test subjects whose health status is unknown; that is, susceptible, asymptomatic infected, and unobserved recovered agents. In our model, an agent can be infected and remain asymptomatic throughout its entire infection. These agents are undiscovered spreaders who keep infecting susceptible agents until they recover. Infected agents are asymptomatic at first and if they are not caught by testing, they keep infecting other susceptible agents in the following periods. Tests do not reveal when a positive agent was infected or whether a negative agent is still susceptible to get infected or has recovered. Results can be false-negative.

Contact tracing is a testing strategy whose aim is to *ex-post* reconstruct as much as possible of the newly symptomatic cases' *infection chain*; i.e., the network of interactions that led the newly symptomatic case to become infected or to infect other agents. How much of the infection chain can be known by health officials defines the *efficiency of the contact tracing technology*. The following two features characterize the efficiency of the tracing technology: i) How many weeks back health officials can trace contacts of the newly symptomatic case; ii) how many rounds of tracing health officials can perform (e.g., second-round tracing means that health officials can trace the contacts of those individuals who interacted with the newly symptomatic cases and tested positive.)

We want to be conservative and, in this paper, we assume that health officials cannot perform multiple rounds of testing. All the tracing technology allows them to do is first-round tracing.[10] We consider two tracing technologies: a *basic technology* that allows health officials to trace only those contacts which occur in the current week and an *comprehensive technology* that allows them to trace contacts up to one week back. We will show that even these fairly rudimentary tracing technologies go a long way toward controlling the spread of the virus even when testing capacity is scant. That said, our methodology can be extended to consider very comprehensive contact tracing technology allowing health officials to trace contacts that happened far back in time and to conduct multiple rounds of contact tracing.

It is useful to resort to some graphical examples to illustrate how contact tracing works in the model. In Figure 1, agent A, which caught the virus in period $t-2$, infects agent B in period $t-1$. In the next period, agent A infects further two agents, which are denoted by C

[10] One practical challenge with multiple round testing is that this is very time-consuming. The second round can only be initiated after the receipt of the test results from the first round, which can take considerable time.

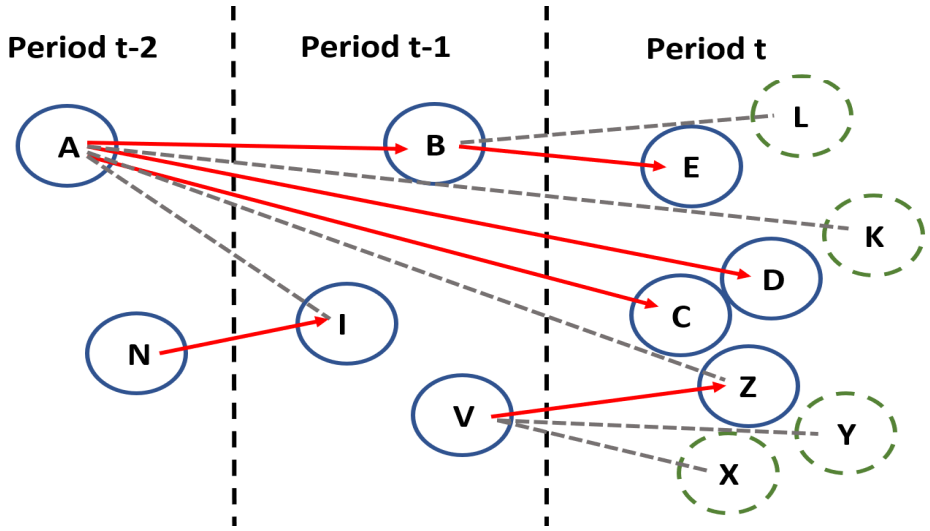

Figure 1: Example of an infection chain. The blue solid circle indicate an asymptomatic person. The green dashed circle are susceptible or recovered agents. The red lines describe an interaction that leads to an infection, while the grey lines describe an interaction that does not lead to an infection.

and D. At the same time, agents B also infects agent E. In period t agent A also met subject Z, who was however infected by subject V. The gray line connecting subject A and Z means that this was a non-infectious meeting. The other subjects, which are denoted with dashed green circles, are agents that were not infected by meeting with one of the asymptomatic infected subjects, which are denoted with blue solid circles.

Let's assume that subject A turns symptomatic in period t. The basic tracing technology would allow health officials to trace the newly infected subjects C, D, and Z. However, subjects B and E, who belongs to the same infection chain originated by the subject A, cannot be traced. If the comprehensive tracing technology is available, then subject B can also be traced. If, in addition to comprehensive contact tracing, second-round testing were feasible, then subject E could also be traced after subject B has tested positive. Furthermore, after subject Z has tested positive, subject V will also be traceable. The latter is an example of how contact tracing allows health officials to exploit a non-infectious link (i.e., the meeting between the newly symptomatic subject A and the newly infected subject Z) to jump on a different infection chain and going backward to reach the spreaders of this new infection chain; that is, subject V.

Now let's assume that subject B turns symptomatic in period t while subject A is still untested asymptomatic. In this case, the basic technology would allow health officials to discover subject E. By allowing to trace subject B's contacts in the earlier period $t-1$, the comprehensive tracing technology allows health officials to discover that subject A is an asymptomatic spreader. Note that the comprehensive tracing technology allows to uncover

who has infected the newly symptomatic case (*backward tracing*). The basic technology does not allow this backward tracing to happen as it takes at least one period for newly infected subjects to become symptomatic. Second-round testing would allow the comprehensive technology to discover subjects C, D and Z via subject A as well as all the subjects infected by subject A in period $t-1$. A third round of tracing would allow health officials to shed light on other infection chains by making subject V traceable via subjects A and Z.

It is worth emphasizing that in the previous case in which subject B becomes symptomatic we assumed that subject A has not recovered either in period $t-1$ or in period t. If subject A's health status is (unobserved) recovered in period t, the comprehensive technology would also fail to identify subject A, her infection chain (i.e., subject C and D in the example), and –in case of multiple round testing– the other infection chain linking subjects V and Z. This suggests that tracing contacts further back in time has decreasing returns in terms of how many infected asymptomatic health authorities can discover. The probability that subject A may have recovered in previous period explains why it is optimal for health officials to first use the available tests to test those contacts of the newly symptomatic subject B that occurred at time t. We will return to this important point later in the paper when we will talk about the Peking order in testing.

Testing Probabilities The probability of catching an untested asymptomatic infected agent depends on (i) the probability of tracing infected subjects, which varies depending on the subject being infected in period t (π_C^T) or earlier (π_C^A); (ii) the testing capacity in period t, Υ_t, relative to the number of people traceable E_t, which depend on the tracing technology; (iii) the accuracy of the testing technology, which in the paper is captured by the probability that tests deliver a false negative response (π_F).

Formally, we can write the probability of testing positive in a given period for subjects infected in period t ($i=T$) and for subject infected earlier than period t ($i=A$) as follows:

$$\pi_{P,t}^i = \pi_{C,t}^i \cdot \pi_{T,t} \cdot (1-\pi_F), \quad i \in \{T, A\} \tag{26}$$

where the probability $\pi_{T,t}$ denotes the probability of being tested conditional on being traced by the government. As we shall explain this probability depends on the testing capacity Υ_t, and the number of agents that are traceable E_t. This decomposition implies that a subject has to be traced before being tested.

Coordination Failure and the Collapse of the Testing System. Agents fail to realize that their consumption and labor decisions have externalities on the number of traceable subjects, E_t, health authorities have to test at the end of the period. First, they do not

appreciate that as they increase their consumption or labor, the overall amount of interactions in the economy will increase and, thereby, newly symptomatic agents will end up having more traceable contacts. Second, untested asymptomatic subjects fail to realize that by consuming or working more, more people will become infected, raising the number of newly symptomatic cases in every period.[11] A larger number of newly symptomatic cases enlarges the pool of subjects who met with them and are, thereby, traceable.

These externalities may lead the number of traceable contacts E_t to rise to the point at which the testing system collapses, with very severe consequences for the economy. When the number of traceable contacts largely exceeds the testing capacity, Υ_t, the probability for traceable people to be tested, $\pi_{T,t}$, falls and, with it, the probability for untested asymptomatic subjects to test positive, $\pi^i_{P,t}, i \in \{T, A\}$ in equation (26). Consequently, the number of asymptomatic spreaders starts increasing out of control and the spread of the virus accelerates. The economy contracts sharply as the heightened probability of becoming infected, τ_t, causes non-quarantined agents to want to reduce economic interactions so as to minimize the probability of catching the virus and dying.

It is worth noting that in overlooking the implications of their consumption and labor decisions on the number of traceable contacts, E_t, agents are not behaving irrationally. Rather, they take the aggregate probability of meeting an asymptomatic spreader as given when they optimally choose their consumption and labor schedules.[12]

As we shall show, contact tracing allows health authorities to quarantine more asymptomatic spreaders in every period for given testing capacity, Υ_t. Less spreaders on the streets today means less symptomatic cases whose contacts have to be tested tomorrow. By lowering the number of traceable subjects, a comprehensive contacts-tracing technology ensures that the testing system remains viable and thereby the probability for asymptomatic spreaders to test positive, $\pi^i_{P,t}, i \in \{T, A\}$ never fall precipitously. An alternative approach to ensure the viability of the testing system is to impose some form of restrictions on economic activities by raising the consumption tax, $\mu^L_{c,t}$), so as to curb the surge in the number of infections.

In the reminder of this section, we will study two contact tracing technologies: the basic contact tracing technology and the comprehensive contact tracing technology, The former allows health authorities to trace those contacts which occur in the current week. The comprehensive technology enables health authorities to trace contacts up to one week ago.

[11] These externalities would not be eliminated if these subjects knew to be asymptomatic spreaders. These externalities are removed only if the asymptomatic cases are tested and quarantined.

[12] There is another source of externalities in the model. Agents do not internalize that their consumption and labor decisions affect how many people will become infected in the economy as a whole and, hence, ultimately their probability of getting infected. Eichenbaum, Rebelo and Trabandt (2020*a*) study the implications of these externalities in great detail. In presence of a well-functioning testing system, contact tracing reduces the importance of these externalities.

3.1 Basic Contact Tracing Technology

The basic contact tracing technology allows health authorities to trace only those contacts which occur in the current week. It is useful to combine the binomial distributions in equation (2), (3), and (4) to obtain the probability for a susceptible individual to meet k-times with the set of asymptomatic subjects while consuming c_t^s, working n_t^s, or performing other activities.

$$f_t^S(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} f_{c,t}(i) f_{n,t}(j) f_{o,t}(k-i-j) \tag{27}$$

We want to derive the probability that *at least* one of the k interactions made by a susceptible individual at time t was with an asymptomatic subject who became symptomatic in the same period. We are interested in this type of interactions as they create a traceable contact. Conditional on meeting k asymptomatic subjects in period t, the probability that at least one of these subjects becomes symptomatic in the same period is $1-(1-\pi_{IS})^k$. Then the probability for a susceptible subject at the beginning of time t to be traced

$$\pi_{C,t}^S = \sum_{k=0}^{\varphi_C(c_t^s)+\varphi_N(n_t^s)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f_t^S(k) \tag{28}$$

We want to compute the probability for a newly infected subject to be traced, $\pi_{C,t}^T$. Note that newly infected subjects are susceptible at the beginning of the period and become infected in the period because they meet an untested asymptomatic individual. Therefore, the newly infected subject must have at least one interaction with an untested asymptomatic. To take this into account, we apply the Bayes theorem to the distribution of contacts that susceptible individuals had with untested asymptomatic people:

$$f_t^T(k) = \frac{f_t^S(k)\tilde{\tau}(k)}{\tau_t} \tag{29}$$

where $\tilde{\tau}(k) \equiv \left[1-(1-\tau)^k\right]$ is the probability to get at least one infectious contact out of k interactions and, recall that τ_t stands for the average probability for susceptible subjects to become infected in period t, which is defined in equation (6).

Following the same reasoning behind the probability in equation (28), we characterize the probability for a newly infected to be traced as

$$\pi_{C,t}^T = \sum_{k=0}^{\varphi_C(c_t^s)+\varphi_N(n_t^s)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f_t^T(k) \tag{30}$$

The final step is to compute the probability for an untested asymptomatic agent to be

traceable. As we already noticed, since untested asymptomatic agents ignore to be infected, they behave exactly as susceptible agents. Furthermore, becoming asymptomatic in an earlier period is an independent event from meeting a newly symptomatic subject at time t. It then follows that the probability for an untested asymptomatic agent to be traced is identical to the one for a susceptible agent at the beginning of the period, which is defined in equation (28). In symbols, $\pi^A_{C,t} = \pi^S_{C,t}$.

Testing with Basic Contact Tracing Technology The efficiency of contact tracing, which depends on the tracing technology, and test availability are perfect complements. Each traced contact cannot be tested if there are not sufficient tests available. Similarly, a test can only be used efficiently if the contacts are known.[13] The contact tracing technology endows the health officials with a list of contacts of newly symptomatic agents in period t. Health authorities look at the contacts with individuals whose health status is unknown (i.e., contacts with observed recovered individuals are discarded). We call this set of traceable individuals *the exposed*. The measure of this set is given by

$$E_t = \pi^S_{C,t} \cdot S_t + \pi^A_{C,t} \cdot (1 - \pi_{IS})\, I^A_t + \pi^R_{C,t} \cdot R^U_t \tag{31}$$

where $\pi^R_{C,t}$ denotes the probability of being traced for an unobserved recovered subject. We adjust the share of the untested asymptomatic by taking out those who have revealed symptoms ($\pi_{IS} I^A_t$) in period t as they are not part of the exposed subjects.

The probability for a susceptible agent and that for an untested asymptomatic agent to be traced were derived earlier. For the same reason, the probability for an unobserved recovered subject to be traced is also identical to the probability for susceptible and an untested asymptomatic subjects to be traced. In symbols, $\pi^R_{C,t} = \pi^S_{C,t} = \pi^A_{C,t}$.

The health authorities do not know the health status of susceptible, untested asymptomatic, and unobserved recovered individuals and hence they cannot tell these three types of subjects apart when it comes to deciding who to test. Therefore, the probability of testing a traceable contact does not depend on the contact's health status and is then defined as

$$\pi_{t,T} = \min\left(1, \frac{\Upsilon_t}{E_t}\right) \tag{32}$$

where the testing capacity $\Upsilon_t \geq 0$. We can plug equations (28) and (32) into equation (26) to evaluate the probability of testing positive for newly infected subjects, $\pi^T_{P,t}$. Plugging equations (30) and (32) into equation (26) allows us to compute the probability of testing

[13] For instance, Germany had in April more testing capacity than actually used tests, which shows that not necessarily all tests need to be used.

positive for subjects infected in earlier periods, $\pi^A_{P,t}$. The probabilities $\pi^A_{P,t}$ and $\pi^T_{P,t}$ can be used to pin down the dynamics of types in equations (20) and (21) for the basic contact tracing technology.

3.2 Comprehensive Contact Tracing Technology

With the comprehensive contact tracing technology, the government can also trace the contacts that occurred in period $t-1$ with subjects who become newly symptomatic at time t. The probabilities of being traced for the newly infected and the untetsted asymptomatic subjects via contacts they have established at time t are the same as those in the previous section. The objective of this section is to characterize the probabilities for newly infected and untested asymptomatic subjects to be traced based on contacts established at time $t-1$.

To derive these probabilities, it is useful to condition to three types of agents and to two types of links. The three types are as follows: (i) Type-A agents are asymptomatic subjects in period t infected earlier than $t-1$; (ii) Type-T agents are asymptomatic subjects in period t who became newly infected in period $t-1$; (iii) Type-S agents are subjects who became newly infected in period t. These letters are chosen to denote the health status of asymptomatic subject in period $t-1$: A for asymptomatic infected, T for newly infected, and S for susceptible. Note that the Type-A and Type-T agents have not been tested positive, or recovered, or developed symptoms before testing is performed in period t.

The two links are as follows: (i) A-links stand for those contacts that three type of subjects had with agents who became infected in a period earlier than $t-1$; (ii) T-links mean those contacts that the three subjects had with agents that become newly infected in period $t-1$. These letters denote the health status of the subjects with which the three types of agents have interacted with in period $t-1$: A for asymptomatic infected and T for newly infected. We care about these two types of links because they connect the three types of subjects to those agents who may become symptomatic in period t.[14]

Type-A agents: asymptomatic subjects in period t who were infected earlier than t-1. The probability of being traced at time t for type-A agents differs from the one for the other two types T and S in one important way. Since type-A subjects were already asymptomatic in period $t-1$, they may have infected susceptible individuals in period $t-1$ and these individuals may become symptomatic in period t. Creating their own infection chain raises the probability for type-A agents to be traced. Indeed, these additional traceable

[14]Recall that it takes at least one period for newly infected agents to develop symptoms. Thus, the probability of meeting in period $t-1$ with subjects who will then become newly infected in period t (Type-S link) does not affect the probability of being traced in period t.

links create the possibility of *backward tracing*. Recall the example, graphically illustrated in Figure 1, where agent B turns symptomatic in period t, making agent A, who infected agent B in period $t-1$, traceable. The probability for a type-A subject to have k T-type links in period $t-1$ can be written as the sum of binomials in equation (27)

$$f_{t-1}^{A,T}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} f_{c,t-1}^{A,T}(i) f_{n,t-1}^{A,T}(j) f_{o,t-1}^{A,T}(k-i-j) \tag{33}$$

where the first superscript of these probability distribution f denotes the agent's type –in this case A – and the second superscript the links' type –in this case T-links. The distributions on the right-hand-side are binomial distributions which are defined as follows:

$$f_{c,t-1}^{A,T}(k) \equiv \mathcal{B}\left(k, \varphi_c\left(c_{t-1}^{S}\right), \frac{[\tau + (1-\tau)\tau_{t-1}]C_{t-1}^{S}}{C_{t-1}}\right), \tag{34}$$

where the distribution regarding labor-based interactions, $f_{n,t-1}^{A,T}$, and that regarding non-economic interactions, $f_{o,t-1}^{A,T}$, are analogously defined.

The probability $[\tau + (1-\tau)\tau_{t-1}]\,\frac{C_t^S}{C_t}$ can be decomposed into two parts. The first part $\tau\frac{C_t^S}{C_t}$ captures the chance for the type-A agent to meet with a susceptible individual and to infect her. In this case, the asymptomatic subject has added one more case to her own infection link which could potentially make her traceable via backward tracing.[15] In the example illustrated in Figure 1, this first case corresponds to the infectious meeting between subject A and subject B.

The second part is the product of the probability of not infecting the susceptible subject $(1-\tau)$ times the probability that some other asymptomatic agents will infect the subject in period $t-1$ (i.e., the average probability τ_{t-1}). Note that in this second case, the type-A agent has a non-infectious meeting with an agent that will be infected by someone elseThis non-infectious meeting creates a traceable link for the type-A agent in period t. In the example illustrated in Figure 1, this second case corresponds to the meeting between the subject A and subject I in period $t-1$, This meeting is not infectious as subject I is actually infected by subject N in the same period.

While both events create a T-link for the A-type agent, in the first case only one event has to happen (the type-A agent infects the susceptible subject) whereas in the second case two events have to jointly happen (the type-A agent does not infect the susceptible individual and the susceptible individual becomes infected by meeting another agent). Thus, the first event is generally more likely than the second chain of events. In our empirical simulation,

[15] The probability τ is the probability of infecting the subject conditional on meeting a susceptible subject. See Assumption 4.

backward tracing raises the probability for a type-A agent to be traced considerably while the probability for a type-A agent to be traced via a non-infectious meeting with an agent that will later become symptomatic is quite tiny. We also call these non-infectious meetings random meetings.

Asymptomatic infected subjects in the periods earlier than $t-1$ has the following probability to have met k-times with other asymptomatic subjects who got infected in periods earlier than $t-1$:

$$f_{t-1}^{A,A}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} \mathcal{B}\left(i, \varphi_C(c_{t-1}^S), \frac{C_{t-1}^A}{C_{t-1}}\right) \mathcal{B}\left(j, \varphi_N(n_{t-1}^S), \frac{N_{t-1}^A}{N_{t-1}}\right) \mathcal{B}\left(k-i-j, \varphi_O, \frac{I_{t-1}^A}{Pop_{t-1}}\right) \tag{35}$$

Since A-links involve subjects who are already infected, all meetings are non-infectious. Hence, this probability distribution does not require us to distinguish between infectious and non-infectious meetings.

Type-T agents: asymptomatic subjects in period t who were infected in period t-1. The probability for type-T agents to have k T-links in period $t-1$ is

$$f_{t-1}^{T,T}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} \mathcal{B}\left(i, \varphi_C(c_{t-1}^S), \frac{c_{t-1}^S T_{t-1}}{C_{t-1}}\right) \mathcal{B}\left(j, \varphi_N(n_{t-1}^S), \frac{n_{t-1}^S T_{t-1}}{N_{t-1}}\right) \times \mathcal{B}\left(k-i-j, \varphi_O, \frac{T_{t-1}}{Pop_{t-1}}\right) \tag{36}$$

where $c_{t-1}^S T_{t-1}$ and $n_{t-1}^S N_{t-1}$ denote the total consumption and labor of the newly infected subjects in period $t-1$.

The probability for type-T agents to have k A-links in period $t-1$ can be constructed from the probability for type-A agents to have k A-links by applying the Bayes theorem

$$f_{t-1}^{T,A}(k) = \frac{f_{t-1}^{A,A}(k)\,\tilde{\tau}(k)}{\tau_{t-1}} \tag{37}$$

where the first distribution at the numerator is defined in equation (35) and now is corrected by applying the Bayes theorem. This correction is required by the fact that, unlike type-A agents, type-T agents must have met at least one untested asymptomatic in period $t-1$; that is, the individual who has infected the type-T agent.

Type-S agents: newly infected subjects in period t. Since, unlike type-A agents, who can expand their own infection chain in period $t-1$, type-S and type-T agents cannot

infect anyone in that period, they will have the same probability to have k t-links in period $t-1$: $f_{t-1}^{S,T} = f_{t-1}^{T,T}$.

The probability for type-S agents to have k A-links in period $t-1$ can be constructed starting from the probability for type-A agents to have k A-links in the same period. However, we need to take into account that for type-S agents none of these meetings with untested asymptomatic subjects triggered an infection. For this, we use again the Bayes theorem

$$f_{t-1}^{S,A}(k) = \frac{f_{t-1}^{A,A}(k)\left(1-\tilde{\tau}(k)\right)}{1-\tau_{t-1}} \tag{38}$$

Conditioning on Type-A and Type-T remaining asymptomatic infected through period t. Since tracing is conducted in period t, the probability distributions for type-A and type-T subjects have to be conditioned on the event that these subjects have remained untested asymptomatic through period t. This event depends on whether the subjects in question have been tested positive at the end of period $t-1$.[16]

The probability of testing positive depends on the probability of being traced, which in turn depends on the number of contacts the subjects have established with asymptomatic subjects in period $t-1$.[17] Therefore, knowing that an asymptomatic subjects have not tested positive at the end of period $t-1$ affects the distribution of their contacts in period $t-1$ with asymptomatic infected people that we have derived earlier. We rely on the Bayes theorem to take care of this time adjustment and obtain the conditional distributions $f_{t-1|t}^{A,A}(k)$ and $f_{t-1|t}^{T,A}(k)$ for type-A agents and type-T agents, respectively. The derivations of these distributions are shown in Appendix A.

All other distributions do not need to be adjusted.[18] It is convenient to write: $f_{t-1|t}^{A,T}(k) = f_{t-1}^{A,T}(k)$, $f_{t-1|t}^{T,T}(k) = f_{t-1}^{T,T}(k)$, $f_{t-1|t}^{S,T}(k) = f_{t-1}^{S,T}(k)$, and $f_{t-1|t}^{S,A}(k) = f_{t-1}^{S,A}(k)$.

Active Links Some of the A-links are not relevant for traceability and testing in period t because infected asymptomatic subjects may become symptomatic or recover or test positive in period $t-1$. T-links could also become non-relevant for traceability and testing in period

[16] This also depends on whether the asymptomatic subject recover or develop symptoms in period $t-1$ or in period t but since the probability of these events does not depend on the number of contacts with asymptomatic subjects in period $t-1$, we do not need to condition the probability distributions derived earlier on these events.

[17] Contacts with the newly infected do not matter as they cannot turn symptomatic in period $t-1$.

[18] The distributions $f_{t-1|t}^{T,T}(k)$ and $f_{t-1|t}^{A,T}(k)$ do not need to be adjusted. The reasons is that meeting with newly infected people in period $t-1$ does not make type-T and type-A agents traceable in period $t-1$ because it takes at least one period for newly infected people to become symptomatic. Testing type-S agents in period $t-1$ does not affect their probabilities of having k T-links or A-links as the outcome of these tests is negative (we do not allow for false positive in test outcomes).

t because some newly infected agents test positive at the end of period $t-1$. Therefore, it is convenient to distinguish between total links (or simply links) and active links, which are those links with infected people who may still reveal symptoms in period t. making the subjects traceable in that period.

Let us start considering the T -links first. The probability that out of k T-links $\underline{k}$ of them will be still active in period t is given by the following binomial distribution:

$$g_{t-1}^{i,T}(\underline{k}_{t-1}|k_{t-1}) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}, \left(1-\pi_{P,t-1}^{T}(i)\right)\right). \tag{39}$$

where the probability of success (i.e., the link remains active) is the probability for the newly infected subjects met by the type A, or type-T, or type-S agents of not testing positive at the end of period $t-1$; that is, $1-\pi_{P,t-1}^{T}(i)$, for each type of agent $i \in \{A,T,S\}$.

Note that this probability depends on the type i of the agent establishing the contact with newly infected agents.. Let us start considering the case of type-A agents. Compared to the probability for an average newly infected agent to test positive in period $t-1$ ($\pi_{P,t-1}^{T}$), the probability of the newly infected agent of the T-link ($\pi_{P,t-1}^{T}(A)$) is lower (for given number of contacts k) as at least one of the untested asymptomatic agents she interacted with in period $t-1$ has not become symptomatic. This is the interaction with the Type-A agent, who does not develop symptoms in period $t-1$ because, by definition, she remains untested asymptomatic through period t. This adjustment is not needed when we compute this probability for type-T and type-S agents as their T-links are never infectious. Hence, $\pi_{P,t-1}^{T}(i) = \pi_{P,t-1}^{T},\ \ i \in \{T,S\}$.

The final step is then to combine this distribution with the appropriate distribution $f_{t-1|t}^{i,j}(k_{t-1})$ derived in the previous section to obtain the marginalized probability distribution of *active* T-links for each type as follows:

$$g_{t-1}^{i,T}(\underline{k}_{t-1}) = \sum_{k=0}^{\varphi_C(c_{t-1}^{s})+\varphi_N(n_{t-1}^{s})+\varphi_o} g_{t-1}^{i,T}(\underline{k}_{t-1}|k) f_{t-1|t}^{i,j}(k), \quad i \in \{A,T,S\} \tag{40}$$

A detailed derivation of the probability that a T-link will become inactive (i.e., no longer relevant for contact tracing), $\pi_{P,t-1}^{T}(i)$, for the three types $i \in \{A,T,S\}$ is in Appendix B.

As far as the active A-links, it is first important to realize that, unlike T-links, A-links can also become inactive as infected asymptomatic subjects may become symptomatic or may recover in period $t-1$. Another difference with T-links is that the probability that the A-link will remain active in period t depends on whether the type-A, or type-T, or type-S individual is traceable at time $t-1$. This is because if Type-A, Type-T, or Type-S agent is traceable in period $t-1$, then at least one of her A-links must have turned symptomatic in that period.

In this case, the probability for the A-link to remain active is lower because it could have been this very A-link to have made the Type-A, or Type-T, or Type-S agent traceable.[19] The derivation of the distribution of the active A-links $g^{i,A}_{t-1}(\underline{k}_{t-1})$ for $i \in \{A, T, S\}$ is tedious and thereby we refer the interested reader to Appendix C.

Tracing Probabilities. It is convenient to aggregate the distribution of having $\underline{k}$ active T-links $g^{i,T}$ and that of having $\underline{k}$ active A-link as follows

$$g^{i}_{t-1}(\underline{k}_{t-1}) = \sum_{j=1}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} g^{i,T}_{t-1}(j) g^{i,A}_{t-1}(\underline{k}_{t-1} - j), \quad i \in \{A, T, S\} \tag{41}$$

We take the same step shown in equation (30) to compute the probability for each type (type-A, type-T, and type-S) to be traceable due to one of their $t-1$ contacts

$$\pi^{1,i}_{C,t} = \sum_{\underline{k}=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} \left[1 - (1 - \pi_{IS})^{\underline{k}}\right] g^{i}_{t-1}(\underline{k}), \quad i \in \{A, T, S\} \tag{42}$$

Testing Probabilities. We use the decomposition in equation (26) to define the probability of being tested positive at time t through contacts established in the previous period

$$\pi^{j,i}_{P,t} = \pi^{j,i}_{C,t} \cdot \pi^{j}_{t,T} \cdot (1 - \pi_F), \quad i \in \{A, T, S\} \quad j \in \{0, 1\}, \tag{43}$$

where j denotes the period $t-j$ the contacts relevant for tracing were established. So we combine the probability of being traced, defined in equation (42), with the probability of testing positive which depends on the ratio of the test availability at time t, i.e., Υ_t, and the number of subjects who were exposed either in period $t-1$ or in period t to people turned symptomatic in period t, and the probability of false negative (π_F). The share of agents exposed to infected subjects showing symptoms in period t is denoted by E^0_t and is defined exactly as E_t in equation (31). We denote the subjects who in period $t-1$ has met subjects who became symptomatic in period t, as E^1_t, which is formally defined in Appendix D.

Tests are administered following a Peking order: First government uses all the available tests to check current period's contacts and if any tests are left, they are used to test previous period's contacts. We can show that this Peking order is optimal because the probability of finding an infected agents by testing one random contact that occurred in the current period

[19] Since it takes at least one period for the newly infected to become symptomatic, this scenario and the ensuing adjustment to the probability distribution of active links do not apply to the T-links.

is higher than testing one random contact that happened in the previous period. This is because some subjects who were untested asymptomatic in the previous period may have recovered before testing is performed in the current period.

The probability of being tested conditional on being traceable in period t is denoted by $\pi^0_{t,T}$ and defined in equation (32). Given the Peking order the probability of being tested conditional on being traceable in period $t-1$ is given by

$$\pi^1_{T,t} = \min\left(1, \frac{\max\left(0, \Upsilon_t - E^0_t\right)}{E^1_t}\right) \tag{44}$$

Note that the probability of testing positive defined in equation (43) is conditioned on the type of the agents in period $t-1$ (i.e., Type-A, Type-T, and Type-S). Recall that what we are ultimately interested in is to pin down the dynamics of types in equations (20) and (21), which requires us to know the *average* the probability for an untested asymptomatic subject to test positive in period t and average probability for a newly infected guys to test positive in period t.

The average probability for an untested asymptomatic subject in period t to test positive in the same period under comprehensive contact tracing is

$$\begin{aligned}\pi^A_{P,t} = &\frac{I^A_{t-1}\left(1-\pi_{IS}-\pi_R\right)\left(1-\pi^A_{P,t-1}\right)}{I^A_t}\cdot\left[\pi^{0,A}_{P,t}+\left(1-\pi^{0,A}_{C,t}\right)\cdot\pi^{1,A}_{P,t}\right] \\ &+\frac{T_{t-1}\left(1-\pi^T_{P,t-1}\right)}{I^A_t}\cdot\left[\pi^{0,A}_{P,t}+\left(1-\pi^{0,A}_{C,t}\right)\cdot\pi^{1,T}_{P,t}\right]\end{aligned} \tag{45}$$

where the first expression within square brackets denotes the probability for a type-A agent to test positive in period t and the expression within the second square bracket is the probability for a type-T subject to test positive in period t.[20] The two bits outside the square brackets re-weight the share of type-A and type-T with respect to the amount of untested asymptomatic cases in period t. This adjustment is needed as the transitions in equation (20) is expressed in terms of the size of the untested asymptomatic subjects at time t.

The average probability for a newly infected subject to test positive in period t under the comprehensive contact tracing technology is given by

$$\pi^T_{P,t} = \pi^{0,T}_{P,t} + \left(1-\pi^{0,T}_{C,t}\right)\cdot\pi^{1,S}_{P,t}. \tag{46}$$

Note that the newly asymptomatic subjects in period t traceable via contacts they established

[20]It should be noted that these probabilities for type-A and type-T to test positive in t reflect the Peking order: If an agent is traced via her time-t contacts, she will not be tested via her time-$(t-1)$ contacts.

in period $t-1$ must have been susceptible in period $t-1$ (Type-S agents). At last, the probabilities in equations (45) and (46) are used to pin down the dynamics of types in equations (20) and (21).

4 Model Solution and Calibration

We use the model to study the response of epidemiological and economic variables following a surprise shock that initially infects a tiny share of the population. To this end, we solve the model iteratively based on a numerical root finder that computes the sequence of policy functions and the evolution of the measure of agents types for a given number of periods. This computation is performed for a given sequence of taxes and for a given initial amount of asymptomatic and symptomatic agents infected by the shock. A key feature of this algorithm is to keep track of the distributions of interactions analyzed in Section 3. The details are in Appendix F.

The probability of getting infected τ_t as a function of consumption and labor decisions enters the decision problem of the susceptible, untested asymptomatic, and unobserved recovered agents (see Section 2.3). This probability, which is defined in equation (6), depends on two step functions $\varphi_c(c_t^s)$ and $\varphi^n(n_t^s)$ that introduce ridges and cliffs in the value function V_t^s of the agents, making the solution to the optimization problem very challenging. To improve the speed and the reliability of the solution algorithm, it is convenient to approximate equation (6) as follows:

$$\tau_t \approx \Xi \left[\varphi_c \cdot c_t^S \left(\frac{C_t^A}{C_t} \right) + \varphi_n \cdot n_t^S \left(\frac{N_t^A}{N_t} \right) + \varphi_O \left(\frac{A_t}{Pop_t} \right) \right] \tag{47}$$

where the coefficient $\Xi \equiv -\ln(1-\tau)(1-\tau)^{\bar{k}_c+\bar{k}_n+\bar{k}_o}$, with $(\bar{k}_c, \bar{k}_n, \bar{k}_o)$ denote the average number of interactions at steady state. In Appendix G, we show the steps taken to approximate τ_t. This specification of the expected probability of becoming infected conditional on consuming c_t^S and working n_t^S is smooth and continuous and thereby greatly simplifies the task of solving for the optimal consumption and labor decisions of agents, described in Section 2.3. We also use this approximation to compute the dynamics of types shown in Section 2.9. To reconstruct the history of interactions needed for pinning down the probability being tested in Section 3, we use the exact definition of the rate τ_t in equation (6).

The approximated infection rate τ_t in equation (47) is isomorphic to the rate used in other leading macro-epidemiological models, such as the one introduced by Eichenbaum, Rebelo and Trabandt (2020*a*). Since the infection rate in equation (47) stems from the choice of modeling economic interactions as binomial trials (Assumptions 1-4), our paper provides

Table 1: Calibration

Parameters	Sign	Value	Target / Source
(a) Economic parameters			
Discount factor	β	$0.96^{1/52}$	Value of life
Labor disutility	θ	0.13%	Weekly working hours of 28
Productivity	A	39.84	Yearly income 58,000$
Frisch labor elasticity	φ	0.5	Literature
(b) Epidemiological parameters			
Interaction via consumption	φ_C	0.99%	Consumption share in total interactions = 1/3
Interaction via labor	φ_N	0.39	Labor share in interactions = 1/3
Interaction independently	φ_O	10	Basic reproduction number $R_0 = 2$
Probability of infection	τ	5%	World Health Organization (2020)
Recovery rate	π_R	7/18	Average recovery rate = 18 days
Symptomatic rate	π_{IS}	7/18	Share of symptomatic cases = 50%
Mortality rate	π_D	0.6%	Infection fatality rate = 0.3%
False negative outcome	π_F	0	False positive probability = 0
Quarantine policy	μ^Q	1	Consumption and labor less 30% in quarantine
Productivity symptomatic	ϕ	0.8	Eichenbaum, Rebelo and Trabandt (2020a)
Lockdown effect in quarantine	α	0	No additional impact besides quarantine
Initial infection	ϵ	0.1%	Infections March 16 2020 for USA

theoretical underpinnings to the infection rate used in other macro-epidemiological models.

Calibration The calibrated parameters of the model are summarized in Table 1. The economic parameters are calibrated based on Eichenbaum, Rebelo and Trabandt (2020a). We set the weekly discount factor to $0.96^{1/52}$ to match the value of a statistical life of 9.3 million 2019 US dollars.[21] Productivity, A, is set to match a yearly income of $58,000. The scale parameter of labor disutility, θ, is calibrated so that agents work on average 28 hours per week. The Frisch labor elasticity φ is 0.5 as standard.

A key epidemiological parameter is τ, which is the probability that one interaction with an infected results in an infection (see Assumption 4). We set this parameter to 5% based on evidence from the World Health Organization (2020).[22] The parameters φ_C, φ_N, φ_O determine the number of interactions required to support levels of individual consumption c_t^S, labor n_t^S, and other non-economic activities, respectively. We set the parameters φ_C and

[21] The present discounted value of a life in current consumption units is $Vu_c = \frac{1}{1-\beta}u(c,n)AN$, where V is the discounted value and u_c is the marginal utility of consumption.

[22] This WHO report analyses the probability of an infection for an individual that had close contact with an individual tested positively with COVID-19 is between 1% and 5%. The study had identified around 40,000 people as close contacts and was conducted in Mid February in three Chinese cities with very active contact tracing.

φ_N so that consumption- and labor-based transmissions of the virus account for a share of 1/3 each, when consumption and labor decisions are fixed to the pre-pandemic level. These targets are chosen consistently with the influenza study by Ferguson et al. (2006).[23] The parameter φ_O is set to target the basic reproduction number R_0, which is the total number of infections caused by one infected person (with measure zero) in his or her lifetime in a population where everybody is susceptible and no containment measures (including testing) are taken.[24] We set the basic reproduction number to 2 in line with the evidence about the early transmission of COVID-19.[25] The calibration implies a total amount of 30 interactions in the pre-epidemic economy, which is broadly in line with surveillance data from infected agents.[26]

In line with evidence from the World Health Organization (2020), we choose that an agent recovers on average after 18 days, which implies $\pi_R = 7/18$.[27] We calibrate the probability of developing symptoms, π_{IS}, so that 50% of infected agents develop symptoms at some point of the pandemic crisis, which is in line with the symptomatic rate estimated by Baqaee et al. (2020).[28] A key metric in parameterizing a SIR model is the infection fatality rate, which measures the amount of deaths relative to all infectious cases. The mortality rate π_D is the infection fatality rate divided by the share of symptomatic agents. This rate is calibrated to target an infection fatality rate of 0.3% based on Hortaçsu, Liu and Schwieg (2020), who adjust the fatility rate to take into account unreported infections.[29]

In the model, symptomatic agents are subject to a labor productivity penalty, ϕ. We

[23]Eichenbaum, Rebelo and Trabandt (2020*a*) provide an alternative interpretation of the same influenza study and argue that labor and consumption interactions are only responsible for 1/6 each. While targeting this lower number would not change significantly our main results, it implies that a plausible lockdown in our model would fail to push the effective reproduction number below one, which is at odds with the evidence shown by Wang et al. (2020).

[24]In our model, the number is defined as $R_0 = \sum_{j=0}^{\infty} \left[\tau_1 (1 - \pi_r - \pi_D)^j\right] = \frac{\tau_1}{\pi_r + \pi_D}$.

[25]For instance, Li et al. (2020) find a basic reproduction number of 2.2 based on the first 425 confirmed patients in Wuhan (China), and Zhang et al. (2020) estimate the reproduction number to be around 2.3 using data based on the Diamond Princess cruise ship in Feburary, where a Covid-19 outbreak occurred.

[26]For the first nine cases in the US, Burke et al. (2020) find that an infected person had up to 45 contacts. Pung et al. (2020) show that a a Covid-19 infected person requires the quarantine of 12 contacts in Singapore in February.

[27]The WHO documents an average recovery rate of 2 weeks for mild cases and 3 to 6 weeks for severe cases.

[28]There is mixed evidence about this rate. Based on a population screening in Iceland, Gudbjartsson et al. (2020) find that 57% of the positive-tested cases report symptoms. However, almost 30% of negatively tested individuals also report symptoms in the same study. Poletti et al. (2020) find that 74% of positively tested contacts of indexed COVID-19 cases did not develop symtoms for individuals with an age below 60. Nishiura et al. (2020*a*) suggest a 69% infection rate based on evacuation flights of Japenese passengers data from China.

[29]This value is supported by further studies such as Nishiura et al. (2020*b*), who find range of 0.3% to 0.6% with Japanese data and Streeck et al. (2020) with an estimate 0.36% based on German data. Fernández-Villaverde and Jones (2020) estimate a higher mortality rate of 1%.

calibrate the penalty $\phi = 0.8$ based on Eichenbaum, Rebelo and Trabandt (2020a). Furthermore, infected symptomatic agents and tested-positive agents are quarantined, which is modeled as a tax on consumption, μ_c^Q. This tax implies that at steady state the consumption and labor of a positively-tested (asymptomatic) agent is lowered than those of a non-quarantined (non-recovered) agents by approximately 30%. We assume that quarantined agents are not affected by the lockdown, that is $\alpha = 0$. We set the probability of a false negative outcome π_F to zero. The initial share of infected agents ϵ is set to 0.1% and is divided evenly between asymptomatic and symptomatic agents. Following Berger et al. (2020), this can be interpreted as the amount of infections adjusted for unreported cases on March 16, 2020.

5 Quantitative Analysis of Contact Tracing

In this section, we evaluate the efficacy of the two technologies of contact tracing (basic and comprehensive) under different scenarios regarding testing capacity (Υ). The testing infrastructure or testing system is made of two parts: (i) the efficiency of the contact tracing technology and (ii) the testing capacity (Υ_t).

Before moving to the scenario analysis, it is useful to define an epidemiological variable that gauges the speed at which the virus is spreading: the effective reproduction number. This number captures how many susceptible people an untested asymptomatic agent infects on average during the spell of her illness.

This number is affected by the testing infrastructure, the amount of economic interactions that depend on non-quarantined agents' decision to consume and work, and the stringency of containment policies (lockdowns) put in place by policymakers. An effective reproduction number above 1 indicates a situation in which the virus is infecting more and more people over time, while a number below 1 signifies that the virus is retracting. The effective reproductive number in our model is defined as

$$\begin{aligned} R_t^E &= (1-\pi_{t-1}^T)\big[\tau_t + (1-\pi_{IS}-\pi_R)\left(1-\pi_t^A\right)\tau_{t+1} + \\ &\qquad (1-\pi_{IS}-\pi_R)^2\left(1-\pi_t^A\right)(1-\pi_{t+1}^A)\tau_{t+2} + \dots\big] \\ &= (1-\pi_{t-1}^T)\sum_{j=0}^{\infty}\left(\tau_{t+j}(1-\pi_{IS}-\pi_R)^j \Pi_{k=0}^j\left(1-\pi_{t+k}^A\right)\right) \end{aligned} \tag{48}$$

The effective reproduction number conflates current and future probabilities for non-quarantined infected agents to be caught. The testing infrastructure influences the effective reproduction number through the probability for a newly infected subject and for an

untested asymptomatic subject to test positive at the end of period t; that is, π^T_{t-1} and π^A_t, respectively. Lockdowns lower the effective reproduction number primarily by reducing the infection rate, τ_t.

It is important to note that the reproduction number is more sensitive to changes in the probability for a newly infected agent to test positive, π^T_{t-1}, than to changes in the future probability for an untested asymptomatic agent to test positive, π^A_{t+k}. The reason is that asymptomatic agents may turn symptomatic or recover in every future period and, when she will do that, she will no longer infect other people. The transience of the status of being asymptomatic, which is captured by the term $(1 - \pi_{IS} - \pi_R)$ in equation (48), implies that increasing the probability of catching asymptomatic agents further in the future has decreasing effects on the effective reproduction number. This suggests that the efficacy of a testing strategy critically hinges on delivering a high probability of capturing newly infected people (i.e., π^T_t close to 1). This is an important point that helps explain some of the results that will be presented in this section.

5.1 Contact Tracing with Unlimited Tests

It is interesting to start with a scenario in which tests are always sufficient to cover all the contacts of newly symptomatic subjects. This scenario sheds light on the efficacy of the two contact-tracing technologies in the most favorable environment where testing capacity is never binding. In addition, this exercise will give us a sense of how many tests would be needed to make contact tracing work at its best.

In this scenario, we also consider random testing as an alternative to contact tracing, which has been advocated by Romer (2020) among other scholars.[30] It is assumed that random testing is run on a testing capacity of 20% of the initial population over the entire simulation horizon. This implies a daily testing capacity of close to 10 million daily tests. To put this number in perspective, in the U.S. the daily testing capacity was around 1 million tests per day in September 2020. We also consider the case in which no testing is performed.

Figure 2 shows the evolution of the key epidemiological, economic, and testing variables.[31] Beginning with the case in which no one is tested (the yellow dashed-dotted line), the pandemic spreads very fast and causes many people to become infected. The pandemic crises fades away when 60% of the population becomes infected and heard immunity is reached. In total 0.4% of the population dies because of the pandemic. In response to the surge in the probability of getting infected, agents reduce their interactions by drastically

[30] How we formalize random testing in our model is explained in Appendix E.

[31] More variables are plotted in Appendix H.

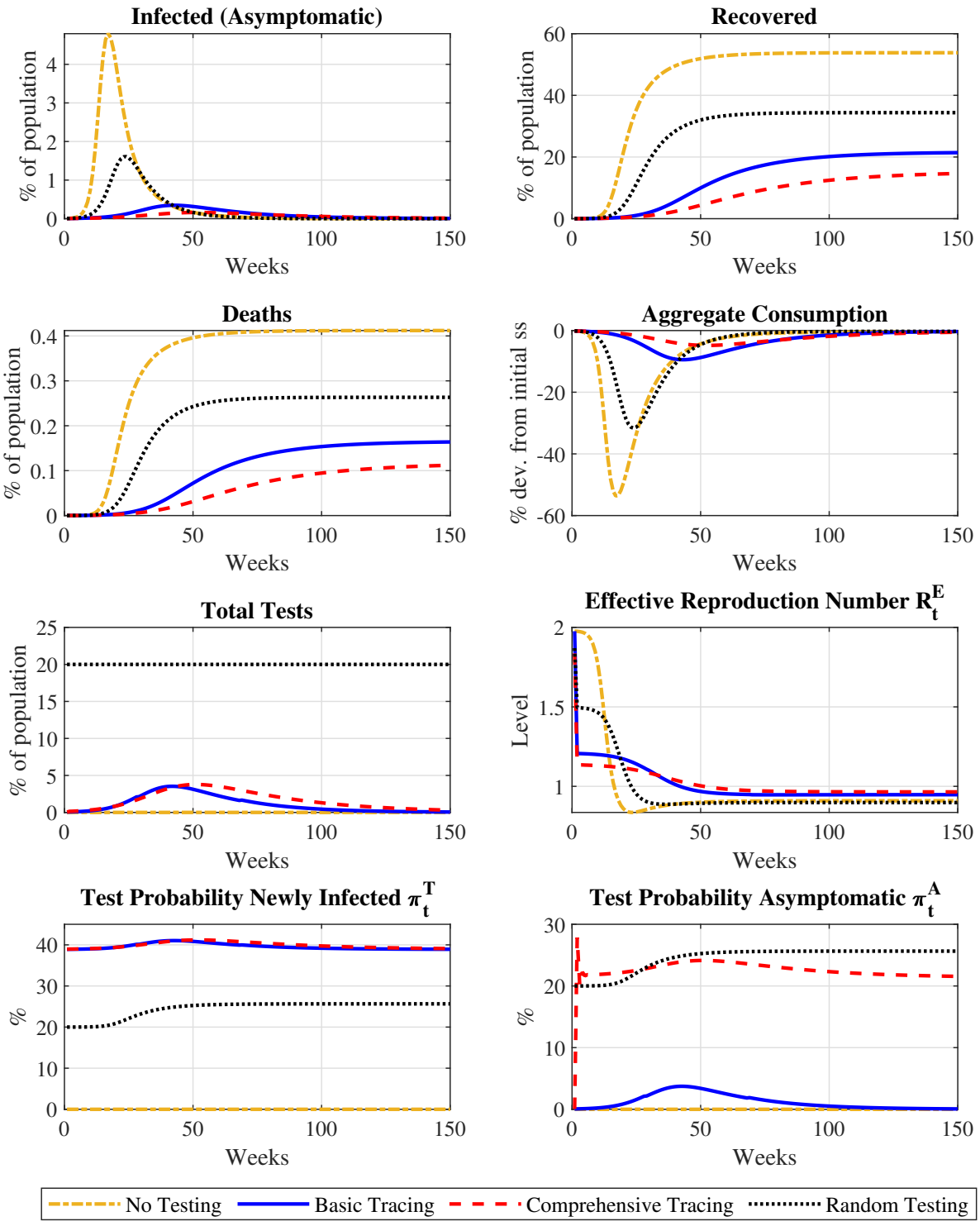

Figure 2: Comparison of different testing strategies with unconstrained number of tests for contact tracing: No testing (blue solid), basic tracing (red dashed) corresponds to current week contact tracing, comprehensive tracing (green dash-dotted) corresponds to current and previous week contact tracing and random testing (black dotted) has an amount tests available for 20% of the entire population each week.

lowering consumption and labor. As a consequence, the economy goes through a very deep recession with aggregate consumption contracting by up to 50%.

The introduction of the basic contact-tracing technology hugely improves outcomes by slowing down the spread of the virus and by reducing the death toll by more than 50%. See the solid blue line in Figure 2. As the virus spreads less quickly (lower effective reproductive number), the chance of getting infected are relatively contained, leading agents to reduce their consumption and labor less dramatically compared to the case of no testing. The reproduction number quickly drops and eventually falls below 1. As a result, heard immunity is reached with around 20% of infected agents –three times less than the share of infected needed in the case of no testing. The death toll is less than half than that under no testing.

While the comprehensive contact-tracing technology (the red dashed line in Figure 2) further mitigates the severe consequences of the pandemic crisis, this improvement is only marginal relative to what is achieved by the basic tracing technology. Both tracing technologies require to test at most 4% of the population in a week, which is substantially less than the number of tests we assume for random testing.

Note that the basic testing technology requires to perform more tests at the early stages of the pandemic relative to the comprehensive tracing technology. While this result may seem odd at first, it is important to realize that the number of tests performed is endogenous since it is determined by the number of traceable subjects, E_t. The number of traceable subjects grows with the speed at which the virus is spreading (the effective reproduction number) which is, in turn, affected by the efficiency of the testing technology. As already noticed, the basic technology is less effective than the comprehensive technology in detecting untested asymptomatic subjects because it does not allow backward tracing. Indeed, in the lower right panel, the share of untested asymptomatic subjects detected in every period by the basic tracing technology is very low compared to that allowed by the comprehensive technology. As a result, in the simulation the effective reproduction number is initially higher in the case of the basic contact-tracing technology, which justifies a bigger number of exposed subjects and hence more tests at the early stages of the pandemic.

Even though random testing (the black dotted line in Figure 2) is assumed to have an implausibly large testing capacity, it proves to be fairly ineffective in mitigating the outcomes of the pandemic. Even if 10 million people could be randomly tested every day, the pandemic would lead to a severe contraction and would kill 0.28% of Americans –more than twice as many the deaths under the comprehensive contact-tracing technology.

What explains the spectacular failure of random testing? To get an answer to that, one should look at the two bottom graphs of Figure 2, which show the share of newly infected

asymptomatic subjects, T_t, (left plot) and the share of untested asymptomatic subjects, IA_t, (right plot) who are detected and quarantined in every period under random testing and under the two tracing technologies. Even though many more tests are performed, random testing can detect only half of the newly infected subjects in every period. Random testing is quite effective in capturing untested asymptomatic subjects. Even so, random testing fails to reduce the effective reproduction number, underscoring the importance of detecting and quarantine the newly infected cases to attain a successful containment of the virus. This last intuition is reinforced by observing that even though the basic contact-tracing technology largely fails to detect untested asymptomatic subjects, it fares relatively well in detecting the newly infected agents.

That the probability of catching the newly infected asymptomatic subjects turns out to be key to control the pandemic should not come as a surprise. We already noted that the reproduction number defined in equation (48) is more sensitive to changes in the probability for newly infected agents to test positive, π_t^T, than to changes in the probability for untested asymptomatic subjects to test positive, π_t^A.

Random testing would have no effect on the severity of the pandemic recession and on the death toll whatsoever if testing capacity would have been calibrated to 4% of the population in every period (i.e., the upper bound of tests performed under the two tracing technologies.) By neglecting the existence of an infection chain connecting the newly symptomatic subjects with the subjects they have infected in the current period, random testing seems to be a blunt tool to stem the spread of the virus.

5.2 Contact Tracing with Limited Tests

In the previous section, we showed that the basic tracing technology does a great job in controlling the spread of the virus. The comprehensive tracing technology improves outcomes only marginally. However, the basic tracing technology calls for a quick increase in the number of tests performed at the early stages of the pandemic. This increase in the number of tests is needed to compensate the poor performance of this technology in catching the untested asymptomatic subjects, as reflected in the low value of π_t^A in the lower right plot of Figure 2. As we will see, if health authorities cannot scale up their testing ability sufficiently quickly, the basic tracing technology fails to contain the pandemic. In this section, we show that this is the case when the testing capacity, Υ_t, is calibrated to the amount of tests performed in the U.S. from March 16, 2020 through October 4, 2020.

The U.S. health authorities had only a daily capacity of 30,000 tests available at the onset of the pandemic crisis. This capacity then has been increased linearly up to 1 million

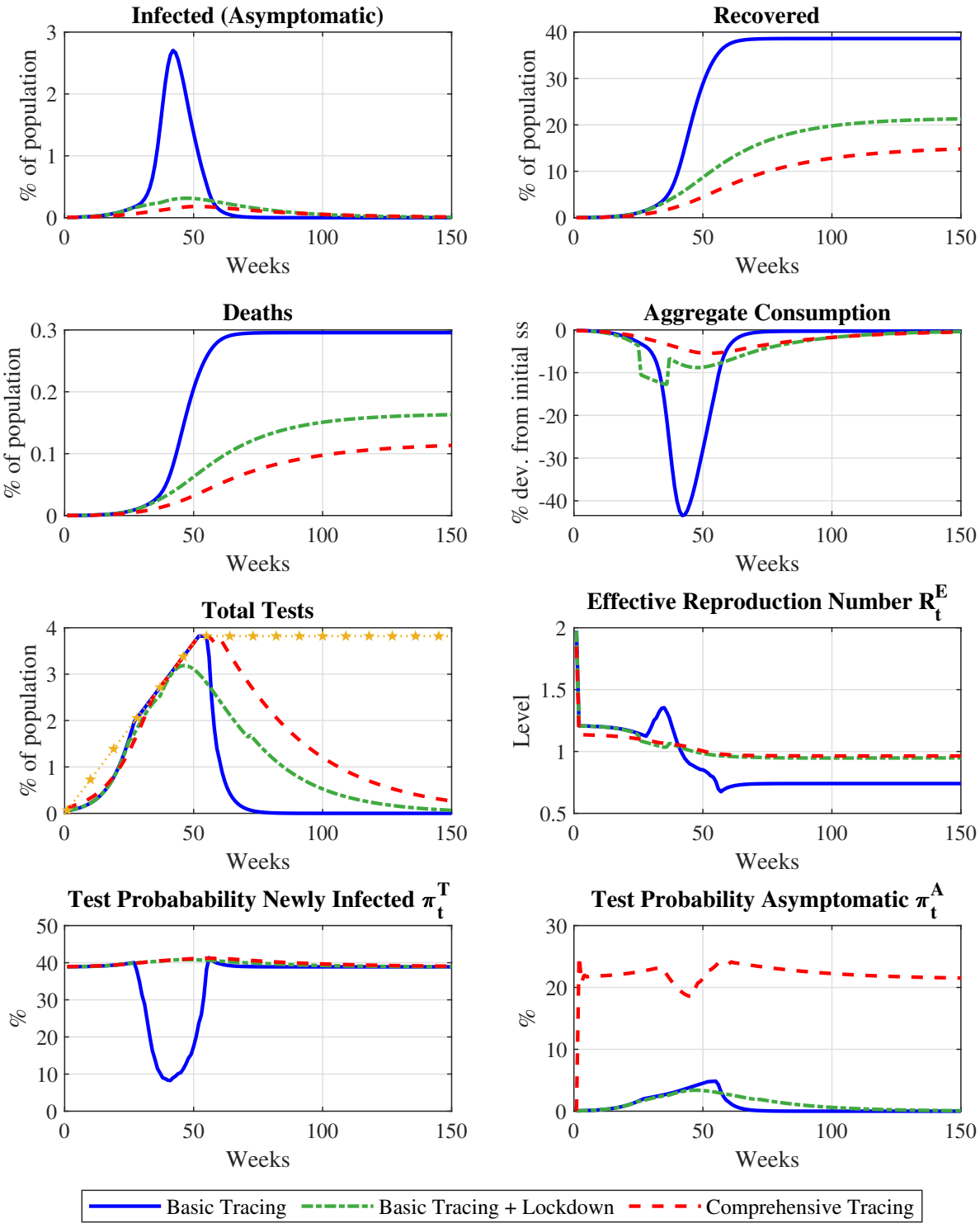

Figure 3: Comparison of different testing strategies with limited tests: Comprehensive tracing (blue solid line) is previous and current week tracing, basic tracing (red dashed line) is current week tracing and in the green dash-dotted basic tracing is combined with a 1 year lockdown. In the fifth plot, the yellow starred line shows the testing capacity Υ_t.

test 28 weeks later.[32] Afterwards, the testing capacity is assumed to be increased at a steady pace until week 52, after which, it will no longer vary.

Looking at the third left plot in Figure 3, the basic contact-tracing technology (blue solid line) requires testing to accelerate after period 10 to compensate for its inability of catching untested asymptomatic subjects. However, testing capacity is not growing fast enough and the blue solid line hits the yellow starred line, denoting the testing availability (Υ_t). As the testing capacity becomes binding, the testing system collapses, as captured by the rapid drop in the probability of catching a newly infected (π_t^T). As a result, the effective reproduction number increases as agents cut their consumption and labor in response to the higher risk of getting infected.

This collapse of the testing system can be averted by introducing a mild lockdown 1 week before the testing capacity would become binding. See the green dashed-dotted line in Figure 3. By lowering the amount of economic interactions, the lockdown reduces the number of tests required, preventing the testing capacity Υ_t (the yellow starred line) to ever become binding. The lockdown greatly mitigates the pandemic recession and reduces the number of final deaths to half. The reason behind this result is that the lockdown solves a coordination failure as agents fail to internalize the effects of their consumption and labor decisions on the viability of the testing system, as explained in Section 3. By preserving the viability of the testing system, the lockdown prevents the effective reproduction number from soaring and, in doing so, improves the outcomes of the pandemic crisis.

The comprehensive tracing technology (the red dashed line in Figure 3) delivers the best outcome among the considered alternative strategies. This better tracing technology allows health authorities to detect and isolate roughly 20% of untested asymptomatic infected agents in every period via backward tracing (see the bottom right graph). In doing so, this technology allows to keep the path of exposed subjects lower, reducing the number of tests required. Consequently, the number of tests performed does not accelerate after period 10 as in the case of the basic tracing technology As a result, under comprehensive contact tracing, the number of required tests does not become constrained by the limited testing capacity Υ_t so early and the testing system remains viable even though no lockdown is imposed.

Nevertheless, the testing availability becomes binding later on, lowering the probability of testing asymptomatic subjects, π_t^A somewhat in subsequent periods. Because of the Peking order, there is no effect on the probability of detecting newly infected, π_t^T, which, as we have already pointed out, is essential for a successful management of the pandemic. Thus, the

[32]The US conducted between 16 and 22 of March 231,081 tests, which is approximately 33,000 daily tests. Between 28 September and 4 October, the US conducted 6,936,961 tests, which correspond approximately to 991,000 daily tests.

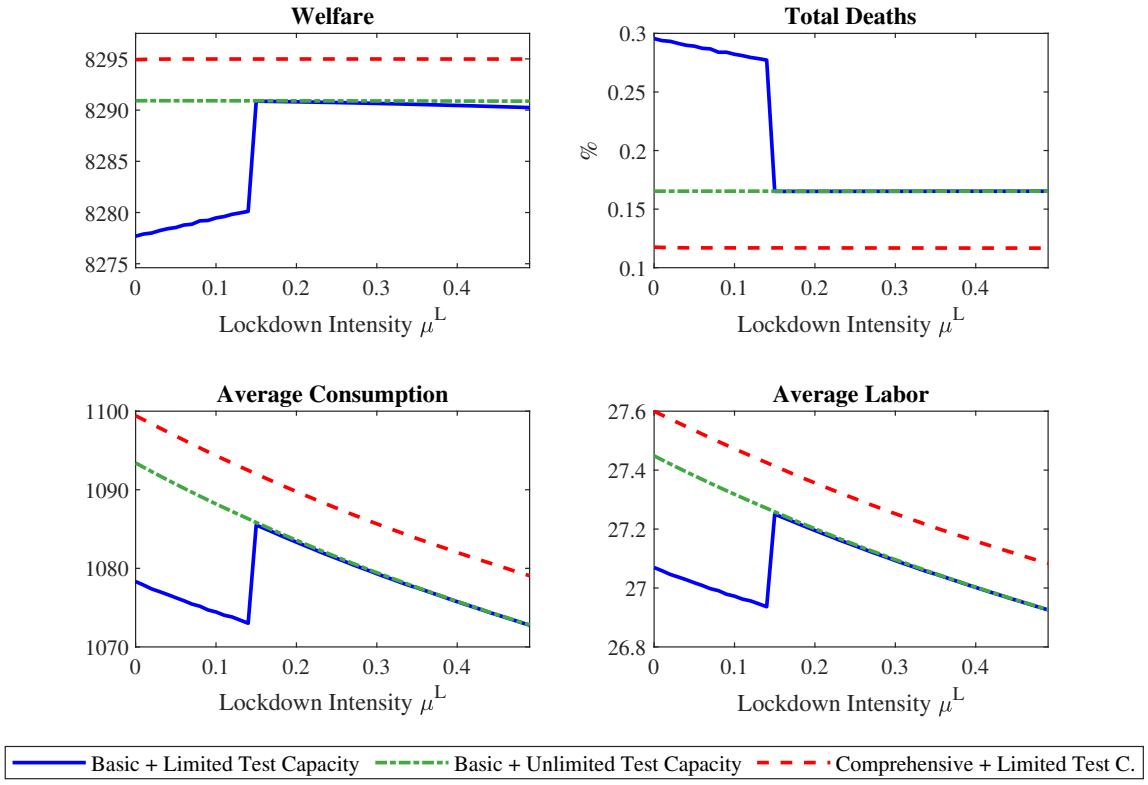

Figure 4: Comparison of different testing strategies under varying lockdown stringency imposed for the first 26 weeks. Welfare in week 1, accumulated deaths, aggregate consumption and aggregate labor averaged over the 250 week horizon are reported.

effective reproduction number hardly budges and the effects on consumption and mortality are only moderate. As we will see in the next section, in this scenario with comprehensive tracing technology only a tiny lockdown is optimal.

5.3 The Optimal Lockdown

We now turn our attention to the optimal lockdown under different contact tracing technologies and testing capacities (unlimited and limited). In our analysis, we focus on one particular dimension of the lockdown: the stringency, which is captured by the size of the consumption tax μ^L. We do not show results when we optimize also with respect to the duration of the lockdown, which is kept fixed at 26 periods, $T_\mu = 26$.[33]

Figure 4 shows the impact of different stringency levels of the lockdown under different contact tracing technologies and testing capacities (unlimited and limited). We show the welfare in the first week, the cumulative deaths, total consumption, and labor.

When no lockdown is imposed ($\mu^L = 0$), the basic tracing technology alone cannot

[33]The results do not depend on the assumption of keeping the lockdown period fixed, as shown in Figure 9 of Appendix H where we consider a longer lockdown duration $T_\mu = 52$.

prevent the demise of the testing system. As a result, consumption and labor are lower and total deaths are higher than those under the case of unlimited testing (the green dashed-dotted line) where, by construction, the testing system cannot collapse. Indeed, when the lockdown intensity is set to zero ($\mu^L = 0$), the vertical distance between the blue solid line and the green dashed-dotted line captures the effects of the collapse of the testing system on welfare, total deaths, aggregate consumption, and labor. As the intensity of the lockdown is increased, social welfare increases as less people will be killed by the pandemic. However, consumption and labor fall steadily.

As the stringency of the lockdown reaches the threshold $\mu^L = 0.18$, social welfare jumps to a higher level as the death toll of the pandemic drops sharply and consumption and labor rise by a discrete amount. This discrete improvement in social welfare is due to the successful preservation of the testing system achieved by the optimal lockdown policy.

This optimal lockdown allows the government to replicate the outcomes of the unlimited testing capacity (the green dahsed-dotted line). This happens because the opitmal lockdown reduces agents' individual consumption and labor *ex-ante* so as to solve the coordination failure threatening the viability of the testing system. The correct functioning of the testing system prompts agents to consume and work more *ex-post*, justifying the jump in consumption and employment when the intensity of the lockdown reaches its optimal level. In addition, by keeping the testing system in one piece, the cumulative death toll looks much less grim when the optimal lockdown is enacted.

Under the comprehensive tracing technology, the viability of the testing system is not threatened by the pandemic (the red dashed line). As a result, raising the intensity of the lockdown (μ^L) monotonically lowers consumption and employment. However, social welfare improves as the lockdown reduces the amount of economic interactions, leading to less infected cases and hence to a lighter death toll. The difference between the red dashed line and the green dashed-dotted line isolates the effects of introducing a more comprehensive contact-tracing technology on welfare, total deaths, aggregate consumption, and labor.

Remarkably, lockdowns have virtually no effect on welfare when the tracing technology is comprehensive. This result stems from the fact that the better tracing technology effectively shores up the testing system against the coordination failure, as shown in Figure 3. However, a tiny lockdown is optimal as it corrects the small drop in the probability of catching asymptomatic subject (π_t^A), shown in Figure 3. Even though this drop is small and, as we noticed, does not bring about any serious consequences for the economy and mortality, welfare is negatively affected by that. In the case of comprehensive contact tracing and testing is unlimited, no lockdown (μ^L) is optimal (not shown).

6 Concluding Remarks

We study contact tracing in a macro-epidemiological model in which some of the infected agents remain asymptomatic for a number of periods during which they contribute to spreading the virus. In the model, agents' consumption and labor decisions have externalities on the viability of the testing system. If agents consume or work more, they will also interact more, increasing the number of contacts to be traced. Too many traceable contacts call for more tests to be performed and, when there are not enough tests available, the testing system starts missing more and more positive cases. In the following periods, there will be more symptomatic cases and more subjects to trace and to test. This vicious circle leads to the demise of testing system and the associated increase in the infection rate. Agents respond to the surge in the infection rate by reducing consumption and labor, which is the prelude to a severe pandemic recession.

A timely, appropriately sized lockdown can correct the coordination failure, averting the demise of the testing system. Unlike the lockdowns that have been enacted by governments in many countries in 2020, the type of lockdowns studied in this paper are generally less stringent and are used preemptively with the objective of moving ahead of the (infection) curve. Indeed, according to our model, a surge in the number of infections is the unequivocal sign that the tracing and testing system is already not working properly.

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A Conditioning on Type-A and Type-T Not Being Tested in Previous Period

We rely on the Bayes theorem to condition the contact probabilities on type-A and type-T agents to not get tested at the end of period $t-1$:

$$f_{t-1|t}^{A,A}(k)=\frac{f_{t-1}^{A,A}(k)\left\{1-\left[1-(1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}(1-\pi_{F})\right\}}{\sum_{k=0}^{\varphi_{c}\left(c_{t-1}^{s}\right)}f_{t-1}^{A,A}(k)\left\{1-\left[1-(1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}(1-\pi_{F})\right\}} \tag{A.1}$$

and

$$f_{t-1|t}^{T,A}(k)=\frac{f_{t-1}^{T,A}(k)\left\{1-\left[1-(1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}(1-\pi_{F})\right\}}{\sum_{k=0}^{\varphi_{c}\left(c_{t-1}^{s}\right)}f_{t-1}^{T,A}(k)\left\{1-\left[1-(1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}(1-\pi_{F})\right\}} \tag{A.2}$$

where $\left[1-(1-\pi_{IS})^{k}\right]$ denotes the probability that at least one of the existing T-links or A-links contacts is with an asymptomatic subject who revealed symptoms in period $t-1$, making the other subject traceable. Conditional on being traced in period $t-1$, the subject will test positive with probability $\pi_{t-1,T}^{0}(1-\pi_{F})$ at the end of the same period. As we will formally define later, $\pi_{t-1,T}^{0}$ is the probability of being tested at the end of period $t-1$ based on tracing the $t-1$ contacts.

All other distributions do not need to be adjusted.[34] It is convenient to write: $f_{t-1|t}^{A,T}(k)=f_{t-1}^{A,T}(k)$, $f_{t-1|t}^{T,T}(k)=f_{t-1}^{T,T}(k)$, $f_{t-1|t}^{S,T}(k)=f_{t-1}^{S,T}(k)$, and $f_{t-1|t}^{S,A}(k)=f_{t-1}^{S,A}(k)$.

B Active T-Links

The objective of this appendix is to derive analytically the probability that a T-link will become inactive (i.e., no longer relevant for contact tracing), $\pi_{P,t-1}^{T}(i)$, for the three types $i\in\{A,T,S\}$. Since type-T and type-S agents cannot infect anyone in period $t-1$, the probability that their T-links will remain active in period t depends on the average probability that a newly infected person in period $t-1$ tests positive at the end of the same period. We denote this probability as $\pi_{P,t-1}^{T}$, which will be derived analytically at the end of this

[34] The distributions $f_{t-1|t}^{T,T}(k)$ and $f_{t-1|t}^{A,T}(k)$ do not need to be adjusted. The reasons is that meeting with newly infected people in period $t-1$ does not make type-T and type-A agents traceable in period $t-1$ because it takes at least one period for newly infected people to become symptomatic. Testing type-S agents in period $t-1$ does not affect their probabilities of having k T-links or A-links as the outcome of these tests is negative (we do not allow for false positive in test outcomes).

section, and then we write

$$\pi^T_{P,t-1}(i) = \pi^T_{P,t-1}, \quad i \in \{T, S\} \tag{B.1}$$

This is the probability to be used in the conditional distribution of active T-links introduced in equation (39) for S-type and T-type agents.

As far as type-A agents are concerned, the derivation of this probability requires a bit more work since some of the T-links of these agents are infectious links. Therefore, the probability for an asymptomatic subject to be tested can be written as the weighted average of the probability of being tested via one of the infection links the asymptomatic subject has created at time $t-1$, $\tilde{\pi}^T_{P,t-1}$, and the probability for the same subject to be tested via non-infectious meetings, $\pi^T_{P,t-1}$; that is,

$$\pi^T_{P,t-1}(A) = \frac{\tau}{\tau + (1-\tau)\,\tau_{t-1}}\tilde{\pi}^T_{P,t-1} + \frac{(1-\tau)\,\tau_{t-1}}{\tau + (1-\tau)\,\tau_{t-1}}\pi^T_{P,t-1} \tag{B.2}$$

where the weights reflect the fraction of infectious T-links. Note that $\pi^T_{P,t-1}$ is the same probability for susceptible and newly infected agents to be tested at the end of period $t-1$, which is shown in equation (B.1).

The probability for a type-A agent to be tested via the infection links she has created at time $t-1$, $\tilde{\pi}^T_{P,t-1}$, has not been derived yet. We tackle this problem by looking at the probability of being traced from the perspective of a subject that became infected as a result of meeting the type-A agent in period $t-1$.

With this change of perspective, the probability $\tilde{\pi}^T_{P,t-1}$ can be obtained by taking three familiar steps. First, we take the step in equation (30) to obtain the probability for the newly infected agents to be tested at the end of the period:

$$\tilde{\pi}^{0,T}_{C,t-1} = \sum_{k=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} \left[1-(1-\pi_{IS})^{k-1}\right] f^T_{t-1}(k) \tag{B.3}$$

where, unlike in equation (30), the probability that none of the contacts of the newly infected agent will become symptomatic, $(1-\pi_{IS})$, is to the power of $k-1$. This tweak is motivated by the fact that it is known that the newly infected agent cannot be traced through the link with the type-A subject who infected her in period $t-1$.[35]

The second step is to obtain the probability of testing positive from the probability of

[35]Type-A agents are, by definition, untested asymptomatic in period t. Consequently, the subject she infected in period $t-1$ cannot be traced via her interaction with the type-A agent. However, the subject can be traced via other non-infectious interactions she entertained in period $t-1$ with other asymptomatic subjects.

being traced, which is precisely the familiar taken in equation (26): $\tilde{\pi}^{0,T}_{P,t-1} = \tilde{\pi}^{0,T}_{C,t-1} \cdot \pi^{0}_{t-1,T} \cdot (1-\pi_F)$. The third step is familiar too: we have to take into account the possibility that the agents infected by the type-A agent in period $t-1$ can be tested because of their contacts in the previous period $t-2$. Thus, we write $\tilde{\pi}^{T}_{P,t-1} = \tilde{\pi}^{0,T}_{P,t-1} + (1-\tilde{\pi}^{0,T}_{P,t-1}) \cdot \pi^{1,T}_{P,t-1}$, where the probability of being tested because of (non-infectious) contacts that occurred in the previous period, $\pi^{1,T}_{P,t-1}$, will be defined later.[36]

C Active A-Links

We now turn to the A-links. It is first important to realize that A-links can also become inactive because the asymptomatic person on the other end of the link recovers or develops symptoms at the end of the previous period. An additional complication is that whether the type-A, or type-T, or type-S individual is traceable at time $t-1$ affects the probability that the A-link will remain active in period t.

If the type-A, type-T, or type-S subject is not traceable in period $t-1$, then no asymptomatic individual she met in period $t-1$ turned symptomatic in that period. Hence, the probability that the link will remain active in the next period is $(1-\pi_R)\left(1-\pi^{A}_{t-1,P}\right)$. Thus, the probability that $\underline{k}_{t-1}$ A-links out of k_{t-1} total links is given by the following binomial distribution:[37]

$$g^{i,A}_{t-1}(\underline{k}_{t-1}|k_{t-1}, A_j = 1) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}, (1-\pi_R)(1-\pi^{A}_{t-1,P})\right) \quad i \in \{A,T,S\} \tag{C.1}$$

where $A_j = 1$ means that the type-A subject is non-traceable at time $t-1$. Note that this probability is the same across the three types of agents considered (type-A, type-T, or type-S), which are denoted by i.

If the type-A, type-T, or type-S subject is traceable in period $t-1$, then at least one of her A-links must have turned symptomatic in that period. Furthermore, other asymptomatic subjects might have also become symptomatic and hence the probability that the link will remain active in the next period is $(1-\pi_{IS}-\pi_R)\left(1-\pi^{A}_{t-1,P}\right)$. All told, the probability that $\underline{k}_{t-1}$ A-links out of k_{t-1} total links is given by the following binomial distribution

$$g^{i,A}_{t-1}(\underline{k}_{t-1}|k_{t-1}, A_j = 2) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}-1, (1-\pi_{IS}-\pi_R)(1-\pi^{A}_{t-1,P})\right) \quad i \in \{A,T,S\} \tag{C.2}$$

[36] We know for sure that these contacts at time $t-2$ were not infectious because we are conditioning on an agent being infected by the type-A agent in period $t-1$.

[37] Since the subjects that met the type-A subject are already untested asymptomatic, they cannot be infected by the type-A agent. Thus, her probability of being tested in period $t-1$ is just the average probability of being tested for an untested asymptomatic, $\pi^{A}_{t-1,P}$.

As before, this probability is the same across the three types of agents considered (type-A, type-T, or type-S), which are denoted by i.

Then we combine the two distributions using the weight for the agents that are not traced in period $t-1$

$$g_{t-1}^{i,A}(\underline{k}_{t-1}|k_{t-1}) = \iota_{t-1}^{i}(k) \cdot \tilde{g}_{t-1}^{i,A}(\underline{k}_{t-1}|k_{t-1}) + (1-\iota_{t-1}^{i}(k)) \cdot \hat{g}_{t-1}^{i,A}(\underline{k}_{t-1}|k_{t-1}) \tag{C.3}$$

where $i \in \{A,T,S\}$ and $\iota_{t-1}^{i}(k)$ denotes the weights, which of course depends on the number of total contacts, k, the agent who met with the asymptomatic has entertained as well as the type (A,T, or S) of agent.

Note that the probability of being traced in period t for a susceptible subjects via her contacts made in the same period is $\pi_{C,t-1}^{S,0}(k) \equiv 1-(1-\pi_{IS})^k$. So, by the law of large numbers, the share for non-traceable susceptible agents is as follows:

$$\iota_{t-1}^{S}(k) = (1-\pi_{IS})^k \tag{C.4}$$

The share of non-traceable A-type and T-type subjects can be derived analogously. However, we need to adjust for the possibility that those traced A-type and T-type agents do not test positive at the end of period $t-1$. In this case, they would no longer been asymptomatic infected in period t and hence they will no longer considered A-type or T-type agents. The share of non-traceable A-type subjects is therefore given by the following

$$\iota_{t-1}^{i}(k) = \frac{(1-\pi_{IS})^k}{(1-\pi_{IS})^k + [1-(1-\pi_{IS})^k]\left(1-\pi_{t-1,T}^{0}(1-\pi_F)\right)}, \quad i \in \{A,T\} \tag{C.5}$$

This adjustment relies on the probability of testing positive conditional on being traced ($\pi_{t-1,T}^{0}(1-\pi_F)$).

At last, we take the step made in equation (40) and obtained the marginalized probability distribution of active A-links for the three types: $g_{t-1}^{i,A}(\underline{k}_{t-1})$ for $i \in \{A,T,S\}$.

D Comprehensive Technology: Exposed in the Previous Period

The measure of the subjects who, in period $t-1$, were exposed to the newly symptomatic individuals is defined below:

$$E_t^1 = (1-\pi_{C,t}^{0,A})\left[\frac{I_{t-1}^{A}(1-\pi_{IS}-\pi_R)\left(1-\pi_{P,t-1}^{A}\right)}{I_t^A}\pi_{C,t}^{1,A} + \frac{T_{t-1}\left(1-\pi_{P,t-1}^{T}\right)}{I_t^A}\pi_{C,t}^{1,T}\right](1-\pi_{IS})I_t^A \tag{D.1}$$

$$+ (1 - \pi_{C,t}^{0,S})\pi_{C,t}^{1,S} S_t + (1 - \pi_{C,t}^{0,R}) \left[\frac{R_{t-1}^A}{R_t^A} \pi_{C,t}^{1,R} + \frac{\pi_R I_{t-1}^A}{R_t^A} \pi_{C,t}^{1,RA} \right] R_t^A$$

where $\pi_{C,t}^{R,1}$ is the probability to be traced for a Type-R agent, which is defined as an agent who became unobserved recovered in period $t-1$ or earlier. $\pi_{C,t}^{RA,1}$ is the probability for a Type-RA agent, which is defined as an agent who became an unobserved recovered agent in period t and hence was an asymptomatic agent in $t-1$. This equation takes into account that different agents of a group can have a different history of interactions due to changes of the type. For instance, there is a difference for asymptomatic infected who became infected in the previous period and the ones who already were infected in the previous period. This is captured in the two terms in the first square bracket of equation (D.1).

The derivation $\pi_{C,t}^{R,1}$ for the Tpye-R agent is the same as for the Type-S agents $\pi_{C,t}^{S,1}$ with one difference. The contacts with asymptomatics in period $t-1$ do not need to be adjusted in contrast to Type S-Agents because the Type-R agent cannot change their health status. Therefore, we have directly $f_{t-1}^{R,A}(k) = f_{t-1}^{A,A}$ and hence the adjustment in equation (38) is not needed. The derivation $\pi_{C,t}^{RA,1}$ for a Type-RA agent is exactly the same as for a Type-A agent except for two exceptions. First, the type-RA agent recovers and becomes an unobserved recovered agent independent of getting tested. For this reason, we can skip the time adjustment in equation (A.1) so that $f_{t-1|t}^{RA,A}(k) = f_{t-1}^{A,A}$. Second, the share of non-traceable subjects does not depend on the probability of getting tested. Replacing equation (C.5) with $\iota_{t-1}^{RA} = (1 - \pi_{IS})^k$ captures this difference. The remaining steps are the same as both types have been asymptotic agents in the previous period. Finally, the probability to be traced due to previous period contacts is the same for susceptible agents regardless of whether they get infected in period t.

E Random Testing

An alternative to a contacting tracing strategy would be to test randomly the population, a strategy that has been also actively discussed. In this strategy, the probability of getting tested is the same for the susceptibles, asymptomatics and unobserved recovered. As before, we assume that agents that are either infected, tested-positive or observed recovered. This can be interpreted as an extreme case of contact tracing, in which every agent gets traced, which can be written is

$$\pi_{C,t}^i = 1, \quad i \in \{A, S, T, R^U\} \tag{E.1}$$

As every agents get traced, the number of subjects to be tested is very large. The pool

of agents that the government tests is given as

$$E_t = S_t + A_t + R_t^U \tag{E.2}$$

The government has the amount of available. Therefore, the probability of getting tested conditionally on being traced depends on the amount of tests Υ_t relative to the pool E_t:

$$\pi_{P,t}^i = \min\left(1, \frac{\Upsilon_t}{E_t}\right), \quad i \in \{A, T\} \tag{E.3}$$

We can plug equations (E.1) and (E.3) into equation (26) to evaluate the probability of testing positive for newly infected subjects, $\pi_{P,t}^T$, and subjects infected in earlier periods, $\pi_{P,t}^A$.

F Model Solution

Solution Algorithm The solution algorithm solves iteratively based on a numerical root finder the model relying on perfect foresight expecations. It computes the sequence of policy functions $\{n_t^R, n_t^{IS}, n_t^P, n_t^{RO}\}_{t=1}^T$ for T = 250 weeks for a given sequence of taxes $\{\mu_{c,t}\}_{t=1}^T$ and given initial asymptomatic and symptomatic infected agents: $\{I_1^A, I_1^S\}$. The algorithm is summarized below:

1. Solve the model for the pre-pandemic economy.

2. Guess a path for the sequence of labor $\{n_t^R, n_t^{IS}, n_t^P, n_t^{RO}\}_{t=1}^T$.

3. Based on the guessed path, solve for the consumption, labor, marginal utilities and intraperiod utility of the susceptibles, infected symptomatic, tested-positive and observed recovered agents, that is $\{c_t^i, \lambda_t^i, u_t^i\}_{t=1}^T,\ i \in \{S, IS, P, OR\}$ and the lump sum transfer from consumption taxes $\{\Gamma_t^L\}_{t=1}^T$.[38]

4. Calculate the interactions of agents (e.g. for susceptibles $f_t^S(k)$) based on their consumption and labor decisions. This allows to calculate probability of getting infected τ_t (for details see paragraph below) and also the probabilities of getting tested for newly infected $\pi_{c,t}^T$ and asymptomatic infected $\pi_{c,t}^A$. Crucially, the latter objects depends on the tracing technology and the testing capacity. In case of the comprehensive tracing technology, the amount of active links from the previous period (e.g for susceptibles

[38]To be precise, the marginal utility of susceptibles is actually calculated later in step 6 as it depends on the testing probabilities.

with T-type agents $g_{t-1}^{S,T}(\underline{k})$) need to be calculated. Based on these objects, the evolvement of the different groups can be computed by forward iteration so that the sequences $\{S_t, T_t, I_t^A, P_t, I_t^S, R_t^U, R_t^O, D_t, Pop_t\}_{t=1}^T$ are obtained.

5. Iterate backwards to solve the utility of the different agents, that is $\{V_t^S, V_t^A, V_t^{UR}, V_t^P, V_t^{IS}, V_t^{OR}\}_{t=1}^T$.

6. Calculate the marginal utility of consumption for a susceptible λ_t^s based on the utilities of the different groups, the probability to get infected and the probability to get tested

7. To solve for the sequences pf $\{n_t^R, n_t^{IS}, n_t^P, n_t^{RO}\}_{t=1}^T$, use a numerical root finder that minimises the error in budget constraint for the positively-tested and infected symptomatic agents, the government budget constraint for the lockdown taxes and the first order condition with respect to labor of susceptibles in each period t

8. Update the path for the sequence of labor slowly and repeat steps 3 - 7 until convergence of $\{n_t^R, n_t^{IS}, n_t^P, n_t^{RO}\}_{t=1}^T$.

G The Individual Risk of Getting Infected

The probability of getting infected τ_t as a function of consumption and labor decisions enters the decision problem of the susceptible, untested asymptomatic, and unobserved recovered agents. See Section 2.3. This probability, which is defined in equation 6, depends on the non-differentiable functions $\varphi_c(c_t^s)$ and $\varphi^n(n_t^s)$ and introduces ridges and cliffs in the value function V_t^s of the agents, making the solution to the optimization problem very challenging. To improve the speed and the reliability of the solution algorithm, it is convenient to take the following two steps.

First, we linearly approximate the probability of getting infected conditional on a susceptible individual entertaining k interactions around the average number of interactions at steady state $(\bar{k_c}, \bar{k_n}, \bar{k_o})$ and obtain

$$\begin{aligned} p &= 1 - (1-\tau)^{k_c+k_n+k_o} \\ &\approx \underbrace{-\ln(1-\tau)(1-\tau)^{\bar{k}_c+\bar{k}_n+\bar{k}_o}}_{\Xi} \cdot (k_c + k_n + k_o) \end{aligned} \tag{G.1}$$

Note also that Ξ is just a constant that depends on parameters and the chosen value for the average number of trials $\bar{k}$.

We then characterize the expected probability for a susceptible individual to get infected conditional on consuming c_t^S and working n_t^S as before using the joint distribution defined

in equation (5) and after some straightforward manipulations we obtain

$$\begin{aligned}\tau_t &= \sum_{k_c=0}^{\varphi_C(c_t^S)} \sum_{k_n=0}^{\varphi_N(n_t^S)} \sum_{k_o=0}^{\varphi_O} \Xi \cdot (k_c + k_n + k_o)\, f_t^c(k_c) \cdot f_t^n(k_n) \cdot f_t^o(k_o), \\ &= \Xi \left[\varphi_c(c_t^S) \left(\frac{C_t^A}{C_t} \right) + \varphi_n(n_t^S) \left(\frac{N_t^A}{N_t} \right) + \varphi_O \left(\frac{A_t}{Pop_t} \right) \right] \end{aligned} \tag{G.2}$$

Second, we consider a linear approximation of the step functions $\varphi_c(c_t^S) \approx \varphi_c \cdot c_t^S$ and $\varphi_n(n_t^S) \approx \varphi_n \cdot n_t^S$. Plugging these linear functions into equation (G.2) leads to equation (47).

H Additional Figures

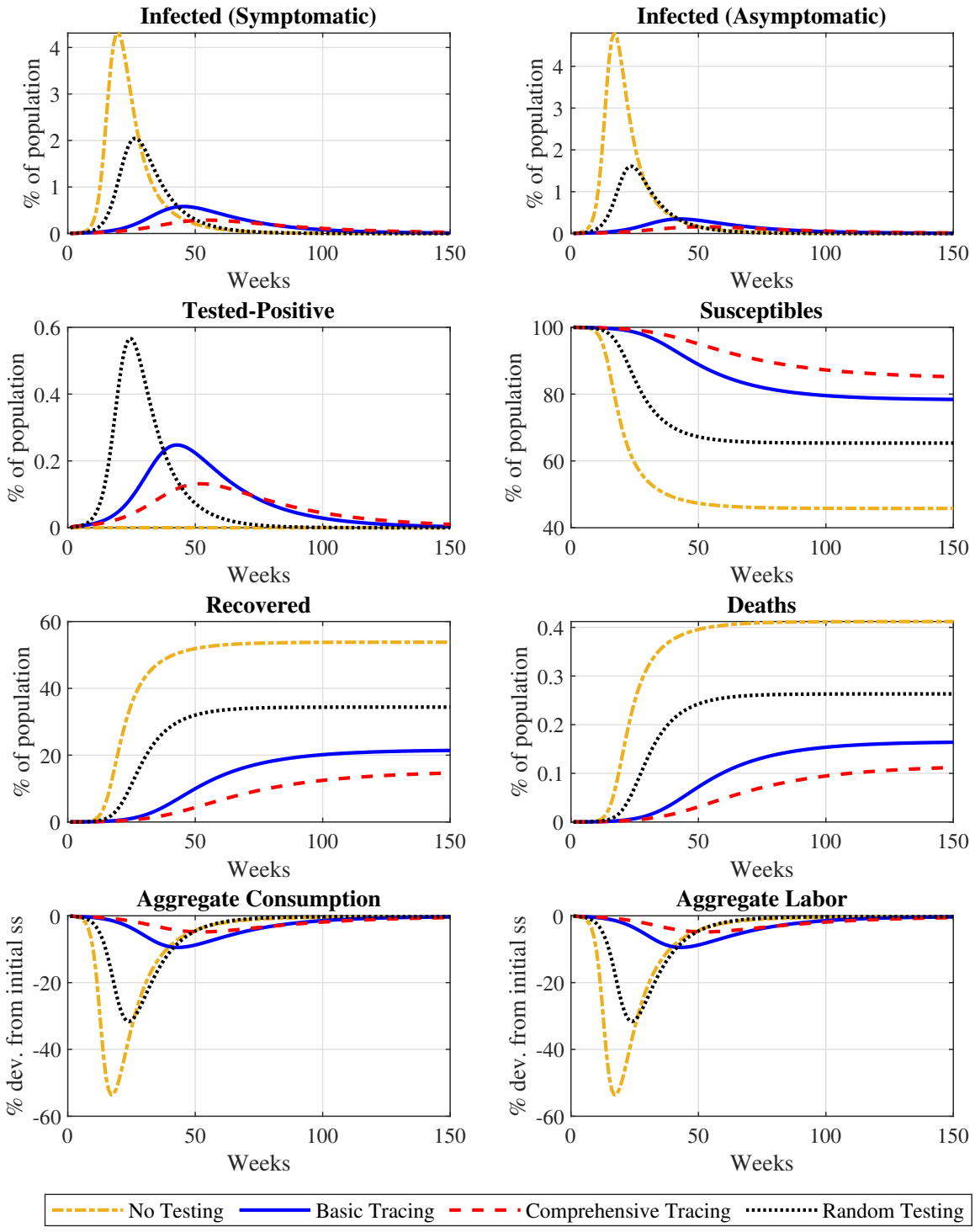

Figure 5: Comparison of different testing strategies with unconstrained number of tests for contact tracing: No testing (blue solid), basic tracing (red dashed) corresponds to current week contact tracing, comprehensive tracing (green dash-dotted) corresponds to current and previous week contact tracing and random testing (black dotted) has an amount tests available for 20% of the entire population each week.

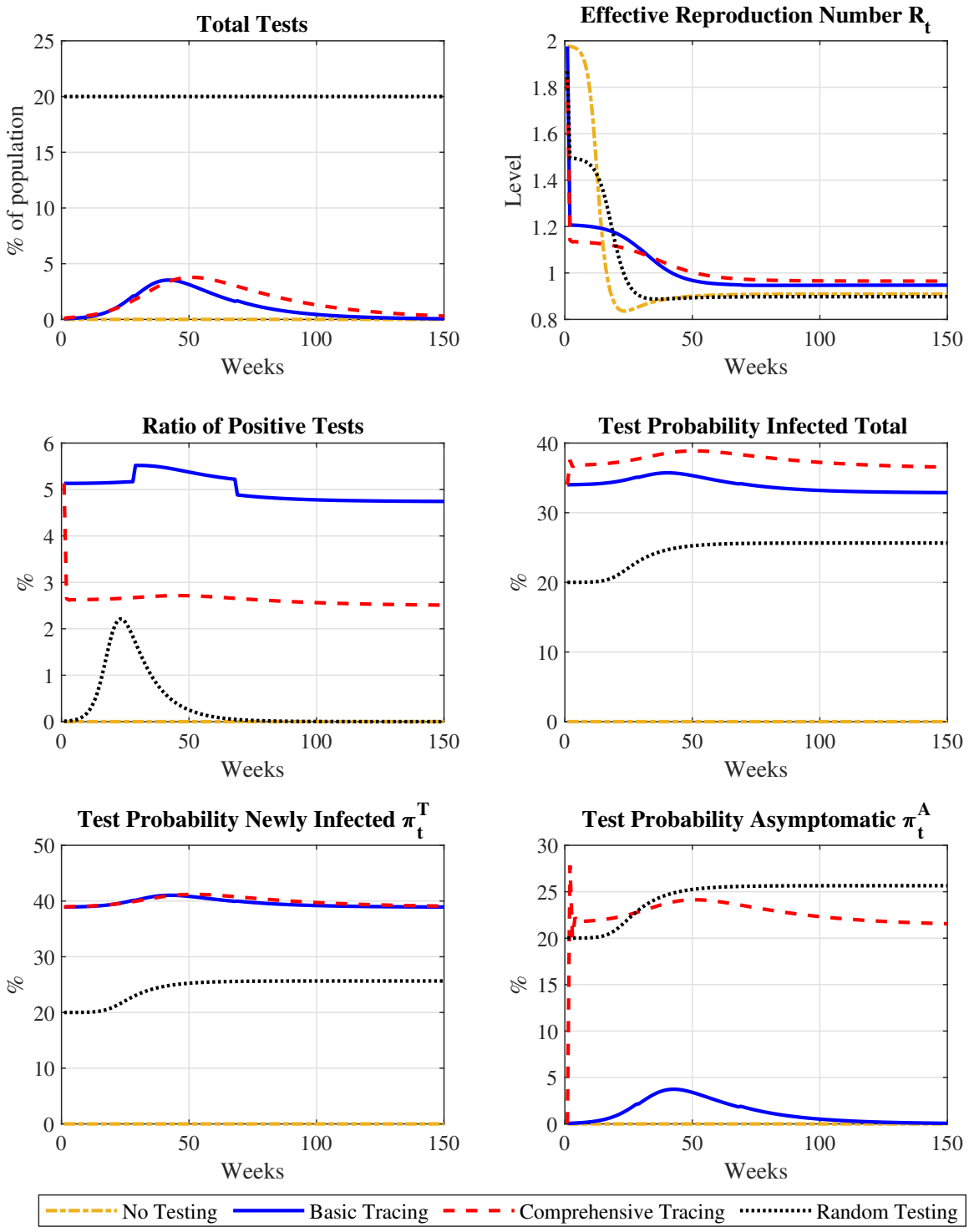

Figure 6: Comparison of different testing strategies with unconstrained number of tests for contact tracing: No testing (blue solid), basic tracing (red dashed) corresponds to current week contact tracing, comprehensive tracing (green dash-dotted) corresponds to current and previous week contact tracing and random testing (black dotted) has an amount tests available for 20% of the entire population each week. The graphs capture different statistics related to testing.

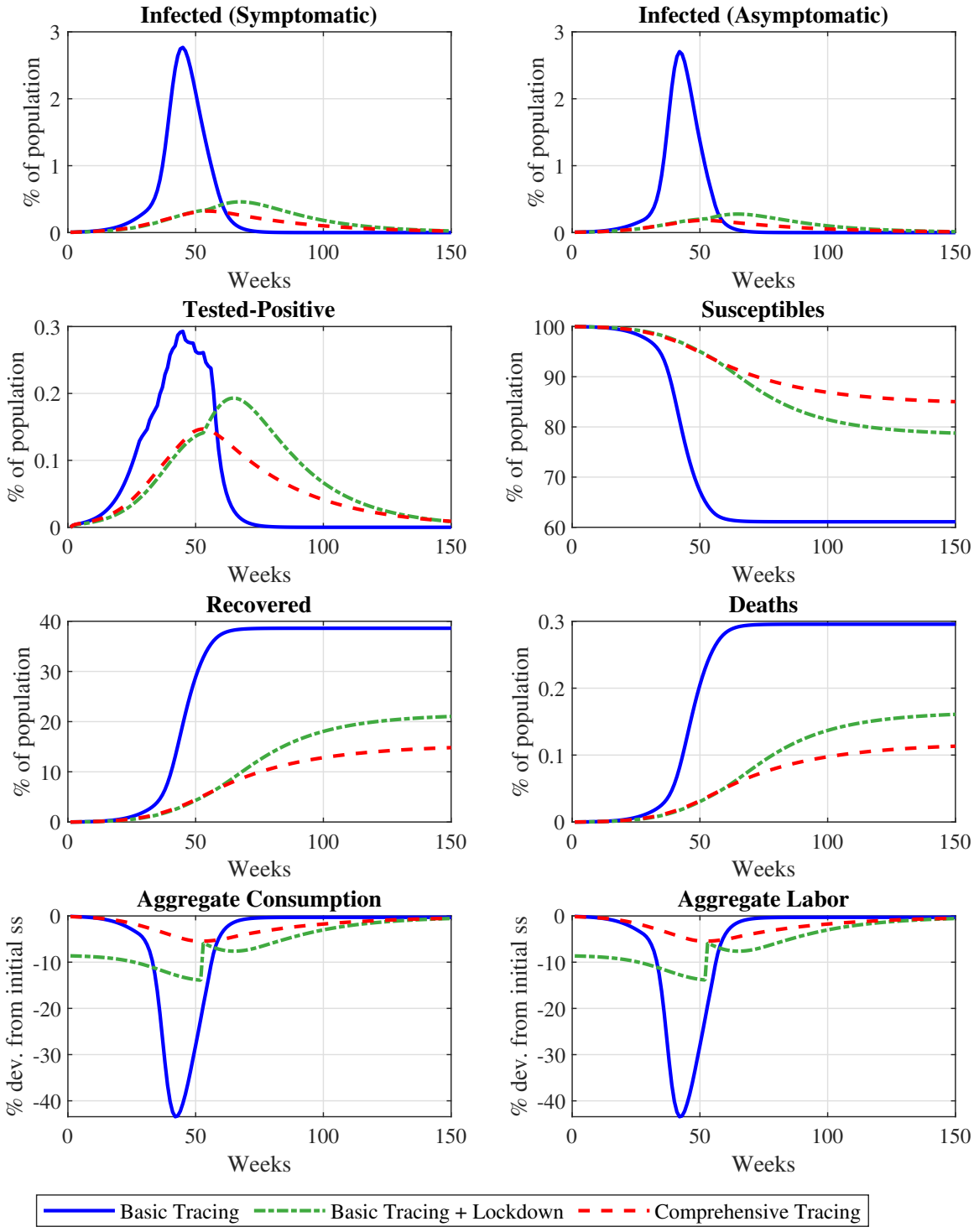

Figure 7: Comparison of different testing strategies with limited tests: Comprehensive tracing (blue solid line) is previous and current week tracing, basic tracing (red dashed line) is current week tracing and in the green dash-dotted basic tracing is combined with a 1 year lockdown.

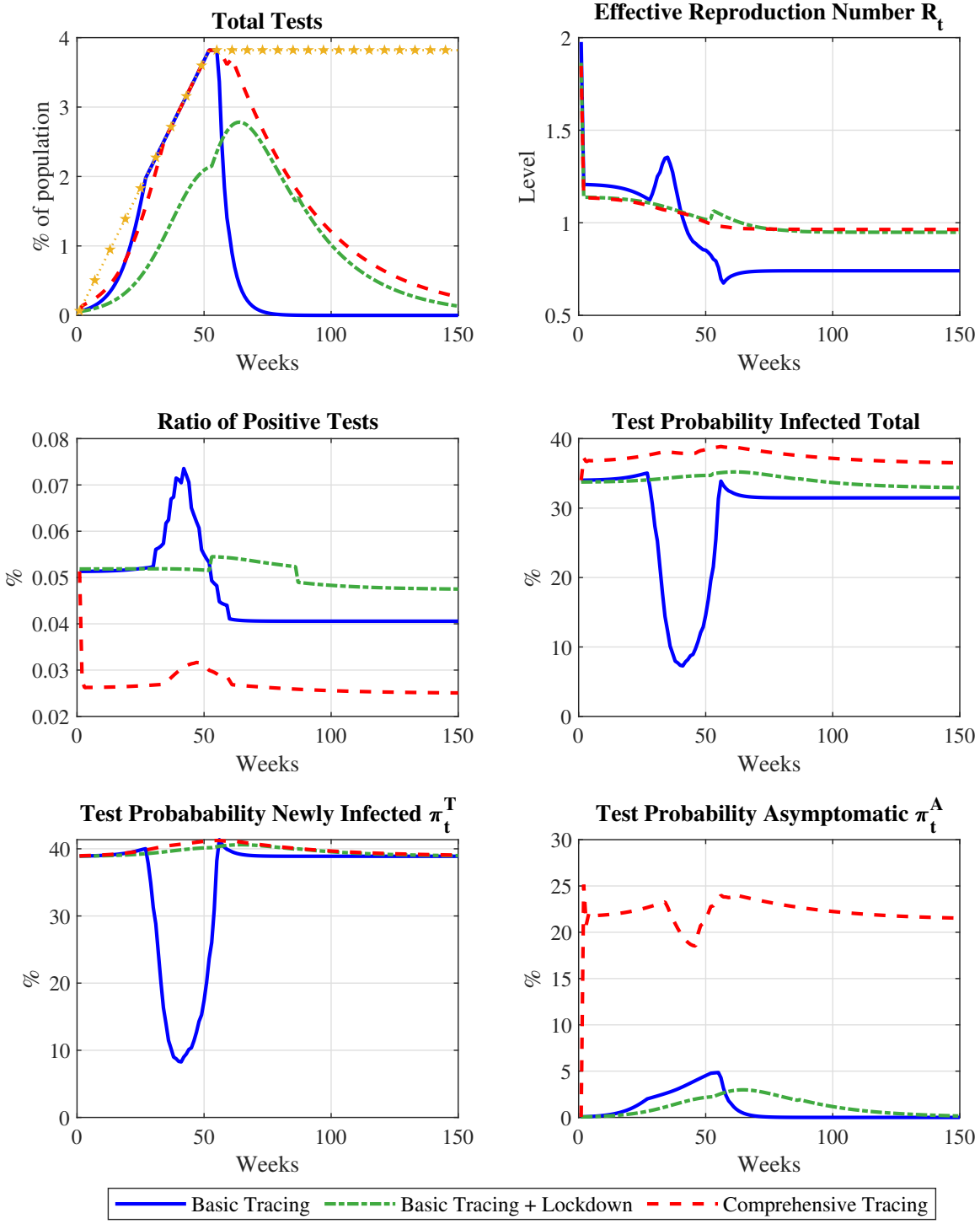

Figure 8: Comparison of different testing strategies with limited tests: Comprehensive tracing (blue solid line) is previous and current week tracing, basic tracing (red dashed line) is current week tracing and in the green dash-dotted basic tracing is combined with a 1 year lockdown. In the first plot, the yellow starred line shows the testing capacity Υ_t.

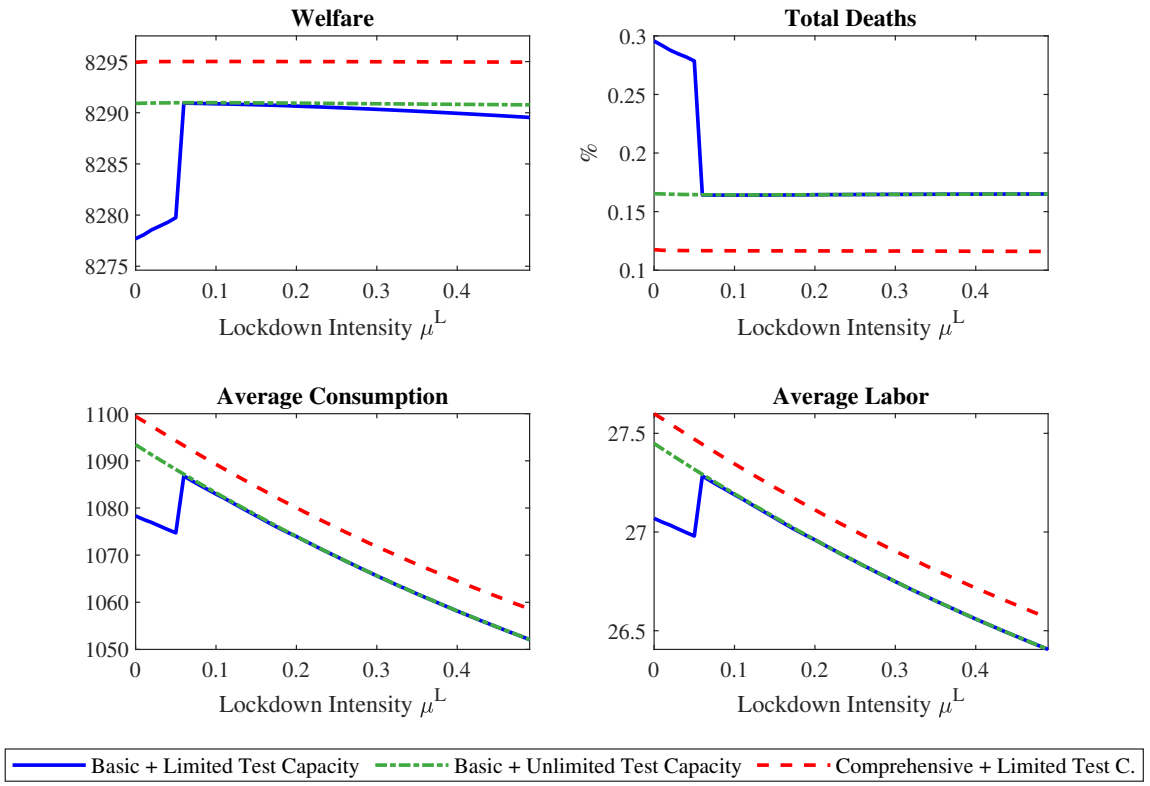

Figure 9: Comparison of different testing strategies under varying lockdown stringency imposed for the first 52 weeks. Welfare in week 1, accumulated deaths, aggregate consumption and aggregate labor averaged over the 250 week horizon are reported.