Pandemic Recessions and Contact Tracing*

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December 31, 2020

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 asymptomatic spreaders is limited. Contact tracing is a testing strategy that aims to reconstruct the infection chain of newly symptomatic agents. We show that contact tracing may be insufficient to stem the spread of infections because agents fail to internalize that their individual consumption and labor decisions increase the number of traceable contacts to be tested in the future. Complementing contact tracing with timely, moderate lockdowns corrects this coordination failure, allowing policymakers to buy time to expand the testing scale so as to preserve the testing system. We provide theoretical underpinnings to the risk of becoming infected in macro-epidemiological models. Our methodology to reconstruct infection chains is not affected by curse-of-dimensionality problems.

JEL Classification: E10, D62, I10.
Keywords: Contact tracing, testing, quarantine, coordination failure, externalities, infection chain, lockdown, epidemics, SIR-macro model, COVID-19.

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1 Introduction

The outbreak of the COVID-19 pandemic set off a worldwide health and economic crisis of unprecedented proportions. Quickly expanding the capacity for testing, isolation, and contact tracing has been suggested by several experts to be a crucial step to alleviate the pandemic's toll on the economy and mortality.[1] For instance, South Korea has combined contact tracing, mass testing, and mild containment measures to achieve one of the lowest infection rates in the world. Nevertheless, other countries, such as the U.S., have been considerably less successful, notwithstanding sizable investments made in contact tracing and mass testing. Dr. Fauci, the director of the National Institute of Allergy and Infectious Diseases, explained the failure of contact tracing in the U.S. at a Milken Institute event held in July: "When you have a situation in which there are so many people who are asymptomatic, that makes that that much more difficult, which is the reason you wanted to get it from the beginning and nip it in the bud. Once you get what they call the logarithmic increase, then it becomes very difficult to do contact tracing. It's not going well."

We construct a macro-epidemiological model to explain why contact tracing can fail and how this failure can be averted. We show that contact tracing can be unsuccessful because of a coordination failure leading people to entertain economic and social interactions at rates exceeding policymakers' ability to trace, test, and isolate the close contacts of confirmed cases. Complementing contact tracing with a timely, moderate lockdown corrects this coordination failure, allowing policymakers to buy time to expand the tracing and testing scale. Preserving the viability of the tracing and testing system is critical to successfully managing pandemics.

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, therefore, 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 a newly symptomatic case to become infected or to infect other agents. This reconstruction

[1] 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 [to a successful response] 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."

forms the basis to decide who to test. The objective of testing is to detect as many asymptomatic spreaders as possible and quarantine them. How much of the infection chain can be reconstructed by health officials defines the efficiency of the contact tracing technology.

Agents' consumption and labor decisions have externalities on the number of subjects that health authorities have to trace and test in future periods. Since agents fail to realize the existence of these externalities, their consumption and labor decisions may end up overburdening the testing system to the point of making it insufficient to contain the spread of the virus a few periods later. As the risk of becoming infected increases, agents want to reduce their economic interactions. To this end, they lower their consumption and labor, causing 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. This is not the only way to shore up the testing system against agents' coordination failure in the model. Improving the efficiency of the contact tracing technology makes the testing system more resilient and reduces the optimal stringency of the lockdown.[2]

When the epidemiological parameters of the model and the availability of tests are calibrated to match the U.S. data during the COVID-19 pandemic, we find several interesting results. Contact tracing –even a very basic one– considerably improves the ability of health authorities to control the spread of the pandemic relative to a strategy based on randomly testing the population. This prediction is in line with empirical findings by Fetzer and Graeber (2020), who show quasi-experimental evidence that contact tracing is very effective in containing the spread of the virus. Unlike randomly testing the population, contact tracing 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 random testing 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 its testing capacity at the beginning of the pandemic would have not been fast enough to stop

[2]A redistributive fiscal policy aimed at taxing the symptomatic agents could also be an effective tool to counter the externalities studied in this paper. This policy penalizes risk taking and compensates for labor productivity losses associated with the symptoms of the disease. We do not study this policy because redistributive issues are beyond the scope of this paper, whose main objective is to formally model contact tracing.

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 testing system can be preserved by imposing a mild lockdown.[3] The lockdown 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 testing system for a successful management of the pandemic.

Lockdowns are typically enacted in response to flare-ups of infection –often to prevent hospitals from becoming overburdened. In this paper, we suggest a quite different strategy that envisions moderate lockdowns as preemptive tools to keep the tracing and testing system viable while policymakers ramp up the testing scale. 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, we show that a surge in the number of infections is the unequivocal sign that the testing system is already not working properly.

When we consider a more efficient technology allowing health authorities to trace contacts that occurred as far back as the previous week (*comprehensive contact tracing technology*), economic and health outcomes improve considerably. The comprehensive tracing technology gives health authorities a second chance to quarantine asymptomatic spreaders who could not be traced and tested in the previous periods.[4] Managing to lower the number of asymptomatic spreaders early on reduces the amount of tests needed to be performed later on. It then turns out that, under this more efficient tracing technology, the pace at which the U.S. built up its testing capacity would have required introducing only minimal restrictions on the economy.

Contact tracing has been used to control the spread of a long list of lethal diseases, such as syphilis, tuberculosis, measles, sexually transmitted infections (including HIV), bloodborne infections, Ebola, H1N1 (swine flu), Avian Influenza, SARS-CoV (SARS), and SARS-CoV-2 (COVID-19).[5] However, formally modeling contact tracing is challenging as the

[3]By mild lockdown, we mean a less stringent lockdown than the optimal one in the absence of testing.

[4]Under the basic tracing technology, these undetected spreaders will not be traceable via their infection chain. They can only be detected if they randomly meet one of the subjects who will then develop the symptoms of the disease. But this is a relatively low-probability event. It is actually worse than that, since the entire infection chain that each of these undetected spreaders will create going forward becomes much harder for the health authorities to uncover. This happens because newly infected subjects are initially asymptomatic and it takes at least one period for them to show symptoms. The comprehensive tracing technology is not affected by these shortcomings.

[5]Contact tracing was originally proposed in 1937 by Surgeon General Thomas Parran for the control of

number of contacts established by an infected subject quickly explodes as the number of past periods considered increases. We solve this dimensionality problem by modeling the probability that a susceptible subject entertains a number of economic interactions with the pool of asymptomatic infected agents as a sequence of Bernoulli trials. The number of trials depends on how much susceptible agents consume (work) and the probability of success (i.e., meeting with an asymptomatic infected subject) is assumed to depend on the total consumption (work) of asymptomatic infected people relative to the total consumption (labor) of non-quarantined subjects. It follows that the probability for a susceptible agent to have met a number of asymptomatic infected agents who can have infected them is a binomial distribution.

This binomial distribution allows us to parsimoniously characterize the endogenous probability of a susceptible agent to become infected in a given period. This probability is isomorphic to that used in the canonical SIR model proposed by Kermack and McKendrick (1927). How we characterize the probability of becoming infected provides theoretical underpinnings to those macro-epidemiological models where this probability is assumed. Moreover, this binomial distribution contains all the necessary information to reconstruct the infection chains in our model, which is key to pinning down agents' probabilities of being traced and tested. This methodology to reconstruct the history of interactions relevant for contact tracing is general and can be applied to models with multiple sectors or heterogeneous agents.[6]

Related literature Our model is related to macro-epidemiological literature. This literature is quickly growing in many different directions. The directions more closely related to our paper are: analyses of the trade-off between saving human lives and mitigating the recession (Gourinchas 2020 and Hall, Jones and Klenow 2020); models to study optimal lockdowns (Alvarez, Argente and Lippi 2020; Atkeson 2020; Bethune and Korinek 2020; Farboodi, Jarosch and Shimer 2020; Eichenbaum, Rebelo and Trabandt 2020*a*; Moser and Yared 2020; Piguillem and Shi 2020); models to study more targeted and smarter policies, such as testing or targeted quarantines, as alternatives to indiscriminate lockdowns (Acemoglu et al. 2020; Akbarpour et al. 2020; Atkeson et al. 2020; Azzimonti et al. 2020; Baqaee et al. 2020*b*; Berger et al. 2020; Brotherhood et al. 2020; Chari, Kirpalani and Phelan 2020; Eichenbaum, Rebelo and Trabandt 2020*b*; Favero, Ichino and Rustichini 2020; Galeotti, Steiner and Surico 2020; Glover et al. 2020; Hornstein 2020; Krueger, Uhlig and Xie 2020); studies of the dis-

syphilis in the U.S. and was later implemented to control the spread of this virus in the following years (Parran, 1937).

[6]See Guerrieri et al. (2020) for an example of multisectoral models to study how an epidemic and associated lockdowns affect aggregate demand and supply. See Kaplan, Moll and Violante (2020) for an example of macro-epidemiological models with income and wealth inequalities.

tributional consequences of various containment policies (Hacioglu, Känzig and Surico 2020; Kaplan, Moll and Violante 2020); empirical evaluations of the effects of COVID-19 across countries (Fernández-Villaverde and Jones 2020*a*,*b*).

Some of these papers develop models that use a network structure combined with various types of agents' heterogeneity to study the spread of the pandemic and its cross-sectional consequences. While we also use a network to model the spread of infections, our primary goal is to use the network to model contact tracing. We also show analytically that our approach to constructing the network is consistent with the SIR and macro-SIR literatures.

Our main contributions relative to the literature are twofold. First, we study a novel type of coordination failure that can disrupt the functioning of the tracing and testing system. A second important point of departure is to use the network of agents' past interactions to keep track of those spreaders who can be traced and tested. While other papers have studied contact tracing, we believe to be the first ones to model contact tracing using this endogenous network structure. These two contributions are intertwined. In order for the coordination failure to arise, the amount of traceable agents who need to be tested has to be linked to agents' consumption and labor decisions. In our model, this link is given by the endogenous network of interactions, which is determined by how much agents have consumed and how much they have worked in the past periods.

The rest of the paper is organized as follows. In Section 2, we present the model. In Section 3, we formalize contact tracing. In Section 4, we discuss the solution method and the calibration of the model. In Section 5, we apply our methodology to study why contact tracing has been largely ineffective in mitigating the COVID-19 crisis in the US and what could have been done to make it work. Some extensions are discussed in Section 6. In Section 7, we conclude.

2 The Model

The model economy is populated by agents who consume and work, firms that 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 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 (2020*a*), we assume there are three types of interactions through which the virus spreads out: consumption interactions, work interactions, and other interactions indepedent of agents' decisions.

Every period is organized as follows: First, agents consume, work, and engage in other interactions. Second, agents' health status can change: agents can get infected or infected agents can recover or die. Third, health officials can administer tests. Tests deliver a binary outcome: positive or negative. Tests do not reveal if an agent has never been infected or has recovered.

There are six types of agents, who differ in their health status. The first type includes *susceptible agents* who have not contracted the disease, are not carriers, and are not immune. Infected agents can be divided into three types: *Untested asymptomatic agents* if they have not shown symptoms and have not 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 previously tested positive. The remaining two types are the recovered agents, who have developed immunity.[7] They are the *observed recovered agents*, who have shown symptoms or have tested positive and the *unobserved recovered agents* who have recovered without having ever shown any symptoms of the disease or having ever tested positive.

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 susceptible agents and that of unobserved recovered subjects is also not observed even if they got tested at the end of the previous period. This is because tests only say whether the tested individual is currently infected or not. The health status of positive-tested, symptomatic infected, and observed recovered agents is publicly observed.

Quarantine. The positive-tested and the symptomatic subjects have their health status revealed and the health authorities immediately quarantine them.[8] Being quarantined means two things. First, in quarantine consumption and labor decisions are subject to restrictions, which are modeled as a consumption tax. Second, quarantined agents are isolated from other subjects and cannot infect anyone.

Note that we use the word quarantine to mean a containment policy targeted to a single subject or a subset of subjects who have been uncovered by the government as potentially capable of spreading the virus. Therefore, quarantine is different from lockdown, which refers

[7]The model is suitable to study the spread of viruses that are non-transmissible by asymptomatic individuals. This requires to set the probability to develop the symptoms of the disease in every period equal to one ($\pi_{IS} = 1$). The coordination failure we study in this paper plays an important role in determining the effectiveness of contact tracing when dealing with this type of viruses as well.

[8]Untested asymptomatic individuals cannot be quarantined because the health authorities cannot distinguish them from susceptible agents.

to an economy-wide containment measure, affecting all the subjects regardless of their health status.

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.[9] 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, meets k-times with agents of a certain type is given by the binomial distribution $\mathcal{B}(k, n, p) = \binom{n}{k} p^k (1-p)^{(n-k)}$, 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 agent to meet with agents 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 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 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 of meeting 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 agents and C_t stands for the aggregate consumption of non-quarantined agents. Analogously, the probability for a worker to meet an untested asymptomatic worker in one hour 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

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

non-quarantined agents. The probability for an individual to meet with an untested asymptomatic agent 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.

Assumption 3. *An individual of health status i who consumes c_t^i units of goods, 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 the total number of interactions a susceptible individual needs to entertain to consume c_t^s, work n_t^s, 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. We can think of the mappings φ_C and φ_N as monotonically increasing step functions.

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:

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

$k \leq \varphi_C(c_t^s)$. We can analogously derive the probability for a susceptible individual to meet k-times with the asymptomatic subjects while working an amount n_t^s of hours

$$f_{n,t}\left(k\right) \equiv \mathcal{B}\left(k, \varphi_N(n_t^s), \frac{N_t^A}{N_t}\right) = \binom{\varphi_N(n_t^s)}{k} \left(\frac{N_t^A}{N_t}\right)^k \left(1 - \frac{N_t^A}{N_t}\right)^{\varphi_N(n_t^s)-k}, \tag{2}$$

$k < \varphi_N(n_t^s)$. 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

$$f_{o,t}(k) \equiv \mathcal{B}\left(k, \varphi_O, \frac{I_t^A}{Pop_t}\right) = \binom{\varphi_O}{k} \left(\frac{I_t^A}{Pop_t}\right)^k \left(1 - \frac{I_t^A}{Pop_t}\right)^{\varphi_O-k}, \tag{3}$$

$k < \varphi_O$.

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

as follows:

$$f_t(k_c, k_n, k_o) \equiv f_{c,t}(k_c) \cdot f_{n,t}(k_n) \cdot f_{o,t}(k_o). \tag{4}$$

Assumption 4. *Conditional on meeting with an untested asymptomatic individual, a susceptible agent will become infected with probability* $\tau \in (0, 1)$.

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, i.e., $(1 - \tau)^{k_c+k_n+k_o}$.

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:

$$\tau_t \equiv \sum_{k_c=0}^{\varphi_C(c_t^s)} \sum_{k_n=0}^{\varphi_N(n_t^s)} \sum_{k_o=0}^{\varphi_O} \left[1 - (1 - \tau)^{k_c+k_n+k_o}\right] f_t(k_c, k_n, k_o), \tag{5}$$

where $f_t(k_c, k_n, k_o)$ denotes the joint binomial distribution defined in equation (4).

The infection rate τ_t can be approximated to obtain

$$\tau_t \approx \Xi \left[\varphi_c \cdot c_t^s \left(\frac{C_t^A}{C_t}\right) + \varphi_n \cdot n_t^s \left(\frac{N_t^A}{N_t}\right) + \varphi_O \left(\frac{A_t}{Pop_t}\right)\right], \tag{6}$$

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.

The approximated infection rate τ_t in equation (6) is isomorphic to the rate used in the canonical SIR model and other leading macro-epidemiological models, in which this rate is assumed (e.g., Eichenbaum, Rebelo and Trabandt 2020*a*). Since the infection rate in equation (6) stems from the choice of modeling economic interactions as binomial trials (Assumptions 1-4), our paper provides theoretical underpinnings to the infection rate used in those models.

2.2 Agents with Unknown Health Status

As discussed earlier, susceptible, untested asymptomatic, and unobserved recovered individuals do not know their health status. To keep the model tractable, we assume that these agents make consumption and labor decisions in the belief that they have never been infected

and thereby are susceptible. While this assumption has a behavioral flavor, it has minimal implications for our conclusions because our analysis is primarily focused on dynamics at the beginning of a pandemic when the economy is far away from achieving herd immunity.[10] Conditional on the belief of having never been infected, the agents compute the probability of future changes in their health status using the model-consistent probabilities. It follows that the agents who do not know their health status choose their 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 the utility function $u\left(c_t, n_t\right) = \ln c_t - \frac{\theta}{1/\eta} n_t^{1/\eta}$ and β denotes the discount factor. We denoted all the variables in equation (7) with the superscript S because these agents believe to be susceptible.

These agents expect to be infected with probability τ_t, which is defined in equation (5). Conditional on this event, the agents expect with probability $\pi_{P,t}^T$ to test positive at the end of period t and thereby to receive the utility V_{t+1}^P of the positive-tested agents in period $t+1$. This value function will be defined in Section 2.3. With probability $(1-\pi_{P,t}^T)$, the agents expect to become untested asymptomatic and receive the utility V_{t+1}^A, which, in period t, is given by

$$V_t^A = u(\tilde{c}_t^s, \tilde{n}_t^s) + \beta\left[\pi_{IS} V_{t+1}^{IS} + \pi_R V_{t+1}^{UR} + \left(1-\pi_{IS}-\pi_R\right)\left(\pi_{P,t}^A V_{t+1}^P + (1-\pi_{P,t}^A) V_{t+1}^A\right)\right], \tag{8}$$

where $\tilde{c}_t^s$ and $\tilde{n}_t^s$ denote the optimal solution to the problem in equation (7) since untested asymptomatic agents do not know their health status. Conditional on becoming untested asymptomatic in period $t+1$, they expect to become infected symptomatic in the next period with probability π_{IS} and receive utility V_{t+2}^{IS} –defined in Section 2.4. They expect to become unobserved recovered with probability π_R and to receive the utility V_{t+2}^{UR}, which is defined for the period t as

$$V_t^{UR} = u(\tilde{c}_t^s, \tilde{n}_t^s) + \beta V_{t+1}^{UR}. \tag{9}$$

The unobserved recovered agents have never showed any symptoms and hence do not know their health status. Hence, they choose consumption and labor by solving the problem in equation (7). If the untested asymptomatic agents neither develop symptoms nor recover,

[10] Solving the imperfect information problem under full rationality requires keeping track of when agents were tested last and thereby is very cumbersome.

they expect to test positive at the end of period $t+1$ with probability $\pi^A_{P,t+1}$ and receive the utility function V^P_{t+2} in the next period.

The probabilities of testing positive for a newly infected, $\pi^T_{P,t}$ in equation (7), and for an asymptomatic agent, $\pi^A_{P,t}$ in equation (8), will be characterized in Section 3.

Budget constraint for the non-quarantined agents. The problem 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.

2.3 Tested-Positive Agents

Tested-positive agents are individuals who know they are infected even though they do not have symptoms. They 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, they will remain in their current status.

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

$$\left(1+\mu^Q_c+\alpha\mu^L_{c,t}\right)c^P_t = w^P_t n^P_t + \Gamma^Q_t, \tag{12}$$

where μ_c^Q proxies the effects of imposing a quarantine on individuals' consumption and labor decisions. 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' consumption. The tax paid by quarantined agents is rebated to them, Γ_t^Q.

2.4 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 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, they will get zero utility forever. If neither events happen, the infected symptomatic individual will not change their 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 lower when the symptoms of the disease are developed. This penalty on labor productivity is given by $\phi < 1$.

2.5 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:

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

subject to the budget constraint for non-quarantined subjects in equation (10).

2.6 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. Recall that C_t denotes consumption of non-quarantined agents. $C_t^{IS}\equiv c_t^{IS}I_t^S$ 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 the agents these taxes are levied on.

2.7 Dynamics of Agents' Types

We now describe the evolution of the six types of agents. The law of motion for the share of susceptible agents reads $S_{t+1}=S_t-T_t$, where T_t denotes the share of newly infected subjects in period t. This share is defined using the law of large number as follows: $T_t=\tau_t\cdot S_t$, where τ_t is the expected probability for susceptible individuals to become infected – defined in equation (5).

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

$$I_{t+1}^{A}=(1-\pi_{P,t}^{T})T_t+(1-\pi_{P,t}^{A})(1-\pi_{IS}-\pi_R)I_t^{A}, \tag{18}$$

This set of agents are given by those who were untested asymptomatic I_t^A 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 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

$$P_{t+1}=(1-\pi_{IS}-\pi_R)P_t+\pi_{P,t}^{T}T_t+\pi_{P,t}^{A}(1-\pi_{IS}-\pi_R)I_t^{A}. \tag{19}$$

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:

$$I^S_{t+1} = (1 - \pi_R - \pi_D)I^S_t + \pi_{IS}(I^A_t + P_t). \tag{20}$$

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

The share of unobserved recovered evolves as follows: $R^U_{t+1} = R^U_t + \pi_R I^A_t$. 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: $R^O_{t+1} = R^O_t + \pi_R(P_t + I^S_t)$. 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 vary because infected people die. The share of agents who have died by period $t + 1$ is given by $D_{t+1} = D_t + \pi_D I^S_t$.

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, $\pi^A_{P,t}$. 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, untested asymptomatic, and unobserved recovered agents. In our model, an agent can be infected and remain asymptomatic throughout their entire infection. These agents are undiscovered spreaders who keep infecting susceptible agents until they recover or get quarantined because they test positive or become symptomatic. Tests do not reveal when a positive agent was infected or whether a negative agent is still susceptible to getting 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 a 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*. We consider two levels of efficiency of the tracing technology: a *basic*

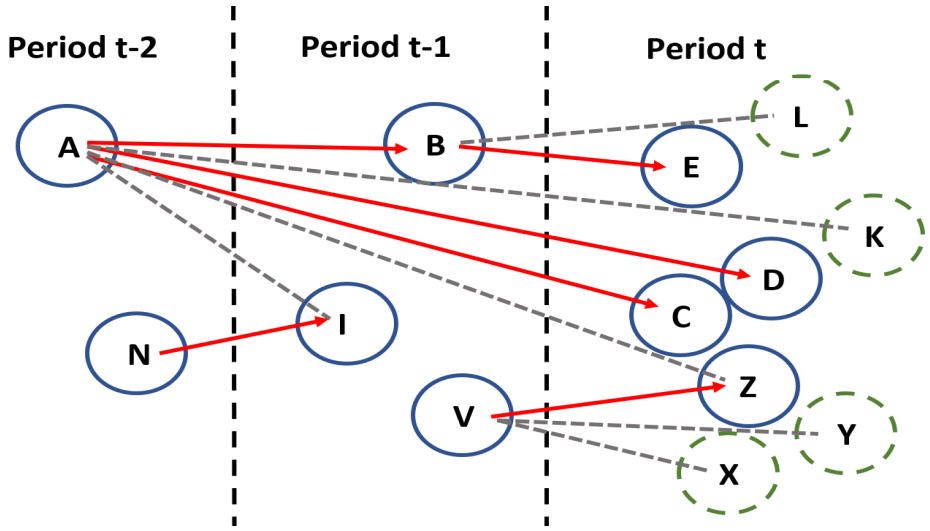

Figure 1: Example of an infection chain. The blue solid circles indicate an asymptomatic person. The green dashed circles are susceptible or recovered agents. The red lines describe an interaction that leads to an infection, while the gray lines describe an interaction that does not lead to an infection.

technology that allows health officials to trace only those contacts that have occurred during the current week and a *comprehensive technology* that allows them to trace contacts up to one week back.

It is useful to resort to a graphical example to illustrate how contact tracing works in the model. In Figure 1, agent A, who caught the virus in period $t-2$, infects agent B in period $t-1$. In the next period, agent A infects further two agents, who are denoted by C and D. At the same time, agent 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, who are denoted by dashed green circles, are agents that were not infected by meeting with one of the untested asymptomatic subjects, who are denoted by 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 belong to the same infection chain originated by subject A, cannot be traced. It is important to note that subject Z does not belong to agent A's infection chain as subject Z was infected by subject V. However, subject Z has randomly met with subject A in period t and is thereby traceable. If the comprehensive tracing technology is available, then subject B can also be traced.

Let's suppose that subject B turns symptomatic in period t while subject A is still untested asymptomatic. The basic technology would discover subject E. By allowing subject B's contacts to be traced in the earlier period $t-1$, the comprehensive technology allows

health authorities to find out that subject A is an asymptomatic spreader. Since subject A infected subject B, the detection of subject A is called *backward tracing*. The basic technology does not allow health authorities to trace backward as it takes at least one period for newly infected subjects to become symptomatic.

It is important to note that the basic tracing technology can catch asymptomatic agents who went untested in the previous periods only if these agents meet randomly with a subject who turn symptomatic in the current period. These random meetings are fairly rare, as we will show in Sections 3.1 and 4. In contrast, the comprehensive technology allows the health authorities to leverage the infection chain of the newly symptomatic agents to detect asymptomatic spreaders that were not caught in previous periods. An example is the backward tracing of agent A when agent B turns symptomatic. Hence, the comprehensive technology is more effective in detecting asymptomatic spreaders the testing system failed to catch in previous periods.

Health authorities could also launch a second round of tests by reconstructing the network of contacts of those agents who tested positive in the first round. We deal with this extension in Section 6.

Testing Probabilities The probability of catching a spreader depends on (i) the probability of tracing this subject; (ii) the testing capacity in period t, Υ_t, relative to the number of people traceable E_t; (iii) the probability of a false negative (π_F). As we will show, the efficiency of the tracing technology influences the probability of being traced and the number of traceable subjects in a given period.

Formally, for given efficiency of the tracing technology, the probability that a newly infected subject infected ($i = T$) or an untested asymptomatic subject ($i = A$) tests positive in period t is

$$\pi^i_{P,t} = \pi^i_{C,t} \cdot \pi_{T,t} \cdot (1 - \pi_F), \quad i \in \{T, A\}, \tag{21}$$

where the probability $\pi^i_{C,t}$ denotes the probability of being traced for a subject of type i and 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. The case in which all the traced subjects are quarantined is discussed in Section 6.

Coordination Failure and the Collapse of the Testing System. The magnitude of the variable Υ_t relative to the number of traceable people, E_t, plays the role of a critical

bottleneck that can lead to the collapse of the tracing and testing system in our model. Agents fail to realize that their consumption and labor decisions have externalities on the number of traceable subjects, E_t, health authorities will have to test a few periods later. This is because of two reasons. First, those agents whose health status is unknown 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 (21). 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.[12]

In the reminder of this section, we will characterize the probability for a newly infected and an untested asymptomatic subject to be traced ($\pi^T_{P,t}$ and $\pi^A_{P,t}$, respectively) under the basic tracing technology and under the comprehensive tracing technology.

3.1 Basic Contact Tracing Technology

The basic contact tracing technology allows health authorities to trace only those contacts that occur in the current week. It is useful to combine the binomial distributions in equation (1), (2), and (3) to obtain the probability for an agent who does not know their health status to meet k-times with the set of untested asymptomatic subjects while consuming, working,

[11] These externalities would not be eliminated if these subjects knew to be asymptomatic spreaders.

[12] There is another source of externality 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 our model with contact tracing and testing, these externalities do not play any significant role.

and performing other activities:

$$f_t(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} f_{c,t}(i) f_{n,t}(j) f_{o,t}(k-i-j). \tag{22}$$

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 $1-(1-\pi_{IS})^k$. Hence, the probability for a subject who does not know their health status to be traced in period t is

$$\pi_{C,t}^{S} = \pi_{C,t}^{A} = \pi_{C,t}^{UR} = \sum_{k=0}^{\varphi_C(c_t^s)+\varphi_N(n_t^s)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f_t(k), \tag{23}$$

implying that the probability of being traced is the same for the three unobserved types: susceptible (S), untested asymptomatic (A), and unobserved recovered (UR). This is because these agents consume and work the same amount as shown in Section 2.2. As a result, they will have the same number of total interactions $\varphi_C(c_t^s)+\varphi_N(n_t^s)+\varphi_O$ and the same probability of meeting with k untested asymptomatic agents.

The probability $\pi_{C,t}^{A}$ in equation (23) is the sought probability for an untested asymptomatic agent to be traced in period t.

We now work out the probability for a newly infected subject to be traced, $\pi_{C,t}^{T}$. Newly infected subjects are susceptible at the beginning of the period and become infected because they have met an untested asymptomatic individual. Thus, we have to condition the probability distribution that a susceptible agent has met k untested asymptomatic subjects in period t – $f_t(k)$ defined in equation (22)– on the fact that the newly infected agent has met at least one untested asymptomatic subject, i.e., the agent who infected them. To do so, we apply the Bayes theorem to obtain:

$$f_t^T(k) = \frac{f_t(k)\tilde{\tau}(k)}{\tau_t}, \tag{24}$$

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 (5). Following the same reasoning behind the probability in equation (23), we characterize the probability for a newly infected individual to be traced as

$$\pi_{C,t}^{T} = \sum_{k=0}^{\varphi_C(c_t^s)+\varphi_N(n_t^s)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f_t^T(k). \tag{25}$$

As noted at the beginning of this section where we analyzed Figure 1, an untested asymptomatic subject can only be traced if they have met a newly symptomatic subject randomly. The application of the Bayes theorem in equation (24) adjusts the probability distribution $f_t^T(k)$ to factor in that the newly infected subject belongs to the infection chain of an agent who was untested asymptomatic at the beginning of the period. This is important as this untested asymptomatic agent may turn symptomatic with probability π_{IS}. The event that the subject who infected the newly infected agent turns symptomatic is more likely than the joint event that an untested asymptomatic agent has randomly met another untested asymptomatic agent ($\sum_{k>1} f_t(k)$) *and* the latter agent turns symptomatic. Therefore, an untested asymptomatic is less likely to be traced than a newly infected agent under the basic tracing technology ($\pi_{C,t}^T > \pi_{C,t}^A$).

In Figure 11 of Appendix H, we show the unconditional and the conditional distributions $f_t(k)$ and $f_t^T(k)$ in one simulation where the basic contact tracing technology leads to a successfully control of the pandemic. As one can see, the probability of catching an untested asymptomatic subject is dwarfed by the fact that these subjects are very unlikely to meet randomly with other untested asymptomatic, who can turn symptomatic. Conditioning on the fact that newly infected agents have met at least one untested asymptomatic subject causes the mode of the probability $f_t^T(k)$ to shift from $k = 0$ to $k = 1$, making tracing more likely.

Probability of Testing Positive under the Basic Tracing Technology. The basic contact tracing technology endows health authorities with the list of contacts of the 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

$$E_t = \pi_{C,t}^S \cdot S_t + \pi_{C,t}^A \cdot (1 - \pi_{IS})\, I_t^A + \pi_{C,t}^{UR} \cdot R_t^U\,, \tag{26}$$

where $\pi_{C,t}^S$, $\pi_{C,t}^A$, and $\pi_{C,t}^{UR}$ are the probabilities of being traced for the three types of agents who do not know their health status. These probabilities were defined in equation (23). We adjusted the share of the untested asymptomatic subjects who were exposed by taking out those who have revealed symptoms ($\pi_{IS} I_t^A$) in period t.

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

$$\pi_{t,T} = \min\left(1, \frac{\Upsilon_t}{E_t}\right), \tag{27}$$

where recall $\Upsilon_t \geq 0$ denotes the testing capacity of policymakers in every period, which is an exogenous variable. We substitute equations (25) and (27) into equation (21) to obtain the probability of testing positive for newly infected subjects, $\pi^T_{P,t}$. Substituting both the probability $\pi^A_{C,t}$ of equation (23) and the conditional probability of being tested of equation (27) into equation (21) allows us to pin down the probability of testing positive for subjects infected in earlier periods, $\pi^A_{P,t}$. The probabilities $\pi^A_{P,t}$ and $\pi^T_{P,t}$, in turn, pin down the dynamics of types in equations (18) and (19) 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 in period t. The objective of this section is to characterize the probabilities for newly infected and untested asymptomatic subjects to be traced based on contacts established in period $t-1$. The probability for these two subjects to be traced based on the contacts they had in period t is identical to the ones derived before under the basic contact tracing technology.

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) and 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 untested asymptomatic, T for newly infected, and S for susceptible. Note that the Type-A and Type-T agents have not 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 the three types of subjects had in period $t-1$ with agents who became infected before period $t-1$; (ii) and T-links mean those contacts that the three subjects had in period $t-1$ with agents that become infected in period $t-1$. These letters denote the health status of the subjects with which the three types of agents have interacted in period $t-1$: A for untested asymptomatic 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.[13]

[13]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

Type-A agents: asymptomatic subjects in period t who were infected earlier than t-1. 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 links create the possibility of *backward tracing*, which was illustrated in the graphical example of Figure 1. 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

$$f_{t-1}^{A,T}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} f_{c,t-1}^{A,T}(i) f_{n,t-1}^{A,T}(j) f_{o,t-1}^{A,T}(k-i-j), \tag{28}$$

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

$$f_{c,t-1}^{A,T}(k) \equiv \mathcal{B}\left(k, \varphi_c\left(c_{t-1}^s\right), \frac{[\tau + (1-\tau)\tau_{t-1}]C_{t-1}^S}{C_{t-1}}\right), \tag{29}$$

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 them. In this case, the asymptomatic subject has added one more case to their own infection link which could potentially make them traceable via backward tracing.[14] 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 random, non-infectious meeting with an agent that will be infected by someone else. This random, non-infectious meeting creates a traceable link for the Type-A agent in period t even though this meeting does not belong to Type-A agent's infection chain. In the example illustrated in Figure 1, this second case corresponds to the meeting between subject A and subject I in period $t-1$. This meeting is not infectious as subject I is infected by subject N in the same period.

link) does not affect the probability of being traced in period t.

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

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, 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 random, non-infectious meeting with an agent that will later become symptomatic is quite tiny.

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

$$f_{t-1}^{A,A}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} \mathcal{B}\left(i, \varphi_C(c_{t-1}^s), \frac{C_{t-1}^A}{C_{t-1}}\right) \mathcal{B}\left(j, \varphi_N(n_{t-1}^s), \frac{N_{t-1}^A}{N_{t-1}}\right) \mathcal{B}\left(k-i-j, \varphi_O, \frac{I_{t-1}^A}{Pop_{t-1}}\right). \tag{30}$$

Since A-links involve subjects who are already infected, all meetings are random (i.e., non-infectious).

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

$$f_{t-1}^{T,T}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} \mathcal{B}\left(i, \varphi_C(c_{t-1}^s), \frac{c_{t-1}^s T_{t-1}}{C_{t-1}}\right) \mathcal{B}\left(j, \varphi_N(n_{t-1}^s), \frac{n_{t-1}^s T_{t-1}}{N_{t-1}}\right) \times \mathcal{B}\left(k-i-j, \varphi_O, \frac{T_{t-1}}{Pop_{t-1}}\right), \tag{31}$$

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 can be constructed from the probability for Type-A agents to have k A-links, $f_{t-1}^{A,A}$ in equation (30), by applying the Bayes theorem

$$f_{t-1}^{T,A}(k) = \frac{f_{t-1}^{A,A}(k)\,\tilde{\tau}(k)}{\tau_{t-1}}, \tag{32}$$

where the variable $\tilde{\tau}(k)$ is defined in equation (24) and the rate τ_t is the average infection rate defined in equation (5). Correcting the distribution $f_{t-1}^{A,A}$ is needed because, unlike Type-A

agents, Type-T agents must have met at least one untested asymptomatic in period $t-1$; i.e., the individual who has infected the Type-T agent.

Analogously to the distribution in equation (24), the application of the Bayes theorem adjusts the distribution $f_{t-1}^{A,A}$, which only reflects random meetings, to factor in that every Type-T agent belongs to the infection chain of an agent who was untested symptomatic in period $t-1$.

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

$$f_{t-1}^{S,A}(k) = \frac{f_{t-1}^{A,A}(k)\,(1-\tilde{\tau}(k))}{1-\tau_{t-1}}. \tag{33}$$

Time Adjustments and Active Links. 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. Furthermore, 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 t because some of the 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.

We show how to condition the six probability distributions, $f_{t-1}^{l,i}$, with $i \in \{A,T,S\}$ $l \in \{A,T\}$ on these two events in Appendix A. These adjustments lead to the probability of being traced in period t for Type-A, Type-T, and Type-S agents because of the contacts they established in period $t-1$. We denote these probabilities by $\pi_{C,t}^{1,i}$, with $i \in \{A,T,S\}$. Notationally, these probabilities have the subscript t to remind that tracing is carried out in period t. The probabilities of being traced for an asymptomatic agent or a newly infected agent through their contacts established in the current week t are denoted by $\pi_{C,t}^{0,i}$, with

$i \in \{A, T\}$ and are the same as $\pi^i_{C,t}$, with $i \in \{A, T\}$, derived in Section 3.1.

Probability of Testing Positive under the Comprehensive Tracing Technology. We use the decomposition in equation (21) to define the probability of being tested positive at time t through contacts established in the previous period[15]

$$\pi^{j,i}_{P,t} = \pi^{j,i}_{C,t} \cdot \pi^{j}_{t,T} \cdot (1 - \pi_F), \quad i \in \{A, T, S\} \quad j \in \{0, 1\}, \tag{34}$$

where j denotes the period $t - j$ when the contacts relevant for tracing were established. So we combine the probability of being traced, $\pi^{j,i}_{C,t}$, 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. 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 (26). We denote the subjects who in period $t-1$ have met agents who become symptomatic in period t, as E^1_t, which is formally defined in Appendix D.

Tests are administered following a Pecking order: First government uses all the available tests to check the current period's contacts and if any tests are left, they are used to test the previous period's contacts. Pecking order is optimal because subjects who were untested asymptomatic in the previous period may have recovered before testing is performed.

The probability of being tested conditional on being traceable in period t is denoted by $\pi^0_{t,T}$ and defined in equation (27). Given the Pecking order, the probability of being tested conditional on being traceable in period $t-1$ is given by

$$\pi^1_{T,t} = \min\left(1, \frac{\max\left(0, \Upsilon_t - E^0_t\right)}{E^1_t}\right). \tag{35}$$

Note that the probability of testing positive defined in equation (34) 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 (18) and (19), which requires us to know the *average* probability for an untested asymptomatic subject to test positive in period t ($\pi^A_{P,t}$) and the average probability for newly infected subjects to test positive in period t ($\pi^T_{P,t}$).

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

$$\pi^A_{P,t} = \frac{I^A_{t-1}\left(1 - \pi_{IS} - \pi_R\right)\left(1 - \pi^A_{P,t-1}\right)}{I^A_t} \cdot \left[\pi^{0,A}_{P,t} + \left(1 - \pi^{0,A}_{C,t}\right) \cdot \pi^{1,A}_{P,t}\right] \tag{36}$$

[15]Note that $\pi^{0,S}_{C,t}$ is the probability for a susceptible agent to test positive in period t, which is zero.

$$+\frac{T_{t-1}\left(1-\pi^{T}_{P,t-1}\right)}{I^{A}_{t}}\cdot\left[\pi^{0,A}_{P,t}+(1-\pi^{0,A}_{C,t})\cdot\pi^{1,T}_{P,t}\right],$$

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.[16] The two bits outside the square brackets weigh 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 transition in equation (18) 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

$$\pi^{T}_{P,t}=\pi^{0,T}_{P,t}+(1-\pi^{0,T}_{C,t})\cdot\pi^{1,S}_{P,t}. \tag{37}$$

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 agent 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. More details are in Appendix F.

We use the approximated infection rate in equation (6) to solve the decision problem of the agents (see Section 2.2) and to compute the dynamics of types in Section 2.7. To pin down the probabilities of getting tested ($\pi^{T}_{P,t}$ and $\pi^{A}_{P,t}$) in Section 3, we use the exact definition of the rate τ_t in equation (5).

The calibrated parameters of the model are summarized in Table 1. The economic parameters are calibrated based on Eichenbaum, Rebelo and Trabandt (2020*a*). We set the weekly discount factor to $0.96^{1/52}$. This number is standard and implies the value of a statistical life of roughly 10 million 2019 U.S. dollars, which is in line with what other studies assume (e.g., Eichenbaum, Rebelo and Trabandt 2020*a*).[17] Productivity, A, is set to match a yearly income of \$58,000. The scale parameter of labor disutility, θ, is calibrated so that

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

[17] 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.

Table 1: Calibration

Parameters	Sign	Value	Target / Source
(a) Economic parameters			
Discount factor	β	$0.96^{1/52}$	Conventional discount factor
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-based interactions 33%
Interaction via labor	φ_N	0.39	Labor-based interactions 33%
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	Quarantine lowers C and L by 30%
Productivity symptomatic	ϕ	0.8	Eichenbaum et al. (2020a)
Lockdown effect in quarantine	α	0	No impact besides quarantine
Initial infection	ϵ	0.1%	Infections March 16 2020

agents work on average 28 hours per week. The Frisch labor elasticity φ is 0.5.

The epidemiological parameters are calibrated to the recent COVID-19 crisis in the US. A key epidemiological parameter is τ, which is the probability that one interaction with an infected subject results in an infection (see Assumption 4). We set this parameter to 5% based on evidence from the World Health Organization (2020).[18] 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. The original step functions $\varphi_C(c_t)$ and $\varphi_N(n_t)$ are shown in the Appendix H (see Figure 10). We set the parameters φ_C and 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).[19]

[18]This WHO report analyses the probability of an infection for an individual that had close contact with an individual who tested positive for 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.

[19]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 our main results significantly, 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).

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 their lifetime in a population where everybody is susceptible and no containment measures (including testing) are taken.[20] We set the basic reproduction number to 2 in line with the evidence about the early transmission of COVID-19.[21] 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.[22]

In line with evidence from the World Health Organization (2020), we choose that an agent recovers on average after 18 days, which implies $\pi_R = 7/18$.[23] 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*a*).[24] 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.[25]

In the model, symptomatic agents are subject to a labor productivity penalty, ϕ. We calibrate the penalty $\phi = 0.8$ based on Eichenbaum, Rebelo and Trabandt (2020*a*). 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 positive-tested agent is lower than those of 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

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

[21] 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.

[22] For the first nine cases in the U.S., 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.

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

[24] 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 positive-tested contacts of indexed COVID-19 cases did not develop symptoms for individuals below 60 years of age. Nishiura et al. (2020*a*) suggest a 69% infection rate based on evacuation flights of Japenese passengers data from China.

[25] This value is supported by Nishiura et al. (2020*b*), who find a range of 0.3% to 0.6% with Japanese data and by Streeck et al. (2020) who estimate 0.36% based on German data.

the amount of infections adjusted for unreported cases on March 16, 2020.

5 Quantitative Analysis of Contact Tracing

To better understand the results shown in this section, 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 their illness.

The effective reproduction rate is affected by the efficiency of the tracing technology, the testing capacity (Υ_t), the amount of economic interactions that depend on non-quarantined agents' decision to consume and work, and the stringency of the 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

$$\begin{aligned} R_t^E &= (1-\pi_{P,t-1}^T)\big[\tau_t + (1-\pi_{IS}-\pi_R)\left(1-\pi_{P,t}^A\right)\tau_{t+1} + \\ &\qquad (1-\pi_{IS}-\pi_R)^2\left(1-\pi_{P,t}^A\right)(1-\pi_{P,t+1}^A)\tau_{t+2} + \dots\big] \\ &= (1-\pi_{P,t-1}^T)\sum_{j=0}^{\infty}\left(\tau_{t+j}(1-\pi_{IS}-\pi_R)^j \Pi_{k=0}^{j}\left(1-\pi_{P,t+k}^A\right)\right). \end{aligned} \tag{38}$$

The effective reproduction number conflates current and future probabilities for non-quarantined infected agents to be caught. The efficiency of the tracing technology and the testing capacity (Υ_t) mainly influence the effective reproduction number by affecting the probability for newly infected subjects and for untested asymptomatic subjects to test positive at the end of period t; that is, $\pi_{P,t}^T$ and $\pi_{P,t}^A$, 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, $\pi_{P,t-1}^T$, than to changes in the future probability for an untested asymptomatic agent to test positive, $\pi_{P,t+k}^A$. The reason is that asymptomatic agents may turn symptomatic or recover in every future period and, when they do, they will stop infecting other people. The transience of the status of being asymptomatic, which is captured by the term $(1-\pi_{IS}-\pi_R)$ in equation (38), 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 shown 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.[26] 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.[27] 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 crisis fades away when 60% of the population becomes infected and herd 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 lowering consumption and labor. As a consequence, the economy goes through an extremely dreadful 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 chances of getting infected are reduced, leading agents to lower 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, herd immunity is reached with around 20% of infected agents –three times less than the share of infected needed in the case of 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 tech-

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

[27] More variables are plotted in Appendix H.

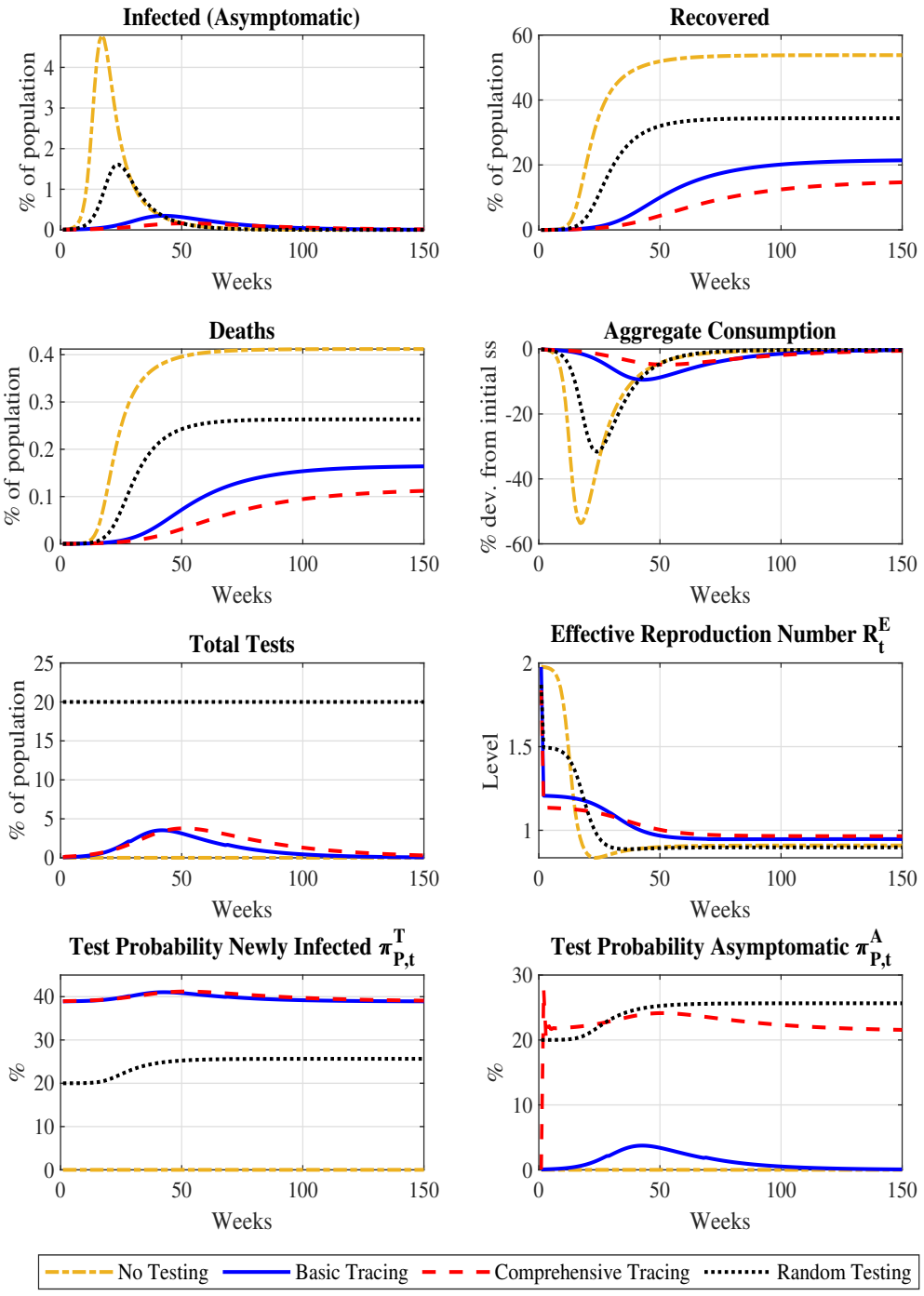

Figure 2: Comparison of different testing strategies with unconstrained number of tests for contact tracing: No testing (blue solid), basic tracing (red dashed) corresponds to current week contact tracing, comprehensive tracing (green dash-dotted) corresponds to current and previous week contact tracing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

nologies require testing at most 4% of the population in a week, which is substantially less than the number of tests we assume for random testing.

The timing of the testing varies somewhat across these two tracing technologies. The basic tracing technology requires performing more tests a few periods after the pandemic has started (around period 30) relative to the comprehensive technology.

While this result may seem odd at first, it is important to recall that the basic technology is less effective than the comprehensive technology in detecting untested asymptomatic subjects because the basic technology can only trace these subjects through random meetings. As explained in Section 3.1, this type of meetings are quite rare.[28] As a result, in the lower right panel of Figure 2, the share of untested asymptomatic subjects detected by the basic tracing technology is very low compared to the levels attained 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 faster increase in the number of traceable subjects, E_t, and hence more tests performed a few periods after the pandemic has started (around period 30). In short, under the basic technology, you trace and test fewer people at the onset of the pandemic and this requires you to test more people later on.

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 the entire population –more than twice as many deaths as under the comprehensive contact-tracing technology.

What explains the spectacular failure of random testing? To answer this question, one should look at the two bottom graphs of Figure 2, which show the share of newly infected asymptomatic subjects (left plot) and the share of untested asymptomatic subjects (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 containing the economic costs and the mortality of the pandemic.

That the probability of catching the newly infected asymptomatic subjects turns out to

[28]The probabilities of these random meetings in period $t = 20$ and in period $t = 40$, when the pandemic picks up a little, are shown in the left plot of Figure 11 of Appendix H.

be key to controlling the pandemic should not come as a surprise. We already noted that the reproduction number defined in equation (38) is more sensitive to changes in the probability for newly infected agents to test positive, $\pi^T_{P,t}$, than to changes in the probability for untested asymptomatic subjects to test positive, $\pi^A_{P,t}$.

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 rapid increase in the testing scale after the thirtieth week of the pandemic. This increase in the number of tests administered is needed to compensate for the poor performance of this technology in catching the untested asymptomatic subjects, as reflected in the low value of $\pi^A_{P,t}$ 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 a daily capacity of only 30,000 tests available at the onset of the pandemic crisis. This capacity then increased linearly up to 1 million tests 28 weeks later.[29] Afterwards, testing capacity is assumed to increase at a steady pace until week 52, after which it stays put.

Looking at the third left plot in Figure 3, the basic contact-tracing technology (blue solid line) requires testing to accelerate after period 30 to compensate for its inability to catch untested asymptomatic subjects. However, testing capacity is not growing fast enough and the blue solid line hits the yellow starred line, denoting the U.S. testing scale (Υ_t). As testing capacity becomes binding, the testing system collapses, as captured by the rapid drop in the probability of catching a newly infected subject ($\pi^T_{P,t}$). 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) from ever becoming binding. The lockdown greatly mitigates the pandemic recession and reduces

[29]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 U.S. conducted 6,936,961 tests, which corresponds approximately to 991,000 daily tests.

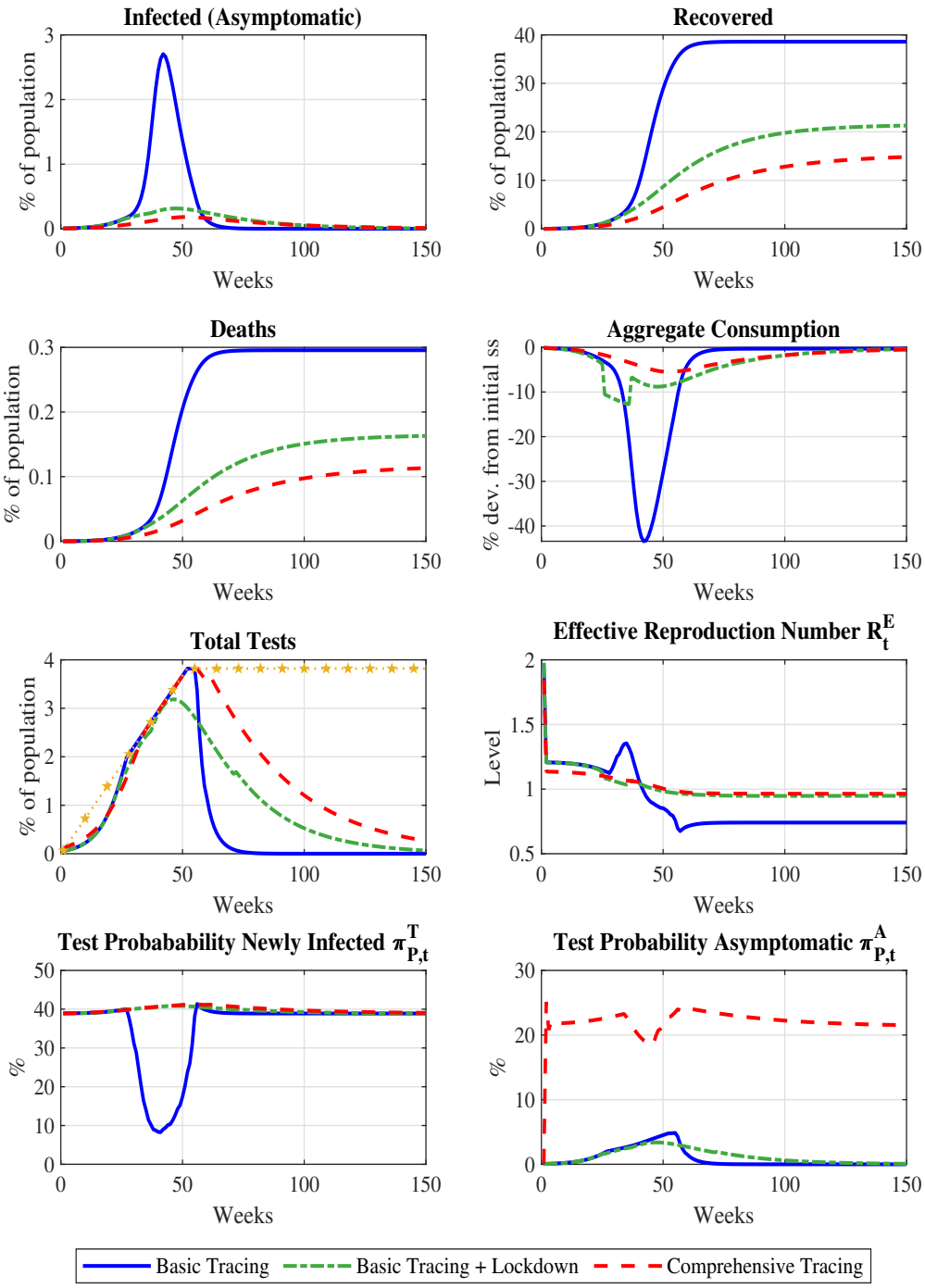

Figure 3: Comparison of different testing strategies with limited tests: Comprehensive tracing (blue solid line) is previous and current week tracing, basic tracing (red dashed line) is current week tracing and in the green dash-dotted basic tracing is combined with a 1 year lockdown. In the fifth plot,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

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 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 30 as in the case of the basic tracing technology. As a result, under the comprehensive tracing technology, 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 Pecking order, there is no effect on the probability of detecting newly infected agents, π_t^T, which, as we have already pointed out, is essential for successful management of the pandemic. Thus, the effective reproduction number hardly budges and the effects on consumption and mortality are only moderate.

5.3 The Optimal Stringency of the Lockdown

We now turn our attention to the optimal stringency of the lockdown. The stringency is captured by the size of the consumption tax μ^L. The duration of the lockdown is kept fixed at 26 periods, $T_\mu = 26$.[30]

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

When no lockdown is imposed ($\mu^L = 0$), the basic tracing technology alone cannot prevent the collapse 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 stringency 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 stringency of the lockdown is increased, welfare increases as fewer people will be killed by the pandemic. However, consumption and labor fall steadily.

[30]Our conclusions 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 $T_\mu = 52$.

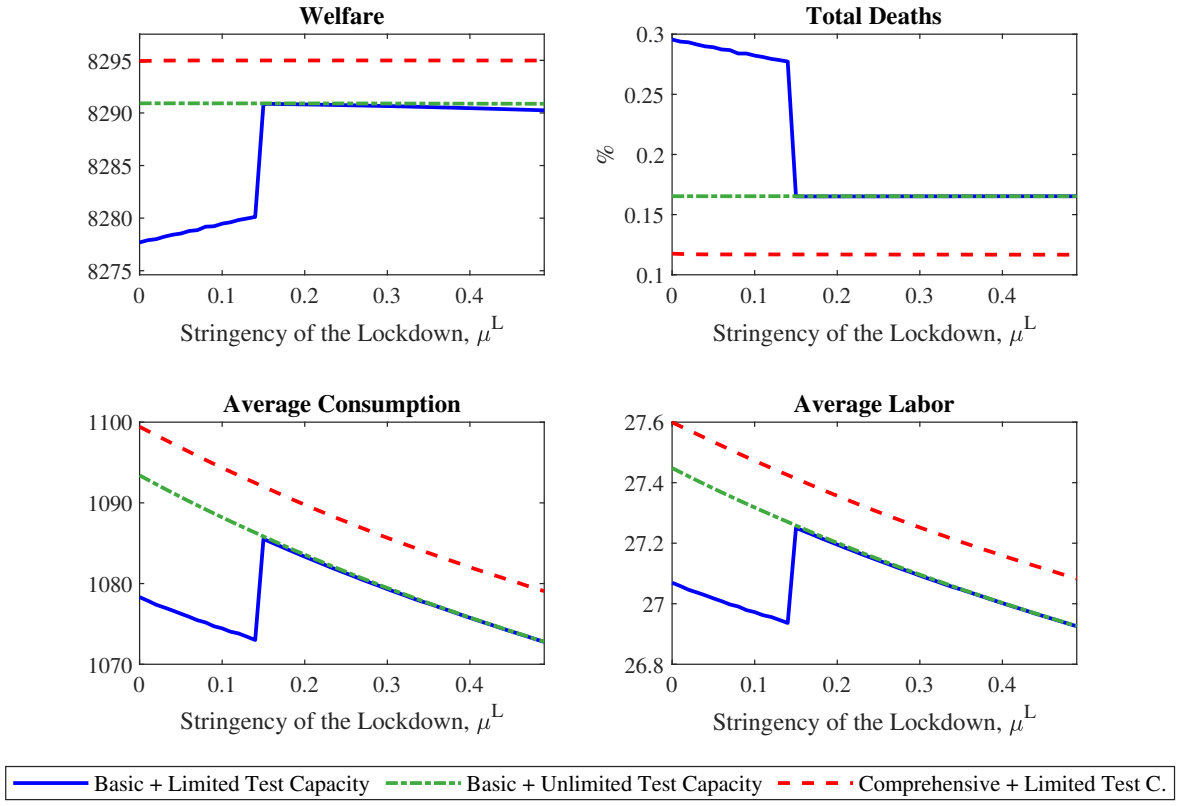

Figure 4: Comparison of different testing strategies under varying lockdown stringency imposed for the first 26 weeks. Welfare in week 1, accumulated deaths, aggregate consumption and aggregate labor averaged over the 250 week horizon are reported.

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 increase in welfare is due to the 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 dashed-dotted line). This happens because the optimal lockdown reduces agents' individual consumption and labor so as to solve the coordination failure threatening the viability of the testing system. By preserving the correct functioning of the testing system, agents can consume and work more later when more tests are available and the infection rate does not increase. This result is reflected in the discrete increase in consumption and employment as the stringency of the lockdown is raised to its optimal level.

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 stringency 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 fewer infected cases and hence to a lower death toll.

Remarkably, lockdowns have virtually no effect on welfare when the tracing technology is comprehensive because this more efficient 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 subjects ($\pi^A_{P,t}$), 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 tracing and unlimited testing, no lockdown is the optimal choice.

6 Extensions

Our objective was to construct a macro-epidemiological model to serve as a general framework to study the efficacy (or lack thereof) of contact tracing and testing. With this goal in mind, we tried to keep the model as clean as possible. That said, our model can be extended in a number of interesting directions. We consider three extensions that can be studied by tweaking our methodology.

Superspreaders. An interesting extension is to consider the case of superspreaders – a small number of carriers ending up infecting many individuals. Superspreading may be linked to subjects who particularly enjoy social activities or have jobs that expose them to a large number of people every day. It may also be linked to large gatherings. Since superspreaders seem to have played a key role in spreading the coronavirus, we could introduce a new type of agents, who either enjoy consumption more or draw less disutility from working than the other set of agents. The presence of superspreader agents would make contact tracing even more effective than random testing. As one of these superspreaders starts showing symptoms, policymakers can detect an outsized number of newly infected agents from tracing the contacts of the superspreader. This is because superspreaders' infection chain is larger than that of normal spreaders.[31] Our methodology is general and can be applied to models featuring households or firms heterogeneity.

All Exposed Contacts Quarantined. We assumed that health authorities can only impose a quarantine on people who get traced and tested positive or start developing some symptoms. We could have assumed instead that all the exposed subjects are quarantined even if the testing capacity is binding and some of them cannot be tested. This extension

[31] If policymakers can observe if an agent is a superspreader, they should first try to trace and test the superspreaders. This strategy would obviously make contact tracing even more effective.

would have made our model less clean by adding a new additional type on top of the currently featured six types. At the same time, it should be noted that this extension would not have added much to our analysis, whose objective is to study a new externality that can explain why contact tracing can fail. For our argument to hold, there just needs to be a constraint on how many people can be traced and isolated in every period. In the real world, there are a large variety of such constraints. An example is the health authorities' capacity to process all contacts traced by the tracing technology. If the "logarithmic increase" of infections is not prevented, these processing constraints will soon become binding. In the presence of this type of constraints, the key externalities studied in this paper will emerge, causing the tracing system to fail. We decided to use the testing capacity as the key constraint in our model because it is relatively easy to calibrate.

Multiple Rounds of Tracing and Testing. We assumed that health officials cannot perform multiple rounds of testing (i.e., testing the contacts of those who tested positive in the previous round). While our methodology can be extended to model multiple rounds of contact tracing and testing, considering this extension in the paper would not change our main conclusions. With the basic tracing technology, multiple rounds of testing can provides only a minimal contribution because policymakers can mostly catch newly infected subjects who did not have time to infect anyone else. With the comprehensive tracing technology, policymakers can catch untested asymptomatic agents who had time to infect someone else in the previous period. However, as shown in Section 4, implementing this technology already attains a close-to-optimal control of the virus. Hence, any gain from performing additional rounds of tracing and testing can only be incremental.

7 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 number of subjects that will need to be traced and tested. These externalities can threaten the viability of the testing system. A timely, appropriately sized lockdown can correct the implications of these externalities for the economic and death toll of the pandemic.

References

Acemoglu, Daron, Victor Chernozhukov, Iván Werning, and Michael D Whinston. 2020. "Optimal targeted lockdowns in a multi-group SIR model." *NBER Working Paper*, 27102.

Akbarpour, Mohammad, Cody Cook, Aude Marzuoli, Simon Mongey, Abhishek Nagaraj, Matteo Saccarola, Pietro Tebaldi, Shoshana Vasserman, and Hanbin Yang. 2020. "Socioeconomic Network Heterogeneity and Pandemic Policy Response." *University of Chicago, Becker Friedman Institute for Economics Working Paper*, , (2020-75).

Alvarez, Fernando E, David Argente, and Francesco Lippi. 2020. "A simple planning problem for covid-19 lockdown." National Bureau of Economic Research.

Atkeson, Andrew. 2020. "What will be the economic impact of covid-19 in the us? rough estimates of disease scenarios." National Bureau of Economic Research.

Atkeson, Andrew, Michael Droste, Michael J Mina, and James H Stock. 2020. "Economic Benefits of COVID-19 Screening Tests."

Azzimonti, Marina, Alessandra Fogli, Fabrizio Perri, and Mark Ponder. 2020. "Pandemic Control in ECON-EPI Networks." National Bureau of Economic Research.

Baqaee, David, Emmanuel Farhi, Michael J Mina, and James H Stock. 2020*a*. "Reopening Scenarios." National Bureau of Economic Research.

Baqaee, David, Emmanuel Farhi, Michael Mina, and James H Stock. 2020*b*. "Policies for a second wave." *Brookings Papers on Economic Activity*.

Berger, David W, Kyle F Herkenhoff, Chengdai Huang, and Simon Mongey. 2020. "An seir infectious disease model with testing and conditional quarantine." National Bureau of Economic Research.

Bethune, Zachary A, and Anton Korinek. 2020. "Covid-19 infection externalities: Trading off lives vs. livelihoods." National Bureau of Economic Research.

Brotherhood, Luiz, Philipp Kircher, Cezar Santos, and Michèle Tertilt. 2020. "An economic model of the Covid-19 epidemic: The importance of testing and age-specific policies."

Burke, Rachel M, Sharon Balter, Emily Barnes, Vaughn Barry, Karri Bartlett, Karlyn D Beer, Isaac Benowitz, Holly M Biggs, Hollianne Bruce, Jonathan Bryant-Genevier, et al. 2020. "Enhanced contact investigations for nine early travel-related cases of SARS-CoV-2 in the United States." *medRxiv*.

Chari, Varadarajan V, Rishabh Kirpalani, and Christopher Phelan. 2020. "The hammer and the scalpel: On the economics of indiscriminate versus targeted isolation policies during pandemics." National Bureau of Economic Research.

Eichenbaum, Martin S, Sergio Rebelo, and Mathias Trabandt. 2020*a*. "The Macroeconomics Of Epidemics." National Bureau of Economic Research.

Eichenbaum, Martin S, Sergio Rebelo, and Mathias Trabandt. 2020*b*. "The Macroeconomics of Testing During Epidemics."

Farboodi, Maryam, Gregor Jarosch, and Robert Shimer. 2020. "Internal and external effects of social distancing in a pandemic." National Bureau of Economic Research.

Favero, Carlo A, Andrea Ichino, and Aldo Rustichini. 2020. "Restarting the economy while saving lives under Covid-19."

Ferguson, Neil M, Derek AT Cummings, Christophe Fraser, James C Cajka, Philip C Cooley, and Donald S Burke. 2006. "Strategies for mitigating an influenza pandemic." *Nature*, 442(7101): 448–452.

Fernández-Villaverde, Jesús, and Charles I Jones. 2020*a*. "Estimating and Simulating a SIRD Model of COVID-19 for Many Countries, States, and Cities." National Bureau of Economic Research.

Fernández-Villaverde, Jesús, and Charles I Jones. 2020*b*. "Macroeconomic Outcomes and COVID-19: A Progress Report." *Brookings Papers on Economic Activity*.

Fetzer, Thiemo, and Thomas Graeber. 2020. "Does Contact Tracing Work? Quasi-Experimental Evidence from an Excel Error in England."

Galeotti, Andrea, Jakub Steiner, and Paolo Surico. 2020. "Merit of Test: Perspective of Information Economics."

Glover, Andrew, Jonathan Heathcote, Dirk Krueger, and José-Víctor Ríos-Rull. 2020. "Health versus wealth: On the distributional effects of controlling a pandemic." National Bureau of Economic Research.

Gourinchas, Pierre-Olivier. 2020. "Flattening the Pandemic and Recession Curves." University California Berkeley mimeo.

Gudbjartsson, Daniel F, Agnar Helgason, Hakon Jonsson, Olafur T Magnusson, Pall Melsted, Gudmundur L Norddahl, Jona Saemundsdottir, Asgeir Sigurdsson, Patrick Sulem, Arna B Agustsdottir, et al. 2020. "Spread of SARS-CoV-2 in the Icelandic population." *New England Journal of Medicine*.

Guerrieri, Veronica, Guido Lorenzoni, Ludwig Straub, and Ivan Werning. 2020. "Macroeconomic Implications of COVID-19: Can Negative Supply Shocks Cause Demand

Shortages?" National Bureau of Economic Research Working Paper 26918.

Hacioglu, Sinem, Diego Känzig, and Paolo Surico. 2020. "The Distributional Impact of the Pandemic."

Hall, Robert E, Charles I Jones, and Peter J Klenow. 2020. "Trading off consumption and covid-19 deaths." National Bureau of Economic Research.

Hornstein, Andreas. 2020. "Social Distancing, Quarantine, Contact Tracing, and Testing: Implications of an Augmented SEIR-Mode." Technical Report, Federal Reserve Bank of Richmond.

Hortaçsu, Ali, Jiarui Liu, and Timothy Schwieg. 2020. "Estimating the fraction of unreported infections in epidemics with a known epicenter: an application to covid-19." National Bureau of Economic Research.

Kaplan, Greg, Benjamin Moll, and Gianluca Violante. 2020. "The great lockdown and the big stimulus: Tracing the pandemic possibility frontier for the US." *NBER Working Paper*, , (w27794).

Krueger, Dirk, Harald Uhlig, and Taojun Xie. 2020. "Macroeconomic dynamics and reallocation in an epidemic." National Bureau of Economic Research.

Li, Qun, Xuhua Guan, Peng Wu, Xiaoye Wang, Lei Zhou, Yeqing Tong, Ruiqi Ren, Kathy SM Leung, Eric HY Lau, Jessica Y Wong, et al. 2020. "Early transmission dynamics in Wuhan, China, of novel coronavirus–infected pneumonia." *New England Journal of Medicine*.

Moser, Christian A, and Pierre Yared. 2020. "Pandemic lockdown: The role of government commitment." National Bureau of Economic Research.

Nishiura, Hiroshi, Tetsuro Kobayashi, Takeshi Miyama, Ayako Suzuki, Sung-mok Jung, Katsuma Hayashi, Ryo Kinoshita, Yichi Yang, Baoyin Yuan, Andrei R Akhmetzhanov, et al. 2020*a*. "Estimation of the asymptomatic ratio of novel coronavirus infections (COVID-19)." *International journal of infectious diseases*, 94: 154.

Nishiura, Hiroshi, Tetsuro Kobayashi, Yichi Yang, Katsuma Hayashi, Takeshi Miyama, Ryo Kinoshita, Natalie M Linton, Sung-mok Jung, Baoyin Yuan, Ayako Suzuki, et al. 2020*b*. "The rate of underascertainment of novel coronavirus (2019-nCoV) infection: estimation using Japanese passengers data on evacuation flights."

Parran, Thomas. 1937. "Shadow on the Land: Syphilis." *Reynal and Hitchcock*.

Piguillem, Facundo, and Liyan Shi. 2020. "Optimal COVID-19 quarantine and testing policies."

Poletti, Piero, Marcello Tirani, Danilo Cereda, Filippo Trentini, Gior-

gio Guzzetta, Giuliana Sabatino, Valentina Marziano, Ambra Castrofino, Francesca Grosso, Gabriele Del Castillo, et al. 2020. "Probability of symptoms and critical disease after SARS-CoV-2 infection." *arXiv preprint arXiv:2006.08471*.

Pung, Rachael, Calvin J Chiew, Barnaby E Young, Sarah Chin, Mark IC Chen, Hannah E Clapham, Alex R Cook, Sebastian Maurer-Stroh, Matthias PHS Toh, Cuiqin Poh, et al. 2020. "Investigation of three clusters of COVID-19 in Singapore: implications for surveillance and response measures." *The Lancet*.

Romer, Paul. 2020. "How to re-start the economy after COIVD-19." *Online Lecture for the Bendheim Center for Finance at Princeton University, 3 April*.

Streeck, Hendrik, Bianca Schulte, Beate Kuemmerer, Enrico Richter, Tobias Höller, Christine Fuhrmann, Eva Bartok, Ramona Dolscheid, Moritz Berger, Lukas Wessendorf, et al. 2020. "Infection fatality rate of SARS-CoV-2 infection in a German community with a super-spreading event." *medrxiv*.

Wang, Chaolong, Li Liu, Xingjie Hao, Huan Guo, Qi Wang, Jiao Huang, Na He, Hongjie Yu, Xihong Lin, An Pan, et al. 2020. "Evolving epidemiology and impact of non-pharmaceutical interventions on the outbreak of Coronavirus disease 2019 in Wuhan, China." *MedRxiv*.

World Health Organization. 2020. "Report of the WHO-China Joint Missionon Coronavirus Disease 2019 (COVID-19)."

Zhang, Sheng, MengYuan Diao, Wenbo Yu, Lei Pei, Zhaofen Lin, and Dechang Chen. 2020. "Estimation of the reproductive number of novel coronavirus (COVID-19) and the probable outbreak size on the Diamond Princess cruise ship: A data-driven analysis." *International journal of infectious diseases*, 93: 201–204.

APPENDICES

A Comprehensive Contact Tracing Technology

In this appendix we complete the derivation of the probability of testing positive for newly infected and untested asymptomatic agents under the comprehensive contact tracing technology.

Conditioning on Type-A and Type-T remaining untested asymptomatic 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 did not test positive at the end of period $t-1$ and, thereby, remain untested asymptomatic through period t.

We rely on the Bayes theorem to condition the probability distributions for Type-A and Type-T agents on not getting 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.[32] 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 $f_{t-1|t}^{S,A}(k)=f_{t-1}^{S,A}(k)$.

[32]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).

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 t because some of the 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 and can make 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:

$$g^{i,T}_{t-1}(\underline{k}_{t-1}|k_{t-1}) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}, \left(1-\pi^{T}_{P,t-1}(i)\right)\right), \tag{A.3}$$

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, $1-\pi^{T}_{P,t-1}(i)$, for each type of agent $i \in \{A,T,S\}$. Note that these probabilities depend on the Type i of the agent establishing the contact with newly infected agents (the T-link). These probabilities are derived in Appendix B.

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

$$g^{i,T}_{t-1}(\underline{k}_{t-1}) = \sum_{k=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_o} g^{i,T}_{t-1}(\underline{k}_{t-1}|k) f^{i,j}_{t-1|t}(k), \quad i \in \{A,T,S\}. \tag{A.4}$$

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 their 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.[33] 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.

[33]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.

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:

$$g^{i}_{t-1}(\underline{k}_{t-1}) = \sum_{j=1}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} g^{i,T}_{t-1}(j) g^{i,A}_{t-1}(\underline{k}_{t-1}-j), \quad i \in \{A,T,S\}. \tag{A.5}$$

We take the same step shown in equation (25) 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

$$\pi^{1,i}_{C,t} = \sum_{\underline{k}=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} \left[1-(1-\pi_{IS})^{\underline{k}}\right] g^{i}_{t-1}(\underline{k}), \quad i \in \{A,T,S\}. \tag{A.6}$$

These are the probabilities that Type-A, Type-T, Type-S agents become traceable in period t because of their contacts in period $t-1$. These probabilities are used in the main text to define the probability of testing positive for these three type of agents. See equation (34).

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), $\pi^T_{P,t-1}(i)$, 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. In the main text we defined this probability, which we denote with $\pi^T_{P,t-1}$, in equation (37).

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

This is the probability to be used in the conditional distribution of active T-links introduced in equation (A.3) 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

random 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 they have 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 (25) 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 (25), 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 them in period $t-1$.[34]

The second step is to obtain the probability of testing positive conditional on being traced, which is precisely the familiar step taken in equation (21): $\tilde{\pi}^{0,T}_{P,t-1} = \tilde{\pi}^{0,T}_{C,t-1} \cdot \pi^0_{t-1,T} \cdot (1-\pi_F)$. 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.[35]

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

[35] 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 $t-1$.

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 they 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:[36]

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

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 their 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

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

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 $t-1$

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

where $i \in \{A,T,S\}$ and $\iota^i_{t-1}(k)$ denotes the weights, which of course depends on the num-

[36] 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, $\pi^A_{t-1,P}$.

ber of total contacts, k, the agent who met with the untested asymptomatic subject 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 subject via their 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 untested asymptomatic in period t and hence they will no longer be considered A-type or T-type agents. The share of non-traceable A-type subjects is therefore given by the following

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

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

At last, we take the step made in equation (A.4) and obtain 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:

$$\begin{aligned} E_t^1 =& (1-\pi_{C,t}^{0,A})\left[\frac{I_{t-1}^{A}\left(1-\pi_{IS}-\pi_R\right)\left(1-\pi_{P,t-1}^{A}\right)}{I_t^A}\pi_{C,t}^{1,A} + \frac{T_{t-1}\left(1-\pi_{P,t-1}^{T}\right)}{I_t^A}\pi_{C,t}^{1,T}\right](1-\pi_{IS})I_t^A \\ &+ (1-\pi_{C,t}^{0,S})\pi_{C,t}^{1,S}S_t + (1-\pi_{C,t}^{0,R})\left[\frac{R_{t-1}^{A}}{R_t^A}\pi_{C,t}^{1,R} + \frac{\pi_R I_{t-1}^{A}}{R_t^A}\pi_{C,t}^{1,RA}\right]R_t^A, \end{aligned} \tag{D.1}$$

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 to be traced 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 the agents of a group may have different histories of interactions due to changes in their health status. For instance, there is a difference for untested asymptomatic agents who became newly infected in the previous period and the ones who already were infected in the previous period. This is captured by the two terms in the first square bracket of equation (D.1).

The derivation $\pi_{C,t}^{1,R}$ for the Type-R agent is the same as for the Type-S agents $\pi_{C,t}^{S,1}$ with one difference. The contacts with untested asymptomatic agents 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. This implies that the adjustment in equation (33) is not needed and, thereby, $f_{t-1}^{R,A}(k) = f_{t-1}^{A,A}$.

The derivation $\pi_{C,t}^{1,RA}$ for a Type-RA agent is exactly the same as for a Type-A agent with 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 asymptomatic agents in the previous period.

Finally, the probability to be traced for susceptible agents due to previous period contacts is the same regardless of whether they get infected in period t. Hence this probability is equal to the probability for an S-type agent to be traced, which is denoted by $\pi_{C,t}^{1,S}$.

E Random Testing

An alternative to a contacting tracing strategy would be to test the population randomly, a strategy that has been also actively discussed. In this strategy, the probability of getting tested is the same for the susceptible, untested asymptomatic, and unobserved recovered agents. 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 tests Υ_t 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 (21) 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 the model iteratively based on a numerical root finder 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 consumption, labor, the marginal utilities and intraperiod utility of the susceptible, 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 $\{\Gamma_t^L\}_{t=1}^T$.[37]

4. Calculate the interactions of agents (e.g. for susceptible agents $f_t(k)$) based on their consumption and labor decisions. This allows us to calculate the 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 untested asymptomatic agents $\pi_{c,t}^A$. Crucially, the latter objects depends on the tracing technology and the testing capacity. In case of the

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

comprehensive tracing technology, the amount of active links from the previous period (e.g for susceptible agents 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 minimizes the error in budget constraint for the positive-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 2.2. This probability, which is defined in equation (5), 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 that Ξ is just a constant that depends on parameters and the average number of trials $\bar{k}$ is implied by the calibration of the structural parameters of the model.

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 (4) and, after some straightforward manipulations, we use the definition of mean of a binomial distribution to obtain

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

Second, we consider a linear approximation of the 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 (6).

H Additional Figures

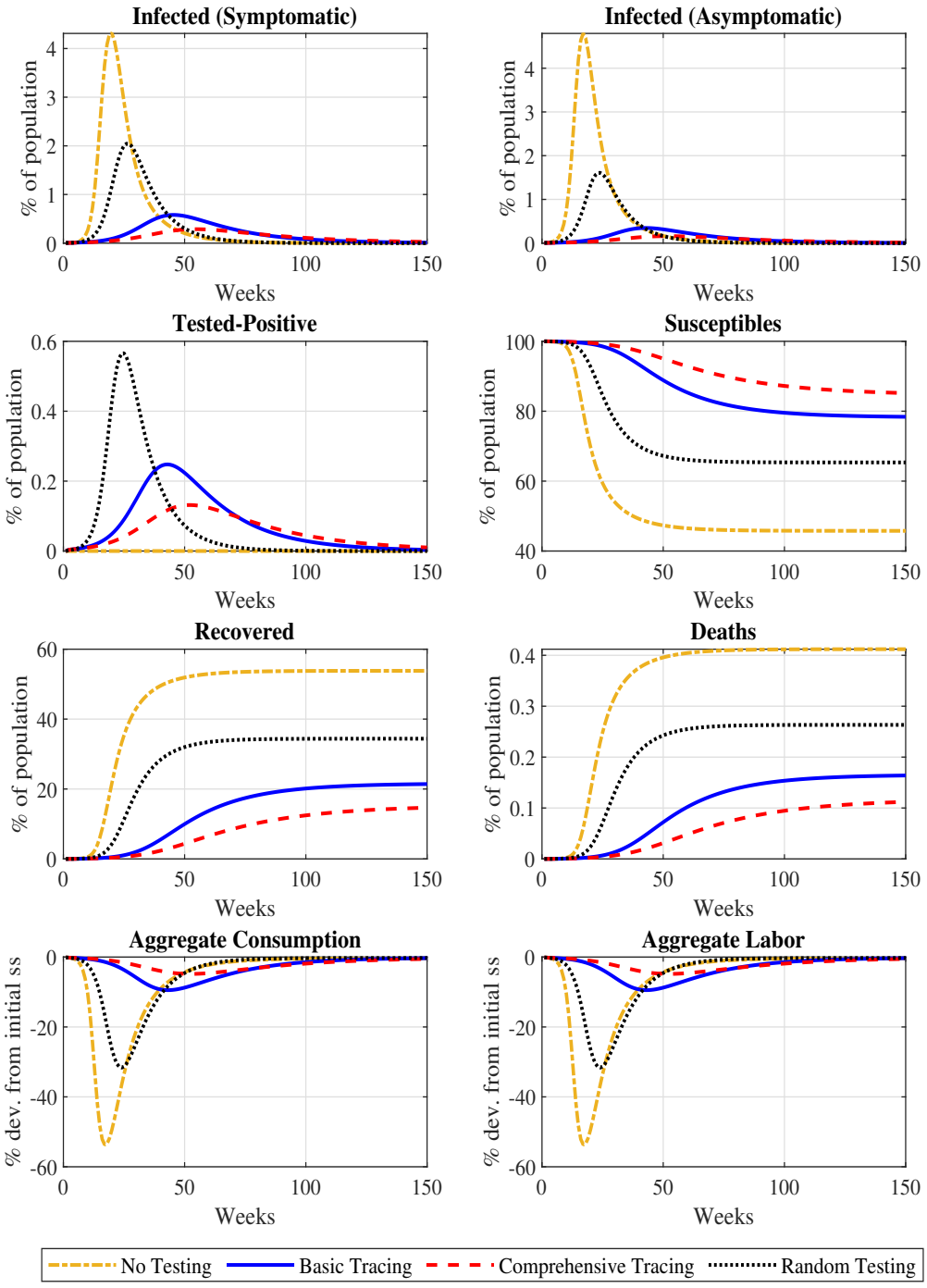

Figure 5: Comparison of different testing strategies with unconstrained number of tests for contact tracing: No testing (blue solid), basic tracing (red dashed) corresponds to current week contact tracing, comprehensive tracing (green dash-dotted) corresponds to current and previous week contact tracing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

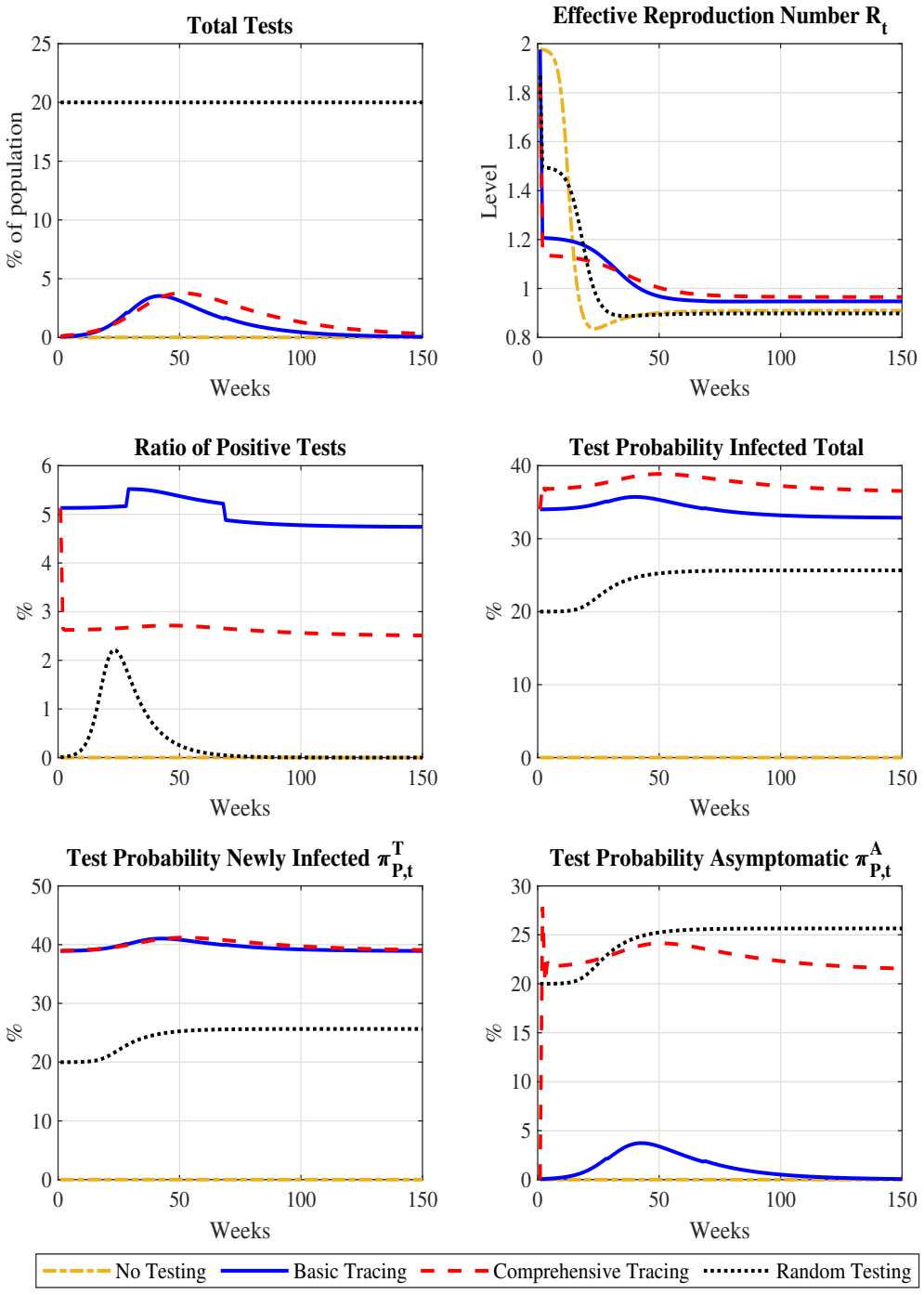

Figure 6: Comparison of different testing strategies with unconstrained number of tests for contact tracing: No testing (blue solid), basic tracing (red dashed) corresponds to current week contact tracing, comprehensive tracing (green dash-dotted) corresponds to current and previous week contact tracing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

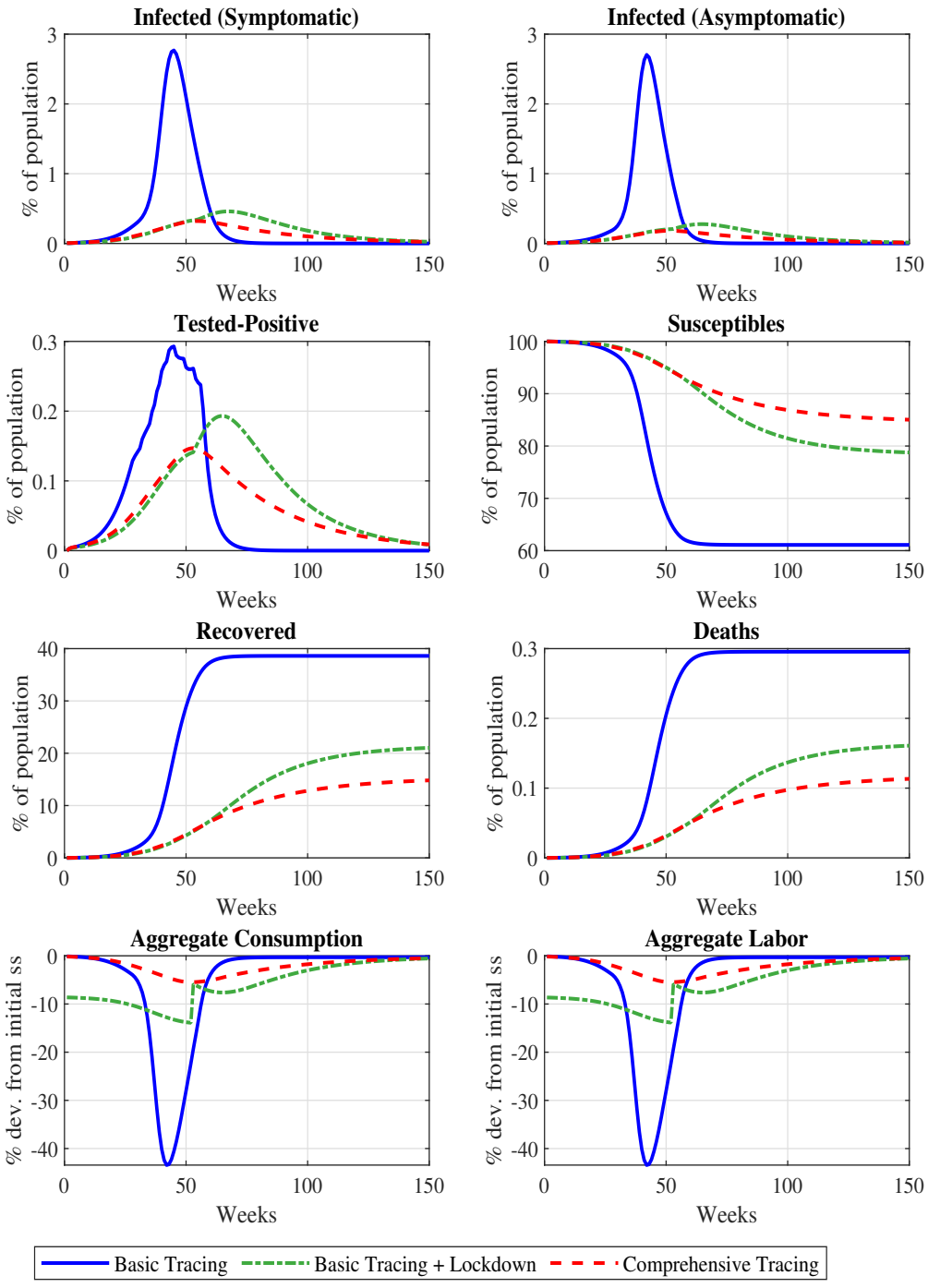

Figure 7: Comparison of different testing strategies with limited tests: Comprehensive tracing (blue solid line) is previous and current week tracing, basic tracing (red dashed line) is current week tracing and in the green dash-dotted basic tracing is combined with a 1 year lockdown.

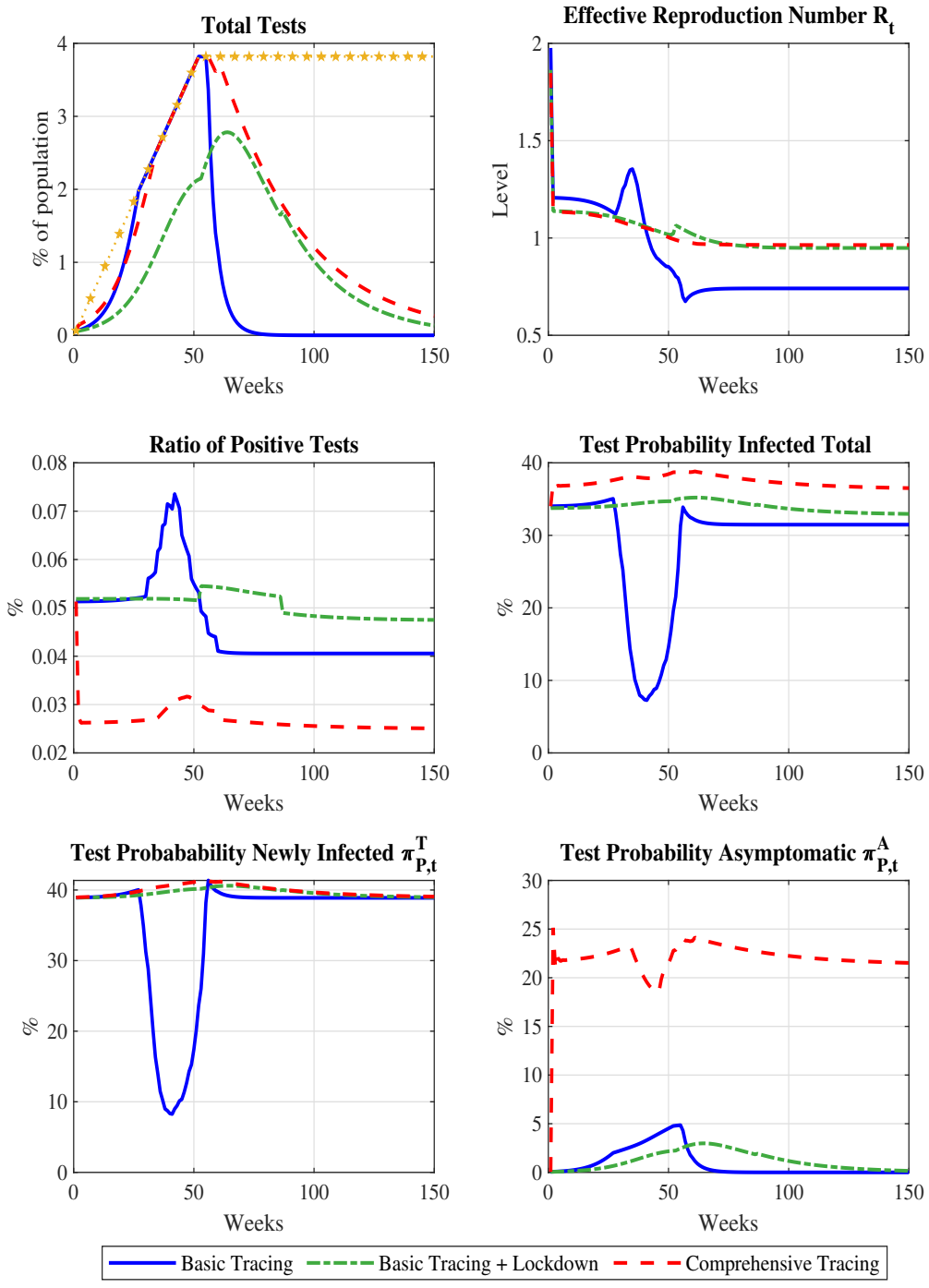

Figure 8: Comparison of different testing strategies with limited tests: Comprehensive tracing (blue solid line) is previous and current week tracing, basic tracing (red dashed line) is current week tracing and in the green dashed-dotted basic tracing is combined with a 1 year lockdown. In the first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

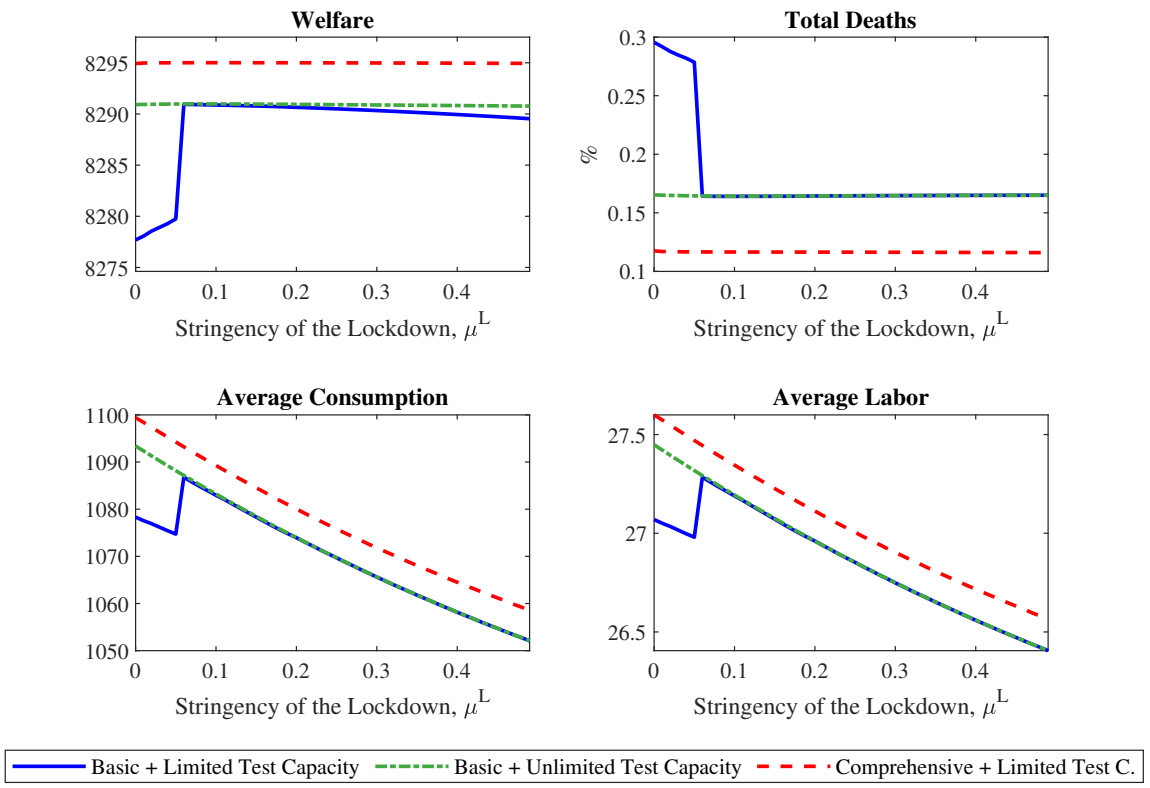

Figure 9: Comparison of different testing strategies under varying lockdown stringency imposed for the first 52 weeks. Welfare in week 1, accumulated deaths, aggregate consumption, and aggregate labor averaged over the 250 week horizon are reported.

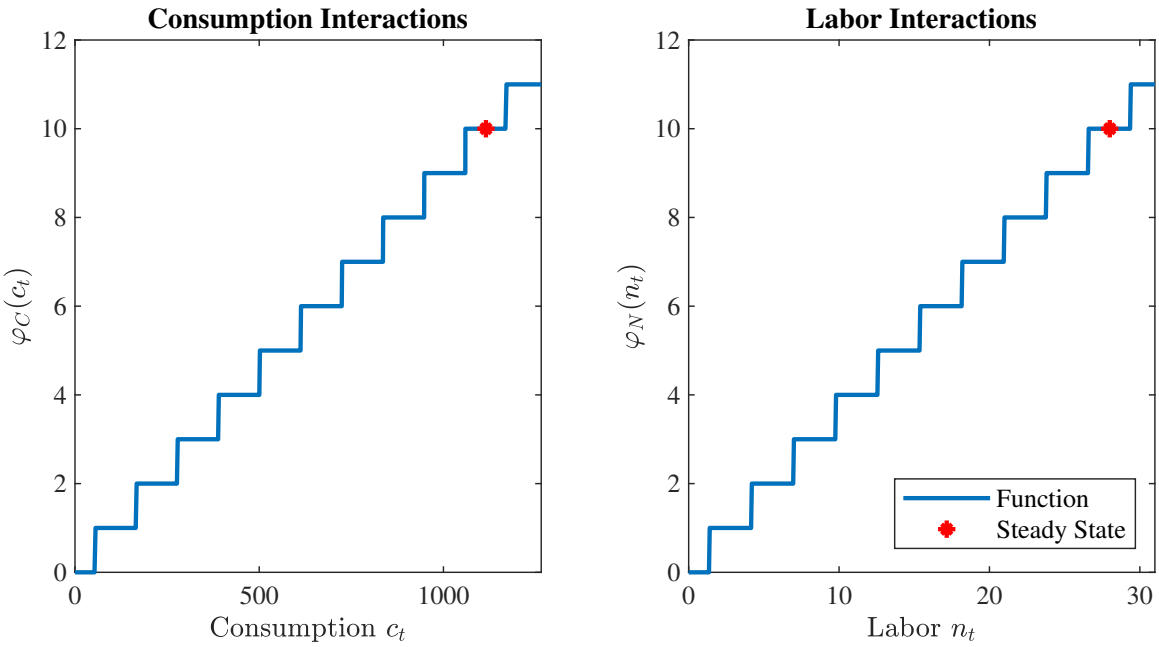

Figure 10: Step functions mapping consumption and labor decisions in total consumption and labor interactions.

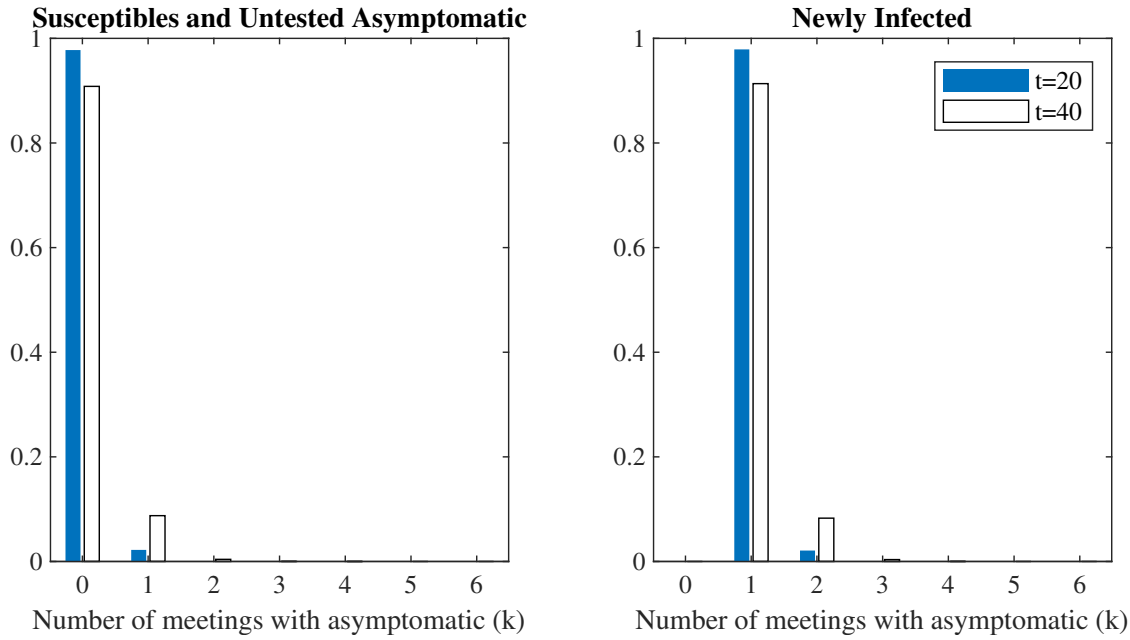

Figure 11: Probability distributions for an agent who does not know their health status to meet with untested asymptomatic subjects k times. The left plot graphs the distribution $f_t(k)$ defined in equation (22) and concerns susceptible agents, who do not turn out to become infected in the period, untested asymptomatic agents, and unobserved recovered agents. The right plot graphs the distribution $f_t^T(k)$ obtained by applying the Bayes theorem as shown in equation (24) and concerns the newly infected agents. The distributions are obtained in period 20 (blue bars) and 40 (white bars) of the simulation in which we assume basic tracing technology and unlimited testing capacity (Section 5.1).