Pandemic Recessions and Contact Tracing*

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January 9, 2023

Abstract

We study contact tracing in a new macro-epidemiological model with asymptomatic transmission and limited testing capacity. Contact tracing is a testing strategy that aims to reconstruct the infection chain of newly symptomatic agents. This strategy may be unsuccessful because of an externality leading agents to expand their interactions at rates exceeding policymakers' ability to test all the traced contacts. Complementing contact tracing with timely-deployed containment measures (e.g., social distancing or a tighter quarantine policy) corrects this externality and delivers outcomes that are remarkably similar to the benchmark case where tests are unlimited. 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: Testing, quarantine, infection chain, externality, social distancing, 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 in alleviating the pandemic's toll on the economy and mortality.[1] For instance, South Korea has combined contact tracing, mass testing, and alternative 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. In this paper, we construct a macro-epidemiological model with asymptomatic transmission and limited testing capacity to study (*i*) the social value of a technology enabling policymakers to trace the close contacts of confirmed infected cases, (*ii*) why this technology may fall short of delivering the expected outcome, and (*iii*) how contact tracing can be combined with alternative containment policies to effectively control a pandemic crisis.

We model contact tracing as a testing strategy that aims to reconstruct 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 allows the policymakers to decide who to test. The objective of testing is to detect and quarantine as many asymptomatic spreaders as possible. The epidemiological parameters of the model and the availability of tests are calibrated to match the U.S. data during the COVID-19 pandemic.

Contact tracing can be unsuccessful because of an externality leading agents to expand economic and social interactions at rates exceeding policymakers' ability to trace, test, and isolate the close contacts of confirmed cases. Complementing contact tracing with timely-deployed containment policies (e.g., social distancing or a tighter quarantine policy) allows policymakers to buy time to expand the tracing and testing scale so as to preserve the viability of the tracing and testing system. Our calibrated model predicts that U.S. testing availability during the COVID-19 pandemic was insufficient to ensure effective contract tracing without other containment policies.

If this externality is addressed properly by policymakers, contact tracing lowers the threshold number of infected agents needed to reach herd immunity by leveraging the information contained in the reconstructed infection chain of confirmed cases. In addition, the

[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."

reconstruction of the confirmed cases' infection channel is critical to enable contact tracing to effectively detect asymptomatic spreaders at the early stages of a pandemic when there are only a few spreaders.[2] In virtue of these two attributes, contact tracing mitigates both the consumption drop due to the pandemic and its death toll, allowing policymakers to move beyond the traditional trade-off between saving human lives and mitigating the economic costs of the pandemic.

We show that preserving the functionality of contact tracing is optimal. When we solve the optimal social distancing problem, we find that the planner wants to tighten social distancing restrictions right before the tracing and testing system would collapse. Scaling up social distancing measures in that period corrects the externality that threatens the smooth functioning of contact tracing and, in doing so, leads to economic and health outcomes that are remarkably similar to to the benchmark case where tests are assumed to be unlimited.

How critical is it for policymakers to be able to run contact tracing smoothly during a pandemic? Our calibrated model predicts that the social value of being endowed with a viable contact tracing and testing system is about \$8.7 trillion. Given that a tracing technology is arguably cheap to develop for most countries, this result suggests that it may be cost-effective for policymakers to invest in such a technology, even if epidemics are expected to be rather infrequent events. A more comprehensive tracing technology enabling policymakers to trace contacts for one additional week further increases social welfare by \$1.5 trillion. We also use the calibrated model to show that the social value of having enough tests to supply to all traced contracts is about \$1.6 trillion.

Contact tracing has been used to control the spread of a long list of lethal diseases, such as tuberculosis, measles, sexually transmitted infections (including syphilis and HIV), blood-borne infections, Ebola, H1N1 (swine flu), Avian Influenza, SARS-CoV (SARS), and SARS-CoV-2 (COVID-19).[3] However, formally modeling contact tracing is very hard, as the 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 share of consumption (work) of

[2]This prediction is in line with empirical findings by Fetzer and Graeber (2021), who show quasi-experimental evidence that contact tracing is very effective in containing the spread of the virus.

[3]Contact tracing was originally proposed in 1937 by Surgeon General Thomas Parran for the control of syphilis in the U.S. and was later implemented to control the spread of this virus in the following years (Parran, 1937).

asymptomatic infected people. It follows that the probability for a susceptible agent to have met a certain number of infected agents is a binomial distribution. This binomial distribution allows us to parsimoniously characterize the endogenous probability of a susceptible agent becoming infected in a given period. This probability turns out to be isomorphic to that in macro-epidemiological models (e.g., Eichenbaum, Rebelo, and Trabandt 2021), thereby providing theoretical underpinnings to that probability, which is typically assumed in those models.[4] Furthermore, this binomial distribution conveniently summarizes all of 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 macro-epidemiological models with multiple sectors or heterogeneous agents.[5]

Our paper belongs to the 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 et al. 2020); models to study optimal social distancing (Alvarez et al. 2021; Atkeson 2020; Bethune and Korinek 2020; Farboodi et al. 2021; Eichenbaum et al. 2021; Moser and Yared 2022; Piguillem and Shi 2022); models to study more targeted and smarter policies, such as testing or targeted quarantines, as alternatives to indiscriminate social distancing measures (Acemoglu et al. 2021; Akbarpour et al. 2020; Atkeson et al. 2020; Aum et al. 2021; Azzimonti et al. 2020; Baqaee et al. 2020a; Berger et al. 2022; Bognanni et al. 2020; Brotherhood et al. 2020; Chari et al. 2021; Eichenbaum et al. 2022; Favero et al. 2020; Galeotti et al. 2020; Glover et al. 2020; Hornstein 2022; Krueger et al. 2022); studies of the distributional consequences of various containment policies (Hacıoğlu-Hoke et al. 2021; Kaplan et al. 2020; Lee et al. 2021); and models to evaluate the efficacy of public policies –not based on tracing and testing– in controlling the spread of HIV (Greenwood et al. 2019).

The implementation of contact tracing is plagued by several bottlenecks. An important part of our analysis is to show that one potential bottleneck –i.e., the limited availability of tests– may lead to the demise of the tracing and testing system and that this event would worsen the pandemic's economic and health outcomes considerably. We study a number of mitigation policies (optimal social distancing, a tighter quarantine policy, and a mask-wearing mandate) that can be deployed in a timely manner to shore up the resilience of the

[4] In the special case in which the virus cannot be spread through consumption and labor interactions, the probability for a susceptible agent to become infected is isomorphic to the canonical SIR model proposed by Kermack and McKendrick (1927).

[5] See Guerrieri et al. (2022) for an example of multisectoral models to study how an epidemic and social distancing affect aggregate demand and supply. See Kaplan et al. (2020) for an example of macro-epidemiological models with income and wealth inequalities.

tracing and testing system. The macro-epidemiological literature has studied the dynamic complementarities of optimal social distancing with other factors: the limited capacity of the health system (e.g., Loertscher and Muir, 2021), the arrival time of an effective vaccine (e.g., Iverson et al., 2022), and the arrival of an effective technology to test and quarantine infected subjects (e.g., Brotherhood et al., 2020).

Given the hurdles to formally modeling the infection chain of confirmed cases, all the papers we know take a reduced-form approach to contact tracing (e.g., Alvarez et al., 2021; Piguillem and Shi, 2022). Typically in these papers, a fraction of agents whose health status is unknown becomes tested by the government in every period.[6] Modeling contact tracing by taking into account the existence of infection chains as we do has three main advantages. First, the central result that a well-functioning contact tracing allows policymakers to improve both economic and health outcomes of a pandemics hinges upon the enhanced ability of contact tracing of successfully detecting asymptomatic spreaders, even at the onset of a pandemic when it is very hard to do so. We find this result because we take into account the existence of infection chains. Second, our approach is preferable when one is concerned about the Lucas critique, which would arise, for instance, if one studies the efficacy of contact tracing under a mutating virus (e.g., a virus mutation resulting in more asymptomatic infections). Third, our structural analysis of contact tracing helps to calibrate smart testing in papers that take a more reduced-form approach.

Our paper is also related to the epidemiological literature that studies contact tracing. Hellewell et al. (2020) model contact tracing based on a branching process, which uses a negative binomial distribution to keep track of the number of secondary infections that a person infected with the virus could potentially produce.[7] In our analysis, the binomial distribution is used to model the probability that an agent meets a number of times with asymptomatic infected subjects while consuming and working. This different approach has important implications: First, the probability for an agent to be traced is endogenous, depending on their consumption and labor decisions. Second, our binomial approach allows us to provide theoretical underpinnings to the infection rate in SIR and Macro-SIR models.

2 The Model

The model economy is populated by agents who consume and work, and firms that hire labor N_t from agents in a competitive market and produce output according to a linear

[6] Chari et al. (2021) study targeted testing assuming that infected agents are more likely to receive a signal about their health status. They interpret the signal as the outcome of a test.

[7] A similar approach is followed by Ferretti et al. (2020) and other epidemiological papers.

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 et al. (2021), we assume there are three types of interactions through which the virus spreads out: consumption interactions, work interactions, and other interactions independent 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, *tested-positive 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. 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 tested-positive, symptomatic infected, and observed recovered agents is publicly observed.

Quarantine. The tested-positive 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,

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

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 social distancing, which refers to an economy-wide containment measure, affecting all 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

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

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 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 are 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 E, we show the steps taken to approximate τ_t.

The approximated infection rate τ_t in equation (6) nests the rate in the canonical SIR model as the special case in which consumption and labor interactions do not transmit the virus. It is also isomorphic to other leading macro-epidemiological models, in which this rate is assumed (e.g., Eichenbaum et al. 2021). 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, agents' beliefs about future changes in their health status are model consistent. 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 they are 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 tested-positive 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

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

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

The unobserved recovered agents have never shown 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, 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 agent, $\pi^T_{P,t}$ in equation (7), and for an asymptomatic agent, $\pi^A_{P,t}$ in equation (8), are characterized in Section 3.

Budget constraint for the non-quarantined agents. The problem is subject to the budget constraint for non-quarantined agents.

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

where $\mu^S_{c,t}$ denotes a tax on consumption proxying the effects of a government-imposed social distancing on consumption and labor. By reducing consumption and labor, social distancing curtails agents' economic interactions. In doing so, social distancing reduces 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

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

where μ_c^Q proxies the effects of imposing a quarantine on individuals' consumption and labor decisions. Social distancing is assumed to affect consumption of quarantined subjects as well. The parameter $\alpha \in (0,1)$ controls the additional effects of social distancing 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 develop, 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 event happens, 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 have developed. This penalty on labor productivity is given by $\phi < 1$.

2.5 Observed Recovered Agents (cont'd)

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

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

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

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

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 tested-positive 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 revenues of the social distancing and quarantine taxes are rebated to the agents these taxes are levied on.[11]

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 numbers 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 is 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 untested asymptomatic subjects in the next period.

[11] We abstract from fiscal policy in this study, which is primarily focused on assessing the efficacy of contact tracing. Bianchi et al. (2020), Mitman and Rabinovich (2021), and Hagedorn and Mitman (2020) study how fiscal policy should respond to pandemic recessions.

The pool of tested positive subjects is given by

$$P_{t+1} = (1 - \pi_{IS} - \pi_R)P_t + \pi^T_{P,t}T_t + \pi^A_{P,t}(1 - \pi_{IS} - \pi_R)I^A_t. \tag{19}$$

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 tested-positive 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

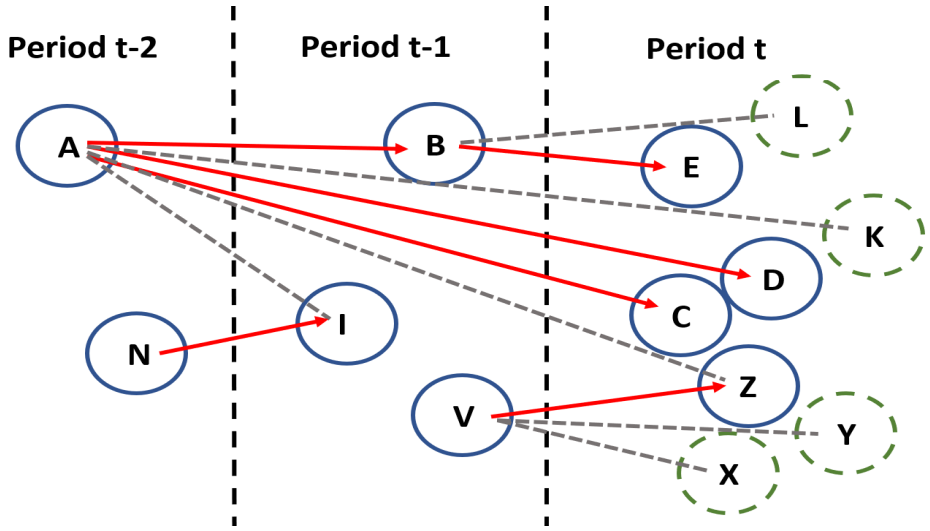

Figure 1: Example of an infection chain. The blue solid circles indicate an asymptomatic person. The green dashed circles are susceptible or recovered agents. The red lines describe an interaction that leads to an infection, while the gray lines describe an interaction that does not lead to an infection.

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 technology that allows health officials to trace only those contacts that have occurred during the current week and a more comprehensive technology that allows them to trace contacts up to one week back. When we say contact tracing, we generally refer to the first technology. When we say *comprehensive contact tracing* or simply comprehensive tracing, we mean the second technology.

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 contact 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 therefore 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 tracing 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 contact tracing 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 contact tracing technology can catch asymptomatic agents who went untested in the previous periods only if these agents meet randomly with a subject who turns 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.

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 tracing and testing capacity in period t, Υ_t, relative to the number of people traceable E_t; and (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 a 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 tracing and 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.

The variable Υ_t should be interpreted broadly as the intensive margin of tracing and testing as opposed to the extensive margin, which is determined by the efficiency of the tracing technology. While the extensive margin affects the number of traceable agents ($\pi^T_{C,t} + \pi^A_{C,t}$), the intensive margin, Υ_t, reflects the government's capacity of to process all the necessary information to test these traceable contacts and quarantine those who test positive. Henceforth, we will refer to Υ_t as testing capacity because this is how we will calibrate the model. This choice reflects the absence of data regarding this broader concept of intensive margin in tracing and testing.

Externality 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 externality on the number of traceable subjects, E_t, that 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 as they consume or work more, more people will become infected, raising the number of newly symptomatic cases in every period.[12] A larger number of newly symptomatic cases enlarges the pool of subjects who met with them and are, therefore, traceable.

This externality 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.[13]

[12]This externality would not be eliminated if these subjects knew to be asymptomatic spreaders.

[13]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 et al. (2021) study the implications of this

Eichenbaum et al. (2021) consider the case in which individuals do not internalize the limited availability of beds in hospitals when they decide how much to consume and work (medical preparedness). While both that externality and the one studied in our paper are about the existence of a bottleneck agents do not internalize, how these two types of externality affect the economic and health outcomes of a pandemic is quite different. When tests are running short, the efficacy of contact tracing falls, the effective reproduction number of the virus soars, and the threshold of recovered agents needed to reach herd immunity increases. As a result, the consumption loss and the number of deaths due to the pandemic worsen considerably. In contrast, the medical-preparedness externality leads to a larger consumption loss and a heavier death toll because the mortality rate sharply rises if there are not enough beds in the hospitals to treat the symptomatic infected agents.

It is also important to note that putting in place a viable system of contact tracing is an effective tool to address the medical-preparedness externality. As we shall show, when we solve the optimal social distancing problem, the planner wants to scale up social distancing measures to shore up the tracing and testing system so as to keep the number of infected cases low. If we expanded the model to introduce medical preparedness, the planner would still want to tighten social distancing in similar fashion to preserve the tracing and testing system. If the planner did not do that, more subjects would become infected and more stress would be put on the health system. An implication of this argument is that the externality concerning medical preparedness becomes less relevant for policymakers when the externality threatening the functionality of the tracing and testing system is properly addressed.

In the next section, we will characterize the probability of being traced and tested ($\pi^i_{C,t}$ and $\pi_{T,t}$) under the assumption that health authorities can trace only those contacts that has occurred in the current week. We show how to obtain those probabilities for the case of the comprehensive tracing technology in Appendix A.

3.1 The Probability of Being Traced

Contact tracing 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, and performing

externality in great detail. In our model with contact tracing and testing, that externality does not play any significant role.

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 of getting 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 agent is less likely to be traced than a newly infected agent under the contact tracing technology ($\pi_{C,t}^T > \pi_{C,t}^A$).

In Appendix I, we show the unconditional and conditional distributions $f_t(k)$ and $f_t^T(k)$ in one simulation where the contact tracing technology leads to successful 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 agents, 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. This result underscores the importance of exploiting the existence of the infection chain to increase the chance of detecting newly symptomatic agents.

3.2 The Conditional Probability of Being Traced

The 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 (B.11) and (19) for the contact tracing technology.

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 with 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. The computation is performed for a given sequence of taxes and an initial amount of asymptomatic and symptomatic agents infected by the shock. More details are in Appendix D.

The calibrated parameters of the model are summarized in Table 1. The economic parameters are calibrated based on Eichenbaum et al. (2021). 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 et al. 2021). Productivity, A, is set to match a yearly income of \$58,000. The scale parameter of labor disutility, θ, is calibrated so that agents work on average 28 hours per week. The Frisch labor elasticity φ is 0.5.

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). The parameters φ_C, φ_N, φ_O determine the number of interactions required to support levels of individual consumption c^s_t, labor n^s_t, and other non-economic activities, respectively. The original step functions $\varphi_C(c_t)$ and $\varphi_N(n_t)$ are shown in Appendix I. 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,

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. (2021)
Social distancing effect on quarantine	α	0	No impact besides quarantine
Initial infection	ϵ	0.1%	Infections March 16 2020

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).

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. We set the basic reproduction number to 2 in line with the evidence about the early transmission of COVID-19 (e.g. Li et al., 2020; Zhang et al., 2020). 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 (Burke et al., 2020; Pung et al., 2020).

In line with evidence from the World Health Organization (2020), we choose that an agent recovers on average after 18 days, which implies $\pi_R = 7/18$. 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. (2020b).[14] A key metric in parameterizing an SIR model is the infection fatality rate, which

[14]There is mixed evidence about this rate. Based on a population screening in Iceland, Gudbjartsson et al. (2020) find that 57% of the tested-positive cases report symptoms. However, almost 30% of negatively tested individuals also report symptoms in the same study. Poletti et al. (2021) find that 74% of tested-positive contacts of indexed COVID-19 cases did not develop symptoms for individuals below 60 years of age. Nishiura et al. (2020a) suggest a 69% infection rate based on flights of Japenese passengers data from China.

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 et al. (2021), who adjust the fatility rate to take into account unreported infections.[15]

In the model, symptomatic agents are subject to a labor productivity penalty, ϕ. We calibrate the penalty $\phi = 0.8$ based on Eichenbaum et al. (2021). 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 tested-positive agent is lower than those of non-quarantined (non-recovered) agents by approximately 30%. We assume that quarantined agents are not affected by social distancing, 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. (2022), this can be interpreted as 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 (e.g., social distancing) 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 retreating. The effective reproductive number in our model is defined as

$$\begin{aligned} R_t^E &= (1-\pi_{P,t-1}^T)\big[\tau_t + (1-\pi_{IS}-\pi_R)\left(1-\pi_{P,t}^A\right)\tau_{t+1} + \\ &\qquad (1-\pi_{IS}-\pi_R)^2\left(1-\pi_{P,t}^A\right)(1-\pi_{P,t+1}^A)\tau_{t+2} + \dots\big] \\ &= (1-\pi_{P,t-1}^T)\sum_{j=0}^{\infty}\left(\tau_{t+j}(1-\pi_{IS}-\pi_R)^j \Pi_{k=0}^{j}\left(1-\pi_{P,t+k}^A\right)\right). \end{aligned} \tag{28}$$

Based on a review of several epidemiological studies, Oran and Topol (2020) suggest an asymptomatic rate between 40-45%, while Byambasuren et al. (2020) estimate a lower asymptomatic rate of 17%.

[15] This is line with Nishiura et al. (2020b). Fernández-Villaverde and Jones (2022) estimate a rate of 1%.

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, $\pi^T_{P,t}$ and $\pi^A_{P,t}$, respectively. Social distancing lowers 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, $\pi^T_{P,t-1}$, than to changes in the future probability for an untested asymptomatic agent to test positive, $\pi^A_{P,t+k}$. 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 transitory nature of being asymptomatic, which is captured by the term $(1 - \pi_{IS} - \pi_R)$ in equation (28), implies that increasing the probability of catching asymptomatic agents further in the future has decreasing effects on the effective reproduction number. Detecting newly infected agents (i.e., increasing $\pi^T_{P,t}$) has the largest (negative) impact on the effective reproduction number because these subjects are quarantined before having the time to infect anyone else. This is an important point that helps explain some of the results shown in this section.

5.1 Contact Tracing with Unlimited Testing Capacity

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 contact tracing technologies in the most favorable environment where policymakers do not face any bottleneck when tracing and testing people. In addition, this exercise will give us a sense of how many tests would be needed to make contact tracing work 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.[16] It is assumed that random testing is run on a weekly testing capacity of 10% of the initial population over the entire simulation horizon. This implies a daily testing capacity of close to 5 million tests. To put this number in perspective, 1 million tests were administered per day in September 2020 in the U.S. We also consider the case in which no testing is performed.

Figure 2 shows the evolution of the key epidemiological, economic, and testing variables. Beginning with the case in which no one is tested (the green 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.

[16]The formalization of random testing in our model is explained in Appendix C.

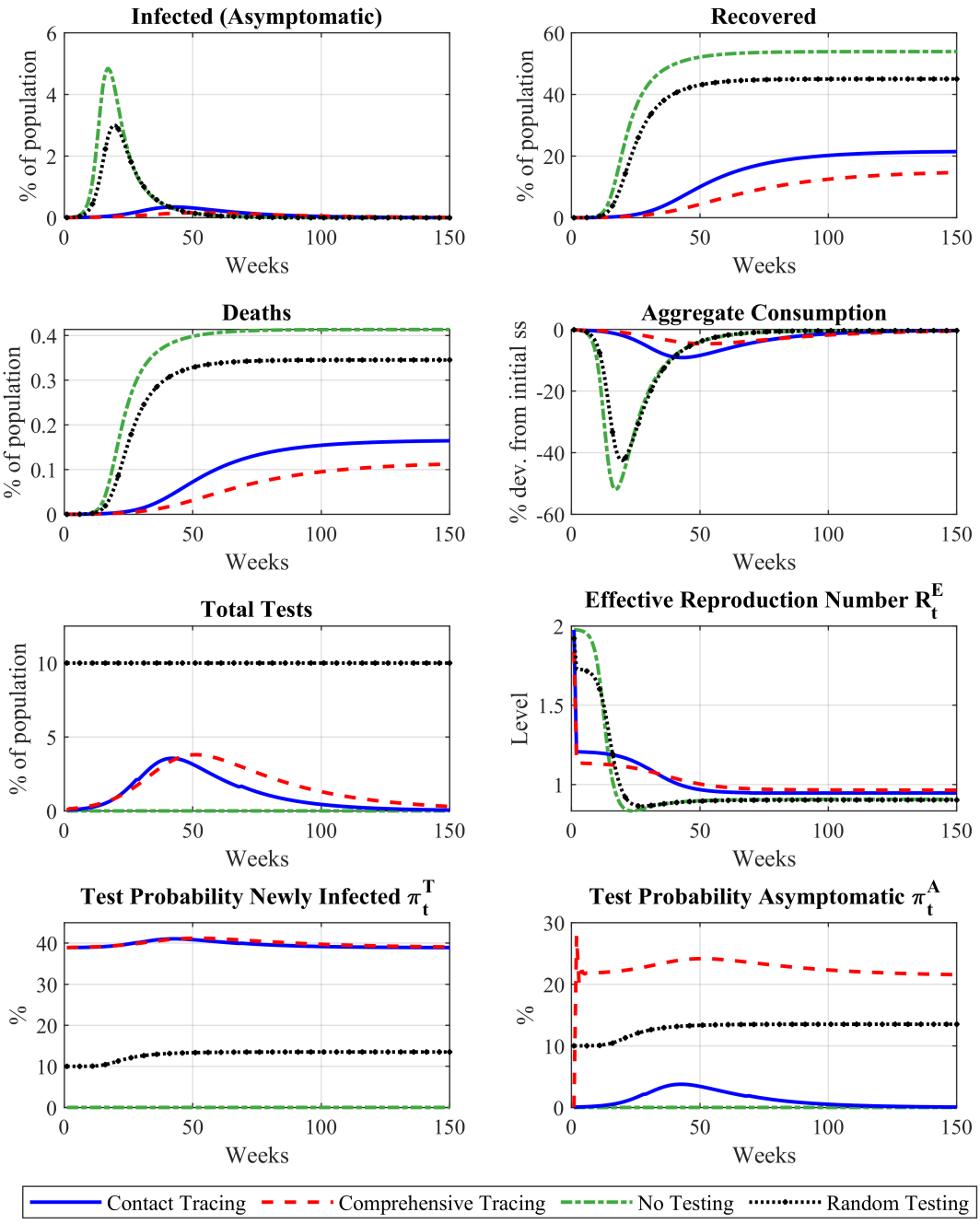

Figure 2: Comparison of different testing strategies with unconstrained number of tests for contact tracing and comprehensive contact tracing. The amount of tests used in random testing is 10% of the entire population each week.

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 extreme recession, with aggregate consumption contracting by up to 50%.

The introduction of the contact tracing technology hugely improves outcomes by slowing down the spread of the virus and reducing the death toll by more than 50%. See the solid blue line in Figure 2. As the virus spreads less quickly (lower effective reproduction 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.

The comprehensive contact tracing technology, which allows tracing of contacts up to one week back, (the red dashed line in Figure 2) further mitigates the severe consequences of the pandemic crisis.[17] However, the improvement is only marginal relative to what is already achieved by the contact tracing technology. Both tracing technologies 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 contact tracing technology requires more tests to be performed a few periods after the pandemic has started (around period 30) relative to the comprehensive one.

While this result may seem odd at first, it is important to recall that the contact tracing technology is less effective than the comprehensive technology in detecting untested asymptomatic subjects. The contact tracing technology can only trace these subjects through random meetings. As explained in Section 3.1, these types of meetings are quite rare.[18] As a result, in the lower right panel of Figure 2, the share of untested asymptomatic subjects detected by the contact tracing technology is very low compared to the levels attained by the comprehensive technology. Thus, the effective reproduction number is initially higher in the case of the 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 contact 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

[17]The formal derivation of comprehensive contact tracing is explained in Appendix A.

[18]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 I.2 of Appendix I.

of the pandemic. Even if 5 million people could be randomly tested every day, the pandemic would lead to a severe contraction and would kill 0.35% of the entire population –more than twice as many deaths as under contact tracing.

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 around 10% of the newly infected subjects in every period. Random testing is rather effective in capturing untested asymptomatic subjects. Even so, random testing fails to reduce the effective reproduction number, underscoring the importance of detecting and quarantining the newly infected cases to attain a successful containment of the virus. This last intuition is reinforced by observing that even though the contact tracing technology largely fails to detect untested asymptomatic subjects, it fares relatively well in containing the economic costs and mortality of the pandemic.

That the probability of catching the newly infected asymptomatic subjects turns out to be key to controlling the pandemic should not come as a surprise. We already noted that the reproduction number defined in equation (28) 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, $\pi^A_{P,t}$.

Why is contact tracing so successful? By leveraging the information contained in the reconstructed infection chains, contact tracing allows policymakers to break the positive relation between the probability of detecting newly infected agents ($\pi^T_{P,t}$) and the infection rate τ_t. In doing so, contact tracing resolves an important challenge faced by random testing: at the beginning of a pandemic – when the infection rate τ_t is low – infected agents who can spread the virus are only a few and are therefore hard to detect. As explained before, the ability of detecting and quarantining newly infected agents has a large effect on reducing the effective reproduction number, allowing contact tracing to nip the pandemic in the bud. Hence, social distancing is not required to quash a surge in the number of infections. Rather, these measures are only adopted if needed to address the externality associated with consumption and labor. The challenges posed by this externality are shown in the next section where we impose an upper bound on the number of tests that can be performed in every period.

5.2 Contact Tracing with Limited Testing Capacity

In the previous section, we showed that the contract tracing technology does a great job in controlling the spread of the virus. The comprehensive tracing technology improves outcomes only marginally. In this section, we show that this is not 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.[19] Afterwards, the 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 contact tracing technology (blue solid line) requires testing to accelerate after period 30 to compensate for its inability to catch untested asymptomatic subjects, as reflected in the low value of $\pi^A_{P,t}$ in the lower right plot of the figure. However, the 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 the 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 and agents cut their consumption and labor in response to the higher risk of getting infected.

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 keeps 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 contact 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.[20]

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

[20] Nevertheless, the testing availability becomes binding later on, lowering the probability of testing asymptomatic subjects, π^A_t, somewhat in subsequent periods. Because of the pecking order (explained in Appendix A), 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.

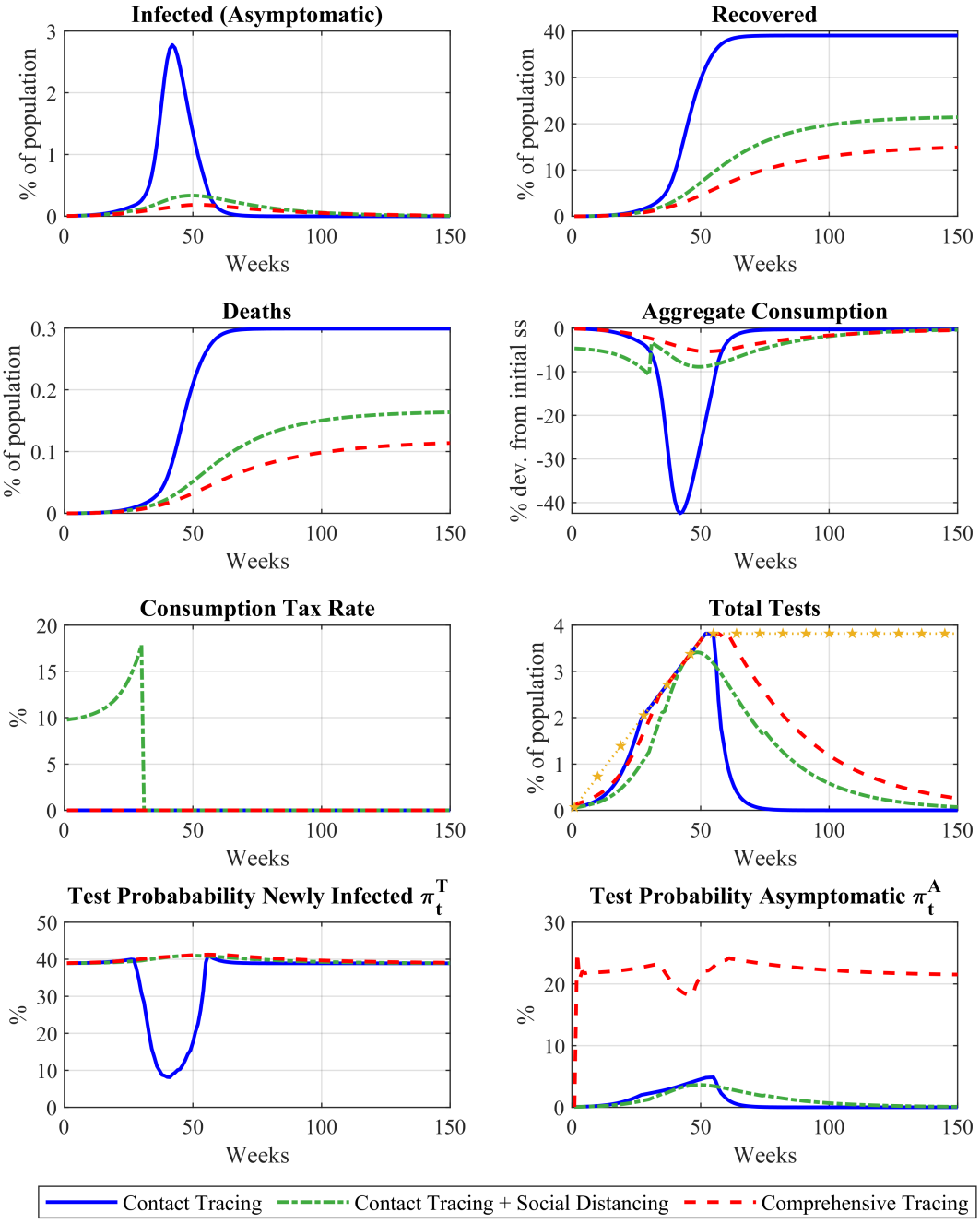

Figure 3: Comparison of different testing strategies with limited testing capacity: Contact tracing (blue solid line), contact tracing combined with social distancing for 30 periods (green dash-dotted line), and comprehensive tracing (red dashed line). In the sixth plot, the yellow starred line shows the testing capacity Υ_t.

Table 2: Welfare, economic, and health outcomes of various containment policies

	Welfare CE %[a]	Consumption %[b]	Mortality %[c]	Recovered %[d]	Social Costs trillion $[e]
Contact tracing with *limited* testing capacity					
No social distancing	−2.07	−3.22	0.30	39	9.67
Optimal social distancing (short)	−1.06	−2.55	0.17	22	4.93
Optimal social distancing (long)	−0.92	−6.56	0.12	16	4.28
Tighter quarantine	−1.06	−1.92	0.17	22	4.94
Alternative contact tracing scenarios					
Unlimited testing capacity	−1.05	−1.89	0.17	22	4.91
Comprehensive tracing	−0.74	−1.38	0.12	15	3.44
All exposed contacts quarantined	−1.40	−2.95	0.21	28	6.51
No contact tracing					
No testing	−2.93	−4.25	0.41	54	13.64
No testing + optimal social distancing	−2.87	−6.76	0.39	51	13.39
Random testing	−2.40	−3.67	0.35	45	11.18

[a] Welfare gain/loss expressed as consumption equivalent relative to a non-pandemic economy.
[b] Cumulated consumption loss at the end of the pandemic relative to a non-pandemic economy.
[c] Cumulated mortality rate at the end of the pandemic.
[d] Fraction of recovered agents at the end of the pandemic.
[e] Social costs in trillion $ relative to a non-pandemic economy.

5.3 Complementarity with Other Containment Policies

In Section 5.1, we showed that a well-functioning contact tracing and testing system allows policymakers to reduce both the consumption loss and the death toll of a pandemic. However, actual implementation of contact tracing turned out to be very challenging for a variety of reasons in many countries across the world. In the previous section, we showed that one reason that can impair the correct functioning of contact tracing is the scarcity of tests, which can be more broadly interpreted as the inability of coordinating tracing and tests when the number of traced close contacts grows too large. In this section, we study how to remedy this situation that leads contact tracing to fail.

To this end, we now consider three containment policies that the government can deploy to shore up a tracing system at risk of collapse. The first policy is social distancing, the second one is a tighter quarantine, and the third one is to randomly allocate the excess testing capacity in the early stages of the pandemic.

Table 2 summarizes the outcomes of these three cases and compares them with those studied in the earlier sections. The case of "All exposed quarantined" will be explained in Section 5.4 and will be used to isolate the social value of testing. The table shows the welfare losses expressed as consumption equivalents relative to the non-pandemic economy. It also shows the average consumption loss over the entire considered horizon of 250 periods relative to the non-pandemic economy, the cumulative mortality rate and the recovery rate at the end of the pandemic. The social costs of the different scenarios are expressed in trillions of dollars. Appendix F shows the derivation of consumption equivalents and the social costs.

Before evaluating the three cases, it is important to notice that, in the idyllic case of unlimited testing capacity (no externality), contact tracing reduces the share of recovered agents needed to reach herd immunity by 32 percentage points. Compare the column reporting the cumulative percentage of fully recovered agents at the end of the pandemic in the case of Unlimited testing capacity (under Alternative contact tracing scenarios) with the No testing (under No contact tracing scenarios) in Table 2. This result arises because tracing and testing permanently lower the effective reproduction number of the virus – as shown in Section 5.1 – decreasing the threshold of recovered people needed to attain herd immunity.

Now we turn to the less idyllic case in which contact tracing is threatened by an externality due to limited testing capacity. In this context, we will show how alternative containment policies can be combined with contact tracing to deliver welfare, economic, and health outcomes that are remarkably similar to those obtained under the idyllic case of unlimited testing capacity (no externality) threatening the implementation of tracing and testing.

Optimal social distancing. We solve for the optimal path of the consumption tax rate $\mu^S_{c,t}$. As standard in this literature, the planner sets the consumption tax to maximize the welfare of the economy at the beginning of the pandemic. Appendix F describes the welfare criteria and the Ramsey problem in detail. For a reason that will be clarified below, we consider two scenarios: The government can either commit its social distancing policy over a period of either 30 weeks (labelled Optimal social distancing (short) in Table 2) or 150 weeks (labelled Optimal social distancing (long) in the table). The green dashed-dotted line in Figure 3 shows the dynamics of the macro and epidemiological variables under the optimal short social distancing policy.

As shown in Figure 3, when we solve for the optimal path of consumption tax rates over the first 30 periods, the collapse of the tracing and testing system is averted by implementing social distancing before the testing capacity would become binding. See the tax increases over the 30 periods aimed to curtail the amount of consumption and labor interactions. By lowering the amount of economic interactions early on, social distancing reduces the number

of tests required, preventing the testing capacity from becoming binding later on. As a result, the effective reproduction of the virus is successfully reduced, allowing the economy to reach herd immunity with fewer cases. To see this, compare the cumulative number of recovered under the case of limited testing capacity, in which the tracing and testing system collapses, to that under the case of limited testing capacity plus the optimal (short) social distancing in Table 2 (the fourth column).

Remarkably, the optimal *short* social distancing policy leads to a cumulative mortality rate and welfare gains that are very similar to those achieved under no constraint on testing (unlimited testing in Table 2), where, by construction, no externality threatens the functioning of contact tracing. As shown in Figure 3, the lower aggregate consumption path at the beginning due to the tightening of social distancing is more than compensated by a higher consumption level throughout the pandemic, relative to the case in which the tracing and testing system collapses – the blue solid line.

How can the government avert the collapse of the tracing and testing system? This is shown in Figure 4, in which the optimal tax rate from the Ramsey problem is displayed. Under both time horizons considered (blue solid and dashed lines), the optimizing tax rate is increased in the run-up to period 37 when the system would have collapsed in the absence of this measure. Yet, if we assume unlimited testing capacity (the red solid and dashed lines), the externality studied in this paper does not arise, the tracing and testing system does not collapse, and the optimal tax rate is not characterized by any increase from period 30 through period 37.

Why is the optimal tax rate increased a second time when we consider a longer commitment period (the solid lines in Figure 4)? The optimal consumption path is raised to sufficiently slow down the spreading of the virus to attain herd immunity gradually over time. This result is not new, and Eichenbaum, Rebelo, and Trabandt (2021) have explained it thoroughly.[21] If one compares the consumption loss and the mortality rate in the case of limited testing capacity and short social distancing with those in the case of limited testing capacity and long social distancing in Table 2, one can see that the second tax hike leads to a quite dramatic contraction in consumption to push the death toll down only a bit. This result is in line with other studies that calibrate the costs of a statistical life similarly to the way we do in our paper.

Importantly, while, of course, social welfare increases relative to the case of the short social distancing policy, much of the welfare gains are reaped in the short run. To see

[21]The second tax hike needed to address the externality related to achieving herd immunity is much larger than the first hike intended to address the externality studied in our paper. This relatively large tax hike is due to the quite large value the literature typically attributes to a human life in the calibration. The magnitude of the first tax hike primarily depends on how many tests are available.

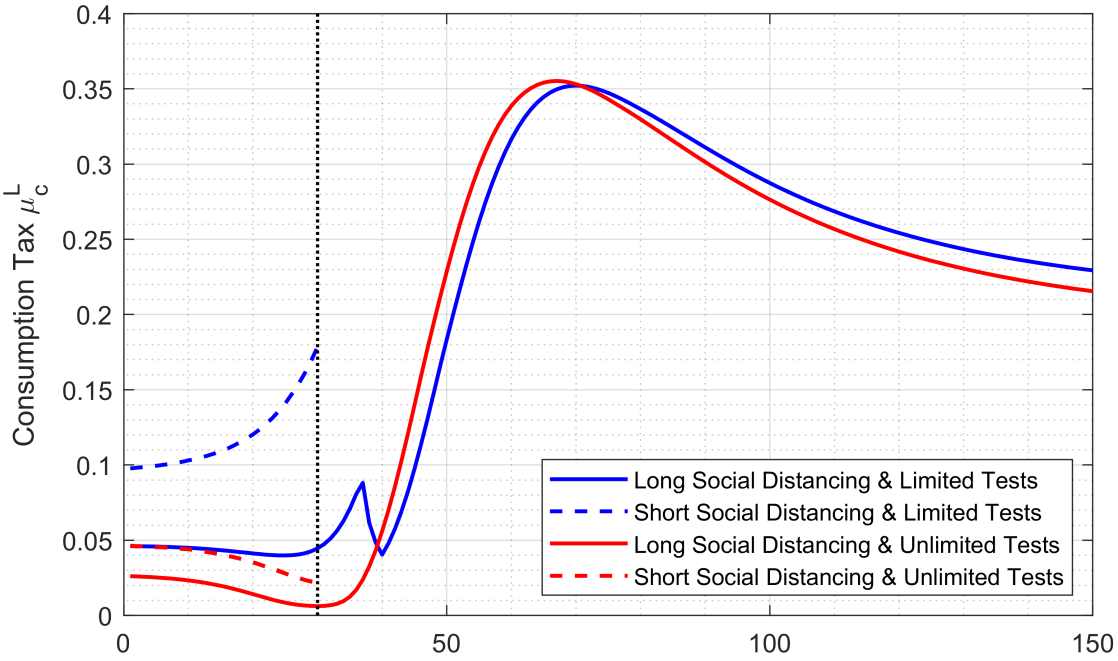

Figure 4: Optimal social distancing – the planner's optimal path of consumption tax rates is shown for different scenarios in an economy with contact tracing. The test availability is either limited (blue lines) or unlimited (red lines). We consider two horizons over which the government can commit to maneuver the tax rate: a long horizon of 150 periods (solid lines – long social distancing) or a short horizon of 30 periods (dashed lines – short social distancing).

this, compare the first column of Table 2 for the cases No social distancing, Optimal social distancing (short), and Optimal social distancing (long) under the first of the three scenarios considered in that table ("Contact tracing with limited testing capacity"). This finding highlights the importance of the type of externality studied in this paper.

To sum up, we showed that (*i*) a combination of mitigation policies (in this case contact tracing and testing + social distancing) is welfare improving; (*ii*) it is optimal for the government to use mitigation policies to lower social interactions right before tests are running short; (*iii*) most of the welfare gains are reaped by only correcting the externality studied in our paper – i.e., by implementing the *short* optimal social distancing policy. Welfare gains from addressing the other externality in the model are relatively small.

Tighter quarantine policy and limited testing capacity. To keep the tracing and testing system afloat when tests are running short, policymakers decide to quarantine all the agents for whom no test is available because the testing capacity constraint is binding.

When the testing capacity constraint is not binding, policymakers quarantine only subjects who test positive, exactly as in the baseline case. For computational reasons, we assume that the duration of the quarantine for the untested agents is stochastic. Agents who were tested before being quarantined leave the quarantine when they test negative, as assumed in the baseline case. The outcomes of this mix of policies is shown in Table 2 as "Tighter quarantine."

The more aggressive quarantine policy leads to welfare, economic, and health outcomes that are remarkably similar to the case of unlimited testing capacity and to the case of (short) optimal social distancing policy under limited testing capacity. The reason is that by quarantining more people, policymakers avert the collapse of contact tracing. Nevertheless, outcomes are slightly worse than those under unlimited testing capacity because the lack of tests prevents policymakers from knowing the true health status of those agents who are quarantined under the tighter quarantine regime. As a consequence, some subjects who leave quarantine are still asymptomatic and able to infect others. As a result, consumption falls and mortality drops. Furthermore, consumption falls because more agents are quarantined and quarantined agents consume less. However, these effects on consumption are rather small quantitatively, as shown in Table 2. Indeed, the tighter quarantine policy leads to a better consumption outcome than the optimal *short* social distancing policy, which, to be effective, has to lower the consumption path considerably in the early stages of the pandemic as shown in Figure 3.

Random testing in combination with contact tracing. When random testing is combined with contact tracing, the marginal contribution of allocating tests randomly is negligible. We combine these two testing strategies by assuming that when the testing capacity exceeds the number of traced subjects to be tested, this excess of tests is allocated randomly across the population. The negligible marginal contribution of random testing is due to the inability of this testing strategy to significantly lower the effective reproduction number beyond what contact tracing already achieves. This failure is largely due to the low probability that asymptomatic spreaders can be detected through their random meetings with newly symptomatic cases, as explained in Section 5.1 and shown graphically in Appendix I. More details on the random testing in combination with contact tracing are shown in Appendix C.

We conclude that both optimal social distancing and a tighter quarantine policy are suitable tools to preserve the viability of the tracing and testing system while random testing is not. In fact, when the government has a limited ability to commit to optimal social distancing (short social distancing), the welfare implications of the two approaches are virtually

identical, as shown in Table 2. Even when the government has the ability to commit for a longer period of time, optimal social distancing policy leads to a quite small increase in social welfare. And this slightly higher level of welfare can be achieved by sacrificing much more consumption than what the tighter quarantine policy requires. This result highlights the critical difference between the two approaches: Social distancing is a very potent pool that allows policymakers to save lives by freezing the economy. The tighter quarantine is less costly in terms of consumption, since it is a measure applied to only a subset of subjects (the quarantined ones). Nevertheless, a tighter quarantine policy when the testing constraint is binding cannot completely eliminate the risk that some asymptomatic spreaders leave quarantine before they have recovered. As a result, the risk for susceptible agents to become infected is higher. These two effects on welfare turn out to cancel each other out.

5.4 The Value of Tracing and Testing

We now want to use our model to study the social value of contact tracing and testing. To this end, we compare the case of unlimited testing capacity plus contact tracing to the case of no tracing and testing in Table 2. This comparison shows that testing and tracing more than halve the consumption loss due the pandemic and the mortality rate. According to our model, the social welfare gains from running a viable tracing and testing system are of the order of \$8.7 trillion (see the column in the far right). This result underscores the importance of preserving the viability of the tracing and testing system.

Let us now focus on the gains from testing alone (conditional on being able to trace the close contacts of confirmed cases in the current week). For this, we need to construct a counterfactual case in which there is no test and hence policymakers have to quarantine all the close contacts traced every week.[22] Note that without testing, lots of susceptible subjects will be quarantined and since tests are not available, some infected agents may leave quarantine still being asymptomatic and infect more susceptible subjects. Furthermore, in the absence of testing, subjects who did not develop symptoms during quarantine do not know their health status. This lack of information lowers social welfare. The case of no testing is called All exposed contacts quarantined in Table 2. If one compares this case with that of unconstrained testing capacity, which corresponds to the case of unlimited testing capacity, the value of having enough tests to check the health status of all the traced contacts is valued by the model to be equal to \$1.6 trillion.

Furthermore, we show that being able to trace contacts for one additional week (comprehensive contact tracing) will further lower the mortality rate (-0.05 percentage points) and

[22]For computational reasons, we assume that quarantine has a stochastic duration in the absence of tests.

the consumption loss ($-.051$ percentage points). The value of a more comprehensive tracing system is roughly $1.5 trillion.

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 four 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.[23] Our methodology is general and can be applied to models featuring households or firms heterogeneity.

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 contact tracing technology, multiple rounds of testing can provide only a minimal contribution because for the most part, policymakers can 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

[23] 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.

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.

Mask-Wearing Mandate to Rescue Contact Tracing. At the end of the previous section, we studied how optimal social distancing and a more stringent quarantine policy may prevent the tracing and testing from collapsing. We now discuss whether a very popular containment measure to combat the spread of the virus –introducing a mask-wearing mandate – could also be an effective tool to avert the collapse of the tracing system.[24]

In our framework, a more stringent mask-wearing mandate can be modeled by reducing the probability for agents to get infected conditional on meeting an asymptomatic spreader, τ. Broadening the set of activities for which wearing masks is mandatory tends to increase social interactions because agents now feel safer and understand that they are less likely to get infected at any interaction. While a lower risk for agents to catch the virus obviously improves the economic and health outcomes of a pandemic, by spurring social interactions a mandatory mask mandate can –all else being equal– increase the number of agents exposed to confirmed cases, requiring more tests to be administered. So a more stringent mask mandate may even add strain to the tracing system in the short run. In this light, this containment measure seems to be less complementary to contact tracing than those studied in Section 5.3. Appendix G provides more details.

Furlough Schemes. Sending agents into quarantine helps contain the health losses, but also imposes economic losses on the quarantined agents. A furlough scheme, which is modeled as a reduction in the quarantine tax μ^Q, might help to contain the economic losses for quarantined agents. However, we find that the social welfare gains of a furlough scheme for a well-functioning contact tracing technology are rather low. The reason is that the fraction of quarantined agents is small (insofar as the contact tracing is not collapsing.) However, the potential gains of introducing a furlough scheme are much larger when the government decides to quarantine a large number of agents, e.g., every traced contact regardless of their test outcome. As a consequence, the importance of introducing a furlough scheme depends on the stringency of the quarantine policy. Our analysis suggests that a very well targeted quarantining approach considerably reduces the necessity of a furlough scheme. Appendix G contains the details and related simulations.

[24] A formal cost and benefit analysis of this matter is very challenging because it requires taking a stand on agents' disutility of wearing masks, which is not well understood.

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 externality on the number of subjects that will need to be traced and tested. This externality can threaten the correct functioning of contact tracing. Timely-deployed containment policies – social distancing or tightening quarantine policies – may correct this externality, allowing policymakers to move beyond the traditional pandemic trade-off between saving human lives and mitigating the economic costs of pandemics. Indeed, we showed that the complementarity between contact tracing and these containment policies is so strong that policymakers can achieve welfare, consumption, and health outcomes that are remarkably similar to the idyllic case in which no externality threatens the implementation of contact tracing.

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A 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 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 $t-1$; and (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.[25]

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*,

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

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

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

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:

$$f_{c,t-1}^{A,T}(k) \equiv \mathcal{B}\left(k, \varphi_c\left(c_{t-1}^{s}\right), \frac{[\tau + (1-\tau)\tau_{t-1}]C_{t-1}^{S}}{C_{t-1}}\right), \tag{A.2}$$

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.[26] 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.

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,

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

backward tracing considerably raises the probability for a Type-A agent to be traced, 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 $t-1$:

$$f_{t-1}^{A,A}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} \mathcal{B}\left(i, \varphi_C(c_{t-1}^s), \frac{C_{t-1}^A}{C_{t-1}}\right) \mathcal{B}\left(j, \varphi_N(n_{t-1}^s), \frac{N_{t-1}^A}{N_{t-1}}\right) \mathcal{B}\left(k-i-j, \varphi_O, \frac{I_{t-1}^A}{Pop_{t-1}}\right). \tag{A.3}$$

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

$$f_{t-1}^{T,T}(k) \equiv \sum_{i=0}^{k} \sum_{j=0}^{k-i} \mathcal{B}\left(i, \varphi_C(c_{t-1}^s), \frac{c_{t-1}^s T_{t-1}}{C_{t-1}}\right) \mathcal{B}\left(j, \varphi_N(n_{t-1}^s), \frac{n_{t-1}^s T_{t-1}}{N_{t-1}}\right) \times \mathcal{B}\left(k-i-j, \varphi_O, \frac{T_{t-1}}{Pop_{t-1}}\right), \tag{A.4}$$

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 (A.3), by applying the Bayes theorem

$$f_{t-1}^{T,A}(k) = \frac{f_{t-1}^{A,A}(k)\,\tilde{\tau}(k)}{\tau_{t-1}}, \tag{A.5}$$

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), applying 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

$$f_{t-1}^{S,A}(k) = \frac{f_{t-1}^{A,A}(k)\,(1-\tilde{\tau}(k))}{1-\tau_{t-1}}. \tag{A.6}$$

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.1. 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 $\pi_{C,t}^{i}$, 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[27]

$$\pi_{P,t}^{j,i} = \pi_{C,t}^{j,i} \cdot \pi_{t,T}^{j} \cdot (1 - \pi_F), \quad i \in \{A, T, S\} \quad j \in \{0, 1\}, \tag{A.7}$$

where j denotes the period $t - j$ when the contacts relevant for tracing were established. So we combine the probability of being traced, $\pi_{C,t}^{j,i}$, 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_t^0 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_t^1, which is formally defined in Appendix A.4.

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 $\pi_{t,T}^0$ 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

$$\pi_{T,t}^1 = \min\left(1, \frac{\max\left(0, \Upsilon_t - E_t^0\right)}{E_t^1}\right). \tag{A.8}$$

Note that the probability of testing positive defined in equation (A.7) 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 (B.11) and (19), which requires us to know the *average* probability for an untested asymptomatic subject to test positive in period t ($\pi_{P,t}^A$) and the average probability for newly infected subjects to test positive in period t ($\pi_{P,t}^T$).

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

$$\begin{aligned}\pi_{P,t}^A = &\frac{I_{t-1}^A \left(1 - \pi_{IS} - \pi_R\right)\left(1 - \pi_{P,t-1}^A\right)}{I_t^A} \cdot \left[\pi_{P,t}^{0,A} + (1 - \pi_{C,t}^{0,A}) \cdot \pi_{P,t}^{1,A}\right] \\ &+ \frac{T_{t-1}\left(1 - \pi_{P,t-1}^T\right)}{I_t^A} \cdot \left[\pi_{P,t}^{0,A} + (1 - \pi_{C,t}^{0,A}) \cdot \pi_{P,t}^{1,T}\right],\end{aligned} \tag{A.9}$$

where the first expression within square brackets denotes the probability for a Type-A agent

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

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.[28] 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 (B.11) 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

$$\pi_{P,t}^{T} = \pi_{P,t}^{0,T} + (1 - \pi_{C,t}^{0,T}) \cdot \pi_{P,t}^{1,S}. \tag{A.10}$$

A.1 Tracing Probabilities

In these subsections, 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 $t-1$:

$$f_{t-1|t}^{A,A}(k) = \frac{f_{t-1}^{A,A}(k)\left\{1 - \left[1 - (1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}\left(1-\pi_{F}\right)\right\}}{\sum_{k=0}^{\varphi_c\left(c_{t-1}^{s}\right)} f_{t-1}^{A,A}(k)\left\{1 - \left[1 - (1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}\left(1-\pi_{F}\right)\right\}}, \tag{A.11}$$

and

$$f_{t-1|t}^{T,A}(k) = \frac{f_{t-1}^{T,A}(k)\left\{1 - \left[1 - (1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}\left(1-\pi_{F}\right)\right\}}{\sum_{k=0}^{\varphi_c\left(c_{t-1}^{s}\right)} f_{t-1}^{T,A}(k)\left\{1 - \left[1 - (1-\pi_{IS})^{k}\right]\pi_{t-1,T}^{0}\left(1-\pi_{F}\right)\right\}}, \tag{A.12}$$

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

[28] 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.

will test positive with probability $\pi^0_{t-1,T}(1-\pi_F)$ at the end of the same period. As we will formally define later, $\pi^0_{t-1,T}$ 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.[29] It is convenient to write: $f^{A,T}_{t-1|t}(k) = f^{A,T}_{t-1}(k)$, $f^{T,T}_{t-1|t}(k) = f^{T,T}_{t-1}(k)$, $f^{S,T}_{t-1|t}(k) = f^{S,T}_{t-1}(k)$, and $f^{S,A}_{t-1|t}(k) = f^{S,A}_{t-1}(k)$.

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

$$g^{i,T}_{t-1}(\underline{k}_{t-1}|k_{t-1}) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}, \left(1-\pi^T_{P,t-1}(i)\right)\right), \tag{A.13}$$

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 A.2.

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:

$$g^{i,T}_{t-1}(\underline{k}_{t-1}) = \sum_{k=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_o} g^{i,T}_{t-1}(\underline{k}_{t-1}|k) f^{i,j}_{t-1|t}(k), \quad i \in \{A,T,S\}. \tag{A.14}$$

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

[29] The distributions $f^{T,T}_{t-1|t}(k)$ and $f^{A,T}_{t-1|t}(k)$ 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).

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.[30] 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 A.3.

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:

$$g^{i}_{t-1}(\underline{k}_{t-1}) = \sum_{j=1}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} g^{i,T}_{t-1}(j) g^{i,A}_{t-1}(\underline{k}_{t-1}-j), \quad i \in \{A,T,S\}. \tag{A.15}$$

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

$$\pi^{1,i}_{C,t} = \sum_{\underline{k}=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} \left[1-(1-\pi_{IS})^{\underline{k}}\right] g^{i}_{t-1}(\underline{k}), \quad i \in \{A,T,S\}. \tag{A.16}$$

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 (A.7).

A.2 Active T-Links

The objective of this subsection 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 (A.10).

$$\pi^{T}_{P,t-1}(i) = \pi^{T}_{P,t-1}, \quad i \in \{T,S\}. \tag{A.17}$$

[30] 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.

This is the probability to be used in the conditional distribution of active T-links introduced in equation (A.13) 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,

$$\pi^T_{P,t-1}(A) = \frac{\tau}{\tau + (1-\tau)\,\tau_{t-1}}\tilde{\pi}^T_{P,t-1} + \frac{(1-\tau)\,\tau_{t-1}}{\tau + (1-\tau)\,\tau_{t-1}}\pi^T_{P,t-1}, \tag{A.18}$$

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 (A.17).

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:

$$\tilde{\pi}^{0,T}_{C,t-1} = \sum_{k=0}^{\varphi_C(c^s_{t-1})+\varphi_N(n^s_{t-1})+\varphi_O} \left[1-(1-\pi_{IS})^{k-1}\right] f^T_{t-1}(k), \tag{A.19}$$

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 $t-1$.[31]

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

[31] 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.

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.[32]

A.3 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 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:[33]

$$g^{i,A}_{t-1}(\underline{k}_{t-1}|k_{t-1}, A_j = 1) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}, (1-\pi_R)(1-\pi^{A}_{t-1,P})\right) \quad i \in \{A,T,S\}, \tag{A.20}$$

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

$$g^{i,A}_{t-1}(\underline{k}_{t-1}|k_{t-1}, A_j = 2) = \mathcal{B}\left(\underline{k}_{t-1}, k_{t-1}-1, (1-\pi_{IS}-\pi_R)(1-\pi^{A}_{t-1,P})\right) \quad i \in \{A,T,S\}. \tag{A.21}$$

As before, this probability is the same across the three types of agents considered (Type-A,

[32] 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 $t-1$.

[33] 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, $\pi^{A}_{t-1,P}$.

Type-T, or Type-S), which are denoted by i.

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

$$g_{t-1}^{i,A}(\underline{k}_{t-1}|k_{t-1}) = \iota_{t-1}^{i}(k) \cdot \tilde{g}_{t-1}^{i,A}(\underline{k}_{t-1}|k_{t-1}) + (1-\iota_{t-1}^{i}(k)) \cdot \hat{g}_{t-1}^{i,A}(\underline{k}_{t-1}|k_{t-1}), \tag{A.22}$$

where $i \in \{A,T,S\}$ and $\iota_{t-1}^{i}(k)$ denotes the weights, which of course depends on the number of total contacts, k, the agent who met with the 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:

$$\iota_{t-1}^{S}(k) = (1-\pi_{IS})^k. \tag{A.23}$$

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

$$\iota_{t-1}^{i}(k) = \frac{(1-\pi_{IS})^k}{(1-\pi_{IS})^k + [1-(1-\pi_{IS})^k]\left(1-\pi_{t-1,T}^{0}(1-\pi_F)\right)}, \quad i \in \{A,T\}. \tag{A.24}$$

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

At last, we take the step made in equation (A.14) and obtain the marginalized probability distribution of active A-links for the three types: $g_{t-1}^{i,A}(\underline{k}_{t-1})$ for $i \in \{A,T,S\}$.

A.4 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:

$$\begin{aligned} E_t^1 =& (1-\pi_{C,t}^{0,A})\left[\frac{I_{t-1}^{A}(1-\pi_{IS}-\pi_R)\left(1-\pi_{P,t-1}^{A}\right)}{I_t^A}\pi_{C,t}^{1,A} + \frac{T_{t-1}\left(1-\pi_{P,t-1}^{T}\right)}{I_t^A}\pi_{C,t}^{1,T}\right](1-\pi_{IS})I_t^A \\ &+ (1-\pi_{C,t}^{0,S})\pi_{C,t}^{1,S}S_t + (1-\pi_{C,t}^{0,R})\left[\frac{R_{t-1}^{A}}{R_t^A}\pi_{C,t}^{1,R} + \frac{\pi_R I_{t-1}^{A}}{R_t^A}\pi_{C,t}^{1,RA}\right]R_t^A, \end{aligned} \tag{A.25}$$

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 (A.25).

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 (A.6) 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.11) 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 (A.24) 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 $\pi_{C,t}^{1,S}$.

B Quarantine Exposed Contacts Without Tests

We extend the model to include a scenario, in which the government can quarantine (a subset of) the traced agents without administering a test. As a consequence, the model features now a new group of quarantined agents. The group contains all agents that have been traced and put into quarantine without being tested. As no test is administered, the health status of quarantined agents is not publicly observed. This implies that the group contains susceptible agents, asymptomatic agents and unobserved recovered agents, which cannot be distinguished by the government. We assume that the imposed quarantine, which

is implemented as a tax on consumption μ^Q is the same as for positive tested agents. Note that without testing now lots of susceptible subject will be quarantined and since tests are not available, some infected agents may leave the quarantine still asymptomatic and infect susceptible subjects.

In particular, we define two different strategies. In the first one, the government has no tests available. Hence, policymakers have to quarantine all the close contacts traced every week. The case of no tests is called "all exposed contacts quarantined". In the second one, the government administers tests. However, the government combines now testing with a tighter quarantine policy if not enough tests are available. In particular, policymakers decide to quarantine all the agents for whom no test is available. Thus, if tests are scarce, a fraction of traced contacts is tested and their health status is observed. The other fraction is sent directly into quarantine without a test. This case is called "tighter quarantine".

B.1 Extended Model

The incorporation of this additional possibility slightly alters the setup of the model, as outlined here. The two scenarios are described in the next subsection.

B.1.1 Agents with Changed Optimization Problem

The additional possibility of quarantine alters the maximization problem of the agents with unobserved health status. Health authorities can directly quarantine susceptible, asymptomatic and unobserved recovered subjects without conducting a test. This implies that the model features now three additional types: quarantined susceptible subjects, quarantined untested asymptomatic subjects and quarantined unobserved recovered subjects.

The possibility of quarantine without tests alters the decision problem for the susceptible, asymptomatic and unobserved recovered. The probability for an individual with unknown health status to get quarantined is $\pi^i_{Q,t},\ i \in \{T, A, S\backslash T, U\}$, where the individuals can be separated as newly infected (T), asymptomatic infected (A), susceptible that did not get infected $(S\backslash T)$ and unobserved recovered U. The value functions for these agents include now the probability of getting quarantined without test:

$$V^S_t = \max_{c^s_t, n^s_t} u\left(c^s_t, n^s_t\right) + \beta \left[\begin{array}{l} (1-\tau_t)\left\{(1-\pi^{S\backslash T}_{Q,t})V^S_{t+1} + \pi^{S\backslash T}_{Q,t} V^{S,Q}_{t+1}\right\} + \dots \\ \tau_t \left\{\pi^T_{P,t} V^P_{t+1} \pi^T_{Q,t} V^{A,Q}_{t+1} + \left(1-\pi^T_{P,t}-\pi^T_{Q,t}\right) V^A_{t+1}\right\} \end{array} \right], \tag{B.1}$$

$$V^A_t = u(\tilde{c}^s_t, \tilde{n}^s_t) + \beta \left[\begin{array}{l} \pi_{IS} V^{IS}_{t+1} + \pi_R V^{UR}_{t+1} + (1-\pi_{IS}-\pi_R) \times \dots \\ \left(\pi^A_{P,t} V^P_{t+1} + \pi^A_{Q,t} V^{AQ}_{t+1} + (1-\pi^A_{Q,t}-\pi^A_{P,t}) V^A_{t+1}\right) \end{array} \right], \tag{B.2}$$

$$V_t^{UR} = u(\tilde{c}_t^s, \tilde{n}_t^s) + \beta \left[(1 - \pi_{Q,t}^{UR}) V_{t+1}^{UR} + \pi_{Q,t}^{U} V_{t+1}^{U,Q} \right] \tag{B.3}$$

where $V_{t+1}^{i,Q},\ i\{S, A, U\}$ is the value function of the three new quarantined types, which are derived below. The probabilities of getting quarantined will be characterized in Section B.2.

Quarantined agents have not been tested by the government so that the health status is not revealed. If quarantined agents do not develop symptoms, it is assumed that agents leave the quarantine with a probability π_Q.[34] We set $\pi_Q = 0.5$ in the quantitative part, which implies an average quarantine duration of 2 weeks. This implies that quarantined agents stay on average two weeks in quarantine. As the agents do not observe their type in quarantine, we keep on assuming that they behave like quarantined susceptible in such a scenario of limited information.[35] Thus, quarantined agents choose consumption, c_t^P and labor n_t^P to maximize their utility as quarantined susceptible:

$$V_t^{S,Q} = \max_{c_t^Q, n_t^Q} u\left(c_t^Q, n_t^Q\right) + \beta \left[\pi_Q V_t^S + (1 - \pi_Q) V_t^{S,Q} \right] \tag{B.4}$$

$$\begin{aligned} V_t^{A,Q} = u\left(\tilde{c}_t^Q, \tilde{n}_t^Q\right) + \beta \Big[& \pi_{IS} V_{t+1}^{IS} + \pi_Q \left\{ \pi_R V_{t+1}^{U} + (1 - \pi_{IS} - \pi_R) V_{t+1}^{A} \right\} + \\ & (1 - \pi_Q) \left\{ \pi_R V_{t+1}^{U,Q} + (1 - \pi_{IS} - \pi_R) V_{t+1}^{A,Q} \right\} \Big], \end{aligned} \tag{B.5}$$

$$V_t^{U,Q} = u\left(\tilde{c}_t^Q, \tilde{n}_t^Q\right) + \beta \left[\pi_Q V_t^U + (1 - \pi_Q) V_t^{U,Q} \right] \tag{B.6}$$

where $\tilde{c}_t^Q$ and $\tilde{n}_t^Q$ denote the optimal solution to the problem of the quarantined susceptible agents. The budget constraint is the same as for the positive tested agents:

$$\left(1 + \mu_c^Q + \alpha \mu_{c,t}^S\right) c_t^Q = w_t^P n_t^Q + \Gamma_t^{QQ}, \tag{B.7}$$

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

The government balances its budget in every period. There is one additional equation now because the revenue of the social distancing and quarantine taxes are rebated to the quarantined agents, on which these taxes are levied on:

$$\mu_{c,t}^S \left[C_t + \alpha \left(C_t^{IS} + C_t^Q \right) \right] = \Gamma_t^L \left(S_t + I_t^A + R_t^U + R_t^O + (1 - \alpha) \left(I_t^S + Q_t + P_t \right) \right), \tag{B.8}$$

$$\mu_c^Q \cdot C_t^Q = \Gamma_t^{QQ} \cdot Q_t, \tag{B.9}$$

[34] For computational reasons, we assume that quarantine has a stochastic duration in the absence of tests.

[35] Conditional on the belief of never having been infected, agents' beliefs about future changes in their health status are model consistent.

where we denote the share of quarantined individuals with $Q_t = Q_t^S + Q_t^A + Q_t^{UR}$, which is aggregate of the susceptible, asymptomatic and unobserved recovered agents in quarantine. $C_t^Q \equiv c_t^Q Q_t$ stands for total consumption of the quarantined (not tested) agents.

The maximization problem for the tested-positive agents, infected symptomatic agents and observed recovered agents as well as the remaining equations are unchanged.

B.1.2 Dynamics of Agents' Types

The possibility of quarantine also alters the dynamics of some types. Since susceptibles can be placed in quarantine and can return from quarantine, the law of motion for susceptibles evolves now as follows:

$$S_{t+1} = (1 - \pi_{Q,t}^{S\setminus T})(S_t - T_t) + \pi_Q Q_t^S \tag{B.10}$$

The size of non-quarantined asymptomatic agents evolves according to the law of motion

$$I_{t+1}^A = (1 - \pi_{P,t}^T - \pi_{Q,t}^T)T_t + (1 - \pi_{P,t}^A - \pi_{Q,t}^A)(1 - \pi_{IS} - \pi_R)I_t^A + \pi_Q(1 - \pi_{IS} - \pi_R)Q_t^A, \tag{B.11}$$

where we account that if asymptomatic agents leave quarantine too early, they belong again to the group of non-quarantined asymptomatic agents. Furthermore, the model features also the new typoe of quarantined subjects, which consists of susceptible, asymptomatic and unobserved recovered subjects and is given by

$$Q_{t+1} = Q_{t+1}^S + Q_{t+1}^A + Q_{t+1}^U \tag{B.12}$$
$$Q_{t+1}^S = (1 - \pi_Q)Q_t^S + \pi_{Q,t}^{S\setminus T}(S_t - T_t) \tag{B.13}$$
$$Q_{t+1}^A = (1 - \pi_Q)(1 - \pi_{IS} - \pi_R)Q_t^A + \pi_{Q,t}^T T_t + \pi_{Q,t}^A(1 - \pi_{IS} - \pi_R)I_t^A. \tag{B.14}$$
$$Q_{t+1}^U = (1 - \pi_Q)Q_t^U + \pi_{Q,t}^U R_t^U + (1 - \pi_Q)\pi_R Q_t^A \tag{B.15}$$

The pool of infected symptomatic agents evolves now as follows:

$$I_{t+1}^S = (1 - \pi_R - \pi_D)I_t^S + \pi_{IS}(I_t^A + P_t + Q_t^A). \tag{B.16}$$

The share of unobserved recovered is given as:

$$R_{t+1}^U = (1 - \pi_{Q,t}^U)R_t^U + \pi_R I_t^A + \pi_Q Q_t^U + \pi_Q \pi_R Q_t^A. \tag{B.17}$$

All other remaining equations are unchanged. The only four variables not yet defined are

the probabilities of getting quarantined without a test for newly infected agents, susceptible agents that did not get infected, untested asymptomatic agents and unobserved recovered agents.

B.2 Probability of Getting Quarantined Without Tests

We are now characterizing the probability to getting quarantined for the two scenarios.

B.2.1 Strategy 1: All Exposed Contacts Quarantined

The strategy is to quarantine all current week exposed contacts without administering any tests. As a consequence, the probability to get tested is zero, that is: $\pi^T_{P,t} = \pi^A_{P,t} = 0$. However, the probabilities to get traced are exactly the same as for the contact tracing technology. As every traced contact gets send into quarantine, we have the following proabilities for the newly infected subjects, asymptomatic infected subjects and unobserved recovered subjects:

$$\pi^A_{C,t} = \pi^{UR}_{C,t} = \sum_{k=0}^{\varphi_C(c^s_t)+\varphi_N(n^s_t)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f_t(k), \tag{B.18}$$

$$\pi^T_{C,t} = \sum_{k=0}^{\varphi_C(c^s_t)+\varphi_N(n^s_t)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f^T_t(k). \tag{B.19}$$

where the tracing probabilities are taken directly from equations (23) and (25). The only additional element that we need to derive is the tracing probability for the susceptibles that did not get infected $\pi^{S\setminus T}_{Q,t}$. Similar to the logic for the newly infected, we now need to get the probability of meeting k asymptomatic agents conditional on not getting infected. To do so, we apply the Bayes theorem to obtain

$$f^{S\setminus T}_t(k) = \frac{f_t(k)(1-\tilde{\tau}(k))}{1-\tau_t}, \tag{B.20}$$

where $1-\tilde{\tau}(k) \equiv (1-\tau)^k$ is the probability to have no infectious contact out of k interactions, and τ_t stands for the average probability for susceptible subjects to get infected. We can then characterize the tracing probability of a suceptible that is not infected as:

$$\pi^{S\setminus T}_{C,t} = \sum_{k=0}^{\varphi_C(c^s_t)+\varphi_N(n^s_t)+\varphi_O} \left[1-(1-\pi_{IS})^k\right] f^{S\setminus T}_t. \tag{B.21}$$

B.2.2 Strategy 2: Tighter Quarantine (Combination of Tests and Quarantine)

This strategy requires to conduct tests for the traced agents first. If no tests are available anymore, the reminding subset of traced individuals is quarantined. Thus, this strategy combines testing and directly quarantining exposed contacts without being tested. Importantly, there is a pecking order as the government first tries to test everyone. This implies that the probability of getting tested $\pi^T_{P,t}$ and $\pi_{P,t}$ is the same as for the contact technology characterized in Section 3. Therefore, a traced agent can only get quarantined if there are more exposed contacts then tests available, which is $E_t > \Upsilon_t$. Importantly, the probability of quarantining a traceable contact does not depend on the contact health status, but in fact on the amount of exposed relative to tests, which is

$$\pi_{Q,t} = max(0, \frac{E_t - \Upsilon_t}{E_t}) \tag{B.22}$$

The quarantine probabilities for the different agents are then given as

$$\pi^i_{Q,t} = \pi^i_{C,t} \cdot \pi_{Q,t}, \quad i \in \{S\backslash T, T, A, U\}, \tag{B.23}$$

where the tracing probabilities are the same as for strategy 1.

B.3 Quantitative Analysis

Figure B.5 compares the implications of having all the contacts of confirmed cases quarantined without testing them first (strategy 1). This is the black dotted line. To assess the importance of the complementarities between tracing and testing, one should compare that line with the blue line that shows the implications of quarantining only those contacts who test positive in a scenario with unlimited testing capacity availability. The difference between these two lines illustrates the gain from having enough tests available to evaluate the health status of close contacts and to decide when quarantined agents can leave quarantine. This shows that testing improves outcomes quite a bit. In particular, testing combined with tighter quarantine (green dash-dotted line) allows policymakers to lower quite considerably the death toll (Deaths graph) and to make the pandemic recession substantially less severe (Aggregate Consumption graph).[36]

[36]Cumulative deaths increases because the stochastic duration of quarantine, which is calibrated to be 2 weeks in expectation, implies that some quarantined agents are still asymptomatic infected when they leave the quarantine.

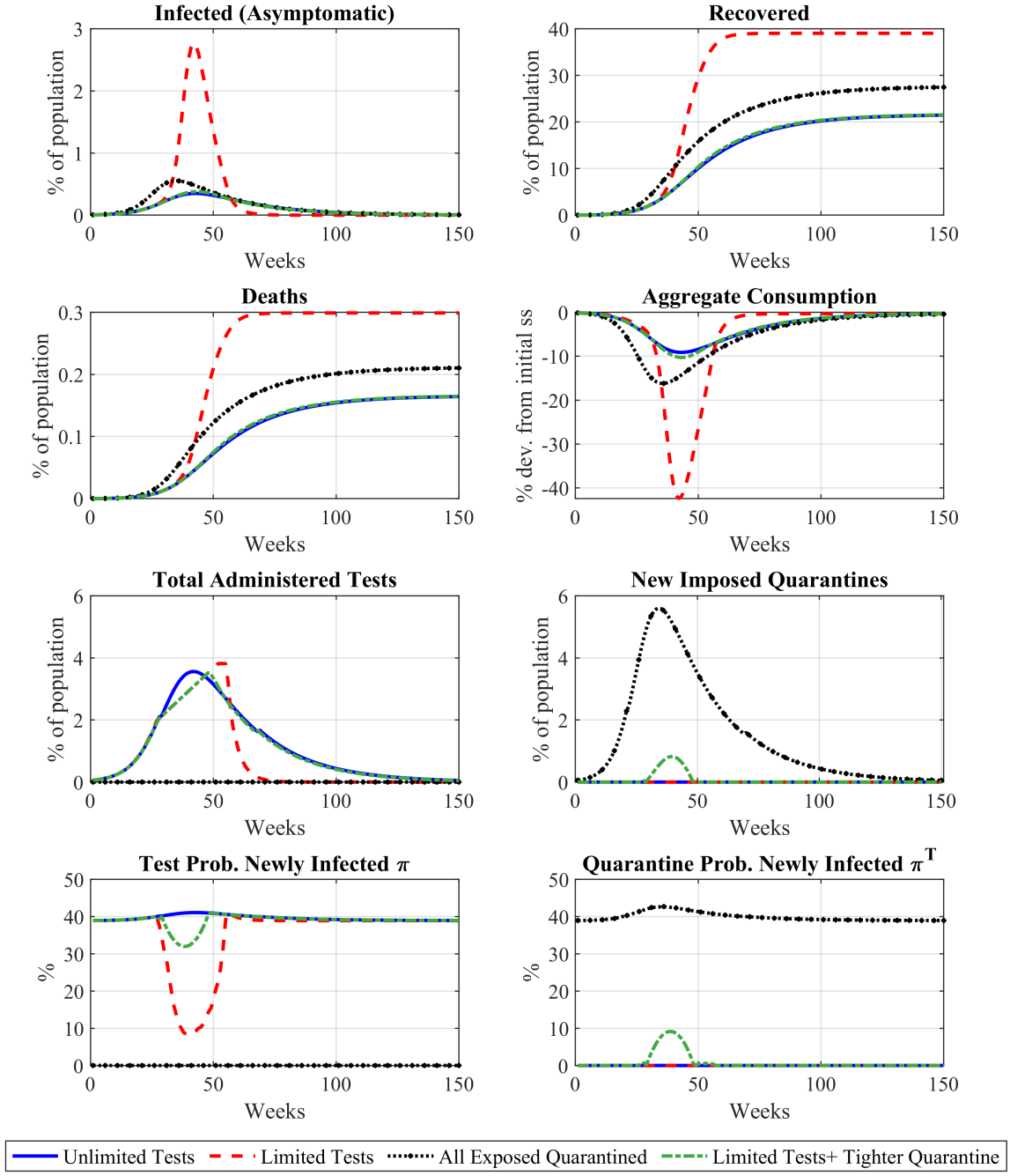

Figure B.5: Analysis of the impact of the testing capacity for contact tracing. The considered scenarios are contact tracing with unlimited testing capacity (blue solid line), contact tracing with limited testing capacity (red dashed line), contact tracing with quarantining all traced conducts without administering any tests (black dotted line) and mixing testing and quarantine, in which only the agents are quarantined for whom no test is available (green dash-dotted line).

C Random Allocation of Tests

An alternative to a contact tracing strategy would be to test (a subset of) the population randomly. For this testing approach, the government does not use or acquire any strategic information from contact tracing on how to efficiently use the available tests.[37] As a consequence, the probability of getting tested is the same for the susceptible, untested asymptomatic, and unobserved recovered agents. We consider two different strategies, in which the government relies (partially) on random testing.

The first strategy allocates its entire testing capacity to test the agents randomly. We denote this scenario as "random testing". The second scenario combines contact tracing and random testing. The idea is to conduct contact tracing in a first step. The government administers then tests for the traced contacts. If there are some leftover tests after this step, the government allocates then, in a second step, the remaining tests randomly among the untraced population, for which the health status is not observed. We denote this scenario as "Contact Tracing + Random Tests".

C.1 Scenario 1: Random Testing

This strategy randomly allocates the tests among the agents, for which the health status is not observed. This implies that the probability of getting tested is the same for the susceptible, untested asymptomatic, and unobserved recovered agents.

To derive the probability of getting tested, it is helpful to interpret this as an extreme case of contact tracing. In this approach, every agent gets traced because random testing considers every agent with unobserved health status for testing. The tracing probabilities are then given as:

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

Compared to contact tracing, the tracing probabilities do not contain any additional strategic information anymore.

As every agent get traced, the number of subjects to be tested is very large. The pool of agents that the government tests is given as

$$E_t = S_t + (1 - \pi_{IS})A_t + R^U_t. \tag{C.2}$$

The government has the number of tests Υ_t available. Therefore, the probability of getting

[37] We assume that the government does not test observed recovered as well as quarantined agents.

tested conditionally on being traced depends on the number of tests Υ_t relative to the pool E_t:

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

We can plug equations (C.1) and (C.3) into equation (21) to evaluate the probability of testing positive for newly infected subjects, $\pi^T_{P,t}$, and subjects infected in earlier periods, $\pi^A_{P,t}$.

C.2 Strategy 2: Contact Tracing Combined with Random Testing

This strategy combines contact tracing and random testing. The government conducts contact tracing and testing in a first step. If some testing capacity is left after contact tracing, the remaining tests are used to randomly test the population. Importantly, the implied pecking order (1. contact tracing + testing, 2. random testing) is optimal since it is more likely to catch an infected agent with contact tracing than with random testing. We also assume that an agent gets tested only once, that means that a traced contact is excluded from the random testing pool.

The setup implies that the probability of getting tested for the newly infected and subjects infected in earlier periods depends now on the probability of testing positive via tracing as well as random testing:

$$\pi^i_{P,t} = \pi^{i,Tr}_{P,t} + \pi^{i,RT}_{P,t}, \quad i \in \{A, T\}. \tag{C.4}$$

where the superscript Tr indicates contact tracing and RT indicates random testing. The pecking order implies that the probabilities to get detected via tracing is directly given from the contact tracing technology derived in Section 3.

The pecking order implies that the probability to get a positive test result via random testing is conditional on not being traced via contact tracing. This can be interpreted as an extreme case of contact tracing, in which every agent, that has not been traced via contact tracing in a first stage, gets traced. We use this definition to derive the adjusted probabilities without introducing new notation. Thus, the probability of being traced via random testing can be written as

$$\pi^{i,RT}_{C,t} = 1 - \pi^{i,Tr}_{C,t}, \quad i \in \{A, S, T, R^U\}. \tag{C.5}$$

Therefore, the random testing part can be interpreted as tracing of all agents that have not

been traced initially, which is then the entire pool of traceable agents minus the agents that have already been traced via contact tracing. Thus, the pool of traced agents for random testing is:

$$E_t^{RT} = S_t + (1 - \pi_{IS})A_t + R_t^U - E_t^{Tr}, \tag{C.6}$$

$$= S_t + (1 - \pi_{IS})A_t + R_t^U - \left(\pi_{C,t}^S S_t + \pi_{C,t}^A (1 - \pi_{IS}) I_t^A + \pi_{C,t}^{UR} R_t^U\right), \tag{C.7}$$

where E_t^{Tr} is the pool of agents that has been traced with the contact tracing technology. The government has at this stage the number of tests $max(\Upsilon_t - E_t^{Tr}, 0)$ available. Therefore, the probability of getting tested randomly (conditionally on not being traced via contact tracing in the first stage) is

$$\pi_{P,t}^{i,RT} = \min\left(1, \frac{max(\Upsilon_t - E_t^{Tr}, 0)}{E_t^{RT}}\right), \quad i \in \{A, T\}. \tag{C.8}$$

Thus, the probability of tesing positive for newly infected subjects and subjects infected in earlier periods via random testing is given as:

$$\pi_{P,t}^{i,RT} = \pi_{C,t}^{i,RT} \cdot \pi_{P,t}^{i,RT} \cdot (1 - \pi_F), \quad i \in \{A, T\}. \tag{C.9}$$

C.2.1 Quantitative Analysis of this Strategy

The contribution of this strategy (combination of contact tracing and random testing) is shown in Figure C.6. The scenario compares contact tracing relative to the outlined strategy, where the excess tests capacity is allocated randomly. The marginal gains of this random allocation is negligible. The probability to test an infected agents increases only slightly and policymakers cannot avert the collapse of the testing system due to shortage of tests. The testing system collapses slightly later when random testing is combined with contact tracing as one can see by noting that the number of infected asymptomatic subjects now peaks two weeks later.

D Model Solution

Solution Algorithm The solution algorithm solves the model iteratively based on a numerical root finder relying on perfect foresight expectations. 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 $\{\mu_{c,t}^S\}_{t=1}^T$ and given initial asymptomatic and symptomatic infected agents: $\{I_1^A, I_1^S\}$. The algorithm is summarized below:

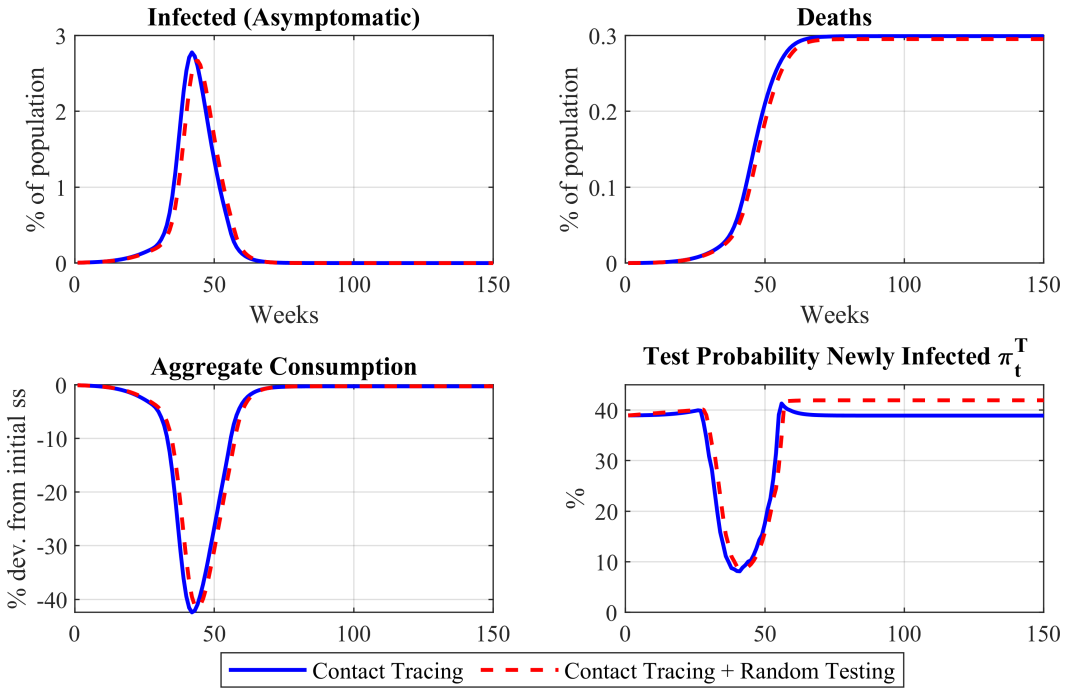

Figure C.6: Combining contact tracing with random testing. The impact of using the remaining test capacity in a second step (after testing each traced contact) for random testing is shown.

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 $\{\Gamma_t^L\}_{t=1}^T$.[38]

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 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

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

on these objects, the dynamics 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 tested-positive and infected symptomatic agents, the government budget constraint for the social distancing 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 $\{n_t^R, n_t^{IS}, n_t^P, n_t^{RO}\}_{t=1}^T$.

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 ($\pi_{P,t}^T$ and $\pi_{P,t}^A$) in Section 3, we use the exact definition of the rate τ_t in equation (5).

E 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

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

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

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

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 (E.2) leads to equation (6).

F Welfare, Ramsey Problem and Optimal Social Distancing

To make explicit the objective function of the government, we use social welfare to evaluate different combinations of containment policies. The welfare changes are expressed as consumption equivalents relative to the non-pandemic economy. We also express the social costs in U.S. dollars. In addition to this, we determine the optimal social distancing policy by solving the Ramsey problem.

Welfare Objective The social welfare of the economy is

$$U_t = S_t V_t^S + I_t^A V_t^A + I_t^S V_t^{IS} + P_t V_t^P + R_t^U V_t^{UR} + R_t^O V_t^{OR}, \tag{F.1}$$

which is the weighted lifetime utility of the different agent types. We compare the welfare gains/losses at the beginning of the pandemic:

$$U_0 = S_0 V_0^S + I_0^A V_0^A + I_0^S V_0^{IS}, \tag{F.2}$$

where the initial measure for some types is zero ($P_0 = R_0^U = R_0^O = 0$).

We express the welfare losses (gains) as consumption equivalents for the non-panemic economy. In particular, we want to determine the maximum fraction of consumption ξ that an agent would be willing to indefinitely forgo in a non-pandemic world to avoid a specific

pandemic scenario. This consumption equivalent measure ξ depends on the social welfare at the beginning of a pandemic scenario U_0 and the utility in the non-pandemic world $U^\star$:

$$\xi = \exp\left((1-\beta)(U_0 - U^\star)\right) - 1, \tag{F.3}$$

where the superscript $\star$ indicates the non-pandemic world and the social welfare of the non-pandemic economy is: $U^\star = \frac{1}{1-\beta}\left(\log(c^\star) - \frac{\theta}{1/\eta}(n^\star)^{1/\eta}\right)$.[39] We use the consumption equivalent measure to calculate the social costs of the pandemic in the U.S.

The social costs are the net discounted consumption equivalent gains/losses of the entire economy:

$$\text{Social Costs} = \frac{\xi C^\star}{1-\beta} \times \text{U.S. Population}_{2019}, \tag{F.4}$$

where the total population in the U.S. was around 328 million people in 2019. The social costs could be interpreted as the willingness to invest in some technology to avoid a specific pandemic course.

Ramsey Problem and Optimal Social Distancing To determine the optimal social distancing path, we solve the Ramsey problem of the economy. The instrument of the Ramsey planner is a non-negative tax on consumption. The government can commit to setting the tax path for H periods. The planner's problem of choosing the optimal sequence of consumption tax rates $\{\mu^S_{c,t}\}_{t=0}^H$ can be expressed as:

$$\underset{\{\mu^S_{c,t}\}_{t=0}^H}{\text{argmax}}\, U_0. \tag{F.5}$$

We find the optimal consumption tax path by using a numerical solver that maximizes the objective function over the sequence of consumption tax rates.

G Mask-Wearing Mandate and Furlough Scheme

G.1 Mask-Wearing Mandate

We consider as alternative policy a more stringent mask-wearing mandate. Such a policy is modeled by reducing the probability of agents getting infected conditional on meeting

[39] Social welfare can be conditioned on the consumption equivalent parameter ξ: $U^{\xi\star} = \log(c^s(1+\xi)) - \frac{\theta}{1/\eta}n^{1/\eta} + \beta U^{\xi\star} = \frac{1}{1-\beta}log(1+\xi^S) + \log(c^s) - \frac{\theta}{1/\eta}n^{1/\eta} + \beta U^\star = \frac{1}{1-\beta}log(1+\xi^S) + U^\star$. Using this result, the costs of a pandemic can be expressed in consumption equivalents: $\xi = \exp\left((1-\beta)(U_0 - U^\star)\right) - 1$.

an asymptomatic spreader, τ. Broadening the set of activities for which wearing masks is mandatory tends to increase social interactions because the agents' probability of getting infected at a single interaction falls. While a lower risk for agents to catch the virus obviously improves the economic and health outcome of a pandemic, by spurring social interactions a mandatory mask mandate can –all else being equal– increase the number of agents exposed to confirmed cases. This requires then to administer more tests. So a more stringent mask mandate may even add strain to the tracing system in the short run. In this light, this containment measure seems to be less complementary to contact tracing than the measures we have analyzed earlier in this paper.

Figure G.7 highlights that a mask mandate can create additional pressure on the tracing and testing system in the short-term. In the displayed simulation, the government unexpectedly introduces a mask mandate in the midst of a pandemic, in which the contact tracing and testing system is already collapsing (period 42). The simulations consider two differently stringent mask mandates, which reduce τ either by 10% or 50%. The imposed mask mandate implies that asymptomatic infected agents are less likely to transmit the virus in an interaction. Thus, the number of new infections is falling, which lowers the pressure on the tracing and testing system. At the same time, agents respond to the reduced threat of the virus by increasing their interactions. This implies that each asymptomatic agent has now more meetings, which enlarges the number of traced contacts. The impact of these opposing channels on the viability of the testing system is unclear. It turns out that the total amount of traced contacts increases in the very short run in the considered scenarios. The increase of traced contacts dampens the positive impact of the mask mandate relative to an economy, in which there would be no shortage of tests. It also highlights that a mask mandate does not necessarily stop the collapse of the testing system in the very short-run. Of course, as mentioned earlier, the Figure also points out that a mask mandate improves the economic and health outcomes. The positive effects also strengthen the viability of the testing system in the longer-term. The analysis suggests that a temporary imposed social distancing policy would support the introduction of a mask mandate if the government faces a shortage of tests.

G.2 Furlough Scheme

We assess how a potential furlough scheme affects contact tracing by focusing on the costs of quarantining agents. In particular, we vary the households' costs of quarantining, which are proxied with a tax on consumption μ_c^Q. Reducing the tax lowers the quarantine costs and captures the essence of introducing a more comprehensive furlough scheme. Figure G.8

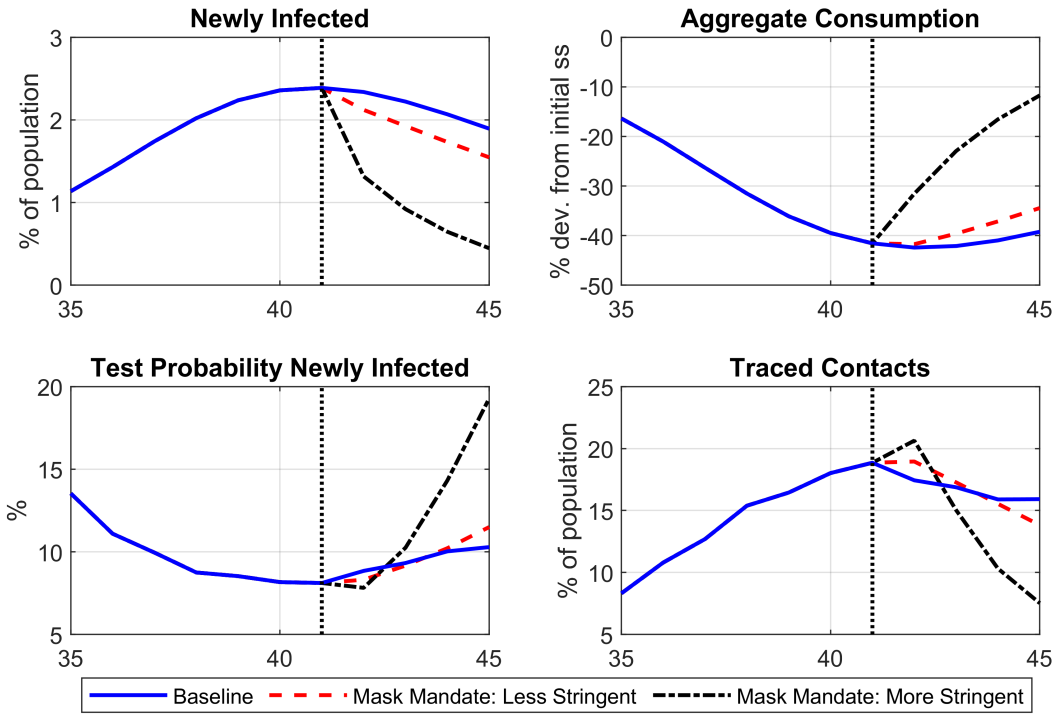

Figure G.7: Mask-wearing mandate and limited testing capacity. Baseline scenario (contact tracing with limited testing capacity) is contrasted to the case, in which mask mandates are unexpectedly introduced in period 42. The simulations show a less stringent mask mandate (reduction in τ by 10%) and more stringent mask mandate (reduction in τ by 50%).

shows the impact on welfare, average consumption and deaths for different tracing scenarios (contact tracing, comprehensive contact tracing, and quarantining all exposed contacts). Lowering the tax increases welfare and average consumption slightly. The reason is that quarantined agents can now consume more. At the same time, the impact on welfare and average consumption is rather low.[40] The reason is that quarantine is a targeted policy that affects agents only for a short period of time (after positive testing throughout their infection period).

The potential gains of introducing a furlough scheme are much larger in the case, where the government quarantines every traced contact regardless of the test outcome. For this reason, the slope is steeper in the scenario, where everyone exposed gets quarantined. The reason is that the policy involves now a broader set of subjects –i.e., it is not targeted to only

[40]To be precise, a lowering of the tax to 0 even reduces welfare slightly compared to a very low tax. The reason is that agents also slightly reduce their consumption and labor supply because they internalize the costs of getting quarantined. This helps to reduce the spread of the inflation and can therefore be welfare improving. However, the effect is very small and is dominated by other effects if all exposed agents are quarantined.

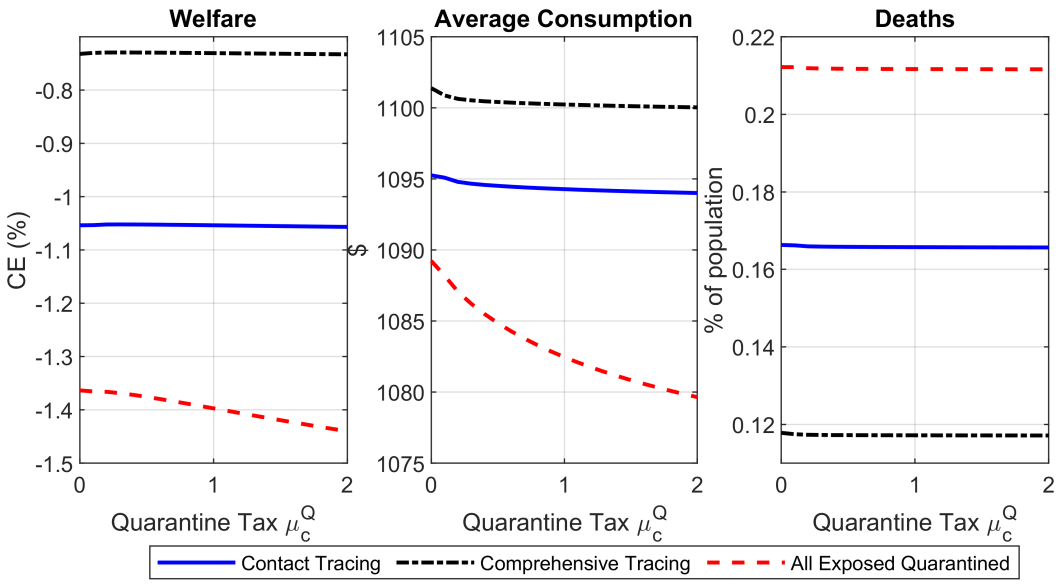

Figure G.8: The economic and epidemiological impact of different furlough schemes, expressed as the level of the quarantine tax μ_c^Q. The impact is compared for the following scenarios: contact tracing, comprehensive contact tracing and quarantine all exposed. Welfare is expressed as the consumption equivalent relative to the non-pandemic economy, consumption is averaged over the simulation horizon and deaths are shown as percent of the initial population.

those traced contacts who test positive. As a consequence, the importance of introducing a furlough scheme depends on the stringency of the quarantine policy. Our analysis suggests that a very well targeted quarantining approach considerably reduces the necessity of a furlough scheme.

H The Stringency of Social Distancing

We now turn our attention to the stringency of social distancing, which is captured by the consumption tax $\mu_{c,t}^S$. For the purpose of the analysis, the tax is imposed in the first 26 periods after the outbreak of the disease and is kept constant throughout this time.

Figure H.9 shows the impact of social distancing on the contact tracing system for varying degrees of stringency. In particular, the consumption tax, which is levied in during the first 26 periods, is increased step by step. We show the response of welfare at the onset of the pandemic, the cumulative deaths at the end of the pandemic as well as consumption and labor averaged over the entire horizon of 250 periods.

When no social distancing is imposed ($\mu_c^S = 0$), the contact tracing technology with a limited testing capacity alone cannot prevent the collapse of the testing system. As a

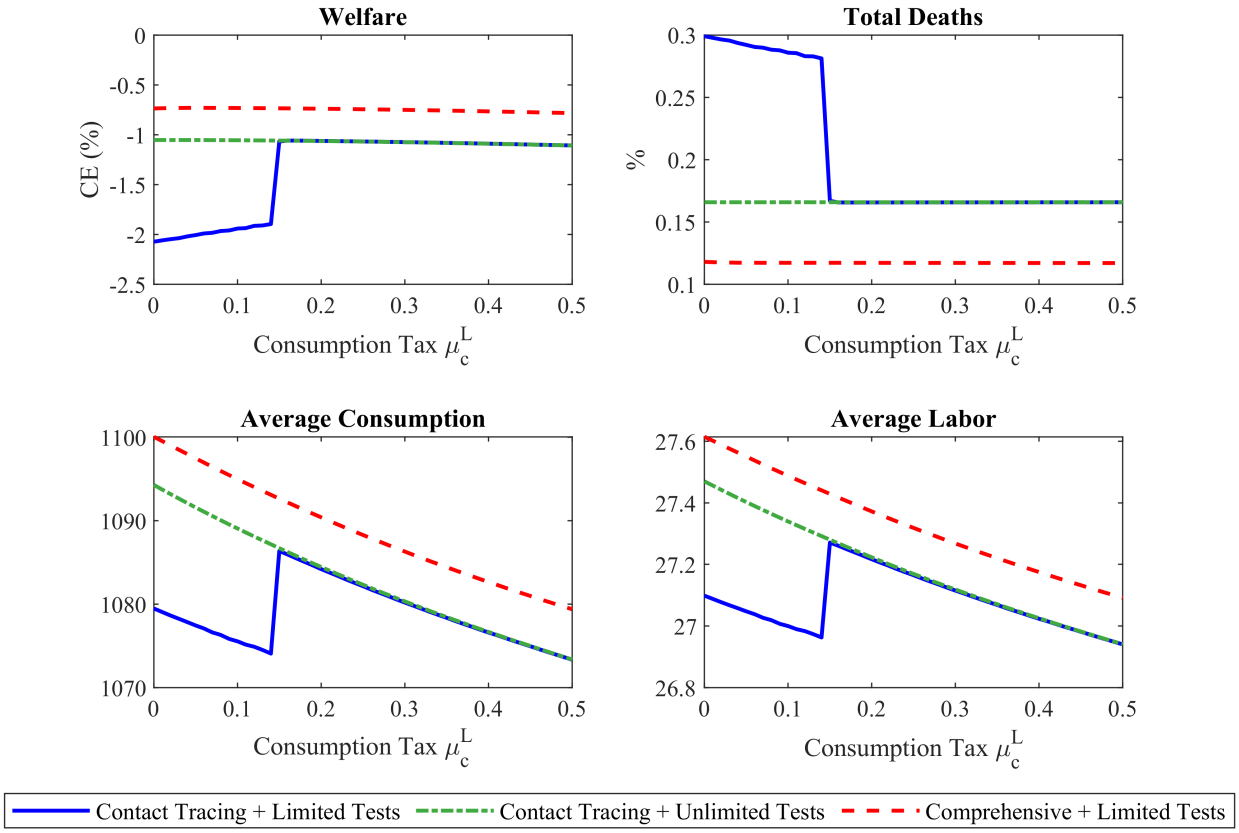

Figure H.9: Comparison of different testing strategies under a varying stringency of social distancing. Social distancing is captured by the consumption tax $\mu_{c,t}^S$, which is levied in the fist 26 periods and is kept constant throughout these periods. Welfare (expressed as consumption equivalent to the non-pandemic economy), accumulated deaths, aggregate consumption and aggregate labor averaged over the 250 week horizon are reported.

result, consumption and labor are lower and total deaths are higher than those in the case of unlimited testing (the green dashed-dotted line) where, by construction, the testing system cannot collapse. Indeed, when the social distancing stringency is set to zero ($\mu_c^S = 0$), the vertical distance between the blue solid line and the green dashed-dotted line captures the effects of the testing system's collapse on welfare, total deaths, aggregate consumption, and labor. As the stringency of the social distancing policy is increased, welfare increases as fewer people will be killed by the pandemic. However, consumption and labor fall steadily.

As the stringency of social distancing reaches the threshold $\mu_c^L = 0.18$, 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 social distancing.

Social distancing, if it is sufficiently constraining, allows the government to replicate the outcomes of the unlimited testing capacity (the green dashed-dotted line). This happens because social distancing reduces agents' individual consumption and labor so as to solve the externality problem 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 social distancing policy is raised.

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 consumption tax monotonically lowers consumption and employment. At the same time, social welfare slightly improves as social distancing reduces the amount of economic interactions, leading to fewer infected cases and hence to a lower death toll.

I Additional Figures

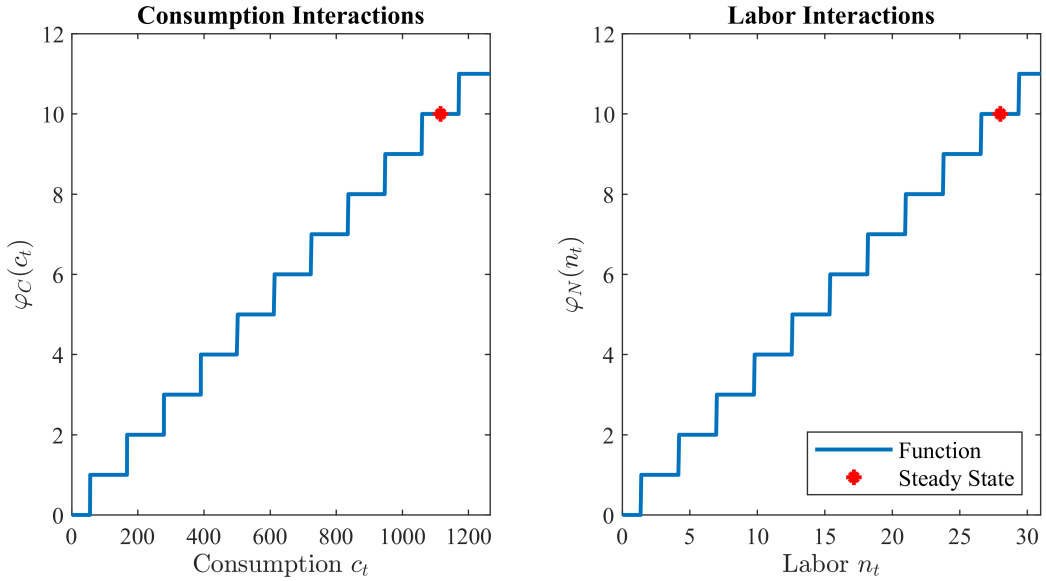

Figure I.10: Step functions mapping consumption and labor decisions in total consumption and labor interactions.

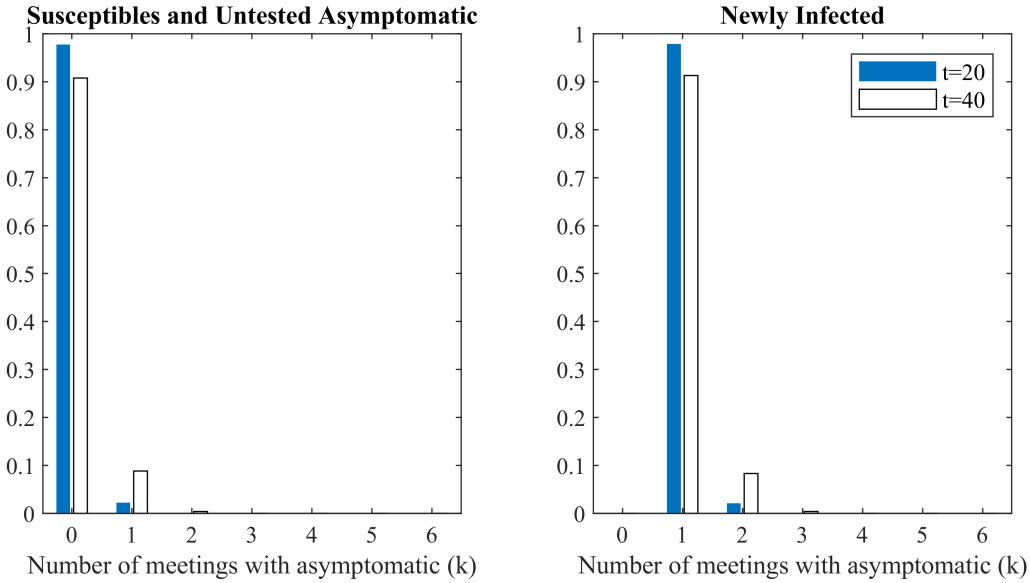

Figure I.11: Probability distributions for an agent, who does not know their health status, to meet with untested asymptomatic subjects k times. The left plot graphs the distribution $f_t(k)$ defined in equation (22) and concerns susceptible agents, who do not turn out to become infected in the period, 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 with a contact tracing technology with unlimited testing capacity.