Reopening in an SEIR model with Testing and Targeted Quarantine*

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Abstract

We show that testing and targeted quarantine make it possible to reopen an economy in such a way that output increases while mortality remains contained at low lockdown levels. We augment a standard Susceptible-Exposed-Infectious-Recovered (SEIR) model with (i) virological testing, (ii) serological testing, (iii) permanently asymptomatic individuals, and (iv) incomplete information. Virological testing allows for targeted quarantine of asymptotic spreaders. Serological testing allows for targeted release of recovered individuals. Fitted to US data, the model demonstrates that these admit a reopening policy that dampens (i) the economic impact of the COVID-19, (ii) substantially flattens the profile of symptomatic infections, and (iii) this can be accomplished almost entirely with virological tests. Implementing testing against a fixed budget, a regime with more frequent use of substantially lower quality (sensitivity) tests is preferred to perfect, but less frequent testing.

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"Once again, our key message is: test, test, test." — @WHO, March 16, 2020

Introduction

This paper shows how, during a pandemic, targeted lockdowns that can be achieved through testing can accommodate more aggressive reopening policies. Such policies can support economic output by freeing up labor supply, while not worsening public health outcomes. We do so by augmenting a standard Susceptible-Exposed-Infectious-Recovered (SEIR) model with (i) virological testing, (ii) serological testing, and (iii) temporarily and permanently asymptomatic individuals. This is important for understanding the response to COVID-19 given two facts.[1] First, there is a high rate of permanently asymptomatic cases conditional on infection. Second, individuals that eventually develop symptoms are nonetheless highly infectious in their pre-symptomatic stages. The existence of large quantities of temporary and permanent asymptomatic infection make testing and targeted quarantine especially useful tools to both lower deaths and raise output.

Given these two types of asymptomatic cases, there is a role for both virological and serological tests to mitigate the spread of the disease. Once infected, both permanent and temporary asymptomatic types are unaware they are infectious. Virological testing of those without symptoms identifies these types and makes possible targeted quarantine. Mortality and disease transmission fall, creating slack to reduce quarantine measures—which we refer to as *reopening*—, mitigating the decline in output. Recovered types that never developed symptoms are also unaware of their recovery. Serological testing of those without symptoms identifies these types and makes possible targeted release. Output increases, and disease transmission falls as recovered types crowd out matches between susceptible and infected types, leading to fewer deaths. Which test provides better health and economic outcomes is a quantitative question, which we attempt to answer.

To quantify these mechanisms, we include the minimal necessary modifications of the SEIR model. Figure 1 provides a model schematic. A standard Susceptible-Exposed-Infectious-Recovered (SEIR) model has four health states: susceptible (S), exposed (E), infected (I) and recovered (R). First, we split the exposed state into two groups: those who are temporarily asymptomatic and those are permanently asymptomatic, where only temporary asymptomatic cases develop symptoms before recovering or dying. Second, we assume that both types spread the disease.[2] Third, we introduce incomplete information. Asymptomatic individuals do not know their own health state, nor does the planner. This creates a

[1] As of June 2020 these have been documented in a number of peer-reviewed publications, e.g. see the meta-analysis of peer-reviewed and unpublished papers in Byambasuren, Cardona, Bell, Clark, McLaws, and Glasziou (2020).

[2] In the textbook SEIR model, exposed (E) types do not spread the disease and play very little role other than slowing down disease dynamics.

role for virological tests. Fourth, our two types of infection generate two types of recovered individuals: known recovered and unknown recovered. Incomplete information implies that unknown recovered and susceptible types cannot be distinguished. This creates a role for serological tests.

We calibrate the model in order to construct counterfactual time-series for health outcomes and economic output, where we assume that economic output is proportional to the number of healthy non-quarantined individuals. We adopt as many agreed-upon medical parameters from the literature as possible and calibrate the remaining model parameters. Since many parameters are unknown we conduct extensive robustness exercises. In our calibration we take as given the shutdown of the US economy over March and April 2020, and choose the rate of disease transmission and effectiveness of the shutdown to match the time-series of deaths between March and mid June.

From this starting point our main counterfactual answers the question, *"If testing is increased, how much can the economy be reopened while containing the losses of lives?"*. Testing saves lives as it comes coupled with targeted quarantine (release) measures in the case of virological (serological testing). We define reopening as the de-quarantining of individuals with unknown health status, which costs lives due to increased disease transmission. In our benchmark without reopening we compute total deaths under the pandemic: D. In economies with testing at frequencies between one to 12 weeks, we then compute the permissible reopening such that deaths do not exceed D. We then compare the non-testing benchmark to the testing counterfactual in terms of the profile of symptomatic infections, and output losses.

Our main result is that by combining elevated testing with targeted quarantine, it is possible to reopen the economy and produce more while maintaining the same long-run deaths as the benchmark U.S. policy. We find that if virological tests are administered on a weekly basis to the U.S. population, then we can lower cumulative output losses by 90 percent. Monthly testing accommodates reopening and lowers output losses by 20 percent. Second, the 'curve' of symptomatic infections flattens, with the balance of quarantining positive cases and releasing unknown cases leading to the same number of infections, but spread out in a way that is more manageable for the healthcare system. Third, we find that serological testing has quantitatively minor effects relative to virological tests. Simply put, with fewer than 10 percent of people ever becoming infected, there is little scope to improve output losses through identifying these individuals.

We provide a range of robustness analysis. In particular we consider a case where serological testing is necessary before release: individuals cannot reliably convey that they had symptoms or provide past virological test results. Even in this setting, serological tests have small effects. We also show that when transmission rates and mortality rates are recalibrated to match the same reproduction number and infection fatality rate, our results are robust.

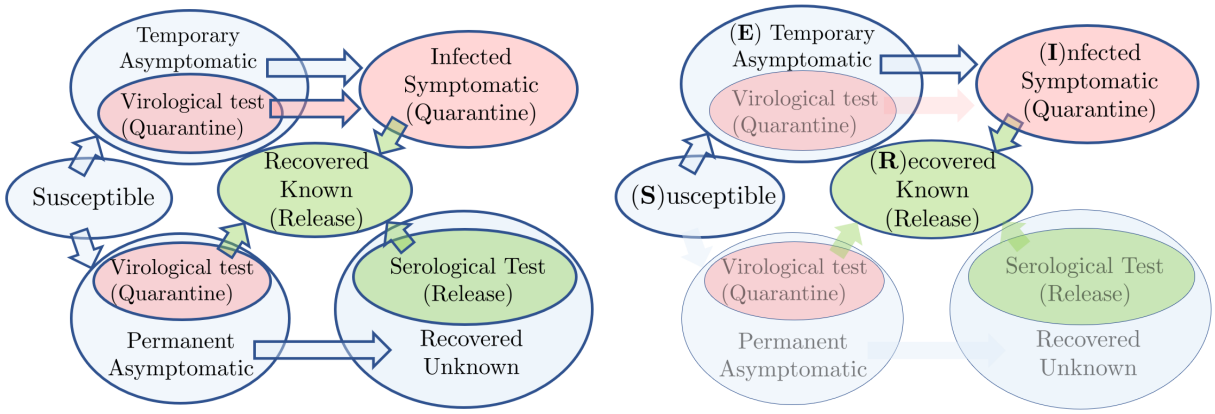

Figure 1: Model schematic (left), and the nested SEIR model (right)

Notes: Blue states are *unknown* to the planner and individual. Red states are *known positive* and green states are *known negative*. Virological (serological) testing takes *unknown* individuals and transits them to the *known positive* (known negative) states. On the right, testing and permanent asymptomatic infection are shut-down, yielding the standard SEIR model.

In implementing a virological testing regime, a government may face trade-offs between test quality and quantity. Our final contribution is to demonstrate that non-linearities in the model imply that test frequency is more important than test quality. We consider two tests: (i) an at-home test that costs \$10 with a 50 percent false negative rate, and (ii) an accurate lab test (Polymerase Chain Reaction or 'PCR' tests) that costs \$40 with a zero false negative rate. For a fixed testing budget, at home testing can be done more frequently, with lower quality. Despite significantly lower accuracy, more frequent at-home tests dominate more accurate and expensive PCR tests. At a budget of \$40 per person per month, at-home tests save 40,000 lives relative to the PCR tests.

Our exercises qualitatively and quantitatively make an important point for economists integrating epidemiological and macroeconomic models. Analysis of an economic model with an SEIR epidemiological block but without public health interventions such as testing and targeted quarantine, will *unavoidably* feature a trade-off between mortality and economic activity. Faster reopening increases output but increases mortality. Kaplan, Moll, and Violante (2020) term this trade-off a *'pandemic possibility frontier'*. Combining widely advocated public health measures of testing and targeted quarantine, the model delivers lower mortality and higher economic activity, shifting this frontier outwards. Theories of economic-activity vs. mortality trade-offs with only a lock-down policy available to the policy-maker are necessarily discussions of second best policies.

This paper has eight sections. Section 1 reviews the related SEIR literature and recent papers using this model to quantify the effects of the corona virus pandemic. Section 2 reviews data on infection, mortality, testing and quarantine measures. Section 3 describes the model. Section 4 details calibration

and model fit. Section 5 provides our main counterfactuals which compute output savings delivered from a combination of elevated testing and targeted reopening. Section 6 considers the quality-frequency trade-off. Section 7 shows how our results are robust to many assumptions regarding the virus. Section 8 concludes. An appendix contains details on additional robustness exercises, figures and tables.

1 Literature review

Brauer and Castillo-Chavez (2012) provide a summary of recent SEIR models. SEIR stands for Susceptible, Exposed (people not yet infectious in the standard model), Infectious, and Removed (quarantined or immune). In particular, they discuss frameworks of quarantine (setting aside individuals who are exposed) and isolation (setting aside individuals who are infectious, often called hospitalization).

A recent policy paper by Imperial College COVID-19 Response Team (2020) incorporates several policy parameters into an SEIR model that is enriched to accommodate geographical transmission and age dependency of transmission and mortality rates. In particular, they consider a model with quarantine, asymptomatic patients, and testing of *hospitalized* patients, with policy thresholds that depend on positive test rates. Their predictions have been reported widely in the press, and the model is being updated in real-time as this article is written. To the best of our knowledge, our contribution is to model (i) the matching process between different subgroups, thus endogenizing R_0, and (ii) highlighting the importance of *testing asymptomatic patients* and, (iii) quarantine policies that are contingent on the testing outcomes. Lastly, we use our measure of the fraction of individuals quarantined as a measure of loss of economic activity. This allows us to evaluate the role of widespread testing which, as a policy, may allow for similar mortality rates but lower quarantine rates.

Recent examples of testing and diagnosis in an SEIR model include Chowell, Fenimore, Castillo-Garsow, and Castillo-Chavez (2003) who model the Severe acute respiratory syndrome (SARS) epidemic in 2002. The purpose of testing and diagnosis in Chowell, Fenimore, Castillo-Garsow, and Castillo-Chavez (2003) is an improvement in healthcare, which reduces the time to recovery by one half.[3] In our model, the role for testing and diagnosis is being able to efficiently target quarantine measures.

Recent examples of quarantine in an SEIR model include Feng (2007) who derive closed form expressions for the maximum and final rates of infection. Feng (2007) has two notions of quarantine: one in which exposed individuals are quarantined (setting aside individuals who are exposed) and isolation

[3]They report a SARs incubation period of 2 to 7 days, with most infected individuals either recovering after 7 to 10 days, or dying. The SARS mortality rate is 4 percent or more. They estimate a basic reproductive number $R_0 = 1.2$. They model a diagnosis rate and diagnosed state. Individuals recover at a fast rate if diagnosed (8 days without diagnosis, 5 days with diagnosis).

(setting aside individuals who are infectious, often called hospitalization). In our model, quarantine is similarly case dependent, but can only depend on observed health status.

Empirically, the literature has begun to document the rate of transmission and incubation periods. Wu, Leung, and Leung (2020) compile a summary of R_0 across various viruses (SARS-CoV, MERS-CoV, Commonly circulating human CoVs (229E, NL63, OC43, HKU1)), and estimate an SEIR model with international travel. Using data from the early days of the outbreak in Wuhan, they report an R_0 of 2.68 and an incubation period of 6.1 days. The World Health Organization (2020) report that the time from symptom recovery to detection fell from 12 days in early January to 3 days in early February 2020. After symptom onset, it typically takes 2 weeks for a mild case to recover, or 3 to 6 weeks for severe cases.

The applied literature has also begun to document the role of quarantine in reducing transmission, and the rate of asymptomatic transmission. Kucharski, Russell, Diamond, Liu, Edmunds, Funk, Eggo, Sun, Jit, Munday, et al. (2020) estimate that in China, the basic reproductive rate R_0 fell from 2.35 one week before travel restrictions on Jan 23, 2020, to 1.05 one week after travel restrictions. They use an estimated SEIR model to forecast the epidemic in China, extending the model to explicitly account for infections arriving and departing via flights. Using data from Wuhan, Wang, Liu, Hao, Guo, Wang, Huang, He, Yu, Lin, Pan, et al. (2020) report a baseline reproductive rate of 3.86, that fell to 0.32 after the vast lock-down intervention. They also find a high rate of asymptomatic transmission, leading us to consider the asymptomatic state to be infectious as opposed to the baseline SEIR model which assumes that the 'exposed' state is non-infectious.[4] A 50% rate of asymptomatic infections has been identified in Iceland, one of the few countries to adopt random testing of asymptomatic individuals.[5] Meta-analysis of both published and unpublished papers in Byambasuren, Cardona, Bell, Clark, McLaws, and Glasziou (2020) includes a wide range of asymptomatic cases, ranging from 6% to 41%.

In the recent economics literature Atkeson (2020b) and Fernández-Villaverde and Jones (2020) provide a review of the SIR model. Fenichel (2013) compares social planner and decentralized solutions for lock-down in an SIR model where all individuals recover. Alvarez, Argente, and Lippi (2020), and Farboodi, Jarosch, and Shimer (2020) study optimal lock-down policy in SIR models with the possibility of death. The latter compares the solution of the planner's problem to a decentralized equilibrium in which individuals choose their level of engagement with the economy understanding the health risks they face. Eichenbaum, Rebelo, and Trabandt (2020), Jones, Philippon, and Venkateswaran (2020) and Glover, Heathcote, Krueger, and Ríos-Rull (2020) nest a similar SIR model in quantitative general equilib-

[4] https://www.medrxiv.org/content/10.1101/2020.03.03.20030593v1

[5] https://www.buzzfeed.com/albertonardelli/coronavirus-testing-iceland. "Early results from deCode Genetics indicate that a low proportion of the general population has contracted the virus and that about half of those who tested positive are non-symptomatic".

rium macroeconomic models of consumption, savings and labor supply. The latter includes heterogeneity by age, income and assets with age-varying transmission and mortality risk. These papers consider lock-down as the only available tool to the policy-marker. Our contribution is to enrich the underlying SEIR model by introducing scope for testing policies which may mitigate the output costs of quarantine policies while not exacerbating the decline in output. It would be relatively straight-forward to integrate the information structure of our model into these models in order to evaluate the economic benefits of broad based testing.

Several contemporaneous papers consider testing in an SEIR model. Recent work by Piguillem and Shi (2020) , Hornstein (2020), Acemoglu, Chernozhukov, Werning, and Whinston (2020) and Brotherhood, Kircher, Santos, and Tertilt (2020) study testing regimes in combination with targeted quarantine. Hornstein (2020) incorporates tracing, while Acemoglu, Chernozhukov, Werning, and Whinston (2020) consider optimal age-dependent lockdown policies under different virological testing regimes. Similarly, Brotherhood, Kircher, Santos, and Tertilt (2020) study various combinations of virological testing and age-dependent quarantine policies in a model with optimizing agents. We contribute to the literature by studying the output-death tradeoff and how it is altered by targeted quarantine, virological testing, and serological testing in a model with permanently asymptomatic individuals.

2 Cases, deaths, quarantine and testing through June 15, 2020

This section provides a short overview of the evolution of the COVID-19 pandemic in the United States through June 15, 2020.

Cases. The first case was reported in the U.S. on January 22, 2020. Panels A through D of Figure 2 plot the evolution of confirmed cases resulting from COVID-19. Despite 30% growth rates of cases throughout March, 2020, the growth rate of cases declined up until June 15, 2020. Due to the general lack of testing (in particular of the asymptomatic) in the U.S., cases are a noisy measure of the spread of COVID-19 (e.g. see Hortaçsu, Liu, and Schwieg (2020) and Stock (2020)). Therefore, we focus on mortality data when calibrating and evaluating our model framework.

Deaths. Panels A through D of Figure 2 also plot the number of deaths attributed to COVID-19. Similar to case counts, the growth rate of deaths declined up until June 15, 2020.

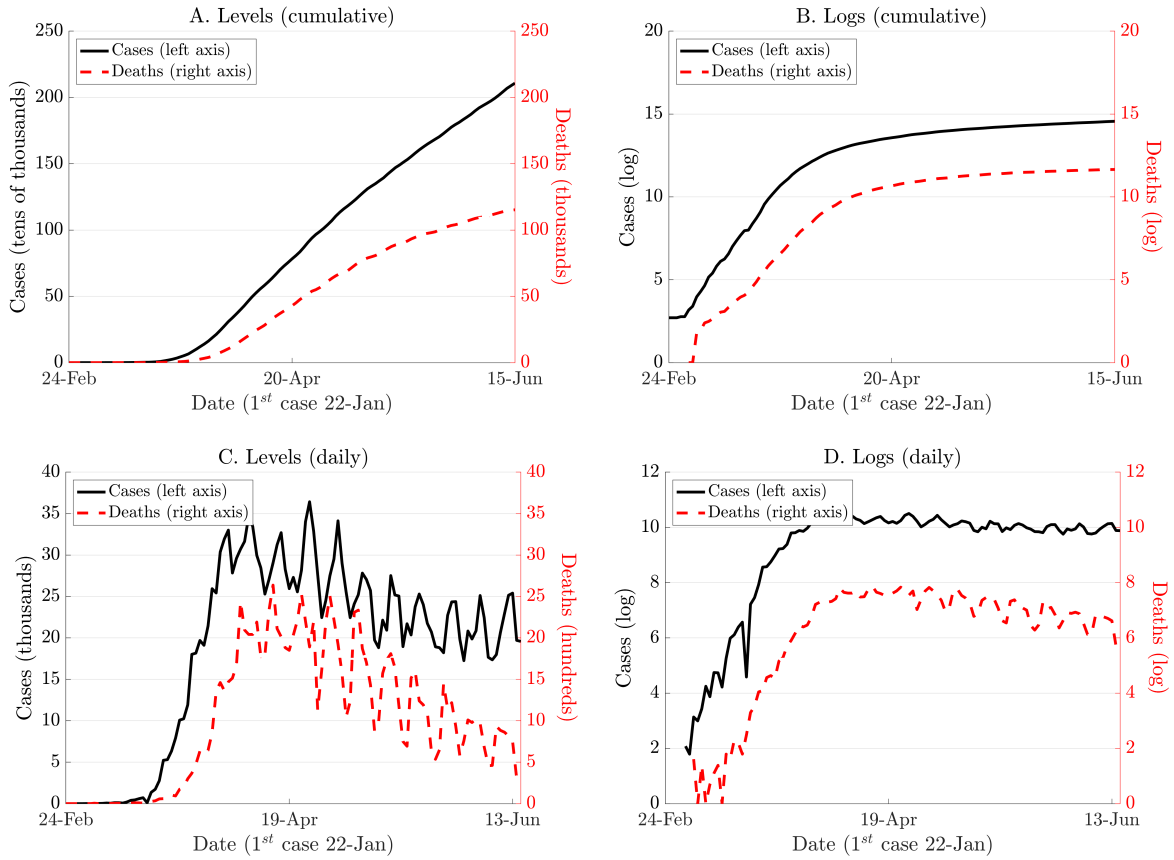

Figure 2: US cumulative cases and deaths

Notes: Source: John Hopkins CSSE, https://github.com/CSSEGISandData/COVID-19. Data reflect non-repatriated cases, and so exclude the cases from the Diamond Princess and Grand Princess cruise ships.

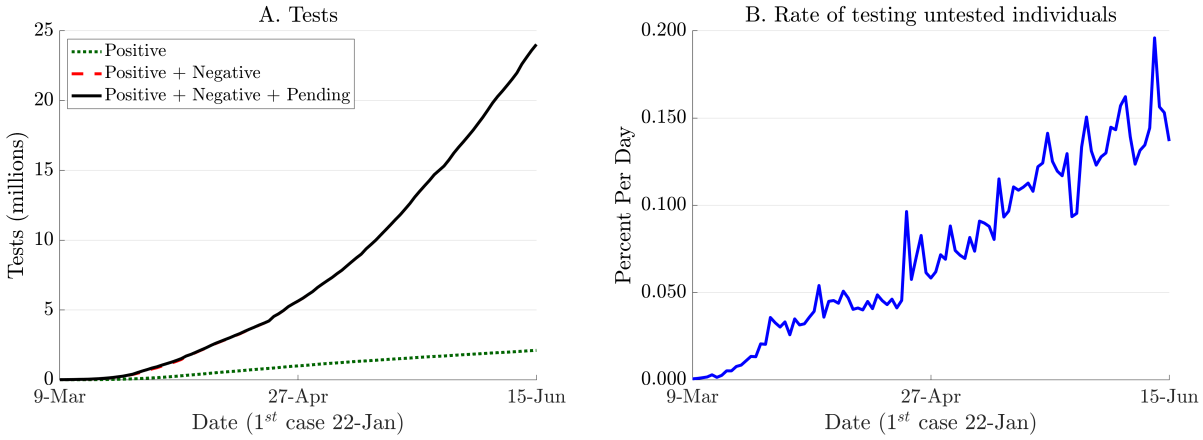

Figure 3: US testing

Notes: Source: John Hopkins CSSE, https://github.com/CSSEGISandData/COVID-19. Panel B plots the fraction of the untested population that is tested each day. Let T_t be total cumulative tests—the black line in Panel A—, then Panel B plots $(T_t - T_{t-1})/(340m - T_t)$.

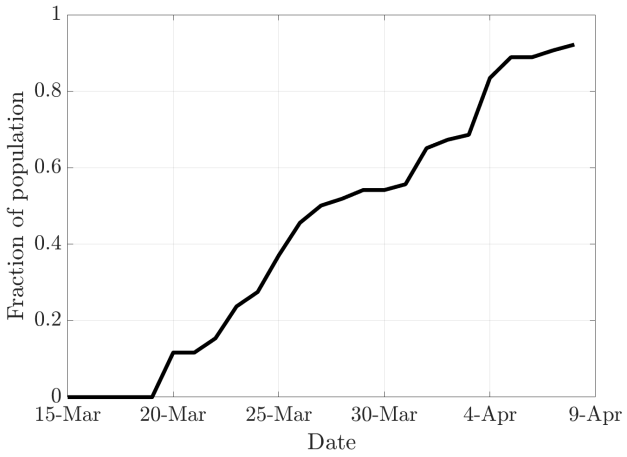

Figure 4: US population living in states with stay-at-home orders

Notes: Based on reporting by the New York Times: https://www.nytimes.com/interactive/2020/us/coronavirus-stay-at-home-order.html.

Testing. Figure 3 reports cumulative tests and the testing rate per day. At its peak to date, the US tested just short of 0.2 percent of its untested population in a single day. If testing continued at this level and were at random, each individual would be tested around once every 16 months. We consider testing levels between weekly and every two months.

Quarantine. Figure 4 reports the fraction of individuals residing in states with stay-at-home orders in the United States. We refer to this as the *quarantine rate*. There are two important aspects of the quarantine rate. First, not all individuals in states with quarantine and shelter-in-place orders complied. Essential workers did not quarantine, and so when we calibrate our framework, we will take this into account. Second, we model quarantine as smoothly increasing even though there are large discrete jumps in the quarantine rate when California, New York, and Illinois announced state-wide shelter in place orders.[6]

3 Model

Throughout this section Figure 1 and Figure 5 may be useful to the reader. Figure 1 is a non-technical schematic of our model (left), and illustrates how the model nests the standard SEIR model (right). Figure 5 provides technical detail underlying the model, including notation and information states.

[6]We will refer to New York's policy as shelter-in-place, despite alternative language used by the government of New York.

3.1 Overview

There are five health states in our model (susceptible, temporary asymptomatic, permanent asymptomatic, infected symptomatic, and recovered), three information states (unknown, known positive, and known negative), and two quarantine states (quarantine, non-quarantine).

Health states. We base this discussion on Figure 1, and we explain how our health states nest the standard SEIR model.

i. Susceptible (S) - These are individuals that have not been exposed to the virus. This corresponds to **S** in the SEIR model.

ii. Temporary Asymptomatic (TA) - Individuals that have contracted COVID-19 and are temporarily asymptomatic. These individuals will eventually develop symptoms. This corresponds to **E** in the SEIR model: *Exposed*. Relative to the SEIR model we allow that these individuals may also transmit the virus albeit at a (potentially) lower rate.

iii. Permanent Asymptomatic (PA) - Individuals that have contracted COVID-19 and will never develop symptoms. These individuals transmit the disease. There is no corresponding state in the SEIR model.

iv. Infected Symptomatic (I) - Individuals that have contracted COVID-19 and are now showing symptoms. These symptoms are such that the individual knows that they have the disease and can respond to this by self-quarantining.[7] This corresponds to **I** in the SEIR model: *Infectious*. These individuals transmit the disease, potentially at a higher rate than the asymptomatic.

v. Recovered (R) - All individuals who previously contracted COVID-19 and have entered the recovery phase. As in the textbook SEIR model we assume these individuals are immune and do not transmit the disease.[8] This corresponds to **R** in the SEIR model: *Recovered*.

Figure 1 (left) tracks an individual case through these states. In terms of *medical transmission*, we assume that temporary asymptomatic, permanent asymptomatic, and infected symptomatic individuals are contagious, although with potentially different rates of transmission.

[7] This appears to attribute a certain amount of altruism to the infected. Quarantine will be imperfect which can be viewed as standing in for less than perfectly altruistic behavior. Regardless, we think such altruism is not a poor assumption. Assuming that infected people instead have nothing left to lose can lead to perverse results such as testing leading to *higher infections*. We don't think this is reasonable.

[8] Ota (2020) tested repeated infection of SARS COV-2 in four Rhesus monkeys and found immunity to reinfection in each case. Existing human studies are inconclusive and suggest that reinfection may occur, e.g. Wen, Su, Tang, Le, Zhang, Zheng, Liu, Xie, Li, Ye, et al. (2020).

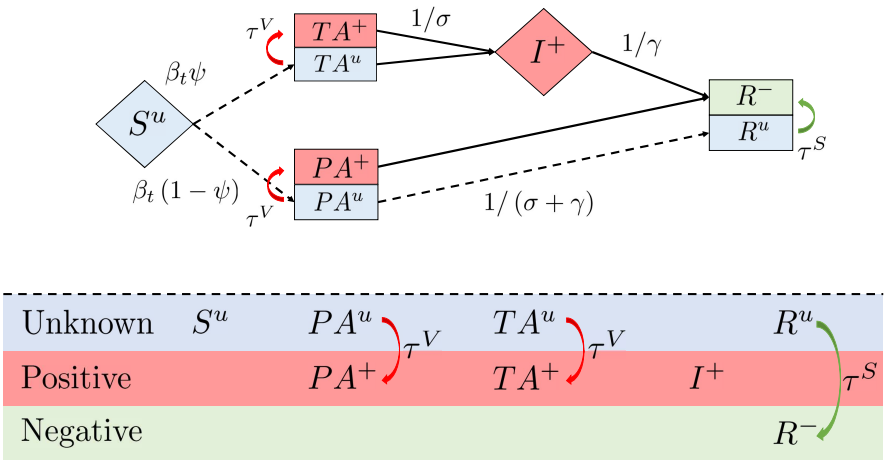

Figure 5: SEIR model with incomplete information and testing of asymptomatic individuals

Figure 1 (right) illustrates how our model nests the SEIR model. The different rates of transmission nest the case that only infected symptomatic (I) individuals can transmit the disease, which is the case in the SEIR model. Recovered (R) and susceptible (S) individuals cannot transmit the disease.

The medical block of the model is very simple and could be enriched in many ways.[9] We assume that susceptible individuals are infected at time varying endogenous rate β_t. A fraction ψ of newly infected individuals will eventually develop symptoms and are thus temporarily asymptomatic. The remaining $1-\psi$ of new infections never develop symptoms and are permanently asymptomatic. Asymptomatic individuals have no mortality risk. At rate $1/\sigma$, the temporary asymptomatic develop symptoms and transition into the infected symptomatic state from which they recover at rate $1/\gamma$ and die at a rate ω_D. Permanently asymptomatic cases recover at rate $1/(\gamma+\sigma)$ so have the same expected duration of infection as those on the temporarily asymptomatic branch.

Information. Information is incomplete, with the environment described in Figure 5. There are three information sets (1) unknown (u), (2) known positive $(+)$, and (3) known negative $(-)$, denoted by super-scripts. Absent testing, the unknown set consists of the susceptible (S^u), both types of asymptomatic (TA^u, PA^u), and those that have recovered from permanently asymptomatic infection (R^u). We are assuming that the development of symptoms is sufficient to infer a known negative recovery (R^-). The set of known positive cases are the identified asymptomatic cases (TA^+, PA^+), and the symptomatic state (I^+). When assessing quarantine and testing policy, we assume that the planner cannot distinguish health-states within information sets.

[9] See http://gabgoh.github.io/COVID/index.html by Gabriel Goh for an example of an SEIR model of *Transmission Dynamics* that appends a rich model of *Clinical Dynamics* which models hospitalization, length of hospital stay, and more. These states would intercede between IS, RA and D, which is not the focus of this paper.

Meeting and transmission rates. The underlying parameters consist of an explicit interaction of *social meeting rates*, which are mutable to quarantine / social distancing policies, and *medical transmission rates*, which determine the probability of transmission between two individuals that meet. We assume a linear matching technology, which we think is important for an assessment of serological testing, since it implies that recovered individuals mixing in the population lower infection rates.

We denote quarantine and non-quarantine states by Q and NQ, respectively. Interacted with our five health states and three information sets, this gives 30 potential states that individuals can be in. We make several assumptions, which keep the state space tractable:

1. **Full quarantine of known positive cases:** Known positive cases (temporary asymptomatic, permanent asymptomatic, symptomatic) are immediately quarantined. Thus there is only one state each for known temporary asymptomatic TA_Q^+, known permanent asymptomatic PA_Q^+ and known symptomatic I_Q^+.

2. **Full release of known immune cases:** Since they are immune, known negative (recovered) individuals are de-quarantined immediately. Thus there is only one known recovered state R_{NQ}^-

Susceptible individuals that test negative to a virological test are only negative that instant, so are not treated differently to untested cases.

The *meeting rates* of non-quarantined individuals is given by $\lambda \in (0,1)$. Quarantine is imperfect, such that the meeting rate for quarantined individuals is $\lambda^Q \in (0,\lambda)$. We interpret the ratio factor by which quarantine reduces the rate of social interaction (λ^Q/λ) as the *quarantine technology* and treat it as a parameter to be calibrated.

We denote the *transmission rates* by $\rho^A\left(\rho^I\right)$ for asymptomatic (infected symptomatic) cases to accommodate the possibility that transmission rates are higher in symptomatic cases; a key robustness exercise. These give the probability that, conditional on meeting an asymptomatic or symptomatic individual, a non-infected individual contracts COVID-19. Crucially, individuals do not know who is infected, and do not know that they have met an infected person.

Testing. The presence of temporary and permanent asymptotic individuals introduces a role for both virological and serological testing. Virological tests identify active cases of the virus. Serological tests identify antibodies and thus past cases of the virus. Our information structure has assumed that when symptoms present, the individual and society know that the individual is infected. In this paper we do not cover testing of symptomatic individuals.[10]

[10]In our model if we were to test symptomatic individuals then all tests would yield positives. In the data a small fraction of tests yield positives. In the US our interpretation of this is not that the US is testing asymptomatic people, but rather that

Virological tests allow the planner to identify and quarantine asymptomatic spreaders. We assume that virological testing of unknown cases takes place at a rate τ^V. Known positives are immediately quarantined. In our benchmark environment, virological tests are 100 percent accurate and do not produce false negatives or false positives.For a given testing rate τ^V, it is straight-forward to model false negatives. Since symptomatic individuals are known, a false negative is only relevant when a test is applied to an asymptomatic individual. A false negative test of an asymptomatic individual results in misclassification of the individual as unknown. This is isomorphic to simply reducing the testing rate by a pre-determined factor. In section 5 we consider false negative rates of up to 50%. False positives are rare and unlikely to pose a threat to public health policy since they simply result in overly strict quarantine, and so we preclude the occurrence of false positives.[11]

The only known negatives we allow for are known recovered individuals. Thus, when an individual is tested virologically and the test yields a negative results, we treat the individual as 'unknown.' This is to capture the notion that the test can only reveal their health state at a given point in time. Therefore, all non-infected individuals are considered unknown, regardless of whether they have just been tested or not.[12]

Serological tests allow the planner to identify and release known negative cases. We assume that serological testing of unknown cases takes place at a rate τ^S. In our benchmark environment, we also assume serological tests are 100 percent accurate and do not produce false negatives or false positives.

Targeted quarantine. We allow for a quarantine policy that is allowed to be targeted toward different information sets. To keep the Markovian structure of the SEIR model, we quarantine individuals at constant rates. We assume in the benchmark a *targeted quarantine* program through which known positives self-quarantine immediately and for the duration of their infection. We also assume a *targeted release* program through which known negative cases are released immediately. The only policy parameters are then the rate at which unknown cases are put into quarantine (ξ^u), and the rate at which they are released (r^u), which we refer to as the *reopening rate*.

individuals with similar symptoms due to common colds and the flu are being tested, as well as individuals testing out of precaution. To introduce testing of symptomatic individuals one would want to extend the model to introduce an additional disease that presents observationally identical symptoms that can then be separated by testing.

[11]Specificity is inversely proportional to false positives (specificity=true negative/(true negative+false positive)). Sensitivity is inversely proportional to false negatives, (sensitivity = true positive/(true positive+ false negative)). In lab settings, PCR tests produce 100% sensitivity and 96% specificity, e.g. https://spectrum.ieee.org/the-human-os/biomedical/diagnostics/testing-tests-which-covid19-tests-are-most-accurate . Although in the field, the specificity and sensitivity are reported to be lower, e.g. https://theconversation.com/coronavirus-tests-are-pretty-accurate-but-far-from-perfect-136671.

[12]In the working paper version of this paper, we consider the opposite extreme in which testing programs reveal subsequent health transitions.

3.2 Transmission

Given the above description of the model, we now describe transition rates of individuals between states. We work in continuous time.[13] There is initially a unit mass of individuals. An initial mass of infected individuals I_0 is distributed across TA^u and PA^u in proportions ψ and $(1-\psi)$, respectively. State variables sub-scripted by t denote masses, e.g. $S^u_{t,NQ}$ is the mass of individuals at date t who are susceptible, unknown, and non-quarantined. We denote aggregated masses of individuals in a state X in period t as M^X_t, e.g. M^Q_t is the mass of individuals in quarantine.

Social interaction. In order to transmit the disease, individuals must first meet. We assume random matching governed by a linear matching technology. Non-quarantined individuals meet other individuals at rate λ, while quarantined individuals meet others at rate λ^Q.

The conditional probabilities of meetings are as follows. The mass of individuals that are out in the world to bump into is given by M_t, which depends on the mass of individuals that are non-quarantined (M^{NQ}_t) and quarantined (M^Q_t): $M_t = \lambda M^{NQ}_t + \lambda^Q M^Q_t$. The masses of non-quarantined and quarantined individuals are given by,

$$\begin{aligned} M^{NQ}_t &= S^u_{t,NQ} + TA^u_{t,NQ} + PA^u_{t,NQ} + R^u_{t,NQ} + R^-_{t,NQ} \quad , \\ M^Q_t &= S^u_{t,Q} + TA^u_{t,Q} + PA^u_{t,Q} + TA^+_{t,Q} + PA^+_{t,Q} + R^u_{t,Q} + I^+_{t,Q}. \end{aligned}$$

Of these, the masses of non-quarantined and quarantined individuals who can transmit the disease are

$$M^{I,NQ}_t = TA^u_{t,NQ} + PA^u_{t,NQ} \quad , \quad M^{I,Q}_t = TA^u_{t,Q} + PA^u_{t,Q} + TA^+_{t,Q} + PA^+_{t,Q} + I^+_{t,Q}.$$

Conditional on meeting someone, the probability the person is infected is

$$\pi_t = \frac{\lambda M^{I,NQ}_t + \lambda^Q M^{I,Q}_t}{M_t}.$$

The conditional probabilities that the meeting is with an asymptomatic (π^A_t) or symptomatic (π^I_t) individual are

$$\pi^A_t = \frac{\lambda(TA^u_{t,NQ} + PA^u_{t,NQ}) + \lambda^Q(TA^u_{t,Q} + PA^u_{t,Q} + TA^+_{t,Q} + PA^+_{t,Q})}{\lambda M^{I,NQ}_t + \lambda^Q M^{I,Q}_t} \quad , \quad \pi^I_t = \frac{\lambda^Q I^+_{t,Q}}{\lambda M^{I,NQ}_t + \lambda^Q M^{I,Q}_t}.$$

Infection. A meeting with an asymptomatic (symptomatic) infected individual has a ρ^A (ρ^S) probability of virus transmission. Once infected, an individual does not know that they are infected as they are

[13] When simulating the model we implement a discrete time approximation in which a period is one hour and days are 14 hours long.

initially asymptomatic. Independent of who they meet, if they contract COVID-19, nature exogenously determines if they will eventually develop symptoms. A newly infected individual becomes temporarily asymptomatic with probability ψ and permanently asymptomatic with probability $1-\psi$. Those who are temporarily asymptomatic develop symptoms with probability $1/\sigma$. Once they develop symptoms they recover at rate $1/\gamma$ or die at rate ω^D.

Transmission rate. Combining the above, the rate of infection of a quarantined (non-quarantined) person is given by $\lambda^Q\alpha_t$, $(\lambda\alpha_t)$, where α_t is the probability of infection conditional on a random meeting:

$$\alpha_t = \pi_t \left[\pi_t^I \rho^I + \pi_t^A \rho^A\right].$$

We then adjust this transmission rate by the quarantine effectiveness to obtain the rate of new infection among non-quarantined β_t^{NQ} and quarantined β_t^Q individuals:

$$\beta_t^{NQ} \equiv \lambda\alpha_t \qquad , \qquad \beta_t^Q \equiv \lambda^Q\alpha_t.$$

3.3 Transition rates

We describe the full set of transition rates between all 13 states in Table 1. Along with initial conditions for the distribution of individuals across health and information states, these transition rates are sufficient to simulate the model. We provide examples of three states: S_{NQ}^u, TA_{NQ}^u and R_Q^u.

Susceptible, unknown. All non-infected individuals are unknown, regardless of whether they have been tested. Three events can affect an unknown susceptible individual. At rate β_t^{NQ} the individual is infected with COVID-19: a product of their meeting rate λ^{NQ}, the probability of a meeting π_t, and the conditional probability of infection $\pi_t^I\rho^I + \pi_t^A\rho^A$. Conditional on infection the individual becomes TA^u with probability ψ, and PA^u with probability $(1-\psi)$. At rate ξ^u the individual is put into quarantine and transitions to S_{NQ}^u. Note that at rate τ^V (τ^S) the individual receives a virological test which reveal that the individual is not (has not been) infected, and so they remain in S_{NQ}^u.

Temporary asymptomatic, unknown. Like the susceptible type the individual goes into quarantine at rate ξ^u. Now following a virological test, which occurs at rate τ^V, the individual tests positive and is quarantined TA_Q^+. From TA_Q^+ they transition to the infected state, still under quarantine, and then become a known recovery and so are released. At rate $1/\sigma$, the individual develops symptoms before being tested, and transitions to I_Q^+.

Recovered unknown, quarantine. Due to porous quarantine, early cases and the permanently asymptomatic route, some individuals will contract Covid-19 without knowing it and be recovered while still

A. Initial state	B. Next instant state												
	Susceptible		Temporary Asymptomatic			Infected Symptomatic	Permanent Asymptomatic			Recovered			Dead
	S^u_{NQ}	S^u_Q	TA^u_{NQ}	TA^u_Q	TA^+_Q	I^+_Q	PA^u_{NQ}	PA^u_Q	PA^+_Q	R^u_{NQ}	R^u_Q	R^-_{NQ}	D
S^u_{NQ}		$[\xi^u]$	$\beta_t^{NQ}\psi$				$\beta_t^{NQ}(1-\psi)$					ν_t	
S^u_Q	(r^u)			$\beta_t^{Q}\psi$				$\beta_t^{Q}(1-\psi)$				ν_t	
TA^u_{NQ}				$[\xi^u]$	τ^V	$1/\sigma$			ν_t				
TA^u_Q			(r^u)		τ^V	$1/\sigma$			ν_t				
TA^+_Q						$1/\sigma$							
I^+_Q												$1/\gamma$	ω^D
PA^u_{NQ}								$[\xi^u]$	$\tau^V+\nu_t$	$\frac{1}{\sigma+\gamma}$			
PA^u_Q							(r^u)		$\tau^V+\nu_t$		$\frac{1}{\sigma+\gamma}$		
PA^+_Q												$\frac{1}{\sigma+\gamma}$	
R^u_{NQ}											$[\xi^u]$	$\tau^S+\nu_t$	
R^u_Q										(r^u)		$\tau^S+\nu_t$	
R^-_{NQ}													

Table 1: Poisson transition rates

Notes: In any instant only one transition can occur. The red $[\xi^u]$ terms are rates *into* quarantine. The green (r^u) terms are rates *out of* quarantine, which represent reopening policy and are varied in our counterfactuals.

being quarantined. While a virological test at rate τ^V reveals nothing, at rate τ^S these individuals receive a serological test which reveals antibodies. At this point the individual moves into the absorbing state R^- which is in the known negative information set and so is released from quarantine.

Nesting the SEIR and SIR models. Formally, the SEIR model is nested under the following parameter restrictions: (i) no quarantine ($\lambda/\lambda^Q = 1$, $\xi^u = 0$, $r^u = \infty$), (ii) no asymptomatic transmission ($\rho_A = 0$), (iii) no permanent asymptomatic infection ($\psi = 1$), (iv) no testing ($\tau^V = \tau^S = 0$). In this case, individuals move from $S^u_{NQ} \to TA^u_{NQ} \to I^+_Q \to R^-_{NQ}$, which substantively correspond to the $SEIR$ states.

3.4 Vaccine and anti-viral

We allow for the arrival of a vaccine/anti-viral at some future date. We assume the vaccine/anti-viral is distributed at rate ν. In the exercises that follow, we model vaccine such that $\nu_t = 0$ up until some date τ, then $\nu_t = \nu$ for $t > \tau$. For the susceptible, treatment delivers a transition to known recovered and non-quarantined. For known and unknown, temporary and permanent asymptomatic cases, treatment delivers a transition to known permanent asymptomatic.

3.5 Measurement

3.5.1 Basic reproduction number

We consider two measures of the basic reproduction numbers summarized by Cori, Ferguson, Fraser, and Cauchemez (2013). One is based on a hypothetical date 0 case, *ceteris paribus*, in particular, holding policy and contact rates fixed: *Cori method*. The other is based on the realized path of infections generated along the transition path: *Wallinga–Teunis method* (Wallinga and Teunis, 2004). The difference parallels a comparison of *predicted* (Cori) and *realized* (Wallinga–Teunis) life expectancy in demographics for any given cohort.

Cori method. Consider a hypothetical 'date-zero' case of an individual that has just contracted COVID-19, and nature is yet to determine if they are temporarily or permanently asymptomatic. A summary statistic of the disease is the expected number of infections caused by this single infected person: R_0. We can write R_0 recursively as follows.

Consider an initial temporary asymptomatic case, before any quarantine measures go into effect. With probability λ they meet another individual and with probability ρ^A they transmit the disease. With probability σ^{-1} the individual develops symptoms. Once the individual has developed symptoms, they meet others at rate λ and transit the disease at rate ρ^S. The resulting recursive equations for the number of infections generated by this date-zero case are:

$$\begin{aligned} R_0^{A,Temp,NQ} &= \lambda\rho^A + \left(1-\sigma^{-1}\right) R_0^{A,Temp,NQ} + \sigma^{-1} R_0^{I,NQ}, \\ R_0^{I,NQ} &= \lambda\rho^S + \left(1-\gamma^{-1}-\omega^D\right) R_0^{I,NQ}. \end{aligned}$$

We can solve this system of equations for the expected number of cases generated by the initial temporarily asymptomatic case:

$$R_0^{A,Temp,NQ} = \left[\sigma\lambda\rho^S \left[\frac{\rho^A}{\rho^S} + \frac{\sigma^{-1}}{\gamma^{-1}+\omega^D}\right]\right] \tag{1}$$

Now consider an initial permanent asymptomatic individual. With probability $\lambda\rho^A$, the individual meets someone and transmits the disease. With probability $\left(1-(\sigma+\gamma)^{-1}\right)$, they do not recover. We can express the number of infections generated by this date zero case recursively and solved for:

$$R_0^{A,Perm,NQ} = \lambda\rho^A + \left(1-(\sigma+\gamma)^{-1}\right) R_0^{A,Perm,NQ} = (\sigma+\gamma)\,\lambda\rho^A \tag{2}$$

Since nature ultimately determines if the initial infection is temporarily or permanent asymptomatic, the

overall Cori-method basic reproductive number R_0^C is given by:

$$R_0^C = \psi R_0^{A,Temp,NQ} + (1-\psi) R_0^{A,Perm,NQ}$$

Combining (1) and (2) delivers the number of infections generated by a date-zero case of COVID-19:

$$R_0^C = \lambda \rho^S \left\{ \psi \left[\sigma \left(\frac{\rho^A}{\rho^S} + \frac{\sigma^{-1}}{\gamma^{-1} + \omega^D} \right) \right] + (1-\psi) \left[(\sigma + \gamma) \frac{\rho^A}{\rho^S} \right] \right\} \tag{3}$$

Wallinga-Teunis method. By contrast, the Wallinga-Teunis method is *forward-looking* and takes into account the full dynamics of disease transmission. Since quarantine measures in the US started in mid-March, around the same time that case and death measurements began, this is the appropriate measure for comparing to data on Covid case transmission. Since we will ultimately solve the model using a discrete time approximation, we express the Wallinga-Teunis method using discrete time. Denote the discrete-time approximation of the transition matrix (Table 1) across the 13 health and information states $\mathbf{P}_t$.[14] Let $\boldsymbol{\rho}_t$ be a 13×1 vector of the expected *contemporaneous* date t infections due to an individual in each state, and let $\boldsymbol{r}_t$ be the *cumulative* number of these infections. After some date T, once the vaccine has completely rolled out, a new infection would generate zero cases: $\boldsymbol{\rho}_T = \boldsymbol{r}_T = \mathbf{0}$. Working backwards

$$\boldsymbol{r}_t = \boldsymbol{\rho}_t + \mathbf{P}_t \boldsymbol{r}_{t+1} \quad , \quad t \in \{1, \ldots, T-1\}. \tag{4}$$

Using the same ordering of states as in Table 1, the expected number of infections due to individuals in each state is[15]

$$\boldsymbol{\rho}_t = \Big[0, 0, \underbrace{\rho^A \lambda}_{\langle TA^u_{NQ} \rangle}, \underbrace{\rho^A \lambda^Q}_{\langle TA^u_Q \rangle}, \underbrace{\rho^A \lambda^Q}_{TA^+_Q}, \underbrace{\rho^S \lambda^Q}_{I^+_Q}, \underbrace{\rho^A \lambda}_{\langle PA^u_{NQ} \rangle}, \underbrace{\rho^A \lambda^Q}_{\langle PA^u_Q \rangle}, \underbrace{\rho^A \lambda^Q}_{PA^+_Q}, 0, 0, 0, 0\Big]' \times \left(\frac{\lambda S^u_{t,NQ} + \lambda^Q S^u_{t,Q}}{M_t} \right) \tag{5}$$

where the final term is the conditional probability that a meeting is with a susceptible individual.

The desired measure is the expected value for an individual infected at date t. An infected individual enters one of the four states in angle brackets (5). Let f_t denote the conditional probability that the susceptible individual that was infected was in quarantine. Then we obtain the scalar Wallinga-Teunis measure by weighting $\boldsymbol{r}_t$ by the probabilities attached to initial infection states:

$$R_t^{WT} = \underbrace{\Big[0, 0, (1-f_t)\,\psi, f_t \psi, 0, 0, (1-f_t)\,(1-\psi), f_t(1-\psi), 0, 0, 0, 0, 0\Big]}_{\boldsymbol{\pi}_t} \times \boldsymbol{r}_t \quad , \quad f_t = \frac{\lambda^Q S^u_{t,Q}}{\lambda S^u_{t,NQ} + \lambda^Q S^u_{t,Q}}$$

The key conceptual difference between the Cori and Wallinga-Teunis methods is that the former does not

[14] With death being an absorbing state, Table 1 yields a 13×13 transition matrix.

[15] In practical terms this can only be computed *backward* after solving the model *forwards*

take into account time varying policy and disease evolution. In the notation we set out for the Wallinga-Teunis method, the Cori method R_t^C is:

$$\boldsymbol{r}_t^C = \boldsymbol{\rho}_t + \mathbf{P}_t \boldsymbol{r}_t^C \quad , \quad \boldsymbol{r}_t^C = (\boldsymbol{I} - \mathbf{P}_t)^{-1} \boldsymbol{\rho}_t \quad , \quad R_t^C = \boldsymbol{\pi}_t \times \boldsymbol{r}_t^C$$

This means that $R_0^{WT} < R_0^C$ since quarantining of types into the future and building of herd immunity reduces future infections.

3.5.2 Activity

When comparing policies, we focus on three metrics: Output, symptomatic infection, and mortality. A reasonable approximation of economic activity is that it scales with the number of non-quarantined workers. We further assume that quarantined workers are $A_{rel} \in [0,1)$ less productive than non-quarantined workers, and that symptomatic workers are incapacitated and so do not produce. We therefore define output Y_t as

$$Y_t = S_{NQ}^u + TA_{NQ}^u + PA_{NQ}^u + R_{NQ}^u + R_{NQ}^- + A_{rel}\left(S_Q^u + TA_Q^u + PA_Q^u + TA_Q^+ + PA_Q^+ + R_Q^u\right).$$

In the initial period all individuals are non-quarantined and the infected are asymptomatic, so $Y_0 = 1$. Therefore Y_t is in units of the percent change in output from the initial period. A reasonable approximation of the load on the hospital system is that it scales with the number of infected, symptomatic individuals. We therefore report symptomatic infections, M_t^I. The number of deaths at any instant of time is given by $\omega^D M_t^I$. We focus on the cumulative death rate.

4 Calibration

4.1 Parameters

Parameter values are given in Table 2. The parameters of the model can be classified into two groups. The first relate to *medical* parameters, which would be the equivalent of technological parameters in an economic model. We use existing medical studies to inform these parameters. Since a number of recent papers have discussed significant measurement challenges for medical parameters, e.g. Atkeson (2020a), Stock (2020) and Hortaçsu, Liu, and Schwieg (2020), we consider robustness of our conclusions to these parameters in Section 7. The second set of parameters are jointly calibrated to match features of the data. Throughout this section, we convert to daily rates.[16]

[16]The discrete time approximation used to solve the model is with one period equal to one hour, and 14 hours per day.

Parameter		Source / Target	Value
A. Medical			
Daily rate at which infected become symptomatic	$1/\sigma$	6 days incubation period	1/ 6
Daily rate at which symptomatic recover	$1/\gamma$	8 day symptomatic period (14 day recovery)	1/ 8
Fraction that develop symptoms	ψ	Literature	0.60
Relative probability of asymptomatic transmission	ρ^A/ρ^S	Kimball et al (2020)	1
B. Externally calibrated			
Daily rate of meeting	λ	Normalized contact rate	1
Initial asymptomatic infections	I_0	0.1 percent of population on March 16, 2020	340,000
Relative output of quarantined	A_{rel}	Decline in output of 10 percent	0.90
C. Internally calibrated			
Daily mortality rate	ω^D	Infection fatality rate of 1 percent	0.0021
Daily quarantine rate	ξ^u	49.5 percent pop. in quarantine on Apr. 7, 2020	0.0364
Effectiveness of quarantine technology	λ^Q/λ	Deaths on June 15, 2020	0.16
Basic reproduction number	R_0^C	Deaths on April 30, 2020	2.6
Probability of transmission	ρ^S	Given other parameters, implies $R_0^C = 2.6$	0.013

Table 2: Model parameters and values

Notes: We compound parameters in the underlying model into daily rates, which are then expressed in the table.

The benchmark features no testing and reopening only under a vaccine. We seed initial infections in the U.S. on March 16, 2020 and assume that quarantine policy begins on March 20, 2020 (see Figure 4). The quarantine policy is not removed until a vaccine is introduced on January 1, 2021, which is distributed to unvaccinated individuals at a rate of 1.5 percent per day ($\nu = 0.015$). There is no virological or serological testing ($\tau^V = \tau^S = 0$) and no reopening ($r^u = 0$). The number of initial infections I_0 on March 16, 2020 is set to 0.1 percent of the U.S. population: $I_0 = 340,000$. Official data reports 4,507 cases on March 16, so we are assuming this represents 1.5 percent of all cases. This is the rate of under-reporting derived by Hortaçsu, Liu, and Schwieg (2020) for March 9, 2020 (Table 3, Panel 2).

Medical. The daily rate at which infected individuals transition from asymptomatic to symptomatic cases, σ, is such that the average incubation period is 6 days (Wu, Leung, and Leung, 2020). World Health Organization (2020) report that the average recovery period is 14 days for mild infections. We therefore set γ so that symptoms last for 8 days so that expected time from infection to recovery is 14 days. We shorten and lengthen these durations in Appendix A.

The fraction of individuals who never develop symptoms is given by evidence that roughly 40 percent of individuals are permanently asymptomatic: $\psi = 0.6$.[17] Recent evidence on the relative transmissibility of asymptomatic and symptomatic cases is provided by Kimball, Hatfield, Arons, James, Taylor, Spicer, Bardossy, Oakley, Tanwar, Chisty, et al. (2020). They study disease transmission in a skilled nurs-

[17] This is the *upper bound* of estimates in the survey by Byambasuren, Cardona, Bell, Clark, McLaws, and Glasziou (2020). We report robustness with respect to the fraction that are permanently asymptomatic in Section 7, considering $\psi \in \{0.50, 0.75\}$.

ing home and are unable to statistically distinguish differences in viral loads between asymptomatic and symptomatic individuals. Therefore in our benchmark asymptomatic and symptomatic individuals transmit the disease at the same rate: $\rho^A/\rho^S = 1$. [18]

Calibrated. We are left with λ, λ^Q, ρ^S, ω^D, A_0 to calibrate. As shown by equation (3), λ and ρ^S are not separately identified. We therefore set $\lambda = 1$ and back out the implied ρ^S that is consistent with a value of R_0^C using the Cori-method. We treat R_0^C as an auxiliary parameter to be estimated. We set $A_{rel} = 0.90$ such that the benchmark decline in US GDP is 10 percent, which replicates the year-on-year decline in second quarter real GDP from the BEA.[19]

We then jointly estimate (i) the infection fatality rate ω^D, (ii) the quarantine rate ξ^u, (iii) the quarantine technology λ^Q, (iv) reproduction number R_0^C; to target (1) the infection fatality rate, (2) the population under quarantine on April 7, 2020, and (3,4) the number of deaths on April 30 and June 15, 2020. Note that given values for ψ, ρ^A/ρ^S, σ, γ, ω^D, and the normalization of $\lambda = 1$, we can always solve for the ρ^S that yields the conjectured value of R_0^C using equation (3).[20]

4.2 Estimates

We target an infection fatality rate of 1 percent, which is in line with the infection fatality rate in New York computed as deaths divided by total infections, where total infections is measured using serological test results.[21,22] We obtain an estimated mortality rate $\omega^D = 0.0021$. In our robustness exercises we show our results are unchanged to alternative targets of the infection fatality rate of 0.5 and 1.5 percent.

[18] Furukawa, Brooks, and Sobel (2020) provides a review of pre-symptomatic and asymptomatic viral loads (cycle thresholds). These cycle thresholds determine infectiousness. The cycle threshold is inversely proportional to the viral load. After conducting viral load tests in a long-term care skilled nursing home, Kimball, Hatfield, Arons, James, Taylor, Spicer, Bardossy, Oakley, Tanwar, Chisty, et al. (2020) describe an inability to distinguish differences in viral loads between asymptomatic and symptomatic individuals, although their samples sizes and power of tests are low. Kimball, Hatfield, Arons, James, Taylor, Spicer, Bardossy, Oakley, Tanwar, Chisty, et al. (2020) write, *"The reverse transcription–polymerase chain reaction (RT-PCR) testing cycle threshold (Ct) values indicated large quantities of viral RNA in asymptomatic, presymptomatic, and symptomatic residents, suggesting the potential for transmission regardless of symptoms... Real-time RT-PCR Ct values for both genetic markers among residents with positive test results for SARS-CoV-2 ranged from 18.6 to 29.2 (symptomatic [typical symptoms]), 24.3 to 26.3 (symptomatic [atypical symptoms only]), 15.3 to 37.9 (presymptomatic), and 21.9 to 31.0 (asymptomatic). There were no significant differences between the mean Ct values in the four symptom status groups (p = 0.3)."*

[19] See: https://fred.stlouisfed.org/graph/?g=tEqU

[20] We can match the first two moments exactly. We minimize the total absolute percent deviations of the data and model across the death moments.

[21] We refer readers to Fernández-Villaverde and Jones (2020) for a thorough discussion of existing infection fatality rate estimates.

[22] It is important to note that ω^D does not correspond to the *case fatality rate*, where the case fatality rate is defined to be the ratio of cumulative deaths to cumulative cases. *"In epidemiology, a case fatality rate (CFR) — sometimes called case fatality risk or case-fatality ratio — is the proportion of deaths from a certain disease compared to the total number of people* diagnosed *with the disease for a certain period of time."* (Wikipedia).

As shown in Figure 4, the fraction of the U.S. population living in states with some form of stay-at-home order peaked at 90 percent on April 7, 2020. Of course, not all individuals in those states actually entered quarantine. Many essential sector workers continued to work, as well as non-compliers. Following Glover, Heathcote, Krueger, and Ríos-Rull (2020), who refer to this as the *'basic sector'*, we assume that 45 percent of workers are in the basic sector and that workers in the basic sector do not enter quarantine. Our estimate $\xi^u = 0.0364$ per day matches 49.5 percent $(= 0.90 \times (1 - 0.45))$ of the population in quarantine as of April 7, 2020.

When matching deaths, we follow Fernández-Villaverde and Jones (2020) and inflate mortality data by 33 percent.[23] A higher R_0^C leads to a steeper path for initial deaths, while a lower λ_Q leads to a quicker flattening off. We estimate $R_0^C = 2.6$ and $\lambda^Q = 0.16$, such that quarantine reduces meetings by a factor of more than six. Our estimated value of R_0^C is in line with estimates of the basic reproductive number at the onset of the Italian and Chinese Covid-19 outbreaks (Li, Guan, Wu, Wang, Zhou, Tong, Ren, Leung, Lau, Wong, et al. (2020) and Gatto, Bertuzzo, Mari, Miccoli, Carraro, Casagrandi, and Rinaldo (2020)). In our robustness exercises we consider alternative values of $R_0^C \in \{2, 3\}$.

4.3 Model fit

In Figure 6, we plot the path of deaths, quarantine, and the Wallinga-Teunis reproductive number R_t^{WT} for the benchmark model. We matched the fraction of the population in quarantine on April 7, 2020 (Panel B). Our simple approach to quarantine also matches the rest of the time-series well. The model comes close to generating the path for deaths in the data, however is unable to simultaneously generate the steep convex to concave time-series of cumulative deaths given initial infections. The model matches deaths on June 15, but understates deaths on April 30.[24] The time-varying path of the Wallenga-Teunis basic reproductive number R_t^{WT} incorporates the future path of quarantine policy and thus lower infection rates. In accounting for this it is markedly lower at the onset of our simulation than 2.6. The implied time varying path of R_t^{WT} is in line with estimates for the U.S. (Fernández-Villaverde and Jones, 2020).

5 Testing and reopening

We now introduce virological (τ^V) and serological testing (τ^S). First, we compute output and deaths under the benchmark quarantine-only policy with no reopening: $\{\xi^U = 0.0364, r^u = \tau^V = \tau^S = 0\}$.

[23] This inflation factor captures under-reporting of deaths, and is based on excess death measures in other countries and New York City.

[24] A higher value of R_0^C could match this, but would require a lower λ^Q to match deaths on June 15.

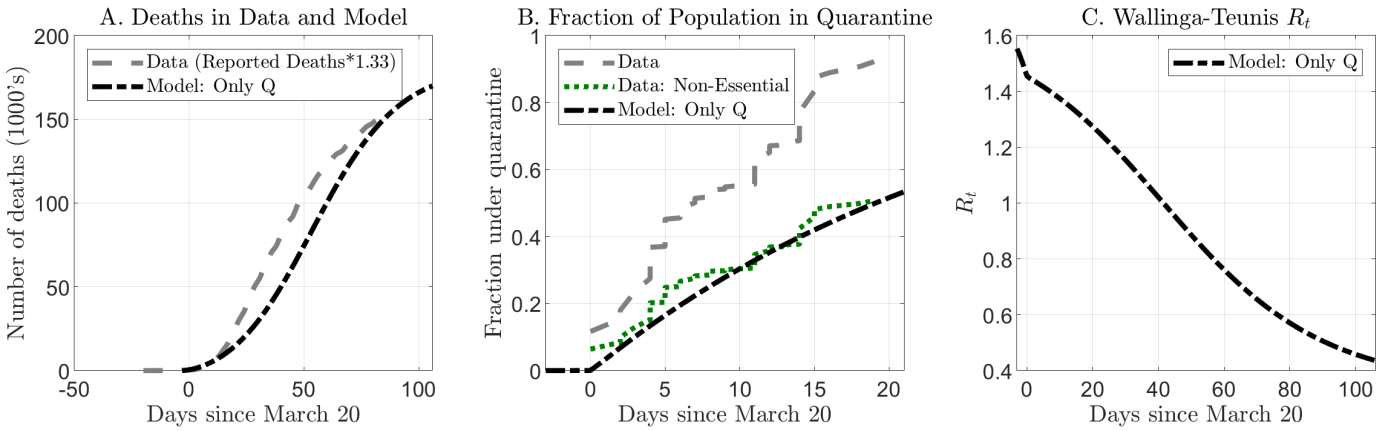

Figure 6: Model fit of deaths, quarantine, and Wallinga-Teunis basic reproductive number

Second, we measure how much we can reopen by increasing the release rate of unknown cases r^u in the testing regime so that long-run cumulative deaths are the same as the benchmark quarantine-only policy. We find muted effects of testing in the former case, however when frequent testing is coupled with targeted quarantine and a reduction in overall levels of lockdown, output gains can be substantial with no additional mortality.

Throughout we consider different rates of testing. To make sense of the testing numbers, we report the average number of weeks between tests for an unknown individual at March 20 if an individual were to remain in their current state. For example, an average number of weeks between tests of 1 week corresponds to roughly 340 million tests in one week. We change τ^S and τ^V such that this ranges between weekly to once every 3 months.

5.1 Testing without reopening

We first illustrate the impact of virological and serological tests on output losses and deaths under our benchmark model with fixed quarantine policy $\xi^u = 0.0364$ and zero reopening $r^u = 0$ ("Only Q"). We consider two cases, (i) quarantine combined with virological tests ("Q+V test"), and (ii) quarantine combined with serological tests ("Q+S test"). Recall that if an individual has a positive virological test, we assume they enter quarantine (see Table 1), and if a serological test identifies antibodies we assume they are released from quarantine (see Table 1).

Weekly testing. Figure 7 plots symptomatic infections, cumulative deaths, and output. The black line in Figure 7 traces the paths of these variables for our benchmark quarantine-only economy $\{\xi^u =$

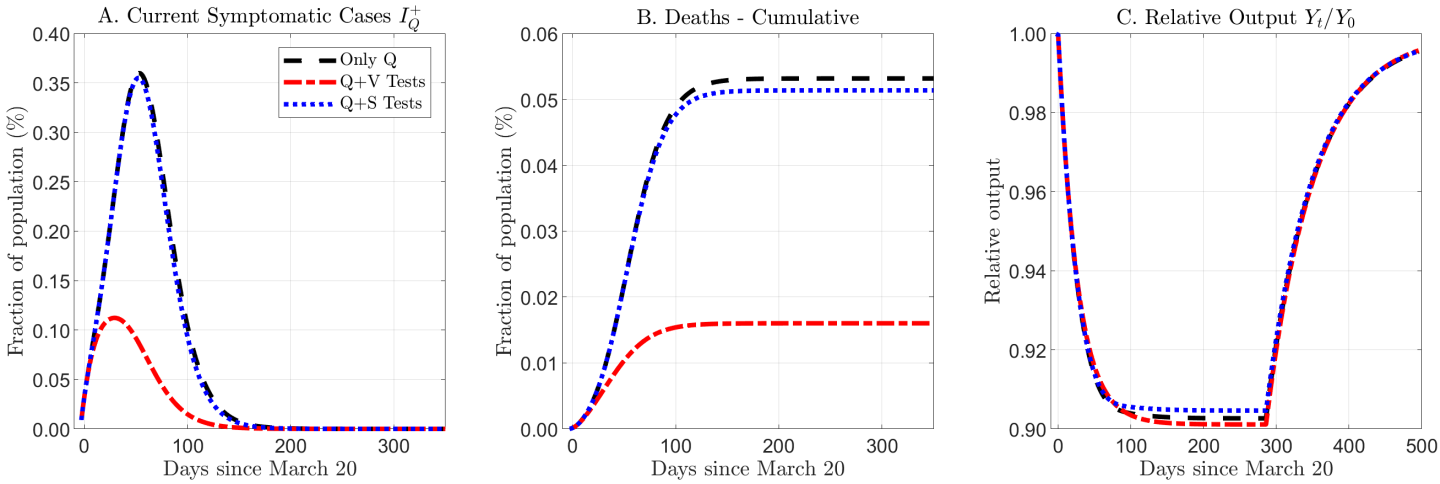

Figure 7: Testing every two weeks under the benchmark quarantine policy

$0.0364, r^u = \tau^v = \tau^s = 0\}$. Under the benchmark policy, panel A shows that approximately 0.4 percent of the population are symptomatic at the peak of the pandemic. Overall 5 percent of the population are infected, just over 17 million in total with 10 million developing symptoms, which seems reasonable given 5 million confirmed cases reported by early August and under-reporting. Approximately 0.05 percent of the U.S. population dies from the disease, around 180,000 deaths. Output is shaped by the quarantine policy and the subsequent vaccination regime. Output declines by our target of 10 percent and then recovers sharply on January 1, 2020 with the arrival of the vaccine which moves individuals to the known recovered state from which they are released.

The dashed red line in Figure 7 traces out the path of the economy when individuals are virologically tested every week $\{\xi^u = 0.0364, r^u = 0, \tau^V > 0, \tau^S = 0\}$. The benchmark quarantine policy remains in place with no reopening, however, those with positive virological tests are quarantined. In quickly quarantining asymptomatic cases, the spread of the disease is reduced and peak infection falls by a factor of four, and cumulative deaths by a factor of around five. The peak also occurs 38 days earlier than the benchmark quarantine-only economy. Since positive virological tests create more quarantine, output losses are larger in Panel C. Here testing is simply a targeted *lockdown* policy and so generates the same type of output-death trade-off as any other lockdown policy.

The blue line in Figure 7 traces out the path of the economy when individuals are serologically tested every week $\{\xi^u = 0.0364, r^u = 0, \tau^V = 0, \tau^S > 0\}$. The benchmark quarantine policy remains in place, however, those who test positive for antibodies (positive serological test) are released. Serological testing has a muted effect on public health outcomes with a small decline in infections and deaths. This might come as a surprise: by identifying and releasing more individuals with antibodies, a greater fraction of

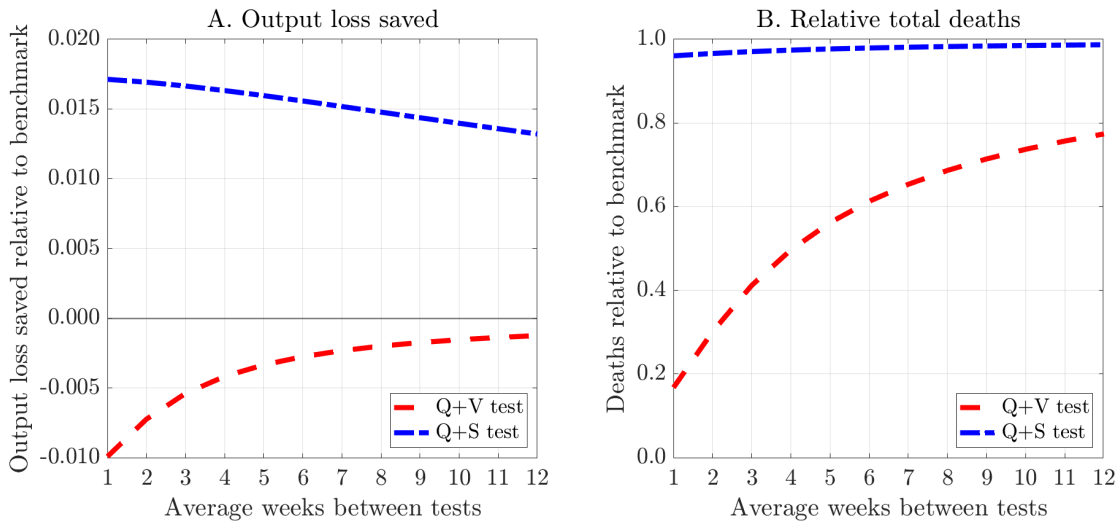

Figure 8: Effects of testing under benchmark quarantine policy

Notes: In this case $\{\xi^u = 0.0364, r^u = 0\}$, such that quarantine policy is as in the benchmark calibration. Output loss is computed as follows: We add up over 365 days from March 20, 2020, the total output loss relative to the non-Covid baseline—under which daily output is equal to 1 ($Y_0 = 1$)—and then divide by the same measure for the benchmark case in which there is only quarantine: $\sum_d \left(1 - Y_d^{WithTests}\right) / \sum_d \left(1 - Y_d^{Bench}\right)$. Relative total deaths over the pandemic under the testing counterfactual, divided by total deaths for the benchmark case in which there is only quarantine: $\sum_d D_d^{WithTests} / \sum_d D_d^{Bench}$.

meetings are with immune individuals which reduces disease spread, especially so given our assumption of a linear matching technology. These effects are quantitatively small for the simple reason that the number of recovered individuals is small. As opposed to the virological test, however, output losses are dampened as the serological test amounts to a targeted *release* policy, increasing labor supply. Thus serological testing coupled with targeted release does *not* generate a tradeoff between output losses and deaths.

Varying test frequency. How do the health and economic outcomes of testing without reopening depend on the frequency of testing? Figure 8 compares output losses and deaths as the frequency of testing varies. The x-axis reports average weeks between testing. Panel A plots the cumulative difference in output losses between March 20, 2020 and March 20, 2021 in the economy with testing relative to the benchmark economy without testing. Positive values imply smaller output losses and a less severe recession relative to the benchmark. For example, a positive value of 0.01 on the y-axis corresponds to a *1 percent smaller reduction in cumulative output* over the subsequent year, i.e. the sum of the differences between the red and black lines in Figure 7C. For example, cumulative GDP falls by 1 trillion between March 20, 2020 and March 20, 2021 without testing, and only 980 billion with serological testing. Negative values imply greater output losses and a more severe recession.

Under the assumption that high, non-targeted levels of lockdowns persist $\{\xi^u = 0.0364, r^u = 0\}$,

tests have small, but very different effects on output. The red dotted line in Panel A illustrates that virological testing generates greater output losses. Individuals are quarantined if they test positive, reducing output. The additional quarantine that comes from cases identified by administering a weekly virological test causes output to decline by approximately 1 percent more than the benchmark. On the other hand, serological testing increases output as individuals are released if revealed to have antibodies, increasing labor supply. With weekly testing, output losses are roughly 1.75 percent less severe. Moving to the right, the test frequency declines, and output differences are less marked in both cases.

From the perspective of output alone, serological tests dominate, but effects are small. However testing policy need not be formulated in a vacuum. With more testing, quarantine rates may be adjusted, increasing labor supply in the case of the virological test, potentially to the point where output is higher under the virological testing regime. The extent to which quarantine can be slackened depends on the public health consequences of the testing regimes.

Panel B of Figure 8 plots the ratio of cumulative deaths between March 20, 2020 and March 20, 2021 in the economies with testing relative to the benchmark without testing. For example, a value of 0.4 on the y-axis implies that the economy with testing has only 40 percent as many deaths as the benchmark economy without testing.

The virological test generates significantly more slack in terms of public health. The red dotted line illustrates that the virological test steeply reduces the number of deaths relative to the benchmark. Weekly virological testing reduces cumulative deaths by 80 percent, and even monthly testing lowers deaths by 50 percent. Benefits are also non-linear: doubling the frequency from 12 to 6 weeks, saves less lives than doubling the frequency from 6 to 3 weeks.

On the other hand, serological tests tend to have limited effects on deaths. Serological tests release immune individuals back into the pool of potential contacts, reducing the infection rate conditional on meeting since more meetings are with immune types. Quantitatively, however, because their numbers are small, this has only a muted effect on disease transmission, and so a muted effect on deaths.

Alone, serological tests increase output and reduce deaths, shifting the so-called *pandemic possibility frontier* that links deaths and output (Kaplan, Moll, and Violante, 2020). The open question, then, is whether in conjunction with a reopening policy, virological testing can do even better in terms of shifting this frontier.

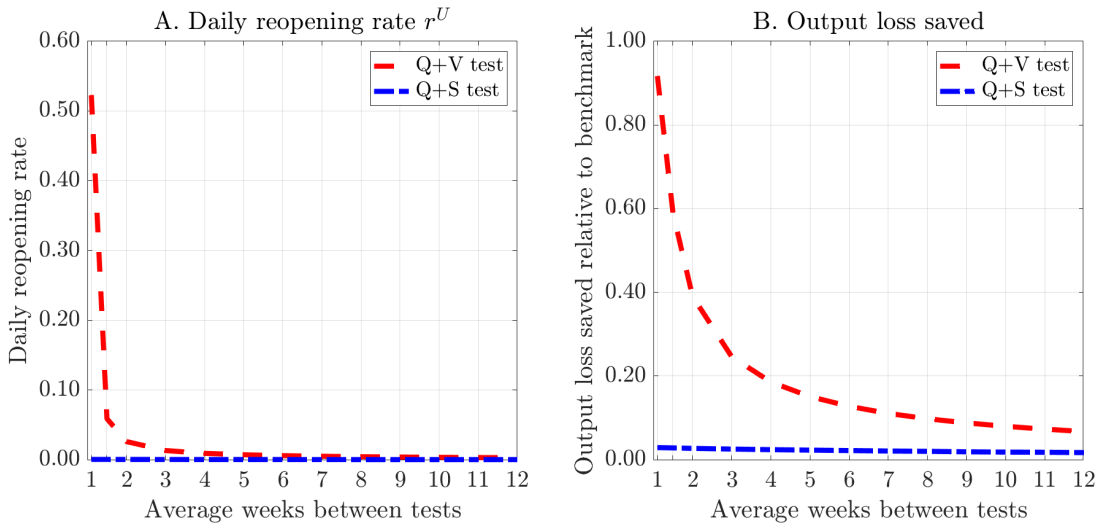

Figure 9: Effects of testing under targeted quarantine policy - Constant long-run deaths

Notes: In this case $r^u > 0$ is set to match long-run deaths as testing frequency varies along x-axis. The reopening rate r^U is expressed at a daily frequency. Panel A plots the output loss saved relative to the benchmark quarantine-only policy. Output loss saved is computed as follows: We add up over 365 days from March 20, 2020 the relative output losses and then divide by the total output loss for the benchmark quarantine-only case: $\sum_d \left(Y_d^{WithTests} - Y_d^{Bench}\right) / \sum_d \left(1 - Y_d^{Bench}\right)$. Panel B plots the value of r^u that generates constant long run deaths.

5.2 Testing and reopening

To answer this question we consider a reopening policy that slackens quarantine measures by setting $r^u > 0$, and ask how much we can reopen without increasing deaths beyond the level of the benchmark economy.

Figure 9 gives the results of this exercise. Panel A plots the admissible value of r^U that generates the same *total* number of deaths as the benchmark economy. Since the targeted quarantine that arose from virological testing reduced deaths substantially, the economy can be reopened substantially even for unknown cases. The small degree of slack generated by the targeted release from serological testing means that even a small amount of reopening increases deaths above the benchmark level.

How do these translate into economic output? Panel B plots the implied output loss saved. With weekly virological testing, around 90 percent of the decline in output over the first year of the pandemic can be erased without incurring additional deaths. Even with monthly testing, 20 percent of the decline in output can be restored. Again, with only mild public health effects, the output effects of reopening in the presence of high frequency serological testing are low.

To visualize their implications for the progress of the pandemic and output, Figure 10 replicates Figure 7 under testing every two weeks and reopening.[25] Panel B verifies that long-run deaths are the

[25] In Appendix B we provide the same figure for weekly testing: Figure B1. Since weekly testing almost entirely removes the

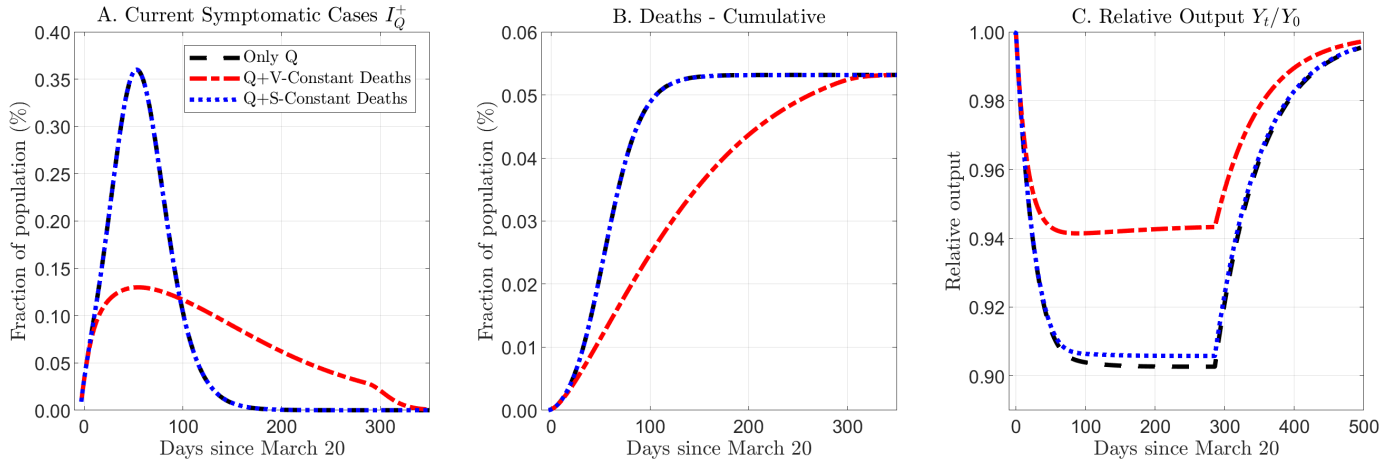

Figure 10: Testing every two weeks and reopening $r^u > 0$ with constant long-run deaths.

Notes: Panel A plots infections, Panel B plots cumulative deaths, and Panel C plots contemporaneous output losses.

same under the benchmark and testing with reopening regimes. Relative to the testing and targeted quarantine policy without reopening, virological testing with reopening leads to a slower progression of the pandemic. Reopening leads to greater infections, which occur slowly, but with a testing and quarantine regime that quickly identifies and isolates asymptomatic carriers, the pandemic remains under control. In 'flattening the curve', additional benefits that we do not model here would also play a role: hospital system preparedness, potential development of anti-virals, and so on. In terms of output loss, the reopening quickly stanches the decline in output, and the economy never reaches the depths of the benchmark case.[26] As now expected, the effects of the serological tests are minor.

In summary, we find that a targeted quarantine policy along with virological testing, and a reopening policy can lead to simultaneously better output and public health outcomes. We now turn to implementation of virological testing given these results.

6 Quality vs. quantity in virological testing - A cheaper lunch

In implementing virological testing, we consider a simple counterfactual that shows that a planner would be willing to trade-off test quality for test frequency.[27] We have shown that the effects of viro-

decline in output it is visually hard to use as a benchmark for robustness exercises.

[26] To understand the quantities, consider the following approximation. Ignoring infection and death, the flows in and out of quarantine would yield steady-state $M^Q = \xi^U/(xi^U + r^U)$. With testing every two weeks, $M^Q \approx 0.04/(0.04 + 0.04) = 0.50$, so output would be $Y = 0.50 + A_{rel} \times 0.50 = 0.95$, which is approximately the value of the output decline in Figure 10, after 50 days.

[27] For a contemporaneous analysis with similar results see the following working paper: Larremore, Wilder, Lester, Shehata, Burke, Hay, Tambe, Mina, and Parker (2020a).

logical testing on public health outcomes are highly non-linear (Figure 9B). In particular, testing and quarantining at much higher frequencies sharply reduce deaths. Suppose that there were a quantity / quality trade-off such that, given a testing budget, better, more expensive tests could be administered infrequently, or poorer, cheaper tests administered more frequently. The non-linearity suggests a planner may be willing to bear *significantly* poorer quality. This is a recent policy considered by the FDA, which on July 29, 2020 issued guidance for *At-Home* testing.[28,29]

We compare two virological tests which we view as representing standard Polymerase Chain Reaction (PCR) tests, and an alternative we call an *At Home Test* (AH test). These tests differ in two dimensions. First, the PCR test has a zero percent false negative rate—consistent with our analysis so far—, while the AH test has extremely poor *sensitivity*, yielding a 50 percent false negative rate. This is substantially worse than usual FDA requirements of a false negative rate of less than 20 percent. An individual in state (TA) or (PA) and *unknown* that receives a false negative simply remains *unknown*. Second, we assume that the cost of the tests scale linearly: the PCR test which we consider being administered monthly costs \$40 per test, and the AH test which we consider being administered weekly costs \$10 per test.[30] Both regimes therefore have the same monthly cost which we view as a useful benchmark. They also have the same *specificity*: they generate false positives at a rate of zero.

Our main result is that even with such poor sensitivity, weekly testing dominates: more lives are saved at a lower cost. Figure 11 presents a simple cost-benefit analysis. In blue circles and red diamonds we compare the two tests. At a cost of \$40/month per person, the less sensitive AH test delivers a saving of around 40,000 lives. To save the same number of lives relative to the benchmark with the PCR test, testing would need to be at least every two weeks and cost more than \$80/month. By lowering deaths by more, the weekly AH test generates more slack in terms of mortality, so re-opening measures could be looser, reducing output losses.

This simple exercise shows that the extreme non-linearities that occur due to testing and targeted quarantine at higher frequencies have important consequences for trade-offs across testing regimes. Importantly, the FDA could approve cheaper tests with very high false negative rates and achieve lower healthcare costs, better healthcare outcomes and better economic outcomes.

[28] https://www.fda.gov/news-events/press-announcements/coronavirus-covid-19-update-fda-posts-new-template-home-and-over-counter-diagnostic-tests-use-non

[29] For a contemporaneous analysis, see Larremore, Wilder, Lester, Shehata, Burke, Hay, Tambe, Mina, and Parker (2020b).

[30] This is the test-price that can be charged to Medicare by contractors https://www.cms.gov/files/document/mac-covid-19-test-pricing.pdf.

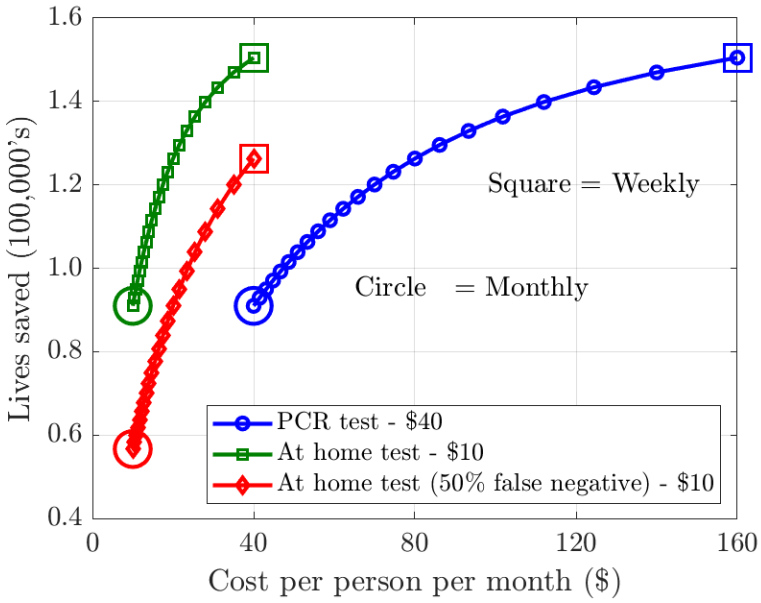

Figure 11: More frequent testing with poorer quality tests dominates

7 Robustness

Here we consider robustness of our conclusion regarding serological and virological testing with respect to a substantive model assumption, and robustness with respect to parameters and moments to the appendix. Throughout we have assumed that infected individuals transition to the *known negative* information state. This assumption may be strong. Without a test verifying that they have had symptoms the government may be unwilling to lift their quarantine. This potentially biases results against serological testing, which could serve a verification role even for those that have developed symptoms.

We show that this is the case, but the effects are quantitatively small. To make this point clearly we go one step further and make virological tests meaningless for the recovered status of an individual. Regardless of past symptoms and virological tests, we assume that an individual must receive a serological test before being granted an *'immunity passport'* and being released. Previously known cases of infected-symptomatic (I_Q^+) and permanently-asymptomatic (PA_Q^+), both transition to recovered-unknown (R_Q^u).

Figure 12C shows that our conclusions with respect to the effectiveness of tests remains qualitatively and quantitatively similar to our main counterfactual (Figure 10). Baseline deaths are now higher as recovered negative cases no longer circulate, but our counterfactual exercise with constant deaths effectively controls for this. Serological tests now permit wider reopening and a smaller drop in output but, again, these effects are small. Virological tests are now less powerful as they no longer grant immu-

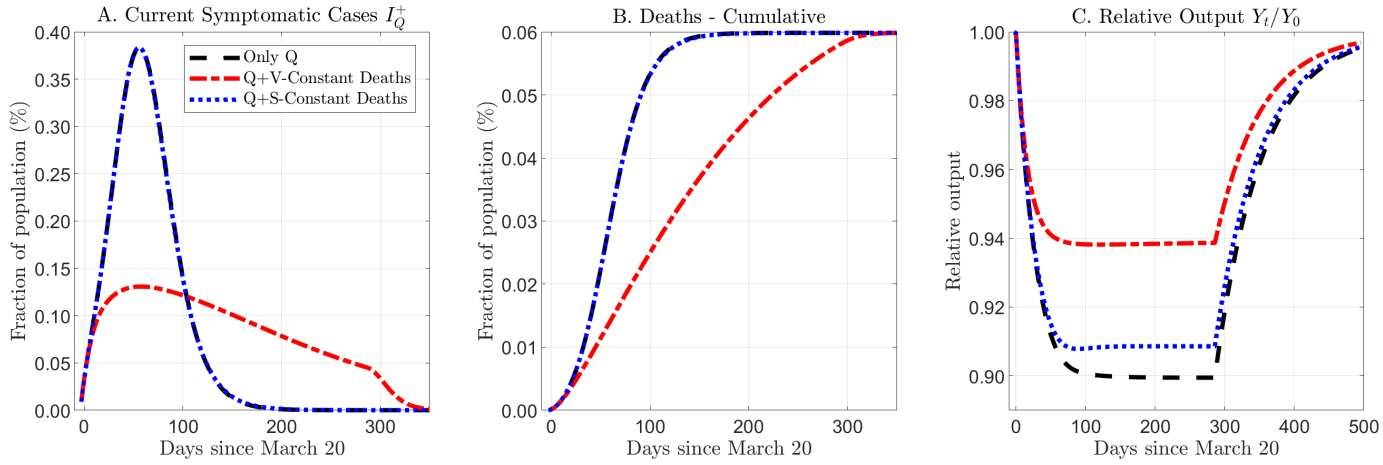

Figure 12: Robustness: Immunity passports require serological testing.

Notes: Panel A plots infections, Panel B plots cumulative deaths, and Panel C plots contemporaneous output losses.

nity passports to those that were tested while asymptomatic. With no role in paths to $R^-_{NQ'}$ the effects of virological tests are slightly smaller, but the additional drop in output is very small. We conclude that neither (i) the double-duty the virological test is doing in our baseline exercises by identifying infected *and* recovered cases, (ii) previous symptoms conveying future recovered status, affect our main conclusions.

Parameters and moments. In the appendix we show how the extent of possible reopening varies under the two testing regimes as we vary targets and parameters. In all cases we recalibrate ρ^S and ω^D such that the model has the same R_0^C and infection fatality rate, and conduct the same 'constant deaths' counterfactual. We show that this benchmarking key for understanding comparative statics. Changes in the target IFR have no effect on the output savings attainable through testing, conditional on matching R_0^C. Under a lower $\rho^A < \rho^S$, lower R_0^C, higher ψ (so *less* permanently asymptomatic), output saved under testing is *higher*. When incubation and infection periods are longer, testing is also more effective in creating capacity for reopening. We present full details and discussion of these robustness exercises in Appendix A.

8 Conclusion

This paper conceptualizes a minor and easily implemented change to the standard SEIR model of infectious disease transmission, and shows that *public health* policies can be of first order importance for *economic trade-offs*. In particular, testing and targeted reopening can slacken the output-mortality trade-

off that is the feature of many economic models of the pandemic.

Quarantine policies can only depend on observed health states, which creates a role for testing in distinguishing between infected and non-infected asymptomatic individuals. Our model, calibrated to the US in March-June 2020, demonstrates that testing asymptomatic individuals can stand-in for economically costly quarantine measures. We make this notion precise by reopening the economy as we test such that the overall mortality rate of the pandemic remains constant. With fewer individuals quarantined, output of the economy would decline substantially less, with testing every two weeks halving the loss in output. Testing allows the government to do better in terms of both *deaths and output*.

Our analysis comes with two important caveats. First, we study an ex-ante homogeneous population. Despite this, our framework demonstrates that incomplete information, testing and targeted quarantine policies can be simply and intuitively integrated into a richer model. Second, our model is *not* a behavioural economic model that integrates an epidemiological model as in Kremer (1996), Greenwood, Kircher, Santos, and Tertilt (2019), or more recently Jones, Philippon, and Venkateswaran (2020), Brotherhood, Kircher, Santos, and Tertilt (2020), Keppo, Quercioli, Kudlyak, Smith, and Wilson (2020) and Toxvaerd (2020). Despite this, we believe our points would survive both qualitatively and quantitatively. In these environments individuals self-quarantine in response to increased mortality risk. A testing and targeted quarantine program reduces the risk of infection and death and would lead individuals to optimally resume activity. How much would they resume activity? Until mortality risk is similar to that which led to their self-quarantine decision in the first place. Since our counterfactuals reopened in a way that kept mortality risk constant, we argue that our exercises are a good approximation of the effect of testing in a model with self-quarantining individuals.

Our exercises show that adding incomplete information and a role for testing through targeted quarantine does not overly complicate the baseline model and allows discussion of testing policies that cannot be discussed in the baseline complete information model. As such this framework could be integrated into models that are richer in terms of both heterogeneity and economic behavior.

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ONLINE APPENDIX NOT FOR PUBLICATION

A Robustness exercises

In this section we test robustness with respect to the infection fatality rate, asymptomatic transmission rate, the share of infections that are permanently asymptomatic, recovery duration, incubation period, and basic reproduction number. Importantly, in each case (except for the last case when we directly move the basic reproductive number), (a) ρ^S is adjusted such that $R_0^C = 2.6$, (b) ω^D is adjusted such that the infection fatality rate is 1 percent (apart from in the case where we target a new infection fatality rate). All figures reflect our main counterfactual exercise, and are directly comparable to Figure 10 which we refer to as our *baseline set of results*. That is, (i) we compute baseline total deaths under "Only Q", (ii) we consider testing every two weeks, (iii) we compute the reopening rate r^U such that total deaths under each test coincide with baseline deaths under "Only Q".

We summarize our findings below, focusing on virological testing and discussing both tests in detail over the following pages. The result of interest is the magnitude of the savings in output that can be achieved by testing and reopening, i.e. where does the red dashed line sit in the output loss figure *relative to* our baseline set of results Figure 10C.

- The target for the infection fatality rate has no affect the output losses under testing and reopening.
- A 50 percent *lower rate* of asymptomatic transmission $\rho^A = \rho^S/2$ *increases* the savings in output. This may seem counterintuitive, however when delivering the same R_0^C, infections are disproportionately driven by symptomatic types which self-quarantine, increasing the scope to reopen.
- Changes in the share of permanently asymptomatic types $(1 - \psi)$ has very small effects due to offsetting forces we account for in the recalibration of ρ^S, ρ^A, ω^D to match R_0^C and the IFR.
- Longer recovery and incubation periods lead to slightly larger output savings from testing, but these are small.
- A lower R_0^C makes testing substantially more effective in reopening.

These exercises demonstrate that our results are robust. They also show the value in two uses of benchmarking. First, *within* each counterfactual exercise we benchmark testing and reopening against deaths from the 'Only Q' case. Second, *across* counterfactuals, we recalibrate the model to match the same value of R_0^C and infection fatality rate.

1. Fatality rate (ω^D)**.** In our benchmark calibration, $\omega^D = 0.0021$ was chosen to deliver an infection fatality rate of 1 percent. Figure A1 reduces this to deliver an IFR of 0.5 percent $(\omega^D = 0.0011)$, and Figure increases this to deliver an IFR of 1.5 percent $(\omega^D = 0.0032)$ With a lower (higher) IFR there are obviously less (more) deaths. However, since our counterfactual exercise reopens the economy up to the point at which deaths are the same as under the benchmark, then we find that the level of reopening is approximately independent of the IFR. This leads to almost identical output losses as our baseline set of results.

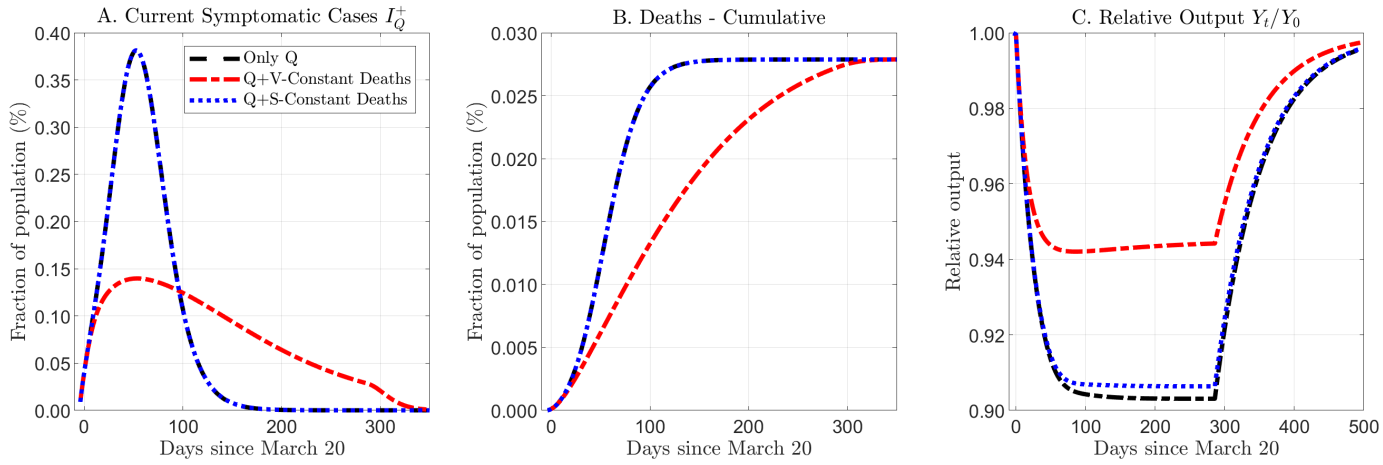

Figure A1: Lower ω^D to deliver lower IFR of 0.5%, all other parameters held at benchmark values.

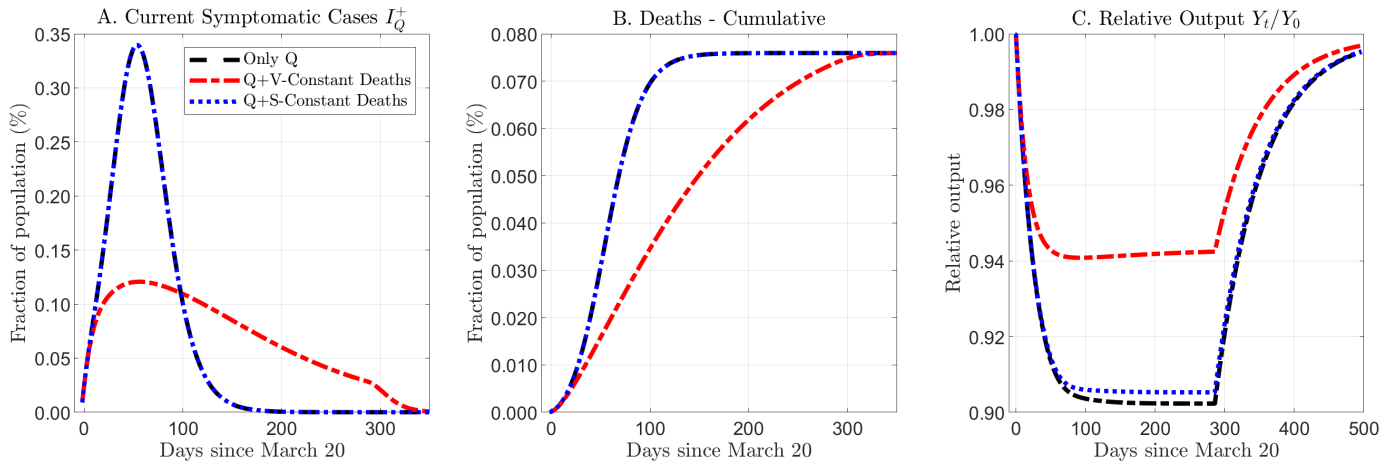

Figure A2: Higher ω^D to deliver higher IFR of 1.5%, all other parameters held at benchmark values.

2. Asymptomatic transmission rate (ρ^A)**.** In our benchmark calibration, $\rho^A/\rho^S = 1$, such that asymptomatic and symptomatic carriers have the same rate of transmissibility. Figure A3 reduces $\rho^A/\rho^S = 0.50$, reducing asymptomatic transmissibility.

Here our recalibration matters. We keep R_0^C constant, since we are asking the question *'Suppose that the data was generated by a lower* ρ^A/ρ^S*, what would this imply for testing?'*, with R_0^C being a feature of this data. To achieve this the new values are such that $\rho^{A\prime} < \rho^A = \rho^S < \rho^{S\prime}$. This implies that infections are being driven by symptomatic cases which are self-quarantining. If this is the case, then reopening can be more aggressive, since the non-symptomatic cases have lower transmissibility. Testing and reopening leads to a *smaller* decline in output relative to our baseline set of results.

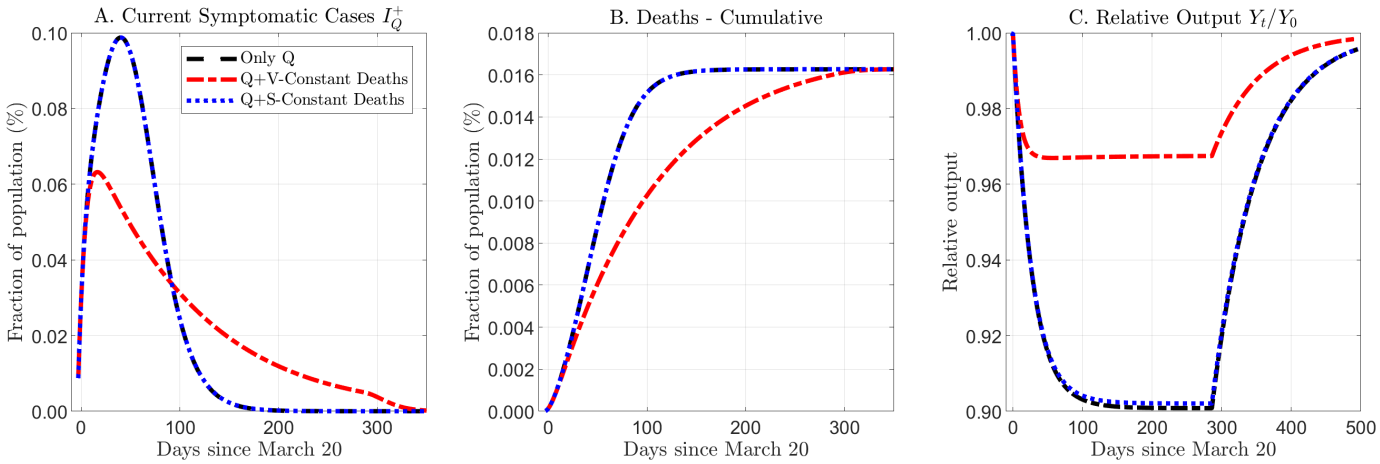

Figure A3: Parameters $\rho^A/\rho^S = 0.50$, all other parameters held at benchmark values.

3. Share of asymptomatic infection $(1-\psi)$**.** In our benchmark calibration, $(1-\psi) = 0.40$, such that 60 percent of infected cases develop symptoms and 40 percent do not. Figure A4 decreases $(1-\psi) = 0.25$, reducing permanently asymptomatic cases, and Figure A5 increases $(1-\psi) = 0.50$, increasing permanently asymptomatic cases.

With more permanently asymptomatic cases (Figure A5), there is a larger role for serological testing, which reduces the decline in output by around 1 ppt relative to the case with fewer permanently asymptomatic cases (Figure A4). Again, these effects are small as the total amount of individuals that are ever infected is small.

The fact that the virological test is accompanied by *less* reopening and a slightly larger output drop may at first seem counter-intuitive, but is explained as follows. With *more* permanently asymptomatic types, which do not die from the disease, then to match the same R_0^C as the baseline requires a higher rate of transmissibility, i.e. an increase in ρ^A and ρ^S. This leads to the higher level of peak infections and total deaths in the 'Only Q' benchmark in Figure A5)A & B. This means there are more permanently asymptomatic types, and they are more infectious, while the symptomatic cases are quarantining as before, so the higher ρ^S has less of an affect through them. Higher levels of virological testing cannot undo this worsening of health outcomes completely.

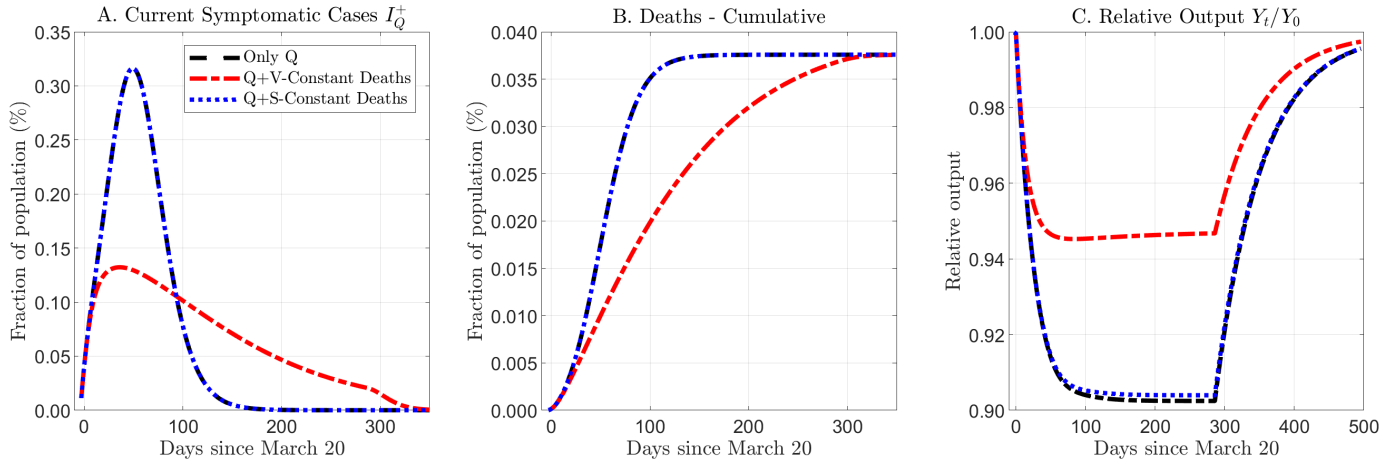

Figure A4: Lower share of permanently asymptomatic: $(1-\psi) = 0.25$, all other parameters held at benchmark values.

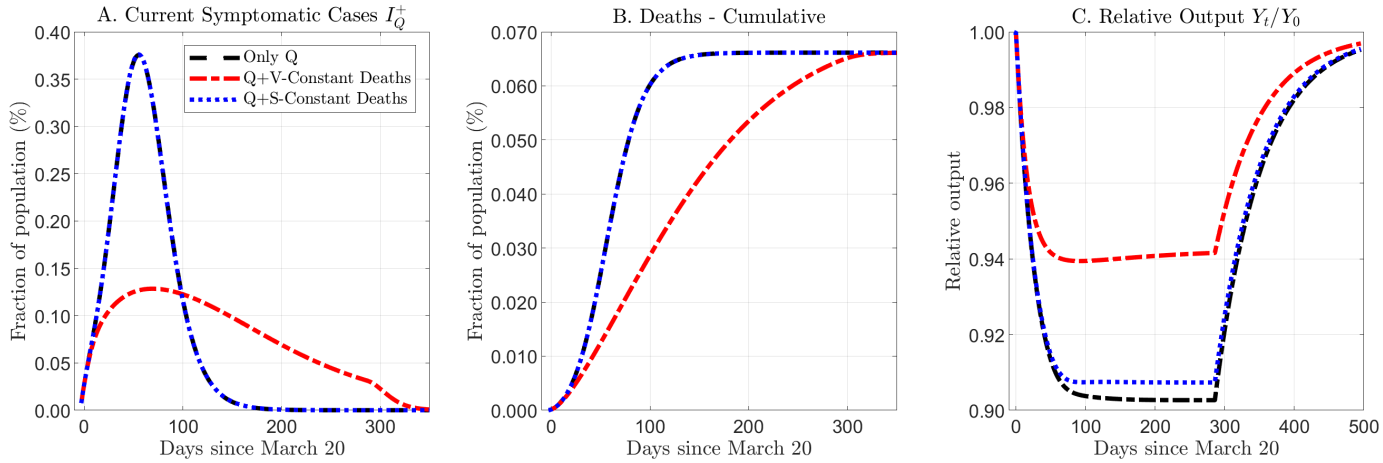

Figure A5: Higher share of permanently asymptomatic: $(1-\psi) = 0.50$, all other parameters held at benchmark values.

4. Recovery (γ)**:** In our benchmark calibration, γ was such that the expected duration of the infected state was 8 days. Figure A6 reduces this to 6 days and Figure increases this to 10 days. Note that since the transition from the *permanently asymptomatic* state to *recovered* was chosen to match the overall same duration of infection as a case that develop symptoms, then this is increased as well.

With a longer overall period of infection of both permanently and temporarily asymptomatic types, virological tests have a larger effect in reducing deaths, which allows for broader reopening. Again, the setup of our counterfactual implies that these effects are not large.

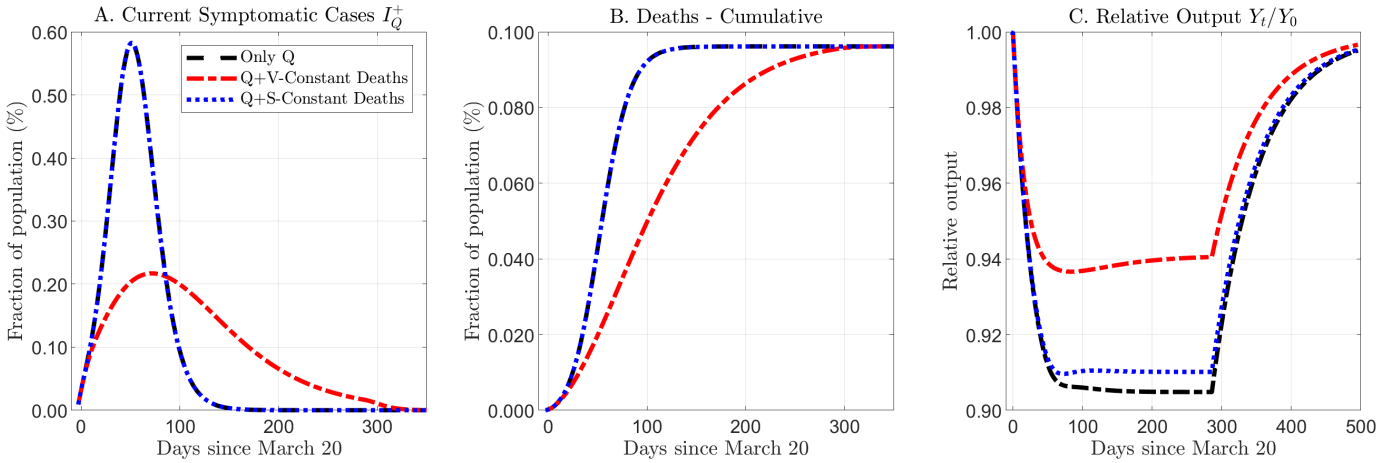

Figure A6: Shorter duration to recovery: $\gamma = 1/6$ days, all other parameters held at benchmark values.

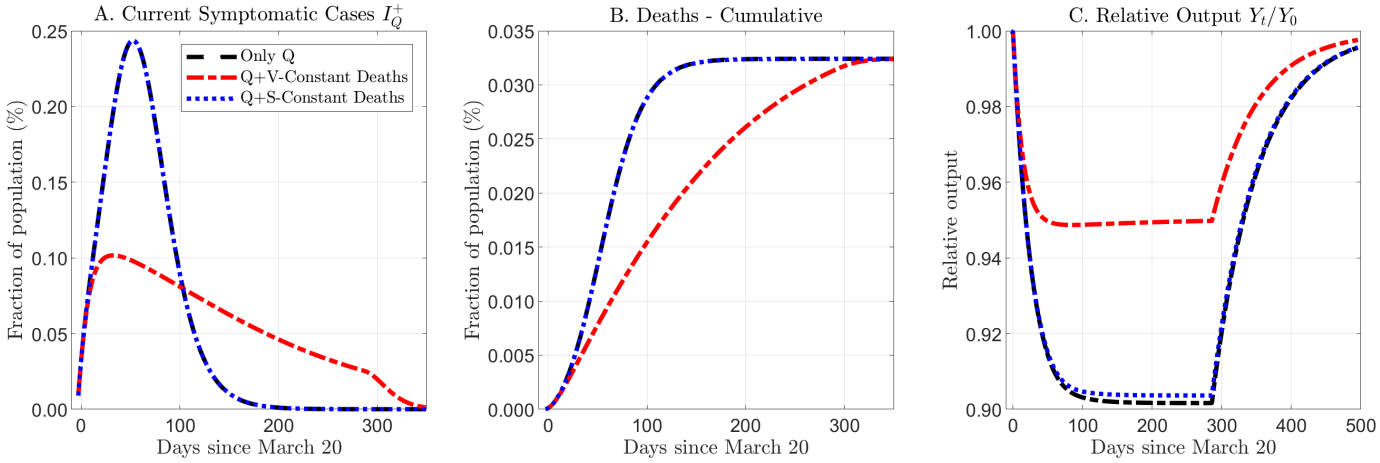

Figure A7: Longer duration to recovery: $\gamma = 1/10$ days, all other parameters held at benchmark values.

5. Incubation period (σ)**:** In our benchmark calibration, σ was such that the expected duration of incubation was 6 days. Figure A8 reduces this to 4 days and Figure increases this to 8 days. Note that since the transition from the *permanently asymptomatic* state to *recovered* was chosen to match the overall same duration of infection as a case that develop symptoms, then this is increased as well.

Similar to the previous exercise, with a longer overall period of infection of both permanently and temporarily asymptomatic types, virological tests have a larger effect in reducing deaths, allowing broader reopening. Again, the setup of our counterfactual implies that these effects are not large.

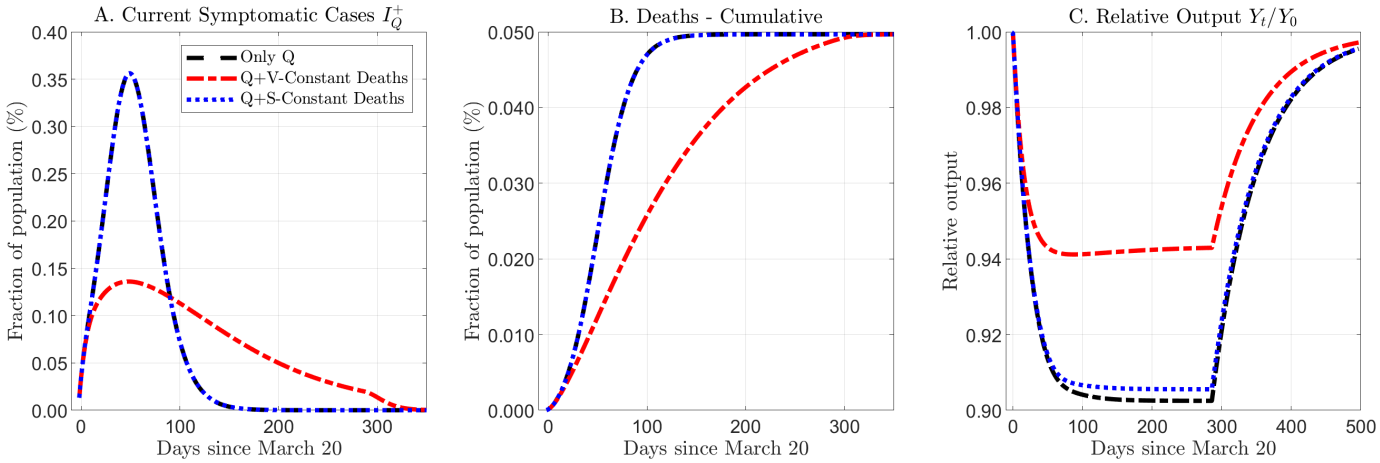

Figure A8: Shorter period of incubation: $\sigma = 1/4$ days, all other parameters held at benchmark values.

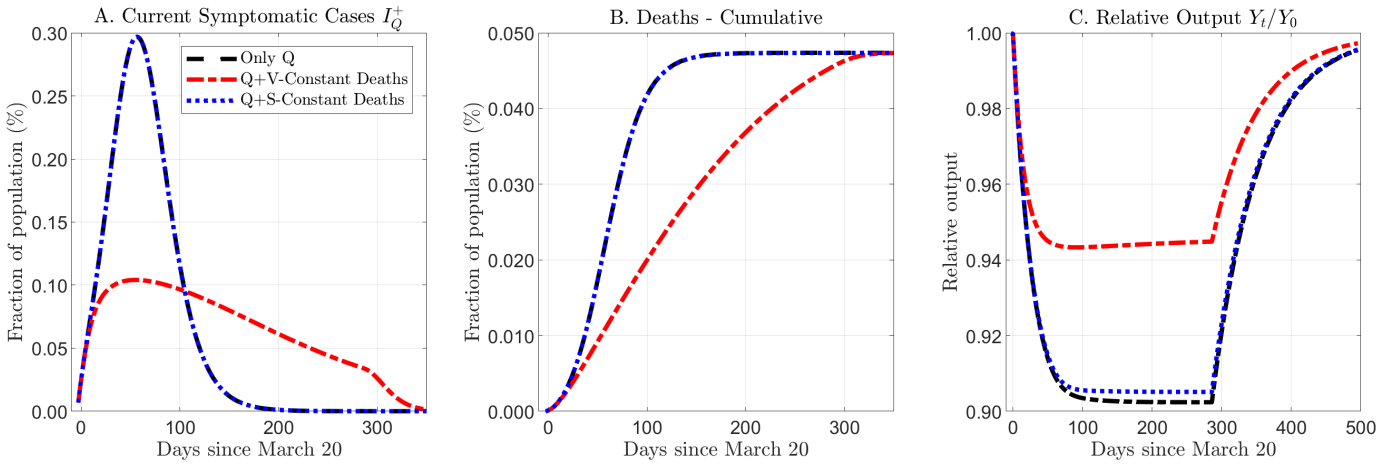

Figure A9: Longer period of incubation: $\sigma = 1/8$ days, all other parameters held at benchmark values.

6. Basic reproduction number (R_0^C): In our benchmark calibration the value of $R_0^C = 2.6$ was chosen to match the steep increase in deaths early in the virus. Figure A10 considers a lower $R_0^C = 2$, which delivers a counterfactually shallower path for total deaths. Figure A11 considers a higher $R_0^C = 3$, which delivers a counterfactually steeper path for total deaths. In both cases ω^D is recalibrated to deliver the benchmark IFR of 1 percent.

With a lower R_0^C, testing is relatively more effective as the number of infections an individual generates while asymptomatic before being tested is lower. Testing has a steeper reduction in deaths, permitting greater reopening and halving the reduction in output relative to our baseline results. With a higher R_0^C the opposite is true, although the effect appears to be highly non-linear, with the only a slight decrease in the ability to reopen and output losses that are not too much larger than our baseline results.

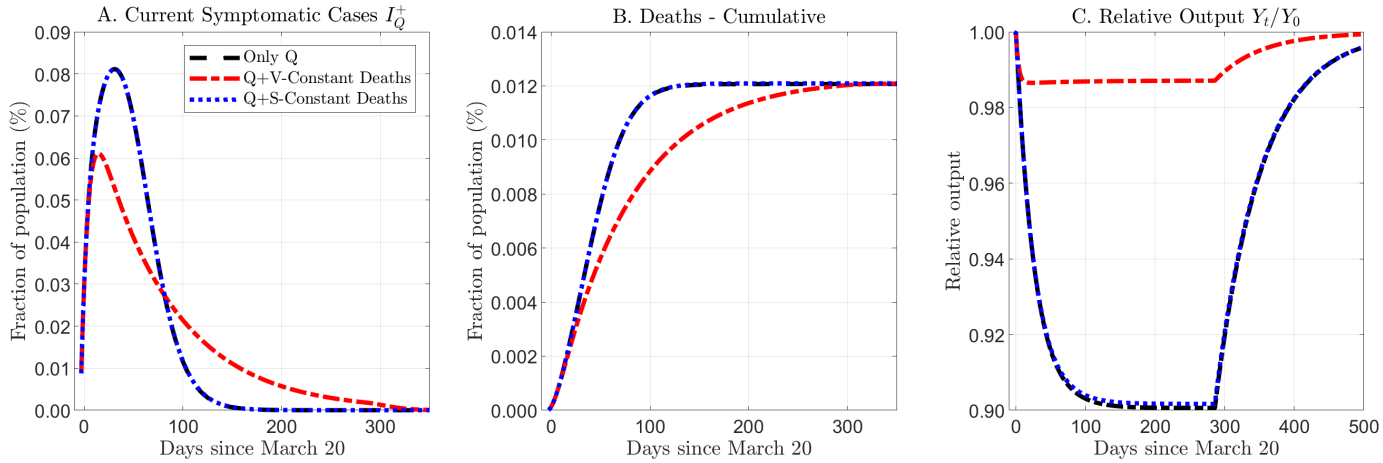

Figure A10: Lower R_0^C: $R_0^C = 2$, all other parameters held at benchmark values.

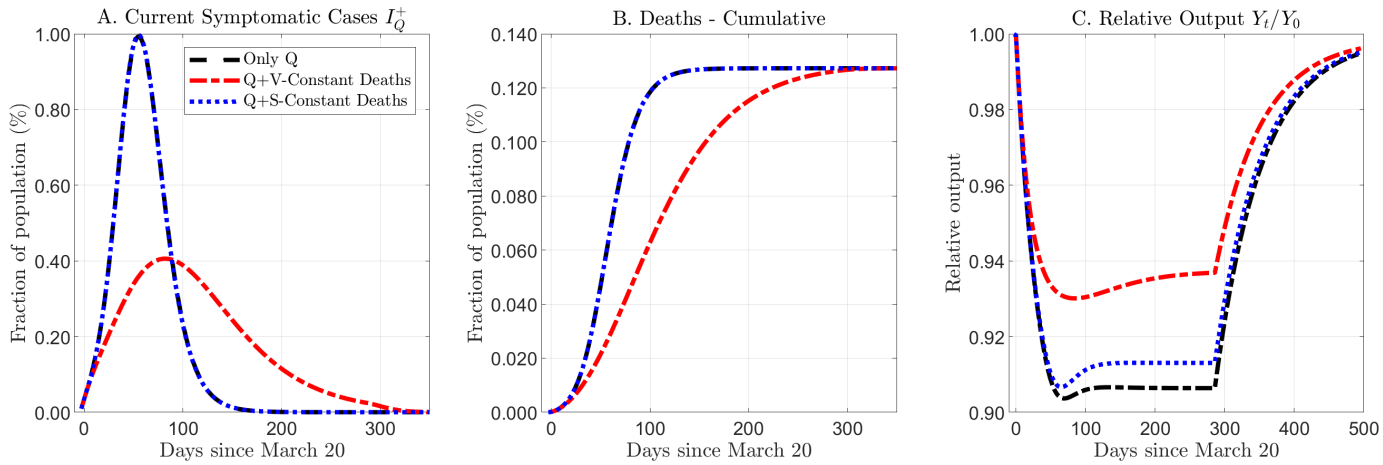

Figure A11: Higher R_0^C: $R_0^C = 3$, all other parameters held at benchmark values.

B Additional tables and figures

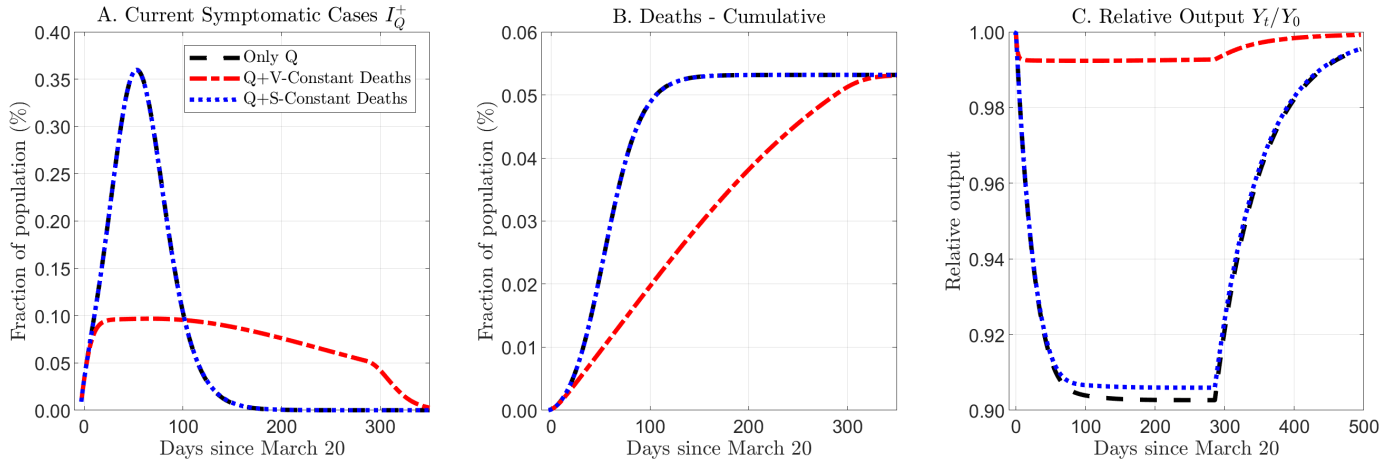

Figure B1: Testing weekly and reopening $r^u > 0$ with constant long-run deaths.