

ONLINE APPENDIX NOT FOR PUBLICATION

Testing and Reopening in an SEIR Model

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This Appendix contains two sections. Section [A](#) contains a number of key robustness exercises referenced in the main text of the paper. 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. Section [B](#) compares the model population in quarantine and that implied by stay-at-home orders in the data under different assumptions regarding the fraction of workers that are essential.

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 8 which we refer to as our *baseline set of results*. That is, (i) we compute baseline total deaths with no testing, (ii) we consider testing every two weeks, (iii) we compute the reopening rate r^U such that total deaths under each test coincides with baseline deaths with no testing.

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 8C.

- 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 benchmark no testing 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.

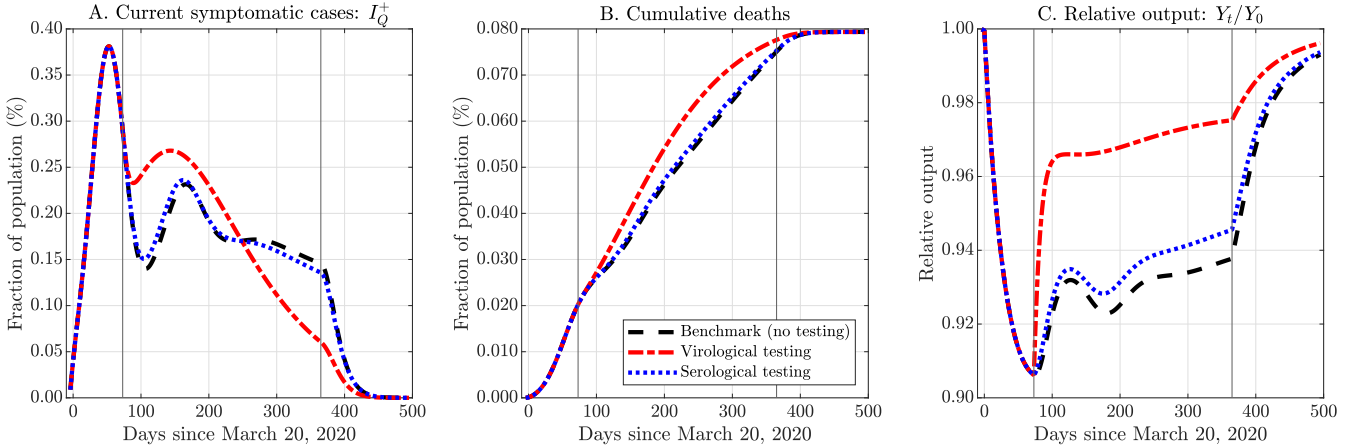


Figure A1: Lower ω^D to deliver lower IFR of 0.5%, all other parameters held at benchmark values.

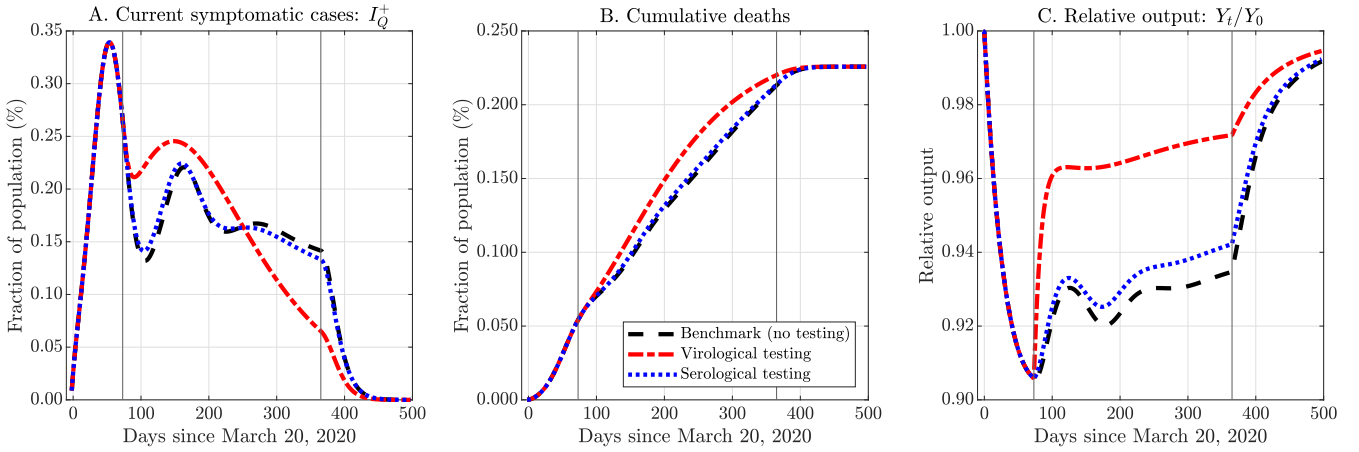


Figure A2: Higher ω^D to deliver higher IFR of 1.5%, all other parameters held at benchmark values.

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 $\rho^{A'} < \rho^A = \rho^S < \rho^{S'}$. This implies that infections are being driven by symptomatic cases which are self-quarantining and hence not leading to higher deaths. Reopening is endogenously more aggressive, yet it does not elevate R_t since the non-symptomatic cases have lower transmissibility. Testing and the reopening response leads to a *smaller* decline in output relative to our baseline set of results.

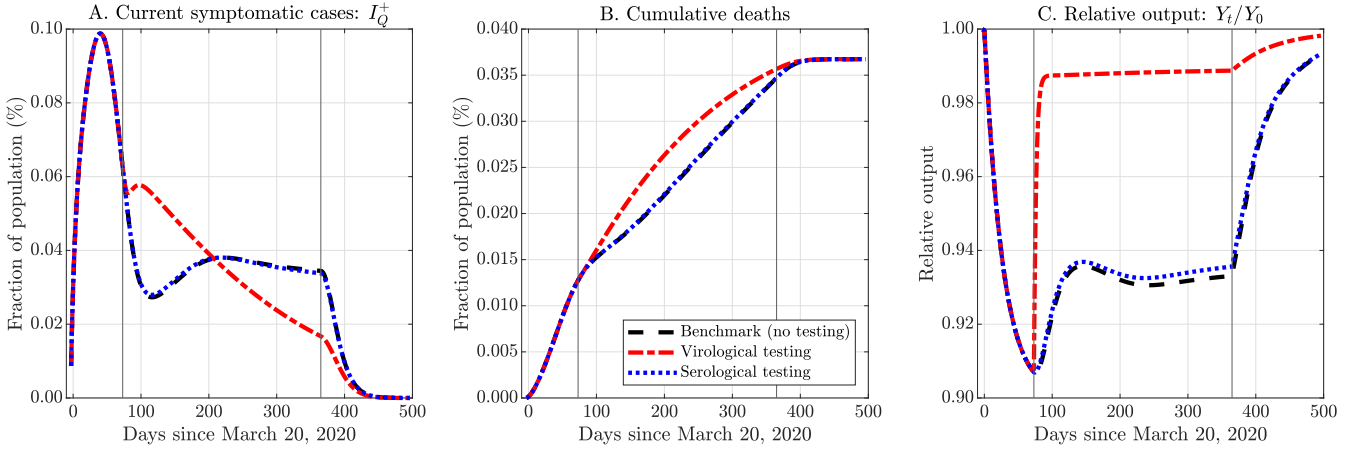


Figure A3: Parameters $\rho^A/\rho^S = 0.50$, all other parameters held at benchmark values.

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 benchmark case in Figure A5)A & B. There are more permanently asymptomatic types, that are also more infectious. The infectious cases are also more infectious, but they are quarantining. Higher levels of virological testing cannot undo this worsening of health outcomes completely.

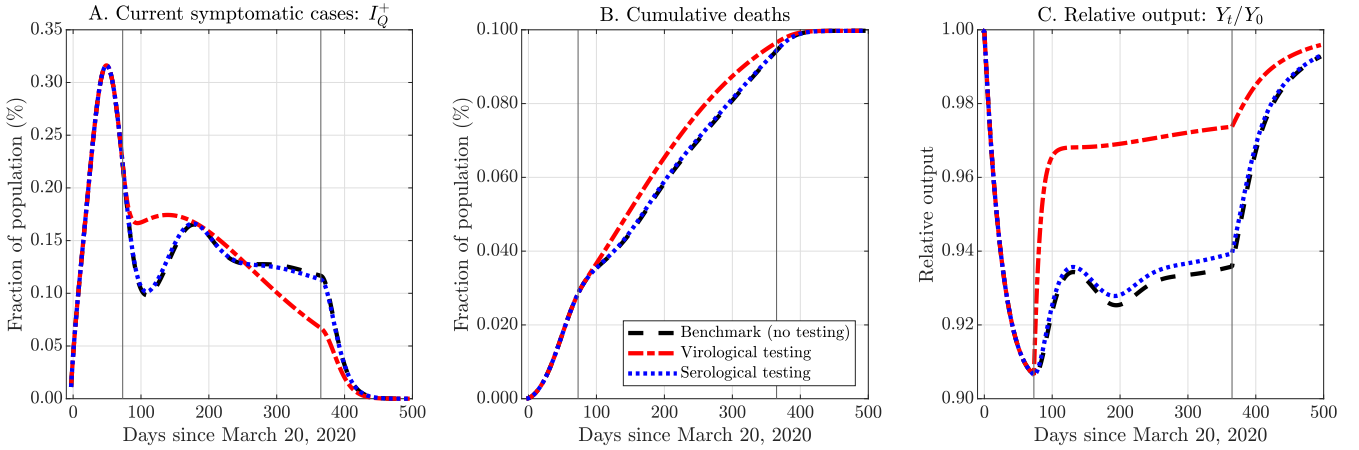


Figure A4: Lower share of permanently asymptomatic: $(1 - \psi) = 0.25$, all other parameters held at benchmark values.

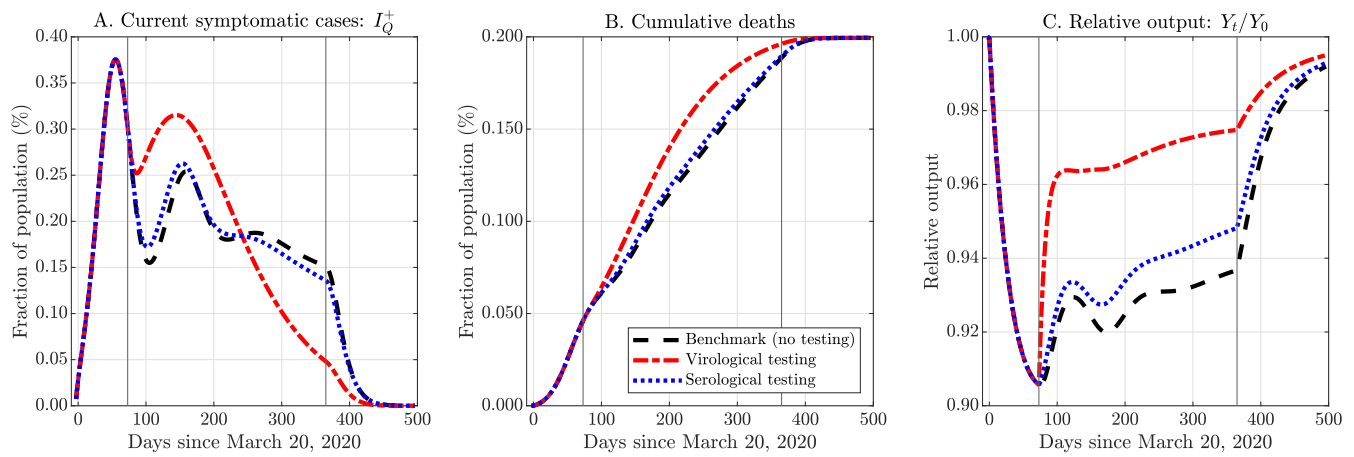


Figure A5: Higher share of permanently asymptomatic: $(1 - \psi) = 0.50$, all other parameters held at benchmark values.

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 A7 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.

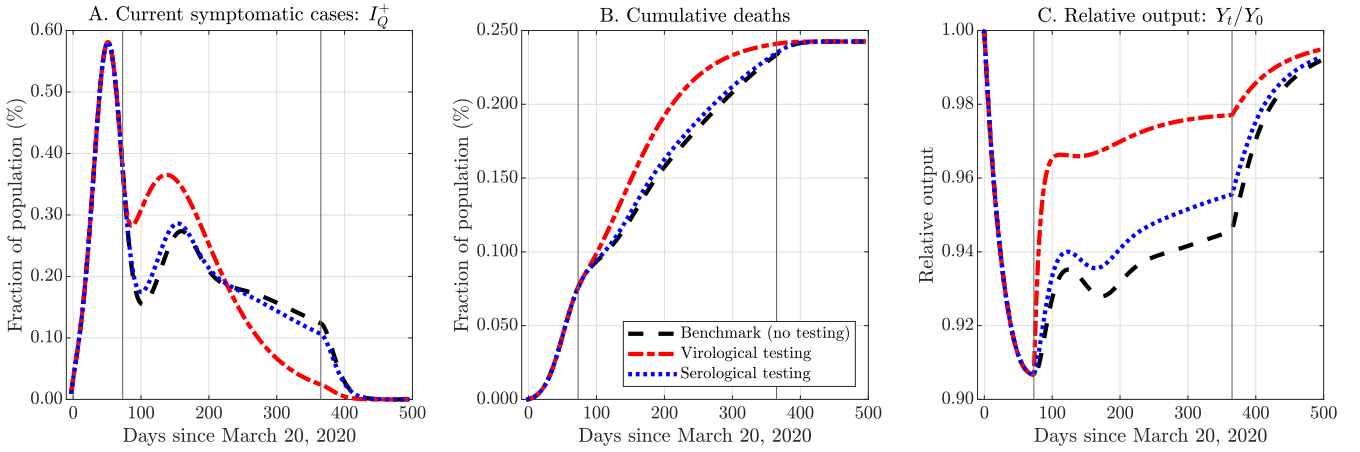


Figure A6: Shorter duration to recovery: $\gamma = 6$ days, all other parameters held at benchmark values.

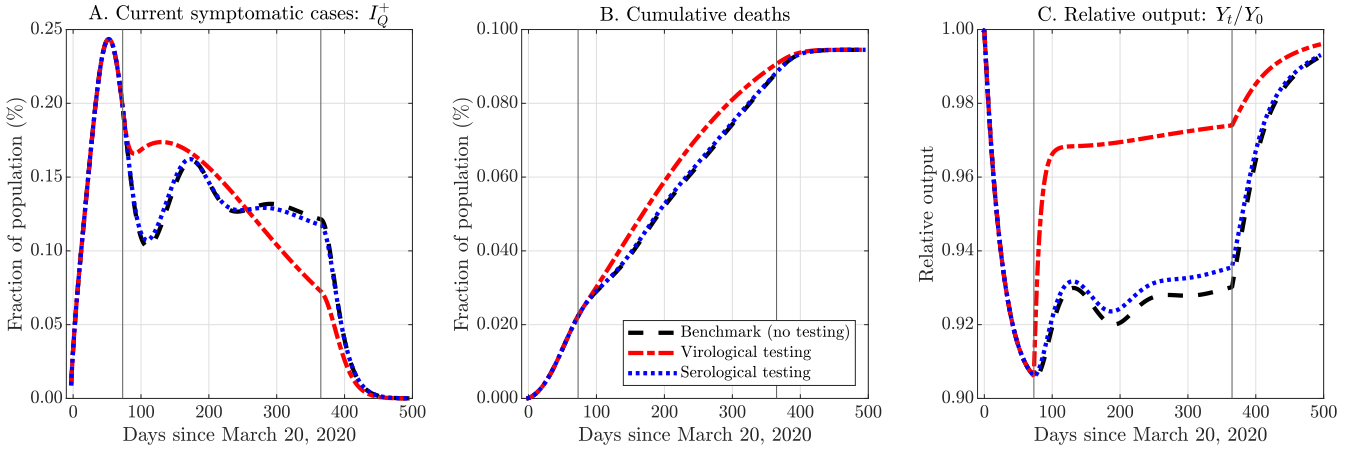


Figure A7: Longer duration to recovery: $\gamma = 10$ days, all other parameters held at benchmark values.

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.

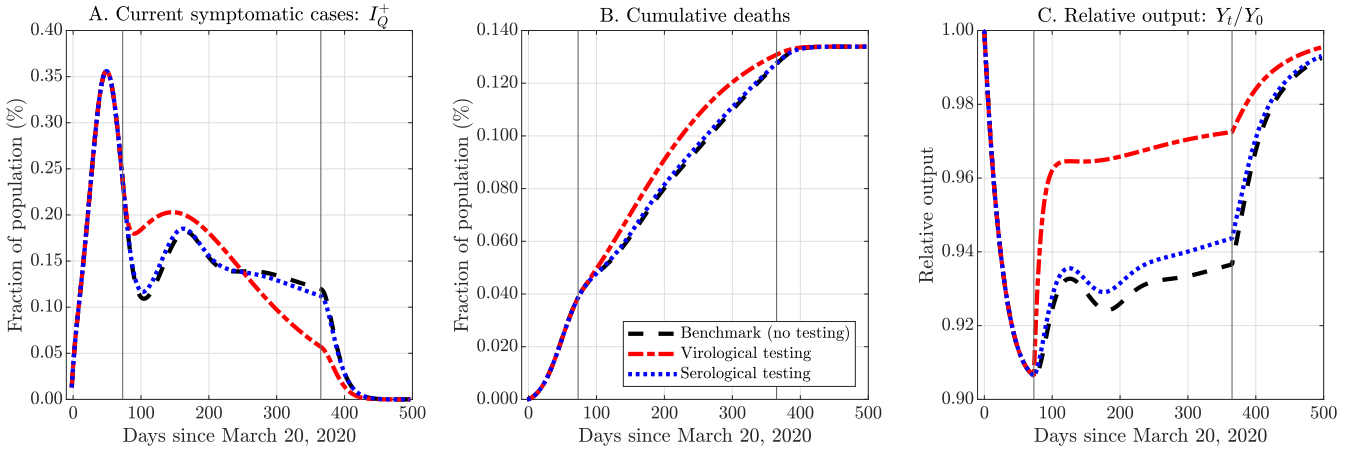


Figure A8: Shorter period of incubation: $\sigma = 4$ days, all other parameters held at benchmark values.

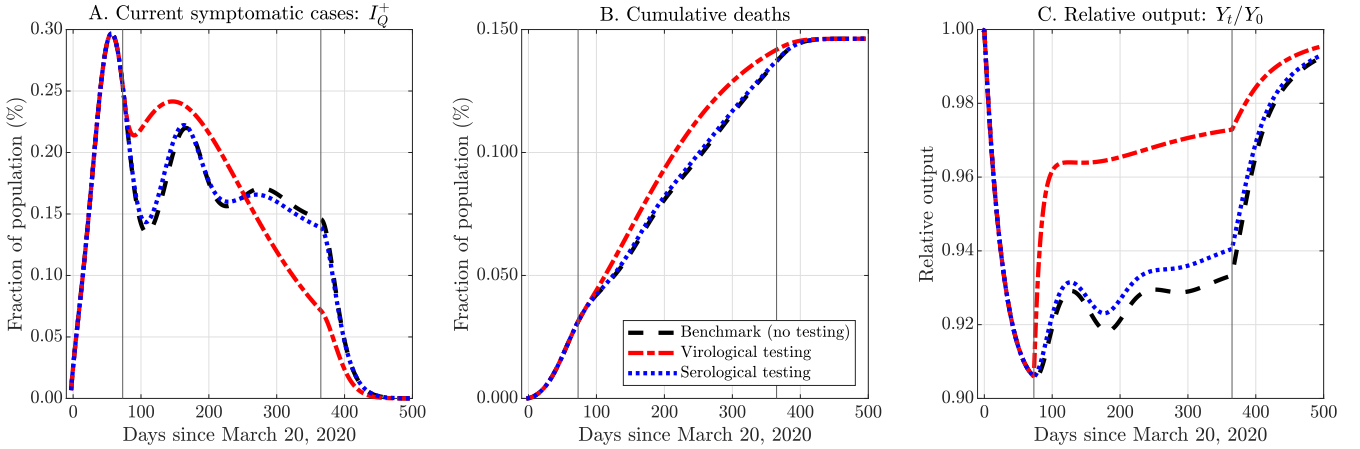


Figure A9: Longer period of incubation: $\sigma = 8$ days, all other parameters held at benchmark values.

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.

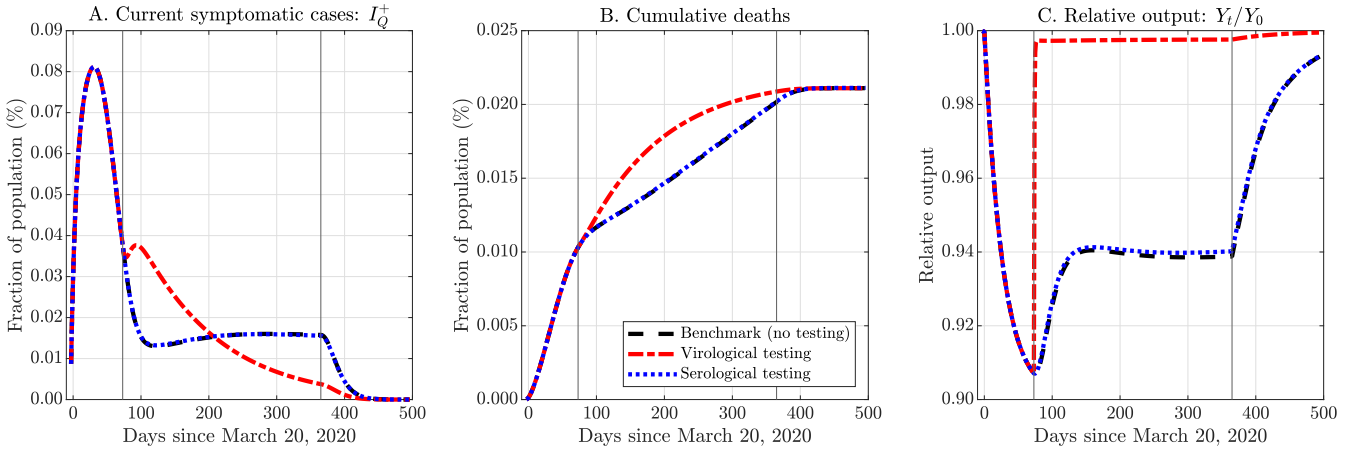


Figure A10: Lower R_0^C : $R_0^C = 2$, all other parameters held at benchmark values.

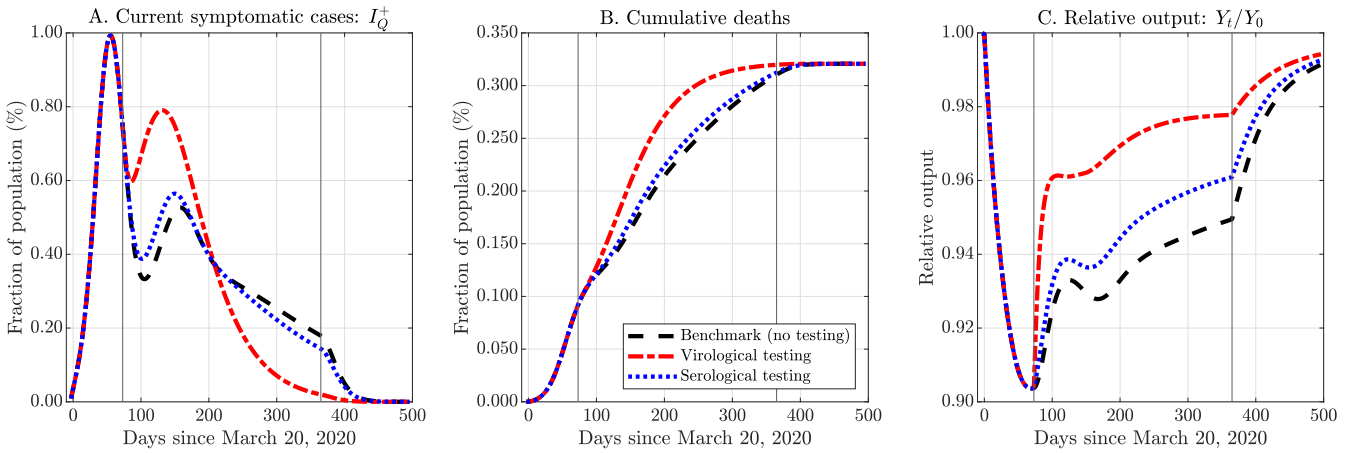


Figure A11: Higher R_0^C : $R_0^C = 3$, all other parameters held at benchmark values.

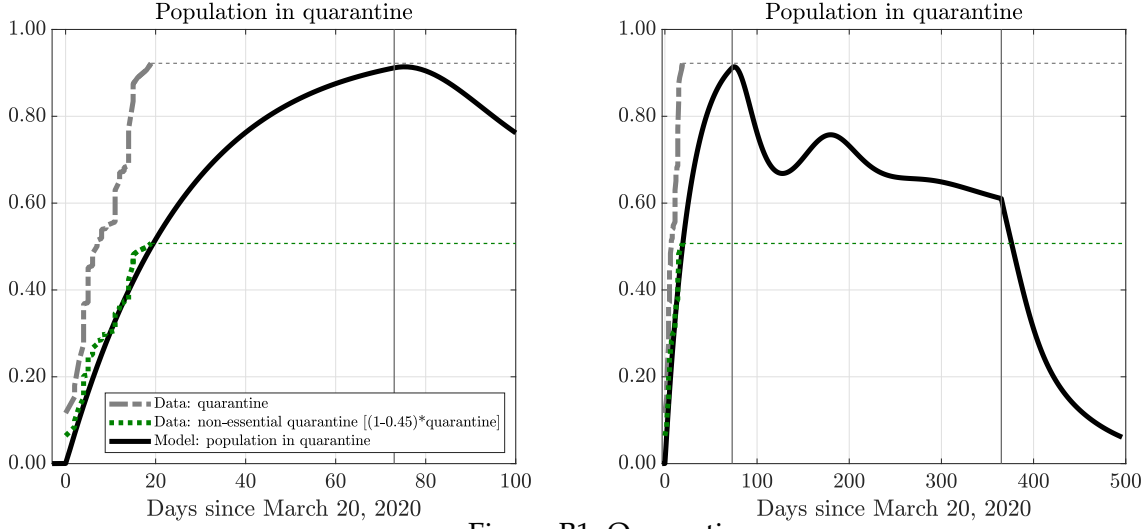


Figure B1: Quarantine

Notes: Vertical lines separate out time into the three phases discussed in the text: (i) lockdown, (ii) reopening, (iii) vaccine.

B Quarantine

Figure B1 plots two bounds of the quarantine rate. The gray dashed line represents the fraction of the U.S. population living in states with some form of stay-at-home order. We plot this measure until it peaks at 90 percent on April 7, 2020. Plotting the data beyond April 7, 2020 requires a stance on compliance with early, aggressive reopening orders. Following [Glover, Heathcote, Krueger, and Ríos-Rull \(2020\)](#), who refer to essential workers as the '*basic sector*', we plot a second line which assumes that 45 percent of workers are in the basic sector and that workers in the basic sector do not initially enter quarantine. The quarantine rate after adjusting for basic sector workers is the green dashed line in Figure B1. These two data series provide plausible bounds for the fraction of individuals in quarantine.

For the range of R_C^0 found in the medical literature, the model requires slow quarantine to match the rapid increase in deaths. By assumption, individuals continue to enter quarantine at rate ξ^u until June 1, 2020 (our reopening date). The continual entry into quarantine until June 1, 2020 is a stand-in for social learning (quarantine/social distancing among essential workers, peer pressure to comply with stay-at-home orders, etc.). Our resulting 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.