# The Testing Multiplier: Fear vs Containment

Francesco Furno[†]

January 18, 2021

**Abstract**

Existing research on the effects of testing during an epidemic outbreak has focused on its ability to slow down transmission thanks to the isolation of the infected. However, when the disease features unobservable infections, testing also informs individuals about the state of the outbreak. Here, I propose a model consistent with key empirical moments where testing affects perceptions of risk. Two insights emerge. First, small-scale testing might "stoke fear", amplify the recession and worsen public deficits. Large-scale testing, instead, successfully contains the epidemic, revives the economy and improves public finances. Second, providing disaggregated testing data so that individuals understand their age-specific death risk has considerable aggregate consequences. For a SARS-CoV-2 calibration, GDP losses and deaths are mitigated by 50% and 30%, respectively, relative to a scenario where risk perceptions are homogeneous across age groups.

[†]New York University, Department of Economics, 19 West 4th Street, 10012, New York. E-Mail: francesco.furno@nyu.edu. I am particularly grateful to Olivier Blanchard, Jaroslav Borovička, Tim Christensen and Bàlint Szőke for their comments and suggestions. This work also benefited from conversations with Valeria Ferraro, Sara Gerstner, Sebastian Hillenbrand, Pietro Monticone, Pietro Reggiani, Matheus Silva and Lena Song. I thank the NYU's High-Performance Computing Center for their support.

# 1 Introduction

There is widespread agreement in the epidemiological profession that, during an epidemic outbreak, testing can help contain the spread of the disease and improve health outcomes thanks to the selective isolation of the infected - see Murray (2020). Along the same lines, the emerging economic literature on testing has focused on its ability to slow down epidemic spread in environments where economic activity almost perfectly correlates with the true size of the infection. In such a setting, testing leads to improved economic outcomes virtually by construction.[1]

However, whenever the epidemic disease features unobservable infections, testing also plays the key role of providing the population with real-time information about the outbreak. This is because testing activity is what allows individuals to assess the *current* state of the epidemic, which is otherwise unobservable. Ignoring this channel might therefore leave sizeable adverse economic consequences of testing activity out of the analysis.

In this paper, I develop an epidemiological model where testing permits selective isolation and informs agents about the risk of infection and the lethality of the novel emerging disease. In the model, all individuals with severe symptoms are always tested and the government decides how many screening tests to perform on the rest of the population. Additional testing widens the range of tested symptoms and mechanically reduces the perceived lethality of the disease, but *can* result in a higher number of detected cases, thereby increasing the perceived risk of infection. Whenever additional testing increases the perceived risk of infection enough that the overall perceived risk of death also increases, it "scares" the population further and compounds the ongoing contraction of economic activity.

The first insight that emerges from the analysis is that testing activity performed at a small-scale can amplify the recession and worsen public deficits. Indeed, screening for infection a small share of the population every day is not enough to successfully contain an epidemic, but would still unveil a larger portion of it. Depending on the properties of the disease and on luck, this could increase the perceived risk of death in the population, cause a further contraction of economic activity and widen public deficits. Interestingly, the fallout in economic activity would occur despite improved health outcomes. Conversely, when a sizeable share of the population is screened daily, epidemic containment succeeds and the perceived risk of death always decreases, improving economic outcomes as well.

The second insight highlights the importance of information provision in situations where the epidemic disease is more lethal for certain segments of the population than for others. Specifically, I extend the model to introduce two age groups ("young" and "old") and calibrate it to the U.S. and SARS-CoV-2, which features a steep risk-gradient across age groups. I consider two benchmark scenarios. In the first, all individuals are assumed to have homogeneous risk perceptions because the government releases only aggregate testing data. In the second, different age groups have het-

[1] It is usually assumed that agents either know the aggregate state of the epidemic or react to observables that are not produced by testing activity. See Eichenbaum et al. (2020c) and Atkeson et al. (2020) for an example of each, respectively.

erogeneous risk perceptions because they can construct age-specific estimates of disease lethality from disaggregated testing data made available by the government. Relative to the homogeneous case, heterogeneous risk perceptions vastly improve aggregate economic and health outcomes, because old agents - who are the most likely to die - protect themselves more while young agents - who generate most of GDP - protect themselves less and return to work.

These two insighs have important implications for policy-making. Arguably, the main one is that large-scale testing programs are likely to be cheaper and produce better economic and health outcomes than small-scale ones. They eliminate not only the trade-off between health and economic activity, but also the one between these two and public finances. Another implication is that - at least for a disease such as SARS-CoV-2 - governments around the world can considerably improve both economic and health outcomes by providing disaggregated data and ensuring that different age groups correctly understand their own age-specific risk.

The model can be summarized as follows. Economic activity is mainly a function of the perceived risk of dying from the epidemic disease, and risk perceptions are constructed using testing data on total cases, active infections and deaths. The testing policies in the model mimic those adopted by health-care systems around the world and are able to match important features of the data - as I show in subsection 5.2. Additional testing systematically improves health outcomes (i.e. it reduces infections and deaths), but has a non-monotone effect on risk perceptions. Indeed, higher testing activity may or may not translate into a higher perceived risk in a dynamic setting: more testing reduces the perceived lethality of the disease, reduces the total number of infections, but reveals a larger share of the epidemic. As a result, the relationship between testing and risk perceptions is highly non-linear.

Risk perceptions are introduced as follows. Agents do not know the true epidemiological process, and testing data is the only source of information about the risk posed by the disease. The risk of dying is given by the product of the probability of dying conditional on infection and the probability of infection. Following the epidemiological approach to an *unknown* disease, I assume that its lethality is assessed with the *case fatality rate*, given by total detected deaths divided by total detected cases. The perceived probability of infection is instead proportional to detected active infections per capita, as a standard epidemiological model - such as the SIR - would suggest. In section 3, I show that this specification is consistent with empirical evidence from U.S. states and counties.

Real-time risk perceptions in the model can systematically differ from the truth, and depend on the level of testing activity. Since it generally takes months or years to produce reliable estimates of the lethality of a novel disease, the case fatality rate necessarily becomes the first best assessment. When there are mild or asymptomatic infections, however, the case fatality rate tends to overestimate the true lethality because real-world testing policies prioritize testing of individuals with severe symptoms, and infected individuals with mild or no symptoms that are less likely to die are not tested. As a result, the perceived lethality of the disease heavily depends on the amount of testing performed, especially in the early stages of the outbreak.

Moreover, agents struggle to estimate the true number of unobservable active infections in real-time without large-scale testing, partly because infections are unobservable and partly because observable variables are not enough to provide correct estimates in real-time. Individuals' ability to estimate the risk of infection therefore relies heavily on the amount of testing performed. In subsection 2.1, I provide a deeper discussion of these issues.

The paper is organized as follows. In section 2, I discuss risk perceptions. In section 3, I assess the empirical relevance of my proposed measure of risk. I present the model in section 4, and provide extensive simulations to better understand its mechanisms in section 5. I define the testing multiplier in section 6, and simulate it for various parameterizations. In section 7, I explore the importance of heterogeneous risk perceptions across age groups.

**Existing Literature** This paper contributes to a fast-growing literature in economics that analyzes the interplay between epidemics and the economy. In terms of its methodological approach, this paper starts from an epidemiological model and extends it with a simple economic component, similar in spirit to Berger et al. (2020), Piguillem and Shi (2020), Taipale et al. (2020), and Atkeson et al. (2020) who also examine the economic and health benefits of testing. In none of these papers, however, does testing provide agents with information about the aggregate state of the epidemic.[2] A complementary approach is to start from a macroeconomic model and extend it with a stylized epidemiological component, as in Eichenbaum et al. (2020b) and Jones et al. (2020). Within this strand of the literature, testing directly affects individual behavior in both Brotherhood et al. (2020) and Eichenbaum et al. (2020c) by resolving uncertainty regarding individual health status, but does not provide information regarding the aggregate state of the epidemic.

In the epidemiological literature, several papers attempt to endogenize behavior in response to "fear" of the disease - see Funk et al. (2010), Verelst et al. (2016) and Wang et al. (2015) for an overview. All these papers assume that agents' behavior reacts to information from various sources, but - to the best of my knowledge - no paper in this literature assumes that agents react to information coming from testing activity.

The empirical evidence in this paper also echoes the findings in Goolsbee and Syverson (2020) that economic activity during an epidemic outbreak falls irrespective of non-pharmaceutical interventions by policy-makers, and the insight in Eichenbaum et al. (2020a) that the probability of dying is a key determinant of individual behavior.

[2] Furthermore, these papers adopt a compartmental modeling strategy which results in better tractability by permitting the aggregation of individuals into epidemiological compartments. I adopt an agent-based framework, which increases complexity but allows me to introduce more realistic testing policies and a more refined modeling of the epidemic disease. For a discussion of compartmental vs agent-based epidemiological models see Murray (2020), Sukumar and Nutaro (2012), Hunter et al. (2018), and Gallagher and Baltimore (2017).

## 2 Perceptions of Risk

Throughout this paper, I assume that the relevant measure of "fear" of an epidemic disease is given by the probability of dying from it:[3]

$$\text{Prob(Death)} = \text{Prob(Death|Infection)} \times \text{Prob(Infection)}$$

where the probability of death ("death risk") is given by the product between the conditional probability of death given infection ("disease lethality" or "infection fatality risk") and the probability of infection ("infection risk").

To see why the probability of dying is the relevant object, consider the following. Imagine first a widely spread epidemic disease which is completely harmless. This would result in a high infection risk but a null disease lethality, implying a null death risk, and thus no fear of the disease. Consider next a very deadly disease which is impossible to catch. This would imply a null infection risk and a null death risk, and thus no fear of the disease.

Since these probabilities are unknown, I assume that individuals attempt to estimate them in real-time using testing data. Specifically, agents look at the case fatality rate to estimate the risk of death conditional on infection:

$$\text{Perceived Lethality}_t = CFR_t = \frac{D_t}{C_t}$$

where $CFR_t$ is the case fatality rate, and $C_t$ and $D_t$ are cumulative cases and deaths reported by the health-care system. To assess the average infection risk in the population, instead, I assume that individuals follow a standard textbook epidemiological model - such as the SIR model - which posits that the probability of infection is proportional to the number of active infections over the population:

$$\text{Perceived Infection Risk}_t = IR_t = \beta \times \frac{I_t}{P_t}$$

where $\beta$ is the transmission coefficient of the disease (which summarizes its contagiousness), $I_t$ is the number of current infections detected by the health-care system, and $P_t$ is the alive population.[4] The perceived risk of death - which will be denoted with the variable $\chi_t$ - can then be re-constructed as follows:

$$\chi_t = \underbrace{CFR_t}_{\text{Perceived Lethality}} \times \underbrace{IR_t}_{\text{Perceived Infection Risk}}$$

In the empirical analysis presented in section 3, I show that this measure of perceived risk predicts

[3] Several epidemiological models with behavioral responses assume that individuals react to the prevalence rate, given by the ratio of cases to the population, a measure of infection risk - see Funk et al. (2010) for a review. In the economics literature, instead, it is usually assumed that behavioral responses depend on the number of deaths - see Kaplan et al. (2020) and Atkeson et al. (2020) for an example.

[4] In standard textbook epidemiological models, active infections - as opposed to cumulative infections - are what matters for transmission because, for most epidemic diseases, individuals who recover or die are no longer infectious.

precisely and robustly economic activity across U.S. states and counties during the SARS-CoV-2 outbreak.

### 2.1 Further Discussion of Risk Perceptions

As mentioned in the introduction, real-time risk perceptions in the model can systematically differ from the truth and it is useful to think about two wedges between perceptions and reality to see why.

The first wedge captures the fact that the perceived probability of dying conditional on infection might exceed the true one. Indeed, whenever the disease features a large share of sub-clinical infections (i.e. not requiring medical attention) that go undetected with narrow testing policies, the denominator of the case fatality rate is under-estimated, and therefore the lethality of the disease is over-estimated - see Lipsitch et al. (2015) for a discussion. This issue is not easily solved even when combining available testing data with epidemiological theory, because of the identification problems outlined in Atkeson (2020) and Korolev (2020) among others. Large-scale testing is a way to solve the problem since it reveals sub-clinical infections that can be then included in the total case count. Another way to solve the problem is to perform a one-off large-scale random experiment, with either a virological test (which detects an active infection) or a serological survey (which detects past infections). The problem is that, for a variety of reasons, these interventions usually take time to be performed - if ever performed. In principle, the wedge between the true lethality of the disease and the perceived one could thus be eliminated even without large-scale testing. In practice, this either takes time or never occurs, forcing individuals to rely on testing data to assess the lethality of the disease.

The second wedge relates to the probability of infection, and arises because agents struggle to estimate it in real-time. This is due to the fact that the risk of infection depends on the number of *current* infections, which is more difficult to estimate than the total number of *past* infections without large-scale testing. For example, a large-scale serological survey can provide a very accurate estimate of the number of infections in the past, but has little to say about current infections. Similarly, observable epidemiological variables such as deaths or hospitalizations are not helpful because they contain information about past infections - as opposed to current infections. For instance, suppose that at some point during the outbreak a certain number of deaths is observed.[5] Without knowledge of the true probability of dying conditional on infection, one is not able to estimate how many infections produced those deaths. Even when these probabilities are known, deaths today are the result of infections days or weeks ago. A similar reasoning holds for hospitalizations. These considerations suggest that the assessment of infection risk is heavily dependent on testing data, exactly as in the specification of beliefs that I propose.

Further support to this view comes from the recent work by Chande et al. (2020). In their paper on the SARS-CoV-2 outbreak in the U.S., the authors construct a location-specific real-time assessment of infection risk using *"recent case reports multiplied by an ascertainment bias informed by*

[5] In practice, not even epidemic deaths are correctly observed unless testing activity is performed at a sufficient level.

*serological surveys"*. In other words, they use testing data on new infections and scale them up by a factor given by the number of cases detected with serological surveys over the number of cases detected by the health-care system through testing. Since the serological surveys are conducted infrequently, daily variation in the estimated infection risk comes exclusively from testing activity. In Online Appendix D.4, I propose an alternative specification of beliefs that mimics this methodology. Agents eventually estimate both the true lethality of the disease and the true number of total cases correctly, but still fail to estimate the number of active infections in real-time without large-scale testing. As a result, my findings are reproduced with this alternative specification as well.

## 3 Fear and Economic Activity: Evidence from the U.S.

To investigate the relationship between the perceived risk of death and economic activity, I combine weekly data on economic activity with testing data on reported cases and deaths across U.S. states and counties during the first stages of the SARS-CoV-2 epidemic outbreak . My preferred proxies of economic activity are the Dallas FED's Mobility and Engagement Index (MEI) and the Google Workplace Mobility Report because of their high-frequency and granularity. The perceived risk of death in location $i$ during week $t$ is given by:

$$\chi_{i,t} = \underbrace{\frac{D_{i,t}}{C_{i,t}}}_{\equiv CFR_{i,t}} \times \underbrace{\beta \cdot \frac{I_{i,t}}{P_{i,0}}}_{\equiv IR_{i,t}}$$

Data on reported total cases ($C_{i,t}$), total deaths ($D_{i,t}$) and population ($P_{i,0}$) come from USA Facts, which are available at a county-level and can be easily aggregated up to the state-level. To estimate reported active infections ($I_{i,t}$), I take new reported cases over a 14-day horizon, although the results are robust to the time window considered. Finally, I assume that the transmission coefficient used to construct the perceived infection risk is $\beta = 0.30$.[6] My dataset stretches from January 2020 to September 2020, and all the details can be found in Online Appendix A.1.

Figure 1 reveals that a higher perceived risk of death is associated with falls in economic activity across U.S. states. However, this is not enough to establish causality for at least two reasons. First, reverse causality might be at play. Second, there might be omitted variable bias: a higher perceived death risk calls for lockdowns and similar non-pharmaceutical interventions, which produce a contraction of economic activity.

[6] Given that the coefficient is constant across time and space, this assumption does not affect the standardized estimated coefficients.

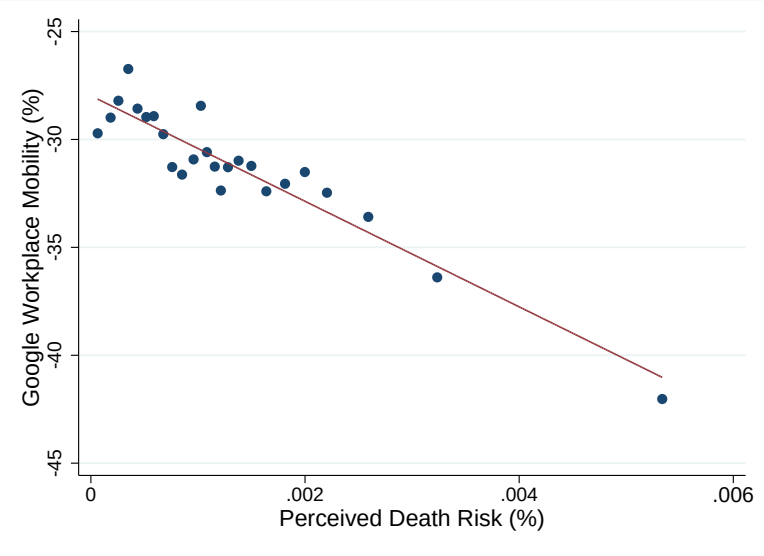


**Figure 1:** Correlation between Perceived Risk and Economic Activity during SARS-CoV-2 in the U.S.
**Notes**: The plot considers the period from 1 May 2020 to 1 September 2020 in order to leave out most of early lockdowns, business closures and similar interventions.

Reverse causality is unlikely to be an issue. SARS-CoV-2 is a disease characterized by lengthy lags between exposure and development of symptoms and/or hospitalization. Given the relatively narrow testing policies adopted in the U.S. during the first phase of the pandemic, the vast majority of detected infections were diagnosed after the appearance of symptoms or even after hospitalization, implying that a new infection was likely to be recorded by the health-care system with a sizeable delay. This implies that economic activity in a given week is likely to increase perceived death risk only in the future. Furthermore, reverse causality would suggest a positive relationship between economic activity and perceived risk, instead of a negative one.

Concerns about omitted variable bias are harder to dissipate. Policy-makers are likely to monitor epidemiological developments and respond to them promptly by implementing lockdowns and other containment policies. To control for such confounders, I employ fixed-effect regression models.

Table 1 reports my regression results at the state-level.[7] The first three columns report the regression results using the FED's MEI measure as dependent variable, while the last three using the Google Workplace Mobility measure. Furthermore, I consider two specifications: one in which I regress economic activity on perceived death risk, and one in which I replace perceived death risk with its two components, namely perceived lethality and perceived infection risk.

[7] All coefficients are standardized. The non-standardized estimates can be found in Online Appendix A.2.

| | FED's MEI | | | Google's Workplace Mobility | | |
|---|---|---|---|---|---|---|
| | (1) OLS | (2) FE | (3) FE | (1) OLS | (2) FE | (3) FE |
| **Spec #1** | | | | | | |
| *Death Risk ($\chi$)* | -37.34*** | -38.19*** | -11.60*** | -43.98*** | -49.46*** | -9.88*** |
| | (3.95) | (3.49) | (1.96) | (4.84) | (5.20) | (1.50) |
| **Spec #2** | | | | | | |
| *Lethality (CFR)* | -36.41*** | -37.76*** | -3.38 | -45.60*** | -50.28*** | -3.44 |
| | (6.10) | (8.30) | (2.29) | (7.48) | (10.74) | (2.10) |
| *Infection Risk (IR)* | -18.81*** | -18.80*** | -10.39*** | -31.05*** | -34.66*** | -9.82*** |
| | (5.16) | (5.48) | (1.80) | (3.92) | (3.78) | (1.28) |
| State FE | N | Y | Y | N | Y | Y |
| Time FE | N | N | Y | N | N | Y |
| Adj. $R^2$ (Spec #1) | 0.15 | 0.17 | 0.96 | 0.20 | 0.25 | 0.97 |
| Adj. $R^2$ (Spec #2) | 0.19 | 0.21 | 0.96 | 0.31 | 0.37 | 0.97 |
| Obs | 1530 | 1530 | 1530 | 1479 | 1479 | 1479 |

**Notes:** Clustered standard errors at the state-level in parenthesis. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. Standardized coefficients (%) obtained by scaling variables by their standard deviation.

**Table 1:** Main Regression Results at the State-Level

The estimates suggest a strong negative relationship between economic activity and perceived death risk, which holds also when the latter is decomposed into its two components. Importantly, the estimated effect is economically meaningful: perceived death risk explains between 35% and 50% of the fall in economic activity when time fixed-effects are excluded, and roughly 10% of the relative fall in economic activity when time fixed-effects are included. State fixed-effects ensure that unobserved heterogeneity in economic activity across states is properly accounted for. Time fixed-effects control for unobserved national developments common across states and countries, such as national containment guidelines, nation-wide communications from policy-makers and so on. The state-level estimates, however, might still suffer from omitted variable bias since they do not control for state-level developments that occur over time and might correlate with both economic activity and perceived risk.

Table 2 reports my regression results at the more granular county-level. This allows me to introduce state-time fixed-effects which absorb state-level developments over time. As the vast majority of lockdowns and containment policies during the first phase of the epidemic outbreak were enacted at a state-level, the state-time fixed-effects should be able to solve any omitted variable bias.[8] The county-level estimates remains negative and statistically significant across all specifications, and the same is true when perceived death risk is decomposed into its two components. The coefficients become smaller as fixed-effects are included, suggesting that the latter are successfully controlling for unobservables. Small coefficients could be due to the importance of local factors to

[8] Goolsbee et al. (2020) construct a dataset of stay-at-home and business closure orders for the first months of the epidemic outbreak. County-level lockdowns are highly correlated with state-level ones, although not perfectly. Moreover, in late 2020 some states started to implement local lockdowns and stay-at-home orders, at a level as granular as the zip-code. This would invalidate the proposed identification for more recent data.

explain local economic activity, but also to the fact that the proposed measure of perceived death risk is only an approximation - and this becomes clearer at a more granular level.[9] Nonetheless, the negative effect of perceived risk on economic activity appears clear.

| | FED's MEI | | | | |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| | OLS | FE | FE | FE | FE |
| **Spec #1** | | | | | |
| *Death Risk ($\chi$)* | -10.13*** | -9.95*** | -4.67*** | -4.19*** | -4.19** |
| | (0.93) | (0.79) | (0.40) | (0.64) | (1.65) |
| **Spec #2** | | | | | |
| *Lethality (CFR)* | -16.16*** | -18.67*** | -1.05*** | -1.91*** | -1.91*** |
| | (1.04) | (1.13) | (0.27) | (0.51) | (0.53) |
| *Infection Risk (IR)* | -2.45*** | -2.14*** | -4.05*** | -3.05*** | -3.05** |
| | (0.58) | (0.50) | (0.65) | (0.67) | (1.21) |
| County FE | N | Y | Y | N | N |
| Time FE | N | N | Y | N | N |
| State-Time FE | N | N | N | Y | Y |
| SE Clustering | County | County | County | County | State |
| Adj. $R^2$ (Spec #1) | 0.01 | 0.14 | 0.90 | 0.81 | 0.81 |
| Adj. $R^2$ (Spec #2) | 0.03 | 0.16 | 0.90 | 0.81 | 0.81 |
| Obs | 90599 | 90599 | 90599 | 90599 | 90599 |

**Notes:** Clustered standard errors in parenthesis. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. Standardized coefficients (%) obtained by scaling variables by their standard deviation.

**Table 2:** Main Regression Results at the County-Level

**Additional Empirical Results** A question that naturally arises is how the proposed measure of death risk compares to alternative measures that have been adopted in the literature, such as total or weekly cases and deaths. Table A3 in Online Appendix A.2 provides a tentative answer. Overall, the estimates suggest that the proposed measure of risk remains precise and robust even when detected cases and deaths are controlled for.

Another important question is whether the level of testing itself has any effects on economic activity. Indeed, one could argue that more testing reduces agents' uncertainty about the accuracy of reported cases and deaths, and that less uncertainty is beneficial to economic activity. In this respect, an indicator which is frequently monitored to assess how much testing is performed relative to the true latent epidemic is the test positivity rate.[10] The results are reported in Table A4 in Online Appendix A.2. The test positivity rate exhibits a significant negative relationship with economic activity, in line with the previous conjecture. Interestingly, however, when the proposed measure of perceived risk is included in the regression, the estimated effect of the test positivity rate becomes indistinguishable from zero, and the magnitude of the estimated standardized coef-

[9] For example, different individuals will perceive death risk differently depending on a wide set of covariates, which could systematically differ across counties.

[10] Testing data come from the COVID Tracking Project, and the test positivity rate is defined as the number of new detected cases over the number of tests performed in a given time period. Due to data limitation, I limit myself to the construction of the test positivity rate at the state-level.

ficient is almost an order of magnitude lower than that of the perceived death risk.

# 4 A Stochastic Epidemiological Model with Fear

The model is agent-based, i.e. each agent is modeled individually, and features two diseases: a novel emerging epidemic disease and an endemic confounding disease.[11] The role of the confounding disease is literally to confound the diagnosis of the epidemic disease, since individuals exhibiting symptoms might be infected with either disease.

Testing policies in the model mimic real-world ones which prioritize testing of severe symptomatic individuals, and play two important roles. First, detected active infections are put into (imperfect) isolation, allowing the government to slow down epidemic transmission. Second, testing provides agents with information about the latent epidemic disease. More precisely, they use reported cases, active infections, and deaths to construct a measure of death risk which embodies the familiar notion of fear. A higher perceived risk of death stokes fear and prompts a reduction in labor supply, causing a fall in economic activity - consistently with the empirical part of the paper.

I assume that all severe symptomatic individuals are always tested by the health-care system and that the government is left with the choice of testing non-severe symptomatic individuals, i.e. those with mild symptoms or no symptoms at all. This is also referred to as "screening" of the population for infections that are not otherwise observable.

## 4.1 Aggregate Epidemic Dynamics

Time is discrete, each time period is interpreted as a day, and the population is studied over an horizon $T$. Consider a homogeneous population of ex-ante identical individuals with initial size $P_0$, and suppose that no individual is added to the population (e.g. no births, no immigration). There are two diseases circulating in the population: the epidemic disease and a confounding disease. The latter is an *endemic disease* which circulates in the population irrespective of the epidemic disease and is named "confounding" because it confounds the diagnosis of the epidemic diseases due to the fact that infected individuals share similar symptoms across the two diseases. I assume the following:

**E1:** *For each disease, individuals who recover obtain immunity.*
**E2:** *Each individual can catch only one of the two diseases.*

Assumption **E1** is often adopted in epidemiological models - since most epidemic diseases offer at least a temporary immunity after recovery - and simplifies the problem from a modeling perspective. Assumption **E2** is a simplification that allows to abstract from what happens when an agent catches both diseases.

[11] From a technical viewpoint, the model presented in this paper is a generalization of standard epidemiological models, and nests the most common ones as special cases. For the sake of illustration, I show in Online Appendix C how to recover the textbook deterministic SIR model.

From now on, latent variables will be denoted with an asterisk and observable ones without. At any point in time, each individual $j$ can find themselves in one of three states:

$$x_t^*(j) + c_t^*(j) + c_T^{f*}(j) = 1$$

where $x_t^*(j)$ denotes susceptibility to the epidemic disease and takes value 1 if individual $j$ has never contracted any disease at time $t$ and will never contract the confounding disease over the time horizon considered. The variable $c_t^*(j)$ takes value 1 if the individual has contracted the epidemic disease at time $t$ or before, while $c_T^{f*}(j)$ takes value 1 if the individual has ever contracted the confounding disease over the time horizon considered.

Given that the confounding disease is an endemic disease, I model new aggregate cases each day as an exogenous stationary process:

$$\Delta C_t^{f*} \sim Normal\left(\frac{\omega^f \cdot P_0}{T}, \left(\sigma^f \cdot \frac{\omega^f \cdot P_0}{T}\right)^2\right)$$

where realizations are rounded to the nearest integer. Notice that $\omega^f$ is the share of the population that on average contracts the confounding disease over the time horizon $T$. So for instance, if $T = 90$ and $\omega^f = 0.20$, then on average twenty percent of the initial population contracts the infection over a 90 day period. Moreoever, $\sigma^f$ is the coefficient of variation of new daily infections.

Turning to the epidemic disease, I assume that the event that a susceptible individual catches the epidemic disease follows a bernoulli random variable:

$$\Delta c_{t+1}^*(j) | x_t^*(j) = 1 \sim Bernoulli\left(IR_t^*\right)$$

where $IR_t^*$ is the true latent infection risk and will be defined shortly. Assuming that individual infection events are independent and aggregating across individuals, one gets new daily aggregate infections

$$\Delta C_{t+1}^* \sim Binomial\left(X_t^*, IR_t^*\right)$$

where $X_t^*$ is the (latent) number of susceptible individuals.[12] Importantly, the true latent infection risk in the model is assumed to be the following:

$$IR_t^* = \underbrace{\beta}_{\text{Transmission Coefficient}} \times \underbrace{\rho_t}_{\text{Contact Rate}} \times \underbrace{\frac{I_I^* - \theta \cdot I_t}{P_t - \theta \cdot I_t}}_{\text{Probability of Meeting an Infected}}$$

[12] Notice that, throughout the paper, variables in capital letters denote aggregate time-series and are recovered as follows:

$$W_t = \sum_{j=1}^{P_0} w_t(j)$$

where $W_t$ denotes a generic time-series variable and $w_t(j)$ denotes the individual-level counterpart.

where $\beta$ is the exogenous transmission coefficient, which is the product of the transmission risk upon contact with an infected and the average number of pre-epidemic contacts; $\rho_t$ is the contact rate, which is normalized to one absent the epidemic; $I_t^*$ is the true latent number of active infections; $I_t$ is the number of detected active infections by the health-care system; $\theta$ is a parameter summarizing the degree of enforcement of the isolation policy adopted by the health care system; and $P_t$ is population.[13]

While isolation of infected individuals directly affects the probability of meeting an infected, behavioral responses directly affect the endogenous contact rate:

$$\rho_t = \pi \cdot \bar{N}_t + (1 - \pi) \cdot \bar{L}_t$$

where $\pi$ is the (exogenous) share of contacts due to work (as opposed to leisure), $\bar{N}_t$ is average labor supply across agents, and $\bar{L}_t$ is average leisure. More precisely:

$$\bar{N}_t = P_t^{-1} \cdot \sum_{j=1}^{P_0} n_t(j), \quad \bar{L}_t = P_t^{-1} \cdot \sum_{j=1}^{P_0} l_t(j)$$

Implicitly, the idea is that labor supply, leisure and interactions across agents are all sides of the same coin. A fall in labor supply and/or leisure reduces interactions among agents, which in turn reduces the true infection risk.

### 4.2 Individuals

Individuals supply labor for production, enjoy leisure and can be infected by either disease. I first describe the reduced-form behavior of labor supply and leisure, and then turn to the evolution of each disease conditional on infection.

**Work and Leisure** Individuals achieve a daily production $y_t(j)$ by supplying labor:

$$y_t(j) = A \cdot n_t(j)$$

where $A$ captures the daily average productivity of an individual, and $n_t(j)$ denotes the individual's labor supply. I assume that labor supply depends on health status, fear of the epidemic, and whether the individual is subject to mandatory isolation. In a reduced-form way, I posit that:

$$n_t(j) = \begin{cases} n_0 \cdot (1 + \chi_t)^{-\varepsilon_n} & \text{if } j \text{ has no or mild symptoms and not isolated} \\ (1 - \theta) \cdot n_0 & \text{if } j \text{ has no or mild symptoms and isolated} \\ 0 & \text{if } j \text{ is dead or has severe symptoms} \end{cases}$$

[13] Notice that when $\theta = 1$ each detected active infection is put into full isolation and when $\theta = 0$ none is. Imperfect isolation obtains when $\theta \in (0, 1)$.

where $n_0$ is labor for a healthy individual in normal times (i.e. absent the epidemic), $\chi_t$ is the perceived risk of death from the epidemic disease, and $\varepsilon_n$ is the (approximate) elasticity of labor supply with respect to the perceived risk of death. The equation above says that dead individuals and those with severe symptoms cannot work, while what those alive do depends on whether they have been tested. An individual with no or mild symptoms that has not been tested, will not be isolated and will not know whether he is infected with the epidemic disease. As a result, she will be assumed to be capable of working, but will protect herself from the epidemic disease. Individuals who have tested positively are put under (imperfect) isolation. Given that they are currently infected, they have no reason to 'protect' themselves from the epidemic disease, and their labor supply will depend exclusively on the strictness of the isolation policy.[14] However, once the isolation is over and they are no longer infected, they keep 'protecting' themselves because they are not sure as to whether past infections guarantee immunity from the epidemic disease.

Consistently with the empirical analysis in section 3, I assume that the test positivity rate does not affect behavior and I model perceived death risk as:

$$\chi_t = \underbrace{\frac{D_t}{C_t}}_{\text{Case Fatality Rate}} \times \underbrace{\beta \cdot \frac{I_t}{P_t}}_{\text{Perceived Infection Risk}}$$

where I assume for simplicity that individuals use the true transmission coefficient $\beta$ when forming their perceptions. The reduced-form behavior of leisure is symmetric to that of labor supply:

$$l_t(j) = \begin{cases} l_0 \cdot (1+\chi_t)^{-\varepsilon_l} & \text{if } j \text{ has no or mild symptoms and not isolated} \\ (1-\theta) \cdot l_0 & \text{if } j \text{ has no or mild symptoms and isolated} \\ 0 & \text{if } j \text{ is dead or has severe symptoms} \end{cases}$$

Together, labor supply and leisure determine the level of interactions between agents.

**Epidemic Disease** The progression of the epidemic disease after infection is visually summarized in Figure 2. Conditional on infection, the epidemic disease evolves as follows: first, the individual enters a *pre-symptomatic period* (a.k.a. *incubation period*), during which they are infected (and can infect others), but do not manifest any symptoms. What happens next is the result of two random events. The first random event determines what type of symptoms the individual will display. I allow for three types of symptoms: severe symptoms, mild symptoms and no symptoms. The second random event determines the terminal outcome of the disease, i.e. whether the individual recovers or dies. The modeling details can be found in Online Appendix B.1.

[14] Individuals in the model engage in a 'selfish' behavior similar to what described in Eichenbaum et al. (2020c).

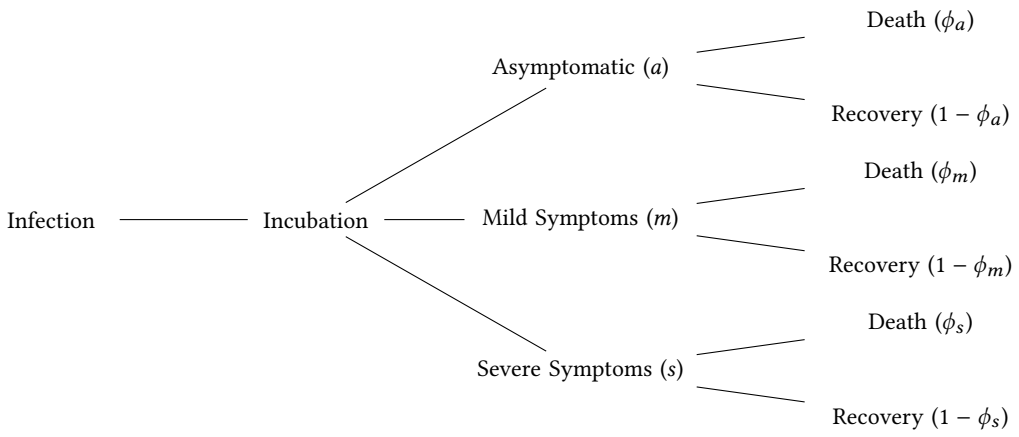


**Figure 2:** Evolution of the Epidemic Disease upon Infection

**Confounding Disease** Since the confounding disease is not the main object of investigation, its characterization is simplified as much as possible. I assume no incubation period and two types of symptoms: severe symptoms and mild symptoms.[15] Importantly, symptoms induced by the confounding disease are similar to those arising from the epidemic disease, so that the former literally acts as a confounder in the diagnostic process of the latter. The modeling details can be found in Online Appendix B.1.

### 4.3 The Government

The government plays two roles in the model. First, it performs testing activity through the health-care system. Second, it collects revenues and engages in health-care spending.

**Symptoms-Based Testing Policies** I assume that the health-care system in the model adopts symptoms-based testing policies that mimic real-world ones. As the World Health Organization puts it, *"the decision to test should be based on clinical and epidemiological factors and linked to an assessment of the likelihood of infection"*, and there are few better indicators of infections than symptoms, especially at the early stages of an epidemic outbreak when the characteristics of the disease are still unknown.[16]

These considerations are introduced into the model by assuming that testing activity is *prioritized* based on the severity of symptoms displayed by individuals: severe symptomatic individuals

[15] The confounding disease can also be thought of as two different diseases which differ in the type of symptoms they induce. For instance, the mild-symptom state can be thought of as seasonal flu, and the severe-symptom state can be thought of as pneumonia.

[16] See https://www.who.int/publications/i/item/10665-331501 for the testing guidelines as of March 19 2020 during the COVID-19 outbreak. As of January 31 2020, right at the start of the outbreak, a suspected case to be tested was required to display either "severe acute respiratory infection requiring admission to hospital" or "any acute respiratory illness". See https://www.who.int/publications/i/item/laboratory-testing-of-2019-novel-coronavirus-(-2019-ncov)-in-suspected-human-cases-interim-guidance-17-january-2020.

are always tested first, then individuals with mild symptoms, and finally asymptomatic ones. Importantly, I assume the following:

**T1:** *There is always enough daily testing capacity to test severe symptomatic individuals.*

Assumption **T1** is motivated by two considerations. First, it reflects testing priority of individuals exhibiting severe symptoms, which is justified by the need to determine the underlying disease in order to decide appropriate medical treatment. Second, it reflects the fact that individuals with severe symptoms are more likely to show up at the hospital, be hospitalized, and tested.

What the government can therefore choose is whether to perform additional tests $T_t^{NS}$ on individuals who do not display severe symptoms (NS stands for 'Non-Severe').[17] Consistently with the idea of symptoms-based policies, these additional tests are administered to mild symptomatic individuals first, and to asymptomatic individuals only if there is any remaining testing capacity.[18]

Depending on the technical characteristics of the existing testing technology and on the properties of the epidemic disease, one can make slightly different assumptions about how testing activity is implemented in the model. I assume the following:

**T2:** *Individuals who have tested positively are not tested again.*

**T3:** *Tests detect only active infections, with a false negativity rate $\alpha$.*

**T4:** *The outcome of the test is known with a fixed delay $d$, and an individual is not tested again until the outcome of the previous test is known.*

Assumption **T2** is justified when immunity is obtained after recovery from the infection.[19] Assumption **T3** implies that a test (imprecisely) detects the infection during the incubation period and irrespective of the type of symptoms while infection is active, but not after death or recovery. The delay $d$ in assumption **T4** could reflect both technological and organizational constraints that create a fixed lag between the time a test is administered and the time its outcome is known. The testing policies are implemented using set theory and the details can be found in Online Appendix B.2.

**Public Deficits** Government expenditure is given by:

$$Exp_t = c_T \cdot T_t + c_s \cdot |\Sigma_t^S|$$

where $c_T$ is the cost of each test performed, $T_t$ is the number of tests performed, $c_S$ is the cost of treating a severe infection, and $|\Sigma_t^S|$ is the number of individuals displaying severe symptoms.[20]

[17] As opposed to standard compartmental epidemiological models, which assume that a (constant) share of some compartment is tested each period, the proposed framework allows for the specification of a daily testing capacity in terms of the number of tests to be performed. This implies, for example, that a certain testing capacity permits testing of a large share of symptomatic individuals at the beginning of the epidemic, but of a very small share during its peak.

[18] Notice that there is no modeling of voluntary testing. However, since a large portion of voluntary testing arguably arises due to the appearance of symptoms, testing policies that prioritize testing of symptomatic individuals should implicitly account - at least partially - for voluntary testing.

[19] It also implicitly assumes that the health care system has a way to detect recovery that does not require the use of an additional test, or that there is another testing capacity dedicated for this purpose.

[20] I assume that individuals with severe symptoms from any disease require costly medical treatment.

The government also collects revenues by taxing economic activity:

$$Rev_t = \tau \cdot Y_t$$

where $\tau$ is a uniform tax rate and $Y_t$ is daily GDP, which is obtained aggregating individuals' daily production. For simplicity, I assume that the government can run budget deficits or surpluses which are freely rolled over.

# 5 Understanding the Mechanism

## 5.1 A Coronavirus-Like Influenza Disease

Table 3 reports the parameterization for a generic coronavirus-disease, which I refer to as the 'baseline parameterization'.[21]

| **Individuals** | | | **Government** | | |
|---|---|---|---|---|---|
| *Baseline Labor Supply* | $n_0$ | 1 | *Test Cost* | $c_T$ | 25 |
| *Baseline Leisure* | $l_0$ | 1 | *Treatment Cost* | $c_S$ | 300 |
| *Daily Productivity* | $A$ | 175 | *Tax Rate* | $\tau$ | 0.30 |
| *Elasticity of Labor to* $\chi$ | $\varepsilon_n$ | 1000 | *Test Outcome Delay* | $d$ | 1 |
| *Elasticity of Leisure to* $\chi$ | $\varepsilon_l$ | 1000 | *Test False Negative Rate* | $\alpha$ | 0.25 |
| *Contact Share from Work* | $\pi$ | 0.5 | *Isolation Effectiveness* | $\theta$ | 0.9 |
| **Epidemic Disease** | | | **Confounding Disease** | | |
| *Transmission Coefficient* | $\beta$ | 0.275 | *Probability of Severe Symptoms* | $s^f$ | 0.10 |
| *Probability of Severe Symptoms* | $s$ | 0.30 | *Infection Fatality Risk* | $\phi^f$ | 0.02 |
| *Probability of Mild Symptoms* | $m$ | 0.40 | *Time from Infection to Recovery* | $q^f$ | 7 |
| *Probability of No Symptoms* | $a$ | 0.30 | *Time from Infection to Death* | $k^f$ | 7 |
| *Infection Fatality Risk for Severe* | $\phi_s$ | 0.15 | *Share of Population Infected* | $\omega^f$ | 0.20 |
| *Infection Fatality Risk for Mild* | $\phi_m$ | 0 | *Volatility of New Daily Infections* | $\sigma^f$ | 0.10 |
| *Infection Fatality Risk for Asymptomatic* | $\phi_a$ | 0 | **General** | | |
| *Unconditional Infection Fatality Risk* | $\phi$ | 0.045 | *Initial Population* | $P_0$ | 5e4 |
| *Incubation Period* | $p$ | 3 | *Time Horizon* | $T$ | 350 |
| *Mean Lag from Symptoms to Recovery* | $\tilde{q}$ | 11 | | | |
| *Mean Lag from Symptoms to Death* | $\tilde{k}$ | 5 | | | |
| *Initial Infections* | $C_0^*$ | 50 | | | |

**Table 3:** Parameterization of a Coronavirus-Like Disease

In this benchmark parameterization, the epidemic disease features a large share of mild symptomatic and asymptomatic infections, an incubation period, and a relatively high probability of death for severe infections, but a null one for non-severe infections. The average time from infection to death is 8 days, and from infection to recovery is 14 days. The confounding disease, instead, is meant to resemble a seasonal flu. Most infections are mild symptomatic, and the infection fa-

[21] Coronavirus diseases, such as SARS-CoV-2, are usually deemed as 'influenza-like' diseases, because of their similarity with influenza. The 2002-2004 SARS epidemic, the 2009 swine flu pandemic, and the ongoing MERS are all examples of influenza-type diseases. On the other hand, measles and ebola are epidemic disease which are not influenza-type.

tality risk for this disease is relatively low. The time from infection to any terminal outcome is assumed to be a week, and 20% of the population is infected over the course of roughly a year.

Individual labor supply and leisure in normal times are normalized to 1. Daily productivity is chosen as to roughly match daily GDP per capita in the U.S., while the elasticities of labor and leisure to the perceived death risk are set to 1000, which generates a sizeable fall in GDP over a 1-year horizon. Half of contacts arise from work, while the other half from leisure activities. I set the cost of a test to 25 dollars, the cost of daily treatment of a severe infection to 300 dollars, and the tax rate to 30%. I assume that it takes one day to learn the outcome of a test, that the probability of a false negative is 25% and that the compliance rate of mandatory isolation is 90%. The population size is set to strike a balance between computational speed and uncertainty of outcomes.[22]

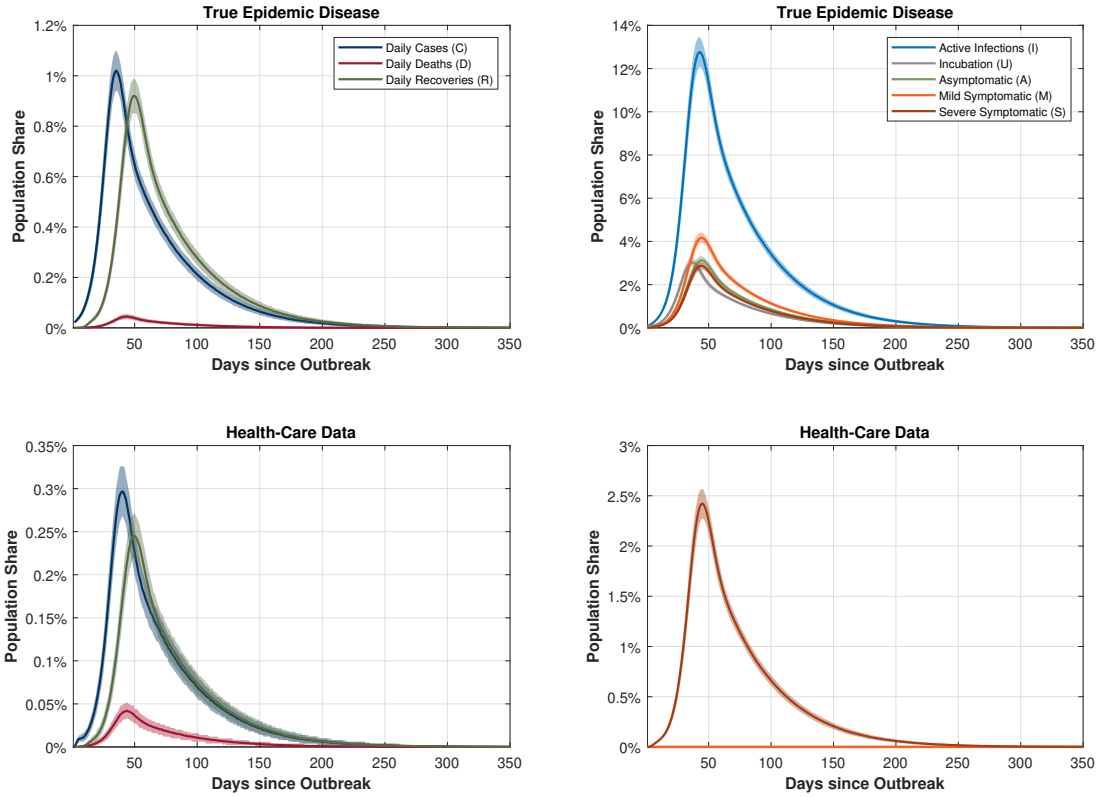


**Figure 3:** Epidemic Dynamics for a Coronavirus-Type Epidemic Disease

Figure 3 displays the epidemic dynamics under the baseline parameterization when the government does not mandate any additional testing on non-severe individuals, i.e. $T_t^{NS} = 0, \forall t$. This implies that the health-care system tests only individuals who exhibit severe symptoms. The top row of the figure summarizes the dynamic evolution of the true latent epidemic. First, most infections result in recovery. Second, active infections can be in the incubation period, asymptomatic, mild symptomatic or severe symptomatic. Third, new daily cases are asymmetric because the

[22] All the simulations in the paper report 68% confidence bands. A larger population size reduces the variance of outcomes, but increases the computational costs. Notice that, unlike standard epidemiological models, even when the population size increases to infinity, uncertainty remains due to the testing activity.

spread of the disease slows down after peak, as a result of agents' endogenous behavior.

The bottom row of the figure displays, instead, what is detected by the health-care system. Only a small portion of true cases is detected, and, since only individuals displaying severe-symptoms are tested, the health-care system does not detect any infection in the incubation period, nor any infection who displays mild or no symptoms. In other words, detected infections are not representative of overall infections and are the most likely to die from the disease.[23]

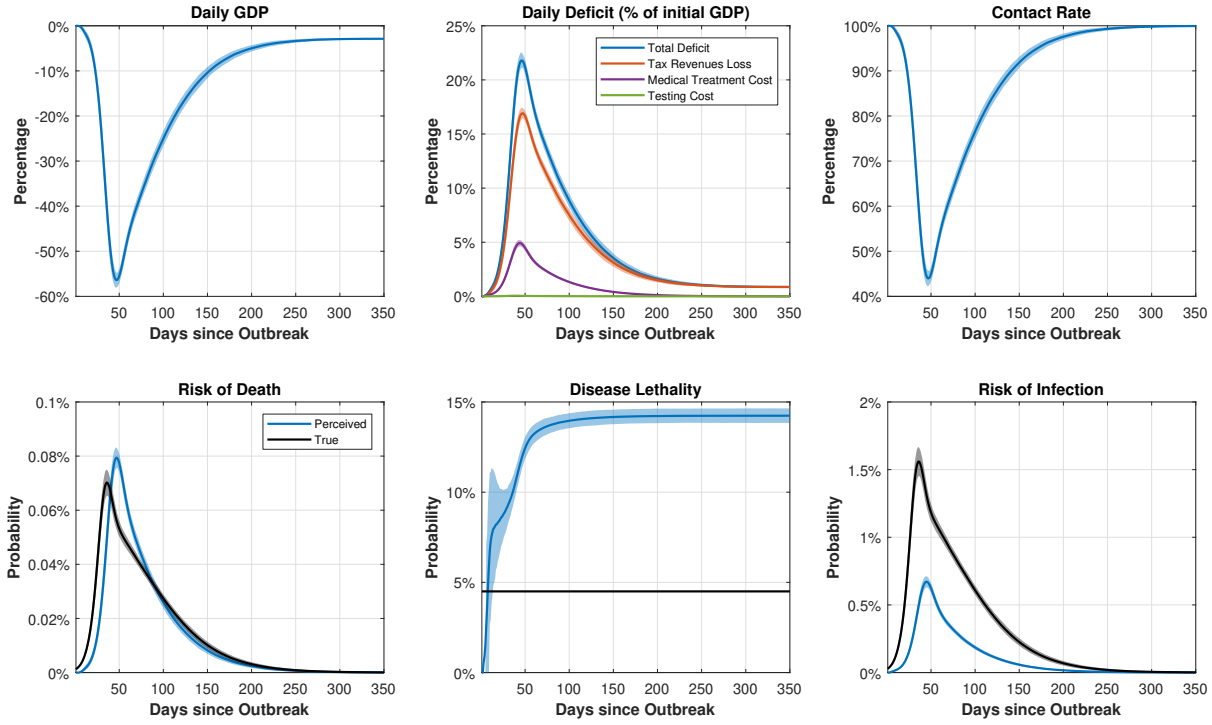


**Figure 4:** Economic Dynamics for a Coronavirus-Type Epidemic Disease

Figure 4 summarizes what happens to the economy. Output contracts, interactions among agents fall and budget deficits soar. This is mainly the result of agents' responses to the perceived risk of death - reported in the second row of the figure. As the outbreak unfolds, the health-care system produces time-series of testing data on cases, deaths and active infections which are used by agents to assess death risk. As the latter increases, agents cut on both work and leisure activities, causing a reduction in both economic activity and epidemic spread.[24] In Online Appendix D.1, I clarify further the role of behavioral responses.

Soaring deficits arise from the combination of several forces. The main one is the loss of tax revenues resulting from the fall in economic activity. The second is the increase in expenditure on medical treatments: each severe symptomatic individual requires costly medical attention and, as a result, health-care expenditure rises. Last, there is the cost of testing. Given that the government

[23] Under the baseline parameterization, the health-care system correctly detects all the deaths due to the epidemic disease.

[24] Under the proposed parameterization, the cumulative GDP loss over the course of one year is around 15%, while the public deficit rises by around 5% of pre-epidemic GDP.

is not mandating any additional testing on non-severe individuals, expenditure on testing is small in this scenario.

The second row of the figure clarifies what happens to agents' perceptions of risk, and compares them to the true latent risk. For this specification, the perceived death risk (in blue) is similar to the true death risk (in black), although this is not a general property of the model. In fact, agents' perceptions about the two determinants of overall risk are incorrect. The perception of disease lethality is off because the case fatality rate substantially over-estimates the true infection fatality risk, as a result of a 'narrow' testing policy that focuses on severe symptomatic individuals, who have the highest conditional infection fatality risk. The perception of infection risk is also off because the health-care system sizably under-estimates the number of true active infections.

### 5.2 Validating Testing Policies: The Case of Northern Italy

To validate the testing policies in the model, I compare the dynamic evolution of key testing variables in the model with their empirical counterparts, using the Italian regions of Lombardy and Veneto during the first SARS-CoV-2 outbreak in early 2020 as a case study. These two regions were the first two territories who experienced the SARS-CoV-2 epidemic outbreak in the West. They border each other, have a similar GDP per capita, similar infrastructures, are both highly populated, and were both among the most hit regions during the epidemic outbreak in Spring 2020.

A key difference between the two regions, however, lies in their approach to testing. At the beginning of the outbreak, Lombardy followed the Italian government's testing guideline, which in turn followed the WHO's initial instructions to limit testing to severe symptomatic individuals. Veneto's approach, instead, was shaped by Professor Andrea Crisanti and consisted in implementing a wider testing policy right away.[25]

For my validation exercise, I therefore parallel testing data from Lombardy to those generated under the baseline parameterization, where the health-care system tests only severe symptomatic individuals. Testing data from Veneto, instead, are paralleled to those generated from the baseline parameterization with the twist that both severe and mild symptomatic individuals are tested daily. The results are presented in Figure 5.[26]

[25] As Science Magazine reports: *"Crisanti persuaded the regional government of Veneto to test anyone with even the mildest of symptoms, and to trace and test their contacts as well"*, while *"[g]uidelines from the World Health Organization and Italy's National Institute of Health said to test only patients with symptoms"*. See https://www.sciencemag.org/news/2020/08/how-italy-s-father-swabs-fought-coronavirus for the full article.

[26] It is also possible to show that the number of total tests per-capita is higher in Veneto, which confirms the narrative that the region enacted a wider testing policy than Lombardy. The model also mechanically reproduces this fact.

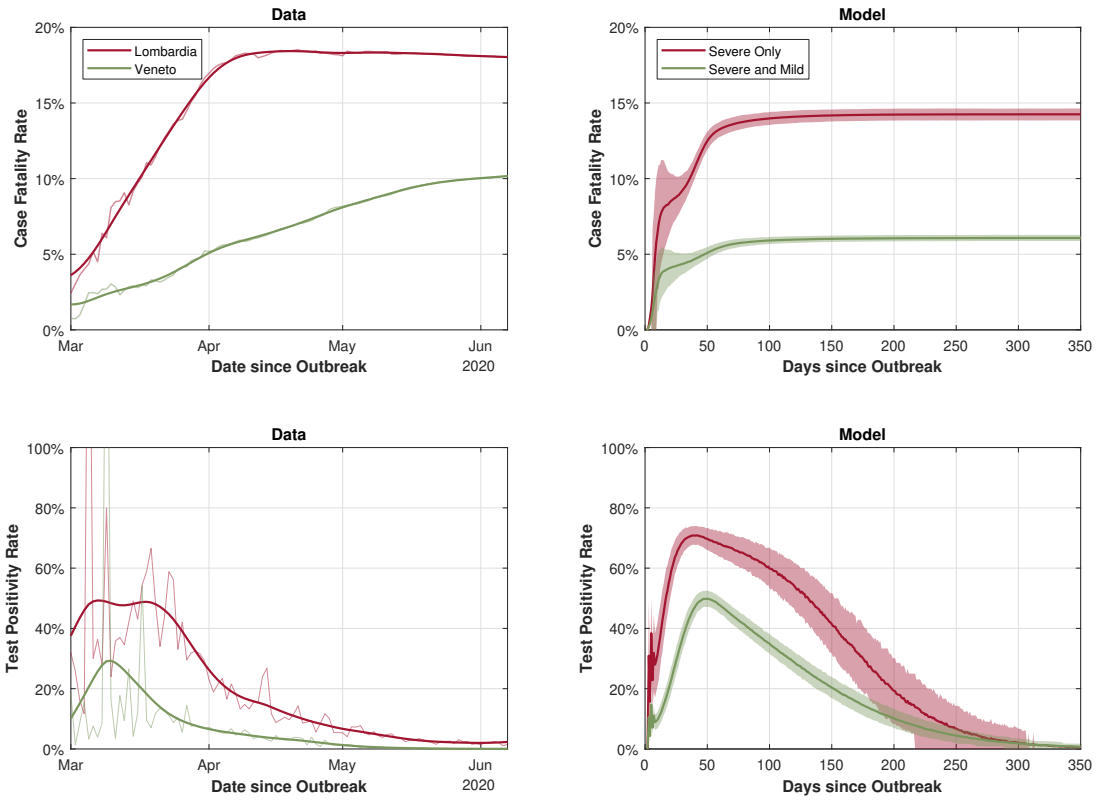


**Figure 5:** Validation of Model's Testing Policies with Data from Northern Italy
**Source:** Protezione Civile. Data are smoothed with an HP-Filter with smoothing parameter set to 200.

The top row looks at the case fatality rate. Under a wider testing policy, the case fatality rate decreases since infected individuals with a lower probability of dying are included in the case count. This is generated by the model and confirmed in the data.

The bottom row shows the test positivity rate. The test positivity rate is generally expected to decrease with a wider testing policy, since the probability of being infected is usually increasing in the severity of symptoms. Under a wider testing policy, it becomes harder to find infected individuals and the test positivity rate falls. The model can reproduce this fact and match the data. Furthermore, the model is also able to replicate the dynamic evolution of the test positivity rate observed in the data.[27]

[27] Standard epidemiological models struggle to generate the dynamic behavior of the test positivity rate observed in the data. In fact, with symptoms-based testing, standard models would generate a constant positivity rate equal to one (assuming that the testing technology is precise). The ability of the proposed model to match the data is due to the presence of a stationary confounding disease. The latter makes detection of epidemic infections hard when there are few of them, resulting in a low test positivity rate. But when epidemic infections peak, the test positivity rate rises.

# 6 The Testing Multiplier

## 6.1 Definitions

Let's assume that the government mandates a constant daily capacity $\bar{T}$ for testing of mild symptomatic and asymptomatic individuals.[28] Let's define cumulative GDP and the cumulative direct cost for a generic testing policy $\bar{T}$ over a horizon $T$ as follows:

$$\mathcal{Y}(\bar{T}) = \sum_{t=1}^{T} Y_t, \quad \mathcal{E}(\bar{T}) = \sum_{t=1}^{T} c_T \cdot T_t \quad \text{with} \quad \{T_t^{NS} = \bar{T}\}_{t=1}^{T}$$

where $T_t$ is the total number of tests performed at time $t$. Notice that both $\mathcal{Y}(\bar{T})$ and $\mathcal{E}(\bar{T})$ are random variables, since the evolution of the epidemic is random. It is then possible to define the testing multiplier for output:

$$\text{GDP-Multiplier} = \frac{\mathcal{Y}(\bar{T}) - \mathcal{Y}(0)}{\mathcal{E}(\bar{T}) - \mathcal{E}(0)}$$

which is an *average multiplier* because it summarizes the effect on GDP of an additional dollar spent on testing *relative* to the case where there is no additional spending on testing mandated by the government. Also, notice that the multiplier is a random variable itself.

Similarly, one can define the testing multiplier for the budget surplus:

$$\text{Surplus-Multiplier} = \frac{\mathcal{B}(\bar{T}) - \mathcal{B}(0)}{\mathcal{E}(\bar{T}) - \mathcal{E}(0)}$$

where $\mathcal{B}(\bar{T})$ represents the cumulative budget-surplus over a horizon $T$.[29] Similarly to the GDP-Multiplier, the Surplus-Multiplier summarizes the effect on the budget of an additional dollar spent on testing.

Its interpretation is nuanced. When the Surplus-Multiplier is positive, an additional dollar spent on testing reduces the public deficit. In the model, this could (and does) happen because testing expenditure reduces the fall in tax revenues and curbs the rise in costly medical treatments. When the Surplus-Multiplier is zero, an additional dollar spent on testing leaves the public deficit untouched, meaning that testing expenditure fully repays itself. When the Surplus-Multiplier is negative but less than 1 in absolute value, an additional dollar spent on testing adds to the deficit less than one dollar.

[28] The government policy does not have to be constant over time, but assuming so is a way to restrict the space of possible government's actions and simplify the comparison of alternative policies.

[29] This is given by:

$$\mathcal{B}(\bar{T}) = -\sum_{t=1}^{T} Def_t \quad \text{with} \quad \{T_t^{NS} = \bar{T}\}_{t=1}^{T}$$

where $Def_t$ is daily deficit.

## 6.2 The Multiplier is Positive for the Baseline Parameterization...

Figure 6 shows the GDP-Multiplier and the Surplus-Multiplier for the baseline parameterization.[30] In Online Appendix D.3, I explore the robustness of the multiplier with respect to the characteristics of the testing and isolation technology.

The GDP-Multiplier is on average always positive and above one. Since luck plays an important role at low testing levels, however, there are realizations of the process where the multiplier takes negative values. With a negative multiplier, additional testing amplifies the recession caused by the epidemic outbreak.

The Surplus-Multiplier is positive for most testing levels, although it turns negative (but greater than $-1$) at very high ones. This means that testing at least partially pays for itself at all testing levels. Luck plays an important role in this case as well, especially at low testing levels.

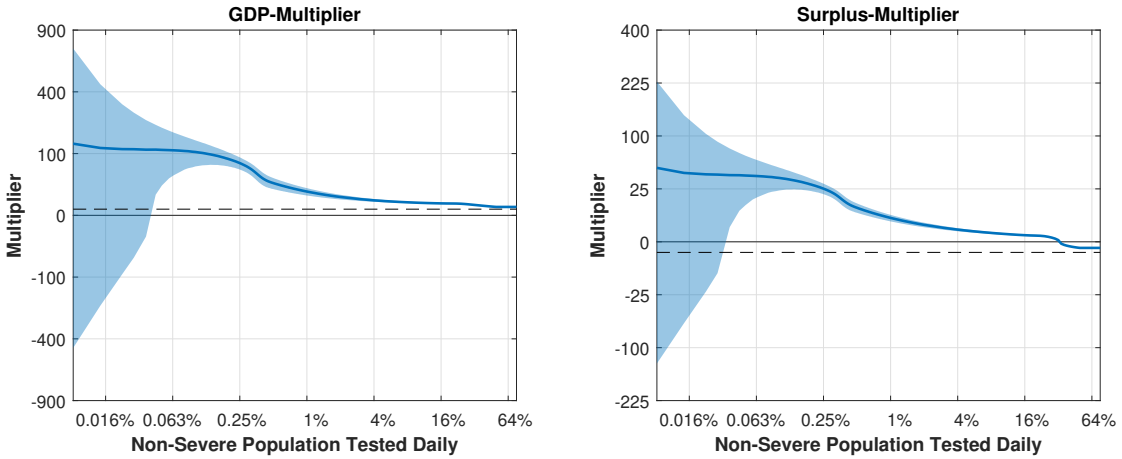


**Figure 6:** The Testing Multiplier under the Baseline Parameterization

While the testing multiplier is a concise summary of the effects of increased testing on the economy, it is not immediate to understand the channels through which it operates. Figure 7 and Figure 8 help clarify what happens by illustrating the dynamic evolution of the disease under three testing levels: low (in red), medium (in orange), and high (in green).

[30] Given the highly non-linear nature of the testing multiplier, I report it on a non-linear scale. The x-axis is on a $log_2$ scale, while the y-axis on a square-root scale.

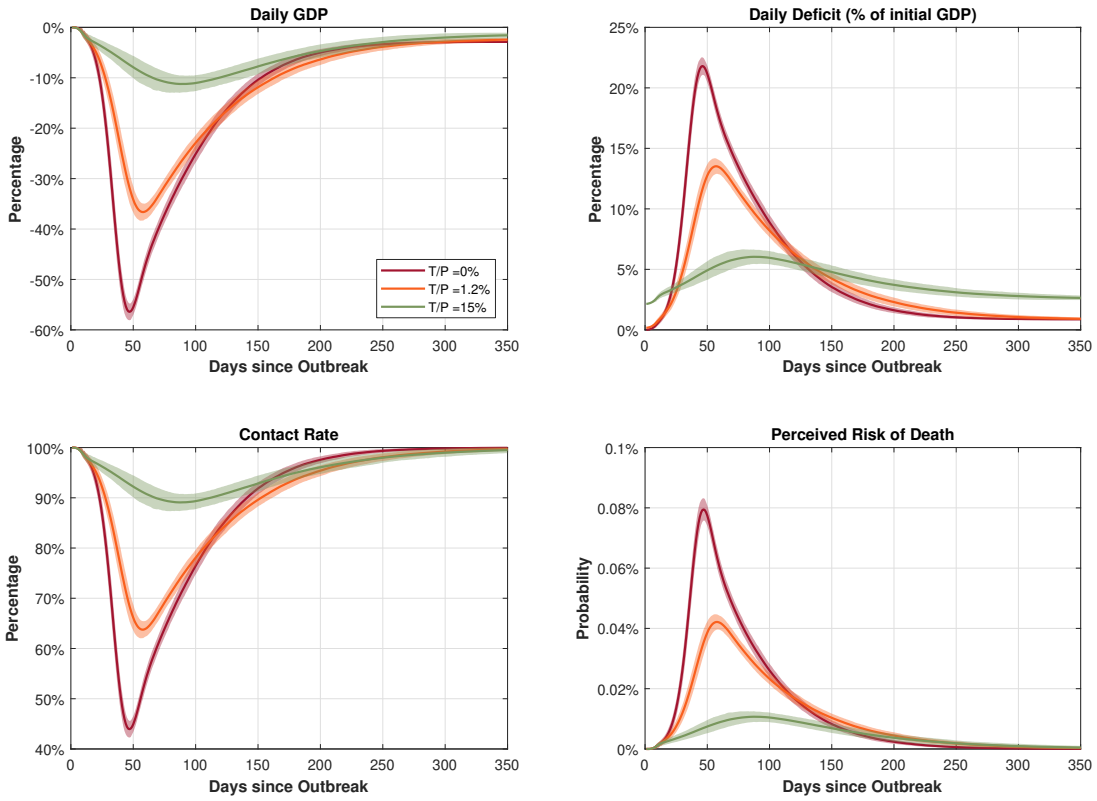


**Figure 7:** Economic Dynamics under Different Testing Levels

Consider Figure 7 first. Under the low testing level, the overall perceived risk of death is highest, which results in lower labor supply, thus higher GDP loss and deficit increase. By expanding testing more and more, the government slows down epidemic transmission thanks to isolation of the infected and behavioral responses. This, in turn, reduces agents' perceived risk, thereby mitigating the fall in GDP and curbing the rising deficit.

What happens to the perceived risk of death is illustrated in Figure 8. As previously explained, because of symptoms-based testing policies who prioritize testing according to the severity of symptoms, individuals with a much lower risk of death are tested when the government mandates additional testing capacity. As a result, the case fatality rate falls, reducing the perceived lethality of the disease, as shown in the top-left panel.[31]

What happens to the perceived infection risk is more complicated. Whenever the government decides to expand testing, the overall number of true latent infections fall, but the share that is detected rises. Which of these two forces prevail is not obvious. In Figure 8, for example, the perceived infection risk rises when ones moves from low to medium testing, but falls when one moves from medium to high testing.

[31] With more and more testing, the case fatality rate keeps falling to the point where it converges to the true infection fatality risk.

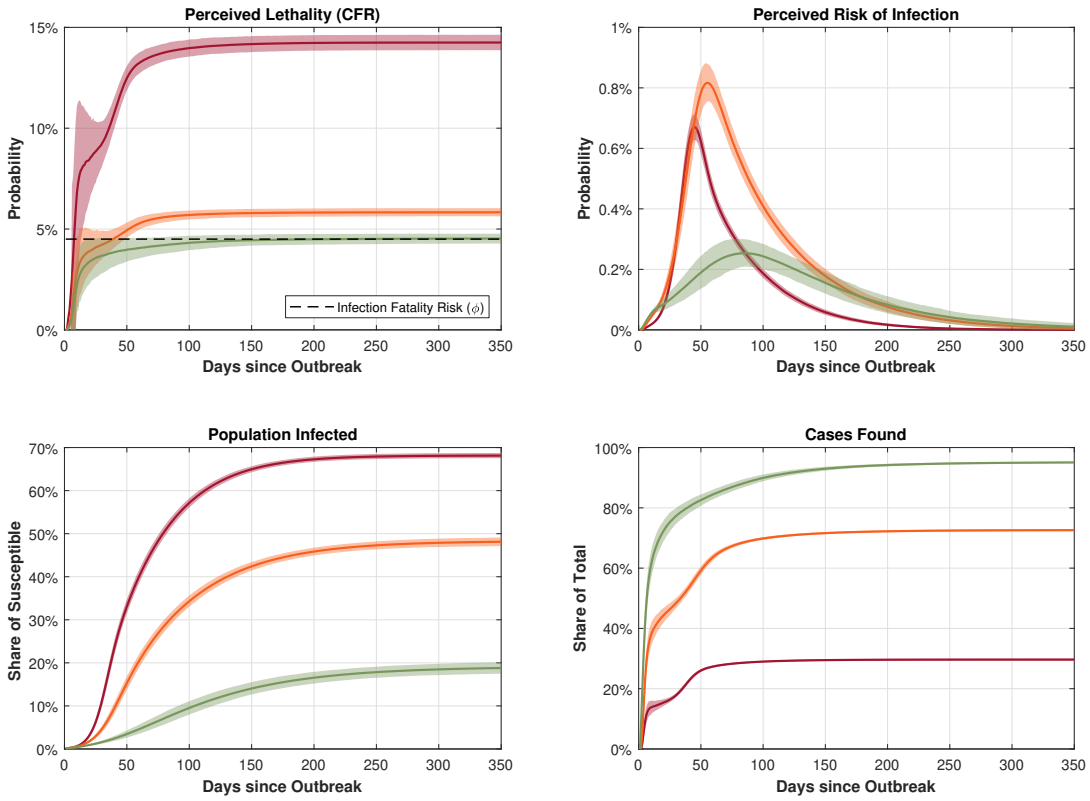


**Figure 8:** Perceptions under Different Testing Levels

In this simulation, even when additional testing increases the perceived risk of infection, the fall in the case fatality rate prevails so that the overall perceived death risk falls.

## 6.3 ... But Can Be Negative for Alternative Diseases

The testing multiplier is a complicated object which does not need to be positive on average. To illustrate this point, I introduce other three influenza-like diseases, each one departing from the baseline parameterization under a specific aspect, as summarized in Table 4. All other parameters of the model stay untouched.

| | **Disease A** **Baseline** | **Disease B** **"Unstoppable"** | **Disease C** **"Less-Lethal"** | **Disease D** **"Never-Ending"** |
|---|---|---|---|---|
| $\beta$ | 0.275 | 0.475 | 0.275 | 0.275 |
| $\phi_s$ | 0.15 | 0.15 | 0.01 | 0.15 |
| $\tilde{k}$ | 5 | 5 | 5 | 20 |
| $\tilde{q}$ | 11 | 11 | 11 | 26 |

**Table 4:** Parameterization of Alternative Influenza-Like Diseases

Disease B is 'unstoppable' because its transmission coefficient is so high that moderate levels of testing and isolation might not be enough to slow down its spread. Disease C is 'less-lethal' because its infection fatality risk for individuals who develop severe symptoms is lower than in

the baseline. This implies that there is little room to reduce the perceived lethality of the disease with additional testing. Finally, disease D is 'never-ending' because the length of infection is longer than in the baseline. This implies that an infected individual remains contagious for more days, increasing the probability of infection for others.

Figure 9 shows the GDP-multiplier across these alternative diseases, while the similar results for the Surplus-Multiplier can be found in Online Appendix D.2. Across all diseases, the GDP-multiplier is positive when a sizeable share of the population is tested every day, but not necessarily otherwise. The multiplier becomes negative when additional testing increases the perceived risk of death, which compounds the contraction of economic activity. The main reason why this happens is that, at a small-scale, additional testing fails to contain the epidemic and results in a higher number of detected cases, increasing the perceived risk of infection.

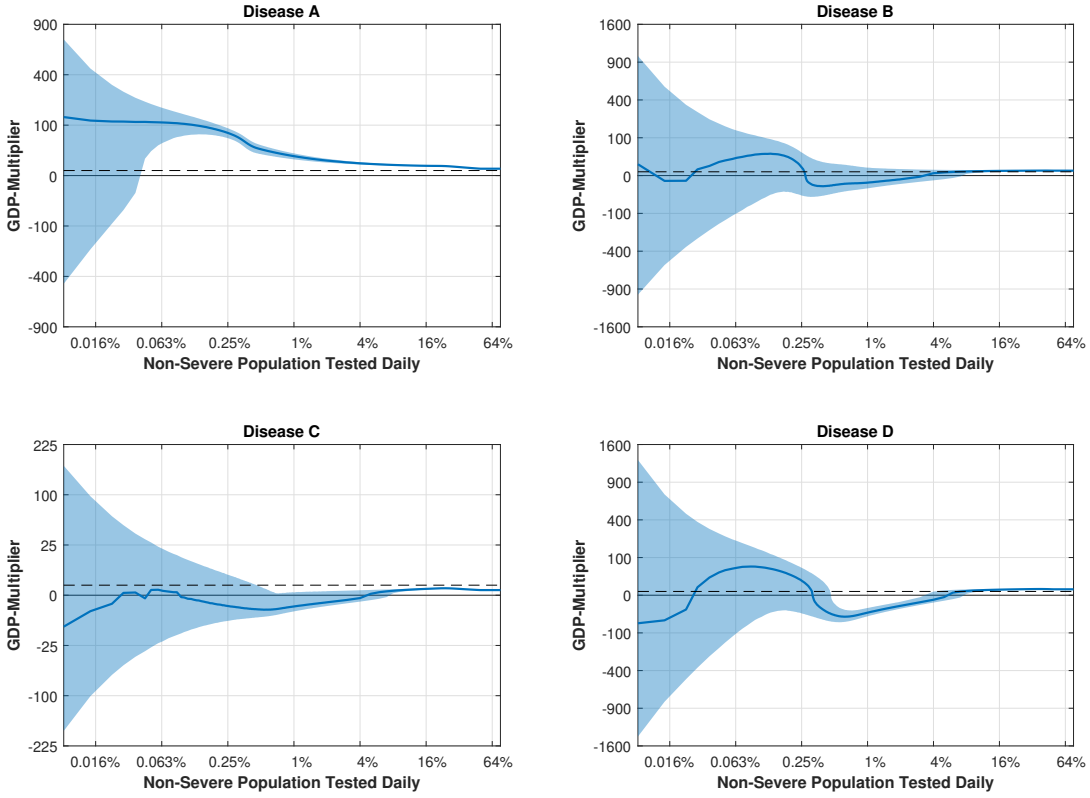


**Figure 9:** The GDP-Multiplier for Alternative Diseases

Importantly, the contraction of economic activity occurs in spite of a systematic improvement in health outcomes, as illustrated in Figure 10.[32]

[32]The number of deaths is on average proportional to the number of total infections, which implies that the former would also monotonically decrease in the number of individuals tested daily.

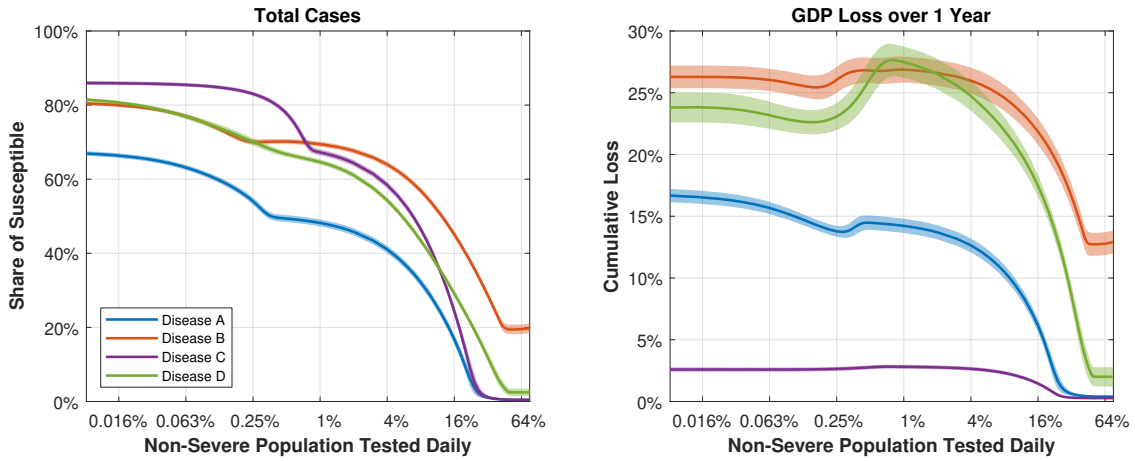


**Figure 10:** Economic and Public Health Outcomes for Alternative Diseases

# 7 Age-Heterogeneity and Risk Perceptions

There are reasons to suspect that how different groups in the population perceive the threat posed by the epidemic disease matters for aggregate health and economic outcomes. For example, referring to the U.S. response to the SARS-CoV-2 outbreak in 2020, Jay Bhattacharya, Professor of Medicine at Stanford University, points out that *"[...] a major public health message that we failed at is describing the [...] age-gradient in the risk. Older people think that they are at lower risk than they actually are, and younger people think they are at higher risk than they actually are. I think that is an enormous public health mistake"*.[33]

To investigate the importance of heterogeneous perceptions across age groups, I divide the population into two groups: young and old. I then calibrate the model to the U.S. and SARS-CoV-2, which features a sharp age-heterogeneity in the infection fatality risk.

I consider two extreme scenarios. In the first, the government releases only testing data that are aggregated across age groups - as it often happens during epidemic outbreaks. Because of this, I assume that the two groups share the same perceptions of risk. In the second scenario, the government provides them with age-specific testing data, and risk perceptions are different across the two groups.[34]

## 7.1 Heterogeneous-Agent Framework

I assume that there are $G = 2$ age groups, young and old:[35]

$$P_0 = P_0^y + P_0^o, \quad P_0^y = \omega^y \cdot P_0, \quad P_0^o = (1 - \omega^y) \cdot P_0$$

[33] The full interview can be watched at https://youtu.be/2tsUTAWBJ9M. The quote can be found at minute 24:20.

[34] While the assumption that heterogeneous individuals could share the same risk perceptions is certainly a stretch, it provides a useful thought-experiment to assess the importance of heterogeneous perceptions.

[35] The generalized model nests the homogeneous case when $G = 1$ or when the various groups are parameterized to be identical.

where $\omega^y$ is the share of young agents in the initial period. In any time period, new true latent epidemic infections for each group are given by:

$$\Delta C^{y*}_{t+1} \sim Binomial(X^{y*}_t, IR^{y*}_t), \quad \Delta C^{o*}_{t+1} \sim Binomial(X^{o*}_t, IR^{o*}_t)$$

where $X^{g*}_t$ is the number of susceptible individuals in group $g$, and $IR^{g*}_t$ is the latent infection risk for group $g$. The two groups interact with each other and these interactions determine the group-specific infection risk as follows:[36]

$$IR^{y*}_t = \beta \times \left[ \rho^{yy}_t \times \frac{I^{y*}_{t-1} - \theta \cdot I^{y}_{t-1}}{P^{y}_{t-1} - \theta \cdot I^{y}_{t-1}} + \rho^{yo}_t \times \frac{I^{o*}_{t-1} - \theta \cdot I^{o}_{t-1}}{P^{o}_{t-1} - \theta \cdot I^{o}_{t-1}} \right]$$

$$IR^{o*}_t = \beta \times \left[ \rho^{yo}_t \times \frac{I^{y*}_{t-1} - \theta \cdot I^{y}_{t-1}}{P^{y}_{t-1} - \theta \cdot I^{y}_{t-1}} + \rho^{oo}_t \times \frac{I^{o*}_{t-1} - \theta \cdot I^{o}_{t-1}}{P^{o}_{t-1} - \theta \cdot I^{o}_{t-1}} \right]$$

which assumes that the infection risk depends on the transmission coefficient (which is assumed to be homogeneous across groups), on the number of interactions within and between groups, and the probability of meeting an infected individual within each group. Pre-epidemic contact rates between groups are given by:

$$\rho_0 = \begin{bmatrix} \rho^{yy}_0 & \rho^{yo}_0 \\ \rho^{oy}_0 & \rho^{oo}_0 \end{bmatrix}$$

where the rows of the matrix sum to 1, and each entry represents the share of contacts that a group entertains with another. Importantly, as the epidemic unfolds, behavioral responses make the matrix of contact rates become endogenous as follows:

$$\rho^{yy}_t = \rho^{yy}_0 \cdot \left[ \pi \cdot \bar{N}^{y}_t + (1-\pi) \cdot \bar{L}^{y}_t \right], \quad \rho^{oo}_t = \rho^{oo}_0 \cdot \left[ \pi \cdot \bar{N}^{o}_t + (1-\pi) \cdot \bar{L}^{o}_t \right]$$
$$\rho^{yo}_t = \rho^{yo}_0 \cdot \left[ \pi \cdot \bar{N}_t + (1-\pi) \cdot \bar{L}_t \right], \quad \rho^{oy}_t = \rho^{oy}_0 \cdot \left[ \pi \cdot \bar{N}_t + (1-\pi) \cdot \bar{L}_t \right]$$

where $\rho^{gg'}_t$ is the contact rate between group $g$ and $g'$, $\bar{N}^{g}_t$ is average labor supply in group $g$, and $\bar{L}^{g}_t$ is average leisure in group $g$, $\bar{N}_t$ is average labor supply in the population, and $\bar{L}_t$ is average leisure in the population. Since between-group interactions are a population-weighted average of labor supply and leisure, the model features a reduced-form infection externality between groups.[37] Labor supply and enjoyment of leisure for a generic individual $j$ in group $g$ are the same as in the homogeneous case, except that the perceived risk of dying from the disease $\chi^g_t$ is now group-specific. Daily production is given by:

$$y_t(j,g) = A^g \cdot n_t(j,g)$$

[36] This specification is similar in spirit to Acemoglu et al. (2020), and to what is used in the epidemiological literature with heterogeneous age groups. See for example Mistry et al. (2020).

[37] The infection externality arises because the labor supply and enjoyment of leisure of one group affects the number of interactions of the other, and thus their infection risk.

where $A^g$ is the productivity of an individual belonging to group $g$.[38]

In the first scenario, the government releases only aggregate testing data and the perceived death risk is given by:

$$\chi_t = \frac{D_t}{C_t} \times \beta \cdot \frac{I_t}{P_t}$$

and is the same across groups, i.e. $\chi_t = \chi_t^y = \chi_t^o$. In the second scenario, the government releases also group-specific testing data, which allows agents to compute age-specific case fatality rates. The perceived risk of death for each group is given by:[39]

$$\chi_t^y = \frac{D_t^y}{C_t^y} \times \beta \cdot \frac{I_t}{P_t}, \quad \chi_t^o = \frac{D_t^o}{C_t^o} \times \beta \cdot \frac{I_t}{P_t}$$

### 7.2 A SARS-CoV-2 Calibration

I calibrate the model to the U.S. and SARS-CoV-2, and the parameters are reported in Table 5. The omitted parameters are the same as in Table 3. The population share of young individuals matches that of individuals younger than 65 years old in the U.S. Census. I assume that 40% of infected individuals are asymptomatic following Oran and Topol (2020), and I assume that the remaining 60% of infections are equally split between mild and severe symptoms. I assume that age does not correlate with the severity of symptoms, following Jung et al. (2020). I keep the assumption that only severe symptomatic individuals can die, and I choose their infection fatality risks so that the implied unconditional infection fatality risk of each group matches the estimates in Levin et al. (2020). The average incubation period comes from Qin et al. (2020). I set the other lags so that they match the period over which an individual can infect others, as reported by the U.S. Centers for Disease Control and Prevention in October 2020. Specifically, non-severe individuals are on average infectious for 14 days, while severe individuals stop to be infectious on average after 10 days since the onset of symptoms.

[38] As a result of these assumptions, the contribution of each group to GDP depends on its size, average labor supply and productivity.

[39] The assumption that the perceived infection risk is the same across groups is made for simplicity, and isolates the importance of heterogeneity in the perceived lethality of the disease.

| SARS-CoV-2 | | | |
|---|---|---|---|
| **Description** | **Parameter** | **Value** | **Source** |
| *Young Population Share* | $\omega^y$ | 0.835 | U.S. Census (2019) |
| *Initial Young Infections* | $C_0^{y*}$ | 42 | |
| *Initial Old Infections* | $C_0^{o*}$ | 8 | |
| *Infection Fatality Risk for Severe Young* | $\phi_s^y$ | 0.005 | Levin et al. (2020) |
| *Infection Fatality Risk for Severe Old* | $\phi_s^o$ | 0.248 | Levin et al. (2020) |
| *Share of Asymptomatic Infections* | $a$ | 0.4 | Oran and Topol (2020) |
| *Share of Mild Infections* | $m$ | 0.3 | Oran and Topol (2020) |
| *Share of Severe Infections* | $s$ | 0.3 | Oran and Topol (2020) |
| *Implied Infection Fatality Risk for Young* | $\phi^y$ | 0.002 | Levin et al. (2020) |
| *Implied Infection Fatality Risk for Old* | $\phi^o$ | 0.074 | Levin et al. (2020) |
| *Incubation Period* | $p$ | 7 | Qin et al. (2020) |
| *Symptoms to Death* | $\tilde{k}_s$ | 10 | U.S. CDC (2020) |
| *Symptoms to Recovery for Severe* | $\tilde{q}_s$ | 10 | U.S. CDC (2020) |
| *Symptoms to Recovery for Non-Severe* | $\tilde{q}_s, \tilde{q}_a$ | 7 | U.S. CDC (2020) |
| *Transmission Coefficient* | $\beta$ | 0.20 | |
| *Daily Productivity for Young* | $A^y$ | 230 | U.S. BLS (2019) |
| *Daily Productivity for Old* | $A^o$ | 46 | U.S. BLS (2019) |
| *Pre-Epidemic Contact Rate Young-Young* | $\rho_0^{yy}$ | 0.95 | Prem et al. (2017) |
| *Pre-Epidemic Contact Rate Young-Old* | $\rho_0^{yo}$ | 0.05 | Prem et al. (2017) |
| *Pre-Epidemic Contact Rate Old-Old* | $\rho_0^{oo}$ | 0.24 | Prem et al. (2017) |
| *Pre-Epidemic Contact Rate Old-Young* | $\rho_0^{oy}$ | 0.76 | Prem et al. (2017) |
| *Elasticity of Labor to Perceived Death Risk* | $\varepsilon_n$ | 5000 | Empirical Estimates |
| *Elasticity of Leisure to Perceived Death Risk* | $\varepsilon_l$ | 5000 | Empirical Estimates |

**Table 5:** A SARS-CoV-2 Calibration

Daily productivity is set as follows. I start from daily GDP per-capita in the U.S. and allocate it to each group taking into account that young individuals comprise roughly 84% of the population and that their employment rate is roughly 5 times higher than that of the old. Contact rates for the U.S. are aggregated from the dataset produced by Prem et al. (2017), which I first correct for non-reciprocity using the pairwise correction suggested by Arregui et al. (2018). Finally, the elasticities to the perceived risk of dying are set equal to the value estimated in the empirical part of the paper.[40]

Let's consider the benchmark case in which the health-care system tests only severe symptomatic individuals. Figure 11 reports the dynamic evolution of total true latent cases and deaths, GDP and the public deficit when the government provides aggregate testing data (in blue) and when it provides disaggregated testing data (in orange). The figure suggests that heterogeneous risk perceptions across the two age groups result in higher total cases, but lower deaths, output losses and budget deficits.[41]

[40] I choose the raw estimate for the Google Mobility Data at the state-level since it corresponds precisely to an elasticity. Moreover, I do not pick the estimate from a specification with time fixed-effects, as that would capture a *relative* elasticity, which is not the object of interest.

[41] The importance of heterogeneous risk perceptions generalizes to all testing levels, as shown by Figure A9 in Online Appendix D.5. Across testing levels and relative to the case where all individuals have the same perceptions of risk, heterogeneous risk perceptions across young and old individuals reduce GDP losses and budget deficits as much as 50%, and the total deaths count as much as 30%. Furthermore, the testing multiplier is on average positive in both scenarios, as shown by Figure A10.

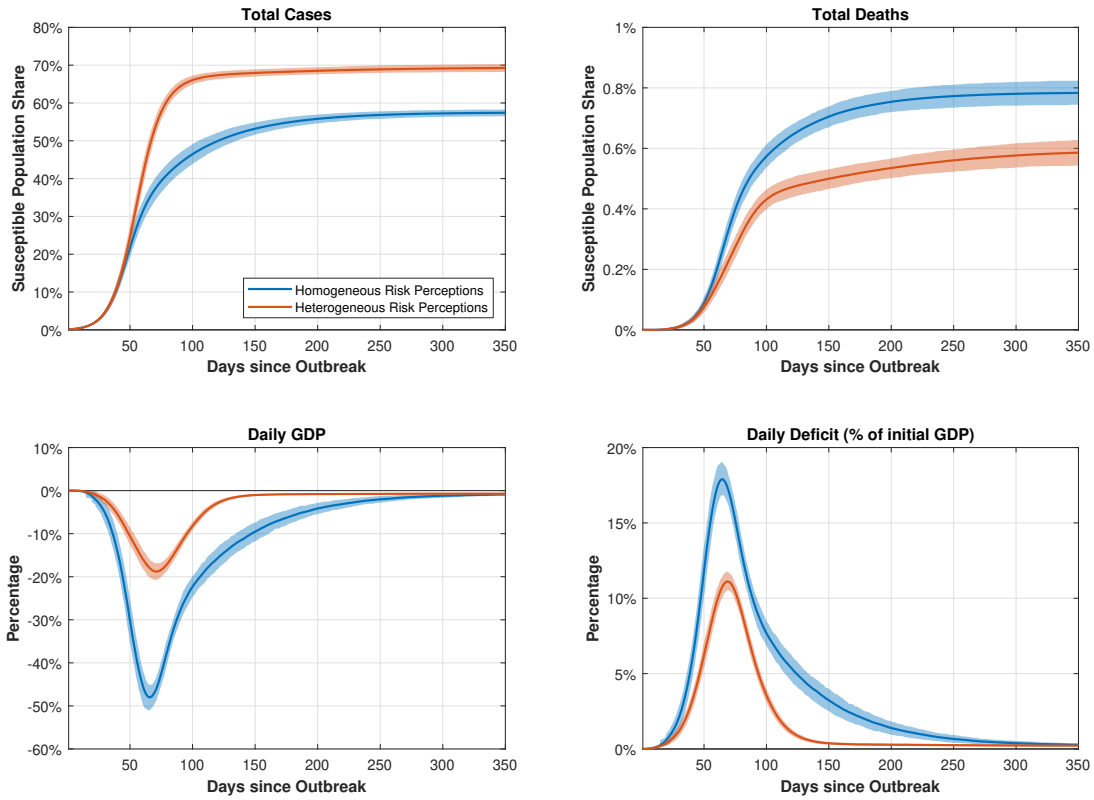


**Figure 11:** Economic and Health Outcomes for the SARS-CoV-2 Calibration of the Model

Figure 12 helps understand why. When the government provides aggregate testing data and both the young and the old share the same perceived death risk, young agents *over-estimate* their true risk, while old agents *under-estimate* theirs. This happens because the "aggregate" case fatality rate estimates the average conditional infection fatality risk across the two groups, which makes the disease appear more lethal than it actually is to the young, and less lethal to the old. This results in less total true cases, since the young (who constitute the largest share of the population) protect themselves a lot, but more deaths, since the old (whose true risk of dying is higher) do not protect themselves enough.

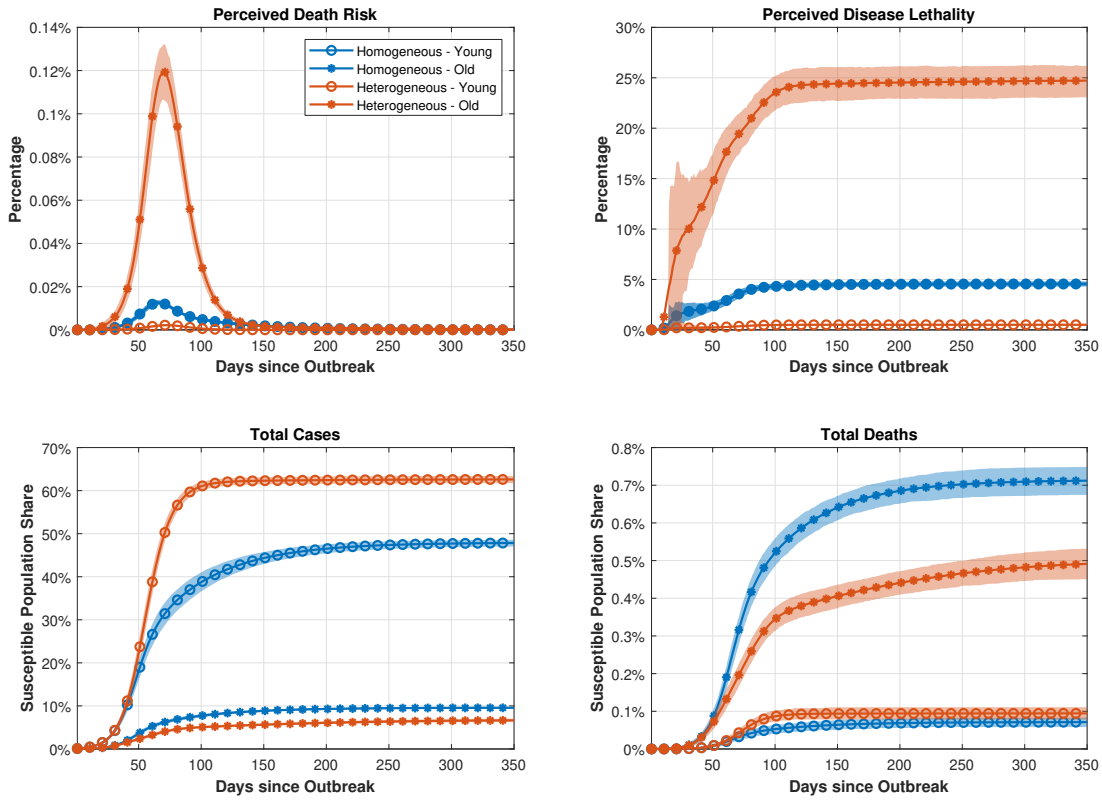


**Figure 12:** The Effect of Heterogeneous Perceptions on Health Outcomes

When the government releases disaggregated testing data, instead, each group develops a more accurate understanding of the disease. As a result, young agents fear the epidemic disease less and reduce their activity less, which results in more cases and deaths among them. The opposite happens with the old, who now fear the disease more.

Figure 13 looks at contact rates and production. With homogeneous risk perceptions, the fall in production and contact rates is homogeneous across groups. With heterogeneous perceptions, instead, production and interactions of the old fall sizeably, but those of the young remain close to pre-epidemic levels. Since young agents account for most of the population and are the most productive, economic activity falls more when they reduce labor supply. This explains why heterogeneous risk perceptions are associated with better economic outcomes than homogeneous perceptions in this calibration of the model.

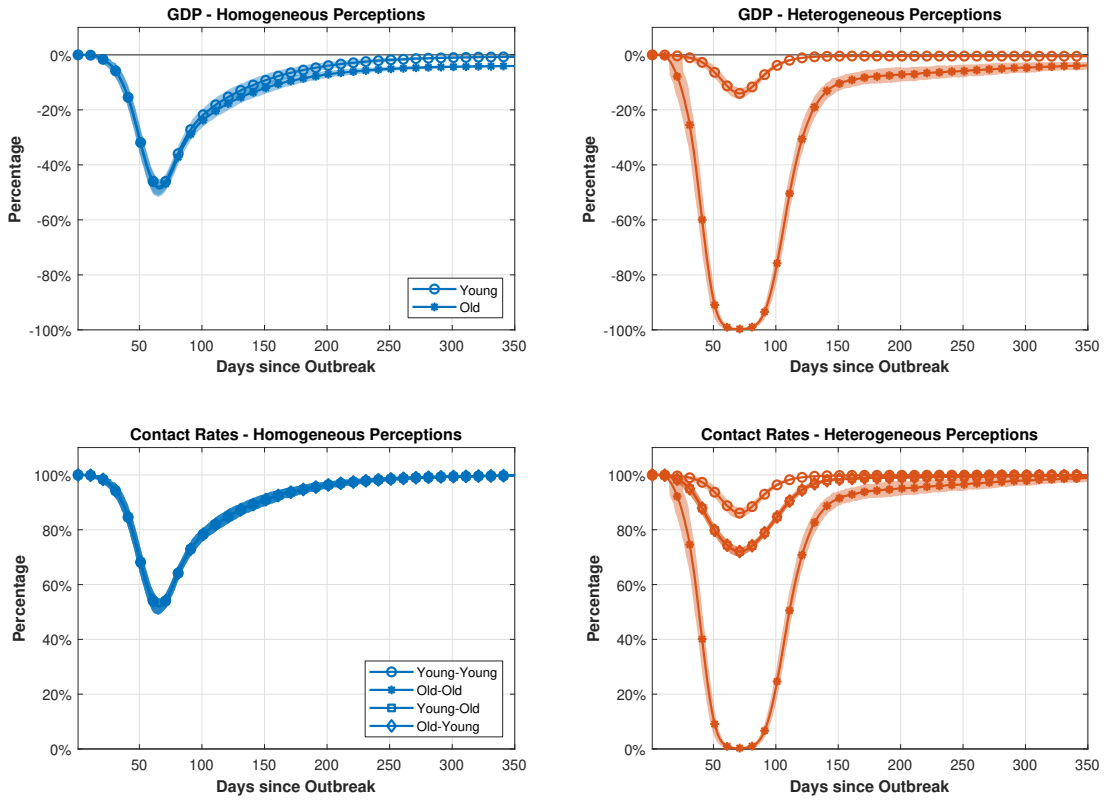


**Figure 13:** The Effect of Heterogeneous Perceptions on Economic Outcomes

Importantly, the bottom-right panel of the figure shows the reduced-form infection externality across groups that is at play in the model. Old individuals, who entertain a sizeable share of their interactions with young ones, are unable to reduce the between-group interactions as much as they can reduce their own, and the opposite happens to the young.[42]

Importantly, the overall effect of heterogeneous risk perceptions across age groups on aggregate economic and public health outcomes depend on the population structure, the productivity of the various groups, the pattern of interactions among them, and the characteristics of the disease. In Online Appendix E, I illustrate how heterogeneous risk perceptions improve health but not economic outcomes for a disease that is more lethal for the young than for the old.

[42] With a little stretch, one can think of heterogeneous risk perceptions as a way to implement an (imperfect) targeted lockdown through behavioral responses. This 'endogenous' lockdown is likely to be sub-optimal because of the infection externality.

# 8 Conclusions

The analysis performed is not aimed at describing any actual epidemic, and its only purpose is to provide insights on the economic effects of testing. It suffers from limitations. The key ingredient in the analysis is the way agents process information, which in turn determines their behavioral responses. The specification proposed in the model is stylized, and takes a strong stance on the source and the type of information exploited by individuals to form their risk perceptions. As such, it should only be seen as a first attempt and more work is needed to understand the details of individual behavior.

The analysis also abstracts from many sources of heterogeneity that could potentially play a role in determining the effects of testing. For example, spatial heterogeneity creates room for geographically-targeted large-scale testing. This could permit epidemic containment without testing a sizeable share of the overall population every day.

These limitations, however, are unlikely to upend the main implications for policy-making. Frugal governments who do not allocate enough resources to large-scale screening of the population might inadvertedly end up damaging the economy and widen public deficits. Allocating enough resources to large-scale testing would improve their financial situation, as unintuitively as it may appear. Moreover, information provision emerges as a low-cost non-pharmaceutical intervention able to support both public health and the economy.

# References


Acemoglu, D., V. Chernozhukov, I. Werning, and M. D. Whinston (2020): "A multi-risk SIR model with optimally targeted lockdown," Tech. rep., National Bureau of Economic Research.

Arregui, S., A. Aleta, J. Sanz, and Y. Moreno (2018): "Projecting social contact matrices to different demographic structures," *PLoS computational biology*, 14, e1006638.

Atkeson, A. (2020): "How Deadly Is COVID-19? Understanding The Difficulties With Estimation Of Its Fatality Rate," Working Paper 26965, National Bureau of Economic Research.

Atkeson, A., M. Droste, M. J. Mina, and J. H. Stock (2020): "Economic Benefits of COVID-19 Screening Tests," *medRxiv*.

Berger, D., K. Herkenhoff, C. Huang, and S. Mongey (2020): "Testing and reopening in an SEIR model," *Review of Economic Dynamics*.

Brotherhood, L., P. Kircher, C. Santos, and M. Tertilt (2020): "An economic model of the Covid-19 epidemic: The importance of testing and age-specific policies," .

Chande, A., S. Lee, M. Harris, Q. Nguyen, S. J. Beckett, T. Hilley, C. Andris, and J. S. Weitz (2020): "Real-time, interactive website for US-county-level COVID-19 event risk assessment," *Nature Human Behaviour*, 1–7.

Cilek, L., G. Chowell, and D. Ramiro Fariñas (2018): "Age-specific excess mortality patterns during the 1918–1920 influenza pandemic in Madrid, Spain," *American journal of epidemiology*, 187, 2511–2523.

Eichenbaum, M., M. G. de Matos, F. Lima, S. Rebelo, and M. Trabandt (2020a): "How do People Respond to Small Probability Events with Large, Negative Consequences?" .

Eichenbaum, M. S., S. Rebelo, and M. Trabandt (2020b): "The macroeconomics of epidemics," Tech. rep., National Bureau of Economic Research.

——— (2020c): "The Macroeconomics of Testing and Quarantining," Working Paper 27104, National Bureau of Economic Research.

Feng, Z. (2007): "Final and peak epidemic sizes for SEIR models with quarantine and isolation," *Mathematical Biosciences & Engineering*, 4, 675.

Feng, Z., D. Xu, and H. Zhao (2007): "Epidemiological models with non-exponentially distributed disease stages and applications to disease control," *Bulletin of mathematical biology*, 69, 1511–1536.

Funk, S., M. Salathé, and V. A. Jansen (2010): "Modelling the influence of human behaviour on the spread of infectious diseases: a review," *Journal of the Royal Society Interface*, 7, 1247–1256.

Gallagher, S. and J. Baltimore (2017): "Comparing compartment and agent-based models," .

Goolsbee, A., N. B. Luo, R. Nesbitt, and C. Syverson (2020): "COVID-19 Lockdown Policies at the State and Local Level," *University of Chicago, Becker Friedman Institute for Economics Working Paper*.

Goolsbee, A. and C. Syverson (2020): "Fear, lockdown, and diversion: Comparing drivers of pandemic economic decline 2020," *Journal of public economics*, 193, 104311.

Hunter, E., B. Mac Namee, and J. D. Kelleher (2018): "A Comparison of Agent-Based Models and Equation Based Models for Infectious Disease Epidemiology." 33–44.

Jones, C. J., T. Philippon, and V. Venkateswaran (2020): "Optimal mitigation policies in a pandemic: Social distancing and working from home," Tech. rep., National Bureau of Economic Research.

Jung, C.-Y., H. Park, D. W. Kim, Y. J. Choi, S. W. Kim, and T. I. Chang (2020): "Clinical characteristics of asymptomatic patients with COVID-19: a nationwide cohort study in South Korea," *International Journal of Infectious Diseases*, 99, 266–268.

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

Korolev, I. (2020): "Identification and Estimation of the SEIRD Epidemic Model for COVID-19," *Journal of Econometrics*.

Larremore, D. B., B. Wilder, E. Lester, S. Shehata, J. M. Burke, J. A. Hay, M. Tambe, M. J. Mina, and R. Parker (2020): "Test sensitivity is secondary to frequency and turnaround time for COVID-19 surveillance," *MedRxiv*.

Levin, A. T., K. B. Cochran, and S. P. Walsh (2020): "Assessing the age specificity of infection fatality rates for COVID-19: Meta-analysis & public policy implications," Tech. rep., National Bureau of Economic Research.

Lipsitch, M., C. A. Donnelly, C. Fraser, I. M. Blake, A. Cori, I. Dorigatti, N. M. Ferguson, T. Garske, H. L. Mills, S. Riley, et al. (2015): "Potential biases in estimating absolute and relative case-fatality risks during outbreaks," *PLoS neglected tropical diseases*, 9, e0003846.

Mina, M. J., R. Parker, and D. B. Larremore (2020): "Rethinking Covid-19 test sensitivity—A strategy for containment," *New England Journal of Medicine*.

Mistry, D., M. Litvinova, M. Chinazzi, L. Fumanelli, M. F. Gomes, S. A. Haque, Q.-H. Liu, K. Mu, X. Xiong, M. E. Halloran, et al. (2020): "Inferring high-resolution human mixing patterns for disease modeling," *arXiv preprint arXiv:2003.01214*.

Murray, E. J. (2020): "Epidemiology's Time of Need: COVID-19 Calls for Epidemic-Related Economics," *Journal of Economic Perspectives*, 34, 105–20.

Oran, D. P. and E. J. Topol (2020): "Prevalence of Asymptomatic SARS-CoV-2 Infection: A Narrative Review," *Annals of Internal Medicine*.

Piguillem, F. and L. Shi (2020): "Optimal COVID-19 quarantine and testing policies," .

Prem, K., A. R. Cook, and M. Jit (2017): "Projecting social contact matrices in 152 countries using contact surveys and demographic data," *PLoS computational biology*, 13, e1005697.

Qin, J., C. You, Q. Lin, T. Hu, S. Yu, and X.-H. Zhou (2020): "Estimation of incubation period distribution of COVID-19 using disease onset forward time: a novel cross-sectional and forward follow-up study," *medRxiv*.

Sukumar, S. R. and J. J. Nutaro (2012): "Agent-based vs. equation-based epidemiological models: A model selection case study," 74–79.

Taipale, J., P. Romer, and S. Linnarsson (2020): "Population-scale testing can suppress the spread of COVID-19," *medRxiv*.

Verelst, F., L. Willem, and P. Beutels (2016): "Behavioural change models for infectious disease transmission: a systematic review (2010–2015)," *Journal of The Royal Society Interface*, 13, 20160820.

Wang, Z., M. A. Andrews, Z.-X. Wu, L. Wang, and C. T. Bauch (2015): "Coupled disease–behavior dynamics on complex networks: A review," *Physics of life reviews*, 15, 1–29.

# Online Appendix

## A Data Appendix

### A.1 Data Sources

The analysis in section 3 is performed combining data from the following data sources:

- *USAFacts*: county-level data on cases, deaths and population
- *COVID Tracking Project*: state-level data on cases, deaths and testing
- *Google Mobility Report*: state-level data on workplace mobility
- *Dallas FED's MEI*: county- and state-level mobility and engagement index

### A.2 Additional Regression Results

| | **FED's MEI** | | | **Google's Workplace Mobility** | | |
|---|---|---|---|---|---|---|
| | (1) OLS | (2) FE | (3) FE | (1) OLS | (2) FE | (3) FE |
| **Spec #1** | | | | | | |
| *Death Risk ($\chi$)* | -9294.2*** | -9503.8*** | -2887.5*** | -5265.0*** | -5921.2*** | -1183.2*** |
| | (983.5) | (868.9) | (487.4) | (579.3) | (622.1) | (179.2) |
| **Spec #2** | | | | | | |
| *Lethality (CFR)* | -4.42*** | -4.59*** | -0.41 | -2.66*** | -2.94*** | -0.20 |
| | (0.74) | (1.01) | (0.28) | (0.43) | (0.63) | (0.12) |
| *Infection Risk (IR)* | -160.6*** | -160.5*** | -88.7*** | -127.5*** | -142.3*** | -40.3*** |
| | (44.0) | (46.8) | (15.4) | (16.1) | (15.5) | (5.3) |
| State FE | N | Y | Y | N | Y | Y |
| Time FE | N | N | Y | N | N | Y |
| Adj. $R^2$ (Spec #1) | 0.15 | 0.17 | 0.96 | 0.20 | 0.25 | 0.97 |
| Adj. $R^2$ (Spec #2) | 0.19 | 0.21 | 0.96 | 0.31 | 0.37 | 0.97 |
| Obs | 1530 | 1530 | 1530 | 1479 | 1479 | 1479 |

**Notes:** Clustered standard errors at the state-level in parenthesis. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$

**Table A1:** Main Regression Results at State-Level, Non-Standardized Coefficients

| | FED's MEI | | | | |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| | OLS | FE | FE | FE | FE |
| **Spec #1** | | | | | |
| *Death Risk ($\chi$)* | -1975.4*** | -1939.8*** | -910.7*** | -817.4*** | -817.4** |
| | (182.2) | (153.6) | (78.7) | (124.7) | (323.3) |
| **Spec #2** | | | | | |
| *Lethality (CFR)* | -1.04*** | -1.20*** | -0.07*** | -0.12*** | -0.12*** |
| | (0.07) | (0.07) | (0.02) | (0.03) | (0.03) |
| *Infection Risk (IR)* | -11.98*** | -10.45*** | -19.79*** | -14.88*** | -14.88** |
| | (2.85) | (2.44) | (3.15) | (3.29) | (5.97) |
| County FE | N | Y | Y | N | N |
| Time FE | N | N | Y | N | N |
| State-Time FE | N | N | N | Y | Y |
| SE Clustering | County | County | County | County | State |
| Adj. $R^2$ (Spec #1) | 0.01 | 0.14 | 0.90 | 0.81 | 0.81 |
| Adj. $R^2$ (Spec #2) | 0.03 | 0.16 | 0.90 | 0.81 | 0.81 |
| Obs | 90599 | 90599 | 90599 | 90599 | 90599 |

**Notes:** Clustered standard errors in parenthesis. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$

**Table A2:** Main Regression Results at County-Level, Non-Standardized Coefficients

| | State-Level | | | | County-Level | | | |
|---|---|---|---|---|---|---|---|---|
| **FED's MEI** | (1) FE | (2) FE | (3) FE | (4) FE | (5) FE | (6) FE | (7) FE | (8) FE |
| *Death Risk* ($\chi$) | | -11.27*** | | -10.23*** | | -3.07*** | | -2.47*** |
| | | (1.92) | | (1.94) | | (0.56) | | (0.56) |
| *Cases* | -5.74*** | -1.55 | | | -7.45*** | -7.48*** | | |
| | (1.69) | (1.48) | | | (2.84) | (2.79) | | |
| *Deaths* | -0.37 | -3.43** | | | -1.05 | -0.61 | | |
| | (1.16) | (1.40) | | | (1.65) | (1.60) | | |
| $\Delta$*Cases* | | | -3.22*** | -2.02 | | | -6.85*** | -6.85*** |
| | | | (1.08) | (1.94) | | | (1.93) | (1.89) |
| $\Delta$*Deaths* | | | -5.60* | -0.34 | | | -1.81* | 1.24 |
| | | | (3.13) | (2.84) | | | (1.08) | (1.04) |
| State FE | Y | Y | Y | Y | N | N | N | N |
| Time FE | Y | Y | Y | Y | N | N | N | N |
| State-Time FE | N | N | N | N | Y | Y | Y | Y |
| SE Clustering | State | State | State | State | County | County | County | County |
| Adj. $R^2$ | 0.96 | 0.96 | 0.96 | 0.96 | 0.82 | 0.82 | 0.82 | 0.82 |
| Obs | 1530 | 1530 | 1530 | 1530 | 90569 | 90569 | 90569 | 90569 |

**Notes:** Clustered standard errors in parenthesis. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. The dependent variable is the FED's Mobility and Engagement Index (MEI). Standardized coefficients (%) obtained by scaling variables by their standard deviation.

**Table A3:** Additional Regression Results on Reported Cases and Deaths

| | FED's MEI | | Workplace Mobility | |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| | FE | FE | FE | FE |
| *Death Risk ($\chi$)* | | -8.96*** | | -8.53*** |
| | | (1.64) | | (1.15) |
| *Test Positivity Rate* | -2.24** | -1.49 | -2.24** | -0.82 |
| | (1.09) | (0.95) | (1.09) | (0.95) |
| State FE | Y | Y | Y | Y |
| Time FE | Y | Y | Y | Y |
| Adj. $R^2$ | 0.96 | 0.96 | 0.97 | 0.97 |
| Obs | 1318 | 1318 | 1318 | 1318 |

**Notes:** Clustered standard errors at the state-level. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. Standardized coefficients (%) obtained by scaling variables by their standard deviation.

**Table A4:** Additional Regression Results on Test Positivity Rate

# B Model Details

## B.1 Epidemic and Confounding Diseases

**Epidemic Disease** For a generic individual $j$ who has been infected by the epidemic disease at a generic time $\tilde{t}$, we have that

$$c_t^*(j) = \begin{cases} 1 & \text{if } t \geq \tilde{t} \\ 0 & \text{if } t < \tilde{t} \end{cases}$$

To model the type of symptoms developed and the terminal outcome for a generic individual $j$, I introduce two random variables: $\text{symptoms}^*(j)$ describing the type of symptoms developed, and $\text{death}^*(j)$ to denote the terminal outcome of the disease. Their *joint probability distribution* is given by:

| | recovers | dies | |
|---|---|---|---|
| severe symptoms | $s \cdot (1 - \phi_s)$ | $s \cdot \phi_s$ | $s$ |
| mild symptoms | $m \cdot (1 - \phi_m)$ | $m \cdot \phi_m$ | $m$ |
| asymptomatic | $a \cdot (1 - \phi_a)$ | $a \cdot \phi_a$ | $a$ |
| | $1 - \phi$ | $\phi$ | |

Notice that $\phi$ is the *unconditional infection fatality risk*, i.e. the probability that an individual who contracts the epidemic disease dies, while $\phi_s$, $\phi_m$ and $\phi_a$ denote the *conditional infection fatality risks*, i.e. the probability that an individual who contracts the epidemic disease and exhibits a certain type of symptoms dies.

The timing of these random events is random itself. In particular, for each individual $j$, the random variable $p^*(j)$ represents the length of the incubation period or, equivalently, the number of days the individual spends in the pre-symptomatic state; $\tilde{k}^*(j)$ represents the number of days between the onset of symptoms and the terminal outcome death; $\tilde{q}^*(j)$ represents the number of days between the onset of symptoms and the terminal outcome recovery. I assume that these lags do not depend on the type of symptoms developed, nor on the terminal outcome, and that they are distributed as Poisson random variables:[1]

$$\begin{aligned} p^*(j), \tilde{k}^*(j), \tilde{q}^*(j) &\perp \text{symptoms}^*(j), \text{death}^*(j) \\ p^*(j) &\sim \text{Poisson}(p - 1) + 1 \\ \tilde{k}^*(j) &\sim \text{Poisson}(\tilde{k}) \\ \tilde{q}^*(j) &\sim \text{Poisson}(\tilde{q}) \end{aligned}$$

[1] Standard stochastic SIR-type models assume that these timings are exponentially distributed, as this assumption allows the aggregation of individuals into compartments, resulting in a noticeable simplification of the problem thanks to the memorylessness property of the exponential random variables. See Feng (2007) and Feng et al. (2007) for more details.

I opt for a shifted-Poisson distribution of $p^*(j)$ so that it takes at least one period - i.e. one day - between infection and the terminal outcome. The number of days between infection and terminal outcomes are therefore given by:

$$k^*(j) = p^*(j) + \tilde{k}^*(j) \sim \text{Poisson}(p + \tilde{k} - 1) + 1$$
$$q^*(j) = p^*(j) + \tilde{q}^*(j) \sim \text{Poisson}(p + \tilde{q} - 1) + 1$$

Analytically, the dynamic evolution of the disease for a generic individual $j$ can be expressed as follows:

$$\begin{aligned}
u_t^*(j) &= c_t^*(j) - c_{t-p^*(j)}^*(j) \\
d_t^*(j) &= \text{death}^*(j) \cdot c_{t-k^*(j)}^*(j) \\
r_t^*(j) &= \left[1 - \text{death}^*(j)\right] \cdot c_{t-q^*(j)}^*(j) \\
s_t^*(j) &= \text{severe}^*(j) \cdot [c_{t-p^*(j)}^*(j) - d_t^*(j) - r_t^*(j)] \\
m_t^*(j) &= \text{mild}^*(j) \cdot [c_{t-p^*(j)}^*(j) - d_t^*(j) - r_t^*(j)] \\
a_t^*(j) &= \text{asymptomatic}^*(j) \cdot [c_{t-p^*(j)}^*(j) - d_t^*(j) - r_t^*(j)] \\
i_t^*(j) &= c_t^*(j) - r_t^*(j) - d_t^*(j) = u_t^*(j) + s_t^*(j) + m_t^*(j) + a_t^*(j)
\end{aligned}$$

where $u_t^*(j)$ is one when the individual in the incubation period (and their symptoms are still Unknown), $d_t^*(j)$ and $r_t^*(j)$ are, respectively, one when the individual dies or recover, $s_t^*(j)$ is one when the individual displays severe symptoms, $m_t^*(j)$ is one when the individual displays mild symptoms and $a_t^*(j)$ is one when the individual is asymptomatic. Furthermore, $i_t^*(j)$ is one when the individual has an active infection, and becomes zero again when the infection is no longer active (either because of recovery or death). At the same time, an active infection can manifest itself in four forms: incubation period, severe symptoms, mild symptoms or lack of symptoms. Finally, notice that $c_t^*(j)$, $d_t^*(j)$, $r_t^*(j)$ are *absorbing states* that, if reached, are never left, while $u_t^*(j)$, $s_t^*(j)$, $m_t^*(j)$, $a_t^*(j)$, $i_t^*(j)$ are *transient states*.

**Confounding Disease** The confounding disease is a simplified version of the epidemic disease. There is no incubation period, and only two types of symptoms. Two random events determine the type of symptoms and the final outcome:

$$\text{severe}^{f*}(j) = \begin{cases} \text{severe}(\equiv 1) & \text{wp } s^f \\ \text{mild}(\equiv 0) & \text{wp } 1 - s^f \end{cases} \qquad \text{death}^{f*}(j) = \begin{cases} 1 & \text{wp } \phi^f \\ 0 & \text{wp } 1 - \phi^f \end{cases}$$

The timing of the terminal outcome is described by $k^{f*}(j)$, which represents the number of periods between infection and death, and by $q^{f*}(j)$, which represents the number of periods between infection and recovery. For simplicity, I assume that these lags are degenerate and independent of the type of symptoms, and that the terminal outcome is independent of the type of symptoms.

Analytically, the evolution of the confounding disease at the individual level is given by:

$$
\begin{aligned}
d_t^{f^*}(j) &= \text{death}^{f^*}(j) \cdot c_{t-k^{f^*}}^{f^*}(j) \\
r_t^{f^*}(j) &= \left[1 - \text{death}^{f^*}(j)\right] \cdot c_{t-q^{f^*}}^{f^*}(j) \\
s_t^{f^*}(j) &= \text{severe}^{f^*}(j) \cdot \left[c_t^{f^*}(j) - d_t^{f^*}(j) - r_t^{f^*}(j)\right] \\
m_t^{f^*}(j) &= \left[1 - \text{severe}^{f^*}(j)\right] \cdot \left[c_t^{f^*}(j) - d_t^{f^*}(j) - r_t^{f^*}(j)\right] \\
i_t^{f^*}(j) &= c_t^{f^*}(j) - r_t^{f^*}(j) - d_t^{f^*}(j) = s_t^{f^*}(j) + m_t^{f^*}(j)
\end{aligned}
$$

## B.2 Testing Policies

The health-care system's testing policy is implemented using set theory. First, by assumption **T1**, all severe symptomatic individuals that need to get tested are tested. The set of severe symptomatic individuals tested at time $t$ is given by

$$\mathcal{T}_t^S = \Sigma_t^S \setminus (C_{t-1} \cup \mathcal{T}_t^p)$$

and consists of the individuals displaying severe symptoms ($\Sigma_t^S$), minus those that have been diagnosed with the disease in the past ($C_{t-1}$), minus those whose test result is still pending ($\mathcal{T}_t^p$).

When the government mandates additional testing capacity (i.e. $T_t^{NS} > 0$), the health-care system expands testing to mild symptomatic individuals. The set of individuals that it would like to test is given by

$$\mathcal{G}_t^M = \Sigma_t^M \setminus (C_{t-1} \cup \mathcal{T}_t^p)$$

where $\Sigma_t^M$ is the set of individuals displaying mild symptoms. A random subset $\mathcal{T}_t^M \subseteq \mathcal{G}_t^M$ of size equal to $T_t^M = |\mathcal{T}_t^M| = \min\{T_t^{NS}, |\mathcal{G}_t^M|\}$ is tested.

After individuals with mild symptoms are tested, the health-care system starts testing asymptomatic individuals if there is additional testing capacity, i.e. $T_t^{NS} - T_t^M > 0$. The set of individuals that it would like to test is given by

$$\mathcal{G}_t^A = \left(\mathcal{P}_t \setminus (\Sigma_t^S \cup \Sigma_t^M)\right) \setminus (C_{t-1} \cup \mathcal{T}_t^p)$$

where $\mathcal{P}_t$ is the set of alive individuals. A random subset $\mathcal{T}_t^A \subseteq \mathcal{G}_t^A$ is tested, and its size is equal to $T_t^A = |\mathcal{T}_t^A| = \min\{T_t^{NS} - T_t^M, |\mathcal{G}_t^A|\}$.

The set of all individuals tested at a generic time $t$ is given by

$$\mathcal{T}_t = \mathcal{T}_t^S \cup \mathcal{T}_t^M \cup \mathcal{T}_t^A$$

and the total number of tests performed is given by $T_t = |\mathcal{T}_t|$. Tests can turn out to be either

positive or negative, i.e. $\mathcal{T}_t = \mathcal{T}_t^+ \cup \mathcal{T}_t^-$, and positive tests are given by:[2]

$$\mathcal{T}_t^+ \subseteq \mathcal{I}_t^* \cap \mathcal{T}_t$$

where $x \in \mathcal{I}_t^* \cap \mathcal{T}_t$ also belongs to $\mathcal{T}_t^+$ with probability $1 - \alpha$, where $\alpha$ is the false negativity rate of the testing technology. Because of the delay in test results, positive cases are known only with a delay:

$$C_t = C_{t-1} \cup \mathcal{T}_{t-d}^+$$

At time $t$, the list of pending test results is given by

$$\mathcal{T}_t^p = \bigcup_{index=1}^{d} \mathcal{T}_{t-index}$$

with $\mathcal{T}_t^p = \emptyset$ if $d = 0$. Finally, given the list of detected cases $C_t$, one can recover any detected set $\mathcal{Z}_t$ as follows:[3]

$$\mathcal{Z}_t = C_t \cap \mathcal{Z}_t^*$$

where $\mathcal{Z}_t^*$ is its latent counterpart. Reported epidemic time-series are then given by $Z_t = |\mathcal{Z}_t|$. In other words, once an individual enters the list of positive cases, her health-status is perfectly known by the health-care system.

[2] I implicitly assume that all tests administered share the same technological characteristics. This can be easily relaxed, for example, by assuming that severe symptomatic individuals receive a different type of test from the rest of the population, as done in Atkeson et al. (2020).

[3] To be clear, what I refer to as 'detected' cases are laboratory-confirmed cases and are not indirect estimates obtained in other ways.

## C Recovering the Deterministic SIR Model

It is possible to recover standard textbook epidemiological models by imposing specific restrictions to the model. In this section, I consider the homogeneous population version of the model and show how to recover the deterministic SIR model. To this end:

- Eliminate the confounding disease by setting $\omega^f = \sigma^f = 0$
- Eliminate severe and mild symptomatic states by setting $s = m = 0$
- Eliminate the incubation period by setting $p(j) = 0$
- Eliminate non-severe testing by setting $T_t^{NS} = 0$
- Eliminate behavioral responses by setting $\varepsilon_l = \varepsilon_n = 0$
- Eliminate any death risk by setting $\phi_s = \phi_m = \phi_a = \phi = 0$
- Assume an exponential form for the time from infection to recovery, i.e. $q(j) \sim Exp(q)$
- Set population size to infinity, i.e. $P_0 \longrightarrow +\infty$

The resulting aggregate epidemic dynamics is given by:

$$\begin{aligned}
\Delta X_{t+1}^* &= -\beta \cdot \frac{I_t^*}{P_0} \cdot X_t^* \\
\Delta I_{t+1}^* &= \beta \cdot \frac{I_t^*}{P_0} \cdot X_t^* - \gamma \cdot I_t^* \\
\Delta R_{t+1}^* &= -\gamma \cdot I_t^*
\end{aligned}$$

where $\gamma = \frac{1}{q}$. Then, define $w_t = \frac{W_t}{P_0}$ for a generic variable $W_t$, and conveniently drop the asterisk denoting latent variables. Divide both sides of each equation by $P_0$ to get:

$$\begin{aligned}
\Delta x_{t+1} &= -\beta \cdot i_t \cdot x_t \\
\Delta i_{t+1} &= \beta \cdot i_t \cdot x_t - \gamma \cdot i_t \\
\Delta r_{t+1} &= -\gamma \cdot i_t
\end{aligned}$$

The equations above are the same as for a deterministic SIR model in discrete time. Figure A1 below provides a visual representation of the aggregate epidemic dynamic under the restrictions above. In the simulation, I set $\beta = 0.30$, $\gamma = \frac{1}{14}$ and increase the population size to $P_0 = 1e6$.

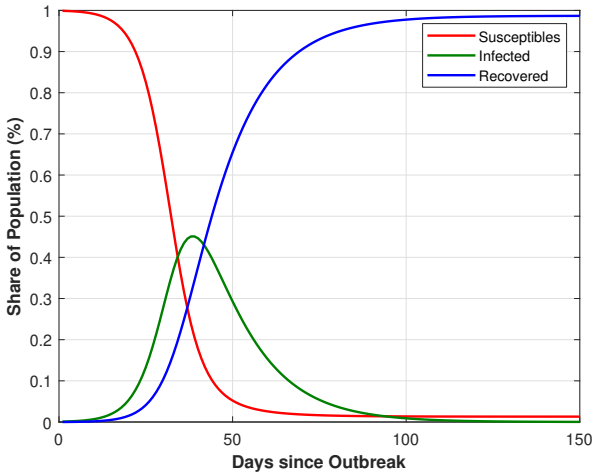


**Figure A1:** Recovering the SIR Textbook Model

# D Additional Results

## D.1 More on the Role of Behavioral Responses

It is insightful to dig deeper into the role of behavioral responses in the model. By construction, the model allows to control the intensity of behavioral responses, and, in Figure A2 below, I show what happens with weaker ($\varepsilon_l = \varepsilon_n = 500$) and stronger ($\varepsilon_l = \varepsilon_n = 1500$) behavioral responses.

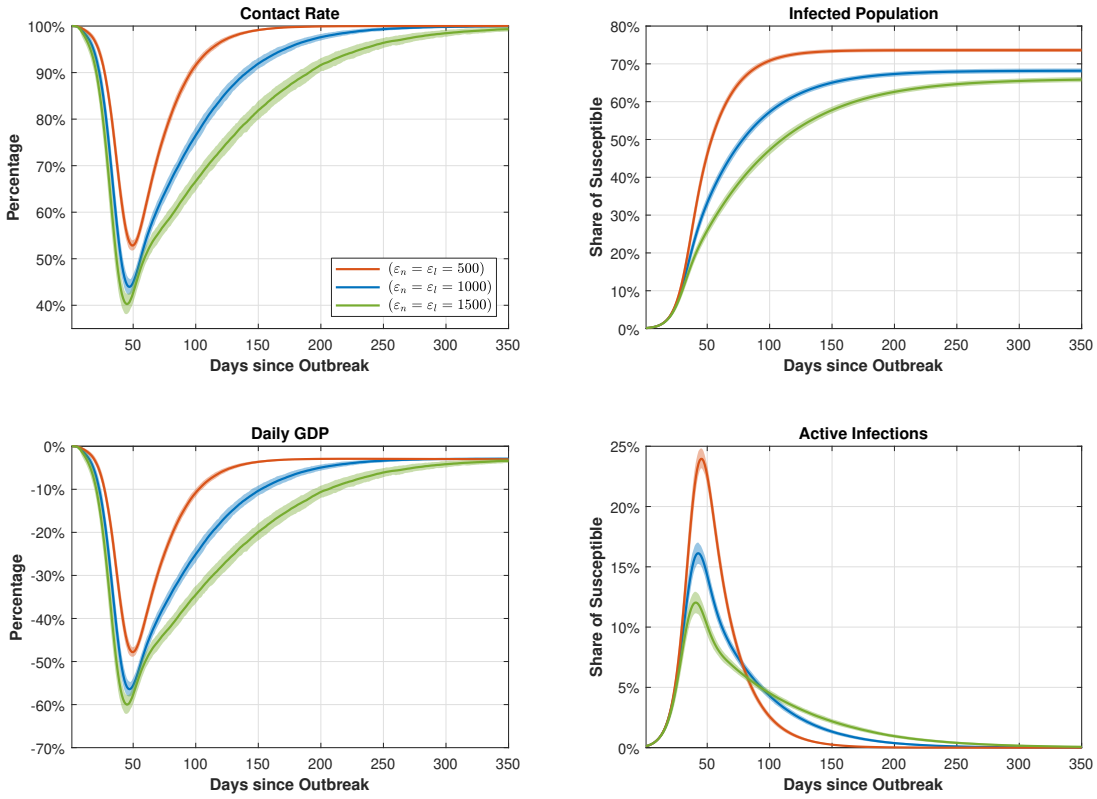


**Figure A2:** The Importance of Behavioral Responses

Stronger behavioral responses generate a larger fall in labor supply and enjoyment of leisure for the same perceived risk of dying. This translates into a greater reduction of the interactions between agents, and, in turn, into a smaller final epidemic size. At the same time, lower labor supply causes a sharper contraction of economic activity. Furthermore, stronger behavioral responses "flatten the curve" and lengthen the horizon over which the epidemic disease naturally disappears, as shown in the bottom-right panel.

## D.2 The Surplus-Multiplier for Alternative Influenza-Like Diseases

Figure A3 below reports the Surplus-Multiplier for alternative epidemic diseases:

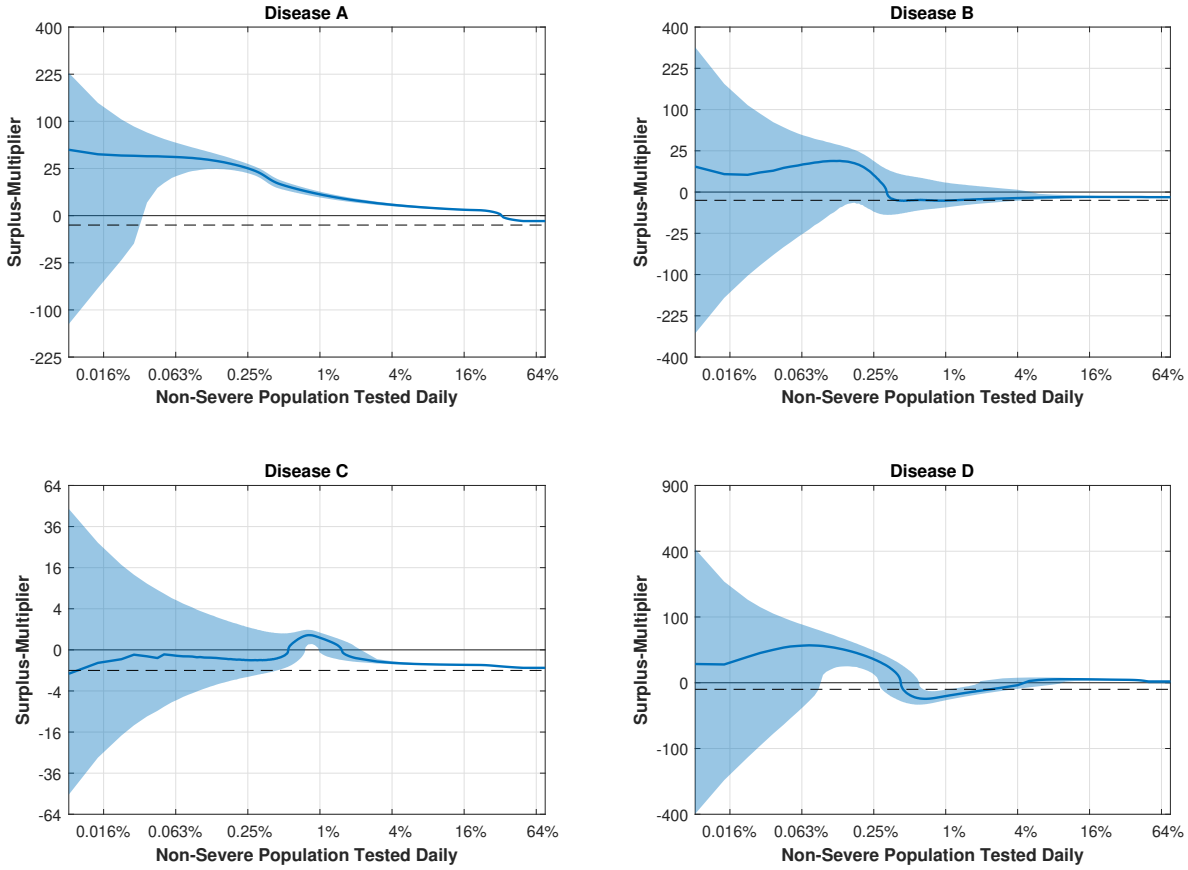


**Figure A3:** The Surplus-Multiplier for Alternative Diseases

## D.3 Technological Determinants of the Testing Multiplier

The testing multiplier depends on the characteristics of the testing and isolation technology. For the epidemic disease of the baseline parameterization, when the testing technology is more precise (i.e. it has a lower false negative rate), cheaper, and more timely (i.e. the lag between the day the test is administered and the day of the result is lower), the multiplier is higher. The multiplier is also higher when isolation of the infected is more rigorously enforced.[4] Figure A4 reports the results for the GDP-Multiplier, while the results for the Surplus-Multiplier are reported in Figure A5.

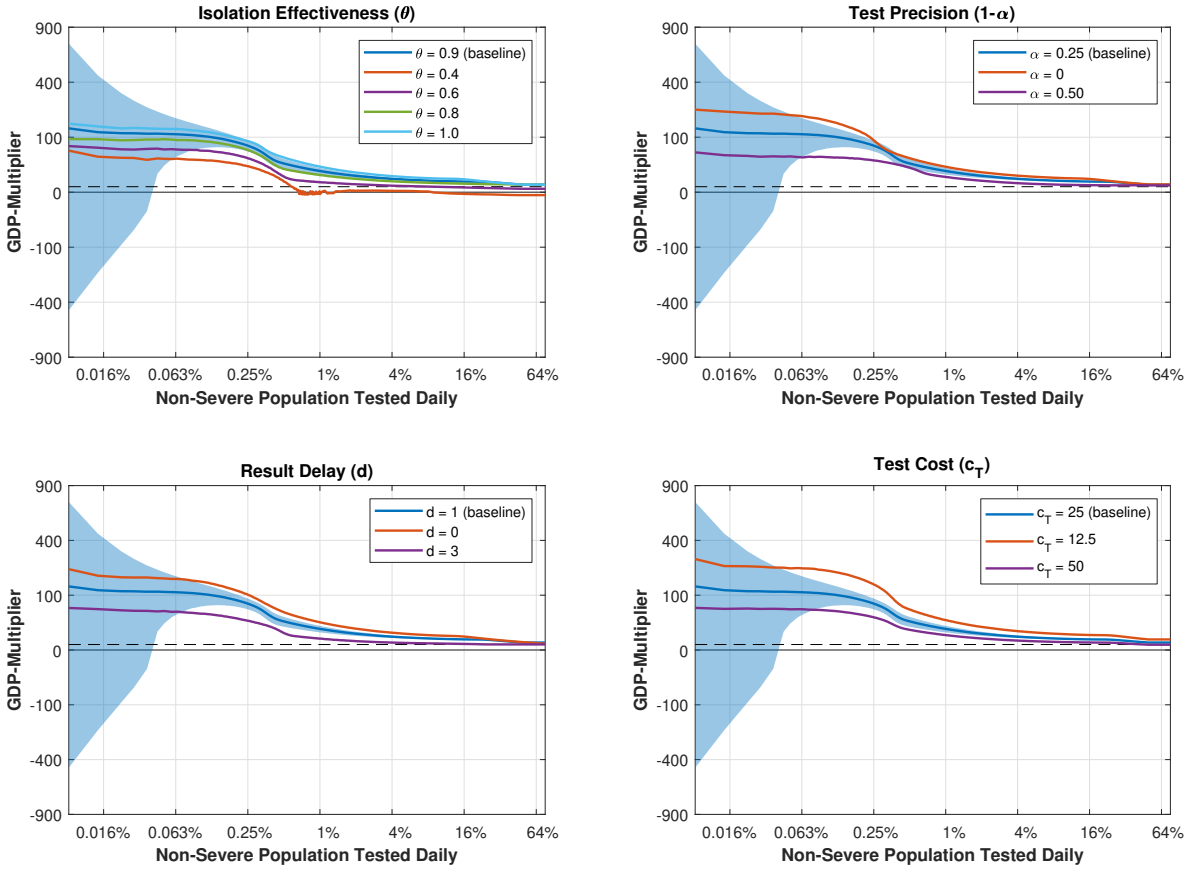


**Figure A4:** Technological Determinants of the GDP-Multiplier

It is important to realize that these sensitivity results are relative to a scenario in which the multiplier is always positive. In scenarios where the multiplier takes negative values, the effect of better technology can actually be detrimental to economic activity - at least until testing reaches a scale large enough that the multiplier becomes positive. To see this, think for example about the cost of each test kit. Around a testing level where the multiplier is negative, an additional

[4] These findings are in line with what found by the existing literature. Importantly, my theoretical analysis assumes that all the tests administered share the same technological characteristics, an assumption that can be easily relaxed. For example, one could allocate very accurate tests to severe symptomatic individuals who require medical attention, and less accurate but faster and cheaper ones to screen the rest of the population. This dual approach is advocated in Mina et al. (2020), Larremore et al. (2020), and Atkeson et al. (2020).

dollar spent on testing produces higher harm to economic activity when the testing technology is cheaper, because the same dollar with translate in more testing being performed. Once again, this highlights the complex non-linearities involved in the analysis.

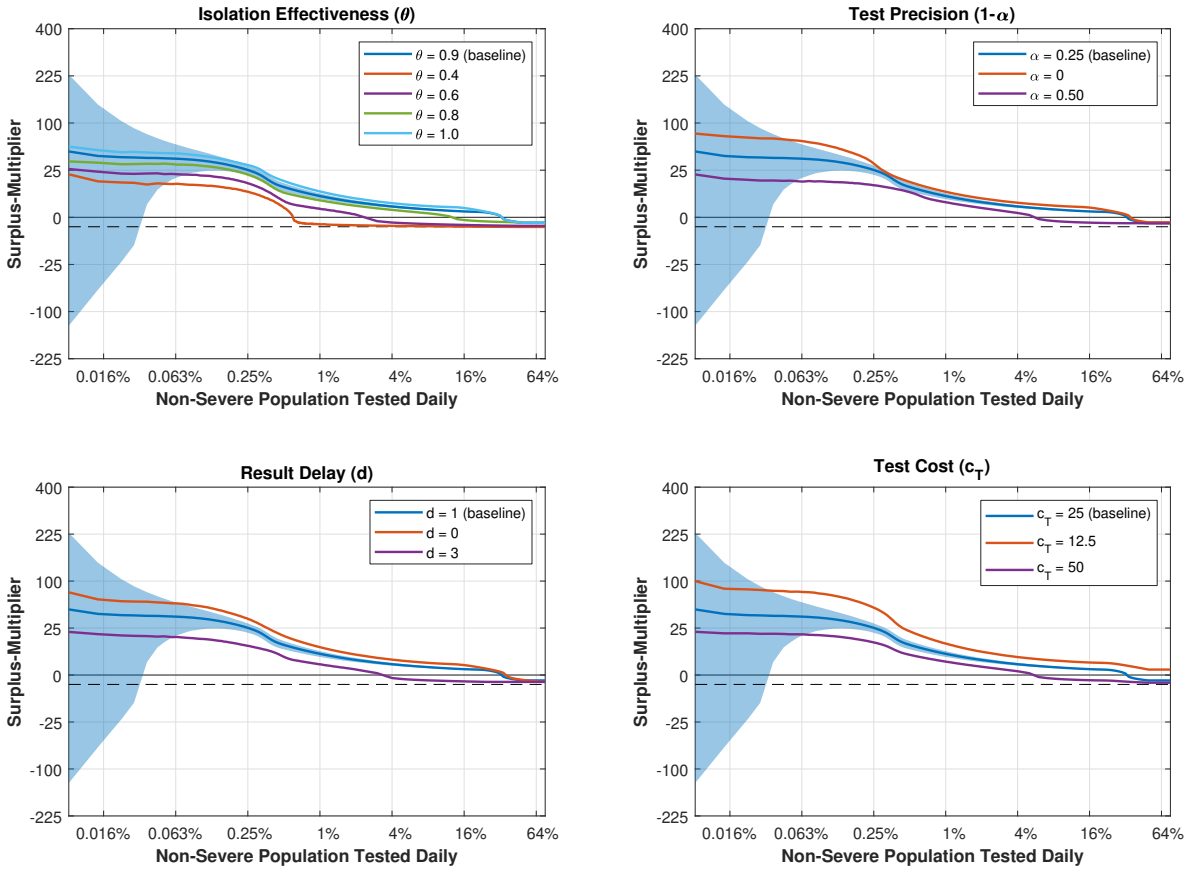


**Figure A5:** Technological Determinants of the Surplus-Multiplier

### D.4 An Alternative Specification of Beliefs

The insight that additional testing has the potential to increase risk perceptions is more general than it may appear. To show this, I twist the original specification of beliefs in the following way. Agents are assumed to learn about the true lethality of the disease over time as follows:

$$\text{Perceived Lethality}_t = (1 - \lambda_t) \cdot CFR_t + \lambda_t \cdot \phi$$

where $\lambda_t = \frac{t}{T}$, where $T$ is the time horizon considered. The perceived lethality of the disease is therefore an average between the case fatality rate and the true infection fatality risk, where the weight of the latter linearly increases over time.[5] Agents then use the total number of deaths from the epidemic disease to construct an estimate of the total number of cases:

$$\hat{C}_t = \frac{D_t}{\text{Perceived Lethality}_t}$$

and compare this estimate with the detected number of cases in order to construct an ascertainment bias factor:

$$\text{Ascertainment Bias}_t = \frac{\hat{C}_t}{C_t}$$

which provides an estimate of the degree to which testing under-estimates the total number of infections. Finally, they estimate the number of current infections by scaling up the detected number of active infections:

$$\hat{I}_t = I_t \times \text{Ascertainment Bias}_t$$

Finally, they form their perceived risk of death as in the original specification:

$$\chi_t = \text{Perceived Lethality}_t \times \beta \cdot \frac{\hat{I}_t}{P_t}$$

The key property of this specification is that, irrespective of testing, agents correctly learn over time the total number of infections.[6] Yet, they still fail to learn the number of active infections in real-time, exactly as in the original specification. Figure A6 illustrates this point for the baseline parameterization when a sizeable share (namely 8%) of the non-severe population is tested daily:

[5] This 'exogenous learning' is meant to capture the idea that agents gradually learn about the true lethality of the disease over time from sources other than testing.

[6] This approach to the estimation of active infections is inspired by and parallels what is proposed in the state-of-the-art work on the SARS-CoV-2 outbreak by Chande et al. (2020). The authors construct an ascertainment bias factor given by the ratio of total cases estimated with serological surveys over detected cases through testing. They then use this factor to scale up newly detected infections.

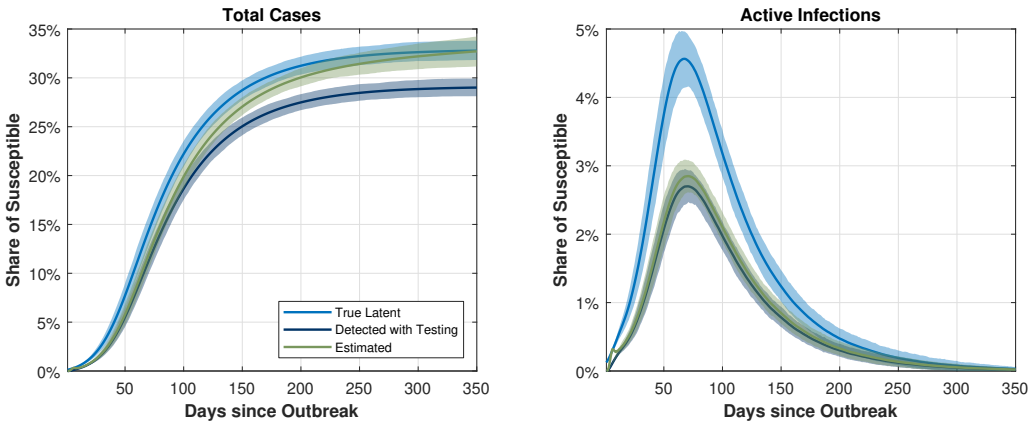


**Figure A6:** Alternative Specification of Beliefs

Since agents need to rely on testing data to form their perceptions of infection risk, the testing multiplier can still be negative, as reported in Figure A7 and Figure A8.

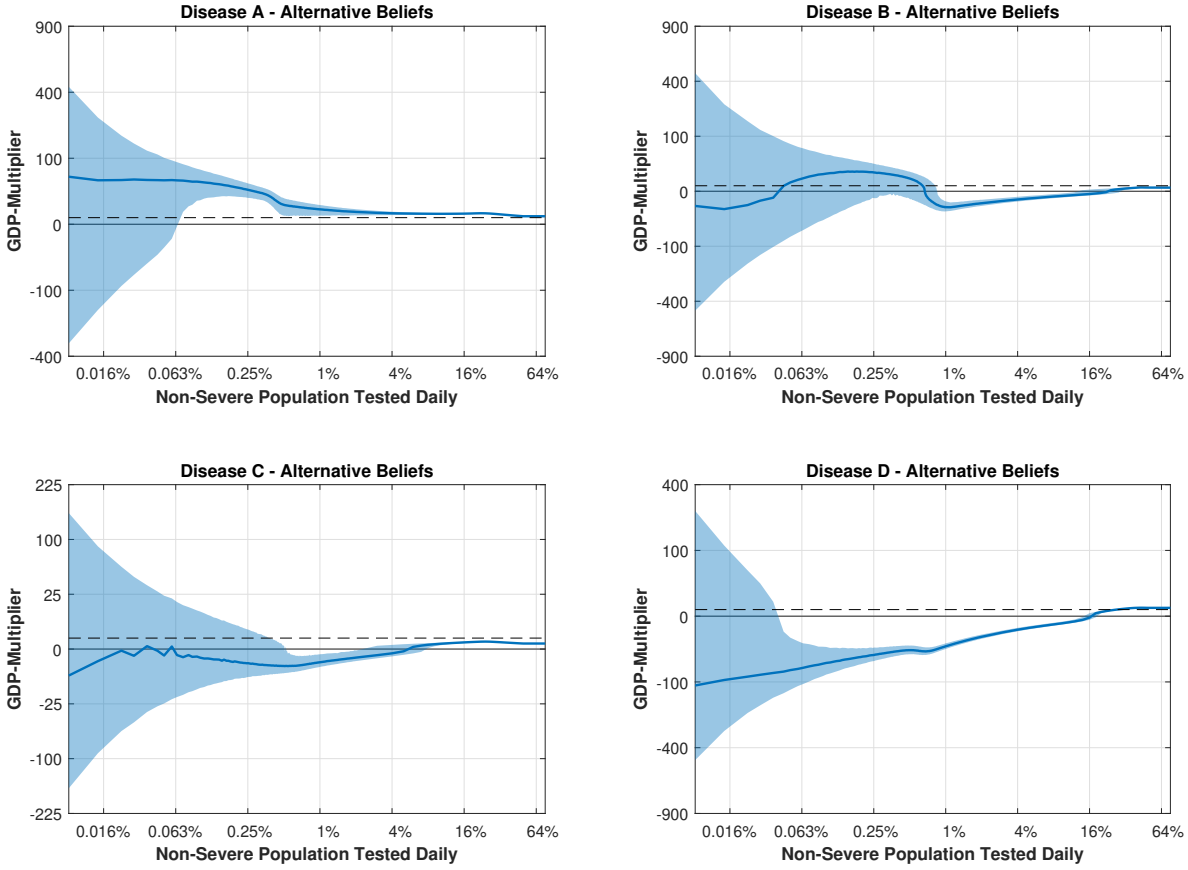


**Figure A7:** The GDP-Multiplier under Alternative Beliefs

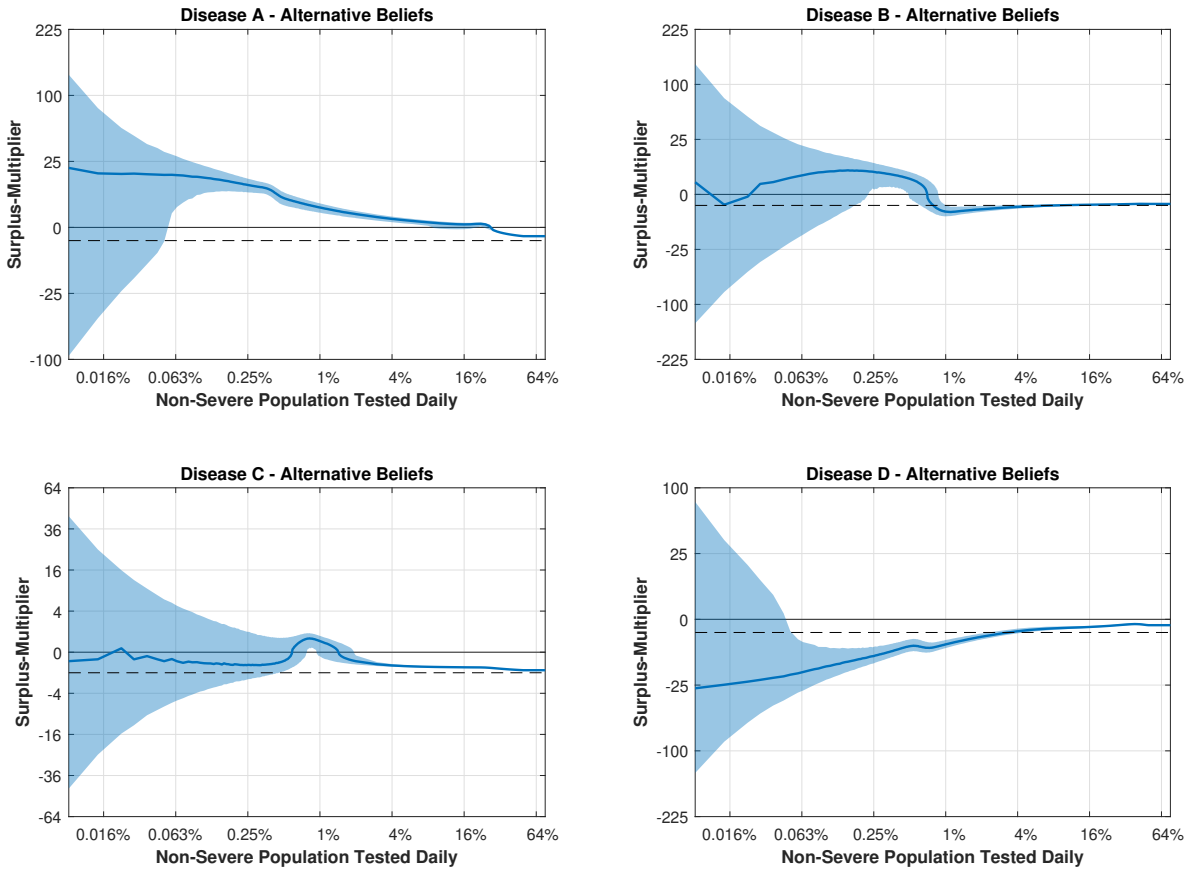


**Figure A8:** The Surplus-Multiplier under Alternative Beliefs

## D.5 A SARS-CoV-2 Calibration of the Model

Figure A9 reports aggregate epidemic and economic outcomes across testing levels under both homogeneous and heterogeneous risk perceptions.

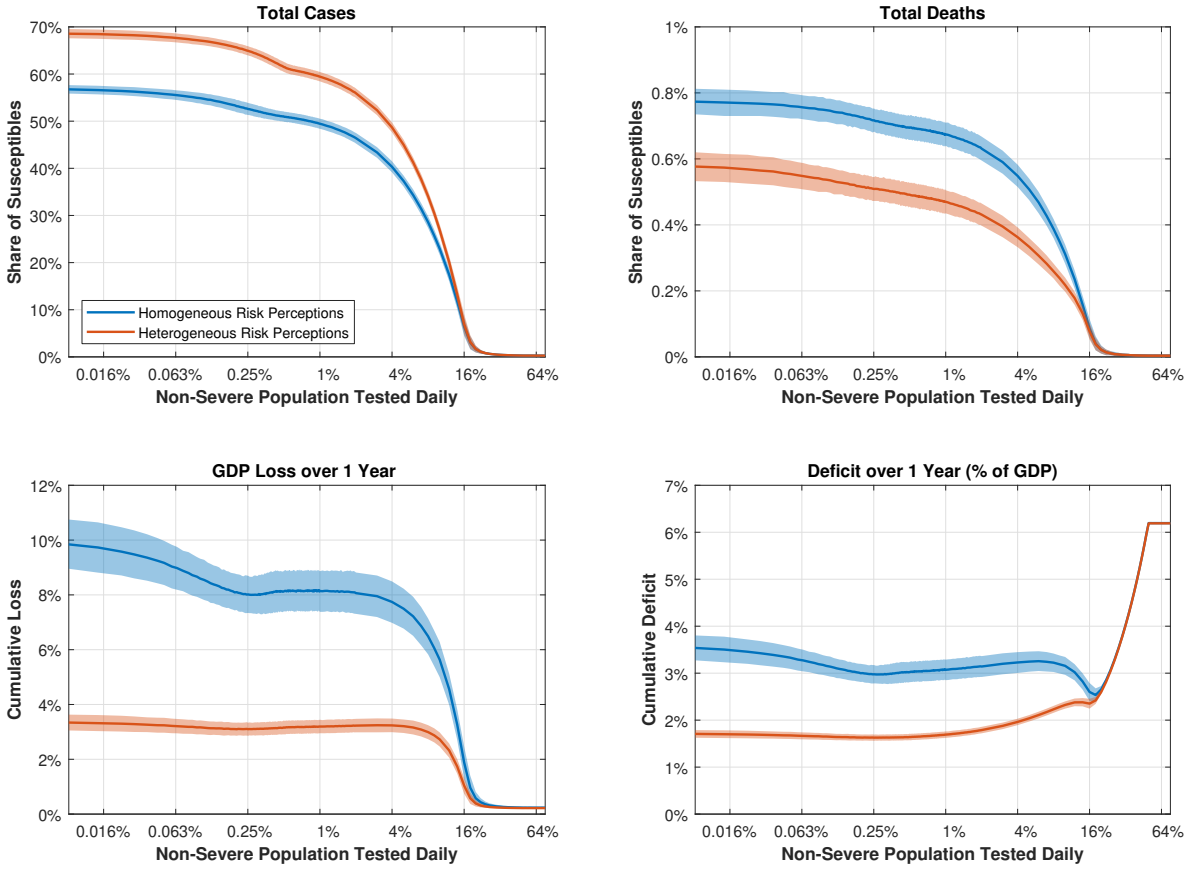


**Figure A9:** Economic and Health Outcomes for SARS-CoV-2 Across Testing Levels

Figure A10 reports both the GDP-Multiplier and the Surplus-Multiplier under both homogeneous and heterogeneous risk perceptions. In both scenarios, testing appears on average beneficial to the economy and (partially) repays for itself. Interestingly, the multiplier is higher with homogeneous risk perceptions, and this happens partly because the 'informational' contribution of additional testing activity is higher in this case.

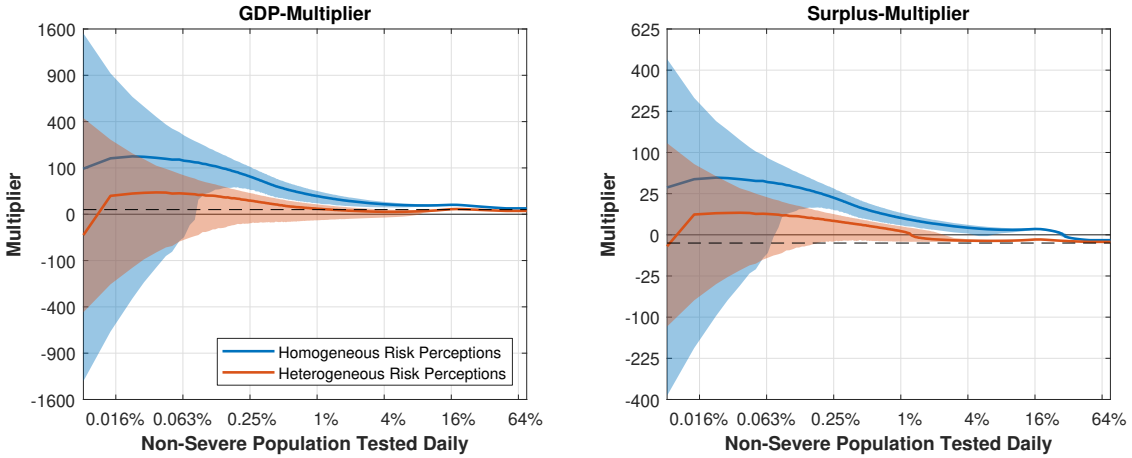


**Figure A10:** The Testing Multiplier for the SARS-CoV-2 Calibration

# E A Pseudo-Spanish-SARS-CoV-2 Parameterization

The simulations for SARS-CoV-2 suggest that heterogeneous risk perceptions improve both economic and public health aggregate outcomes, and this happens because high-risk agents 'protect' themselves more and low-risk agents - who contribute the most to economic activity - 'protect' themselves less and return to work. Consider now a disease such that the individuals at high risk are those that contribute the most to economic activity. Interestingly, the so-called 'Spanish Flu' of 1918-1919 is considered to be characterized precisely by this property. Figure A11 below reports standardized mortality risks across age groups for the Spanish Flu, as estimated by Cilek et al. (2018).

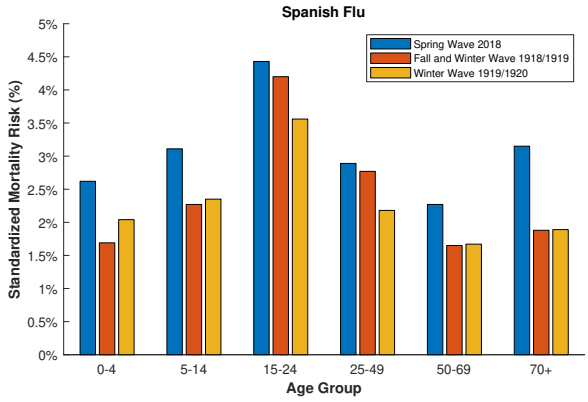


**Figure A11:** Standardized Mortality Risk Across Age-Groups for the Spanish Flu

Given the scarcity of reliable information, calibrating the model to the Spanish Flu would be a daunting task beyond the scope of this paper. I therefore perform the simplest thought-experiment one could think about: taking the calibration for SARS-CoV-2 and swapping the infection fatality risks across the two groups. More precisely:

$$\phi_s^y = 0.248$$
$$\phi_s^o = 0.005$$

All the other parameters stay untouched. Figure A12 reports economic and health outcomes under this calibration.

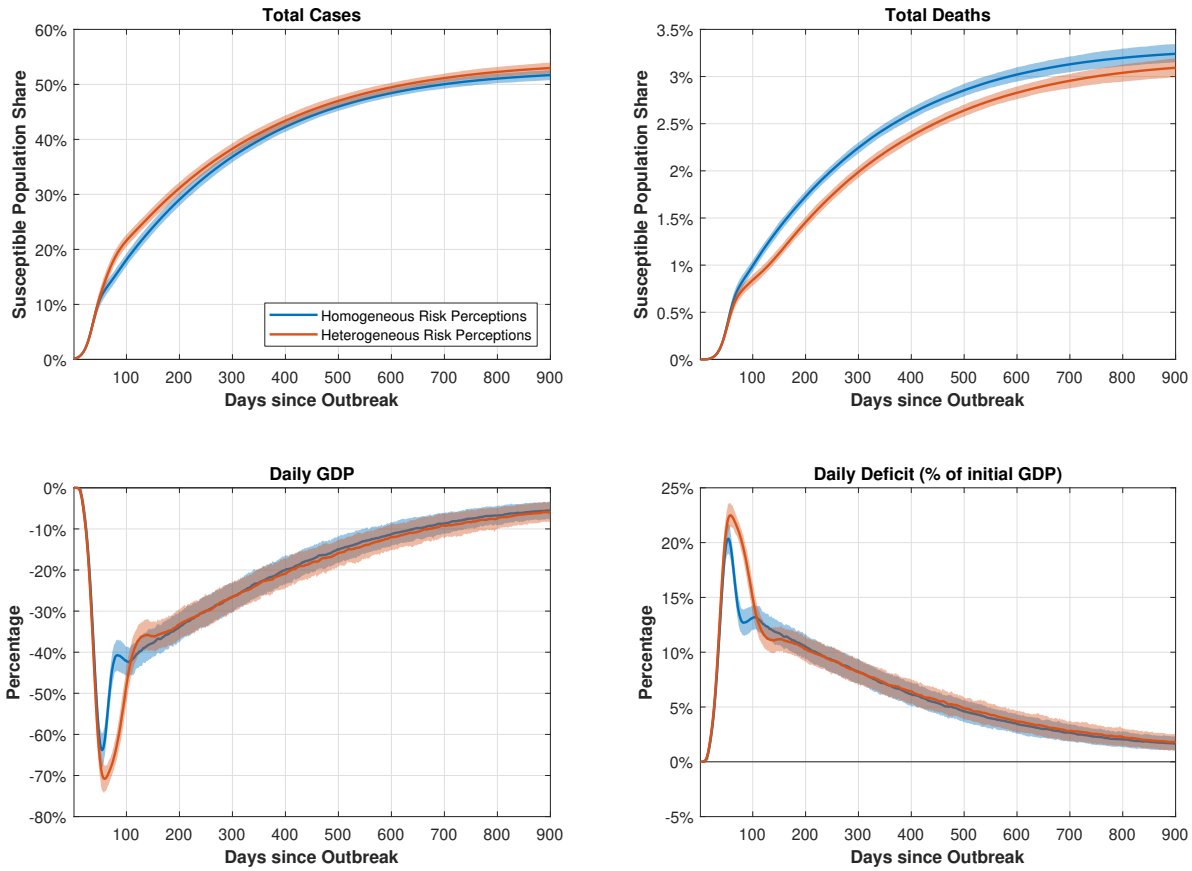


**Figure A12:** Heterogeneous Risk Perceptions for the Pseudo-Spanish-SARS-CoV-2 Calibration

Heterogeneous risk perceptions still result in less overall deaths, but the economic loss is now slightly higher. This happens because young agents, who generate the vast majority of GDP and are not at high risk, reduce their labor supply more when provided with disaggregated data. This, in turn, produces a larger fall in GDP relative to the scenario in which the government provides aggregate data. Figure A13 confirms this intuition across all testing levels, whereas Figure A14 displays the testing multiplier.

It is interesting to notice how powerful the behavioral responses of the young are in slowing down epidemic transmission. Because the young population comprises most of the population, the case fatality rate is very high with both aggregate and disaggregated testing data. As a result, in an attempt to 'protect' themselves, the young produce a catastrophic collapse of economic activity and sizeably increase the time necessary for the population to acquire herd immunity.[7]

[7]Because of this, I am forced to increase the time horizon considered from 350 to 900.

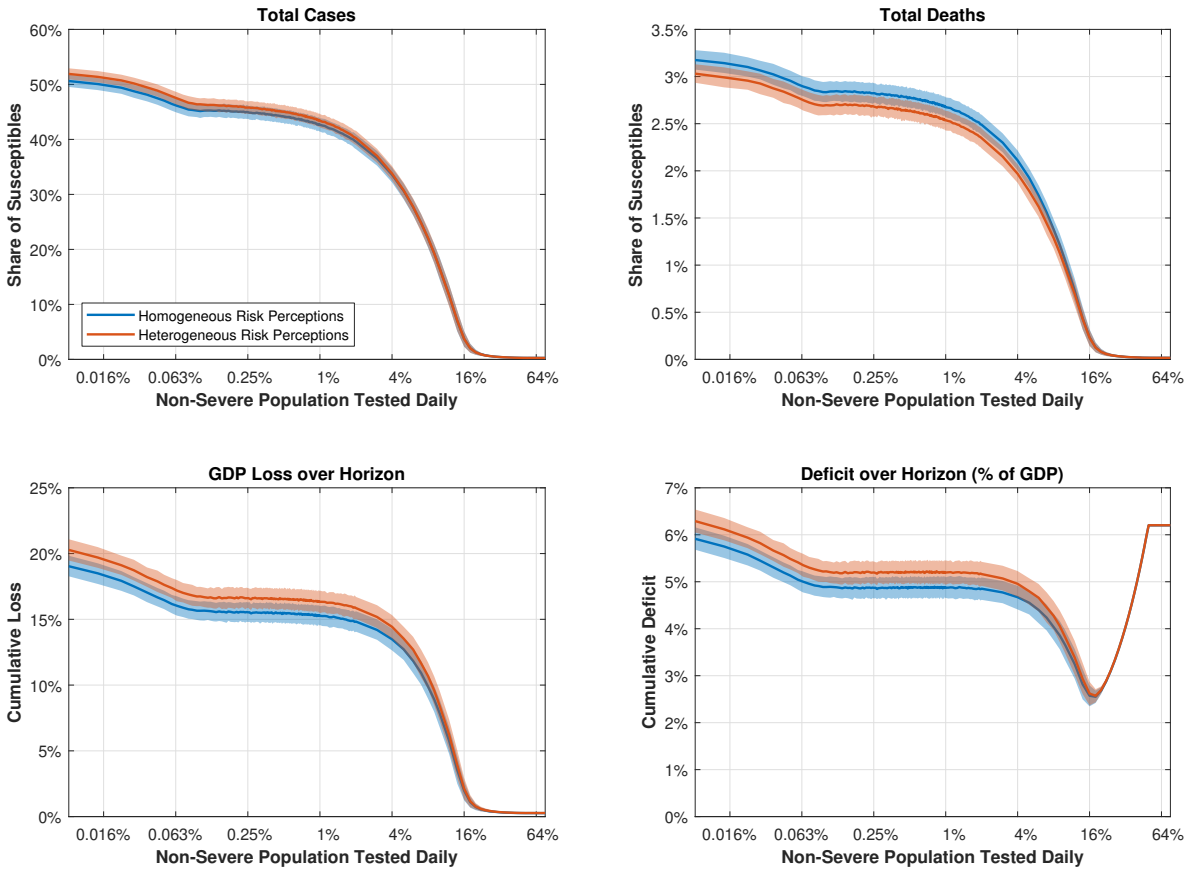


**Figure A13:** Heterogeneous Risk Perceptions Across Testing Levels for the Pseudo-Spanish-SARS-CoV-2 Calibration

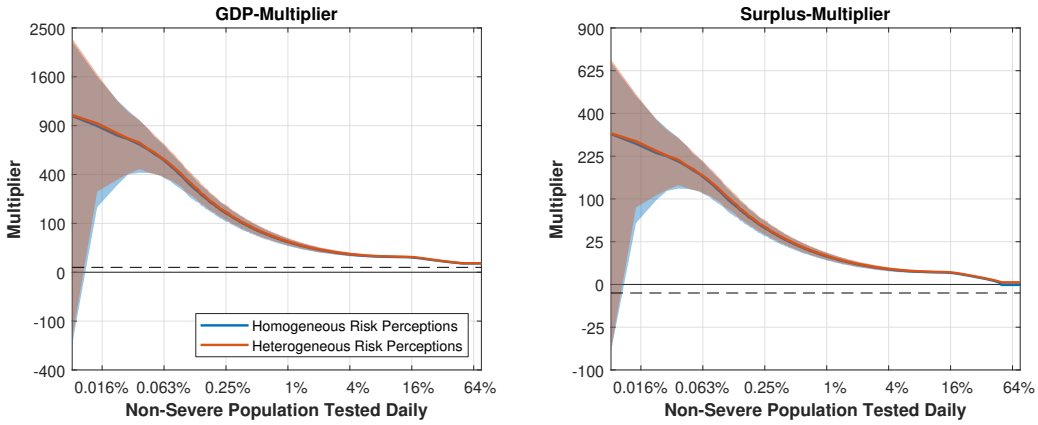


**Figure A14:** The Testing Multiplier for the Pseudo-Spanish-SARS-CoV-2 Calibration