

Guimaraes, Luis

Working Paper

Antibody Tests: They are More Important than We Thought

QMS Research Paper, No. 2020/07

Provided in Cooperation with:

Queen's University Belfast, Queen's Business School

Suggested Citation: Guimaraes, Luis (2020) : Antibody Tests: They are More Important than We Thought, QMS Research Paper, No. 2020/07, Queen's University Belfast, Queen's Management School, Belfast,
<https://doi.org/10.2139/ssrn.3640105>

This Version is available at:

<https://hdl.handle.net/10419/271238>

Standard-Nutzungsbedingungen:

Die Dokumente auf EconStor dürfen zu eigenen wissenschaftlichen Zwecken und zum Privatgebrauch gespeichert und kopiert werden.

Sie dürfen die Dokumente nicht für öffentliche oder kommerzielle Zwecke vervielfältigen, öffentlich ausstellen, öffentlich zugänglich machen, vertreiben oder anderweitig nutzen.

Sofern die Verfasser die Dokumente unter Open-Content-Lizenzen (insbesondere CC-Lizenzen) zur Verfügung gestellt haben sollten, gelten abweichend von diesen Nutzungsbedingungen die in der dort genannten Lizenz gewährten Nutzungsrechte.

Terms of use:

Documents in EconStor may be saved and copied for your personal and scholarly purposes.

You are not to copy documents for public or commercial purposes, to exhibit the documents publicly, to make them publicly available on the internet, or to distribute or otherwise use the documents in public.

If the documents have been made available under an Open Content Licence (especially Creative Commons Licences), you may exercise further usage rights as specified in the indicated licence.



QUEEN'S
UNIVERSITY
BELFAST

MANAGEMENT
SCHOOL

QUEEN'S MANAGEMENT SCHOOL RESEARCH PAPER SERIES

QMS Research Paper 2020/07

Antibody Tests: They Are More Important Than We Thought?

Luís Guimarães – Queen's University Belfast

1 July 2020

<https://ssrn.com/abstract=3640105>

Queen's University Belfast
Queen's Management School
<https://www.qub.ac.uk/mgt/>

Queen's Management School Research Paper Series
<http://ssrn.com/link/Queens-Management-BSR.html>

ANTIBODY TESTS: THEY ARE MORE IMPORTANT THAN WE THOUGHT *

LUÍS GUIMARÃES†

June 30, 2020

Abstract

Antibody testing is a non-pharmaceutical intervention – not recognised so far in the literature – to prevent COVID-19 contagion. I show this in a simple economic model of an epidemic in which agents choose social activity under health state uncertainty. In the model, susceptible agents are more socially active when they *think* they might be immune. And this increased activity escalates infections, deaths, and welfare losses. Antibody testing, however, prevents this escalation by revealing that susceptible agents are not immune. Through this mechanism, in exercises calibrated to the UK, I find that antibody testing can save about 8% of COVID-19 related deaths within 12 months.

JEL Classification: D62; E17; I12; I18.

Keywords: COVID-19; Antibody Tests; Health State Uncertainty; Non-Pharmaceutical Interventions.

*I am deeply grateful for the helpful feedback I received from friends and colleagues.

†Queen's University Belfast. E-mail address: l.guimaraes@qub.ac.uk.

1 Introduction

The COVID-19 pandemic has virtually stopped the world economy and has led to the death of more than 400 thousand people worldwide by June 2020. In the hope of constraining the pandemic, governments around the world restricted movement, imposed lockdowns and quarantines, forced the closure of many businesses and activities, and increased the scale of viral testing and contact-tracing. In this paper, I argue that there is another non-pharmaceutical intervention to curb down the pandemic: large-scale antibody testing.

There are two obvious reasons to support antibody testing. First, antibody testing helps in understanding the extent of the pandemic, its infection-fatality rate, the duration of immunity, and the proportion of asymptomatic. It has been conducted in countries like the UK, US, and Spain. And the estimates of these studies have guided policy and the calibration of epidemiological models. Second, by identifying immune individuals, antibody testing may facilitate reopening the economy after lockdown (e.g., by issuing the so-called “immunity passports”). Yet, there is another reason to support antibody testing: by revealing that susceptible and asymptomatic individuals are not immune, it tends to reduce their social activity lowering the scale of the epidemic.

The economics literature has long emphasised the role of incentives and individual decision making in shaping the extent and welfare costs of an epidemic. In the context of the AIDS pandemic, some examples are [Philipson and Posner \(1993\)](#), [Kremer \(1996\)](#), [Lakdawalla, Sood and Goldman \(2006\)](#), [Delavande and Kohler \(2015\)](#), [Gong \(2015\)](#), [Friedman \(2018\)](#), and [Greenwood et al. \(2019\)](#). More recently, in the context of the COVID-19 pandemic, some examples are [Eichenbaum, Rebelo and Trabandt \(2020a\)](#), [Farboodi, Jarosch and Shimer \(2020\)](#), [Garibaldi, Moen and Pissarides \(2020\)](#), [Keppo et al. \(2020\)](#), [Krueger, Uhlig and Xie \(2020\)](#), and [Toxvaerd \(2020\)](#). Economic agents in these models face a trade-off that can be broadly summarised as the following: economic and social activity generates a gain but might generate a loss by increasing exposure to the virus. Thus, unsurprisingly, economic agents refrain themselves from many of the activities that they would pursue absent the epidemic, increasing the duration of the epidemic but significantly lowering life and welfare losses.

Individuals decide under uncertainty, and particularly in the context of an epidemic, they decide without knowing their health state. For example, more than half of those exposed to COVID-19 are estimated to be asymptomatic (see references in [Eikenberry et al., 2020](#)). And among the symptomatic, many only develop mild symptoms. Furthermore, in June 2020, it is estimated that about 6.8% of the UK population have developed antibodies against COVID-19 but less than 10% have been identified and documented. Similar numbers apply to Spain: antibody tests suggest that 5.2% of the Spanish population have antibodies against COVID-19 but less than 15% have been identified.¹ All this evidence compellingly suggests that many individuals must decide without knowing whether they are susceptible, (infected but) asymp-

¹For the UK figures, I used the COVID-19 Infection Survey of 12 June 2020 conducted by the ONS and available in <https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/conditionsanddiseases>. For Spain, I consulted the link <https://english.elpais.com/society/2020-06-05/spains-macro-study-show-just-52-of-population-has-contraction> on 17 June 2020.

tomatic, and (in many cases) immune. Antibody tests end part of this uncertainty by revealing whether individuals are immune or not.²

In this paper, I build a simple economic model in which agents choose social activity to maximise their lifetime utility under health state uncertainty. In the model, agents can be in one of six health states: susceptible, asymptomatic, symptomatic, recovered undocumented, recovered documented, or dead. Susceptible agents do not have antibodies against the virus, which puts them at risk of an infection. Asymptomatic and symptomatic agents are infected and may infect others. Recovered undocumented and recovered documented have antibodies against COVID-19 and are, thus, immune. Individuals' optimal social activity depends on their health state and uncertainty. In a world with perfect information, susceptible agents would constrain social activity to reduce exposure to the virus, while recovered agents (both documented and undocumented) would not. But, under health state uncertainty, agents are forced to decide based on expectations of their health state: some recovered agents may choose excessively low social activity and, most importantly for contagion, some susceptible agents may choose excessively high social activity.

In the model, exposed susceptible agents become asymptomatic. Asymptomatic agents are infected but show no symptoms; thus, they are unaware of the infection and behave as susceptible agents. A few days after exposure, some asymptomatic agents develop symptoms and learn that they are infected while the rest recover without ever developing symptoms. This latter group of agents is, thus, unaware of the recovery/immunity because they were unaware of the infection; these agents – which I call recovered undocumented – can only learn that they are immune if they do an antibody test. Recovered undocumented agents differ substantially from agents that recover after developing symptoms. If symptomatic agents recover, they know that they were infected and, thus, know that they are immune; I refer to these agents as recovered documented.

My model also distinguishes between two groups of economic agents: tested and untested. The difference between the two is that the former have continuous access to antibody tests while the latter do not.³ Thus, tested agents always know whether they are immune, whereas untested agents only know that they are immune if they had symptoms of the infection (i.e., are recovered documented). By the same token, tested susceptible agents choose social activity knowing that they are not immune, while untested susceptible agents choose social

²The human body can develop immunological memory (become immune) by developing special B and T cells, which defend the body against pathogens that it has previously encountered (Punt et al., 2018). Antibodies are produced by B-cells and are detected by antibody (serological) tests; but antibody tests do not detect T cells and it is theoretically possible that an individual is immune to COVID-19 without developing antibodies thanks to T cells. As there is not yet, best to my knowledge, concrete evidence of that in the context of COVID-19 infections, I simplify my analysis and abstract from the possibility that immune individuals do not develop antibodies.

³It is likely that agents have continuous access to antibody tests if there are home antibody tests that can be used without medical supervision and with very low marginal cost; in this regard, there are public and private investments to develop a 'finger-prick' type antibody test for home use, similar to a diabetes test, that would satisfy these conditions (<https://www.gov.uk/government/publications/coronavirus-covid-19-antibody-tests/coronavirus-covid-19-antibody-tests> visited on 17 June 2020).

activity with the doubt that they might be immune. This distinct health state uncertainty changes optimal decisions. Given that restraining social activity is unwarranted when immune/recovered, untested susceptible agents tend to be more socially active than tested ones, especially when the probability of being recovered undocumented is large.

Numerical simulations of my model confirm that large-scale antibody testing can substantially raise welfare and save lives. When an epidemic begins, tested and untested susceptible agents behave similarly for some time. But as the probability of being recovered undocumented builds up, their optimal choices uncouple and untested susceptible agents raise their social activity. Moreover, as susceptible and asymptomatic agents choose the same social activity, the higher social activity of untested asymptomatic agents increases exposure of all susceptible agents. Therefore, if agents are unaware of recovery, total exposure rises, causing more deaths and welfare losses. Widespread antibody testing, however, prevents this from happening: by revealing to susceptible (and asymptomatic) agents that they are not immune, these agents optimally restrain their social activity. Through this channel and using my preferred calibration, antibody testing can avoid about 8% (7000) of COVID-19 related deaths in the UK from June 2020 to June 2021.

The gains from antibody testing rise with the number of recovered undocumented agents, which depends on (i) social activity choices, (ii) the proportion of symptomatic agents, (iii) COVID-19 contagiousness, and (iv) the scale and effectiveness of other non-pharmaceutical interventions. For example, my preferred calibration assumes that policies like mandatory mask use in the UK public transports permanently reduce COVID-19 contagiousness by 40%; absent that assumption, antibody testing prevents almost 13% (28000) of COVID-19 related deaths. Furthermore, large scale viral testing and contact-tracing reduce the importance of antibody tests because these policies tend to identify those who are infected, reducing the number of recovered undocumented. Yet, as identified by many others (e.g., [Hall, Jones and Klenow, 2020](#) and [Hornstein, 2020](#)), there is much uncertainty surrounding the parameters of epidemiological models, which critically changes optimal choices and the implications of non-pharmaceutical interventions. Therefore, we should not take my estimates literally. But my message that antibody testing can save lives is robust.

Relation with the Literature

The channel in my model through which antibody tests affect decision making has support in data. [Gong \(2015\)](#) assesses how HIV testing affects individual decision making in Sub-Saharan Africa and documents that individuals surprised by a negative HIV test decrease their risky sexual behaviour. Similarly, in my model, agents who think that they are immune and test negative for antibodies against COVID-19 restrain their social activity.

As in [Farboodi, Jarosch and Shimer \(2020\)](#), [Garibaldi, Moen and Pissarides \(2020\)](#), [Keppo et al. \(2020\)](#), and [Toxvaerd \(2020\)](#), my model assumes that economic agents directly choose their social activity to maximise their lifetime utility.⁴ Among these four models, my model

⁴Other models like, e.g., [Bodenstein, Corsetti and Guerrieri \(2020\)](#), [Eichenbaum, Rebelo and Trabandt \(2020a\)](#), and [Krueger, Uhlig and Xie \(2020\)](#) consider more complex economic blocks including a production function,

is closer to that in [Farboodi, Jarosch and Shimer](#) because they assume that economic agents cannot distinguish between being susceptible or infected whereas the other three assume, as common in the literature, that agents always know their health state. Nonetheless, [Farboodi, Jarosch and Shimer](#) crucially assume that agents are always aware whether they are recovered/immune. In such a setting, it is impossible to study the role of antibody testing in revealing the health state to economic agents.

The effects of viral testing in the context of COVID-19 have attracted the attention of the literature ([Berger, Herkenhoff and Mongey, 2020](#); [Brotherhood et al., 2020](#); [Eichenbaum, Rebelo and Trabandt, 2020b](#); [Piguillem and Shi, 2020](#)).⁵ These papers acknowledge the importance of health state uncertainty and argue that viral tests play a relevant role in resolving that uncertainty. They, and in particular [Eichenbaum, Rebelo and Trabandt \(2020b\)](#), offer an enlightening lesson: viral testing only increases welfare if it comes hand-in-hand with quarantining of infected agents because these agents do not face a trade-off. Viral testing, however, differs from antibody testing, which is the focus of my paper. A viral test, i.e., a test that identifies the current presence of an infection, allows individuals to distinguish the states of susceptible and infected without informing individuals of a past infection. An antibody test, i.e., a test that identifies the past presence of an infection, allows individuals to know whether they are immune and to distinguish the recovered state from all the other states without informing individuals of a current infection. This distinction has critical implications because, in contrast with viral testing, I find that antibody testing always increases welfare. There are two reasons for that: first, positive antibody tests allow recovered agents to enjoy more social activity and, second, negative antibody tests reduce the social activity of those that matter in the propagation of the virus – susceptible and infected agents.

In the context of epidemics, an externality arises from individuals not considering the risk of infecting others. This externality, which has been emphasised in the literature, leads to sizeable theoretical gains from central-planning interventions like lockdowns to avoid contagion. For example, in [Farboodi, Jarosch and Shimer \(2020\)](#), a central planner that optimally sets social activity avoids 75% of deaths relative to the decentralised equilibrium and, in [Alvarez, Argente and Lippi \(2020\)](#), a central planner that optimally confines individuals avoids about 40% of deaths relative to a model with no individual choice.⁶ My paper also incorporates the externality emphasised in the literature. But, unlike the literature, my paper em-

labour supply decision, and a decision among differentiated goods implying different social-contact levels. Due to the epidemic, then, susceptible agents refrain from buying goods that imply much social contact. In essence, the trade-off is the same as choosing directly social activity. Given my focus on how economic decisions under uncertainty affect the propagation of the COVID-19 pandemic, I abstract from more complex economic setups.

⁵To be precise, [Eichenbaum, Rebelo and Trabandt \(2020b\)](#) is, to some extent, different from the other papers because they simultaneously assess the roles of viral and antibody testing. Yet, their approach does not allow to single out the role of antibody testing that I emphasise in this paper.

⁶The result in [Farboodi, Jarosch and Shimer \(2020\)](#) is driven only by the externality whereas the result in [Alvarez, Argente and Lippi \(2020\)](#) also includes any gains that could arise from individual choices. Yet, [Farboodi, Jarosch and Shimer](#) assume that the central planner has complete control over social activity whereas [Alvarez, Argente and Lippi](#) assume that the central planner cannot confine all individuals and is not entirely effective in its lockdown policy. For other papers studying how a social planner can maximise welfare in the context of an epidemic, see, e.g., [Acemoglu et al. \(2020\)](#), [Glover et al. \(2020\)](#), and [Hall, Jones and Klenow \(2020\)](#).

phasises a new mechanism: absent antibody tests, susceptible and asymptomatic individuals that think they might be immune prefer to enjoy more social activity. As mentioned above, by cancelling this mechanism, large scale antibody testing avoids about 8% of COVID-19 related deaths. This is clearly lower than the numbers in the papers above, but it is not negligible. Furthermore, and very importantly, these gains from antibody testing arise in the context of a decentralised equilibrium in which agents are free to choose their social activity. Such a context becomes even more important as many countries gradually lift lockdown policies.

The rest of the paper is organised as follows. Section 2 presents my model. Section 3 calibrates it and Section 4 simulates the epidemic from its start and shows how antibody testing affects contagion and welfare. Section 5 recalibrates the model to the UK as of June 2020 and presents a few scenarios for the epidemic evolution in that country. Section 6 discusses how often agents need to test their antibodies for antibody testing to meaningfully change social activity. Section 7 concludes the paper.

2 Model

I build a simple economic model in discrete time with two blocks, an epidemiological block and an utility-maximization block. The epidemiological block is an extended version of the canonical SIR (Susceptible-Infected-Recovered, [Kermack and McKendrick, 1927](#)) model to include two types of infected individuals, asymptomatic and symptomatic, and the possibility of death. All individuals who are infected start as asymptomatic but may become symptomatic. And, as common in the literature, all recovered individuals gain permanent immunity.⁷ This block of my model is similar to advanced epidemiological models reviewed in [Hethcote \(2000\)](#) and used in [Ferguson et al. \(2020\)](#) and [Wang et al. \(2020\)](#) in the context of the COVID-19 pandemic. Similar epidemiological models are also used in the economics literature (e.g., [Atkeson, 2020](#), [Hornstein, 2020](#), and [Piguillem and Shi, 2020](#)).

To model antibody tests, I assume that there are two groups of economic agents trying to maximise their lifetime utility.⁸ Their trade-off is essentially the same: they can increase social activity to enjoy higher current utility but, by doing so, they raise the probability of an infection and dying, which lowers their future utility. The only distinction between the two groups of agents regards their uncertainty about health state. A share ω of agents, whose variables are denoted by the superscript τ , have their antibodies continuously tested and, thus, *always* know whether they are immune/recovered; I think of these agents as having access to home antibody tests that can be used without medical supervision and with zero marginal cost; I refer to these agents as tested agents. The remaining $1 - \omega$ share of the population, whose variables are denoted by the superscript o , never test their antibodies and, thus, *may* not know whether they are immune/recovered; I refer to these agents as untested agents. My assumptions imply that some untested agents are immune but, because they are unaware of it, they still behave as if they face exposure risk; furthermore, some untested susceptible

⁷For this reason, I use recovered and immune interchangeably throughout the paper.

⁸In modelling antibody tests, I abstract from imperfect testing; I study antibody tests that perfectly distinguish those that have antibodies against COVID-19 from those that do not.

agents may expose themselves excessively because they think they might already be immune. Most of my analysis will draw on how different levels of ω – affecting how widespread antibody testing is – affect the welfare loss and propagation of the epidemic.

The way I model antibody testing is naturally a simplification but necessary for tractability. By partially revealing health state, antibody tests offer information to economic agents. Either agents learn that they are immune and start behaving as if there is no epidemic or they learn that they are not immune and adjust the probability of being in each state based on the time elapsed since the most recent antibody test. This implies that, if agents are tested in different periods and with different frequency, there is a growing heterogeneity of optimal choices, which makes it intractable to solve the model. [Eichenbaum, Rebelo and Trabandt \(2020b\)](#) find a similar problem. They analyse the role of testing (of both a current infection and antibodies) in a macroeconomic model. And to allow for testing, they are forced to make two simplifying assumptions: (i) a fixed proportion of the population is tested every period and (ii) once tested, economic agents always know their health state. In slight contrast, I assume that some agents have continuous access to antibody tests while others do not, which is only realistic if agents have access to home antibody tests. Yet, in [Section 6](#), I discuss how relaxing the assumption of continuous testing likely affects the results.

2.1 Epidemiological Block

In the canonical SIR model, new infections (i.e., transitions from susceptible to infected) are governed by the number of susceptible and infected individuals and by β (a measure of the virus' contagiousness and the number of contacts per person per period absent any behavioural change or policy). The economics literature has adapted the SIR model to allow for interactions between the economic and epidemiological blocks, endogenising new infections. For example, in [Bodenstein, Corsetti and Guerrieri \(2020\)](#), [Eichenbaum, Rebelo and Trabandt \(2020a,b\)](#) and [Krueger, Uhlig and Xie \(2020\)](#), new infections are a function of consumption choices of individuals; in [Alvarez, Argente and Lippi \(2020\)](#), [Barnett, Buchak and Yannelis \(2020\)](#), and [Piguillem and Shi \(2020\)](#) new infections depend on the lockdown and quarantine measures; in [Glover et al. \(2020\)](#) new infections also depend on employment and co-worker relationships; and in [Farboodi, Jarosch and Shimer \(2020\)](#), [Garibaldi, Moen and Pissarides \(2020\)](#), [Keppo et al. \(2020\)](#), and [Toxvaerd \(2020\)](#) new infections depend on agents' social activity choices. I follow the latter group and assume that new infections depend on the number of susceptible and infected (both asymptomatic and symptomatic) individuals and their respective social activity choices.

In my model, an agent can be in one of six health states: susceptible, s , asymptomatic, e , symptomatic, i , recovered undocumented, re , recovered documented, ri , and dead, d . I normalise the size of the population to unity and denote the number of agents in group $y = \{\tau, o\}$ in state $x = \{s, e, i, re, ri, d\}$ at time t by $N_{x,t}^y$. The same logic applies to social activity, denoted by $0 \leq A_{x,t}^y \leq 1$.

The number of new infections of economic agents in group y at time t is $\beta A_{s,t}^y N_{s,t}^y I_t$, where

I_t denotes all the social activity of infected agents:

$$I_t = (A_{e,t}^r N_{e,t}^r + A_{e,t}^o N_{e,t}^o + A_{i,t}^r N_{i,t}^r + A_{i,t}^o N_{i,t}^o). \quad (1)$$

All new infections start as asymptomatic, which is a state that lasts an average of $1/\gamma_e$ periods. Then, a proportion σ develop symptoms while the rest recover without developing symptoms and move to $N_{re,t}^y$. Economic agents that develop symptoms remain infected for an additional $1/\gamma_i$ periods on average. And when they leave this state, a proportion π dies while the rest recover and move to $N_{ri,t}^y$. The transitions between all states are summarised by:

$$\Delta N_{s,t+1}^y = -\beta A_{s,t}^y N_{s,t}^y I_t, \quad (2)$$

$$\Delta N_{e,t+1}^y = \beta A_{s,t}^y N_{s,t}^y I_t - \gamma_e N_{e,t}^y, \quad (3)$$

$$\Delta N_{i,t+1}^y = \gamma_e \sigma N_{e,t}^y - \gamma_i N_{i,t}^y, \quad (4)$$

$$\Delta N_{re,t+1}^y = (1 - \sigma) \gamma_e N_{e,t}^y, \quad (5)$$

$$\Delta N_{ri,t+1}^y = \gamma_i (1 - \pi) N_{i,t}^y, \quad (6)$$

$$\Delta N_{d,t+1}^y = \pi \gamma_i N_{i,t}^y. \quad (7)$$

I assume that symptomatic agents are sure to be infected while asymptomatic agents do not know.⁹ When symptomatic agents recover, they are aware of the recovery but asymptomatic agents are not as they did not develop symptoms. Thus, I use the two recovered states to distinguish those who know that they are recovered (because they developed symptoms before) from those who do not know. Then, antibody tests creates a wedge between the two groups of agents: all agents know whether they are recovered documented but only tested agents know whether they are recovered undocumented.

2.2 Agents' Utility Maximization

The utility-maximization block of the model is very close to that in [Farboodi, Jarosch and Shimer \(2020\)](#) and also close to that in [Garibaldi, Moen and Pissarides \(2020\)](#), [Keppo et al. \(2020\)](#), and [Toxvaerd \(2020\)](#). The trade-off in my model and in these models is similar: economic agents can increase social activity to increase current utility but only at the expense of lower expected lifetime utility due to higher infection risk. There are, however, important distinctions. First, in [Garibaldi, Moen and Pissarides, Keppo et al.](#), and [Toxvaerd](#), agents are always aware of their health state while in [Farboodi, Jarosch and Shimer](#) they are only aware of their health state when recovered. In my model, some agents know if they are infected and not all agents know if they are recovered. Second, their models assume homogeneous agents whereas I consider two groups of agents with access to different information. These nuances lead to different optimization problems, which allow me to study the implications of antibody testing.

⁹For example, [Brotherhood et al. \(2020\)](#) and [Piguillem and Shi \(2020\)](#) make a similar assumption.

In what follows, I continue to use capital letters to denote aggregate variables and use small letters to denote the variables of a specific agent. Thus, $a_{x,t}^y$ denotes the social activity of one agent in group $y = \{\tau, o\}$ in state $x = \{s, e, i, re, ri\}$ at time t . And $n_{x,t}^y$ denotes the (subjective) probability of an agent in group y of being in state x at time t . As [Farboodi, Jarosch and Shimer](#), I assume that the utility derived from social activity a is given by $u(a)$, which is single-peaked; I also normalise the maximum of $u(a)$ at $a^* = 1$ with $u(a^*) = u'(a^*) = 0$. Moreover, each agent takes aggregate variables as given when deciding social activity.

2.2.1 Tested Economic Agents

Antibody tests partially reveal the health state of economic agents: if the test is positive, agents learn that they are immune; if negative, then agents do not know whether they are susceptible or asymptomatic (because I assume that all agents are aware of the infection if symptomatic). Thus, tested agents can distinguish among all states except between susceptible and asymptomatic and choose the same social activity when in these two states: $a_t^\tau = a_{s,t}^\tau = a_{e,t}^\tau$. When these agents are immune (i.e., recover from the infection), they no longer face a trade-off and stop restraining their social activity; in this case, $a_{re,t}^\tau = a_{ri,t}^\tau = 1$. If these agents are allowed to freely choose their social activity when (infected) symptomatic, they choose $a_{i,t}^\tau = 1$ as they also do not face a trade-off and are not altruistic.¹⁰ Yet, there are a few reasons to think that they could not maximise social activity. One is that they may be hospitalised or their symptoms prevent them from appreciating the benefits of social activity. Another is that the government imposes quarantines to these individuals. Thus, I assume that social activity when symptomatic is fixed at $a_i^\tau < 1$, implying a per-period cost of being in this state.

Tested economic agents choose a_t^τ , $n_{s,t+1}^\tau$, $n_{e,t+1}^\tau$, and $n_{i,t+1}^\tau$ to maximise

$$\sum_{t=0}^{\infty} \Lambda^t \left[(n_{s,t}^\tau + n_{e,t}^\tau) u(a_t^\tau) + n_{i,t}^\tau u(a_i^\tau) - \nu \gamma_i \pi n_{i,t}^\tau \right] \quad (8)$$

subject to

$$\Delta n_{s,t+1}^\tau = -\beta a_t^\tau n_{s,t}^\tau I_t, \quad (9)$$

$$\Delta n_{e,t+1}^\tau = \beta a_t^\tau n_{s,t}^\tau I_t - \gamma_e n_{e,t}^\tau, \quad (10)$$

$$\Delta n_{i,t+1}^\tau = \gamma_e \sigma n_{e,t}^\tau - \gamma_i n_{i,t}^\tau. \quad (11)$$

Using ν to denote the value of life, I include the term $\nu \gamma_i \pi n_{i,t}^\tau$ in Eq. (8) to account for the cost of dying due to the virus.¹¹ Λ denotes the discount factor. The optimal behaviour of

¹⁰[Eichenbaum, Rebelo and Trabandt \(2020b\)](#) reach the same conclusion and add that testing (of a current infection or antibodies) without quarantines can reduce welfare. This result, however, is solely driven by viral testing.

¹¹In this maximization problem as well as that of untested agents below, I implicitly assume that agents do not readjust the probability of being in each health state based on the probability of dying. Yet, given that π is low, this approximation is not very costly and helps to avoid clutter in my exposition. It would be equivalent to assume that dead agents pay the value of life to stay as recovered in the model. Another alternative would be to merge recovered documented and dead, which is close to the option followed in [Garibaldi, Moen and Pissarides \(2020\)](#).

agents with access to antibody tests is then determined by

$$(n_{s,t}^\tau + n_{e,t}^\tau)u'(a_t^\tau) = (V_{s,t}^\tau - V_{e,t}^\tau)\beta n_{s,t}^\tau I_t, \quad (12)$$

$$\Lambda^{-1}V_{s,t}^\tau = u(a_{t+1}^\tau) + V_{s,t+1}^\tau - (V_{s,t+1}^\tau - V_{e,t+1}^\tau)\beta a_{t+1}^\tau I_{t+1}, \quad (13)$$

$$\Lambda^{-1}V_{e,t}^\tau = u(a_{t+1}^\tau) + V_{e,t+1}^\tau - \gamma_e(V_{e,t+1}^\tau - \sigma V_{i,t+1}^\tau), \quad (14)$$

$$\Lambda^{-1}V_{i,t}^\tau = u(a_i^\tau) + V_{i,t+1}^\tau - \gamma_i(v\pi + V_{i,t+1}^\tau), \quad (15)$$

where $V_{x,t}^y$ is the (shadow) value of agents in group $y = \{\tau, o\}$ of being in state $x = \{s, e, i, re\}$. Because a_i^τ is fixed, the value of being symptomatic, $V_{i,t}^\tau$, is constant and clearly negative due to the imposed social distance and the possibility of dying.

The trade-off faced by economic agents is summarised in Eq. (12). This equation shows that economic agents equal the marginal gain from social activity (which is the marginal utility of social activity weighted by the probability of being susceptible and asymptomatic) with the marginal cost of social activity (which depends on the shadow values, β , and the total social activity of infected agents, I_t , and is weighted by the probability of being susceptible). Furthermore, because I restrict my analysis to the cases in which the value of being susceptible, $V_{s,t}^\tau$, exceeds that of being asymptomatic, $V_{e,t}^\tau$, (as in [Garibaldi, Moen and Pissarides, 2020](#)) economic agents will optimally reduce their social activity to prevent exposure. As shown by [Farboodi, Jarosch and Shimer \(2020\)](#) this decentralised response of economic agents critically lessens the costs and the propagation of the pandemic. And it is in line with their evidence and the evidence reported in, e.g., [Kaplan, Moll and Violante \(2020\)](#) and [Maloney and Taskin \(2020\)](#) of behavioural changes in the US prior to lockdown and quarantining policies.

2.2.2 Untested Economic Agents

The maximization problem of untested economic agents is similar to the one in the previous section. There is, however, one key difference. Because untested agents do not test their antibodies and only know that they are recovered when they develop symptoms, they cannot distinguish the state of recovered undocumented from the states of susceptible and asymptomatic. Thus, they optimally choose the same level of social activity when in these three states: $a_t^o = a_{s,t}^o = a_{e,t}^o = a_{re,t}^o$. It is also optimal for them to choose maximum social activity when they are recovered documented, $a_{ri,t}^o = 1$. And, again, I assume that their social activity when symptomatic is restricted ($a_i^o < 1$). Thus, untested economic agents maximise

$$\sum_{t=0}^{\infty} \Lambda^t \left[(n_{s,t}^o + n_{e,t}^o + n_{re,t}^o)u(a_t^o) + n_{i,t}^o u(a_i^o) - v\gamma_i \pi n_{i,t}^o \right] \quad (16)$$

subject to

$$\Delta n_{s,t+1}^o = -\beta a_t^o n_{s,t}^o I_t, \quad (17)$$

$$\Delta n_{e,t+1}^o = \beta a_t^o n_{s,t}^o I_t - \gamma_e n_{e,t}^o, \quad (18)$$

$$\Delta n_{i,t+1}^o = \gamma_e \sigma n_{e,t}^o - \gamma_i n_{i,t}^o, \quad (19)$$

$$\Delta n_{re,t+1}^o = (1 - \sigma)\gamma_i n_{i,t}^o. \quad (20)$$

The optimal behaviour of these agents is then determined by

$$(n_{s,t}^o + n_{e,t}^o + n_{re,t}^o)u'(a_t^o) = (V_{s,t}^o - V_{e,t}^o)\beta n_{s,t}^o I_t, \quad (21)$$

$$\Lambda^{-1}V_{s,t}^o = u(a_{t+1}^o) + V_{s,t+1}^o - (V_{s,t+1}^o - V_{e,t+1}^o)\beta a_{t+1}^o I_{t+1}, \quad (22)$$

$$\Lambda^{-1}V_{e,t}^o = u(a_{t+1}^o) + V_{e,t+1}^o - \gamma_e \left[V_{e,t+1}^o - \sigma V_{i,t+1}^o - (1 - \sigma)V_{re,t+1}^o \right], \quad (23)$$

$$\Lambda^{-1}V_{i,t}^o = u(a_i^o) + V_{i,t+1}^o - \gamma_i(v\pi + V_{i,t+1}^o), \quad (24)$$

$$\Lambda^{-1}V_{re,t}^o = u(a_{t+1}^o) + V_{re,t+1}^o. \quad (25)$$

There are three differences between the set of equations governing the optimal behaviour of tested and untested agents. First, untested agents must keep track of the value of being recovered undocumented, Eq. (25), whereas tested agents know that their value in the recovered undocumented state is permanently the maximum possible (normalised to zero). Second, comparing Eqs. (12) and (21) shows that untested agents attach, *ceteris paribus*, a higher weight to the marginal utility of social activity than tested agents. This increases the incentives of untested agents to raise social activity (relative to that of tested agents), especially when $n_{re,t}^o$ is large. Third, comparing Eqs. (14) and (23) shows that the value of being recovered undocumented reduces the value of being asymptomatic. This, in turn, raises the right-hand side of Eq. (21), which tends to reduce the social activity of untested agents. Yet, in my numerical analysis, I find that this last force usually plays a minor role and untested agents end up choosing higher social activity when $n_{re,t}^o$ is slightly positive. All these three differences result from health state uncertainty and the information asymmetry between the two groups and tend to dwarf the lifetime utility of untested agents relative to tested agents.

2.3 Equilibrium

All agents within each group have the same preferences and access to the same information. Thus, at all t , their social activity, a_t^y and a_i^y , equals aggregate social activity, $A_t^y = A_{s,t}^y = A_{e,t}^y$ and $A_i^y = A_{i,t}^y$. This, in turn, implies that the probability of being in each state must equal the proportion of the population in that state: $n_{x,t}^y = N_{x,t}^y$ at all t .

A decentralised equilibrium corresponds to a path of social activities, $\{A_t^T, A_t^O, I_t\}$, state variables, $\{N_{s,t}^T, N_{s,t}^O, N_{e,t}^T, N_{e,t}^O, N_{i,t}^T, N_{i,t}^O, N_{re,t}^O\}$, and shadow values, $\{V_{s,t}^T, V_{s,t}^O, V_{e,t}^T, V_{e,t}^O, V_{i,t}^T, V_{i,t}^O, V_{re,t}^O\}$, that satisfy Eqs. (1-5), (12-15), and (21-25).

3 Calibration

I calibrate the model to daily data and summarise my parameter choices in Table 1. I set $\Lambda = \frac{1}{1+\rho} \frac{1}{1+\delta}$, where ρ is the time discount rate and δ is the probability of finding a cure-for-all (a treatment that would work simultaneously as vaccine and cure). I set $\rho = 0.05/365$ and $\delta = .67/365$ reflecting an yearly discount rate of 5% and the probability of finding the cure-for-all of 67% within a year (e.g., Alvarez, Argente and Lippi, 2020; Farboodi, Jarosch and Shimer,

2020).

Table 1: Benchmark Calibration

Discount factor:	$\Lambda = \frac{1}{1+0.05/365} \frac{1}{1+0.67/365}$
Curvature of the utility function:	$\alpha = 1$
Value of Life:	$v = 30,000$
Measure of contacts and infectiousness:	$\beta = 3/14$
Average number of days as symptomatic:	$\gamma_i^{-1} = 18$
Average number of days as asymptomatic:	$\gamma_e^{-1} = 14 - \sigma\gamma_i^{-1}$
Proportion of asymptomatic agents that develop symptoms:	$\sigma = 0.3$
Death probability conditional on being symptomatic:	$\pi = 0.0064\sigma^{-1}$
Social activity of symptomatic agents:	$A_i^o = A_i^r = 0.6$

I assume the following functional form for the utility of social activity:

$$u(A) = \frac{A^{1-\alpha} - 1}{1-\alpha} - A + 1, \quad \alpha > 0, \quad (26)$$

which guarantees that $u(a)$ is single-peaked with maximum at $A = 1$ and $u(1) = u'(1) = 0$. As my benchmark, I set $\alpha = 0$, which delivers the same utility function as in [Farboodi, Jarosch and Shimer](#). I also closely follow [Farboodi, Jarosch and Shimer](#) to find the value of life, v , in model units. I start by using a value of a prevented fatality in the UK of £1.8 Million ([Thomas and Waddington, 2017](#)) and by interpreting this number as agents being willing to pay £1.8 thousand to avoid a death probability of 0.1% or, equivalently, £0.247 per day (using the yearly discount rate of 5%). Given UK consumption per capita of £22.2k, this implies that agents are willing to permanently trade 0.41% of their consumption for a permanent reduction in their death probability of 0.1%. Assuming that the utility of consumption refers to the first term on the right-hand side of Eq. (26) (and using $\alpha = 1$) implies that the value of life must satisfy the following indifference condition:

$$\frac{\log(1)}{1 - \frac{1}{1+0.05/365}} - 0.001v = \frac{\log(1 - 0.0041)}{1 - \frac{1}{1+0.05/365}},$$

and, thus, $v = 30,000$. This is lower than the value found by [Farboodi, Jarosch and Shimer](#) because I assume a relatively low value of prevented fatality.

[Atkeson \(2020\)](#) and [Wang et al. \(2020\)](#) calibrate an epidemiological model close to that in Section 2.1 by assuming that asymptomatic individuals take on average 5.2 days to develop symptoms and, after developing symptoms, they take on average 18 days to recover or die. This calibration has influenced much of the parameter choices in the economics literature. For example, [Acemoglu et al. \(2020\)](#), [Alvarez, Argente and Lippi](#), and [Eichenbaum, Rebelo and Trabandt \(2020a\)](#) abstract from the asymptomatic state and assume that it takes on average 18 days for an infected individual to either recover or die. [Glover et al. \(2020\)](#) add an

hospitalization state to the model in Wang et al. (besides other changes) and split the 18 days from infection to recovery (or death) in 10 days of flu-like symptoms and 8 days of hospitalization. But there is one important difference between my model and that of Wang et al.: my model allows for the possibility that some asymptomatic agents recover without developing symptoms whereas, in their model, all asymptomatic agents eventually develop symptoms.¹² In light of this difference, I set $\gamma_i^{-1} = 18$ but, instead of setting $\gamma_e^{-1} = 5.2$, I find γ_e as a residual to target an average exposure time of 14 days, which is the WHO guideline for the duration of COVID-19-related quarantines. Given that individuals stay exposed asymptomatic for γ_e^{-1} periods and a proportion σ of them remain infected (but symptomatic) for an additional γ_i^{-1} days, I obtain γ_e using $\gamma_e^{-1} + \sigma\gamma_i^{-1} = 14$.

In epidemiological models, the *basic* reproduction number, R_0 , is key to determine the number of infections and herd immunity to a virus. R_0 equals the number of new infections from one infected individual absent any behavioural change or non-pharmaceutical interventions and assuming that all the population is susceptible. And I use it to calibrate β . In the epidemiological literature surveyed by Atkeson, R_0 is estimated between 2 and 3.25. But the lowest estimates are likely already affected by behavioural changes and by lockdown and quarantine measures. On the other hand, Alvarez, Argente and Lippi set $R_0 = 3.6$ while Farboodi, Jarosch and Shimer target an initial growth rate of the stock of infected of 30%, implying $R_0 = 3.1$ given their choice of an average infection duration of seven days. Based on this, I target $R_0 = 3$, which implies $\beta = 3/14$.

I fix $\sigma = 0.3$, implying that 70% of all infected agents do not develop symptoms or, at least, do not have access to a viral test confirming the change in health state. $\sigma = 0.3$ is close to the lower bound (0.15) of the early estimates reviewed in Eikenberry et al. (2020) and Stock (2020). But $\sigma = 0.3$ agrees with the recent findings of Emery et al. (2020) that more than 70% of individuals who tested positive for COVID-19 on board of the Diamond Princess cruise ship did not develop symptoms. And it also agrees with the proportion of estimated asymptomatic individuals in the UK based on the ONS (Office for National Statistics) survey.¹³ The estimated share of asymptomatic in Spain is, however, lower (less than 40%). But given that I assume that symptomatic agents are sure of being infected, it is also important to look at the proportion of those with antibodies that were diagnosed with the infection when they were infected. The ONS survey suggests that, in June 2020, about 7% of the UK population had developed antibodies against COVID-19 but less than 0.5% of the population was diagnosed with an infection. In Spain, comparable numbers are 5.2% and 0.6%, respectively. In light of these numbers, I believe it is reasonable to set $\sigma = 0.3$ but I test how a higher σ affects my results.

Meyerowitz-Katz and Merone (2020) conduct a meta-analysis of published estimates of COVID-

¹²Another difference between the two models is that only symptomatic can infect susceptible agents in Wang et al. (2020) while all exposed agents can infect susceptible agents in my model. In this way, my model is closer to that in Piguillem and Shi, 2020. I could assume that there is a prior exposed state in which agents cannot infect others but, for simplicity, I abstract from that.

¹³This evidence is reported in <https://www.ft.com/content/033745f3-2d78-4869-9690-ea46fcc9cb3d>, which I consulted on 17 June 2020.

19 infection-fatality rates and conclude that about 0.064% of those infected with the virus, die. I use this number to fix $\pi = 0.0064/\sigma$, implying that slightly more than 2% of symptomatic agents end up dying due to the virus. To calibrate the social activity of symptomatic agents, I follow the calibrations of lockdown measures and lockdown effectiveness in [Acemoglu et al. \(2020\)](#) and [Alvarez, Argente and Lippi \(2020\)](#). Based on that, I set $A_i^o = A_i^t = 0.6$.

I use a shooting algorithm as detailed in [Garibaldi, Moen and Pissarides \(2020\)](#) to solve the model. I start the algorithm by assuming that there is an initial number of asymptomatic agents of $N_e^t + N_e^o = 1e-6$ while the rest of the population is susceptible. To put this number into perspective, looking at the UK, I start the algorithm in a scenario in which approximately 67 individuals have been exposed the virus but do not yet display symptoms.

I do not impose a value for the proportion of untested agents, ω . My objective is to test for the interval of $\omega \in [0, 1]$ to see how different values affect the results. I also conduct several robustness checks to changes in the parameters of the model.

4 Results

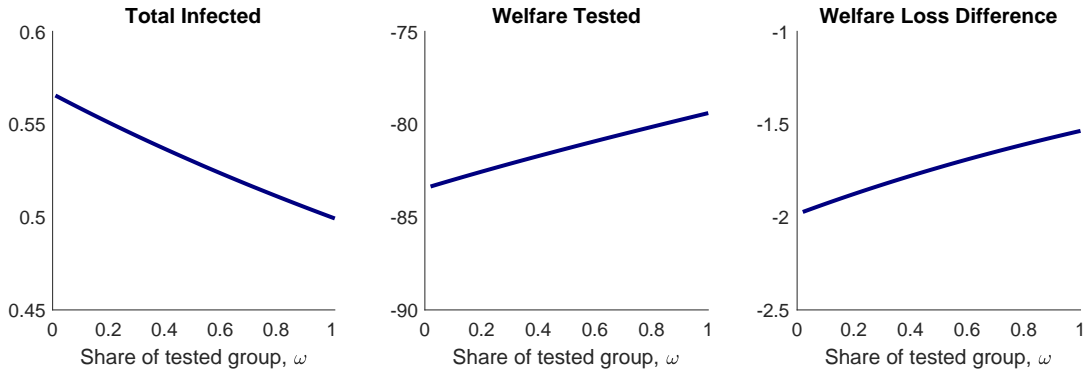
How important are antibody tests in response to a pandemic? Do they significantly affect the number of infected individuals and welfare? In this section, I use my model to answer these questions.

4.1 Main Results

Figure 1 summarises how the the share of tested agents, ω , affects three indicators of the effects of an epidemic under the benchmark calibration. On the left, it presents the total number of infected agents within the first year, irrespective of their current health state, which corresponds to $1 - N_{s,365}^o - N_{s,365}^t$; although some of these agents are infected precisely after one year, most are recovered or dead. In the middle, it presents a measure of welfare loss of tested agents; in particular, the proxy that I report is $V_{s,1}^t$, which measures how costly it is for a tested agent to be susceptible in the beginning of the epidemic. On the right, it presents a measure of the difference in welfare loss between tested and untested agents; as an indicator I use $V_{s,1}^o - V_{s,1}^t$, which measures how more costly it is for an untested agent (relative to a tested agent) to face the epidemic when it begins.

The model suggests that, if all agents are continuously tested, 49.9% of the population is exposed to the virus within one year, which contrasts with 56.6% if no agent is ever tested. In other words, within the first year of the epidemic, it is possible to reduce the number of exposed individuals and, thus, deaths by 11.7% if there is widespread and continuous testing, a scenario that would likely occur if virtually all households in the economy had easy access to home antibody tests. Using again the UK to put numbers into perspective, widespread and continuous testing would prevent about 29000 deaths. Therefore, as shown in Figure 1, large-scale antibody testing (i.e., high ω) leads to sizeable welfare gains for both tested and

Figure 1: Main Results



Note: This figure shows total infected after one year, $1 - N_{s,365}^o - N_{s,365}^t$, welfare of tested susceptible agents in period 1, $V_{s,1}^t$, and difference in welfare loss between tested and untested susceptible agents in period 1, $V_{s,1}^o - V_{s,1}^t$, as a function of the share of tested agents, ω . The model is calibrated using the benchmark calibration.

untested agents because it reduces exposure to the virus and life losses.¹⁴

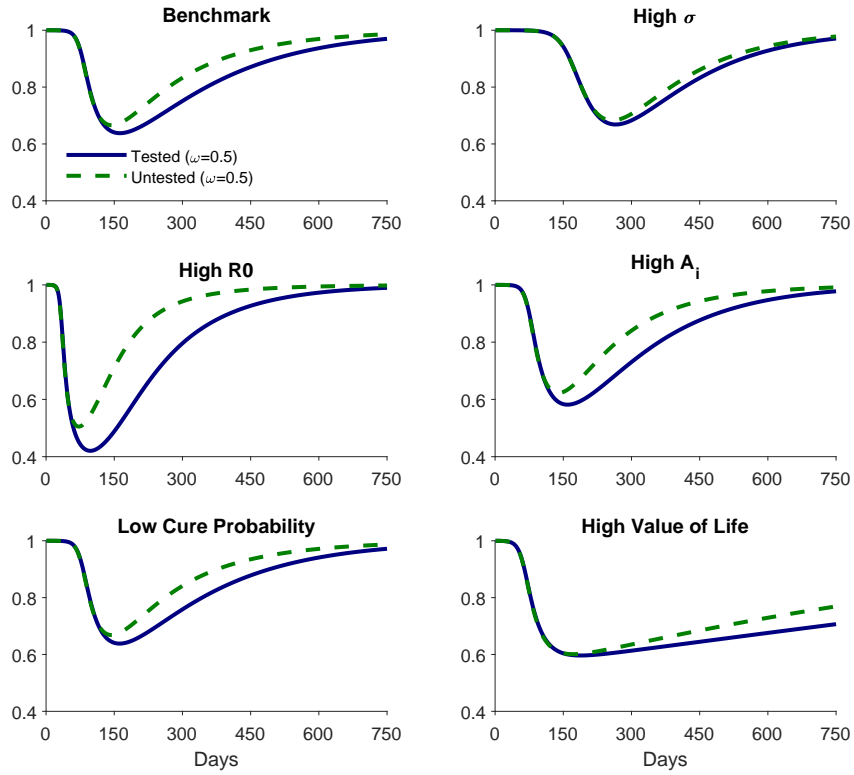
To understand why antibody testing reduces total exposure and welfare losses, Figure 2 contrasts the social activity of tested agents with that of untested agents when half of the population is tested ($\omega = 0.5$) and under multiple calibrations of the model. The key takeaway from this figure is that tested agents restrain their social activity by more than untested agents, $A^t < A^o$, especially about 100 days after the beginning of the epidemic (in the benchmark case). The reason was already hinted in Section 2.2: as the probability of being recovered undocumented enlarges, untested susceptible and asymptomatic agents start increasing social activity with the hope that they are immune but are unaware of it. Therefore, by replacing untested with tested agents, antibody testing lowers the average social activity of susceptible agents and, very importantly, the total social activity of infected agents, I_t ; this implies, respectively, a direct and an *externality* effect reducing total exposure to the virus and the welfare losses of all agents. Thus, even if there is no centralised offer of antibody tests, individuals that take advantage of private sourcing of antibody tests end up benefiting themselves but also others.¹⁵

Figure 1 also shows that untested agents suffer more from the epidemic than tested agents (Welfare Loss Difference is negative). Even though untested agents enjoy more social activity, the losses from the higher exposure to the virus and higher probability of death escalate their losses. Yet, somewhat surprisingly, antibody testing increases the welfare of untested agents by more than that of tested agents (the slope of the Welfare Loss Difference curves is

¹⁴There is also a direct welfare gain by replacing untested agents with tested agents as the latter enjoy more social activity when recovered undocumented.

¹⁵Many private clinics in the UK and around the world are already offering their services for antibody testing at a relatively low price. An example of that is in <https://www.londondoctorsclinic.co.uk/services/private-coronavirus-test/> consulted in 17 June 2020.

Figure 2: Social Activity Under Different Calibrations



Note: This figure shows social activity of tested and untested susceptible agents under six calibrations of the model and assuming that half the population is tested, $\omega = 0.5$. The six calibrations are, respectively, benchmark, $\sigma = 0.7$, $\beta = 6/14$, $A_i^o = A_i^r = 0.9$, $\Lambda = \frac{1}{1+0.05/365} \frac{1}{1+0.33/365}$, and $\nu = 120000$.

positive). In other words, untested agents benefit marginally more than tested agents if there is an increase in the share of tested agents, ω . The cause is, once more, the *externality* effect of antibody testing on I_t , which reduces exposure of all susceptible agents to the virus and allows them to enjoy more social activity.¹⁶ Because $A_i^o > A_i^r$, the fall in I_t turns out to be especially important to reduce exposure of untested agents.

To sum up, antibody testing lowers the contagion of the virus, increases welfare, and reduces inequality. It does so by increasing the information available to economic agents. And this information improves the well-being of all agents even if only a few agents have access to it. The reason is simple: susceptible and asymptomatic agents who know that they are not immune tend to curtail their social activity.

¹⁶There are some opposing indirect effects as well but they are of second-order importance. For example, tested agents restrain their social activity by less when ω is high as I_t is lower due to the higher weight of tested agents. Yet, these general-equilibrium effects do not change the qualitative results.

4.2 Robustness Checks

This section presents my sensitivity analysis to changes in the parameters of the model. It demonstrates that my qualitative results are robust to different calibrations but also that some parameters substantially change the quantitative results.

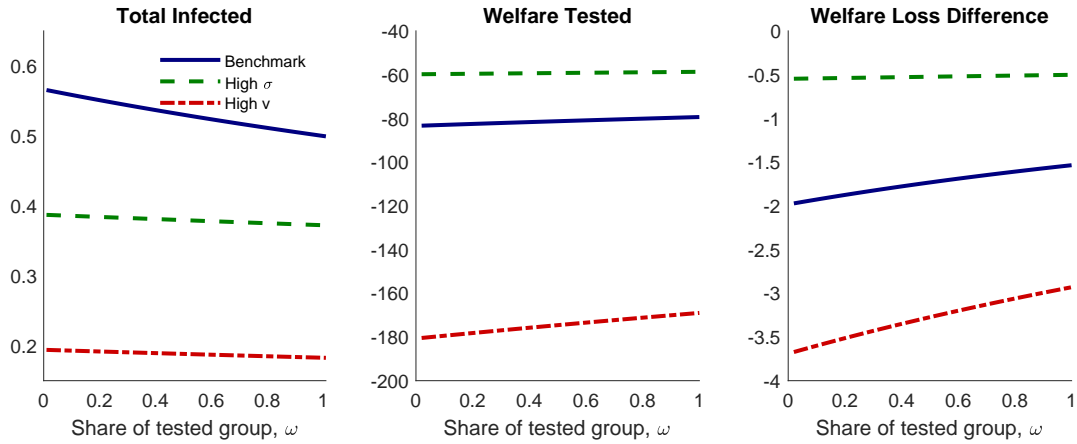
The green dashed lines in Figure 3 present the case of $\sigma = 0.7$, i.e., 70% of asymptomatic individuals eventually become symptomatic. This may be the case if most individuals develop symptoms that are perfectly distinguishable from those of other diseases. But I also use this experiment to assess the consequences of large-scale viral testing (to detect the presence of an infection in many asymptomatic agents) and very efficient contact-tracing allowing for an early identification of most infected individuals even if asymptomatic.¹⁷ Comparing the results under the benchmark calibration (blue solid lines) with those with $\sigma = 0.7$ shows a reduction in the (absolute) slope of the lines; this indicates that the gains from antibody testing depend negatively on σ . The reason is simple: if σ is large, most recovered individuals know that they are immune even without antibody tests. Thus, antibody tests offer much less information to economic agents if (i) most agents easily identify that they are infected, and/or (ii) viral testing is conducted in large scale, and/or (iii) contact tracing is very efficient. But, even if $\sigma = 0.7$, my simulations suggest that antibody testing can prevent up to 4% of deaths (corresponding to 6400 individuals in the UK) in the first year.

The value of a prevented fatality that I use to reach a measure of the value of life, ν , is relatively low. It is slightly higher than in Alvarez, Argente and Lippi (2020) and slightly lower than in Hall, Jones and Klenow (2020) but much lower than in, e.g., Eichenbaum, Rebelo and Trabandt (2020a,b) and Farboodi, Jarosch and Shimer (2020). I use the red dot-dashed lines in Figure 3 to show the implications of $\nu = 120,000$, which is four times larger than my benchmark and implies a value of life closer (relative to consumption terms) to the higher targets used in the literature. The implications for the number of susceptible agents after one year are staggering. Within the first year, the total number of exposed agents is about 36% of that implied by $\nu = 30,000$ because all economic agents persistently strongly constrain their social activity (see Figure 2). But, more importantly for the purposes of this paper, the welfare gains of antibody testing are still substantial: even though antibody testing prevents less deaths, the value attached to each life is four times higher.

I also experimented with a basic reproduction number of $R_0 = 6$ (implying a β twice that in my benchmark), which is extreme. I report the results using the green dashed lines in Figure 4. Unsurprisingly, the effect on the number of infected agents is very large: about 80% of agents are infected within one year. In this case, moving from no antibody testing to continuous and widespread antibody testing is even more important; it would save about

¹⁷This is naturally a simplification. First, I assume that only asymptomatic agents are tested for the presence of the virus such that viral tests do not offer information to susceptible agents. If I do not make this assumption, then I would need to conduct a different optimization problem, which is beyond the scope of the paper. Second, in the model, asymptomatic agents are infected for less days (on average) than symptomatic agents. Thus, modelling the effects of large-scale viral testing and very efficient contact-tracing as a transition from asymptomatic to symptomatic is also not entirely accurate. Yet, I adjust the death probability according to σ and I interpret the extra days of infection as quarantine imposed by the national authorities.

Figure 3: Robustness Checks I



Note: This figure shows total infected after one year, $1 - N_{s,365}^o - N_{s,365}^T$, welfare of tested susceptible agents in period 1, $V_{s,1}^T$, and difference in welfare loss between tested and untested susceptible agents in period 1, $V_{s,1}^o - V_{s,1}^T$, as a function of the share of tested agents, ω . Solid blue lines present the results under the benchmark calibration. The other two lines change one parameter relative to benchmark: green dashed lines assume $\sigma = 0.7$ and red dot-dashed lines assume $\nu = 120000$.

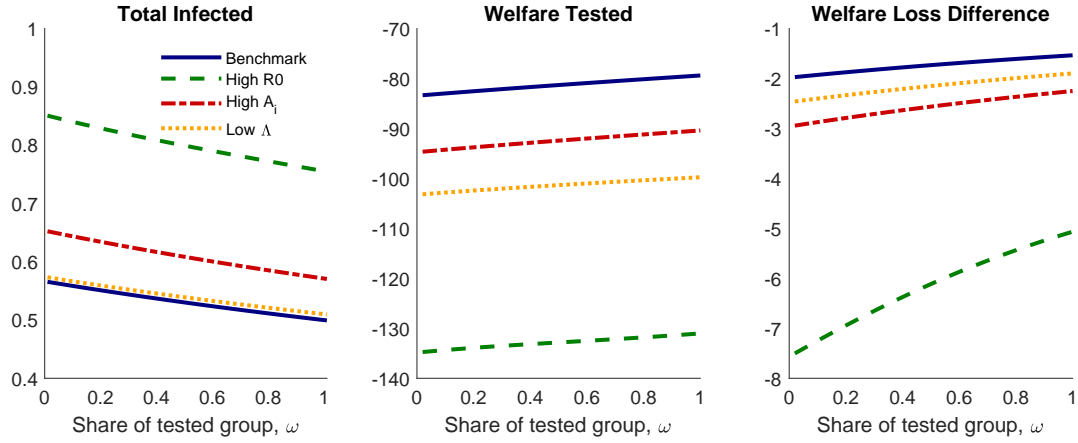
41000 lives within the first year (using the UK as benchmark). Thus, the more contagious is the virus, the more important are antibody tests.

Figure 4 also reports the implications of a social activity of symptomatic agents of $A_i^o = A_i^T = 0.9$ (50% larger than in my benchmark) and a cure-for-all probability of 33% per year (half that in my benchmark). Increasing $A_i^o = A_i^T$ (red dot-dashed lines) increases the total exposure to the virus and reduces welfare, which agrees with [Eichenbaum, Rebelo and Trabandt \(2020b\)](#): quarantining of detected infected individuals is paramount for viral testing to increase welfare. But this experiment shows that the gains from antibody testing do not change substantially. I also do not find substantial changes to the benefits of antibody testing if the probability of a vaccine is relatively low (see orange dotted lines).

My final robustness checks relate with α , the parameter governing the curvature of the utility of social activity. I report the results in Figure 5. One important message is that the extent of the epidemic depends substantially on α . For example, relative to $\alpha = 1$, assuming $\alpha = 0.5$ lowers total infections by about 30 percentage points while assuming $\alpha = 2$ rises infections by about 15 percentage points in the first 12 months. The logic is that a lower α reduces the curvature of the utility function; thus, it implies that agents are willing to significantly adjust their social activity in response to the epidemic.¹⁸ The gains of antibody testing are still sizeable: widespread and continuous antibody testing would prevent between 8% (in the case of $\alpha = 0.5$) and 10% (in the case of $\alpha = 1$) of COVID-19 related deaths.

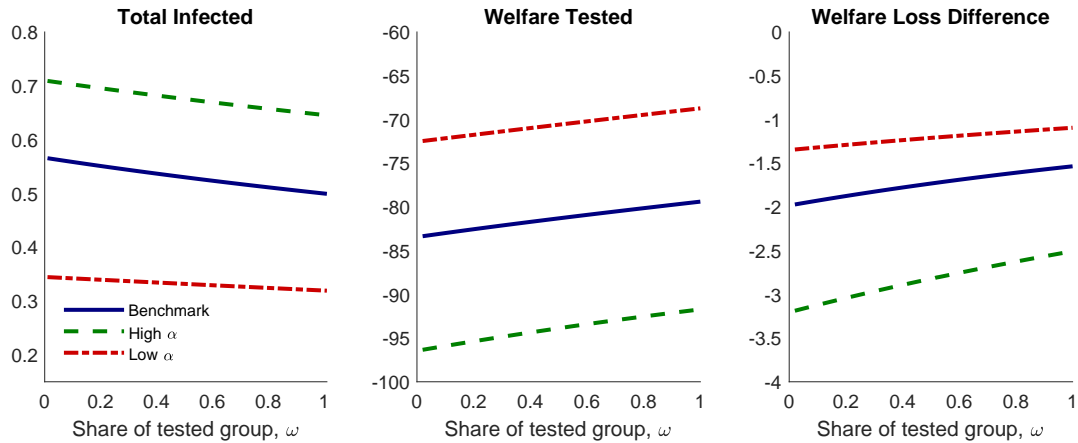
¹⁸In the limit, if $\alpha = \infty$, then agents would not adjust their social activity and my model would only include its epidemiological block.

Figure 4: Robustness Checks II



Note: This figure shows total infected after one year, $1 - N_{s,365}^o - N_{s,365}^T$, welfare of tested susceptible agents in period 1, $V_{s,1}^T$, and difference in welfare loss between tested and untested susceptible agents in period 1, $V_{s,1}^o - V_{s,1}^T$, as a function of the share of tested agents, ω . Solid blue lines present the results under the benchmark calibration. The other three lines change one parameter relative to benchmark: green dashed lines assume $\beta = 6/14$, red dot-dashed lines assume $A_i^o = A_i^T = 0.9$, and orange dotted lines assume $\Lambda = \frac{1}{1+0.05/365} \frac{1}{1+0.33/365}$.

Figure 5: Robustness Checks III



Note: This figure shows total infected after one year, $1 - N_{s,365}^o - N_{s,365}^T$, welfare of tested susceptible agents in period 1, $V_{s,1}^T$, and difference in welfare loss between tested and untested susceptible agents in period 1, $V_{s,1}^o - V_{s,1}^T$, as a function of the share of tested agents, ω . Solid blue lines present the results under the benchmark calibration. The other two lines assume different values of α : green dashed lines assume $\alpha = 1.5$ and red dot-dashed lines assume $\alpha = 3$.

My results also help to distinguish the roles of antibody and viral testing. [Eichenbaum, Rebelo and Trabandt \(2020b\)](#) study tests with a dual purpose: they simultaneously indicate viral presence (viral tests) and immunity (antibody tests). Instead, I focus on antibody tests, which only indicate immunity. This difference implies divergent results. [Eichenbaum, Rebelo and Trabandt](#) conclude that their dual-purpose tests can be nefarious for welfare unless there are quarantining measures. But this result only follows from the fact that diagnosed infected agents face no trade-off and (if unconstrained) increase their activity, leading to a rise in infections and lower welfare. In contrast, antibody testing unambiguously reduces the average social activity of susceptible and asymptomatic agents, implying an unambiguous welfare gain.

5 UK Scenarios

In this section, I apply my model to the UK as of 12 June 2020. I believe my model can offer a good reference point for how the COVID-19 pandemic might evolve as the UK government is gradually lifting lockdown measures and individual decision making becomes more important in the propagation of the virus. In this regard, my analysis complements that in [Pichler et al. \(2020\)](#) and [Costa Dias et al. \(2020\)](#).¹⁹ Both take different approaches to assess how to lift lockdown policies and reopen the UK economy while I focus on the pandemic evolution taking into account how agents are likely to react over time. In this section, I also examine how important antibody tests can be in shaping the remaining evolution of the COVID-19 pandemic in the UK. My conclusion is that antibody testing can avoid about 8% of COVID-19 related deaths from June 2020 to June 2021.

5.1 Starting Point

Relative to Section 3, I recalibrate the starting point of my simulations. As of June 12, the UK government reported approximately 41000 deaths associated with COVID-19.²⁰ I use this number to set $N_{d,1}^o + N_{d,1}^t = 0.0006$ and use ω to split deaths between the groups. On the same day, the ONS reported that about 6.8% of the population had antibodies (95% confidence interval: [5.21%, 8.64%]). These estimates are, however, from the period between 26 April and 24 May and might be low. Thus, I use a slightly higher target for recovered individuals: $N_{re,1}^o + N_{re,1}^t + N_{ri,1}^o + N_{ri,1}^t = 0.075$. To split the recovered among the two health states of the two groups, I use ω and σ ; in particular, I set $N_{re,1}^o = (1 - \sigma)(1 - \omega)0.075$. Also on June 12, the ONS reported that about 0.06% of the English population had COVID-19 between May 25 and June 7 (95% confidence interval: [0.02%, 0.12%]). Yet, given that these estimates are based on only 11 individuals testing positive for COVID-19 in a survey with 19933 participants, I prefer to be conservative and set $N_{e,1}^o + N_{e,1}^t + N_{i,1}^o + N_{i,1}^t = 0.0012$. I use ω to split between the two groups and assume that $N_{i,1}^y = 0.5N_{e,1}^y$. Susceptible agents, $N_{s,1}^y$, are the residual.

¹⁹See also [Ferguson et al. \(2020\)](#) for an early epidemiological model for the evolution of COVID-19 in the UK.

²⁰<https://coronavirus.data.gov.uk>, consulted in 17 June 2020.

5.2 Scenarios

I consider two main scenarios. One is the *unconstrained* scenario in which I use the benchmark calibration as in Section 3 and still include quarantine for symptomatic agents. The other is a *constrained* scenario in which I assume a permanently lower β because the UK government imposes policies that might restrict contagion like mandatory mask use in public transport.²¹ For example, [Mitze et al. \(2020\)](#) document that mandatory mask use strongly reduced contagiousness in Germany and [Eikenberry et al. \(2020\)](#) estimate and simulate an epidemiological model and find that mask use could significantly reduce new cases in New York and Washington.

It is very hard to quantify how much lower should β be in the constrained scenario relative to the unconstrained one. And this uncertainty has huge implications for any simulations that I can make. For example, if β falls to the point that $R_0 < 1$, then any epidemiological model would suggest that the epidemic will soon vanish in the UK. That, however, seems unlikely; for example, countries like China and South Korea (that were very efficient in attenuating contagion in the beginning of the epidemic) imposed stringent policies than the UK and still repeatedly report surges in new cases. Furthermore, if policies stop before herd immunity is achieved and all possible contagion sources are eradicated, then the epidemic restarts.

To find a reasonable β in constrained scenarios, I build on [Eikenberry et al. \(2020\)](#). They review the evidence on the filtering efficiency of different types of masks (from home-made to N95) to design multiple scenarios for the impact of mask use on β . Their simulations suggest that mask use can significantly reduce β : for example, in the extreme circumstance that all individuals properly wear N95 masks at all times, β would fall to virtually zero. Yet, mask use is only mandatory in public transport in England (it recommended in Northern Ireland, Scotland, and Wales) and the government does not recommend the use of surgical masks. Thus, these policies might not be very effective especially if most workers cannot continue to work from home. Given that, and the simulations in [Eikenberry et al.](#), my constrained scenario assumes that β is 40% lower than in the unconstrained one: $\beta = 1.8/14$. This, arguably, might be optimistic. But my main point is to devise a scenario that takes into account, at least to some extent, the effect of the policies of the UK government.

For completeness, I also analyse how a high value of life, $v = 120000$, and a higher proportion of symptomatic (and identified) agents, $\sigma = 0.5$, change the results. I study the case of a high v because, as argued above, the value of a prevented fatality that I use to calibrate the model is relatively low; it is likely that agents attach a higher value to their own life. I also study $\sigma = 0.5$ because there might be substantial improvements in the scale of viral testing (that identify the presence of a COVID-19 infection) and in the efficiency of contact tracing.

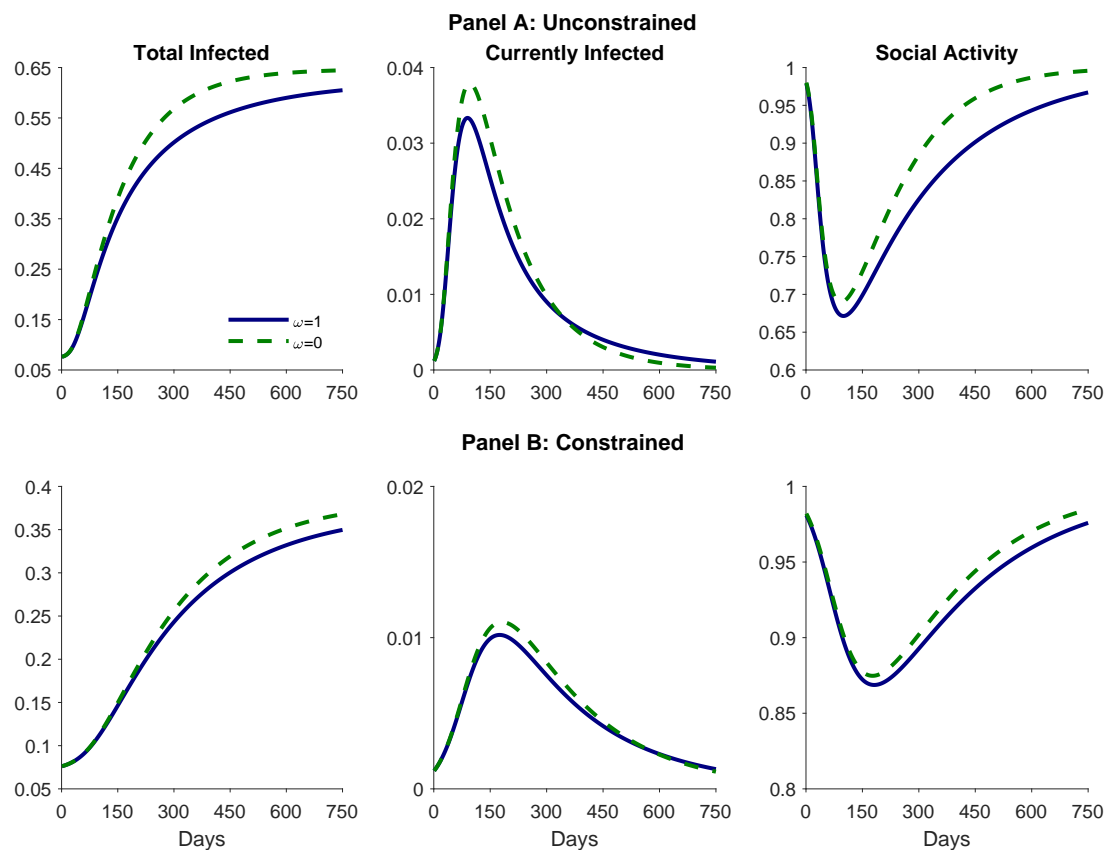
Finally, to grasp the implications of antibody testing, I split each scenario into two: continuous and widespread antibody testing ($\omega = 1$) and zero antibody testing ($\omega = 0$).

²¹<https://www.bbc.com/news/health-51205344>, consulted in 17 June 2020.

5.3 Simulations for the UK as of 12 June 2020

The results of my main simulations of the COVID-19 pandemic in the UK until July 2022 are presented in Figure 6. Blue solid lines represent the scenarios with continuous and widespread antibody testing ($\omega = 1$) and the green dashed lines represent the scenarios without antibody testing ($\omega = 0$). In Panels A and B, I show the unconstrained scenarios ($\beta = 3/14$) and constrained scenarios ($\beta = 1.8/14$), respectively. Each panel shows total exposure to the virus, the number of currently infected (both symptomatic and asymptomatic), and social activity of susceptible and asymptomatic agents.²²

Figure 6: Simulations for the UK as of 12 June 2020



Note: Each panel shows total infected irrespective of current status, $1 - N_{s,t}^o - N_{s,t}^t$, infected agents, $N_{e,t}^o + N_{e,t}^t + N_{i,t}^o + N_{i,t}^t$, and social activity of susceptible agents in the cases of $\omega = 1$ (all agents are tested; blue solid lines) and $\omega = 0$ (all agents are untested; green dashed lines). Panels A and B assume, respectively, that $\beta = 3/14$ and $\beta = 1.8/14$.

Both scenarios suggest that there will be a large second wave of infections unless a cure or

²²In the case of $\omega = 0$, the social activity of recovered undocumented equals that of susceptible and asymptomatic agents; in the case of $\omega = 1$, their social activity is the maximum possible, normalised to unity.

vaccine is discovered soon. This result already takes into account that agents reduce their social activity, which significantly reduces the scale of the epidemic.²³ And antibody testing does not have the firepower to prevent it; it only has the firepower to mitigate it.

As is well-known, the choice of β plays a fundamental role in epidemiological models. Based on my simulations, the constrained scenario implies a reduction of 20 percentage points in total exposure after two years and reduces the peak infection rate to less than a third while postponing it in time. Moreover, economic agents do not need to constrain their social activity by as much, which improves welfare.²⁴ Thus, if the policies conducted by the UK government do reduce β by 40%, those policies save many lives and gain time to search for a cure or vaccine.

Antibody testing has a non-negligible effect in reducing exposure and peak infections. In particular, the simulations of the model suggest that widespread and continuous antibody testing lower additional exposure and deaths by 12.6% and 7.7% in the unconstrained and constrained scenarios in the first 12 months, respectively. In absolute terms, this corresponds to 28000 and 7000 lives. The gains of antibody testing become less impressive with time but are persistent; thus, antibody testing crucially gains some time for a cure and/or vaccine to be discovered.

In Figures A1 and A2 in the Appendix, I report the simulations for the UK if $\nu = 120000$ and $\sigma = 0.5$, respectively. In both cases, the extent of the epidemic in the UK is lower. And this is especially clear in the case of high ν due to very strong and persistent reduction in social activity. Furthermore, compared with Figure 6, antibody testing saves less lives in the two cases for the reasons identified in Section 4.2. Yet, in both cases, there are still visible gains from large scale antibody testing especially after the first 10 months.

6 Random & Periodic Testing

Arguably, the gains from antibody testing in revealing the health state to individuals suggested by my model are hard to reach if home antibody tests that can be used without medical supervision and with very low marginal cost are not available. Even though some antibody testing is better than nothing, most of the gains of antibody testing seem to require that a sizeable share of the population is tested every day (at least until they develop antibodies). Without home antibody tests, this seems impracticable; it would be more realistic to assume that agents are tested in different days and with different periodicity. As explained in Section 2, however, considering this scenario makes the model intractable. The main reason is general equilibrium effects: all agents' decisions affect all other agents' decisions through total social activity of infected agents, I_t . But, in this section, I bypass this problem to show

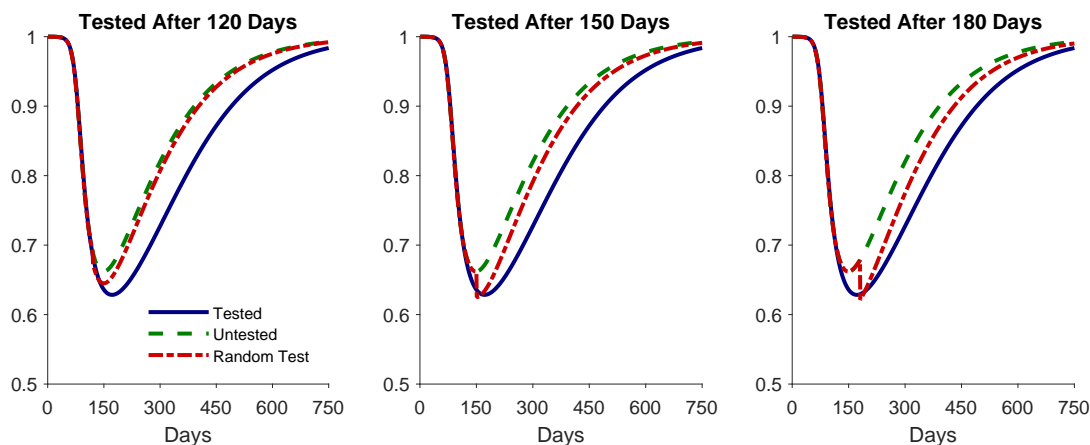
²³I simulated, in parallel, an epidemiological model like mine with $A_t^o = A_t^r = 1$ for all t and continued to impose that $A_h^o = A_h^r = 0.6$. In the unconstrained scenario, 90% of agents are exposed in less than 200 days. And in the constrained one, 56% are exposed in 400 days.

²⁴Arguably, the policies imposed by the UK government like mandatory mask use in public transport might reduce welfare. I am not measuring those costs in my analysis.

that there are sizeable gains to testing agents at least once and that most of the gains from continuous antibody testing can be harvested if agents are periodically tested.

In this section, I study the problem of a marginal untested susceptible agent who is unsure whether she is currently asymptomatic or whether she has recovered without developing symptoms. Because this agent corresponds to a very tiny fraction of the population, her decisions have insignificant effects on I_t ; thus, to continue, I assume that those effects are zero, implying that the optimal decisions of all other agents are unchanged. I consider two scenarios. In one scenario, I study how the marginal agent reacts if she unexpectedly tests her antibodies at some point in time; I present the results in Figure 7. In the other scenario, I study how the marginal agent reacts if she knows that she is tested every T periods; I present the results in Figure 8. In both scenarios, the marginal agent decides given an equilibrium path for I_t , which I obtain using my benchmark calibration and setting $\omega = 0.01$ (only 1% are continuously tested).²⁵

Figure 7: Random Antibody Testing of a Marginal Economic Agent



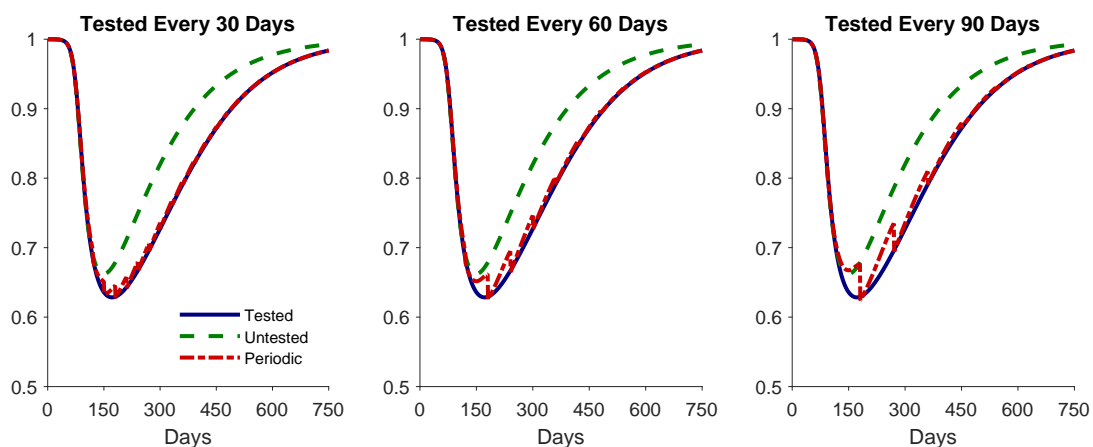
Note: This figure contrasts the social activity of the marginal untested agent when tested only once (red dot-dashed lines) with the social activity of continuously tested agents (blue solid line) and of untested agents (green dashed lines).

To grasp how random and periodic testing affects decision making, Figures 7 and 8 contrast the social activity of the marginal agent (red dot-dashed line) with that of continuously tested agents (blue solid line) and that of untested agents (green dashed line). Figure 7 shows that if an agent is randomly tested at some period, she permanently reduces her social activity relative to never being tested because she learns that she is in a riskier position than previously thought (she can either be susceptible or asymptomatic; she is not immune). This concurs

²⁵In practice, after obtaining I_t , I reset $N_{re,j}$ where j denotes the dates in which the agent is tested. I do it because if the agent tests positive, her utility is normalised to zero from that period on, while if she tests negative, she learns that she is not recovered undocumented. If the agent is aware that she will be tested, I also set $V_{re,j-1} = 0$ because, in the day before the test, the agent is unconstrained. Furthermore, given that the agent knows that she is under a periodic testing regime, setting $N_{re,j} = 0$ and $V_{re,j-1} = 0$ affects all other periods recursively.

with the results documented in [Gong \(2015\)](#) as he reports that if individuals are surprised by a negative HIV test, they reduce their risky sexual exposure. Figure 7 also shows that most of the reduction in social activity caused by random immunological testing occurs if the test is not conducted when the epidemic starts but rather a few months later. The reason is that, in the first few months, the probability of being recovered undocumented is low; thus, agents behave similarly irrespective of testing. It is only when the probability of being recovered undocumented builds up that random antibody testing significantly lowers social activity. Another interesting result in Figure 7 is that the marginal agent temporarily constrains her social activity by more than continuously tested agents. She does that because she internalises the future loss of being recovered undocumented, which dominates for low n_{re} (see the discussion in Section 2.2.2).

Figure 8: Periodic Antibody Testing of a Marginal Economic Agent



Note: This figure contrasts the social activity of the marginal untested agent when tested periodically (red dot-dashed lines) with the social activity of continuously tested agents (blue solid line) and of untested agents (green dashed lines).

Figure 8 shows that periodic antibody testing remarkably lowers social activity. In fact, if the marginal agent is tested every 30 days, her social activity is almost indistinguishable from agents tested every day. Furthermore, even if the marginal agent is tested every 90 days, her optimal path of social activity falls significantly and is notably close to that of tested agents after the first nine months of the epidemic. Therefore, given that the marginal agent behaves similarly to tested agents, these experiments suggest that the benefits of large scale antibody testing can be collected if all agents are tested every 30 or 60 days. Furthermore, these experiments suggest that it is possible to approximate the social activity of the marginal agent to that of the tested agent if: 1) there are no immunological tests in the first few months of the epidemic; 2) there is frequent antibody testing during the period in which there are more infections; and 3) there is a gradual reduction in the periodicity of testing as the epidemic fades.²⁶

²⁶The actual timing of these phases depend on the calibration of the model.

7 Concluding Remarks

In the context of the COVID-19 pandemic, the literature has advocated many non-pharmaceutical interventions to reduce the scale of the pandemic and, if possible, its economic costs. [Alvarez, Argente and Lippi \(2020\)](#) and [Glover et al. \(2020\)](#) argue that lockdowns and quarantines can be optimised to increase welfare; [Acemoglu et al. \(2020\)](#) and [Gollier \(2020\)](#) argue that age-specific lockdown and quarantine policies are far better than age-indifferent ones; [Mitze et al. \(2020\)](#) document that mandatory mask use reduces deaths; [Berger, Herkenhoff and Mongey \(2020\)](#), [Brotherhood et al. \(2020\)](#), [Eichenbaum, Rebelo and Trabandt \(2020b\)](#), and [Piguillem and Shi \(2020\)](#) argue that the combination of testing and quarantining increases welfare. In this paper, I argue that antibody testing is another non-pharmaceutical intervention – not recognised so far in the literature – that can save lives and increase welfare.

I use a simple economic model in which agents are uncertain about their health state to show the importance of antibody testing. In the model, agents are optimally more socially active when they have a slightly positive perceived probability of having recovered from a COVID-19 infection without developing symptoms. Although some did recover and are immune, most are still susceptible or asymptomatic individuals. Thus, this surge in social activity increases both directly and indirectly the exposure of susceptible agents. The final effects of this health state uncertainty is a higher proportion of the population exposed to the virus, more death, and greater welfare losses. Yet, antibody testing, by revealing whether individuals are immune or not, stops this chain of events at the start. In simulations calibrated to the UK, my model suggests that antibody testing prevents about 8% of COVID-19 related deaths between June 2020 and June 2021.

My model also hints that the welfare gains of antibody testing and its efficacy in preventing COVID-19 contagion depends:

- Positively on the contagiousness of the virus because the effects of increased activity in the absence of antibody tests compound quickly;
- Positively on the probability of being asymptomatic because it increases the probability that agents are unaware that they are immune;
- Negatively on viral testing and contact tracing because (i) these policies increase the number of individuals aware of recovery absent antibody tests and (ii) also reduce contagiousness;
- Negatively on other interventions that likely lower contagiousness like mandatory mask use.

My simulations suggest that antibody testing is not a relevant tool to lower contagion in the beginning months of the epidemic. It only becomes important when a sufficiently large proportion of the population is immune but unaware of its immunity. This is precisely the case in countries like the UK and Spain but, even more clearly, in cities like London and New York where more than 15% of the population is estimated to be immune. Those numbers, although staggering, fall significantly short of herd immunity. Thus, my model flags the importance to distinguish those who are immune from those who are not to curb down the

future propagation of COVID-19.

There is much uncertainty about COVID-19. For example, what is the actual case fatality rate? What is the proportion of asymptomatic? How contagious is the virus? How does it propagate? We have learned a lot in the past six months and I have used that knowledge as best as I could in designing and calibrating my model. But one critical source of uncertainty, which will likely not be resolved soon, regards the duration of immunity. I follow the economics literature and assume that recovered individuals are permanently immune. Yet, there is not yet conclusive evidence that immunity is permanent. If it is not, then the role of antibody testing likely depends on the type of tests available. In particular, if immunity is short-lived, then large public and private investments in developing easy-to-use and reliable home antibody tests seems extremely important for agents to identify when they become immune and when they lose that immunity.

References

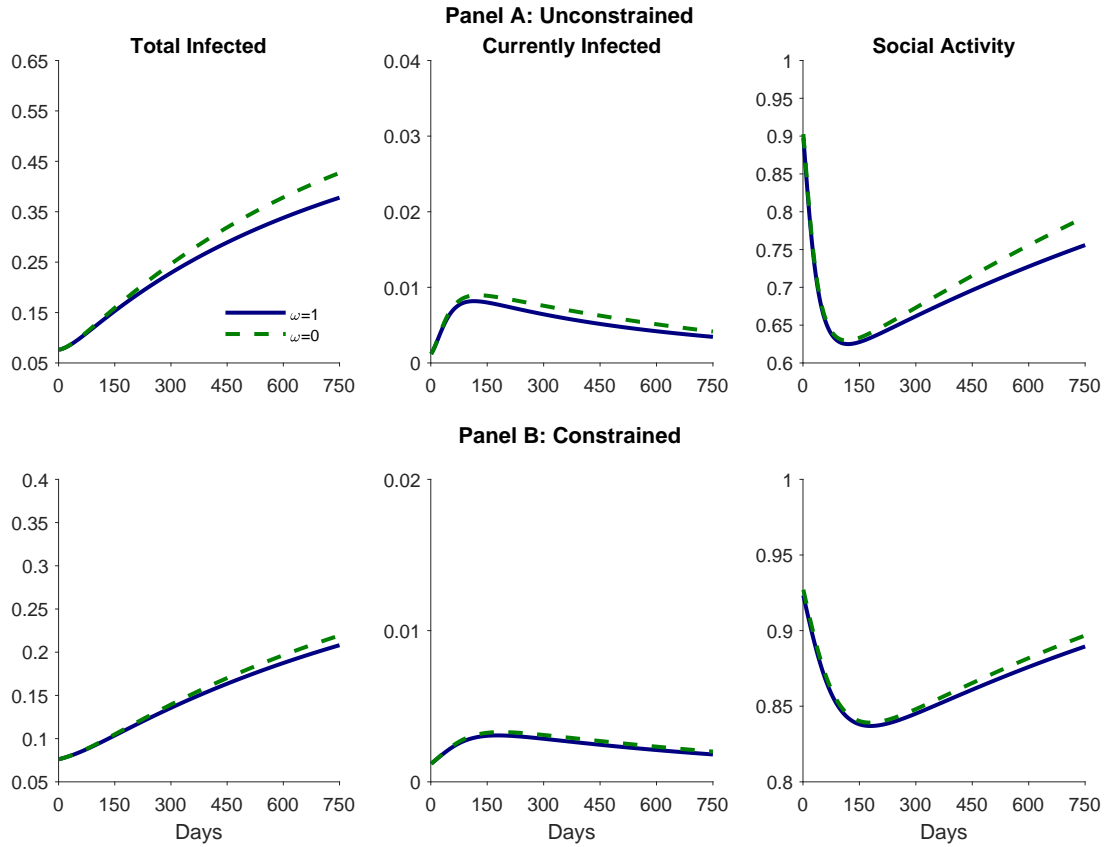
- Acemoglu, Daron, Victor Chernozhukov, Iván Werning, and Michael D Whinston.** 2020. “Optimal Targeted Lockdowns in a Multi-Group SIR Model.” National Bureau of Economic Research Working Paper 27102.
- Alvarez, Fernando E, David Argente, and Francesco Lippi.** 2020. “A Simple Planning Problem for COVID-19 Lockdown.” National Bureau of Economic Research Working Paper 26981.
- Atkeson, Andrew.** 2020. “What Will Be the Economic Impact of COVID-19 in the US? Rough Estimates of Disease Scenarios.” National Bureau of Economic Research Working Paper 26867.
- Barnett, Michael, Greg Buchak, and Constantine Yannelis.** 2020. “Epidemic Responses Under Uncertainty.” National Bureau of Economic Research Working Paper 27289.
- Berger, David W, Kyle F Herkenhoff, and Simon Mongey.** 2020. “An SEIR Infectious Disease Model with Testing and Conditional Quarantine.” National Bureau of Economic Research Working Paper 26901.
- Bodenstein, Martin, Giancarlo Corsetti, and Luca Guerrieri.** 2020. “Social Distancing and Supply Disruptions in a Pandemic.”
- Brotherhood, Luiz, Philipp Kircher, Cezar Santos, and Michèle Tertilt.** 2020. “An Economic Model of the Covid-19 Epidemic: The Importance of Testing and Age-Specific Policies.”
- Costa Dias, Monica, Christine Farquharson, Rachel Griffith, Robert Joyce, and Peter Levell.** 2020. “Getting People Back Into Work.”
- Delavande, Adeline, and Hans-Peter Kohler.** 2015. “HIV/AIDS-related Expectations and Risky Sexual Behaviour in Malawi.” *The Review of Economic Studies*, 83(1): 118–164.
- Eichenbaum, Martin S, Sergio Rebelo, and Mathias Trabandt.** 2020a. “The Macroeconomics of Epidemics.” National Bureau of Economic Research.
- Eichenbaum, Martin S, Sergio Rebelo, and Mathias Trabandt.** 2020b. “The Macroeconomics of Testing and Quarantining.” National Bureau of Economic Research Working Paper 27104.
- Eikenberry, Steffen E., Marina Mancuso, Enahoro Iboi, Tin Phan, Keenan Eikenberry, Yang Kuang, Eric Kostelich, and Abba B. Gumel.** 2020. “To Mask Or Not to Mask: Modeling the Potential for Face Mask Use by the General Public to Curtail the COVID-19 Pandemic.” *Infectious Disease Modelling*, 5: 293 – 308.
- Emery, Jon C, Timothy W Russel, Yang Liu, Joel Hellewell, Carl AB Pearson, Gwen M Knight, Rosalind M Eggo, Adam J Kucharski, Sebastian Funk, Stefan Flasche, et al.** 2020. “The Contribution of Asymptomatic SARS-CoV-2 Infections to Transmission – A Model-Based Analysis of the Diamond Princess Outbreak.” Mimeo.

- Farboodi, Maryam, Gregor Jarosch, and Robert Shimer.** 2020. "Internal and External Effects of Social Distancing in a Pandemic." National Bureau of Economic Research Working Paper 27059.
- Ferguson, Neil, Daniel Laydon, Gemma Nedjati Gilani, Natsuko Imai, Kylie Ainslie, Marc Baguelin, Sangeeta Bhatia, Adhiratha Boonyasiri, Zulma Cucunuba Perez, Gina Cuomo-Dannenburg, Amy Dighe, Iliaria Dorigatti, Han Fu, Katy Gaythorpe, Will Green, Arran Hamlet, Wes Hinsley, Lucy Okell, Sabine van Elsland, Hayley Thompson, Robert Verity, Erik Volz, Haowei Wang, Yuanrong Wang, Patrick GTWalker, Caroline Walters, Peter Winskill, Charles Whittaker, Christl Donnelly, Steven Riley, and Azra Ghani.** 2020. "Report 9: Impact of Non-Pharmaceutical Interventions (NPIs) to Reduce COVID-19 Mortality and Healthcare Demand."
- Friedman, Willa Helterline.** 2018. "Antiretroviral Drug Access and Behavior Change." *Journal of Development Economics*, 135: 392 – 411.
- Garibaldi, Pietro, Espen R. Moen, and Christopher A. Pissarides.** 2020. "Modelling Contacts and Transitions in the SIR Epidemics Model." *Covid Economics Vetted and Real-Time Papers, CEPR*.
- Glover, Andrew, Jonathan Heathcote, Dirk Krueger, and José-Víctor Ríos-Rull.** 2020. "Health versus Wealth: On the Distributional Effects of Controlling a Pandemic." National Bureau of Economic Research.
- Gollier, Christian.** 2020. "If the Objective is Herd Immunity, On Whom Should It Be Built?" *EconPol Policy Brief*, 29(4).
- Gong, Erick.** 2015. "HIV Testing and Risky Sexual Behaviour." *The Economic Journal*, 125(582): 32–60.
- Greenwood, Jeremy, Philipp Kircher, Cezar Santos, and Michèle Tertilt.** 2019. "An Equilibrium Model of the African HIV/AIDS Epidemic." *Econometrica*, 87(4): 1081–1113.
- Hall, Robert E, Charles I Jones, and Peter J Klenow.** 2020. "Trading Off Consumption and COVID-19 Deaths." National Bureau of Economic Research Working Paper 27340.
- Hethcote, Herbert W.** 2000. "The Mathematics of Infectious Diseases." *SIAM review*, 42(4): 599–653.
- Hornstein, Andreas.** 2020. "Social Distancing, Quarantine, Contact Tracing, and Testing: Implications of an Augmented SEIR Model." *Federal Reserve Bank of Richmond Working Papers*, 20: 1–38.
- Kaplan, Greg, Benjamin Moll, and Gianluca Violante.** 2020. "Pandemics According to HANK." Mimeo.
- Keppo, Jussi, Marianna Kudlyak, Elena Quercioli, Lones Smith, and Andrea Wilson.** 2020. "The Behavioral SIR Model." Mimeo.

- Kermack, William Ogilvy, and Anderson G McKendrick.** 1927. "A Contribution to the Mathematical Theory of Epidemics." *Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character*, 115(772): 700–721.
- Kremer, Michael.** 1996. "Integrating Behavioral Choice into Epidemiological Models of AIDS*." *The Quarterly Journal of Economics*, 111(2): 549–573.
- Krueger, Dirk, Harald Uhlig, and Taojun Xie.** 2020. "Macroeconomic Dynamics and Reallocation in an Epidemic." National Bureau of Economic Research Working Paper 27047.
- Lakdawalla, Darius, Neeraj Sood, and Dana Goldman.** 2006. "HIV Breakthroughs and Risky Sexual Behavior*." *The Quarterly Journal of Economics*, 121(3): 1063–1102.
- Maloney, William, and Temel Taskin.** 2020. "Determinants of Social Distancing and Economic Activity During COVID-19: A Global View." mimeo.
- Meyerowitz-Katz, Gideon, and Lea Merone.** 2020. "A Systematic Review and Meta-Analysis of Published Research Data on COVID-19 Infection-Fatality Rates." *medRxiv*.
- Mitze, Timo, Reinhold Kosfeld, Johannes Rode, and Klaus Wälde.** 2020. "Face Masks Considerably Reduce COVID-19 Cases in Germany: A Synthetic Control Method Approach."
- Philipson, Tomas J., and Richard A. Posner.** 1993. *Private choices and public health: The AIDS epidemic in an economic perspective*. Harvard University Press.
- Pichler, Anton, Marco Pangallo, R Maria del Rio-Chanona, François Lafond, and J Doynne Farmer.** 2020. "Production Networks and Epidemic Spreading: How to Restart the UK Economy?" Mimeo.
- Piguillem, Facundo, and Liyan Shi.** 2020. "Optimal COVID-19 Quarantine and Testing Policies."
- Punt, Jenni, Sharon Stranford, Patricia Jones, and Judith A. Owen.** 2018. *Kuby Immunology*. Eighth ed., Macmillan Learning.
- Stock, James H.** 2020. "Data Gaps and the Policy Response to the Novel Coronavirus." *Harvard Environmental Economics Program Discussion Paper Series*.
- Thomas, Philip, and Ian Waddington.** 2017. "What Is the Value of Life? A Review of the Value of a Prevented Fatality Used by Regulators and Others in the UK." *Nuclear Future*, 13(1): 32–39.
- Toxvaerd, Flavio.** 2020. "Equilibrium Social Distancing." Mimeo, University of Cambridge.
- Wang, Huwen, Zezhou Wang, Yinqiao Dong, Ruijie Chang, Chen Xu, Xiaoyue Yu, Shuxian Zhang, Lhakpa Tsamlag, Meili Shang, Jinyan Huang, et al.** 2020. "Phase-Adjusted Estimation of the Number of Coronavirus Disease 2019 Cases in Wuhan, China." *Cell discovery*, 6(1): 1–8.

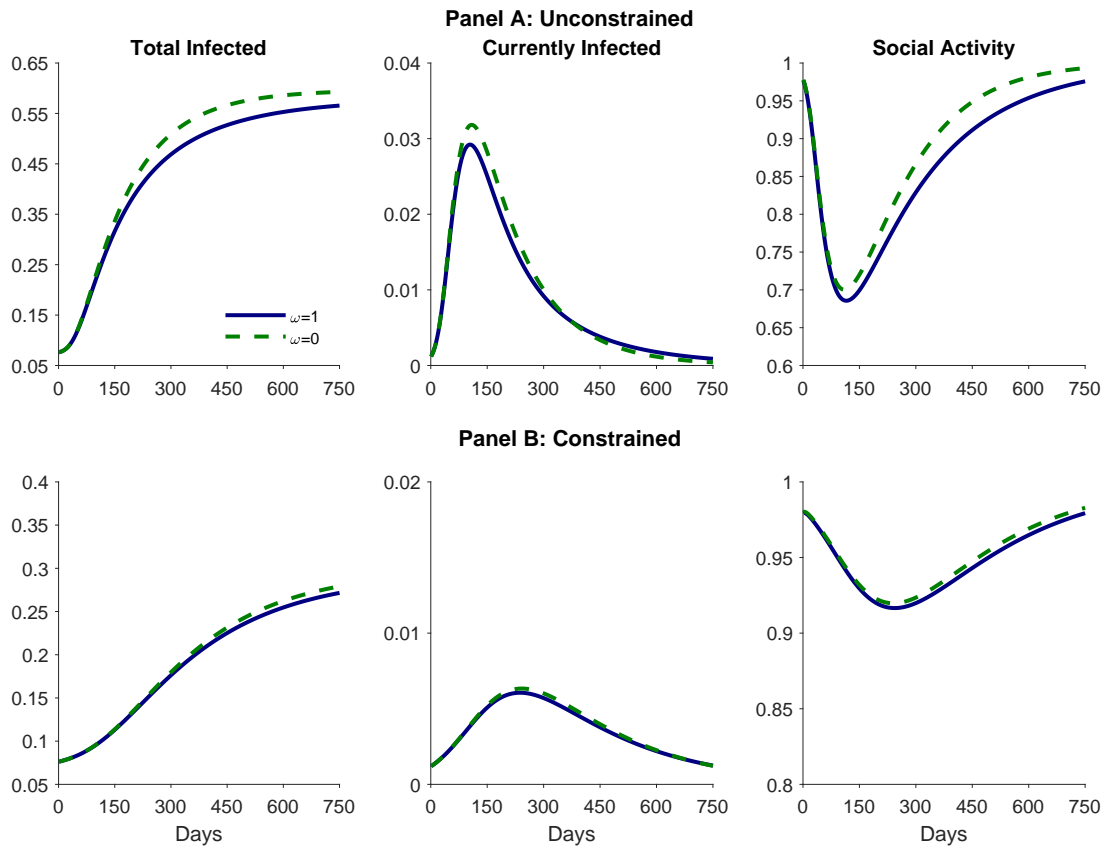
A Other UK Scenarios

Figure A1: Simulations for the UK as of 12 June 2020 – High ν



Note: Each panel shows total infected irrespective of current status, $1 - N_{s,t}^o - N_{s,t}^T$, infected agents, $N_{e,t}^o + N_{e,t}^T + N_{i,t}^o + N_{i,t}^T$, and social activity of susceptible agents in the cases of $\omega = 1$ (all agents are tested; blue solid lines) and $\omega = 0$ (all agents are untested; green dashed lines). Panels A and B assume, respectively, that $\beta = 3/14$ and $\beta = 1.8/14$. Both panels assume that $\nu = 120000$.

Figure A2: Simulations for the UK as of 12 June 2020 – High σ



Note: Each panel shows total infected irrespective of current status, $1 - N_{s,t}^o - N_{s,t}^t$, infected agents, $N_{e,t}^o + N_{e,t}^t + N_{i,t}^o + N_{i,t}^t$, and social activity of susceptible agents in the cases of $\omega = 1$ (all agents are tested; blue solid lines) and $\omega = 0$ (all agents are untested; green dashed lines). Panels A and B assume, respectively, that $\beta = 3/14$ and $\beta = 1.8/14$. Both panels assume that $\sigma = 0.5$.