A theory of voluntary testing and self-isolation in an ongoing pandemic

Thomas Hellmann1 | Veikko Thiele2

1Saïd Business School, University of Oxford, Oxford, UK
2Smith School of Business, Queen's University, Kingston, Ontario, Canada

Abstract

Beyond Covid-19, there is a growing interest in what economic structures will be needed to face ongoing pandemics. In this paper, we focus on the diagnostic problem and examine a new paradigm of voluntary self-testing by private individuals. We develop a dynamic model where individuals without symptoms face daily choices of either taking the risk of going out (to work and socialize), staying at home in self-isolation, or using a test to verify whether they are infected before going out. Our central insight is that the equilibrium public infection risk falls when home-based testing becomes cheaper and easier to use, even if they generate both false-positive (type I error) and false-negative (type II error) test outcomes. We also show that the presence of naïve individuals actually reduces the equilibrium infection risk in the economy. Overall our model shows that, even if inaccurate, home-based tests are vital for an economy facing an ongoing pandemic.

1 INTRODUCTION

The Covid-19 pandemic, which started in November 2019, has caused tremendous economic and social challenges globally. However, Covid-19 is not, and will not be, the only pandemic that poses a threat to individuals and the economy. Covid-19 was preceded by SARS, MERS, Ebola, and AIDS, and is likely to be followed by further mutations of the Coronavirus and/or other infectious diseases. An important policy question is what structures are needed to curb the impact of an on-going pandemic on the economy as a whole, as well as on the social interactions of individuals?
In this paper, we focus on voluntary testing by private individuals as a way to curtail the infection risk in an ongoing pandemic. We develop an economic theory to answer the following three sets of questions:

(i) What is the structure of the private demand for testing in a pandemic? What types of people are most interested in regularly buying tests to verify their health status? How does the price and ease of use affect this?

(ii) How important is the accuracy of the test? What are the implications of false negatives and false positives? How do rational people respond to testing inaccuracies? What if people are naïve and do not understand that tests are inaccurate?

(iii) What determines the equilibrium infection risk in an ongoing pandemic? How does the availability of home-based tests affect this risk?

There is considerable disagreement about the proper approach to testing. At the risk of simplifying, there are two schools of thought that we will call the “clinical mainstream view” versus the “public health view.” The clinical mainstream view is that diagnostic tests are the responsibility of the medical system. Testing should be performed by medically trained staff and should occur at the point of care (e.g., hospitals), with the help of approved labs. Diagnostic accuracy is considered extremely important, as it guides all subsequent medical interventions. Two key measures are “sensitivity” which concerns the absence of false negatives (where infected people are erroneously found to be free of the virus) and “specificity” which concerns the absence of false positives (where uninfected people are erroneously found to have the virus). The clinical mainstream view is deeply sceptical about any testing outside of a clinical setting. For example, in September 2020 the homepage of the Federal Trade Commission included the following warning:¹ “Be wary of ads for test kits. Most test kits being advertised have not been approved by the FDA and aren’t necessarily accurate.”

Some public health experts beg to differ. They argue that testing accuracy is not necessarily the most important aspect. Instead what matters is to quickly identify as many infected people as early as possible before they further spread the disease. Dr. Michael Mina, from the Harvard Chan School of Public Health, argues:² “As long as you’re using the test on a pretty frequent basis, you will be more likely than not to catch the person on the day they might go out and transmit. And they’ll know to stay home.” Carroll (July 28, 2020, NYT) further argues: “These tests aren’t perfect, but that’s not the point [---] every single case we identify is better than not. We can isolate that person from the population and prevent infections.”

This debate raises some interesting questions about the economic role of voluntary testing by private individuals. Is testing accuracy the most important factor, or do other aspects such as ease of use and price matter more from an economic perspective? How important is testing accuracy if all people are rational and fully understand the limitations of a test? What if some naïve consumers do not understand these imperfections?

Numerous start-ups and established companies have developed new testing technologies for the Coronavirus that are faster and cheaper than the standard polymerase chain reaction tests, although they typically are not more accurate (Hellmann, 2020; Meyer & Madrigal, 2020). Most important, testing is no longer just a medical tool for diagnosis, it is also becoming an economic

¹See https://www.ftc.gov/coronavirus/scams-consumer-advice.
²Quoted by Richard Harris, NPR, July 22, 2020; see https://www.npr.org/sections/health-shots/2020/07/22/893931848/rapid-cheap-less-accurate-coronavirus-testing-has-a-place-scientists-say.
decision tool. There is a new demand for testing that is administered outside of clinical settings, including self-testing at home. Unlike clinical tests which are used when patients have symptoms, and are administered by qualified staff, home-based tests are for asymptomatic private individuals. They self-administer them to make daily decisions about whether to “go out” and participate in public life, or “stay at home” and self-isolate. This simple choice is at the core of an ongoing pandemic. People who stay at home do not infect others, and find out in due time whether they get ill or not. By contrast, people who go out risk getting infected, and risk infecting others. The choice of “going out” should be interpreted broadly, to include going out to work (to generate income), going out to socialize (e.g., visit friends, look after relatives), and going out to consume (e.g., go shopping, eat out).

Agrawal et al. (2020), and Gans (2020a) argue that pandemics are essentially an information problem, especially when infections are transmitted by people without symptoms (aka asymptomatic transmission). For example, Johansson et al. (2021) estimate that about 59% of all Covid-19 transmissions are caused by people not showing any symptoms. The major disruptions to economic life occur because people lack information about who is infected, and when. The key message from our paper is that voluntary testing, even if not fully accurate, constitutes an effective tool to mitigate this information problem, and can therefore curtail the infection risk in a pandemic.

In this paper, we develop a theoretical model to understand the role of voluntary testing by private individuals to reduce the infection risk in an ongoing pandemic. Our model resembles the so-called susceptible-infectious-susceptible (SIS) model, where individuals can move from “susceptible” to “infectious” to “susceptible.” We focus on long-term steady-state equilibria where people make recurring decisions in the face of ongoing risks. Specifically, we consider private individuals making recurring decisions whether to go out into public places, versus staying at home to self-isolate. Going out permits the usual economic activities and gives people positive utility, whereas self-isolation avoids getting infected and infecting others. Importantly, the decision to go out versus self-isolate is made by people who show no symptoms. We first consider a benchmark model where these individuals make this decision without testing. In the main model we then introduce voluntary self-testing. However, tests can be inaccurate and may generate type I (false positive) and type II (false negative) errors.

We obtain three main sets of results. First, in our base model without testing, some individuals choose to go out (and may get infected or may infect others), while everyone else stays home and self-isolates. When introducing testing, we show that some of the individuals that would have always gone out in the absence of testing, now use a test and stay home and self-isolate when the test turns out to be positive. Moreover, some of the individuals that would have always stayed home in the absence of testing, now use a test and go out when the test result is negative. The lower the price of a test, and the easier its use, the more people of both types switch to regular testing.

Second, self-testing plays an important role because it affects who goes out and who self-isolates. This changes how contagious on average people in public places are, and thus, what the equilibrium infection risk is. Our two most important results are that lowering the price of testing (i) reduces the risk of getting infected when going out, and (ii) increases social welfare, as measured by the sum of utilities of all agents in the economy. An important caveat is that the

3Technically, a steady-state means a constant infection rate, also known as “$R = 1$”. Gans (2020b) argues that most behavioural models of pandemics converge to $R = 1$ equilibria.

4Note that we only consider these two choices (“go out” vs. “stay home”) in our base model.
total infection rate in the economy (which includes people going out and self-isolating) may go up or down, because cheaper testing brings some people out of self-isolation, exposing them to infection risk.

Third, we show that tests remain economically valuable even if not perfectly accurate. The clinical mainstream view frequently warns against false negatives, where people go out erroneously thinking they are not infectious. However, the implicit comparison in this argument is a perfect test where no false negatives occur. Our model makes the more realistic comparison, where the alternative to an imperfect test is no test at all. Some of the people with false negatives would have stayed at home, but others would have gone out anyway. As long as an imperfect test provides some information, it helps to keep higher-risk people at home, and allows lower-risk people to go out.

Rational people who fully understand inaccuracies reduce their demand if tests are less accurate. However, naïve consumers may not understand inaccuracies and put too much trust into the tests. A surprising result is that having more naïve people in the economy increases self-testing and reduces the equilibrium infection risk.

Our analysis has two important public policy implications. First, it suggests a rationale for governments to encourage voluntary self-testing in pandemics, subsidizing the price of tests, and/or making them more easily accessible. There is also a rationale for encouraging the development of better testing solutions. Second, our theory shows that less accurate tests can still make a positive contribution to reducing infection risks, and should therefore not be blocked by overly stringent regulations. The traditional regulatory argument is that any new home-based tests should be compared against the alternative of using the best prevailing clinical test. However, a key insight from our model is that this is not an economically relevant comparison. The alternative to home-based tests is asymptomatic people having no test at all. The policy implication from our paper is therefore that regulators should calibrate their benchmark used to allow home-based testing devices.

The remainder of this paper is organized as follows. In the next section, we discuss the related literature. In Section 3 we introduce our base model. In Section 4 we derive the equilibrium choices of individuals and the public infection risk in the absence of testing. In Section 5 we introduce (imperfect) testing and show how it changes the equilibrium outcome. Section 6 considers several model extensions. Section 7 discusses the robustness of our results and the limitations of our model. Section 8 concludes. All proofs are in the appendix.

2 RELATED LITERATURE

Our model structure is inspired by the so-called compartment models, commonly used by mathematical epidemiologists to analyze the dynamics of infectious diseases. The standard compartment model is the so-called SIR model, where individuals can move from “susceptible,” to “infectious,” to “recovered.” In these models individuals have some immunity after recovering from an infection. In our model, however, individuals do not develop any immunity after an infection, and can get infected again. The closest variant of the SIR model to

---

5See, for example, Brauer and Castillo-Chavez (2012) and Tang et al. (2020) for overviews of the general compartment disease models, and Boucekkine et al. (2021) for a review of the economics literature using compartment models to analyze disease progressions.
our model, in spirit, is therefore the SIS model where individuals can move from “susceptible,” to “infectious,” to “susceptible,” and do not develop any immunity. Our main focus is on how the availability of (imperfect) tests changes individual behavior, and therefore the transition rates between the different “compartments.”

An important feature of our model is that infected individuals, when going out, impose a negative externality on others, as this increases the infection risk in the economy. Gersovitz and Hammer (2004) and Goenka and Liu (2020) also include such a negative externality problem in an SIS model, and show that it is optimal for the government to subsidize the prevention and therapy of the disease (Gersovitz & Hammer, 2004) and public health care more generally (Goenka & Liu, 2020). In our model we assume that individuals have altruistic preferences of not wanting to infect others (e.g., their relatives, friends, or work colleagues). They therefore partially internalize the negative externality of transmitting the disease to others. Moreover, our main focus is on how private testing can mitigate infection risks in the economy.

Naturally, our paper is closely related to the economics literature using compartment models to analyze the effects of testing. Eichenbaum et al. (2020) show that test-based quarantines can significantly reduce the social costs compared to nontest-based lockdowns during the Covid-19 pandemic. Moreover, Gori et al. (2022) show that extensive testing, contact tracing, and mandatory isolation, can be used to avoid future lockdowns to curb the transmission of the virus. Using French data, Gollier (2020) shows that infections in the population can be reduced as a significantly lower economic cost through mass testing compared to stringent lockdowns. Brotherhood et al. (2020) show how the number of deaths due to Covid-19, can be significantly reduced by testing the young, combined with a mandatory quarantine for everyone who tested positive. Berger et al. (2020) show that extensive random testing of asymptomatic people can mitigate the number of Covid infections. This in turn allows policymakers to relax quarantine restrictions, which reduces the negative implications for the economy. Finally, Guimarães (2021) shows that making antibody tests available to a larger share of the population, reduces social interactions, lowers the mortality from the disease in the population, and increases welfare.

In all of the aforementioned models with testing, diagnostic testing is introduced as an exogenous factor. Our model differentiates itself from the prior literature by considering the endogenous adoption of testing. We look at individual choices and derive what kind of individuals choose to purchase (possibly inaccurate) tests, and under what circumstances. For this we focus not on medical but economic choices, similar to Gersovitz and Hammer (2004).
3 MODEL ASSUMPTIONS

Consider an economy with many individuals, an infinite number of dates \( t \), and a discount factor \( \delta \) (and let \( \delta \equiv 1 - \delta \)).

At every date people face a choice between either “self-isolating” or “going out.” Self-isolation means avoiding contact with other people, such as staying (and possibly working) at home.\(^9\) We assume that anyone self-isolating cannot get infected, nor can they infect others. We normalize the date utility of self-isolation to zero. The date utility of going out is denoted by \( y \). Naturally the utility of going out can differ across individuals, so we assume that \( y \) has the distribution \( \Omega(y) \).

In our model infections occur without people realizing it (aka asymptomatic transmission). We use the following stylized disease progression. Suppose an individual who chooses to go out, gets infected at date \( t \). At date \( t + 1 \) this individual remains asymptomatic, that is, s/he does not know if s/he is infected (unless self-testing as considered in Section 5). If s/he goes out at \( t + 1 \), s/he may infect others. However, further exposure to contagious people does not change his/her infection status. At \( t + 2 \), infected people become ill and cannot go out. Being ill is associated with a disutility \( z \). We assume that all infected people recover at \( t + 3 \). They are no longer contagious and can go out again. However, we assume no immunity, so that people can get infected again. While these assumptions are not medically accurate, they help us to illustrate the underlying key trade-offs. Moreover, the assumptions that no one dies, and that there is no immunity, are necessary to guarantee steady states in our dynamic model. We denote the infection risk in the economy by \( \phi \) (and let \( \phi \equiv 1 - \phi \)). This is defined as the probability that someone uninfected going out, gets infected. The main objective of the model is to derive the equilibrium properties of \( \phi \).

Infecting others is an externality, but we allow people to partially internalize it through an altruistic utility function. For our base model we deliberately assume that the test has no medical role, so that anyone purely selfish would never self-test. In fact, the only reason people voluntarily test themselves in our model, is to protect others. We deliberately focus on the altruistic motive of self-testing, because this is where the interesting questions about externalities and economic efficiency lie. However, in Section 6.4 we show that our key insights continue to hold if we also allow for some selfish benefits of testing.

We model the altruistic motive as a social conscience that can be equally interpreted as a forward looking desire not to infect others, or a backward-looking feeling of remorse. To get the intuition, consider first the remorse logic. A person feels remorse if s/he realizes that s/he might have infected others (e.g., infecting a relative or a friend). Specifically, if someone discovers at date \( t + 1 \) that s/he is ill (which means s/he got infected at date \( t - 1 \)), and s/he went out at date \( t \), then the ex post remorse for going out is characterized by a disutility \( x \) at \( t + 1 \). Rationally anticipating this at date \( t \), where there is a probability \( \phi \) of being infectious, the expected disutility of going out is \( \phi \delta x \). Importantly, this disutility is increasing in \( \phi \), reflecting the higher risk of being infectious. We can also interpret \( \phi \delta x \) as a forward-looking desire not to infect others, that is, we can simply define \( \phi \delta x \) is the disutility of taking an action that risks infecting others.

\(^9\)In our context one can also interpret social distancing as a lesser form of self-isolation (Greenstone & Nigam, 2020).
4 | BENCHMARK MODEL WITHOUT TESTING

4.1 | Equilibrium choices of individuals

In this section, we identify the optimal choices of individuals in the economy. The benchmark model deliberately omits the possibility of self-testing, which we will introduce in Section 5.

In our benchmark model individuals can choose one of two possible strategies. The first strategy is to go out whenever possible—we call this the “GO” strategy. In this case individuals go out even if they know they might be contagious to others. The expected utility when choosing the “GO” strategy is given by

\[ U^{GO} = \phi[y + \delta(-x - z + \delta(y + \delta U^{GO}))] + \bar{\phi}[y + \delta U^{GO}], \]

which in steady-state implies

\[ U^{GO} = \frac{(1 + \phi\delta^2)y - \phi\delta(x + z)}{\bar{\delta} + \phi\delta(1 - \delta^2)}. \]

The second strategy is to stay home—we call this the self-isolation (“SI”) strategy. The expected utility for an individual choosing the “SI” strategy is normalized to \( U^{SI} = 0 \).

The next proposition identifies the optimal strategy for an individual, given the specific value of going out \( y \) for that individual.

**Proposition 1.** There exists a critical value \( Y_1 \) such that

(i) For all \( y < Y_1 \) individuals self-isolate (SI).
(ii) For all \( y \geq Y_1 \) individuals always go out (GO).

The critical value \( Y_1 \) is increasing in \( \phi, x, \) and \( z \).

Individuals with a high utility of going out will always try to do so; individuals with a low utility of going out will self-isolate. Individuals switch from GO to SI (higher \( Y_1 \)) with a higher chance to get infected (higher \( \phi \)), a stronger desire not to infect others (higher \( x \)), or a more severe illness (higher \( z \)).

4.2 | Equilibrium infection risk

We now derive the resulting equilibrium infection risk \( \phi \) in the economy. This requires the derivation of two equilibrium curves that we call the contagiousness curve (CO-curve) and the infection curve (IN-curve). The CO-curve measures how contagious people are that go out. Specifically, we define \( \mu \) as the average contagiousness of an individual going out. We will explain how this depends on the infection risk \( \phi \), so that the CO-curve is a function \( \mu_{CO}(\phi) \). The IN-curve measures how likely it is for an uninfected individual going out, to become infected. This depends on the contagiousness of the individuals s/he gets exposed to, so that the IN-curve is a function \( \phi_{IN}(\mu) \). The equilibrium is defined by the intersection of the CO-curve and the IN-curve.
We first derive the CO-curve. We assume that people cannot observe the history of others, and get exposed to a set of people that are each contagious with probability \( \mu \). In the appendix we show that in the steady-state, a fraction \( \varpi^{GO,0} = \frac{1}{1 + 2\phi} \) of GO people is not infected, a fraction \( \varpi^{GO,1} = \frac{\phi}{1 + 2\phi} \) is infected but asymptomatic, and a fraction \( \varpi^{ILL} = \frac{\phi}{1 + 2\phi} \) is ill and stays home. Moreover, we know from Proposition 1 that the number of GO people is \( 1 - \Omega(Y_t) \). The CO-curve is therefore given by

\[
\varpi^{CO}(\phi) = \frac{\varpi^{GO,1}[1 - \Omega(Y_t)]}{\varpi^{GO,0}[1 - \Omega(Y_t)] + \varpi^{GO,1}[1 - \Omega(Y_t)]} = \frac{\phi}{1 + \phi}. \quad (1)
\]

The numerator of (1) contains the number of people that choose to go out and are infected (but not yet ill). The denominator contains all people that choose to go out, both uninfected and infected. Note that the number of GO people, \( 1 - \Omega(Y_t) \), cancels out, so that the CO-curve only depends on the infection risk \( \phi \). This says that all GO people are equally contagious, irrespective of how many of them there are. This is because all GO people face the same fundamental risk of being asymptomatically infected. The CO-curve is concave increasing in \( \phi \), and satisfies \( \varpi^{CO}(0) = 0 \).

To derive the probability of getting infected (i.e., the IN-curve), we use a simple infection model. Specifically, if an individual goes out, s/he meets \( M \geq 1 \) other people, each of which may be contagious with probability \( \mu \). The measure \( M \) therefore reflects the contact level for people going out. There is also an imperfect shielding technology which for any encounter prevents infection with probability \( \lambda \) (e.g., wearing face mask or sanitizing hands). The probability of getting infected by any encounter is thus \( \lambda \mu (1 - \lambda) \). For simplicity we treat \( M \) and \( \lambda \) as exogenous for now, but show in Section 6.7 how \( M \) and \( \lambda \) can be endogenized without changing the key insights from our model.

The probability of not getting infected by any encounter at a given date is \( \lambda \mu [1 - (1 - \lambda)\mu]^M \).

Thus, the probability of getting infected at a given date is

\[
\phi = 1 - [1 - (1 - \lambda)\mu]^M. \quad (2)
\]

This equation defines the IN-curve \( \mu_{IN}(\phi) \). In the appendix, we derive an explicit expression for the IN-curve \( \mu_{IN}(\phi) \) by solving (2) for \( \mu \), and then show that \( \mu_{IN}(\phi) \) is decreasing in \( M \) and increasing in \( \lambda \). Moreover, \( \mu_{IN}(0) = 0 \), and \( \mu_{IN}(\phi) \) is always increasing in \( \phi \).

Figure 1 illustrates the CO-curve \( \varpi^{CO}(\phi) \) and the IN-curve \( \mu_{IN}(\phi) \). We can see from Figure 1 that there are two possible steady-state equilibria defined by the intersections of the two curves: One equilibrium with a zero infection risk \( \phi^* = 0 \), and one with a strictly positive infection risk \( \phi^* > 0 \). We can infer from (1) and (2) that the equilibrium with the strictly positive infection risk, \( \phi^* > 0 \), only depends on \( M \) and \( \lambda \) in our benchmark model.

The next proposition derives stability conditions for the two possible steady-state equilibria.

**Proposition 2.** There exists a threshold \( \bar{M} \) such that for \( M \leq \bar{M} \) the unique stable steady-state equilibrium exhibits a zero infection risk, that is, \( \phi^* = 0 \). For \( M > \bar{M} \) the unique stable steady-state equilibrium exhibits a strictly positive infection risk, that is, \( \phi^* > 0 \). The equilibrium steady-state infection risk \( \phi^* > 0 \) for \( M > \bar{M} \) is then increasing in \( M \) and decreasing in \( \lambda \).
Proposition 2 shows that if the contact level $M$ for people going out is low (i.e., $M \leq \bar{M}$), then $\phi^* = 0$ is the unique stable steady-state equilibrium. This is the case where, unlike in Figure 1, the $\mu_{CO}$ curve always lies below the $\mu_{IN}$ curve, only intersecting at $\phi = 0$. However, for higher levels of $M$ (i.e., $M > \bar{M}$), the $\mu_{CO}$ curve initially rises above the $\mu_{IN}$ curve, as shown in Figure 1. This creates two intersections, one at $\phi = 0$ and one for some $\phi^* > 0$. In this case, the $\phi = 0$ intersection is no longer a stable equilibrium. This is because a small flare-up in infections quickly raises the average contagiousness of the public pool, moving the economy onto a path to the $\phi^* > 0$ equilibrium. In this case, the only stable steady state has a positive infection risk $\phi^* > 0$. For the remainder of this paper we focus on the more interesting case with $M > \bar{M}$, that is, there is an ongoing pandemic in equilibrium ($\phi^* > 0$).

For the $\phi^* > 0$ equilibrium, higher contact levels (higher $M$), and less effective shielding technologies (lower $\lambda$), shift the IN-curve outward, thereby increasing the equilibrium steady-state infection risk $\phi^*$. Intuitively, more contact and less shielding make going out riskier because of higher transmission risk.

5 | MODEL WITH VOLUNTARY TESTING

5.1 | Imperfect testing assumptions

We now introduce the possibility that individuals use a test to verify whether they are infected. Here we are not interested in clinical tests for symptomatic people, but tests that asymptomatic people regularly self-administer to decide whether to go out or self-isolate.\footnote{In Section 6.5, we also consider clinical tests as an alternative to home-based tests.} Such tests may be inaccurate, generating both false positives (type I error) and false negatives (type II error).
We ask who would use such tests, what effect they have on the infection risk, and what the implications of type I/II errors are.

Let $c$ be the disutility of using a test, which depends on its price and the difficulty of administering it. Let $\alpha$ be the probability of a false-positive test result, and $\beta$ the probability of a false-negative test result. There are four possible outcomes:

(i) With $(1 - \phi)(1 - \alpha)$: the person is uninfected, test correctly negative.
(ii) With $(1 - \phi)\alpha$: the person is uninfected, test falsely positive.
(iii) With $\phi(1 - \beta)$: the person is infected, test correctly positive.
(iv) With $\phi\beta$: the person is infected, test falsely negative.

For parsimony we define $\alpha = 1 - \alpha$ and $\beta = 1 - \beta$. Moreover, we assume that the test is informative, that is, $\alpha, \beta \in [0, \frac{1}{2})$.

Testing is only useful if it generates information that affects decisions. A person who will always go out irrespective of the test result, would never purchase a test; similar for someone who always self-isolates. We can therefore focus on individuals who go out with a negative test result, but self-isolate with a positive test outcome. Moreover, these individuals would only use a test if they went out the date before (and are possibly infected).

The net effect of an imperfect test should be evaluated against the alternative of no test. For example, people who would otherwise have self-isolated, now choose to go out if they get a false negative test result. However, for people who would have gone out otherwise, a false negative test does actually not change their behavior. Figure 2 summarizes how and when an imperfect test affects behavior, and how this compares to the alternative of no testing.

For now we assume that individuals are fully rational and understand test limitations when making their decisions. We relax this assumption in Section 6.1.

### 5.2 Equilibrium with voluntary testing

Suppose an individual chooses to use a test when s/he went out the date before, and thus faces a risk of being infected. We call this the $TE$ strategy. The expected utility of using a test is then given by

\[
U^{TE} = -c + \phi \alpha (y + \delta U^{TE}) + \phi \alpha \delta (y + \delta U^{TE}) + \phi \beta (\delta (-z + \delta (y + \delta U^{TE}))) + \phi \beta (y + \delta (-x - z + \delta (y + \delta U^{TE}))).
\]  

The first term measures the disutility cost of the test. The remaining four terms correspond to the four possible outcomes as described above. Specifically, the second term is the expected utility

| Person is infected | Testing says | Decision | Without test would have self-isolated | Without test would have gone out |
|--------------------|--------------|----------|--------------------------------------|----------------------------------|
| Infected           | Correct positive | Self isolate | Same outcome as without test | Better outcome than without test |
| False negative     | Go out       | Worse outcome than without test | Same outcome as without test |
| Not infected       | False positive | Self isolate | Same outcome as without test | Worse outcome than without test |
| Correct negative   | Go out       | Better outcome than without test | Same outcome as without test |

**FIGURE 2** Effect of false and correct tests
when going out after a correct negative test. The third term concerns a false-positive test: the individual self-isolates although with the correct information would have gone out. This individual then realizes at the next date that s/he is not infected and therefore goes out, and tests again at the next date. The fourth term is the expected utility if the test is correctly positive. The individual then stays home and self-isolates. The fifth term concerns a false-negative test result: the individual goes out although with the correct information would have self-isolated.

Solving (3) for $U^{TE}$ we find that the expected utility of testing in the steady-state, is given by

$$U^{TE} = \frac{[\phi (1 - \alpha \delta) + \phi \delta^2]y - \phi [\beta (\delta x - y) + \delta z]}{1 - \delta [\phi (1 - \alpha \delta) + \phi \delta^2]} - c.$$ 

We can immediately see that the expected utility of testing is decreasing in its costs $c$. Moreover, it is decreasing in $\alpha$ and $\beta$.11 This implies that less accurate tests are less valuable to rational people.

In the appendix we show that there are always some individuals who choose the testing strategy $TE$ provided the disutility of being ill, $z$, is not too high (otherwise they would choose the self-isolation strategy $SI$, even if $c = 0$). Formally, this requires $z < z^*$, where $z^*$ is characterized in the Appendix. Since we are interested in how the availability of (imperfect) tests affect equilibrium outcomes, we focus on the case where at least some individuals choose the testing strategy $TE$ (i.e., $z < z^*$).

The next proposition identifies the optimal choices for individuals when self-testing is available.

**Proposition 3.** There exists a critical testing cost $c(\alpha, \beta)$ such that for $c \geq c(\alpha, \beta)$ no one does testing. For $c < c(\alpha, \beta)$ the optimal choices are as follows:

(i) For all $y < \hat{Y}_1$ individuals self-isolate ($SI$).
(ii) For all $\hat{Y}_1 \leq y < \hat{Y}_2$ individuals test ($TE$).
(iii) For all $y \geq \hat{Y}_2$ individuals go out ($GO$).

The critical value $\hat{Y}_1$ is increasing in $c$, $\alpha$, and $\beta$, and $\hat{Y}_2$ is decreasing in $c$, $\alpha$, and $\beta$.

Figure 3 illustrates the equilibrium choices for the benchmark model, and the model with testing, for different values of $y$.

The key insight from Proposition 3 is that testing is adopted along two margins. First, people with higher values of going out, $y$, trade-off testing against going out without a test. Testing is altruistic, in that these people would otherwise have gone out, but now with a positive test result self-isolate to avoid infecting others. Second, people with lower values of $y$ trade-off testing against self-isolation. A negative test result gives them the required assurance that they are not contagious. The cheaper tests become, the more people switch to testing, along both margins. Moreover, the more accurate the test (lower $\alpha$ and/or $\beta$), the more people switch to testing, too.12 Overall, testing adds an option to individuals, and therefore increases the

11 Specifically, $U^{TE}$ is decreasing in $\beta$ as long as $\delta x > y$. This condition is always satisfied for any individual that chooses testing. To see why, note that for $\delta x < y$ the remorse factor is sufficiently small, so that these individuals always go out, even if they are infected, and would never take a test. Consequently, all people who test satisfy the condition $\delta x > y$.

12 We also formally show in the Appendix (see Proof of Proposition 3) that $c(\alpha, \beta)$ is decreasing in both $\alpha$ and $\beta$. This implies that less accurate tests are only used in equilibrium if they involve a lower disutility $c$. 

HELLMANN AND THIELE | 11
utility of those who adopt it. The question remains what effect testing has on the equilibrium infection risk $\phi^*$.

In the appendix we rederive the CO-curve, now allowing individuals to also choose the testing strategy $TE$.\(^{13}\) The CO-curve is now given by

$$
\mu_{CO}(\phi) = \frac{\phi}{1 + 2\phi} [1 - \Omega(Y_2)] + \frac{\phi \beta}{1 + 2\phi + \beta} [\Omega(Y_2) - \Omega(Y_1)].
$$

An important intuition (and the reason why this function looks somewhat complex) is that in addition to the $GO$ types, the pool of people now going out in public includes the $TE$ types. Many of them tested negative correctly, and they help to reduce the average contagiousness of the public pool (and only enter in the denominator of the above expression). Those who had a false negative test, however, enter both the numerator and the denominator, indicating that they increase the average contagiousness of the public pool. The question is thus how the addition of both uninfected (correct negatives) and infected (false negatives) people going out affects the equilibrium infection risk $\phi^*$.

Proposition 4. Higher values of $c, \alpha,$ and $\beta$ shift the CO-curve upward. The equilibrium infection risk $\phi^*$ is thus increasing in $c, \alpha,$ and $\beta$.

Voluntary testing by private individuals has an important effect on the equilibrium infection risk. The more accurate and the cheaper tests are, the more people use them. This means that fewer of the people who go out are contagious, and thus fewer infections occur. This is true even if tests are imperfect, that is, if some people go out based on a false negative test result. The intuition is as follows. A pool of tested individuals who choose to go out, contains both uninfected people (who had correct negative test results) and infected people (who had false-negative test results). As long as testing remains informative (i.e., $\alpha, \beta < \frac{1}{2}$), the pool of tested people is less contagious than the pool of untested people. Testing, therefore, helps to bring down the average contagiousness of the people going out, that is, it shifts the CO-curve downward. All that is required for this downward shift to occur is that the tests are informative, so they do not need to be perfectly accurate.

\(^{13}\)See Proof of Proposition 4 in the appendix.
5.3 | Social welfare with voluntary testing

Unlike the typical SIR models, our model is based on utility maximization. Consequently, we can analyze social welfare by looking at the sum of utilities across all agents in the economy. The social welfare function is as follows:

\[
W = \int_0^{\bar{Y}_1} U^{SI}d\Omega(y) + \int_{\bar{Y}_1}^{\bar{Y}_2} U^{TE}d\Omega(y) + \int_{\bar{Y}_2}^{\infty} U^{GO}d\Omega(y).
\]

The next proposition analyzes the effect of (imperfect) tests on social welfare, and therefore provides the central finding of this paper.

**Proposition 5.** The social welfare function is decreasing in \(c, \alpha, \) and \(\beta\).

Proposition 5 says that social welfare is increasing as the price of tests comes down. Those individuals buying the tests clearly prefer lower prices. Moreover, as shown in Proposition 4, the risk of infection comes down with lower test prices, which benefits everyone else too. Maybe the most remarkable part of Proposition 5 is that this simple logic holds true irrespective of testing inaccuracies, that is, it holds for all values of \(\alpha\) and \(\beta\). The key intuition is that imperfect testing is better than no testing at all, as it helps to reduce the average contagiousness of the people going out. Obviously, higher accuracy is still highly desirable. Indeed, Proposition 5 finds that social welfare always increases when tests become more accurate (i.e., they have lower \(\alpha\) and \(\beta\)).

Proposition 5 implies that making tests available has a positive effect on welfare. This is because tests prevent some infected individuals from going out and infecting others, reducing the steady-state infection risk \(\phi^*\), and therefore the number of individuals getting ill. Even inaccurate tests, causing type I and type II errors, are better than no tests, as they still help to reduce the infection risk. Our analysis therefore provides two important implications for public policy. First, the government should provide home-based tests, even if they are not perfectly accurate. To encourage as many individuals as possible to use these tests, the government needs to ensure that the associated costs are sufficiently low (e.g., by subsidizing the tests). Second, the government could encourage and support the development of more accurate tests, which helps to curb the infection risk in the economy.

Finally, an interesting question is whether home-based testing can completely eradicate the disease? In principle, this would be possible if all people always took the test before going out, and the tests had no false negatives (i.e., \(\beta = 0\)). However, these two conditions are unlikely to be satisfied with voluntary home-based testing. Our model shows that there can always be some people with high values of \(y\) who would prefer to go out even with a positive test result. This, and the fact that home-based tests are likely to be imperfect (\(\beta > 0\)), suggests that home-based testing can reduce the equilibrium infection risk, but is unlikely to eradicate the disease entirely.

6 | EXTENSIONS

6.1 | Naïve individuals

One of the central concerns is that inaccurate tests can mislead the public. The argument is that some people do not understand the inaccuracy of test results and believe them to be fully
reliable. In this section, we provide a simple model extension where a fraction $\xi$ of the population is naïve and believes tests to be perfect. Such naïve people do not understand type I/II errors and thus put too much faith into test results. We then ask how this affects individual choices and the equilibrium infection risk.

Figure 4 shows the differences between the equilibrium choices for our main model (with fully rational individuals) and our model extension where a fraction $\xi$ of the population is naïve. We provide the formal proofs in the appendix. The key insight is that two types of naïve people buy the test, even though they would have not done so if they fully understood its accuracy: (i) those who would have self-isolated (lower $y$, on the left side in Figure 4), and (ii) those who would have gone out anyway (higher $y$, on the right side in Figure 4). The first types are precisely those that the medical community typically worries about, where a false negative test encourages them to go out when they should not. However, our analysis shows that there is also a second type that would otherwise have gone out. Their naïveté makes them buy the test, even though they would not have done so if they fully understood its accuracy. Whenever they get a positive test result (correct or false), they self-isolate, something that everyone would welcome.

Ceteris paribus it is always better to be rational than naïve from an individual utility perspective. The interesting question is how the presence of naïve people affects the equilibrium infection risk.

**Proposition 6.** A higher fraction of naïve individuals (higher $\xi$) shifts the CO-curve downward. The equilibrium infection risk $\phi^*$ is therefore decreasing $\xi$.

This surprising result says that if more people are naïve, the equilibrium infection risk goes down. We show in the Appendix that this result holds for all $\alpha, \beta \in (0, \frac{1}{2})$ and $\xi > 0$, that is, as long as tests have some informational value, and some individuals are naïve. The key intuition is that naïve people overestimate the power of the test. They use tests even though for some of them their rational alter-ego would not do so. This increases the number of people testing themselves, which helps to reduce contagion. This is because testing, as long as tests are informative (i.e., $\alpha, \beta < \frac{1}{2}$), keeps some infected people at home (correct positives), even if some infected people still go out (false negatives). Proposition 6 formally shows that the additional naïve testers who go out after a negative test result, are on average less contagious than the other untested people who also go out. These naïve testers, therefore, help to reduce the average contagiousness of the people going out, as represented by a downward shift of the CO-curve. Since the IN-curve is not affected, this results in a reduction of the equilibrium infection risk $\phi^*$. 

**FIGURE 4**  Comparison of equilibrium choices with Naïve customers
6.2 Required tests

Our model assumes that testing affects the decision to go out, but not the utility of doing so. In reality, testing may change what people do, and thus may also affect the utility of going out. For example, people may do certain things only once they had a negative test result, such as visiting a fragile relative. Access to certain events (e.g., music concerts) might also require having a negative test result.

Formally, suppose that the value of going out without a negative test result is now \( (1 - \tau)y \). A higher \( \tau \) represents greater limitations to people without negative tests. The value of going out with a negative test result is still \( y \). The next proposition shows how the requirement of a negative test result affects the equilibrium outcome.

**Proposition 7.** More individuals choose testing when \( \tau \) increases. This reduces the equilibrium infection risk. Formally, \( Y_\tau(\tau) \) is increasing, and \( \phi^*(\tau) \) is decreasing in \( \tau \).

Facing more limitations convinces more people to test. This stops more infected people from going out, leading to a lower equilibrium infection risk.

6.3 Total infection rate

Our analysis focuses on the equilibrium infection risk \( \phi^* \), which measures the likelihood that someone who is going out gets infected. Another measure, commonly used by health officials, is the total infection rate of the entire population (which we denote by \( \Phi \)). This measure also includes (in the denominator) individuals who self-isolate (SI). Cheaper testing allows some GO people to get a test before going out, but it also encourages some SI people to get a test and go out if the test results are negative. While our model generates an unambiguous prediction about the infection risk (\( \phi^* \)), we show in the appendix that the effect of cheaper testing on the total infection rate \( \Phi \) is ambiguous. There are the following three main effects: First, more testing reduces the equilibrium infection risk \( \phi^* \). Second, more testing reduces the number of infected people in the public—thanks to a correct positive test, more infected people stay at home instead of going out. And third, more testing increases the number of infected people who go out with a false negative test, who would otherwise have self-isolated. The net effect of these three effects is ambiguous and depends on the distribution of \( y \). However, one special case is worth mentioning. If there are no false negatives (i.e., \( \beta = 0 \)) then the third effect is zero; and using continuity, if there are sufficiently few false negatives (i.e., \( \beta \) is sufficiently close to 0), then the third effect remains sufficiently small so not to overpower the first two effects. We then get the unambiguous result that more testing reduces the total infection rate \( \Phi \).

One reason why the total infection rate plays an important role in practice is that it serves as an indicator of capacity constraints faced by the medical system. The higher the total infection rate, the more likely are some congestions at key medical intervention points, especially hospitals. Let us take this last argument one step further and ask how such congestion externalities might affect equilibrium behaviours. Consider a simple parameterization where the disutility of being ill, \( z \), is an increasing function of the total infection rate \( \Phi \). Specifically, we assume that \( z(\tau \Phi) \) with \( z' > 0 \), where \( \tau \) measures the importance of congestion externalities.
We first examine how a congestion externality affects the equilibrium without testing. We show in the Appendix that $Y_1$ is an increasing function of $\tau$, suggesting that more people stay home ($SI$) instead of going out ($GO$) when congestion matters more. For the model with testing, we show that both $\bar{Y}_1$ and $\bar{Y}_2$ are increasing in $\tau$, suggesting that some people switch from testing ($TE$) to self-isolation ($SI$), and others from going out ($GO$) to testing ($TE$). These results imply that the private demand for asymptomatic testing adapts in response to congestion externalities.

The presence of congestion externalities can also affect the social welfare properties of home-based testing. Consider first the case where $\beta$ is sufficiently small, so that the total infection rate always decreases with more home-based testing (i.e., $\Phi^*$ is increasing in $c$). In this case, the benefit of home-based testing is reinforced by congestion externalities: home-based testing not only reduces infection rates, but also helps to alleviate congestion. For larger values of $\beta$, however, this finding can be reversed. In particular, it is possible to construct examples where more home-based testing can lead to more infected people going out because of false-negative test results. This could increase the total number of infected people, worsen the congestion externality, and reduce social welfare. The more general point is that in the presence of congestion externalities, the total infection rate can have a more direct impact on social welfare.

6.4 Medical benefit of testing

For our base model we deliberately assumed that self-testing only provides information, which helps altruistic people to assess the risk of infecting others, but does not deliver any direct personal benefits. In practice self-testing may also have direct medical benefits, for example, if an early diagnosis helps to abate illness progression. Such direct medical benefits would increase the utility of testing and therefore increase the range of people who voluntary self-test. In this section, we briefly show that adding such medical benefits does not change the key message of the model.

To allow for a medical benefit of testing, suppose that a correct test result changes the disutility of being infected to $z - \kappa$, where $\kappa$ is the medical benefit of a correct positive test. The expected utility of testing then changes to

$$U^{TE} = -c + \phi \bar{\alpha} (y + \delta U^{TE}) + \phi \alpha \delta (y + \delta U^{TE}) + \phi \bar{\beta} (\delta (-(z - \kappa) + \delta (y + \delta U^{TE}))) + \phi \beta (y + \delta (-x - z + \delta (y + \delta U^{TE}))).$$

Solving for $U^{TE}$ we get the new expected utility of testing in the steady state:

$$U^{TE} = \frac{[\bar{\phi} (1 - \alpha \delta) + \phi \delta^2]y - \phi [\beta (\delta x - y) + \delta z - \delta \kappa \bar{\beta}]}{1 - \delta [\bar{\phi} (1 - \alpha \delta) + \phi \delta^2]} - c.$$

**Proposition 8.** A higher medical benefit of a correct positive test, $\kappa$, leads to more testing in equilibrium, that is, $\bar{Y}_1$ is decreasing in $\kappa$, and $\bar{Y}_2$ is increasing in $\kappa$.

A medical benefit ($\kappa > 0$) makes testing ($TE$) relatively more attractive compared to self-isolating ($SI$) and going out ($GO$). This implies that the range of $y$, where individuals choose
testing, increases. Other than that, the main insights of the base model remain intact in the presence of a medical benefit of testing.

6.5 | Clinical tests

In our main model, we analyzed how imperfect home-based testing, which may produce false-positive and false-negative test results, affects the behaviour of individuals, and therefore the infection risk in the economy. An alternative to home-based testing is clinical testing, administered by healthcare workers. An important argument for clinical testing is that the test results are more accurate. Our model implicitly assumes that clinical testing occurs once people have developed symptoms, but not before. In this section, we briefly show how also using clinical tests at the asymptomatic stage, as an alternative to home-based tests, affects the equilibrium.

Suppose that individuals now have the additional option of clinical testing before having any symptoms, at a cost \( \chi \). The cost parameter \( \chi \) also reflects the inconvenience for individuals associated with clinical testing, including driving to public test centers and waiting to get tested. For simplicity, we assume that clinical tests are accurate, that is, they do not produce false-positive or false-negative test results (i.e., \( \alpha = \beta = 0 \)).

The expected utility when using a clinical test is

\[
U^{CT} = \frac{[\phi + \phi \delta^2]y - \phi \delta z - \chi}{1 - \delta[\phi + \phi \delta^2]}.
\]

The question is whether and when asymptomatic people prefer perfect clinical over imperfect home-based testing? The next proposition identifies the optimal strategies for individuals when accurate clinical tests are available.

**Proposition 9.** For clinical testing there exist two cost thresholds \( \tilde{\chi} \) and \( \check{\chi} \), with \( \tilde{\chi} < \check{\chi} \), such that no one uses clinical tests for \( \chi \geq \check{\chi} \). For \( \tilde{\chi} \leq \chi < \check{\chi} \) the optimal choices are as follows:

(i) For all \( y < \check{Y}_1 \) individuals self-isolate (SI).
(ii) For all \( \check{Y}_1 \leq y < \check{Y}' \) individuals use home-based tests (TE).
(iii) For all \( \check{Y}' \leq y < \check{Y}_2 \) individuals choose clinical tests (CT).
(iv) For all \( y \geq \check{Y}_2 \) individuals go out (GO).

For \( \chi < \tilde{\chi} \) the optimal choices are as follows:

(i) For all \( y < \check{Y}_1 \) individuals self-isolate (SI).
(ii) For all \( \check{Y}_1 \leq y < \check{Y}_2 \) individuals choose clinical tests (CT).
(iii) For all \( y \geq \check{Y}_2 \) individuals go out (GO).

The critical values \( \check{Y}_1 \) and \( \check{Y}' \) are increasing in \( \chi \), and \( \check{Y}_2 \) is decreasing in \( \chi \).

\(^{14}\)We obtain the expression for \( U^{CT} \) by setting \( \alpha = \beta = 0 \) in \( U^{TE} \), and replacing \( c \) by \( \chi \).
Figure 5 illustrates the key insights from Proposition 9 for the case where the cost of clinical testing is sufficiently low so that some individuals choose clinical testing, while others choose home-based testing ($\overline{\chi} \leq \chi < \overline{\chi}$). The interesting question then is who in the population switches to clinical testing when it becomes available at a reasonable cost? We can see in Figure 5 that there are two types that switch to clinical testing first: some people who would have otherwise chosen home-based testing, and some who would have gone out without testing. Further lowering the cost $\chi$ encourages more individuals to switch from home-based testing to clinical testing, and more GO people to use clinical tests. For a sufficiently low cost ($\chi < \overline{\chi}$), home-based testing is completely replaced by clinical testing (and some SI people now switch to clinical testing).

The main benefit of clinical testing is its accuracy, that is, the fact that it reliably filters out infected asymptomatic people that may have otherwise gone out and infected others. Having easily accessible and affordable clinical tests alongside home-based tests, can thus further help to reduce the equilibrium infection risk in the economy.\footnote{One may also ask how the equilibrium with clinical testing would change if some individuals are naïve as in Section 6.1. Note that naïve individuals would treat the results of home-based tests and clinical tests the same, as they believe that home-based tests are fully accurate just like clinical tests. They would therefore always choose the type of test (home-based or clinical) that has the lower cost. For $\chi \geq \overline{\chi}$ they choose home-based tests, and the main insights about equilibrium choices from Section 6.1 remain intact. However, there may be some inefficiencies now, namely when naïve individuals prefer a home-based test that is slightly cheaper but could be much less accurate. In the extreme case of slightly cheaper but highly inaccurate home-based tests, combined with a large fraction of naïve individuals, it may even become optimal to only allow clinical and no home-based testing. Moreover, educating naïve individuals about the lack of accuracy of such tests would also become desirable in that case. For $\chi < \overline{\chi}$ all naïve individuals choose clinical tests. Rational individuals then also prefer clinical tests, and all individuals correctly consider them as perfectly accurate. Thus, for $\chi < \overline{\chi}$, the presence of naïve individuals does not affect the equilibrium outcome at all.}

### 6.6 Partial isolation

So far we considered three possible strategies for individuals: (i) go out indiscriminately (GO), (ii) use a test and only go out if test is negative (TE), and (iii) always self-isolate (SI). In this section, we consider an additional strategy that we call the partial isolation (PI) strategy. This combines elements of the GO and SI strategies. Specifically, after going out...
at some date $t$, an individual stays at home at $t + 1$ to see whether s/he is infected. If s/he is still healthy at $t + 2$, s/he is certain not to be contagious and goes out again without fear of infecting others.

The expected utility for the partial isolation strategy ($PI$) is given by

$$U^{PI} = \phi \delta [-z + \delta (y + \delta U^{PI})] + \delta (y + \delta U^{PI}),$$

which in steady-state implies

$$U^{PI} = \frac{\delta [(1 - \phi \delta) y - \phi z]}{1 - (1 - \phi \delta) \delta^2}.$$  

The next proposition characterizes the optimal choices of individuals when allowing them to also choose partial isolation.

**Proposition 10.** There exists a threshold $z'$, with $z' < z^*$, such that partial isolation ($PI$) is chosen by some individuals when $z \leq z'$. The optimal choices are then as follows:

(i) For all $y < \hat{Y}_1'$ individuals completely self-isolate ($SI$).
(ii) For all $\hat{Y}_1' \leq y < \hat{Y}_2''$ individuals partially self-isolate ($PI$).
(iii) For all $\hat{Y}_2'' \leq y < \hat{Y}_2$ individuals test ($TE$).
(iv) For all $y \geq \hat{Y}_2$ individuals go out ($GO$).

The critical value $\hat{Y}_1'$ does not depend on $c$, $\alpha$, and $\beta$, while $\hat{Y}_2''$ is increasing in $c$, $\alpha$, and $\beta$.

Proposition 10 shows that partial isolation would only be an equilibrium choice for some individuals if the illness is sufficiently mild (low $z$). In this case, we get a new region in Figure 3 ($\hat{Y}_1' \leq y < \hat{Y}_2''$) where some individuals choose partial self-isolation. However, everything else remains qualitatively the same.16

### 6.7 Endogenous encounters and shielding

To derive the infection curve we assumed a fixed number of encounters $M$, and an exogenous shielding technology $\lambda$. In reality, we might expect these to depend on the infection risk. For instance, people will try to reduce the number of meetings when the infection risk goes up, or they may avoid some of the most crowded places. Similarly, people might shield themselves more if the infection risk rises, such as (voluntarily) wearing masks, sanitizing hands more often, and keeping a safe distance.

The next proposition shows how the equilibrium infection risk $\phi^*$ changes when $M$ or $\lambda$ are endogenous.

---

16One question is whether allowing for partial isolation changes our key insights concerning the equilibrium infection risk $\phi^*$ (see Proposition 4). With partial isolation, the model becomes much more cumbersome because the public pool (and thus the contagiousness curve) involves additional types. Still, in the appendix, we show that our key insights from Proposition 4 continue to hold, provided a mild sufficient condition of $\beta$ being not too large, is satisfied.
Proposition 11. **An endogenous number of encounters, M, or an endogenous shielding technology, λ, reduces the equilibrium infection risk ϕ*.**

In the appendix, we show that the main effect of making M and λ endogenous, is that the IN-curve becomes steeper. This in turn reduces ϕ*, but has no other effect on the model.

7 | **DISCUSSION**

We derive our key results using an analytically tractable steady-state model. This allows us to analyze the economic decisions of private individuals, and how they get aggregated in equilibrium. This naturally requires some limiting assumptions, which we briefly discuss here. To begin with, our steady-state model cannot capture the dynamics of a pandemic over time, such as its peaks and troughs. However, our focus concerns scenarios where pandemics become an on-going economic threat. Put differently, our objective here is to analyze the economic drivers of voluntary self-testing in a simple transparent setting, without the additional complexities of individuals forecasting how pandemic threat levels will evolve dynamically.

Our model makes some simplifying assumptions about the medical progression of the disease. Specifically, in our model no one ever dies and recovered individuals do not develop immunity from the disease. Our model, therefore, resembles the so-called SIS disease model, which builds the foundation for several economic studies of disease transmissions.17 Adding death and/or immunity to our model would negate any steady-state analysis, because the density of people in the various regions of Figure 3 would change over time. However, as long as the immunity and death rates remain low, these density changes are slow-moving, and have minimal impact on short-run equilibrium outcomes.

Vaccination is not part of our model. Historically, some viruses such as SARS and AIDS never had any approved vaccines. In the case of Covid-19, vaccines clearly help to contain the virus. However, vaccine hesitancy and imperfect vaccine efficacy still mean that infection risks persist, and that testing remains a relevant tool for containment.18

Our model naturally makes simplifying assumptions about underlying economic behaviours. For analytical tractability, we assume common knowledge and consider only one dimension of heterogeneity, namely the utility of going out (y). This is an important dimension because different people have different opportunity costs of self-isolating (e.g., some people can easily work from home, others risk losing their economic livelihood). However, there could also be heterogeneity in other model dimensions: different people could have different levels of altruism (x), fragility (z), or difficulties self-administering a test (c).19 Future research could enrich our model along several of these dimensions.

---

17See, for example, Goldman and Lightwood (2002), Gersovitz and Hammer (2004), Sims et al. (2016), and Goenka and Liu (2020).

18As of December 21, 2021, in the United States only 61% of the population is fully vaccinated, and in the United Kingdom 69%; see https://ourworldindata.org/covid-vaccinations.

19See, for example, Gollier (2020) and Acemoglu et al. (2021), for models where different age groups have different costs associated with the disease (i.e., where z differs across individuals).
8  |  CONCLUSION

This paper provides a theoretical framework for assessing the economic role of voluntary self-testing in the context of an on-going pandemic. Our theory generates three broad insights. First, the model uncovers two margins along which private individuals opt into testing. There are those people who, in the absence of testing, would self-isolate, and those who would always go out. Second, the price and ease of use of a test play an important role for the equilibrium infection risk. A positive test result convinces high-risk people to self-isolate. Moreover, a negative test result enables low-risk people to go out, which reduces the average contagiousness in public places. Third, the model sheds light on the role of diagnostic accuracy, showing that imperfect tests still help to reduce the equilibrium infection risk. A surprising result is that the presence of naïve people actually reduces the equilibrium infection risk.

ACKNOWLEDGMENTS

We would like to thank Rabah Amir (the editor), an Associate Editor, two anonymous referees, Peter Drobac, and Joshua Gans for their many helpful comments and suggestions. We are not epidemiologists, and view this paper as a contribution to health economics and management.

ORCID

Veikko Thiele  http://orcid.org/0000-0002-1316-3322

REFERENCES

Acemoglu, D., Chernozhukov, V., Werning, I., & Whinston, M. D. (2021). Optimal targeted lockdowns in a multigroup SIR model. American Economic Review: Insights, 3(4), 487–502.
Agrawal, A., Gans, J., Goldfarb, A., & Lederman, M. (2020). The CEO's guide to safely reopening the workplace. MIT Technology Review, May 28th.
d'Albis, H., & Augeraud-Véron, E. (2020). Optimal prevention and elimination of infectious diseases. Journal of Mathematical Economics, 93(2), 102487.
Alfaro, L., Faia, E., Lamersdorf, N., & Saidi, F. (2020). Social interactions in pandemics: fear, altruism, and reciprocity (NBER Working Paper 27134).
Althouse, B. M., Bergstrom, T. C., & Bergstrom, C. T. (2010). A public choice framework for controlling transmissible and evolving diseases. Proceedings of the National Academy of Sciences of the United States of America, 107, 1696–1701.
Baril-Tremblay, D., Marlats, C., & Ménager, L. (2021). Self-isolation. Journal of Mathematical Economics, 93, 102483.
Berger, D. W., Herkenhoff, K. F., & Mongey, S. (2020). An SEIR infectious disease model with testing and conditional quarantine. Mimeo, Duke University.
Bethune, Z. A., & Korinek, A. (2020). Covid-19 infection externalities: Trading off lives vs. livelihoods (NBER Working Paper 27009).
Bhattacharya, J., Chakraborty, S., & Yu, X. (2021). A rational-choice model of covid-19 transmission with endogenous quarantining and two-sided prevention. Journal of Mathematical Economics, 93, 102492.
Boozer, M. A., & Philipson, T. J. (2000). The impact of public testing for human immunodeficiency virus. Journal of Human Resources, 35(3), 419–446.
Bosi, S., Camacho, C., & Desmarchelier, D. (2021). Optimal lockdown in altruistic economies. Journal of Mathematical Economics, 93, 102488.
Bosi, S., & Desmarchelier, D. (2018). Pollution and infectious diseases. International Journal of Economic Theory, 14(4), 351–372.
Boucekkine, R., Carvajal, A., Chakraborty, S., & Goenka, A. (2021). The economics of epidemics and contagious diseases: An introduction. Journal of Mathematical Economics, 93, 102498.
Brauer, F., & Castillo-Chavez, C. (2012). Mathematical models in population biology and epidemiology, vol. 2 of Texts in Applied Mathematics. Springer.

Brotherhood, L., Kircher, P., Santos, C., & Tertilt, M. (2020). An economic model of the Covid-19 pandemic with young and old agents: behavior, testing and policies. University of Bonn and University of Mannheim, Discussion Paper No. 175.

Carroll, A. E. (2020). We made a mistake with masks. Now it could be tests. The New York Times, July 28, 2020. https://www.nytimes.com/2020/07/28/opinion/coronavirus-testing-antigen-pooling.html

Chang, R., & Velasco, A. (2020). Economic policy incentives to preserve lives and livelihoods (NBER Working Paper 27020).

Chowell, G., Fenimore, P., Castillo-Garsow, M., & Castillo-Chavez, C. (2003). SARS outbreaks in Ontario, Hong Kong and Singapore: The role of diagnosis and isolation as a control mechanism. Journal of Theoretical Biology, 224(1), 1–8.

Eichenbaum, M. S., Rebelo, S., & Trabandt, M. (2020). The macroeconomics of testing and quarantining (NBER Working Paper No. 27104).

Farboodi, M., Jarosch, G., & Shimer, R. (2021). Internal and external effects of social distancing in a pandemic. Journal of Economic Theory, 196, 105293.

Federico, S., & Ferrari, G. (2021). Taming the spread of an epidemic by lockdown policies. Journal of Mathematical Economics, 93, 102453.

Gaffeo, E. (2003). The economics of HIV/AIDS: A survey. Development Policy Review, 21(1), 27–49.

Gallic, E., Lubrano, M., & Michel, P. (2022). Optimal lockdowns for COVID-19 pandemics: Analyzing the efficiency of sanitary policies in Europe. Journal of Public Economic Theory (forthcoming).

Gans, J. S. (2020a). Economics in the age of COVID-19. MIT Press.

Gans, J. S. (2020b). The economic consequences of $\hat{R} = 1$: Towards a workable behavioural epidemiological model of pandemics (NBER Working Paper 27632).

Garibaldi, P., Moen, E. R., & Pissarides, C. (2020). Modelling contacts and transitions in the SIR epidemics model. Covid Economics 5, 1–20.

Gersovitz, M., & Hammer, J. S. (2004). The economical control of infectious diseases. The Economic Journal 114(492), 1–27.

Glick, P. (2005). Scaling up HIV voluntary counseling and testing in Africa. Evaluation Review, 29(4), 331–357.

Godlonton, S., & Thornton, R. L. (2013). Learning from others’ HIV testing: Updating beliefs and responding to risk. American Economic Review, 103(3), 439–444.

Goenka, A., & Liu, L. (2012). Infectious diseases and endogenous fluctuations. Economic Theory, 50(1), 125–149.

Goenka, A., & Liu, L. (2020). Infectious diseases, human capital and economic growth. Economic Theory, 70, 1–47.

Goenka, A., Liu, L., & Nguyen, M.-H. (2014). Infectious diseases and economic growth. Journal of Mathematical Economics, 50, 34–53.

Goldman, S. M., & Lightwood, J. (2002). Cost optimization in the SIS model of infectious disease with treatment. Topics in Economic Analysis & Policy, 2(1), 1–24.

Gollier, C. (2020). Cost-benefit analysis of age-specific deconfinement strategies. Journal of Public Economic Theory, 22(6), 1746–1771.

Gonzalez-Eiras, M., & Niepelt, D. (2020). Optimally controlling an epidemic (CESifo Working Paper No. 8770).

Goodkin-Gold, M., Kremer, M., Snyder, C. M., & Williams, H. L. (2002). Optimal vaccine subsidies for endemic and epidemic diseases (NBER Working Paper 28085).

Gori, L., Manfredi, P., Marsiglio, S., & Sodini, M. (2022). COVID-19 epidemic and mitigation policies: Positive and normative analyses in a neoclassical growth model. Journal of Public Economic Theory (forthcoming).

Greenstone, M., & Nigam, V. (2020). Does social distancing matter? Covid Economics, 7, 1–22.

Grimm, V., Mengel, F., & Schmidt, M. (2021). Extensions of the SEIR model for the analysis of tailored social distancing and tracing approaches to cope with COVID-19. Scientific Reports, 11, 4214.

Guimarães, L. (2021). Antibody tests: They are more important than we thought. Journal of Mathematical Economics, 93, 102485.
Johansson, M. A., Quandelacy, T. M., Kada, S., VenkataPrasad, P., Steele, M., Brooks, J. T., Slayton, R. B., Biggerstaff, M., & Butler, J. C. (2021). SARS-CoV-2 transmission from people without COVID-19 symptoms. *JAMA Netw Open*, 4(1), e2035057.

Meyer, R., & Madrigal, A. C. (2020). How to test every American for COVID-19, every day—The plan that could give us our lives back. The Atlantic, August 14, 2020.

Morin, B. R., Kinzig, A. P., Levin, S. A., & Perrings, C. A. (2018). Economic incentives in the socially optimal management of infectious disease: When $R_0$ is not enough. *EcoHealth*, 15, 274–289.

Rachel, L. (2020). *An analytical model of covid-19 lockdowns*. Mimeo, London School of Economics and Political Science.

Rothert, J. (2022). Optimal federal transfers during uncoordinated response to a pandemic. *Journal of Public Economic Theory* (forthcoming).

Sims, C., Finnoff, D., & O’Regan, S. M. (2016). Public control of rational and unpredictable epidemics. *Journal of Economic Behavior & Organization*, 32, 161–176.

Tang, L., Zhou, Y., Wang, L., Purkayastha, S., Zhang, L., He, J., Wang, F., & Song, P. X.-K. (2020). A review of multi-compartment infectious disease models. *International Statistical Review*, 88(2), 462–513.

Thornton, R. L. (2008). The demand for, and impact of, learning HIV status. *American Economic Review*, 98(5), 1829–1863.

Toxvaerd, F. (2020). *Equilibrium social distancing* (Cambridge-INET Working Paper Series No: 2020/08).

vonThadden, E.-L. (2020). A simple, non-recursive model of the spread of Covid-19 with applications to policy. *Covid Economics*, 10, 24–43.

---

**How to cite this article**: Hellmann, T., & Thiele, V. (2022). A theory of voluntary testing and self-isolation in an ongoing pandemic. *Journal of Public Economic Theory*, 1–39. https://doi.org/10.1111/jpet.12584

**APPENDIX A**

**Proof of Proposition 1.** Using the steady-state utilities we find that $U^{SI} > U^{GO}$ when $y < Y_1 \equiv \phi \delta (x + z)/(1 + \phi \delta^2)$. We can see that $\frac{dY_1}{d\phi}, \frac{dY_1}{dx} > 0$. Moreover,

$$
\frac{dY_1}{d\phi} = \frac{\delta(x + z)(1 + \phi \delta^2) - \phi \delta(x + z)\delta^2}{[1 + \phi \delta^2]^2} = \frac{\delta(x + z)}{[1 + \phi \delta^2]^2} > 0. \quad \square
$$

**Derivation of Contagiousness Curve (CO).** We know from Proposition 1 that for $y < Y_1$ individuals choose SI, and therefore never go out. For $y \geq Y_1$ individuals choose GO. Let $\omega_{t}^{GO0}$ denote the fraction of these individuals at time $t$ that is not infected and goes out, $\omega_{t}^{GO1}$ the fraction that is infected (but asymptomatic) and goes out, and $\omega_{t}^{ILL}$ the fraction that is ill and stays home. By definition, $\omega_{t}^{GO0} + \omega_{t}^{GO1} + \omega_{t}^{ILL} = 1$. Moreover, everyone who is ill in $t$ was asymptomatic in $t - 1$: $\omega_{t}^{ILL} = \omega_{t-1}^{GO1}$. The portion $\phi$ of people that were uninfected in $t - 1$ and went out, are infected (but asymptomatic) in $t$: $\omega_{t}^{GO1} = \phi \omega_{t-1}^{GO0}$. In the steady state we have $\omega^{k} \equiv \omega_{t}^{GO0} = \omega_{t-1}^{GO0} = \omega_{t-k}^{GO0} = \ldots, k = \{GO0, GO1, ILL\}$. Consequently, the equilibrium is defined by (i) $\omega^{GO0} + \omega^{GO1} + \omega^{ILL} = 1$, (ii) $\omega^{ILL} = \omega^{GO1}$, and (iii) $\omega^{GO1} = \phi \omega^{GO0}$. Using (ii)
in (i) we get \( \varpi^{GO0} = 1 - 2\varpi^{GO1} \). Using this in (iii) yields \( \varpi^{GO1} = \frac{\phi}{1 + 2\phi} \). Consequently, \( \varpi^{GO0} = \frac{1}{1 + 2\phi} \).

Using the expressions for \( \varpi^{GO0} \) and \( \varpi^{GO1} \), we get

\[
\mu_{CO}(\phi) = \frac{\varpi^{GO1}[1 - \Omega(Y_1)]}{\varpi^{GO0}[1 - \Omega(Y_1)] + \varpi^{GO1}[1 - \Omega(Y_1)]} = \frac{\frac{\varpi^{GO1}}{1 + 2\phi}[1 - \Omega(Y_1)]}{\frac{\varpi^{GO0}}{1 + 2\phi}[1 - \Omega(Y_1)]} = \frac{\phi}{1 + \phi}.
\]

Note that \( \mu_{CO}(0) = 0 \). Moreover,

\[
\frac{d\mu_{CO}(\phi)}{d\phi} = \frac{1}{[1 + \phi]^2} > 0.
\]

We can then immediately see that \( \frac{d^2\mu_{CO}(\phi)}{d\phi^2} < 0 \).

**Proof of Proposition 2.** Solving (2) for \( \mu \) we get

\[
\mu_{IN}(\phi) = \frac{1}{1 - \lambda} \left[ 1 - (1 - \phi)^\lambda \right].
\]

Note that \( \mu_{IN}(0) = 0, \mu_{IN}(1 - \lambda^M) = 1 \), and

\[
\frac{d\mu_{IN}(\phi)}{d\phi} = \frac{1}{1 - \lambda} \frac{1}{M} (1 - \phi)^{1/M - 1} > 0.
\]

Thus, \( \mu_{IN}(1 - \lambda^M) = 1 > \phi > \mu_{CO}(\phi) \). Moreover, we have \( \mu_{IN}(\phi) \geq \mu_{CO}(\phi) \) at \( \phi = 0 \) when

\[
\left. \frac{d\mu_{IN}(\phi)}{d\phi} \right|_{\phi=0} \geq \left. \frac{d\mu_{CO}(\phi)}{d\phi} \right|_{\phi=0}.
\]

This condition is equivalent to

\[
M \leq \tilde{M} \equiv \frac{1}{1 - \lambda}.
\]

Thus, for \( M \leq \tilde{M} \) we have \( \mu_{IN}(\phi) > \mu_{CO}(\phi) \) for all \( \phi > 0 \). Consequently, \( \mu_{IN}(\phi) \) and \( \mu_{CO}(\phi) \) only intersect at \( \phi = 0 \), that is, \( \mu_{IN}(0) = \mu_{CO}(0) = 0 \). This implies that for \( M \leq \tilde{M} \) the unique stable steady-state equilibrium is \( \phi^* = 0 \). For \( M > \tilde{M} \), \( \mu_{IN}(\phi) \) and \( \mu_{CO}(\phi) \) intersect at \( \phi = 0 \) and at some \( \phi^* > 0 \). Note that then \( \left. \frac{d\mu_{IN}(\phi)}{d\phi} \right|_{\phi=0} < \left. \frac{d\mu_{CO}(\phi)}{d\phi} \right|_{\phi=0} \), so that \( \phi^* = 0 \) is not a stable steady-state equilibrium, as a higher contagiousness forces the infection rate to increase over time, until \( \mu_{IN}(\phi) = \mu_{CO}(\phi) \). Thus, for \( M > \tilde{M} \) the unique stable steady-state equilibrium is \( \phi^* > 0 \), where \( \phi^* \) satisfies \( \mu_{IN}(\phi^*) = \mu_{CO}(\phi^*) \).

Finally note that \( \frac{d\mu_{IN}(\phi)}{dM} < 0 \) and \( \frac{d\mu_{IN}(\phi)}{d\lambda} > 0 \). Consequently, \( \frac{d\phi^*}{dM} > 0 \) and \( \frac{d\phi^*}{d\lambda} < 0 \) for \( M > \tilde{M} \). \( \square \)
Proof of Proposition 3. Recall from Proof of Proposition 1 that $U^{SI} > U^{GO}$ when $y < Y_1 = \phi \delta (x + z)/(1 + \phi \delta^2)$. Moreover, comparing the other expected utilities we get

$$U^{SI} > U^{TE} \text{ if } y < \hat{Y}_1 \equiv \frac{\phi \delta [\beta x + z] + c}{\phi (1 - \alpha \bar{\delta}) + \phi (\beta + \delta^2)},$$

$$U^{TE} > U^{GO} \text{ if } y < \hat{Y}_2 \equiv \frac{\phi \delta [1 - \delta \bar{\delta} (1 - \alpha \bar{\delta}) - \phi \delta^3] (x + z) - [\delta + \phi \delta - \phi \delta^3] [\phi \delta \beta x + \phi \delta z + c]}{(1 + \phi \delta^2) [1 - \delta \bar{\delta} (1 - \alpha \bar{\delta}) + \phi \delta^3] - [\delta + \phi \delta - \phi \delta^3] [\phi (1 - \alpha \bar{\delta}) + \phi \delta^2 + \phi \bar{\delta}]}.$$

Note that $\hat{Y}_1$ is defined by $U^{TE} - U^{SI} = 0$, where $U^{SI} = 0$. Implicitly differentiating $\hat{Y}_1$ w.r.t. $k = c, \alpha, \beta$, we get $\frac{d\hat{Y}_1}{dk} = -\frac{\partial U^{TE}}{\partial y} / \frac{\partial U^{TE}}{\partial k}$, $k = c, \alpha, \beta$, with $\frac{\partial U^{TE}}{\partial k} < 0$ and $\frac{\partial U^{TE}}{\partial y} > 0$ (recall that $\frac{\partial U^{TE}}{\partial \delta} < 0$ as $\delta x > y$ for any individual that chooses testing in equilibrium). Thus, $\frac{d\hat{Y}_1}{dk} > 0$. Moreover, $\hat{Y}_2$ is defined by $U^{GO} - U^{TE} = 0$. Implicit differentiating $\hat{Y}_2$ w.r.t. $k = c, \alpha, \beta$,

$$\frac{d\hat{Y}_2}{dk} = \frac{\frac{<0}{\partial U^{TE}}}{\frac{1 + \phi \delta^2}{\bar{\delta} + \phi \delta (1 - \delta^2)}} - \frac{\frac{<0}{\partial U^{TE}}}{\frac{\bar{\delta} (1 - \alpha \bar{\delta}) + \phi \delta^2}{1 - \delta [\bar{\delta} (1 - \alpha \bar{\delta}) + \phi \delta^2]}} \equiv T(\alpha, \beta)$$

$T(\alpha, \beta)$ is decreasing in $\alpha$ and increasing in $\beta$. Consequently, it is sufficient to evaluate the denominator at $\alpha = 0$ and $\beta = 1$ to show that it is positive. We get

$$T(0, 1) = \frac{1 + \phi \delta^2}{\bar{\delta} + \phi \delta (1 - \delta^2)}.$$

Thus, the denominator is strictly positive for all $\alpha, \beta \in [0, 1)$. Consequently, $\frac{d\hat{Y}_2}{dk} < 0$.

Next, we derive the optimal choices for different values of $z$. Suppose for a moment that $c$ is sufficiently low so that some individuals choose $TE$ (i.e., $\hat{Y}_2 > \hat{Y}_1$ for some $z > 0$). For $c = 0$ and $z = 0$ we find that $\hat{Y}_1(z = 0) < \hat{Y}_2(z = 0)$ if

$$0 < \phi \left(1 - \frac{1}{\alpha \bar{\delta}}\right) - \beta \bar{\delta} + \phi \delta^2 (1 - \beta).$$

Note that the RHS of this condition is decreasing in $\alpha$ and $\beta$. Evaluating this condition at the highest possible values of $\alpha$ and $\beta$, $\alpha = \beta = 1/2$, this condition becomes

$$0 < \phi \left(1 - \frac{1}{2}\right) - \frac{1}{2} \bar{\delta} + \frac{1}{2} \phi \delta^2 \quad \Leftrightarrow \quad 0 < \phi (1 - \bar{\delta}) + \phi \delta^2,$$

which is clearly satisfied. Thus, $\hat{Y}_1(z = 0) < \hat{Y}_2(z = 0)$ for all $\alpha, \beta \in [0, 1/2)$. Next, we show that $Y_1(z = 0) > \hat{Y}_1(z = 0)$ for $c = 0$, which is equivalent to
\[ \Phi(1 - \alpha \delta) + (1 - \beta)\phi \delta^2 > \Phi \beta. \]  

(A1)

The LHS of this condition is decreasing in \( \alpha \) and \( \beta \), and the RHS is increasing in \( \beta \). Evaluating this condition at the highest possible values of \( \alpha \) and \( \beta \), \( \alpha = \beta = 1/2 \), we get

\[ \frac{1}{2} \Phi(1 - \delta) + \frac{1}{2} \phi \delta^2 > 0, \]

which is always satisfied. Thus, condition (A1) is satisfied for all \( \alpha, \beta \in [0, 1/2] \). Thus, \( Y_1(z = 0) > \hat{Y}_1(z = 0) \) for \( c = 0 \). Moreover, \( \frac{d\hat{Y}_1}{dz} > \frac{dY_1}{dz} \) if

\[ \frac{\phi \delta}{\Phi(1 - \alpha \delta) + \phi(\beta + \delta^2)} > \frac{\phi \delta}{1 + \phi \delta^2} \iff 1 > \Phi(1 - \alpha \delta) + \phi \beta. \]

Note that the RHS is decreasing in \( \alpha \) and increasing in \( \beta \). Thus, if this condition is satisfied for \( \alpha = 0 \) and \( \beta = 1/2 \), then it is also satisfied for all \( \alpha, \beta \in [0, 1/2] \). Evaluating this condition at \( \alpha = 0 \) and \( \beta = 1/2 \), we get \( 0 > -\frac{1}{2} \phi \), which is clearly satisfied. Thus, \( \frac{d\hat{Y}_1}{dz} > \frac{dY_1}{dz} \).

This implies that for sufficiently low \( c \), there exists a threshold \( z^* \), so that \( Y_1(z^*) = \hat{Y}_1(z^*) \); see Proof of Proposition 1 for the definition of \( Y_1 \). At \( z = z^* \) we have \( U^{GO} = U^{TE} = U^{SI} = 0 \), and therefore, \( Y_1(z^*) = \hat{Y}_1(z^*) = \hat{Y}_2(z^*) \), with \( \hat{Y}_1(z) < \hat{Y}_2(z) \) for \( z < z^* \). Consequently, individuals choose (i) SI for all \( y < \hat{Y}_1 \), (ii) TE for all \( \hat{Y}_1 \leq y < \hat{Y}_2 \), and (iii) GO for all \( y \geq \hat{Y}_2 \). For \( z \geq z^* \) no one chooses TE.

Note that \( z^* \) is defined by \( Y_1(z^*) = \hat{Y}_1(z^*) = Y_2(z^*) = 0 \) (as \( U^{GO}(z^*) = U^{TE}(z^*) = U^{SI} = 0 \)). Implicit differentiating \( z^* \) w.r.t. \( c \) we get

\[ \frac{d z^*}{dc} = -\frac{\frac{d\hat{Y}_1}{dc} - \frac{dY_1}{dc}}{\frac{d\hat{Y}_1}{dz} - \frac{dY_1}{dz}}, \]

where \( \frac{d\hat{Y}_1}{dc} > 0 \) and \( \frac{dY_1}{dc} = 0 \). Moreover, we have \( \frac{d\hat{Y}_1}{dz} > \frac{dY_1}{dz} \), because

\[ \frac{\phi \delta}{\Phi(1 - \alpha \delta) + \phi(\beta + \delta^2)} > \frac{\phi \delta}{1 + \phi \delta^2} \iff \phi(1 - \beta) > -\Phi \alpha \delta, \]

which is clearly satisfied. Consequently, \( \frac{d z^*}{dc} < 0 \). Moreover, note that \( \lim_{c \to \infty} \hat{Y}_1 = \infty \) and \( \lim_{c \to \infty} \hat{Y}_2 = -\infty \). Thus, there exists a threshold \( \hat{c}(\alpha, \beta) \) so that \( z^*(\hat{c}(\alpha, \beta)) = 0 \). For \( c \geq \hat{c}(\alpha, \beta) \) we have \( \hat{Y}_1(z) \geq \hat{Y}_2(z) \) for all \( z > 0 \); in this case, no individual chooses TE for all \( z \geq 0 \).

Finally, note that \( \hat{c}(\alpha, \beta) \) is defined by \( \hat{Y}_1(\hat{c}(\alpha, \beta)) = \hat{Y}_2(\hat{c}(\alpha, \beta)) \). Implicit differentiating \( \hat{c}(\alpha, \beta) \) w.r.t. \( k = \alpha, \beta \),

\[ \frac{d\hat{c}(\alpha, \beta)}{dk} = -\frac{\frac{d\hat{Y}_1}{dk} - \frac{d\hat{Y}_1}{dc}}{\frac{d\hat{Y}_1}{dc} - \frac{dY_1}{dc}} k = \alpha, \beta. \]
We have already shown that $\frac{dY_1}{dc} > 0$ and $\frac{dY_2}{dc} < 0$, with $k = \alpha, \beta$. Moreover, it is easy to see that $\frac{dY_1}{dk} > 0$ and $\frac{dY_2}{dk} < 0$. Consequently, $\frac{d\epsilon(\alpha, \beta)}{dx} < 0$ and $\frac{d\epsilon(\alpha, \beta)}{d\beta} < 0$.

**Proof of Proposition 4.** Consider the individuals that choose TE (i.e., $Y_1 \leq y < Y_2$ for $c < \hat{c}(\alpha, \beta)$). Let $\eta^{GOI}_t$ denote the fraction of these individuals at time $t$, that is, not infected and goes out (correct negative test), $\eta^{GOI}_t$ the fraction that is infected and goes out (incorrect negative test), $\eta^{SNI}_t$ the fraction that is not infected and self-isolates (incorrect positive test), $\eta^{SNI}_t$ the fraction that is infected and self-isolates (correct positive test), and $\eta^{ILL}_t$ the fraction that is ill and stays home. By definition, $\eta^{GOI}_t + \eta^{GOI}_t + \eta^{SNI}_t + \eta^{SNI}_t + \eta^{ILL}_t = 1$. Moreover, everyone who is ill in $t - 1$ was infected (but asymptomatic) in $t - 1$: $\eta^{ILL}_t = \eta^{GOI}_{t-1} + \eta^{SNI}_{t-1}$. Everyone going out and being infected in $t$, went out in $t - 1$, got infected, and got a false-negative test: $\eta^{GOI}_t = \phi \beta \eta^{GOI}_{t-1}$. Everyone self-isolating and being infected in $t$, went out in $t - 1$, got infected, and got a correct positive test: $\eta^{SNI}_t = \phi (1 - \beta) \eta^{GOI}_{t-1}$. Everyone self-isolating in $t$ and being uninfected, went out in $t - 1$, did not get infected, and got an incorrect positive test: $\eta^{SNI}_t = \phi \alpha \eta^{GOI}_{t-1}$. In the steady-state we have $\eta^k \equiv \eta^k = \eta^k_{t-1} = \eta^k_{t-2} = ..., k = \{GOI0, GOI1, SNI0, SNI1, ILL\}$. Thus, the equilibrium is defined by the following equations: (i) $\eta^{GOI}_t + \eta^{GOI}_t + \eta^{SNI}_t + \eta^{SNI}_t + \eta^{ILL}_t = 1$, (ii) $\eta^{ILL} = \eta^{GOI}_t + \eta^{SNI}_t$, (iii) $\eta^{GOI}_t = \phi \beta \eta^{GOI}_t$, (iv) $\eta^{SNI}_t = \phi (1 - \beta) \eta^{GOI}_t$, (v) $\eta^{SNI}_t = \phi \alpha \eta^{GOI}_t$. Using (ii), (iii), (iv), and (v), in (i), we get $\eta^{GOI}_t = \frac{1}{1 + 2\phi + \phi \alpha}$. Consequently, $\eta^{GOI}_t = \frac{\phi \beta}{1 + 2\phi + \phi \alpha}$.

The CO-curve is then given by

$$
\mu_{CO}(\phi) = \frac{\phi}{1 + 2\phi} \left[ 1 - \Omega(Y_2) \right] + \frac{\phi \beta}{1 + 2\phi + \phi \alpha} \left[ \Omega(Y_2) - \Omega(Y_1) \right]
$$

$$=
\phi \frac{1 + 2\phi + \phi \alpha + \beta (1 + 2\phi) \Theta}{(1 + \phi)(1 + 2\phi + \phi \alpha) + (1 + \phi \beta)(1 + 2\phi) \Theta}, \quad \Theta = \frac{\Omega(Y_2) - \Omega(Y_1)}{1 - \Omega(Y_2)}.
$$

(A2)

We get

$$
\frac{d\mu_{CO}(\phi)}{dk} = \frac{\partial \mu_{CO}(\phi)}{\partial Y_2} \frac{\partial Y_2}{\partial k} + \frac{\partial \mu_{CO}(\phi)}{\partial Y_1} \frac{\partial Y_1}{\partial k} + \frac{\partial \mu_{CO}(\phi)}{\partial k}, \quad k = c, \alpha, \beta.
$$

Note that

$$
\frac{\partial \mu_{CO}(\phi)}{\partial \Theta} = \phi \beta \frac{(1 + 2\phi)(1 + \phi \beta)(1 + 2\phi) \Theta}{(1 + \phi)(1 + 2\phi + \phi \alpha) + (1 + \phi \beta)(1 + 2\phi) \Theta}\left[\Omega(Y_2) - \Omega(Y_1)\right] - \phi \beta \frac{(1 + 2\phi + \phi \alpha + \beta (1 + 2\phi) \Theta)(1 + \phi \beta)(1 + 2\phi)}{(1 + \phi)(1 + 2\phi + \phi \alpha) + (1 + \phi \beta)(1 + 2\phi) \Theta}.
$$

This is negative if
\[
\beta (1 + 2\phi) [(1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (1 + \phi\beta)(1 + 2\phi)\Theta ] < [1 + 2\phi + \bar{\phi} \alpha + \beta (1 + 2\phi)\Theta ] \\
\Rightarrow \beta (1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) < (1 + \phi\beta)(1 + 2\phi + \bar{\phi} \alpha) \\
\Rightarrow \beta < 1.
\]

This is clearly satisfied. Thus, \( \frac{\partial \mu_{CO}(\phi)}{\partial \Theta} < 0 \). We can also see that \( \frac{\partial \Theta}{\partial Y} > 0 \) and \( \frac{\partial \Theta}{\partial k} < 0 \). Thus, \( \frac{\partial \mu_{CO}(\phi)}{\partial Y_2} < 0 \) and \( \frac{\partial \mu_{CO}(\phi)}{\partial Y_1} > 0 \). We also know from Proposition 3 that \( \frac{\partial Y_i}{\partial k} > 0, k = c, \alpha, \beta \). And we can see that \( \frac{\partial \mu_{CO}(\phi)}{\partial c} = 0 \). Moreover,

\[
\frac{\partial \mu_{CO}(\phi)}{\partial \alpha} = \phi \left[ (1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (1 + \phi\beta)(1 + 2\phi)\Theta \right] \\
- \left[ (1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (1 + \phi\beta)(1 + 2\phi)\Theta \right]^2.
\]

This is positive if

\[
(1 + \phi\beta)(1 + 2\phi)\Theta > \beta (1 + 2\phi)\Theta (1 + \phi) \quad \Rightarrow \quad 1 > \beta,
\]

which is clearly satisfied. Thus, \( \frac{\partial \mu_{CO}(\phi)}{\partial \alpha} > 0 \). Moreover,

\[
\frac{\partial \mu_{CO}(\phi)}{\partial \beta} = \left[ (1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (1 + \phi\beta)(1 + 2\phi)\Theta \right] \\
- \left[ (1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (1 + \phi\beta)(1 + 2\phi)\Theta \right]^2.
\]

This is positive if

\[
(1 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (1 + \phi\beta)(1 + 2\phi)\Theta > [1 + 2\phi + \bar{\phi} \alpha + \beta (1 + 2\phi)\Theta ] \phi \\
\Rightarrow (1 + 2\phi + \bar{\phi} \alpha) + (1 + 2\phi)\Theta > 0,
\]

which is always satisfied. Thus, \( \frac{\partial \mu_{CO}(\phi)}{\partial \beta} > 0 \). All this implies that \( \frac{\partial \mu_{CO}(\phi)}{\partial k} > 0, k = c, \alpha, \beta, \) and therefore \( \frac{d\phi^*}{dk} > 0 \). \( \square \)

**Proof of Proposition 5.** Let \( \omega(y) \) denote the pdf of \( y \). Note that \( U^{SI} = 0 \). Taking the total derivative of \( W \) w.r.t. \( k, k = c, \alpha, \beta, \) we get

\[
\frac{dW}{dk} = \frac{\partial W}{\partial \phi} \frac{d\phi^*}{dk} + \frac{\partial W}{\partial k}.
\]

Recall from Proposition 4 that \( \frac{d\phi^*}{dk} > 0 \). Moreover,
\[
\frac{\partial W}{\partial \phi} = [U^{TE}(\bar{Y}_1) - U^{GO}(\bar{Y}_2)]\omega(\bar{Y}_2)\frac{d\bar{Y}_2}{d\phi} - U^{TE}(\bar{Y}_1)\omega(\bar{Y}_1)\frac{d\bar{Y}_1}{d\phi} + \int_{\bar{Y}_1}^{\bar{Y}_2} \frac{\partial U^{TE}}{\partial \phi} d\Omega(y) \\
+ \int_{\bar{Y}_2}^{\infty} \frac{\partial U^{GO}}{\partial \phi} d\Omega(y).
\]

At \( y = \bar{Y}_1 \) we have \( U^{TE}(\bar{Y}_1) = U^{SI} = 0 \), and at \( y = \bar{Y}_2 \) we have \( U^{TE}(\bar{Y}_2) = U^{GO}(\bar{Y}_2) \). Moreover,

\[
\frac{\partial U^{GO}}{\partial \phi} = \frac{[\delta^2y - \delta(x + z)][\delta + \phi\delta(1 - \delta^2)] - [(1 + \phi\delta^2)y - \phi\delta(x + z)]\delta(1 - \delta^2)}{[\delta + \phi\delta(1 - \delta^2)]^2},
\]

which is negative if

\[
[\delta^2y - \delta(x + z)][\delta + \phi\delta(1 - \delta^2)] < [(1 + \phi\delta^2)y - \phi\delta(x + z)]\delta(1 - \delta^2).
\]

This condition can be simplified to \( 0 < (1 - \delta)y + (x + z)\delta \), which is always satisfied. Thus, \( \frac{\partial U^{GO}}{\partial \phi} < 0 \). Recall that \( U^{TE} \) is defined by (3). Let

\[
F \equiv -c + \bar{\phi} \alpha(y + \delta U^{TE}) + \bar{\phi} \alpha\delta(y + \delta U^{TE}) + \phi\beta(\delta(-z + \delta(y + \delta U^{TE})) \\
+ \phi\beta(y + \delta(-x - z + \delta(y + \delta U^{TE}))) - U^{TE}.
\]

We get

\[
\frac{\partial F}{\partial U^{TE}} = \bar{\phi} \alpha \delta + \bar{\phi} \alpha\delta\delta + \phi\beta\delta\delta\delta + \phi\beta\delta\delta\delta - 1 \\
= -[1 - \delta + \phi\delta(1 - \delta^2) + \bar{\phi} \alpha\delta(1 - \delta)] < 0.
\]

Moreover, defining \( A \equiv y + \delta U^{TE} \), we get

\[
\frac{\partial F}{\partial \phi} = -\bar{\alpha} A - \alpha\delta A + \bar{\beta}(\delta(-z + \delta A)) + \beta(y + \delta(-x - z + \delta A))
\]

This is negative if

\[\bar{\alpha} A + \alpha\delta A + \delta(z - \delta A) + \beta(\delta x - y) > 0.\]

Note that the LHS is increasing \( z \). Thus, if this condition is satisfied for \( z = 0 \), it is also satisfied for all \( z > 0 \). Evaluating this condition at \( z = 0 \), we get

\[\alpha A + \alpha\delta A - \delta^2 A + \beta(\delta x - y) > 0.\]

We know that \( \delta x > y \) for individuals that choose \( TE \). Thus, this condition is satisfied if
\[ \pi A + \alpha \delta A - \delta^2 A > 0 \iff 1 - \alpha (1 - \delta) - \delta^2 > 0. \]

The LHS is decreasing in \( \alpha \). Thus, we can evaluate this condition at the highest possible \( \alpha, \alpha = 1/2 \), and get

\[
\frac{1 + \delta - 2\delta^2}{\equiv \Psi(\delta)} > 0.
\]

Note that \( \Psi(\delta) \) is increasing in \( \delta < 1/4 \), and decreasing in \( \delta > 1/4 \). Moreover, we have \( \Psi(0) = 1 > 0 \) and \( \Psi(1) = 0 \). Thus, this condition is satisfied for all \( \delta \in [0, 1) \), so that \( \frac{\partial F}{\partial \phi} < 0 \). Consequently,

\[
\frac{\partial U^{TE}}{\partial \phi} = -\frac{<0}{\frac{\partial \phi}{\partial \Psi}} < 0.
\]

This implies that \( \frac{\partial W}{\partial \phi} < 0 \).

Likewise, noting that \( \frac{\partial U^{GO}}{\partial k} = 0 \), we get

\[
\frac{\partial W}{\partial k} = [U^{TE}(\hat{Y}_2) - U^{GO}(\hat{Y}_2)] \frac{d\hat{Y}_2}{dk} - U^{TE}(\hat{Y}_1) \omega(\hat{Y}_1) \frac{d\hat{Y}_1}{dk} + \int_{\hat{Y}_1}^{\hat{Y}_2} \frac{\partial U^{TE}}{\partial k} d\Omega(y).
\]

Again, at \( y = \hat{Y}_1 \) we have \( U^{TE}(\hat{Y}_1) = U^{SI} = 0 \), and at \( y = \hat{Y}_2 \) we have \( U^{TE}(\hat{Y}_2) = U^{GO}(\hat{Y}_2) \). We can also immediately see that \( \frac{\partial U^{TE}}{\partial k} < 0 \). Thus, \( \frac{\partial W}{\partial k} < 0 \). All this implies that \( \frac{dW}{dk} < 0, k = c, \alpha, \beta. \)

**Proof of Proposition 6.** First, recall from Proposition 3 that \( \frac{\partial Y(\alpha, \beta)}{\partial k} > 0 \) and \( \frac{\partial Y_1(\alpha, \beta)}{\partial k} < 0, k = \alpha, \beta \). Thus, \( \hat{Y}_1(0, 0) < \hat{Y}_1(\alpha, \beta) \), and \( \hat{Y}_2(0, 0) > \hat{Y}_2(\alpha, \beta), \) for \( \alpha, \beta > 0 \).

The number of individuals that go out (GO) and are infected, is given by

\[
N^{GO}_1 = \omega^{GO1}[(1 - \xi)[1 - \Omega(\hat{Y}_2(\alpha, \beta))] + \xi [1 - \Omega(\hat{Y}_2(0, 0))]]
\]

The number of individuals that test (TE) and go out, and are infected (false-negative test), is

\[
N^{TE}_1 = \eta^{GO1}[(1 - \xi)[\Omega(\hat{Y}_2(\alpha, \beta)) - \Omega(\hat{Y}_1(\alpha, \beta))] + \xi [\Omega(\hat{Y}_2(0, 0)) - \Omega(\hat{Y}_1(0, 0))]]
\]

The total number of people going out (GO and TE with negative tests), is given by

\[
N = (\omega^{GO10} + \omega^{GO1})[(1 - \xi)[1 - \Omega(\hat{Y}_2(\alpha, \beta))] + \xi [1 - \Omega(\hat{Y}_2(0, 0))]) + (\eta^{GO10} + \eta^{GO1})[(1 - \xi)[\Omega(\hat{Y}_2(\alpha, \beta)) - \Omega(\hat{Y}_1(\alpha, \beta))] + \xi [\Omega(\hat{Y}_2(0, 0)) - \Omega(\hat{Y}_1(0, 0))]]
\]
The CO-curve is therefore given by

\[ \mu_{CO}(\phi) = \frac{N_G^1 + N_{TE}^1}{N}. \]

We get

\[
\frac{d\mu_{CO}(\phi)}{d\xi} = \left[ -\omega^{GO1} \Delta \Omega(\hat{Y}_2) + \eta^{GO1} \Delta \Omega(\hat{Y}_2) \right] \left[ \frac{N_G^1 + N_{TE}^1}{[N]^2} \right]
\]

where \( \Delta \Omega(\hat{Y}_1) = \Omega(\hat{Y}_1(0,0)) - \Omega(\hat{Y}_1(\alpha, \beta)) < 0 \) and \( \Delta \Omega(\hat{Y}_2) = \Omega(\hat{Y}_2(0,0)) - \Omega(\hat{Y}_2(\alpha, \beta)) > 0 \). Using the expressions for \( N_G^1, N_{TE}^1 \), and \( N \), we find that \( \frac{d\mu_{CO}(\phi)}{d\xi} < 0 \) if

\[
\frac{d\mu_{CO}(\phi)}{d\xi} = \left[ -\omega^{GO1} \Delta \Omega(\hat{Y}_2) + \eta^{GO1} \Delta \Omega(\hat{Y}_2) \right] \left[ \frac{N_G^1 + N_{TE}^1}{[N]^2} \right]
\]

Simplifying this condition we get

\[
\eta^{GO1} [AC + B\Delta \Omega(\hat{Y}_2)](\omega^{GO0} + \omega^{GO1}) < \eta^{GO1} [AC + B\Delta \Omega(\hat{Y}_2)](\eta^{GO0} + \eta^{GO1}).
\]

Using the expressions for \( \omega^{GO0}, \omega^{GO1}, \eta^{GO0}, \) and \( \eta^{GO1} \) (see the derivation of the CO-curve in this appendix, and Proof of Proposition 4), we can write this condition as

\[
\frac{1 + \phi}{\phi} < \frac{1 + \phi \beta}{\phi \beta} \quad \Leftrightarrow \quad \beta(1 + \phi) < 1 + \phi \beta \quad \Leftrightarrow \quad \beta < 1,
\]

which is clearly satisfied. Thus, \( \frac{d\mu_{CO}(\phi)}{d\xi} < 0 \), and therefore, \( \frac{d\phi}{d\xi} < 0 \).

Proof of Proposition 7. Rederiving the expected utility \( U^{GO} \) we get
\[ U_{GO} = \frac{(1 + \phi \delta^2)(1 - \tau) y - \phi \delta (x + z)}{\delta + \phi \delta (1 - \delta^2)}. \]

The expected utilities \( U^{TE} \) and \( U^{SI} \) do not depend on \( \tau \), and therefore do not change. We then find that \( U^{TE} > U^{GO} \) if

\[
y < \bar{Y}_2(\tau)
\]

\[\equiv \frac{\phi \delta [1 - \delta (\bar{\phi}(1 - \alpha \bar{\delta}) + \phi \delta^2)](x + z) - [\bar{\delta} + \phi \delta (1 - \delta^2)][\phi \delta (\beta x + z) + c]}{(1 - \tau)(1 + \phi \delta^2)[1 - \delta (\bar{\phi}(1 - \alpha \bar{\delta}) + \phi \delta^2)] - [\bar{\delta} + \phi \delta (1 - \delta^2)]}
\]

\[[\bar{\phi}(1 - \alpha \bar{\delta}) + \phi \delta^2 + \phi \beta]]

Using (A2) (see Proof of Proposition 4) we get

\[
\frac{d\mu_{CO}(\phi)}{d\tau} = \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_2} \frac{\partial \bar{Y}_2}{\partial \tau}.
\]

We know from Proof of Proposition 4 that \( \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_2} < 0 \). Moreover, we can immediately see that \( \frac{\partial \bar{Y}_2(\tau)}{\partial \tau} > 0 \). Consequently, \( \frac{d\mu_{CO}(\phi)}{d\tau} < 0 \). This implies that \( \frac{d\phi(\tau)}{d\tau} < 0 \). □

**Total Infection Rate with Testing.**

The fraction of GO-people that is infected, is given by \( \varpi^{GO} = \frac{2\phi}{1 + 2\phi} \) (see the derivation of the CO-curve in the appendix). Likewise, the fraction of TE-people that is infected, is given by \( \eta^{TE} = \frac{2\phi}{1 + 2\phi + \phi \alpha} \) (see Proof of Proposition 4). SI-people are never infected. Thus, the total infection rate, \( \Phi \), is

\[
\Phi = \frac{\Omega(\bar{Y}_2) - \Omega(\bar{Y}_1) + \frac{2\phi}{1 + 2\phi} [1 - \Omega(\bar{Y}_2)]}{\Omega(\bar{Y}_1) + [\Omega(\bar{Y}_2) - \Omega(\bar{Y}_1)] + [1 - \Omega(\bar{Y}_2)] + 1}.
\]

We can use \( c \) to measure the effect of a more accessible test on \( \Phi \), where a lower \( c \) implies more testing. We get

\[
\frac{d\Phi}{dc} = \frac{\partial \Phi}{\partial \bar{Y}_1} \left( \frac{d\bar{Y}_1}{dc} + \frac{d\bar{Y}_1}{dc} \right) + \frac{\partial \Phi}{\partial \bar{Y}_2} \left( \frac{d\bar{Y}_2}{dc} + \frac{d\bar{Y}_2}{dc} \right) + \frac{\partial \Phi}{\partial \bar{Y}_2} \left( \frac{d\bar{Y}_2}{dc} + \frac{d\bar{Y}_2}{dc} \right).
\]

We can see that \( \frac{\partial \Phi}{\partial \bar{Y}_1} < 0 \). Likewise,

\[
\frac{\partial \Phi}{\partial \Omega(\bar{Y}_2)} = \frac{2\phi}{1 + 2\phi + \phi \alpha} - \frac{2\phi}{1 + 2\phi}.
\]

This is negative if

\[
\frac{2\phi}{1 + 2\phi + \phi \alpha} < \frac{2\phi}{1 + 2\phi} \Leftrightarrow 0 < \phi \alpha,
\]
which is clearly satisfied. Thus, \( \frac{\delta \Phi}{\delta Y_2} < 0 \). We also know from Proposition 4 that \( \frac{d\alpha}{dc} > 0, \frac{\delta Y}{\delta c} > 0 \), and \( \frac{\delta Y}{\delta c} < 0 \). Moreover,

\[
\frac{\partial}{\partial \Phi} \left[ \frac{2\Phi}{1 + 2\Phi + \Phi \alpha} \right] = \frac{2[1 + 2\Phi + \Phi \alpha] - 2\Phi[2 - \alpha]}{[1 + 2\Phi + \Phi \alpha]^2} = \frac{2 + 2\alpha}{[1 + 2\Phi + \Phi \alpha]^2} > 0
\]

\[
\frac{\partial}{\partial \Phi} \left[ \frac{2\Phi}{1 + 2\Phi} \right] = \frac{2[1 + 2\Phi]}{[1 + 2\Phi]^2} = \frac{2}{[1 + 2\Phi]^2} > 0.
\]

Consequently, \( \frac{\delta \Phi}{\delta \Phi} > 0 \).

Next, recall that \( \tilde{Y}_1 \) is defined by \( U^{TE} (\tilde{Y}_1) - U^{SI} = 0 \). Noting that \( U^{SI} = 0 \), we get

\[
\frac{\partial \tilde{Y}_1}{\partial \Phi} = -\frac{\frac{\delta U^{TE}}{\delta \Phi}}{\frac{\delta U^{TE}}{\delta \Phi}}
\]

It is easy to see that \( \frac{\delta U^{TE}}{\delta \Phi} > 0 \). Moreover, we know from Proposition 5 that \( \frac{\delta U^{TE}}{\delta \Phi} < 0 \). Thus, \( \frac{\delta \tilde{Y}_1}{\delta \Phi} > 0 \).

All this implies that the sign of \( \frac{d\Phi}{dc} \) is ambiguous. A numerical simulation, which shows that depending on parameter values, \( \frac{d\Phi}{dc} \) can be positive or negative, is available from the authors upon request.

Model with Congestion Externality.

Consider first the base model without testing. In the presence of a congestion externality we have \( U^{SI} = 0 \) and

\[
U^{GO} = \frac{(1 + \Phi \delta^2)y - \Phi \delta(x + z(\Phi))}{\delta + \Phi \delta(1 - \delta^2)}.
\]

Thus, we have \( U^{SI} > U^{GO} \) when

\[
y < \tilde{Y}_1 = \frac{\Phi \delta(x + z(\Phi))}{1 + \Phi \delta^2}, \text{ with } \frac{d\tilde{Y}_1}{d\tau} = \frac{\Phi \delta z'(\Phi)\Phi}{1 + \Phi \delta^2} > 0.
\]

Now consider the model with testing. With a congestion externality we find that

\[
U^{TE} = \frac{[\Phi (1 - \alpha \delta) + \Phi \delta^2]y - \Phi [\beta (\delta x - y) + \delta z(\Phi)] - c}{1 - \delta[\Phi (1 - \alpha \delta) + \Phi \delta^2]}.
\]

We then have \( U^{SI} > U^{TE} \) when

\[
y < \tilde{Y}_1 = \frac{\Phi \delta [\beta x + z(\Phi)] + c}{\Phi (1 - \alpha \delta) + \Phi (\beta + \delta^2)}, \text{ with } \frac{d\tilde{Y}_1}{d\tau} = \frac{\Phi \delta z'(\Phi)\Phi}{\Phi (1 - \alpha \delta) + \Phi (\beta + \delta^2)} > 0.
\]
Moreover, we have \( U^{TE} > U^{GO} \) when \( y < \bar{Y}_2 \), where \( \bar{Y}_2 \) is defined by \( U^{GO} - U^{TE} = 0 \). Implicit differentiating \( \bar{Y}_2 \) w.r.t. \( \tau \) yields

\[
\frac{d\bar{Y}_2}{d\tau} = \frac{\phi \delta \xi'(\tau \Phi) \Phi}{\delta + \phi \delta (1 - \delta^2)} - \frac{\phi \delta \xi'(\tau \Phi) \Phi}{1 - \delta [\Phi (1 - \alpha \delta) + \phi \delta^2]}
\]

We have already shown in Proof of Proposition 3 that the denominator is strictly positive for all \( \alpha, \beta \in [0, 1) \). Furthermore, the numerator is strictly positive because

\[
0 < \phi \delta \xi'(\tau \Phi) \Phi < 1 - \delta [\Phi (1 - \alpha \delta) + \phi \delta^2]
\]

Consequently, \( \frac{d\bar{Y}_2}{d\tau} > 0 \).

Proof of Proposition 8. We can immediately see that \( U^{TE} \) is increasing in \( \kappa \). This implies that \( \frac{dY_1}{d\alpha} < 0 \) and \( \frac{dY_2}{d\alpha} > 0 \). □

Proof of Proposition 9. Using \( \hat{Y}_1 \) and \( \hat{Y}_2 \) with \( \alpha = \beta = 0 \), and replacing \( c \) by \( \chi \), we find that

\[
U^{SI} > U^{CT} \quad \text{if} \quad y < \hat{Y}_1 \equiv \frac{\phi \delta \xi + \chi}{\phi + \phi \delta^2},
\]

\[
U^{CT} > U^{GO} \quad \text{if} \quad y < \hat{Y}_2 \equiv \frac{\phi \delta [1 - \delta \Phi - \phi \delta^3](x + z) - [\delta + \phi \delta - \phi \delta^3][\phi \delta \xi + \chi]}{(1 + \phi \delta^2)[1 - \delta \Phi - \phi \delta^3] - [\delta + \phi \delta - \phi \delta^3][\Phi + \phi \delta^2]}.
\]

Moreover, we have \( U^{CT} > U^{TE} \) if

\[
y < \bar{Y}'
\]

\[
= \frac{[1 - \delta (\Phi (1 - \alpha \delta) + \phi \delta^3)][\phi \delta \xi + \chi] - [1 - \delta (\Phi + \phi \delta^2)][\beta \delta \Phi x + \delta \Phi z + c]}{\Phi \alpha \delta - \phi \beta [1 - \delta (\Phi + \phi \delta^2)]}
\]

Clearly, \( \frac{d\bar{Y}}{d\alpha} > 0 \), \( \frac{d\bar{Y}}{d\chi} < 0 \), and \( \frac{d\bar{Y}'}{d\chi} > 0 \).

Next, because \( \frac{d\bar{Y}}{d\alpha} < 0 \) and \( \frac{d\bar{Y}'}{d\chi} > 0 \), with \( \lim_{\chi \to \infty} \bar{Y} = -\infty \) and \( \lim_{\chi \to \infty} \bar{Y} = \infty \), there exists a threshold \( \bar{\chi} \) such that \( \bar{Y}' \geq \bar{Y}_2 \) for \( \chi \geq \bar{\chi} \). The threshold \( \bar{\chi} \) satisfies \( \bar{Y}'(\bar{\chi}) = \bar{Y}_2(\bar{\chi}) \). Moreover, because \( \frac{d\bar{Y}'}{d\chi} > 0 \) with \( \lim_{\chi \to \infty} \bar{Y}' = \infty \), there exists a threshold \( \bar{\chi} \), with \( \bar{\chi} < \bar{\chi} \), such that \( \bar{Y} \leq \bar{Y}_1 \) for \( \chi \leq \bar{\chi} \). Thus, for \( \chi \geq \bar{\chi} \) no one chooses clinical testing. For \( \chi \leq \chi < \bar{\chi} \) individuals choose (i) SI for all \( y < \bar{Y}_1 \), (ii) TE for all \( \bar{Y}_1 \leq y < \bar{Y}' \), (iii) CT for all \( \bar{Y}' \leq y < \bar{Y}_2 \), and (iv) GO for all \( y \geq \bar{Y}_2 \). For \( \chi < \bar{\chi} \)
individuals choose (i) SI for all \( y < \hat{Y}_1 \), (ii) CE for all \( \hat{Y}_1 \leq y < \hat{Y}_2 \), and (iii) GO for all \( y \geq \hat{Y}_2 \).

**Proof of Proposition 10.** Using the steady-state utilities we find that \( U^{SI} > U^{PI} \) when \( y < \hat{Y}_1' \equiv \phi z/(1 - \phi \delta) \). Moreover, we have \( U^{PI} > U^{TE} \) when

\[
y < \hat{Y}_1'' \equiv \frac{[1 - (1 - \phi \delta)\delta^2]([\phi \beta (\beta x + z) + c] - [1 - \delta (\phi (1 - \alpha \delta) + \phi \delta^2)]\delta \phi z}{\phi \delta (1 - \alpha) + [1 - (1 - \phi \delta)\delta^2]\phi \beta}.
\]

We can immediately see that \( \frac{d\hat{Y}_1'}{dk} = 0, k = c, \alpha, \beta \). Moreover, noting that \( \hat{Y}_1'' \) is defined by \( U^{TE} - U^{PI} = 0 \), we get

\[
\frac{d\hat{Y}_1''}{dk} = -\frac{\frac{\partial U^{TE}}{\partial k} < 0 - \frac{\partial U^{PI}}{\partial k} = 0}{\frac{\delta (1 - \phi \delta)}{1 - (1 - \phi \delta)\delta^2} - \frac{\delta (1 - \phi \delta)}{1 - (1 - \phi \delta)\delta^2}} = 0, k = c, \alpha, \beta.
\]

Note that \( S(\alpha, \beta) \) is decreasing in \( \alpha \) and increasing in \( \beta \). Thus, to show that the denominator is positive, it is sufficient to evaluate the denominator at \( \alpha = 1 \) and \( \beta = 0 \). We then find that \( S(1, 0) = \frac{\delta (1 - \phi \delta)}{1 - (1 - \phi \delta)\delta^2} \). This implies that the denominator is strictly positive for all \( \alpha, \beta \in [0, 1/2] \). Consequently, \( \frac{d\hat{Y}_1''}{dk} > 0 \).

Next, we derive the optimal choices for different values of \( y \). Note that \( \hat{Y}_1'(z = 0) = \hat{Y}_1''(z = \alpha = \beta = c = 0) = 0 \). We also know that \( \frac{d\hat{Y}_1'}{dk} > 0, k = \alpha, \beta, c \). Thus, \( \hat{Y}_1'(z = 0) < \hat{Y}_1''(z = 0) \) for \( \alpha, \beta, c > 0 \). Moreover, it is easy to see that \( \frac{d\hat{Y}_1'}{dz} > 0 \) and \( \lim_{z \to \infty} \hat{Y}_1' = \infty \). Thus, there exists a \( z' \) such that \( \hat{Y}_1'(z') = \hat{Y}_1''(z') \), and therefore, \( U^{TE}(z') = U^{PI}(z') = U^{SI} = 0 \). The threshold \( z' \) is defined by \( U^{PI}(z') = U^{SI} = 0 \), which implies that \( z' > 0 \). Moreover, note that at \( z = z' \) we have \( U^{TE}(z') = U^{PI}(z') = U^{SI} = 0 < U^{GO}(z') \), so that \( \hat{Y}_1'(z') = \hat{Y}_1''(z') < \hat{Y}_2(z) \). This implies that \( z' < z^* \). Consequently, for \( z \leq z' \), individuals choose (i) SI for all \( y < \hat{Y}_1' \), (ii) PI for all \( \hat{Y}_1' \leq y < \hat{Y}_1'' \), (iii) TE for all \( \hat{Y}_1'' \leq y < \hat{Y}_2 \), and (iv) GO for all \( y \geq \hat{Y}_2 \).

**Total Infection Rate with Partial Isolation.**

We first derive the CO-curve with partial isolation. We know that for \( z \leq z' \) and \( \hat{Y}_1' \leq y < \hat{Y}_1'' \), individuals choose PI. Let \( \varphi^T_{i}^{GO} \) denote the fraction of these individuals at time \( t \) that go out, \( \varphi^T_{i}^{SI} \) the fraction that self-isolates, and \( \varphi^T_{i}^{ILL} \) the fraction that is ill and stays home. By definition, \( \varphi^T_{i}^{GO} + \varphi^T_{i}^{SI} + \varphi^T_{i}^{ILL} = 1 \). Moreover, everyone who went out in \( t - 1 \) is self-isolating in \( t \): \( \varphi^T_{i}^{SI} = \varphi^T_{i-1}^{GO} \). Individuals that were ill in \( t - 1 \) and recovered, and individuals that went out in \( t - 2 \) but did not get infected (and only self-isolated in \( t - 1 \)), go out in \( t \): \( \varphi^T_{i}^{GO} = \varphi^T_{i-1}^{ILL} + (1 - \phi)\varphi^T_{i-1}^{GO} \). In the steady-state we have \( \varphi^T_{i}^{k} = \varphi^T_{i-1}^{k} = \varphi^T_{i-2}^{k} = \ldots, k \in \{GO, ILL, SI\} \). Thus, the equilibrium is defined by the following equations: (i)
φ^{GO} + φ^{SI} + φ^{ILL} = 1, (ii) φ^{SI} = φ^{GO}, and (iii) φ^{GO} = φ^{ILL} + (1 - φ)φ^{GO}. Solving (i) for φ^{ILL}, and using this expression with (ii) in (iii), we get φ^{GO} = \frac{1}{2 + \phi}.

The CO-curve is then given by

\[ \mu_{CO}(\phi) = \frac{\frac{\phi}{1 + 2\phi}[1 - \Omega(\hat{Y}_2)] + \frac{\phi\beta}{1 + 2\phi + \phi\alpha}[1 - \Omega(\hat{Y}_2) - 2\Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \frac{1}{2 + \phi}[1 - \Omega(\hat{Y}_1'') - \Omega(\hat{Y}_1')]}{(1 + \phi)(1 + \Omega(\hat{Y}_2)) + (1 + \phi)k[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \frac{mk}{2 + \phi}[1 - \Omega(\hat{Y}_1'') - \Omega(\hat{Y}_1')]}, \]

where \( m = (1 + 2\phi + \phi\alpha) \) and \( k = (1 + 2\phi) \). We can see that \( \frac{\partial \mu_{CO}(\phi)}{\partial \Omega(\hat{Y}_2)} > 0 \). Moreover,

\[ \frac{\partial \mu_{CO}(\phi)}{\partial \Omega(\hat{Y}_2)} = \frac{[-\phi m + \phi\beta k][(1 + \phi)(1 + \Omega(\hat{Y}_2)] + (1 + \phi\beta)k[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \Psi]}{[(1 + \phi)(1 + \Omega(\hat{Y}_2)] + (1 + \phi\beta)k[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \Psi^2}, \]

This is negative if

\[ \phi\beta k(1 + \phi) m[1 - \Omega(\hat{Y}_2)] - \phi m(1 + \phi\beta) k[1 - \Omega(\hat{Y}_2)] - \phi k[1 - \Omega(\hat{Y}_2)] - (1 + \phi) m\phi\beta k[1 - \Omega(\hat{Y}_2)] - \Omega(\hat{Y}_1'')] < 0, \]

which can be simplified to

\[ -(1 - \beta + 2\phi(1 - \beta) + \phi\alpha)\Psi < [1 - \beta]mk[1 - \Omega(\hat{Y}_2)] + [1 - \beta]mk[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')]. \]

The LHS is negative, while the RHS is positive. Thus, \( \frac{\partial \mu_{CO}(\phi)}{\partial \Omega(\hat{Y}_2)} < 0 \). This also implies that \( \frac{\partial \mu_{CO}(\phi)}{\partial \Omega(\hat{Y}_2)} < 0 \).

Next, we can write the CO-curve as

\[ \mu_{CO}(\phi) = \frac{\phi(2 + \phi)m[1 - \Omega(\hat{Y}_2)] + \phi(2 + \phi)\beta k[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \phi m[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \phi k[1 - \Omega(\hat{Y}_2)] - \Omega(\hat{Y}_1')] + \phi m[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \phi k[1 - \Omega(\hat{Y}_2)] - \Omega(\hat{Y}_1')] + \phi m[1 - \Omega(\hat{Y}_2) - \Omega(\hat{Y}_1'')] + \phi k[1 - \Omega(\hat{Y}_2)] - \Omega(\hat{Y}_1')] \]

\[ \equiv \tilde{T}_1. \]
We get

\[
\frac{\partial \mu_{CO}}{\partial \Omega(\bar{Y}_1''')} = k \frac{-\phi(2 + \phi)\beta T_2 + T_1[(1 + \phi\beta)(2 + \phi) - m]}{[T_2]^2}.
\]

Using \( m = (1 + 2\phi + \bar{\phi}\alpha) \) we find that this is positive if

\[
T_1[(1 + \phi\beta)(2 + \phi) - (1 + 2\phi + \bar{\phi}\alpha)] > \phi(2 + \phi)\beta T_2
\]

\[
\Leftrightarrow T_1\bar{\phi}(1 - \alpha) > \phi(2 + \phi)\beta[T_2 - T_1].
\]

Note that this condition is satisfied for a sufficiently small \( \beta \). Thus, \( \frac{\partial \mu_{CO}}{\partial \Omega(\bar{Y}_1''')} > 0 \), and therefore \( \frac{\partial \mu_{CO}}{\partial Y_1} > 0 \), for a sufficiently small \( \beta \).

Totally differentiating \( \mu_{CO}(\phi) \) w.r.t. \( c \),

\[
\frac{d\mu_{CO}(\phi)}{dc} = \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_2} \frac{d\bar{Y}_2}{dc} + \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_1''} \frac{d\bar{Y}_1''}{dc} + \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_1'} \frac{d\bar{Y}_1'}{dc} + \frac{\partial \mu_{CO}(\phi)}{\partial c}.
\]

Recall that \( \frac{d\bar{Y}_2}{dk} > 0 \) and \( \frac{d\bar{Y}_1'}{dk} = 0 \), \( k = c, \alpha, \beta \). Moreover, we know from Proposition 3 that \( \frac{d\bar{Y}_1'}{dk} < 0 \). Thus, \( \frac{d\mu_{CO}(\phi)}{dc} > 0 \) for sufficiently small \( \beta \).

Next, we get

\[
\frac{d\mu_{CO}(\phi)}{d\alpha} = \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_2} \frac{d\bar{Y}_2}{d\alpha} + \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_1''} \frac{d\bar{Y}_1''}{d\alpha} + \frac{\partial \mu_{CO}(\phi)}{\partial \bar{Y}_1'} \frac{d\bar{Y}_1'}{d\alpha} + \frac{\partial \mu_{CO}(\phi)}{\partial \alpha}.
\]

Writing the CO-curve as

\[
\mu_{CO}(\phi) = \frac{\phi(1 + 2\phi + \bar{\phi}\alpha)(2 + \phi) + \phi\beta(1 + 2\phi)(2 + \phi)\Theta}{(1 + \phi)(2 + \phi)(1 + 2\phi + \bar{\phi}\alpha) + (1 + \phi\beta)(2 + \phi)(1 + 2\phi)\Theta}
\]

\[
+ (1 + 2\phi + \bar{\phi}\alpha)(1 + 2\phi)\Gamma,
\]

where

\[
\Theta = \frac{\Omega(\bar{Y}_2) - \Omega(\bar{Y}_1'')}{1 - \Omega(\bar{Y}_2)}, \quad \Gamma = \frac{\Omega(\bar{Y}_1'') - \Omega(\bar{Y}_1')}{1 - \Omega(\bar{Y}_2)},
\]

we get
\[ \frac{\partial \mu_{CO}(\phi)}{\partial \alpha} = \frac{\phi \bar{\phi} (2 + \phi)F_2 - F_1 [(1 + \phi)(2 + \phi)\bar{\phi} + \bar{\phi} (1 + 2\phi)\Gamma]}{[F_2]'} \]

This is positive if
\[ \phi \bar{\phi} (2 + \phi)F_2 > F_1 [(1 + \phi)(2 + \phi)\bar{\phi} + \bar{\phi} (1 + 2\phi)\Gamma] \]
\[ \Leftrightarrow \phi F_2 > F_1 \left[ 1 + \phi + \frac{1 + 2\phi}{2 + \phi} \Gamma \right]. \]

Using \( F_1 \) and \( F_2 \) we can write this condition as
\[ (1 + \phi)(2 + \phi)(1 + 2\phi + \bar{\phi} \alpha) + (2 + \phi)(1 + 2\phi)\Theta + (1 + 2\phi + \bar{\phi} \alpha)(1 + 2\phi)\Gamma \]
\[ > (1 + 2\phi + \bar{\phi} \alpha)(2 + \phi) \left[ (1 + \phi) + \frac{1 + 2\phi}{2 + \phi} \Gamma \right] + \beta (1 + 2\phi)(2 + \phi)\Theta \]
\[ + \beta (1 + 2\phi)\Theta (1 + 2\phi)\Gamma, \]
which can be simplified to
\[ 1 - \beta > \beta \frac{1 + 2\phi}{2 + \phi} \Gamma. \]

This condition is satisfied for a sufficiently small \( \beta \). Thus, \( \frac{\partial \mu_{CO}(\phi)}{\partial \alpha} > 0 \) when \( \beta \) is sufficiently small. We then have \( \frac{d\mu_{CO}(\phi)}{d\alpha} > 0 \) for sufficiently small \( \beta \).

Finally,
\[ \frac{d\mu_{CO}(\phi)}{d\beta} = \frac{\partial \mu_{CO}(\phi)}{\partial \bar{\phi}} \frac{d\bar{\phi}}{d\beta} + \frac{\partial \mu_{CO}(\phi)}{\partial \phi} \frac{d\phi}{d\beta} + \frac{\partial \mu_{CO}(\phi)}{\partial \alpha} \frac{d\alpha}{d\beta} + \frac{\partial \mu_{CO}(\phi)}{\partial \Theta} \frac{d\Theta}{d\beta}. \]

We get
\[ \frac{\partial \mu_{CO}(\phi)}{\partial \beta} = \frac{\phi (1 + 2\phi)(2 + \phi)\Theta F_2 - F_1 \phi (2 + \phi)(1 + 2\phi)\Theta}{[F_2]'} \]

This is positive if
\[ \phi (1 + 2\phi)(2 + \phi)\Theta F_2 - F_1 \phi (2 + \phi)(1 + 2\phi)\Theta \Leftrightarrow F_2 > F_1, \]
which is satisfied, so that \( \frac{\partial \mu_{CO}(\phi)}{\partial \beta} > 0 \). Consequently, \( \frac{d\mu_{CO}(\phi)}{d\beta} > 0 \) for sufficiently small \( \beta \).

Proof of Proposition 11. As a benchmark we first use (2) and differentiate \( \mu \) w.r.t. \( \phi \):
\[
\frac{d\mu_{IN}(\phi)}{d\phi} = \frac{1}{(1 - \lambda)M[1 - (1 - \lambda)\mu]^{M-1}}.
\]

Now suppose that \(M\) is a function of \(\phi\), with \(M'(\phi) < 0\). Using (2) we can differentiate \(\mu\) w.r.t. \(\phi\):

\[
\frac{d\mu_{IN}(\phi, M(\phi))}{d\phi} = \frac{1}{(1 - \lambda)M[1 - (1 - \lambda)\mu]^{M-1}} \left[ 1 + \ln(1 - (1 - \lambda)\mu)[1 - (1 - \lambda)\mu]^{M-1}M'(\phi) \right].
\]

This implies that \(\frac{d\mu_{IN}(\phi, M(\phi))}{d\phi} > \frac{d\mu_{IN}(\phi)}{d\phi}\).

Now suppose that \(\lambda\) is a function of \(\phi\), with \(\lambda'(\phi) > 0\). Using (2) we get

\[
\frac{d\mu_{IN}(\phi, \lambda(\phi))}{d\phi} = \frac{1}{(1 - \lambda)M[1 - (1 - \lambda)\mu]^{M-1}} \left[ 1 + M[1 - (1 - \lambda)\mu]^{M-1}\mu\lambda'(\phi) \right].
\]

Consequently, \(\frac{d\mu_{IN}(\phi, \lambda(\phi))}{d\phi} > \frac{d\mu_{IN}(\phi)}{d\phi}\).

Overall this implies that the IN-curve becomes steeper when either \(M\) or \(\lambda\) depends on \(\phi\), thereby reducing \(\phi^*\). However, this does not change the basic equilibrium structure. \(\square\)