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Abstract

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JEL Classification: C73, I18

Keywords: Economic epidemiology, fatalism, diagnostic testing

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Silent Spreaders: Behavior and Equilibrium Under Asymptomatic Infection*

Flavio Toxvaerd†

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1. Background

The current health crisis, caused by the SARS-CoV-2 virus, is unprecedented in modern times. As governments and health authorities scramble to contain and manage the outbreak, individuals face difficult tradeoffs and must balance infection risk from social proximity against the social and financial costs of mitigation and self-isolation.

A complicating factor during the COVID-19 epidemic, like during the HIV/AIDS epidemic before it, is that a significant proportion of infected individuals are asymptomatic or pre-symptomatic, which means that they can continue being infected for prolonged pe-

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riods without knowing that they are infected.[1] During this time, they may unknowingly pass the infection on to others. Recent research shows that a large proportion of tested subjects turn out to be recovered from a previous spell of infection and are thus partially immune to new infection in the short run, without having had any noticeable symptoms. Estimates vary widely but most studies suggest that as many as half of all infected individuals display no symptoms.[2] Asymptomaticity is often credited with making it easier for diseases to spread in the population.[3]

But what exactly is the mechanism that makes it easier for asymptomatic infection to spread? First, note that by definition, symptoms are subjective and thus cannot serve as a warning to others to keep their distance and take precautionary measures around the infected person. Visible signs of infection would of course help alert those at risk and could help people self-protect. With asymptomatic infection though, neither those at risk of infection nor those that are the source of infection know their own health status and thus both may change behavior accordingly.

This possibility raises a number of questions. First, absent possibilities for testing, how will individuals behave when facing a potentially asymptomatic epidemic when the only available protective measure is social distancing? Second, how does the possibility of testing change individual decisions and what are people's incentives to take tests in the first place? Third, how do these incentives change across the stages of the epidemic? Fourth, as pre-test infection probabilities—which mirror the population frequencies of health states in a homogeneous population—change over the epidemic, how does the interpretation of test results depend on aggregate disease prevalence?

This paper provides a tractable framework for studying the aggregate effects that asymptomaticity can have on individual beliefs and incentives and on how these interact with aggregate equilibrium disease dynamics. The model nests two extreme models, namely those with full health state information like Toxvaerd (2019), Rowthorn and Toxvaerd (2020) and Toxvaerd (2020) and the purely mechanistic, non-behavioral SIR model studied in mathematical epidemiology. Depending on the fraction of asymptomatic individuals in the population, the dynamics of these two idealized benchmarks emerge in my setup as special cases. Second, the analysis contributes to the literature by endogenizing the matching function. In full-information frameworks like those referenced above, equilibrium necessarily leads to so-called linear matching in which only suscep-

[1] A *pre-symptomatic* individual is someone who is infected with COVID-19, has not yet shown symptoms, but eventually will. An *asymptomatic* individual is someone infected with COVID-19, but who will never exhibit symptoms. A *symptom-naive* (or naive) individual is someone who has not yet shown any symptoms. Naive individuals are sometimes referred as *non-symptomatic*.

[2] See https://www.cebm.net/covid-19/covid-19-what-proportion-are-asymptomatic/ for a review of recent studies.

[3] See e.g. https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/asymptomatic-infection

tible individuals protect themselves, while infected people do not. In contrast, in some recent analyses of equilibrium dynamics in economic-epidemic frameworks, people are assumed not to know their infection status (see e.g. Farboodi et al., 2021 and Dasaratha, 2021). In such cases, both infected and susceptible individuals self-protect, leading to so-called quadratic matching. Notably, individuals in those papers do not form beliefs about their health status and so the matching function remains unchanged throughout the course of the epidemic. In the present analysis, people continually update their beliefs about their health status. This leads to state-dependent self-protection decisions for non-symptomatic individuals (infected and susceptible alike), which results in a force of infection that is an endogenous composite of linear and quadratic matching. As the fraction of asymptomatic people in the population is varied from one extreme to the other, the two matching functions emerge as special cases.

In terms of individual decision making, the introduction of potential asymptomaticity has several distinct effects. The first, which I term the *severity effect*, works directly through preferences. If preferences over health states are tied to the presence or absence of symptoms (rather than to the underlying health state), then the wellbeing from being asymptomatically infected is no different than that from being healthy and non-infected. This means that when there is a probability that one is never to experience any symptoms regardless of one's health state, then the prospect of becoming infected is altogether less unpleasant. This will tend to reduce individuals' incentive to mitigate infection.

The second, which I term the *fatalism effect*, is that individuals now face an inference problem which changes across the stages of the epidemic.[4] Specifically, because individuals cannot distinguish states in which they are still susceptible and at risk with states in which they are asymptomatically infected or recovered, they must make inferences about their likely susceptibility and hence the extent to which they will engage in social distancing. As time passes, lack of symptoms is increasingly ascribed to asymptomaticity, thereby decreasing the probability attached to being susceptible and at risk.

Third, there is what I term the *force of infection effect*. This effect comes from the fact that some infected individuals are naive and thus engage in social distancing. This reduces the force of infection faced by those who are actually at risk. In other words, while individuals who know that they are infected may have reduced personal incentives to self-protect, this reduction is attenuated under asymptomatic infection. This means that aggregate self-protection by infected individuals may be higher under asymptomatic infection, ceteris paribus. The fatalism effect and the force of infection effect can be

[4]The term *fatalism* is used here in the sense commonly employed in the literature, namely that people in high-risk environments may have higher propensity to engage in transmissive behavior. Of course, being less susceptible may be regarded as a positive thing, even if it is a consequence of having been infected. This should cause no ambiguities in the analysis that follows.

seen as two opposite reflections of the same underlying force, namely that individuals who have imperfect health state information have difficulty matching behavior to their personal health circumstances.

In addition, the possibility that asymptomatically infected people are less infectious to others introduces a mechanistic impact on disease propagation.

The upshot of these effects is that aggregate mitigation decreases relative to the full information benchmark, with knock-on effects on the number of infected people. While overall cumulative infections may be higher under asymptomaticity, the number of *infected with symptoms* is in fact lower than in the full information benchmark. This fact is reflected in overall social welfare, which turns out to be higher with asymptomatic infection, despite the higher infection numbers.

Last, I use my framework to analyse the effects of both individual and mass testing. For the individual, diagnostic tests for infection and immunity change behavior and thus have a value of information. Except at early stages, tests for immunity have higher private value than tests for infection. This also implies that mass testing for immunity may lead to stronger effects on aggregate behavior and disease dynamics than mass tests for infection, ceteris paribus. I show that depending on the sensitivity and specificity of the tests and on the stage of the epidemic, mass testing may lead to changes in aggregate behaviour that either increases or decreases disease incidence, thereby complicating the welfare assessment of such testing programs.

1.1. Related literature. This paper contributes to three distinct but related literatures. First, there is a strand of literature that considers decentralized social distancing. Examples include Reluga (2010), Fenichel et al. (2011), Fenichel (2013), Chen et al. (2011), Chen (2012) and Toxvaerd (2020). Second, the paper contributes to the literature on asymptomatic infection. Examples include Matthies and Toxvaerd (2016), Toxvaerd (2014) and references therein. Last, the paper contributes to the literature on testing, exemplified by Boozer and Philipson (2000), Gersovitz (2010), Godlonton and Thornton (2013). Berger et al. (2020) study testing in a non-behavioral model in which quarantine planning can be informed by the results of mass testing. Eichenbaum et al. (2020) study the macroeconomics of testing. Ely et al. (2020) consider the optimal allocation of a set of heterogeneous but scarce tests in a static setting. Last, there are a few papers that feature social distancing under incomplete information. Farboodi et al. (2021) and Dasaratha (2021) assume that individuals don't know whether they are susceptible or infected, but become perfectly informed once they exogenously recover. In Farboodi et al. (2021), individuals are assumed not to form beliefs about their health state, although this information is directly payoff relevant to them. In Dasaratha (2021), individuals pay a cost upon becoming infected, yet this does not induce individuals to

update their beliefs until they recover. Keppo et al. (2021) set up a model with potential learning about payoff relevant health states, but proceed to analyze the model under the simplifying assumption that beliefs are degenerate. In particular, while individuals cannot determine whether they are susceptible or infected, they behave as if they are in fact at risk of becoming infected. Berger et al. (2020) consider socially optimal quarantines and testing when individuals can be presymptomatic. They show how testing allows the health authority to better target quarantines to individuals' health states. Piguillem and Shi (2020) similarly show how testing can improve the targeting of public quarantine policies. Eichenbaum et al. (2022) assume that individuals cannot directly observe their health states and so must all act alike. They assume that testing, which perfectly reveals infection status, is gradually rolled out and this allows authorities to better target quarantines. Brotherhood et al. (2020) also study the interaction between testing and mitigation policies under presymptomatic infection and, unlike other papers in the literature, consider the possibility that individuals have confounding symptoms from other sources than the main disease under consideration.

2. THE MODEL

The model builds on the classical *susceptible-infected-recovered* model, but is extended to include the possibility of asymptomatic infection. Time is continuous and runs indefinitely. A closed population consists of a continuum $\mathcal{P} = [0, 1]$ of infinitely lived individuals who can at each instant $t \geq 0$ be in one of three states, namely *susceptible*, *infected* or *recovered*. The sets of individuals who are susceptible, infected or recovered are denoted by $\mathcal{S}(t)$, $\mathcal{I}(t)$ and $\mathcal{R}(t)$ and have measures $S(t)$, $I(t)$ and $R(t)$, respectively. The population size is normalized to one, so these measures can be interpreted as fractions.

At each instant, the population mixes homogeneously. This corresponds to pair-wise random matching where each individual has an equal chance of meeting any other individual, irrespective of the health status of the two matched individuals. A match between an infected and a susceptible individual may infect the susceptible. The rate at which infection is transferred in such a match, absent social distancing, is denoted by $\beta > 0$. This parameter captures the infectivity of the disease. Recovered individuals are immune to further infection and also cannot carry the disease. Coupled with the assumption of homogeneous mixing, this means that the *aggregate* rate at which susceptible individuals become infected, or *disease incidence*, is given by $\beta I(t)S(t)$.

Last, infected individuals spontaneously recover at rate $\gamma \geq 0$. This means that on aggregate, the rate at which recovery occurs is $\gamma I(t)$. Throughout, I will maintain the assumption that $\beta > \gamma \geq 0$. Note that the analysis is easily extended to allow for the possibility of disease-induced mortality. The basic model compartments with states and flows is illustrated in Figure 1.

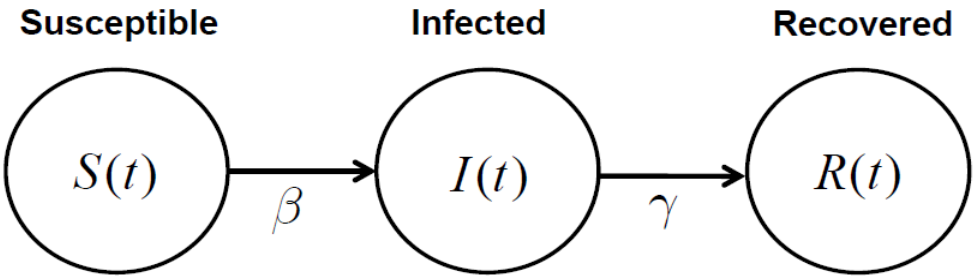

Figure 1: States and flows in the SIR model.

To model the possibility of engaging in social distancing, assume that the individuals can affect their personal rate of infection by controlling the extent to which they expose themselves to others in the population. In particular, at each instant $t \geq 0$, each individual non-cooperatively chooses some social distancing level $d_i(t) \in [0, 1]$, at personal cost $c \times d_i(t)^2/2$ with $c > 0$ a constant. Effectively, this reduces the rate of infection for the individual to $(1 - d_i(t))\beta I(t)$. This formalization captures the notion that, ceteris paribus, engaging in social distancing is costly to the individual. In this analysis, infected and recovered (and therefore immune) individuals who know their health status have no private benefits from social distancing and are thus taken to not engage in any preventive efforts. To complete the economic model, assume that susceptible and recovered individuals earn flow payoffs $\overline{\pi}$, while infected individuals earn $\underline{\pi} < \overline{\pi}$.

For simplicity, I will assume that individuals are myopic in the sense that while they understand the behaviour of aggregate dynamics, they do not maximize an intertemporal objective function. This considerably reduces the complexity of the analysis, while allowing me to focus on the ways in which introducing asymptomatic infection modifies the incentives to socially distance and to submit to diagnostic tests. As will become clear, the main insights remain valid in a setting with forward-looking behavior. This will be shown formally in Section 7.

2.1. Aggregate Epidemic Dynamics and the Matching Function. The dynamics of the epidemic are described by the following system of differential equations:

$$\dot{S}(t) = -\beta I(t)S(t) \tag{1}$$
$$\dot{I}(t) = I(t)\left[\beta S(t) - \gamma\right] \tag{2}$$
$$\dot{R}(t) = \gamma I(t) \tag{3}$$
$$S(t) = 1 - I(t) - R(t) \tag{4}$$
$$S(0) = S_0 > \gamma/\beta, \quad I(0) = I_0 \approx 0, \quad S_0 + I_0 = 1 \tag{5}$$

To understand the different effects of asymptomaticity on behavior, it is useful to start by recalling that behavior is driven by a tradeoff between the costs and benefits of protective measures. While the costs of social distancing are independent of people's health state (i.e. whether susceptible, infected or recovered), the expected benefits are not. As a benchmark, consider an individual i's infection risk under full information. It can be modeled as

$$\beta I(t)(1 - d_i(t))(1 - \bar{d}(t)) \tag{6}$$

where $d_i(t) \in [0, 1]$ is individual i's personal social distancing decision and $\bar{d}$ is the average social distancing decision of those who are currently infected. Note that the social distancing activity of those who are infected is a substitute for the social distancing of those at risk (i.e. the susceptible), in the sense that transmission is decreasing in the mitigation effort of *either* type. This formulation is known in the literature as *quadratic matching*, because the exposure level of the matched individuals are multiplied.[5] Now let us consider incentives. Under full information, infected individuals have no personal benefit from self-protection and so in any equilibrium it must be that $\bar{d}(t) = 0$, leaving susceptible individuals to do all the mitigation of their own accord. This reduces the transmission rate to

$$\beta I(t)(1 - d_i(t)) \tag{7}$$

which is referred to in the literature as *linear matching*.[6] It is important to note that the incentives to self-protect of infected and susceptible people are misaligned unless they have other-regarding preferences. Now consider a world in which only a fraction $(1 - \alpha)$ of the population show symptoms if infected, while a fraction α never experience any symptoms. In such a situation, all symptomatically naive individuals (whether they are susceptible, infected or recovered) will behave the same way and distance at some common level $d^*(t)$. The resulting equilibrium infection risk rate for a susceptible individual will therefore be

[5] See e.g. Alvarez et al. (2021).
[6] See e.g. Toxvaerd (2019).

	Susceptible	Infected	Recovered
Asymptomatic	$\alpha S(t)$	$\alpha I(t)$	$\alpha R(t)$
Symptomatic	$(1-\alpha)S(t)$	$(1-\alpha)I(t)$	$(1-\alpha)R(t)$

Table 1: Fractions of the population in different classes.

$$\beta I(t)(1-d^*(t))(1-\alpha d^*(t)) \tag{8}$$

From this expression, it's clear that as the asymptomatic ratio varies from zero to one, the linear and quadratic formulations obtain as special cases.[7]

2.2. Symptoms, Beliefs and Updating. To model the presence of asymptomatic individuals, I assume that there are two types of individuals, namely *asymptomatic* and *(potentially) symptomatic*. A fraction $\alpha \in [0,1]$ of the population is asymptomatic, which means that they will never find out whether they are or have been infected (unless they take a test, which will be analyzed in Sections 5 and 6). However, they can still become infected and infect others. I assume that someone who is infected but asymptomatic transmits infection to susceptible contacts at rate $\sigma\beta$, where $\sigma \in [0,1]$ captures the possibility that asymptomatic infection has reduced transmissibility. Potentially symptomatic individuals do not show any symptoms while susceptible, but do so once infected. Since they become aware of their infection status as soon as they become infected, they also know once they recover that they are then immune. I will maintain the assumption that asymptomatic individuals have the same utility as susceptible or recovered individuals at all times, so that experiencing symptoms is equivalent to bearing the disease burden due to infection.

Since people do not know ex ante whether they are of the symptomatic or the asymptomatic type, they all behave alike unless they start showing symptoms. Symptomatic infected and symptomatic recovered individuals do not engage in any social distancing, as they know that they face no risk of further infection.

As a fraction α of the population show no symptoms irrespective of their health status, there are effectively six compartments in the model, namely susceptibles, infected or recovered and each of these can be symptomatic or asymptomatic, respectively. The population frequencies for the different types of individual (asymptomatic versus symptomatic) across health compartments are shown in Table 1.

[7] In the analysis that follows, this formula will be further generalized to accommodate the possibility that asymptomatic individuals have reduced infectiousness.

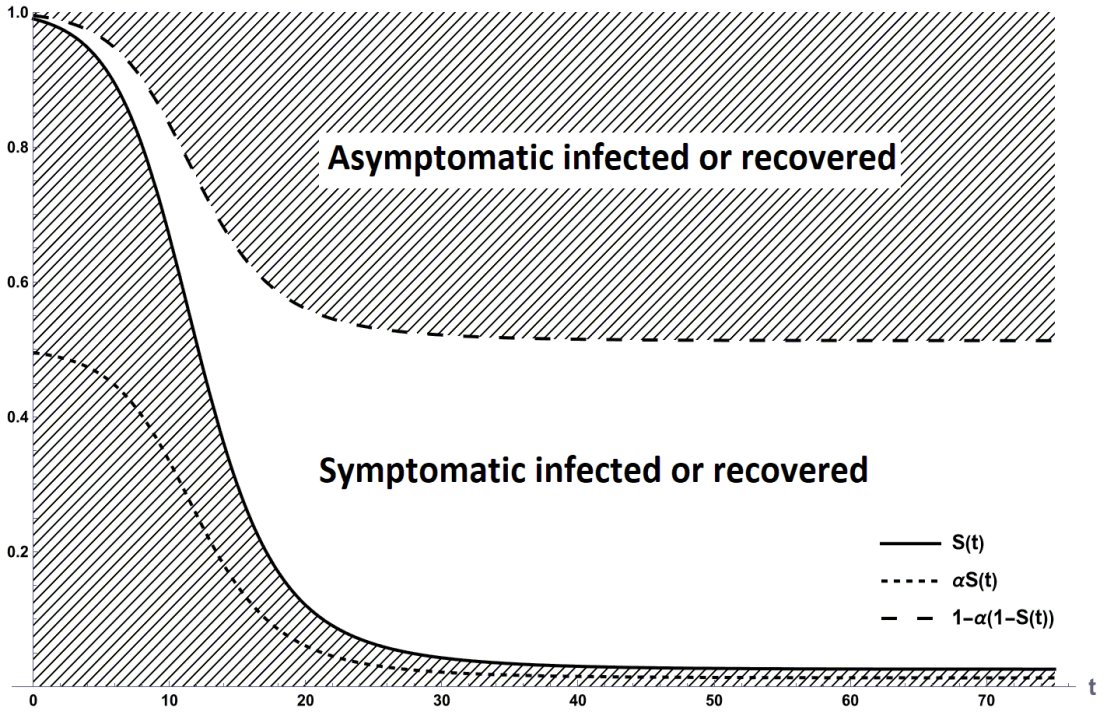

Figure 2: Relative probabilities of being susceptible and non-susceptible, conditional on not showing symptoms. Upper hatched area is asymptomatic non-susceptible, lower hatched area is susceptible (symptomatic and non-symptomatic) and white region is symptomatic non-susceptible (infected and recovered).

The measures of individuals in each compartment are common knowledge amongst individuals. But although everyone knows the population averages, i.e. how many people are in each of these compartments, asymptomatic individuals cannot distinguish between being susceptible or being asymptomatically infected or recovered. This is important, because the inference problem, which changes across the stages of the epidemic, influence people's incentives to engage in social distancing.

Absent any additional information, say learned through testing, how many people would potentially engage in social distancing? Since social distancing is costly, only those individuals who do not positively know that they are not at risk, i.e. that are or have been symptomatically infected, will socially distance. Denote by $\mathcal{A}(t)$ the set of individuals who have never observed any symptoms by time $t \geq 0$. The set of the symptomatically naive individuals has measure

$$\underbrace{S(t)[\alpha + (1-\alpha)]}_{\text{All susceptible}} + \underbrace{\alpha I(t)}_{\text{Asympt. infected}} + \underbrace{\alpha R(t)}_{\text{Asympt. recovered}} = S(t) + \alpha(1 - S(t)) \tag{9}$$

where the last equality follows since $I(t) + R(t) = 1 - S(t)$.

Conversely, individuals who do not engage in social distancing consists of all those who either know they are infected or know that they have been infected but have now recovered. This set has measure

$$\underbrace{(1-\alpha)I(t)}_{\text{Sympt. infected}} + \underbrace{(1-\alpha)R(t)}_{\text{Sympt. recovered}} = (1-\alpha)(1-S(t)) \tag{10}$$

The beliefs of a naive individual are thus

$$p_S(t) \equiv \Pr(i \in \mathcal{S}(t) | i \in \mathcal{A}(t)) = \frac{S(t)}{S(t)+\alpha(1-S(t))} \tag{11}$$

$$p_I(t) \equiv \Pr(i \in \mathcal{I}(t) | i \in \mathcal{A}(t)) = \frac{\alpha I(t)}{S(t)+\alpha(1-S(t))} \tag{12}$$

$$p_R(t) \equiv \Pr(i \in \mathcal{R}(t) | i \in \mathcal{A}(t)) = \frac{\alpha R(t)}{S(t)+\alpha(1-S(t))} \tag{13}$$

Note that the beliefs $p_S(t)$ are increasing in the measure of susceptibles $S(t)$ but decreasing in the proportion of asymptomatic individuals α. As the susceptibles necessarily decrease in measure over time, this implies that the probability that an individual assigns to him or herself being susceptible, conditional on not having observed any symptoms, must decrease as time progresses.

The probability of becoming infected per unit of exposure, conditional on not having had any symptoms, is then

$$p_S(t)\beta I(t) \tag{14}$$

The last thing that must be emphasised is that for an individual to engage in social distancing, it is not enough that he or she perceives him or herself to be susceptible. In fact, for social distancing to make sense, the individual must be *both* susceptible and of the symptomatic type. The probability that a symptom-naive individual is asymptomatically susceptible is

$$(1-\alpha)p_S(t) \tag{15}$$

Note that

$$\lim_{\alpha\to 0}(1-\alpha)p_S(t) = 1, \quad \lim_{\alpha\to 1}(1-\alpha)p_S(t) = 0 \tag{16}$$

As will be shown below, these limit results will imply that the equilibrium under asymptomatic infection will nest the two extreme models in which there is perfect health state information and in which there is no behavioral responses to risks at all (i.e. the outcome coincides with the purely biological model). In particular, as $\alpha \to 0$, the equilibrium path coincides with the full information equilibrium while as $\alpha \to 1$, individuals cease all mitigation and the dynamics follow the mechanical, non-behavioral dynamics.

α	β	γ	$\mathbb{R}_0$	σ	$\overline{\pi}$	$\underline{\pi}$	c	$S(0)$	$I(0)$	$R(0)$	q_I	q_R
1/3	1/2	1/7.5	3.75	0.9	0	-1	1/10	0.99	0.01	0	0.95	0.95

Table 2: Parameter values used for simulations.

Throughout, I use the model parameters in Table 2 to generate plots.

For the disease dynamics parameters, I use numbers that yield a basic reproduction ration $\mathbb{R}_0 = 3.75$, as this is similar to estimates made for the COVID-19 pandemic (Kucharski et al., 2020; Wu et al., 2020). Additionally, I use a recovery rate of $\gamma = 1/7.5$, which implies an average infectious period of 7.5 days. This is also close to some estimates made for the COVID-19 pandemic (Byrne et al., 2020).[8]

3. Equilibrium Disease Dynamics

In this section, I first characterize the individual best responses and then proceed to characterize aggregate equilibrium disease dynamics and their properties.

3.1. Best Responses and Equilibrium. The objective of a representative naive individual $i \in \mathcal{A}(t)$ at time $t \geq 0$ is to choose a level of social distancing $d_i \in [0,1]$ to solve the following problem:

$$\max_{d_i(t)\in[0,1]} U(d_i(t), p_S(t)) = \max_{d_i(t)\in[0,1]} \left\{ -(1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t)(\overline{\pi}-\underline{\pi}) - c \times \frac{d_i(t)^2}{2} \right\} \tag{17}$$

where

$$\phi(t) \equiv (1-\alpha)\times 1 + \sigma\alpha(1-d(t)) = [1-\alpha(1-\sigma(1-d(t)))] \tag{18}$$

is the adjusted force of infection coefficient under asymptomatic infection and $d(t)$ is the average social distancing of all naive individuals in the population.[9] Note that

$$\lim_{\alpha\to 0}\phi(t) = 1, \quad \lim_{\alpha\to 1}\phi(t) = \sigma\left(1-d(t)\right) \tag{19}$$

Definition 1. *An equilibrium is a set of paths*

$$\left\{ \left\{ d_i^*(t) \right\}_{i\in\mathcal{A}(t)}, S(t), I(t), R(t) \right\}_{t\geq 0} \tag{20}$$

with the following properties:

[8] The simulations in this paper were done in *Mathematica 13.0*. All code is available upon request.

[9] This objective function is identical to the objective $[1-(1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t)]\,\overline{\pi} + (1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t)\underline{\pi} - c\times d_i(t)^2/2$, except for the constant $\overline{\pi}$, which does not alter the maximizer.

(a) Given $\{S(t), I(t), R(t)\}$, $d_i^*(t)$ solves problem (17) for each individual $i \in \mathcal{A}(t)$ at each instant $t \geq 0$.

(b) Given $\{d_i^*(t)\}_{i\in\mathcal{A}(t)}$, the paths $\{S(t), I(t), R(t)\}$ satisfy the set of differential equations for the aggregate system

$$\begin{aligned}
\dot{S}(t) &= -(1-d^*(t))(1-\alpha(1-\sigma(1-d^*(t)))\beta I(t)S(t) && (21)\\
\dot{I}(t) &= I(t)\left[(1-d^*(t))(1-\alpha(1-\sigma(1-d^*(t)))\beta S(t) - \gamma\right] && (22)\\
\dot{R}(t) &= \gamma I(t) && (23)\\
S(t) &= 1 - I(t) - R(t) && (24)\\
S(0) &= S_0 > \gamma/\beta, \quad I(0) = I_0 \approx 0, \quad S_0 + I_0 = 1 && (25)
\end{aligned}$$

Next, I characterize the equilibrium path. The first-order condition for the individual's (17) problem is given by[10]

$$\frac{\partial U(d_i(t), p_S(t))}{\partial d_i(t)} = p_S(t)(1-\alpha)\phi(t)\beta I(t)(\overline{\pi} - \underline{\pi}) - c d_i(t) = 0 \tag{26}$$

Thus the best-response social distancing decision of the individual, if it is interior, is defined by the equation

$$d_i(t) = p_S(t)(1-\alpha)\phi(t)\beta I(t)\left(\frac{\overline{\pi} - \underline{\pi}}{c}\right) \tag{27}$$

This equation states that the individual will engage in social distancing to the extent that it equalizes the marginal cost of social distancing with its expected marginal benefit. In turn, the latter is a function of beliefs about susceptibility, of the adjusted force of infection and of the disease burden from infection (i.e. its severity). For comparison, the best response of a susceptible individual under perfect information without asymptomatic infection is obtained by setting $p_S(t) = \phi(t) = (1-\alpha) = \sigma = 1$, namely

$$d_i(t) = \beta I(t)\left(\frac{\overline{\pi} - \underline{\pi}}{c}\right) \tag{28}$$

The first-order condition clearly shows the attenuation effect of asymptomaticity, as it reduces the disease burden from $(\overline{\pi} - \underline{\pi})$ to $(1-\alpha)(\overline{\pi} - \underline{\pi})$. It also shows that ceteris paribus, the incentive for social distancing of a symptom-naive individual is moderated by the perceived susceptibility $p_S(t)$ and by the population-wide behavioral response of symptom-naive infected people, captured by $\phi(t)$. While the former is monotone decreasing over time, the latter is typically not.

[10]The second-order condition is satisfied as $\partial^2 U(d_i(t), p_S(t))/\partial d_i(t)^2 = -c < 0$.

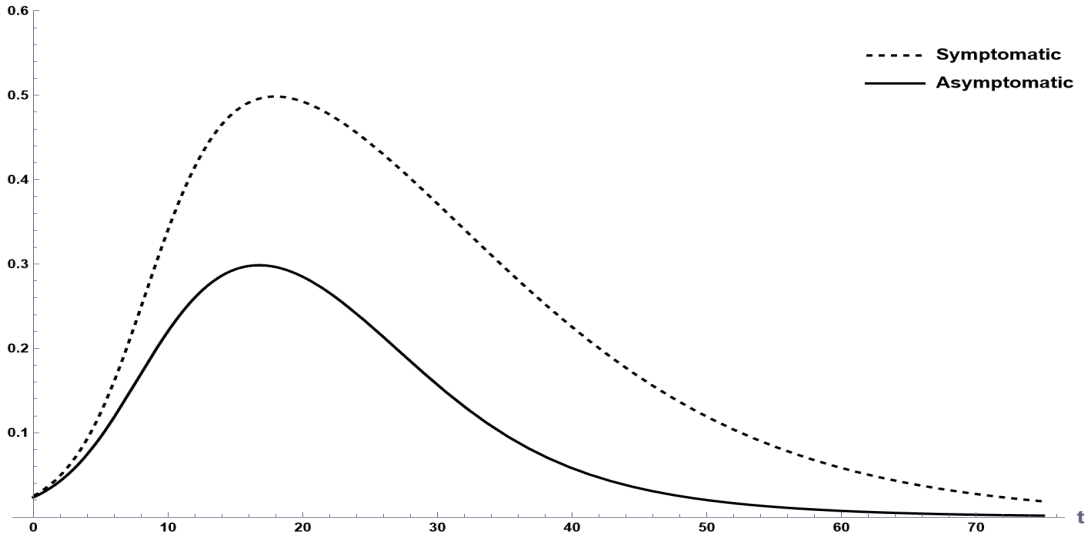

Figure 3: Equilibrium social distancing paths under symptomatic and asymptomatic infection.

Using the definition of $\phi(t)$ and the fact that in symmetric equilibrium $d_i(t) = d(t)$, the best response can be rewritten as

$$d(t) = \frac{S(t)}{S(t) + \alpha(1 - S(t))}(1 - \alpha(1 - \sigma(1 - d(t)))(1 - \alpha)\beta I(t) \left(\frac{\overline{\pi} - \underline{\pi}}{c}\right) \tag{29}$$

This can be solved to yield

$$d(t) = \frac{(1 - \alpha(1 - \sigma))(1 - \alpha)S(t)\beta I(t)(\overline{\pi} - \underline{\pi})}{c\alpha + (1 - \alpha)(c - \alpha\beta(\overline{\pi} - \underline{\pi})\sigma I(t))S(t)} \tag{30}$$

The equilibrium extent of social distancing for a symptom-naive individual at time $t \geq 0$, including the possibility of a corner solution, is then

$$d^*(t) = \min\left\{1, \frac{(1 - \alpha(1 - \sigma))(1 - \alpha)S(t)\beta I(t)(\overline{\pi} - \underline{\pi})}{c\alpha + (1 - \alpha)(c - \alpha\beta(\overline{\pi} - \underline{\pi})\sigma I(t))S(t)}\right\} \tag{31}$$

Figure 3 compares the equilibrium paths of social distancing under asymptomatic and under perfect health state information, respectively. In turn, Figure 4 displays the resulting equilibrium paths of disease prevalence and compares these with the disease path under the mechanistic (non-behavioral) dynamics.Note that for fixed beliefs $p_S(t)$, social distancing is increasing in the force of infection $\beta I(t)$ because an individual who is not certain that he or she is either (asymptomatically) infected or recovered, will use this as a measure of the probability of becoming infected. Furthermore, it also follows that for any disease prevalence level $I(t)$, the privately optimal social distancing effort is decreasing

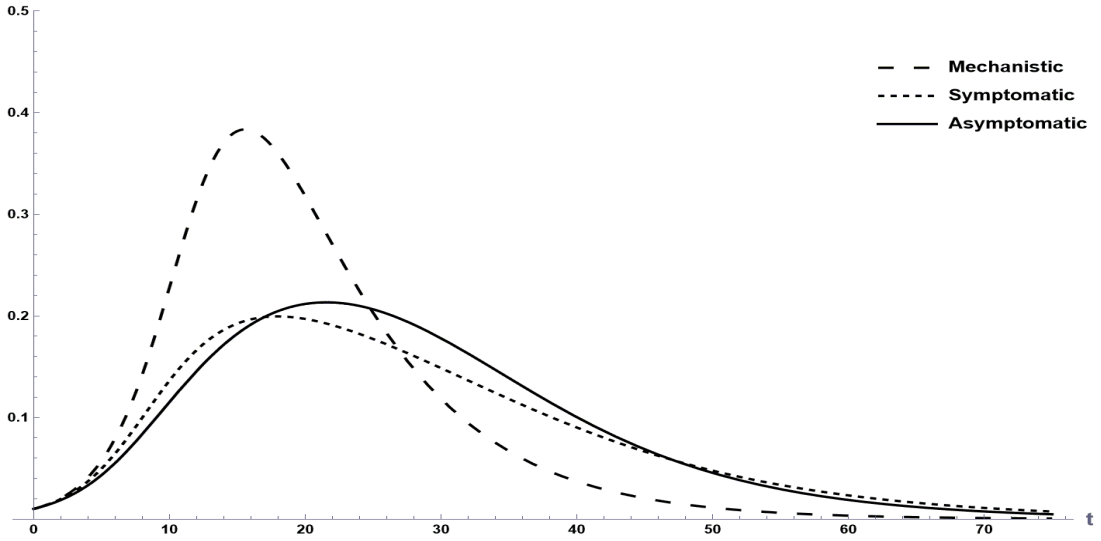

Figure 4: Equilibrium disease prevalence paths under symptomatic and asymptomatic infection.

in the individual's perceived susceptibility $p_S(t)$.

3.2. Effect Decomposition. From the individual's best response function (29) it is readily verified that relative to the full-information benchmark (28), the individual's best response to disease risk is decreased through three distinct channels, illustrated in Figure 5. First, the factor $p_S(t)$ captures a *fatalism effect*, whereby individuals decrease their mitigation efforts because they become increasingly confident that they are no longer at risk. This effect is monotone decreasing as the epidemic progresses. Second, the factor $\phi(t)$ constitutes a *force of infection effect*, which is U-shaped. This effect stems from the fact that a fraction α of infected individuals are symptomatically naive and therefore behave as if they were still at risk. This leads these individuals to engage in mitigation, thereby effectively decreasing the force of infection for those who are actually susceptible. Last, the constant factor $(1 - \alpha)$ constitutes a *severity effect*, because a fraction α of the population will never experience any symptoms. This means that ex ante, when people don't know whether they are of the symptomatic type, the overall expected disease burden from becoming infected is diminished. This lowers the benefits of mitigation.[11] Last, if transmissibility of asymptomatic infection is reduced and $\sigma < 1$, then there is an additional mechanistic *infectivity reduction effect* of asymptomaticity.

In Section 6, I will consider the aggregate effects on disease incidence of mass testing

[11]Note that the absence of symptoms does not necessarily imply the absence of harm, because infection can have long-term consequences regardless of the short-term symptoms. In those cases, the additional disease burden from symptomatic infection should be thought of as the level that is over and above the long-term effects of infection. For more detail, see e.g. https://www.acpjournals.org/doi/10.7326/M20-3012

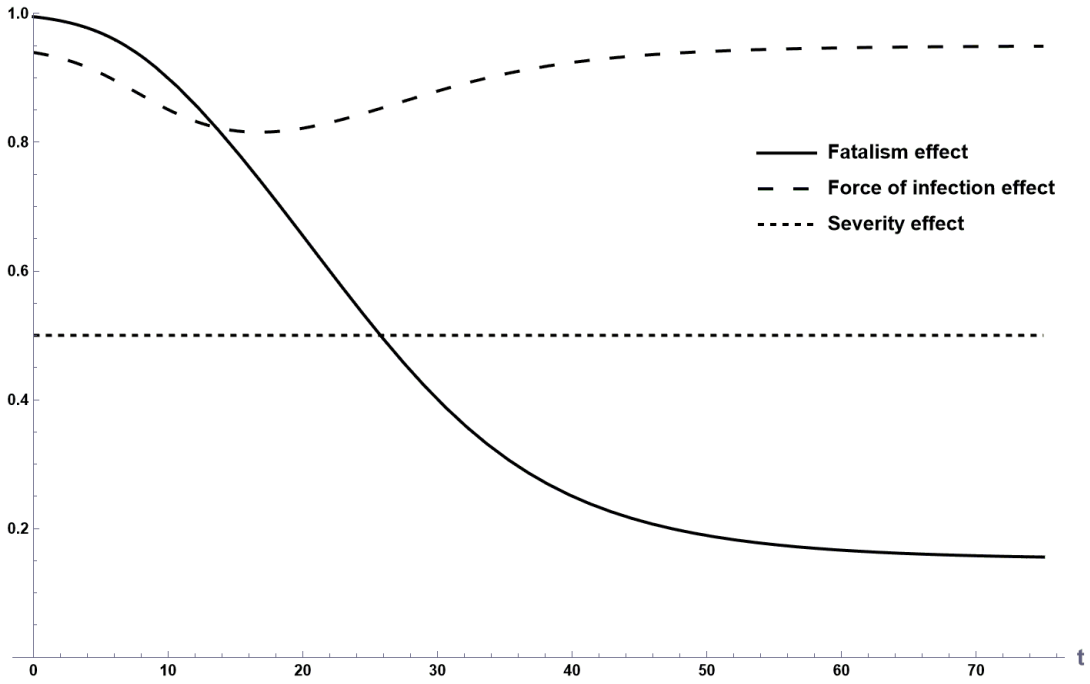

Figure 5: Decomposition of effects of asymptomaticity. The fatalism effect, the force of infection effect and the severity effect.

for infection and immunity. It turns out that these aggregate changes stem from the impact that testing has on the fatalism and force of infection effects (while the severity effect is unchanged).

3.3. Aggregate Welfare. To compare social welfare under asymptomatic infection with the full information benchmark, I compute the undiscounted sum of individual payoffs across the epidemic. Aggregate welfare at time $t \geq 0$ is simply

$$W(t) = [S(t) + \alpha(I(t) + R(t))] (\overline{\pi} - c(d^*(t))) + (1 - \alpha) [I(t)\underline{\pi} + R(t)\overline{\pi}] \tag{32}$$

Overall social welfare is then

$$W_T = \int_0^T W(t)dt \tag{33}$$

Note that asymptomaticity has multiple influences on aggregate welfare.[12] First, a fraction α of all non-susceptible individuals engage in costly mitigation. Under full information, these individuals would know that they are at no risk and would therefore engage in no protective behavior. Second, a measure $\alpha I(t)$ of individuals are infected but do not know it, earning them payoffs $\overline{\pi}$ rather than the reduced payoffs $\underline{\pi} < \overline{\pi}$. Third, under asymptomaticity, the entire infection path is changed, as is the concomitant path of social distancing by those who are actually at risk. This involves less mitigation throughout, which in turn causes higher aggregate infection. Figure 6 and Figure 7 show the cumulated number of infections and aggregate social welfare, respectively. Two features stand out. First, aggregate infections may be substantially higher under asymptomatic infection than under the full information benchmark (unless the infection reduction effect is substantial). This follows naturally from the fact that the three effects of asymptomatic disease all work in the direction of diminishing equilibrium mitigation. Yet despite this, we see that aggregate social welfare is actually higher under asymptomatic infection, despite higher aggregate infection numbers. This stems from the severity effect, i.e. that the disease burden for those who are infected is discounted by a factor $(1-\alpha)$. Figure 7 shows that the cumulative number of *infected with symptoms* under asymptomatic infection (i.e. a subset of those infected in the model with asymptomatic infection) is lower than the corresponding number under full information. Thus, while it is true that asymptomatic infection unambiguously increases disease prevalence, the analysis shows that on welfare grounds, decreased severity counterweights that effect.

4. Diagnostic Tests and Predictive Values

In this section, I consider the application of two diagnostic tests, namely an I-test, which detects whether infection is present in the individual at the time of testing and an R-test, which detects whether the individual is immune. Note that tests for infection and immunity are indirectly informative about the individual's susceptibility at a point in time, although they are not direct tests for susceptibility. Each test will have the following structure. For each test $k = I, R$, the individual will either be in the given category or not at time $t \geq 0$, indicated by a state of the world $\theta_k(t) \in \{0, 1\}$. The test will return a

[12] Two notes are in order. First, the definition of $W(t)$ contains the term $(1-\alpha)R(t)\overline{\pi}$, which means that the planner values the utility of recovered individuals although people themselves ignore the possibility of recovery from their own (myopic) decision making. But since there is a one-for-one relationship between infected and recovered individuals, the inclusion of this term is just a rescaling of the value of the disease burden. In addition, $\underline{\pi}$ can be reinterpreted as the expected net present value of an individual who has just become infected and who will recover at the constant rate γ (see e.g. Toxvaerd, 2020). Second, the welfare criterion is calculated over some finite but large horizon T in order to avoid having to compare infinite undiscounted sequences of payoffs.

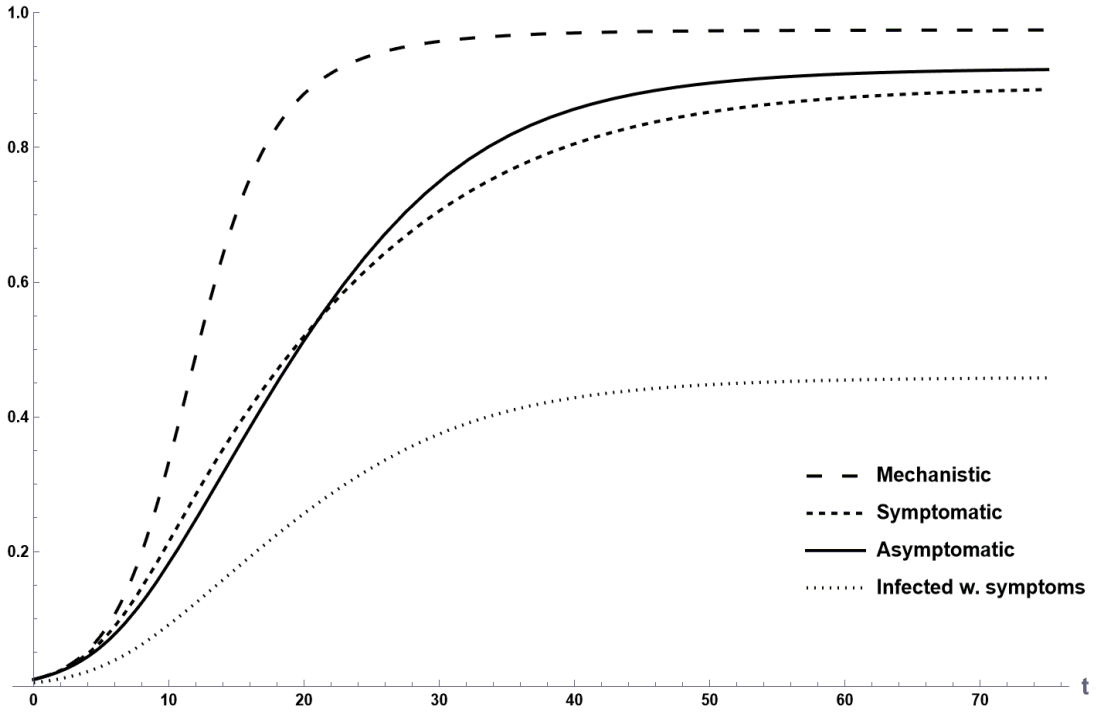

Figure 6: Cumulative infection cases under full information, under asymptomatic infection and in non-behavioral benchmark.

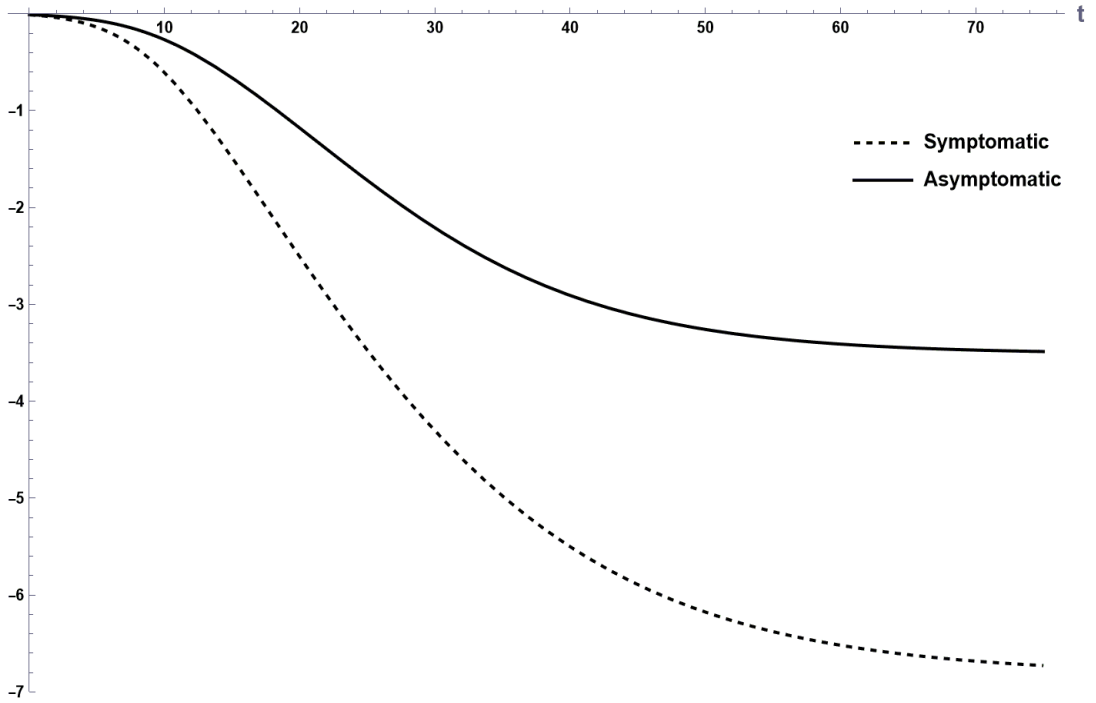

Figure 7: Cumulative welfare under full information and under asymptomatic infection.

	$m_k(t)=1$	$m_k(t)=0$
$\theta_k(t)=1$	q_k	$1-q_k$
$\theta_k(t)=0$	$1-q_k$	q_k

Table 3: Health states, test results and precisions.

test result $m_k(t) \in \{0,1\}$ with precision

$$\Pr(m_k(t)=1|\theta_k(t)=1) = \Pr(m_k(t)=0|\theta_k(t)=0) = q_k > 1/2 \tag{34}$$

Without loss of generality, I impose symmetry so that the probability of a true positive equals the probability of a true negative. The testing setup is illustrated in Table 3.

The I-test is essentially a virological test, while the R-test is a serological (or antibody) test. In practice, tests for infection and tests for antibodies or T cells may differ in their accuracy. As will become clear, the predictive value and value of information for the two tests will change during the epidemic as the proportion of the population in the different classes change. To focus on these changes, I will put the two tests on an equal footing and assume that $q_I = q_R$ so that the tests are equally informative about the state for which they test.[13]

In this model, risk-mitigation is the central concern of individuals and since their incentives depend on how susceptible they believe themselves to be, beliefs play a key role in driving behavior. Diagnostic testing, either for infection or for immunity, can be indirectly informative about the risks that symptom naive individuals face. This means that diagnostic tests can induce changes in behavior and are thus valuable in the value of information sense.[14] But the value of the tests changes across the states of the epidemic, because belief formation is part and parcel of the equilibrium dynamics. To understand how the value of the different diagnostic tests changes over time, I characterize the testing of a single individual at arbitrary moments along the equilibrium path. In practice, I take the aggregate evolution of the disease as given and consider the effects of testing a single individual who is symptomatically naive.

Consider an individual who has not shown any previous symptoms and who receives a positive result $m_k(t) = 1$ on a k-test at time $t \geq 0$. From Bayes' rule, the probability

[13] https://academic.oup.com/bjaed/article/8/6/221/406440

[14] See e.g. work by Boozer and Philipson (1996, 2000).

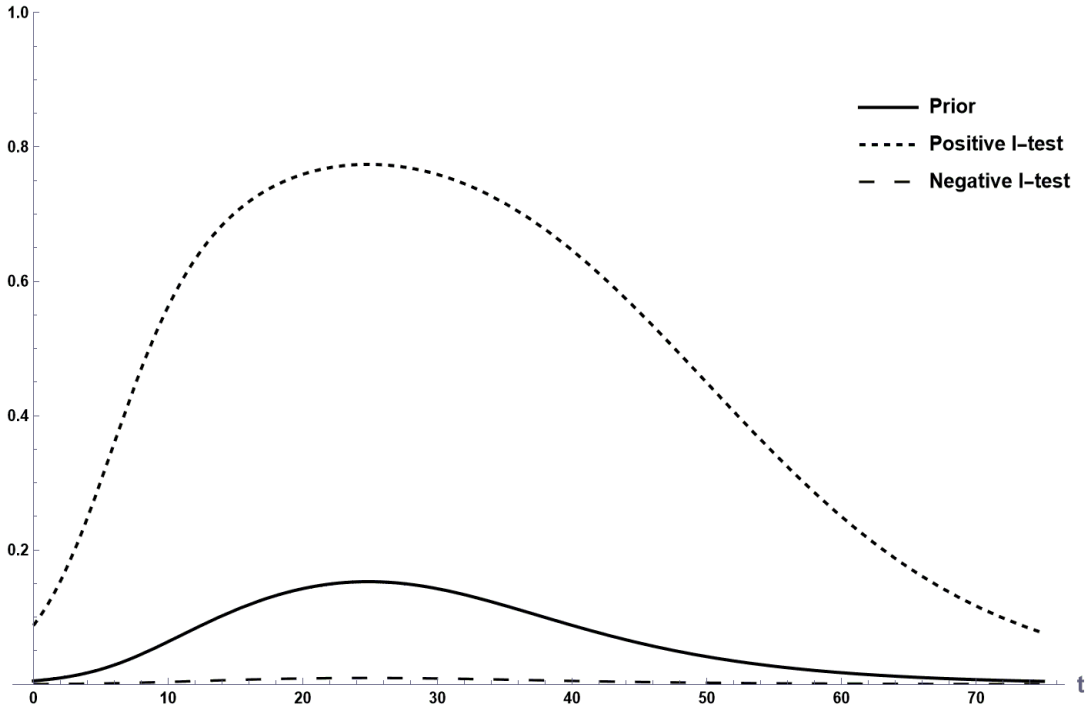

Figure 8: Positive and negative predictive values of tests for infection. Graph shows pre-test probabilities and corresponding posterior beliefs after positive and negative test results at different stages of epidemic.

of being in state $k = I, R$ is

$$\begin{aligned} p_k^1(t) &\equiv \Pr(\theta_k(t) = 1 | m_k(t) = 1) \\ &= \frac{\Pr(\theta_k(t) = 1 \cap m_k(t) = 1)}{\Pr(m_k(t) = 1)} \qquad (35) \\ &= \frac{\Pr(m_k(t) = 1 | \theta_k(t) = 1) \Pr(\theta_k(t) = 1)}{\Pr(m_k(t) = 1)} \\ &= \frac{q_k \Pr(\theta_k(t) = 1)}{q_k \Pr(\theta_k(t) = 1) + (1 - q_k)(1 - \Pr(\theta_k(t) = 1))} \qquad (36) \end{aligned}$$

Similarly, the posterior probabilities after receiving a negative result $m_k(t) = 0$ on a k-test are

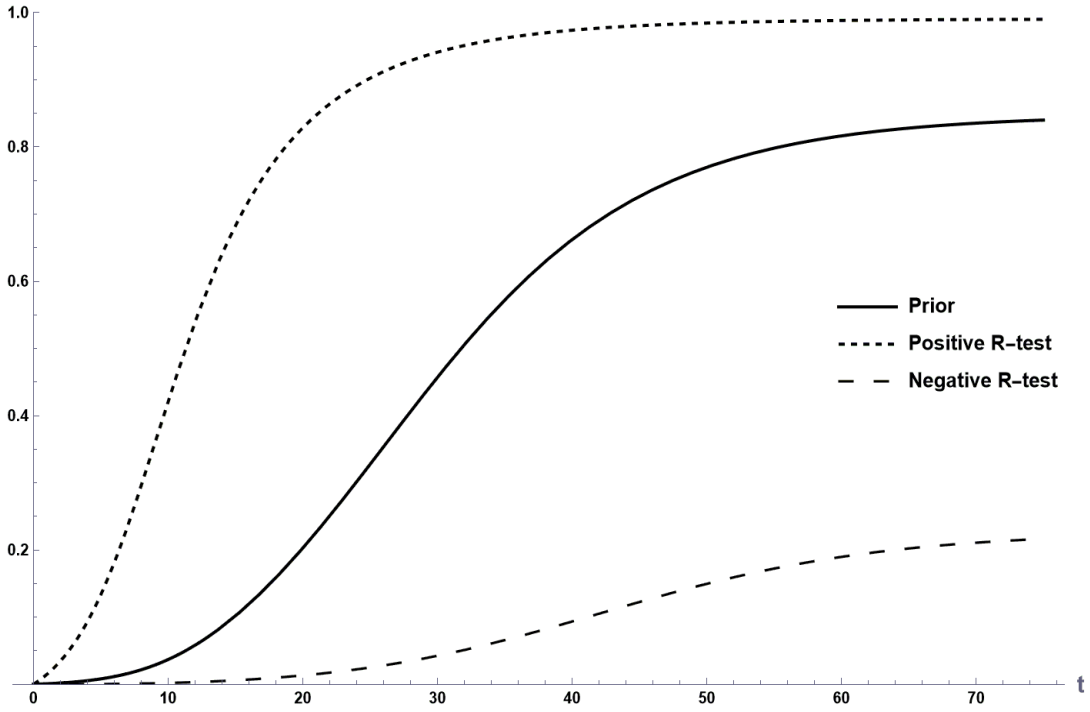

Figure 9: Positive and negative predictive values of tests for immunity. Graph shows pre-test probabilities and corresponding posterior beliefs after positive and negative test results at different stages of epidemic.

$$\begin{aligned} p_k^0(t) &\equiv \Pr(\theta_k(t)=1|m_k(t)=0) \\ &= \frac{\Pr(\theta_k(t)=1 \cap m_k(t)=0)}{\Pr(m_k(t)=0)} \qquad (37) \\ &= \frac{\Pr(m_k(t)=0|\theta_k(t)=1)\Pr(\theta_k(t)=1)}{\Pr(m_k(t)=0)} \\ &= \frac{(1-q_k)\Pr(\theta_k(t)=1)}{(1-q_k)\Pr(\theta_k(t)=1)+q_k(1-\Pr(\theta_k(t)=1))} \qquad (38) \end{aligned}$$

Since beliefs are a martingale, we know that

$$\left[p_k(t)q_k+(1-p_k(t))(1-q_k)\right]p_k^1(t)+\left[(1-p_k(t))q_k+p_k(t)(1-q_k)\right]p_k^0(t)=p_k(t) \qquad (39)$$

It should be noted that the inference drawn from different test results varies with the state of the epidemic, because the state determines the pre-test probabilities of being in different classes. Figures 8 and 9 compare the pre-test probabilities with the posterior beliefs after positive and negative tests for infection and immunity, respectively, at different points on the equilibrium path of the epidemic. As would be expected, a positive test

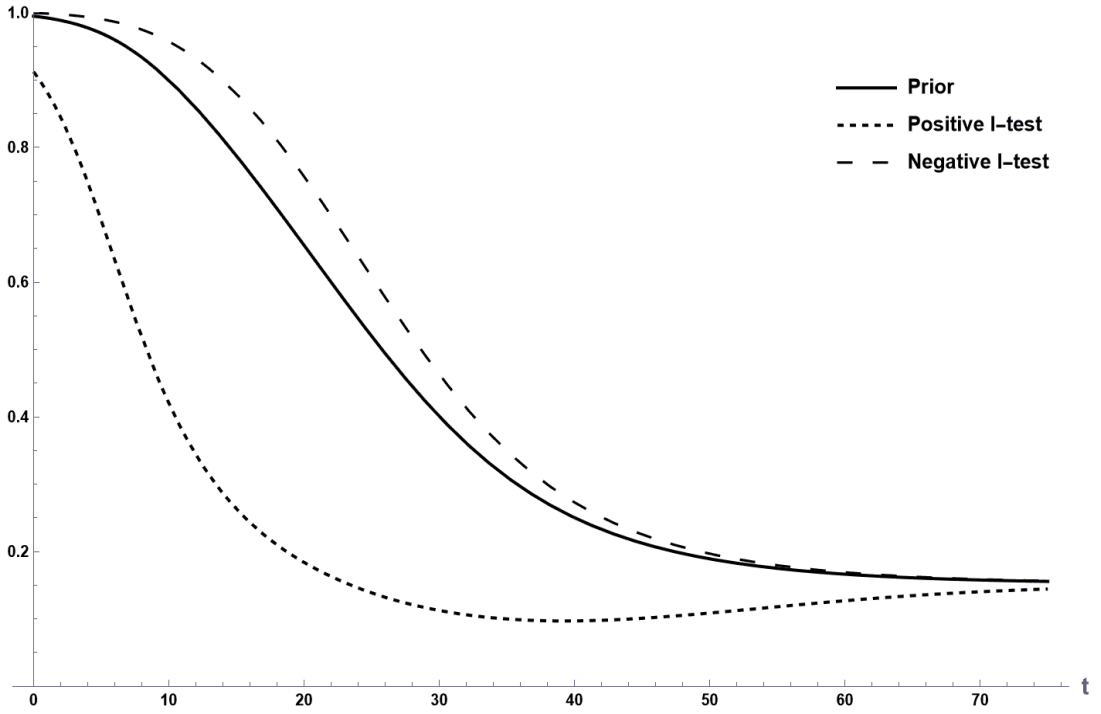

Figure 10: Positive and negative predictive values of susceptibility of tests for infection. Graph shows pre-test probabilities and corresponding posterior beliefs after positive and negative test results at different stages of epidemic.

increases the posterior, while a negative test decreases it. Having said that, the extent to which the posteriors differ from the prior clearly depends on the prior, which in turn varies greatly as the epidemic progresses. E.g., a positive test result for infection has only moderate effects on beliefs at early and late stages of the epidemic, but significant effects at intermediate stages, because disease prevalence is hump-shaped and thus the pre-test probability of infection is highest at intermediate stages. Similarly, testing negative for immunity towards the tail-end of the epidemic moves beliefs considerably, because by then the individual's pre-test beliefs of being recovered but asymptomatic are high.

To trace the effects that testing has on individuals' behavior, we need to determine the information content of different tests. Recall that there is no direct test for susceptibility but that tests for infection and immunity are indirectly informative about the extent to which an individual is at risk. It is therefore useful to distinguish between *test states* and *target states*. The test state is the state that we are testing for, e.g. infection or immunity. The target state is the state we want to make inferences about and this can coincide or be different from the test state. In the previous two formulae, the states coincided and we found the predictive value of tests for infection and immunity. We now consider the equivalent formulae for tests where the target state and the test state are different.

For some state $h \neq k$, the posterior beliefs after a negative result on a $k = I, R$ test

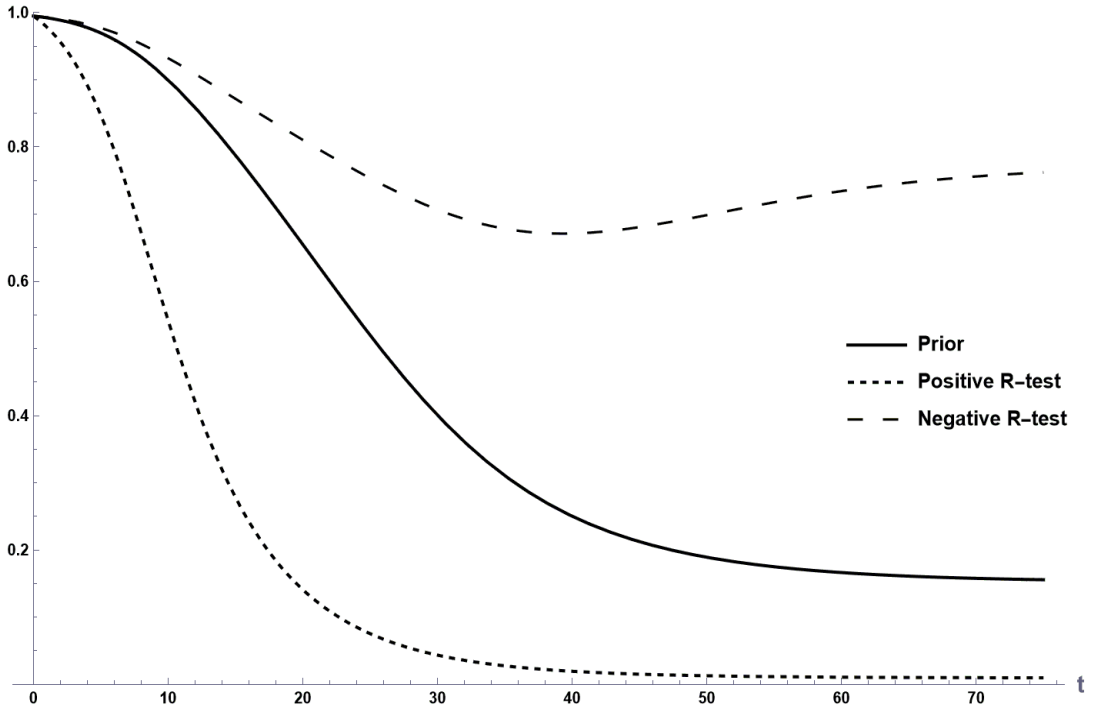

Figure 11: Positive and negative predictive values of susceptibility of tests for immunity. Graph shows pre-test probabilities and corresponding posterior beliefs after positive and negative test results at different stages of epidemic.

are

$$
\begin{aligned}
p_{hk}^{10}(t) &\equiv \Pr(\theta_h(t)=1|m_k(t)=0) && (40)\\
&= \frac{\Pr(m_k(t)=0|\theta_h(t)=1)\Pr(\theta_h(t)=1)}{\Pr(m_k(t)=0)} && (41)\\
&= \frac{q_k \Pr(\theta_h(t)=1)}{q_k \Pr(\theta_k(t)=0)+(1-q_k)(1-\Pr(\theta_k(t)=0))} > \Pr(\theta_h(t)=1) && (42)
\end{aligned}
$$

This inequality means that when an individual tests negative for being in a state k, then the posterior probability assigned to being in state $h \neq k$ increases and is proportional to the prior probability of being in state h.

Similarly, beliefs after a positive result on a $k = I, R$ are

$$
\begin{aligned}
p_{hk}^{11}(t) &\equiv \Pr(\theta_h(t)=1|m_k(t)=1) && (43)\\
&= \frac{\Pr(m_k(t)=1|\theta_h(t)=1)\Pr(\theta_h(t)=1)}{\Pr(m_k(t)=1)} && (44)\\
&= \frac{(1-q_k)\Pr(\theta_h(t)=1)}{q_k \Pr(\theta_k(t)=1)+(1-q_k)(1-\Pr(\theta_k(t)=1))} < \Pr(\theta_h(t)=1) && (45)
\end{aligned}
$$

Figures 10 and 11 show the pre-test probabilities and posterior beliefs for susceptibility

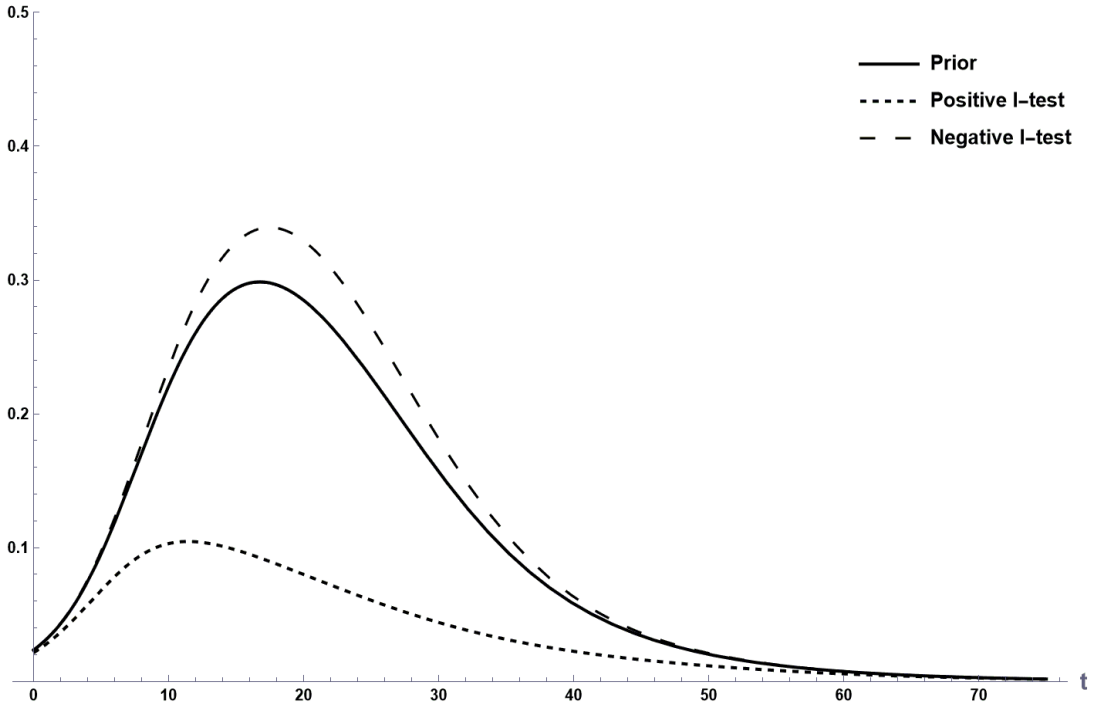

Figure 12: Best responses after tests for infection. Graph shows pre-test best response and corresponding best responses after positive and negative test results at different stages of epidemic.

after negative and positive tests for infection and immunity, respectively, at different points on the equilibrium path of the epidemic. Because the test states and the target states no longer coincide with these tests, the interpretation of these graphs is slightly different from the ones showing predictive values. A positive test result for either infection or immunity causes the posterior probability of susceptibility to *decrease*, whereas a negative result on either of those tests causes the posterior probability of susceptibility to *increase*.

It is noteworthy that because there are three health states, the informativeness of a test for one test state varies not only with the pre-test probability of the target state, but also on the prior for the residual state (see Birkett, 1988). This causes non-monotonicities seen in Figures 10 and 11.

5. Private Demand for Tests and the Value of Information

When individuals are potentially asymptomatic, they may remain uncertain about their health status for prolonged periods of time unless they experience unambiguous symptoms showing infection. For this reason, the model is ideally suited for analyzing the desirability and effects of diagnostic tests. In this section, I do so in two different settings, namely individual testing and mass testing.

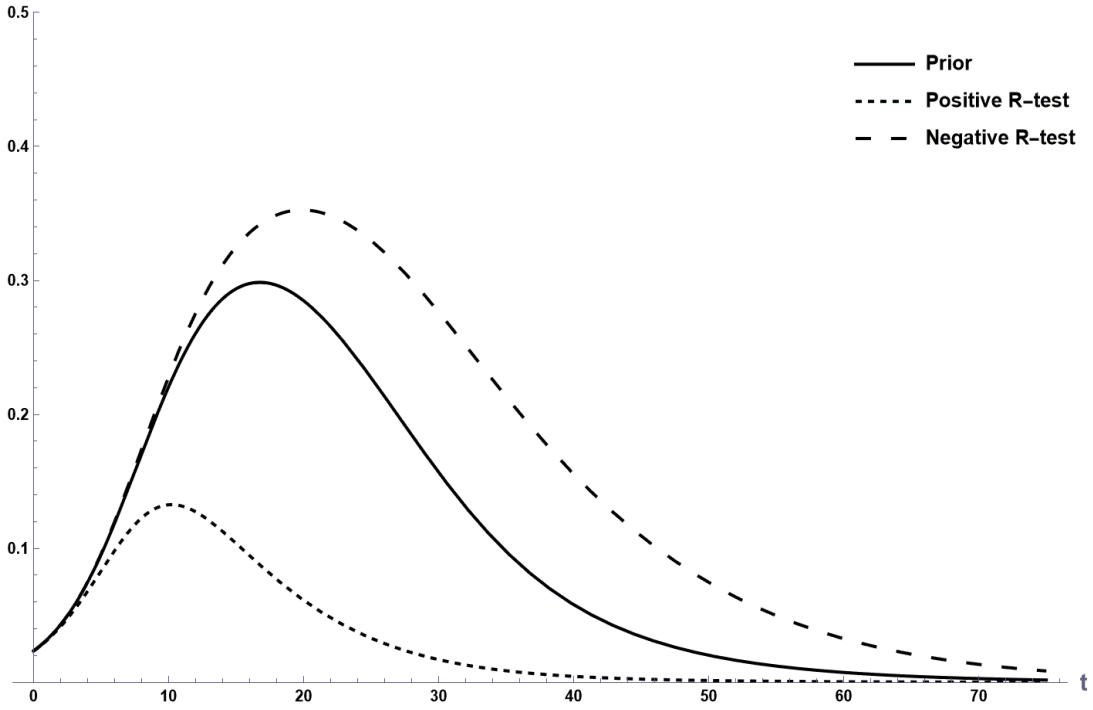

Figure 13: Best responses after tests for immunity. Graph shows pre-test best response and corresponding best responses after positive and negative test results at different stages of epidemic.

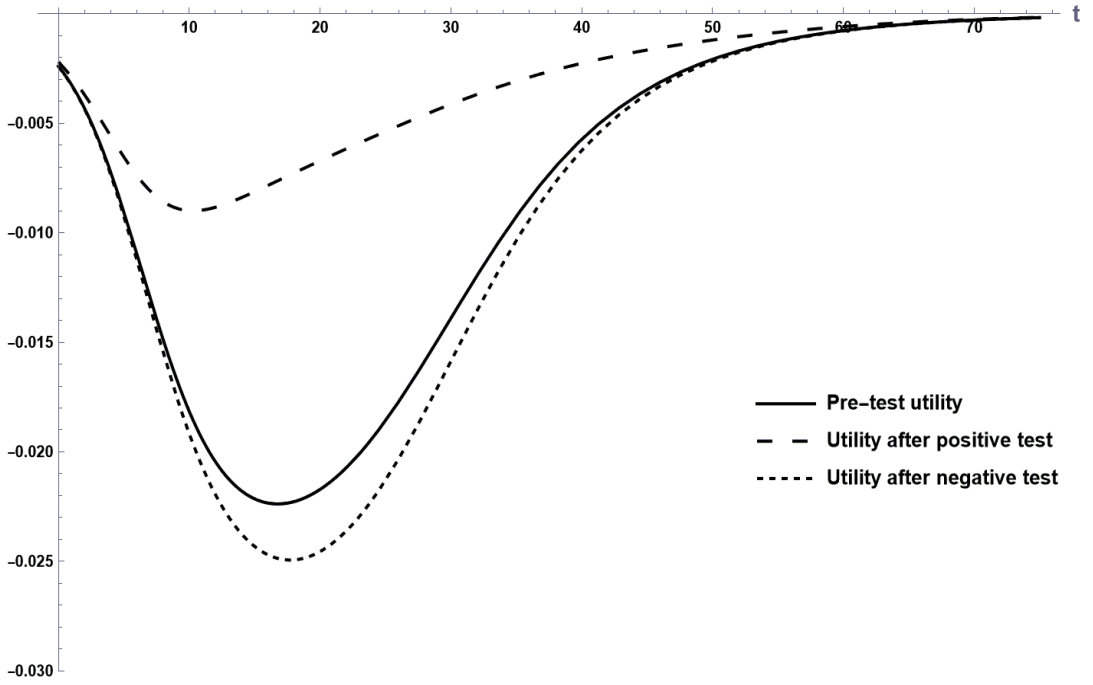

Figure 14: Expected utilities after tests for infection. Graph shows pre-test utility and corresponding utilities after positive and negative test results at different stages of epidemic.

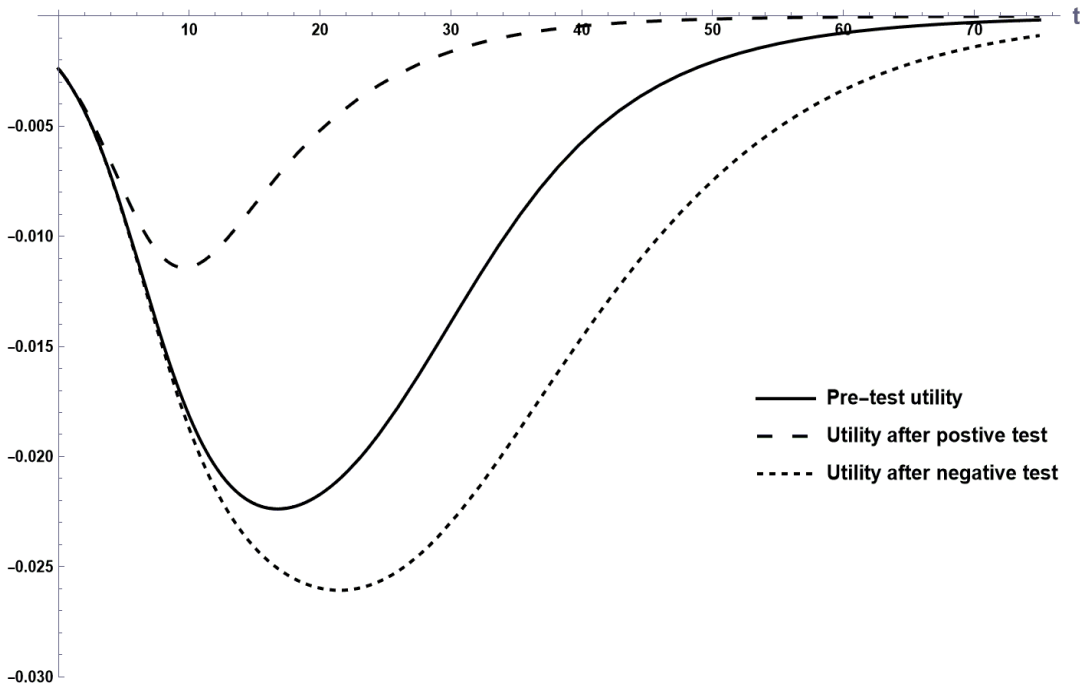

Figure 15: Expected utilities after tests for immunity. Graph shows pre-test utility and corresponding utilities after positive and negative test results at different stages of epidemic.

5.1. Individual Testing. Individual mitigation behavior is driven by infection risks and thus intimately tied to perceptions of susceptibility. Diagnostic testing is a key tool to improve such information and can therefore help guide people's decisions. For that reason, I now analyze how test results can alter behavior and how such changes can make testing valuable in the first place.

To this end, recall that for arbitrary social distancing decision $d_i(t)$ and "personal" beliefs $\hat{p}_S(t)$, an individual's utility at some point $t \geq 0$ on the equilibrium path is

$$U(d_i(t), \hat{p}_S(t)) = -(1 - d_i(t))\hat{p}_S(t)(1-\alpha)\phi^*(t)\beta I(t)(\overline{\pi} - \underline{\pi}) - c \times \frac{d_i(t)^2}{2} \tag{46}$$

where the force of infection effect is the equilibrium amount

$$\phi^*(t) = [1 - \alpha(1 - \sigma(1 - d^*(t)))] \tag{47}$$

$$d^*(t) = \frac{(1 - \alpha(1-\sigma))(1-\alpha)S(t)\beta I(t)(\overline{\pi} - \underline{\pi})}{c\alpha + (1-\alpha)(c - \alpha\beta(\overline{\pi} - \underline{\pi})\sigma I(t))S(t)} \tag{48}$$

Here, the aggregate social distancing is evaluated at the "aggregate" beliefs

$$p_S(t) = \frac{S(t)}{S(t) + \alpha(1 - S(t))} \tag{49}$$

I distinguish between personal and aggregate beliefs so that I can consider the effects on behavior of only a single individual who gets tested.

Denote by $d_i(\hat{p}_S(t))$ the privately optimal decision of an individual with beliefs $\hat{p}_S(t)$. Figures 12 and 13 compare the best responses after positive and negative results on tests for infection and immunity, respectively, together with the pre-test best responses, at different stages of the epidemic. Since mitigation efforts are monotone in perceived susceptibility, the shifts in beliefs evident in Figures 10 and 11 are mirrored in the post-test best responses. In turn, these changes in behavior, prompted by information gained from the tests, are reflected in corresponding changes in post-test expected utilities, as shown in Figures 14 and 15. These changes in information, best responses and post-test utilities can be valued ex ante.

At the moment of taking a k-test, the expected utility of the individual is

$$V_T \equiv \Pr(m_k(t)=1)U(d_i(p_S^1(t)),p_S^1(t)) + \Pr(m_k(t)=0)U(d_i(p_S^0(t)),p_S^0(t)) \tag{50}$$

where

$$\Pr(m_k(t) = 1) = p_k(t)q_k + (1-p_k(t))(1-q_k) \tag{51}$$

$$\Pr(m_k(t) = 0) = (1-p_k(t))q_k + p_k(t)(1-q_k) \tag{52}$$

are the ex ante probabilities of receiving positive and negative results on a k-test, respectively. Let the no-test expected utility be

$$V_N \equiv U(d_i(p_S(t)),p_S(t)) \tag{53}$$

The value of information of a k-test is calculated as

$$V_T - V_N \geq 0 \tag{54}$$

Note that this value is a function of what is being tested for (i.e. whether the test detects infection or immunity), the aggregate state of the system $(S(t),I(t),R(t))$ and of the population-wide contemporaneous social distancing $d^*(t)$. Figure 16, shows the value of information for a test for infection and immunity, respectively. The figure has several noteworthy features. First, since the value of information derives from the potential changes that new information induce in behavior, the value of information of tests for infection and immunity are not necessarily highest when there is the highest pre-test levels of mitigation. Rather, the values are highest when the difference in the post-test mitigation levels are highest, as can be verified in Figures 12 and 13 for infection and immunity tests, respectively. Second, except at early stages of the epidemic, the value

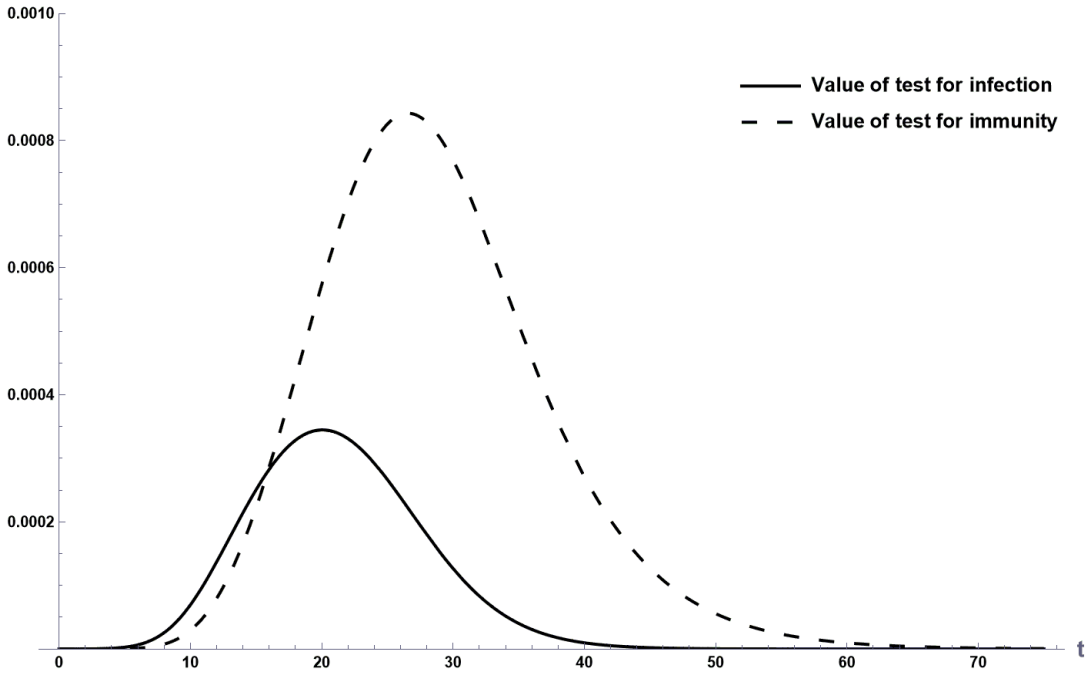

Figure 16: Value of information of tests for infection or immunity at different stages of epidemic.

of information from a test of immunity is significantly higher than the value of a test of infection. Furthermore, the value of the former reaches its highest point later in the epidemic. Here it should be recalled that in these plots, the tests for infection and immunity are equally precise.

5.2. Mass Testing. To this point, I have considered only the testing of a single individual. This allowed me to focus on the effects of tests on the behavior of the individual, while sidestepping the issue of how tests may affect aggregate behavior and thus the overall dynamics of the epidemic. In this section, I take a different perspective and ask what the aggregate effects of mass testing are. While the effects will depend on the exact time-path of aggregate testing, I will illustrate the basic principles by way of a simple thought experiment. In particular, I will trace the effects on disease incidence at a given point in time of mass diagnostic tests and determine how these effects vary with the state of the epidemic.

For individual testing, the sensitivity and specificity of diagnostic tests influences the value of information, but these properties have no effect on aggregate disease dynamics in this case. This is no longer true under mass testing. As there is a continuum of individuals in each class (susceptible, infected, recovered), this means that for a k-test with precision q_k, a fraction q_k of individuals who are actually in this class will test positive (true positives), while the remaining fraction $(1 - q_k)$ will test negative (false negatives).

I-test	True positives	False negatives	False positives	True negatives
Measure	$\alpha I(t)q_I$	$\alpha I(t)(1-q_I)$	$S(t)(1-q_I)$	$S(t)q_I$
Effect	$+$	$-$	$+$	$-$

Table 4: Effects of test for infection on incidence.

Similarly, for those individuals who are tested but who are not actually in this class, $(1-q_k)$ will test positive (false positives) while a fraction q_k will test negative (true negatives). This means that to understand the aggregate effects of mass tests, we need to keep track both of the precision of the tests and of the magnitude of the subpopulation that is submitted to the test. The latter is always going to be those individuals who have never shown any symptoms, which we have determined have measure

$$S(t) + \alpha \left[I(t) + R(t) \right] \tag{55}$$

Mass tests for infection. Suppose that mass testing for infection is performed on all symptom-naive individuals at some moment $t \geq 0$. Equilibrium disease incidence will then be

$$\begin{aligned} \dot{I}(t) &= \beta I(t) S(t) \left(1 - q_I d(p_{SI}^{11}(t)) - (1-q_I) d(p_{SI}^{10}(t)) \right) \\ &\quad \times \left(1 - \alpha \left\{ 1 - \sigma \left[1 - (1-q_I) d(p_{SI}^{11}(t)) - q_I d(p_{SI}^{10}(t)) \right] \right\} \right) \end{aligned} \tag{56}$$

where p_{hk}^{ij} is the posterior belief for realization $i = 0, 1$ of target state $h \neq k$ after result $j = 0, 1$ on a k-test.

This expression is the extension of the law of motion (22) to a population with heterogeneous beliefs. What happens with mass testing is that rather than having one homogeneous naive class who all mitigate at the common level $d^*(t)$, the tests create two classes with different posterior beliefs (because some test positive while others test negative) and hence with different ex post best responses. Depending on whether a given individual is actually susceptible or infected, these different best responses lead to different changes in behavior in different directions, the impact of which is determined by the magnitude of the different segments of the population in each situation. The magnitudes are in turn determined by the state of the epidemic and on the properties of the tests. Table 4 summarizes the different effects for the case of a test for infection.

It is useful to think of the changes in aggregate behavior due to mass testing in terms of the fatalism and force of infection effects, discussed in Section 4.2. Those who get true positive results are in fact infected and will now become confident that they are no longer

at risk. They will therefore increase their activity level and increase transmission, all else equal. In contrast, those receiving false negative results are also infected, but they now think it even more likely that they are susceptible, thereby increasing social distancing and in effect reducing transmission. Similarly, those who receive false positive results are in fact susceptible but think they're not, thus lowering their guard. Last, people receiving true negative results are at risk and have their beliefs reinforced, therefore increasing protection. What this means is that when mass tests are rolled out, there are a myriad different, offsetting effects on behavior and this creates an ambiguous effect on the aggregate number of matches between infected and susceptible individuals. As a consequence, disease incidence can in principle increase or decrease, depending on parameters and on the state of the epidemic. In addition, note that because each individual's best response is a function of contemporaneous aggregate social distancing in the population, individuals must also trace the effects listed in the table.

To find exact values for the ex-post equilibrium mitigation levels $(d(p_{SI}^{11}(t)), d(p_{SI}^{10}(t)))$, I solve the system of best responses for the two test outcomes in symmetric equilibrium, namely

$$\begin{aligned} d(p_{SI}^{11}(t)) &= p_{SI}^{11}(t)(1-\alpha)\beta I(t)\left(\frac{\overline{\pi}-\underline{\pi}}{c}\right) && (57)\\ &\quad\times\left(1-\alpha\left\{1-\sigma\left[1-(1-q_I)d(p_{SI}^{11}(t))-q_I d(p_{SI}^{10}(t))\right]\right\}\right) && (58)\\ d(p_{SI}^{10}(t))) &= p_{SI}^{10}(t))(1-\alpha)\beta I(t)\left(\frac{\overline{\pi}-\underline{\pi}}{c}\right) && (59)\\ &\quad\times\left(1-\alpha\left\{1-\sigma\left[1-(1-q_I)d(p_{SI}^{11}(t))-q_I d(p_{SI}^{10}(t))\right]\right\}\right) && (60)\end{aligned}$$

These best responses are the equivalent of (29), corrected for heterogeneous ex post beliefs and an amended force of infection effect. The solution to this system is the pair of equilibrium social distancing levels

$$\begin{aligned} d(p_{SI}^{11}(t)) &= \frac{p_{SI}^{11}(t)(1-\alpha)\beta I(t)(1-\alpha(1-\sigma))(\overline{\pi}-\underline{\pi})}{c+\left[(1-q_I)p_{SI}^{11}(t)+q_I p_{SI}^{10}(t)\right]\alpha\sigma(1-\alpha)\beta I(t)(\overline{\pi}-\underline{\pi})} && (61)\\ d(p_{SI}^{10}(t)) &= \frac{p_{SI}^{10}(t)(1-\alpha)\beta I(t)(1-\alpha(1-\sigma))(\overline{\pi}-\underline{\pi})}{c+\left[(1-q_I)p_{SI}^{11}(t)+q_I p_{SI}^{10}(t)\right]\alpha\sigma(1-\alpha)\beta I(t)(\overline{\pi}-\underline{\pi})} && (62)\end{aligned}$$

Figure 17 compares disease incidence at different stages of the equilibrium path of the epidemic, before and after a mass test for infection. Under the benchmark parameterization, mass testing for infection increases disease incidence at any stage of the epidemic. Note however that this does *not* mean that the overall effect of mass testing is an upward shift in infection throughout the epidemic, for it is a statement only about disease incidence at a moment in time. As is known from the control of SIR type diseases, mitigation that

R-test	False positives	True negatives
Measure	$(1-q_R)\left[S(t)+\alpha I(t)\right]$	$q_R\left[S(t)+\alpha I(t)\right]$
Effect	$+/-$	$+/-$

Table 5: Effects of test for immunity on incidence.

suppresses infections early on may cause increases in infections later in the epidemic.

Mass tests for immunity. Under the assumption of density dependent transmission $\beta I(t)S(t)$, tests may influence the behavior of asymptomatically recovered people, of which there are $\alpha R(t)$. But these changes in behavior have no effects on aggregate disease incidence and I can therefore ignore the effects on those who receive true positive or false negative results on a test for immunity. Table 5 shows the effects in the case of a test for immunity.

As can be seen, the effects are somewhat simpler than those under a mass test for infection, yet the aggregate effects on disease incidence are still ambiguous.

When there is mass testing for immunity of all symptom-naive individuals at some moment $t \geq 0$, disease incidence is given by

$$\begin{aligned}\dot{I}(t) &= \beta I(t)S(t)\left(1-(1-q_R)d(p^{11}_{SR}(t))-q_R d(p^{10}_{SR}(t))\right)\\ &\quad\times\left(1-\alpha\{1-\sigma\left[1-(1-q_R)d(p^{11}_{SR}(t))-q_R d(p^{10}_{SR}(t))\right]\}\right) \qquad (63)\end{aligned}$$

To find the exact values for the ex-post equilibrium mitigation levels $(d(p^{11}_{SR}(t)), d(p^{10}_{SR}(t)))$, I solve the system of best responses for the two test outcomes in symmetric equilibrium, namely

$$\begin{aligned}d(p^{11}_{SR}(t)) &= p^{11}_{SR}(t)(1-\alpha)\beta I(t)\left(\frac{\overline{\pi}-\underline{\pi}}{c}\right) \qquad (64)\\ &\quad\times\left(1-\alpha\left\{1-\sigma\left[1-(1-q_R)d(p^{11}_{SR}(t))-q_R d(p^{10}_{SR}(t))\right]\right\}\right) \qquad (65)\\ d(p^{10}_{SR}(t))) &= p^{10}_{SR}(t))(1-\alpha)\beta I(t)\left(\frac{\overline{\pi}-\underline{\pi}}{c}\right) \qquad (66)\\ &\quad\times\left(1-\alpha\left\{1-\sigma\left[1-(1-q_R)d(p^{11}_{SR}(t))-q_R d(p^{10}_{SR}(t))\right]\right\}\right) \qquad (67)\end{aligned}$$

The solution to this system is the pair of equilibrium social distancing levels

$$\begin{aligned}d(p^{11}_{SR}(t)) &= \frac{p^{11}_{SR}(t)(1-\alpha)\beta I(t)(1-\alpha(1-\sigma))(\overline{\pi}-\underline{\pi})}{c+\left[(1-q_R)p^{11}_{SR}(t)+q_R p^{10}_{SR}(t)\right]\alpha\sigma(1-\alpha)\beta I(t)(\overline{\pi}-\underline{\pi})} \qquad (68)\\ d(p^{10}_{SR}(t)) &= \frac{p^{10}_{SR}(t)(1-\alpha)\beta I(t)(1-\alpha(1-\sigma))(\overline{\pi}-\underline{\pi})}{c+\left[(1-q_R)p^{11}_{SR}(t)+q_R p^{10}_{SR}(t)\right]\alpha\sigma(1-\alpha)\beta I(t)(\overline{\pi}-\underline{\pi})} \qquad (69)\end{aligned}$$

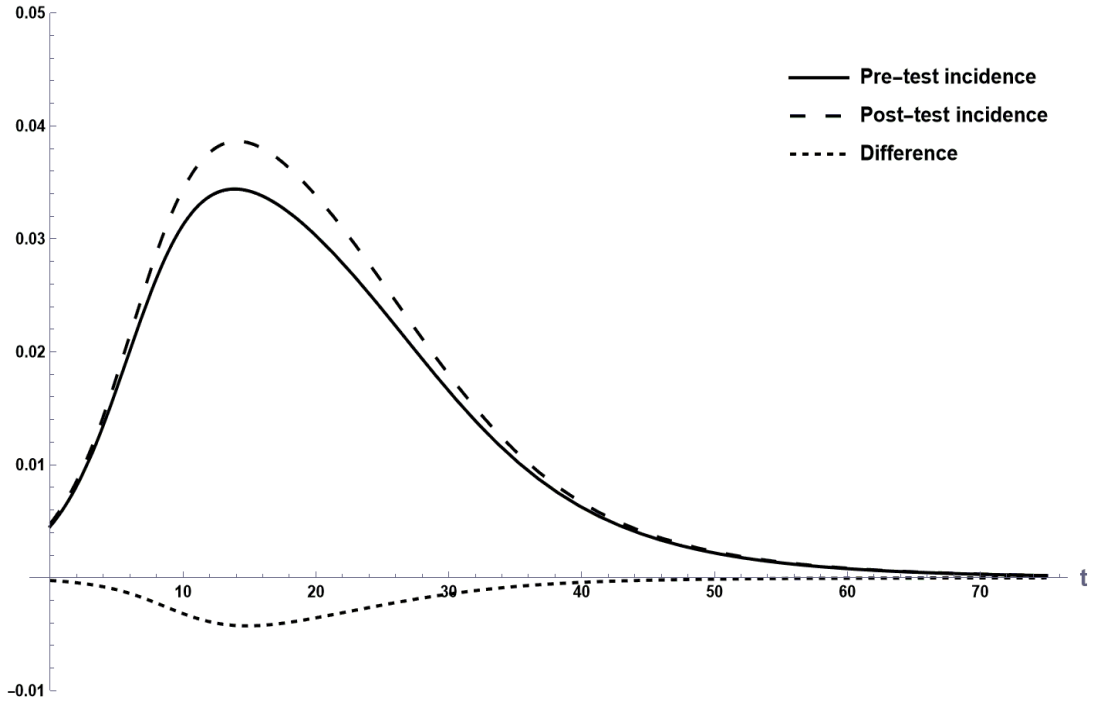

Figure 17: Disease incidence after mass testing for infection at different stages of epidemic.

Figure 18 compares disease incidence at different stages of the equilibrium path of the epidemic, before and after a mass test for immunity. In this case, I find that mass testing for immunity may reduce or increase disease incidence, depending on the stage of the epidemic.What this analysis makes clear is that mass testing cannot be seen simply as a means to gauge the state of the epidemic and to identify who is in which health state. The mere act of mass testing, to the extent that the results are communicated to people and these are allowed to change behavior in response, can itself influence disease dynamics and change the situation on the ground. As a consequence, rollouts of mass testing must be carefully considered on a par with other public health interventions such as mass vaccination or generalized lockdowns..

6. Forward-Looking Behavior

The main analysis has been conducted under the assumption of myopic decision making by individuals. This restriction was made purely for convenience, as it allows me to get closed-form solutions for equilibrium behavior and straightforward comparative statics results. Assuming instead that individuals are fully forward-looking will introduce additional considerations into decision making, in particular intertemporal tradeoffs known from complete information models like those surveyed in the literature review. Yet the basic insights from the myopic setting remain valid under forward-looking behavior. To show this, I will in this section set out the problem of a representative forward-looking

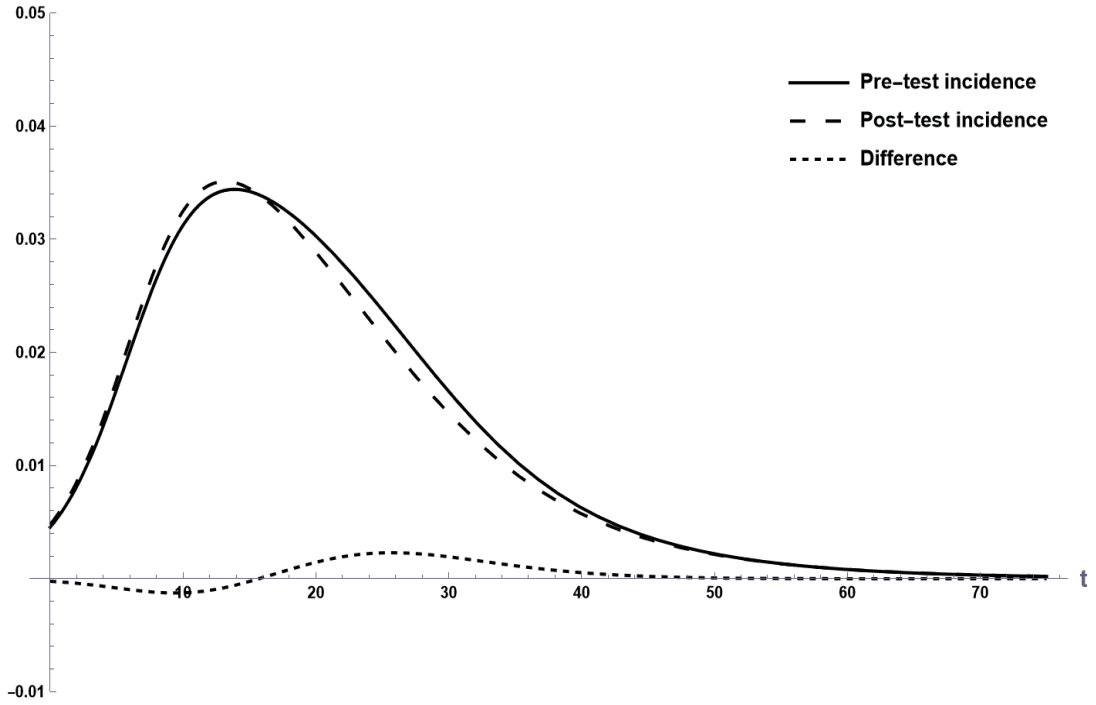

Figure 18: Disease incidence after mass testing for immunity at different stages of epidemic.

individual and derive the best response function. I show that the nature of this best response function closely mirrors that of the myopic individual, relative to these individuals' complete information equivalents in the benchmark models with no asymptomatic infection.

To consider forward-looking behavior, I restrict attention to so-called open-loop strategies in which individuals only condition their decisions on time. This amounts to considering only passive learning whereby individuals do not intentionally deviate from their best responses in order to experiment.

Assume that individuals discount the future at rate $\rho > 0$. The individual's maximization problem is then

$$\max_{d_i(t)\in[0,1]} \int_0^\infty e^{-\rho t} \left\{ p_S(t) \left[\overline{\pi} - c \times \frac{d_i(t)^2}{2} \right] + p_I(t)\underline{\pi} + p_R(t)\overline{\pi} \right\} dt \tag{70}$$

where the state variables $p_j(t)$, $j = \mathcal{S}, \mathcal{I}, \mathcal{R}$ are the health state probabilities of the individual at time t. The individual's problem is solved subject to the following system

of differential equations for the "individual" state variables:

$$\dot{p}_S(t) = -(1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t) \quad (71)$$

$$\dot{p}_I(t) = (1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t) - \gamma p_I(t) \quad (72)$$

$$\dot{p}_R(t) = \gamma p_I(t) \quad (73)$$

In addition to these laws of motion, equations for the evolution of the aggregate state variables (21)-(23) complete the description of the individual's problem.

To make a direct comparison between the best responses under forward-looking decision making and under myopic decision-making, the payoffs from infection $\underline{\pi}$ in the latter setting must be replaced by some value

$$\underline{\pi}' \equiv \frac{1}{\rho+\gamma}\left[\underline{\pi} + \gamma\frac{\overline{\pi}}{\rho}\right] \quad (74)$$

The value $\underline{\pi}'$ is simply the expected discounted lifetime utility of an individual in the infected state and can be understood as follows.[15] Once infected, the individual experiences flow utility $\underline{\pi}$ until he or she recovers. From then on, the individual earns flow utility $\overline{\pi}$ in perpetuity. The recovery date is governed by a Poisson process with rate γ and cannot be influenced by the individual. Last, observe that

$$\lim_{\gamma\to 0}\underline{\pi}' = \frac{\overline{\pi}}{\rho}, \quad \lim_{\gamma\to\infty}\underline{\pi}' = \frac{\overline{\pi}}{\rho} \quad (75)$$

It is worth emphasizing that under decentralized decision making, each individual takes the aggregate dynamics as given and chooses a path of social distancing in order to maximize his or her individual expected discounted utility. The outcome is thus one of perfect foresight equilibrium, in which the aggregate dynamics that the individuals anticipate when choosing their social distancing policies actually materializes.

Let $\lambda_j(t)$ denote the "individual" costate variables for the state variables $p_j(t)$, $j = \mathcal{S}, \mathcal{I}, \mathcal{R}$. Then the individual's current-value Hamiltonian is given by

$$H = p_S(t)[\overline{\pi} - c \times d_i(t)^2/2] + p_I(t)\underline{\pi} + p_R(t)\overline{\pi} \quad (76)$$

$$-\lambda_S(t)(1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t) \quad (77)$$

$$+\lambda_I(t)[(1-d_i(t))p_S(t)(1-\alpha)\phi(t)\beta I(t) - \gamma p_I(t)] \quad (78)$$

$$+\lambda_R(t)\gamma p_I(t) \quad (79)$$

[15] See Toxvaerd (2020) for a derivation.

A necessary condition for individual maximization is that

$$\frac{\partial H}{\partial d_i(t)} = -p_S(t)cd_i(t) + p_S(t)(1-\alpha)\phi(t)\beta I(t)[\lambda_S(t) - \lambda_I(t)] = 0 \tag{80}$$

which can be re-written as

$$d_i(t) = p_S(t)(1-\alpha)\phi(t)\beta I(t)\left(\frac{\lambda_S(t) - \lambda_I(t)}{c}\right) \tag{81}$$

$$d_i(t) = p_S(t)(1-\alpha)\phi(t)\beta I(t)\left(\frac{\overline{\pi} - \underline{\pi}}{c}\right) \tag{82}$$

The costate variables satisfy the laws of motion

$$\begin{aligned}
\dot{\lambda}_S(t) &= \lambda_S(t)\rho - \frac{\partial H}{\partial p_S(t)} && (83)\\
&= \lambda_S(t)\left[\rho + (1-d_i(t))(1-\alpha)\phi(t)\beta I(t)\right] - \lambda_I(t)(1-d_i(t))(1-\alpha)\phi(t)\beta I(t) \\
&\quad - \left[\overline{\pi} - c \times d_i(t)^2/2\right] && (84)\\
\dot{\lambda}_I(t) &= \lambda_I(t)\rho - \frac{\partial H}{\partial p_I(t)} && (85)\\
&= \lambda_I(t)\left[\rho + \gamma\right] - \lambda_R(t)\gamma - \underline{\pi} \\
\dot{\lambda}_R(t) &= \lambda_R(t)\rho - \frac{\partial H}{\partial p_R(t)} = \lambda_R(t)\rho - \overline{\pi} && (86)
\end{aligned}$$

Note that relative to the best response under myopic decision-making (27), reproduced here for reference, the only difference is that in the best response (81) under forward-looking behavior, the constant health premium $(\overline{\pi} - \underline{\pi})$ is replaced by the time-varying equivalent $(\lambda_S(t) - \lambda_I(t))$. We know from e.g. Makris and Toxvaerd (2021) that in equilibrium, $\beta I(t)(\lambda_S(t) - \lambda_I(t))$ is hump-shaped and so the qualitative properties of equilibrium mitigation are robust to this extension. Furthermore, it is still true under forward-looking behavior that an individual's beliefs about susceptibility $p_S(t)$ are monotone decreasing.

7. DISCUSSION

In this paper, I introduced a parsimonious setting in which the effects of asymptomatic infection on equilibrium behavior can be fruitfully analyzed. In addition, I use the model to analyze the demand for and effects of individual and mass testing, both on individual and on aggregate behavior and disease dynamics. While the analysis has been kept as simple as possible for tractability, the main ideas and tools extend straightforwardly to more complicated and realistic settings, such as forward-looking behavior and the possibility of disease-induced mortality. Some enhancements to the disease model would be particularly interesting and these can also be made within the framework developed in this paper.

First, I have for simplicity assumed that once infected, symptomatic individuals show symptoms without delay. In practice, many infected individuals are presymptomatic, i.e. they become aware that they're infected only after some time. This extension seems worthwhile pursuing, especially for future quantitative work.

Second, I have assumed that once recovered, individuals become permanently immune to further infection. We now know that diseases such as COVID-19 have waning immunity, which changes the underlying disease dynamics in interesting ways (see e.g. Giannitsarou et al., 2022). The possibility of waning and/or imperfect immunity changes the analysis of belief formation because perceived susceptibility is then no longer monotone decreasing, as is the case in the present work based on the SIR model. With an SIRS setting where recovered people slowly move back to susceptibility, the beliefs of those who do not experience any symptoms may fluctuate over time. In addition, asymptomatic people may come to learn asymptotically that they are of the asymptomatic types, with knock-on effects on both behavior and aggregate dynamics. Similarly, if being of the asymptomatic type is a permanent property, then anyone who experiences symptoms at any time learn that they are of the symptomatic type going forward. This will also influence their future mitigation decisions.

Last, it should be acknowledged that an individual's incentives to test his or her health status may be different from those of a medical practitioner, a public health official or the government. In addition to the value of information to individuals from diagnostic testing, there are roughly three main reasons to test people:

(i) There may be a direct clinical benefit from testing, to help to determine the appropriate therapy. If therapy is available for the condition in question, then it can be applied as appropriate. Testing can also be used to exclude certain causes of symptoms and so other tests can be conducted to determine alternative causes. In the diagnostic testing literature, this is known as ruling in/ruling out.

(ii) Testing is an integral part of researching and learning about a disease, to better understand the medical issues involved and to understand the scale of the problem. E.g., testing can help researchers determine the infectiousness of the disease and also the asymptomatic ratio (see Nishiura et al., 2020 for an application to COVID-19).

(iii) Testing can help manage the epidemic through better targeting of policy measures, such as imposing curfews, lock-downs and quarantines, but also to project future demand for critical infrastructure such as hospitals and ICU capacity.

Including the value of these alternative objectives into the calculation seems worthwhile, but is not pursued in the present paper.

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