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The Hammer and the Scalpel: On the Economics of Indiscriminate versus Targeted Isolation Policies during Pandemics

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ABSTRACT

We develop a simple dynamic economic model of epidemic transmission designed to be consistent with widely used SIR biological models of the transmission of epidemics, while incorporating economic benefits and costs as well. Our main finding is that targeted testing and isolation policies deliver large welfare gains relative to optimal policies when these tools are not used. Specifically, we find that when testing and isolation are not used, optimal policy delivers a welfare gain equivalent to a 0.6% permanent increase in consumption relative to no intervention. The welfare gain arises because under the optimal policy, the planner engineers a sharp recession that reduces aggregate output by about 40% for about 3 months. This sharp contraction in economic activity reduces the rate of transmission and reduces cumulative deaths by about 0.1%. When testing policies are used, optimal policy delivers a welfare gain equivalent to a 3% permanent increase in consumption. The associated recession is milder in that aggregate output declines by about 15% and cumulative deaths are reduced by .3%. Much of this welfare gain comes from isolating infected individuals. When individuals who are suspected to be infected are isolated without any testing, optimal policy delivers a welfare gain equivalent to a 2% increase in permanent consumption.

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1. Introduction

Pandemics force households, firms, and governments to make cruel choices between unhappy alternatives. Much economic activity is enhanced by close person-to-person contact. Unfortunately, this kind of contact typically allows viruses to be more easily transmitted from person to person. Much of the economic literature on epidemics studies the trade-offs between the losses to economic activity associated with limiting contact and the gains from reduced transmission of the virus, including reduced healthcare costs, lower strains on hospitals, and fewer deaths. The response to the coronavirus epidemic in most Western countries has been to limit contacts by limiting economic activity. Some countries—most notably, South Korea, Singapore, Hong Kong, and Taiwan—have limited economic activity to a lesser extent and have supplemented the modest limitations with aggressive policies of targeted testing, contact tracing, and isolation. In this paper, we develop a version of a fairly standard macroeconomic model of epidemics, incorporate testing and isolation policies into it, and ask to what extent testing policies of targeted testing and isolation can achieve better outcomes. In a quantitative version of our model, we find that even a policy of targeted testing and isolation that is substantially less aggressive than that conducted in South Korea can yield substantial welfare gains. If testing and isolation policies are optimally designed, economic activity must be curtailed to a much more limited extent, and the number of deaths is substantially smaller than if testing and isolation policies are not available. We argue that relative to no testing, untargeted testing yields only modest benefits. We also argue that, even if testing resources are not available, targeted isolation without testing yields about two-thirds of the welfare gains from a targeted testing and isolation policy.

This paper develops a simple dynamic economic model of epidemic transmission. The model is designed to be consistent with widely used SIR biological models of the transmission of epidemics (see Atkeson (2020b) for a primer), while incorporating economic benefits and costs as well. We choose a formulation that makes it possible to analyze the benefits and costs of various policies. The main stand we take is that social proximity has benefits by allowing for economic activity to take place. We have in mind that certain types of production activities require groups of people to work in close proximity to one other. The obvious example of such an activity is assembly line production. In other activities, individuals derive value from

social proximity in consumption. Examples of such activities are watching live performances of plays or rock concerts. While substitutes are available for production and consumption with social proximity (artisanal production as opposed to assembly line production, or televised rock concerts versus live ones), revealed preference shows that people value social proximity in many aspects of production and consumption. Social proximity has costs when such proximity allows viruses to be transmitted relatively easily.

The standard SIR model in epidemiology has three types of agents: susceptible (or not yet infected) agents, infected agents, and recovered agents (who may be alive or dead). To allow for testing, we extend the model to allow for two types of infected agents: those known to be infected and those not known to be infected. In our economic model, agents engage in a variety of economic activities. Each economic activity is associated with a given number of "meetings" with other agents. These activities are combined to produce a final output good. The virus is transmitted with an exogenous activity-specific probability in a meeting between an infected and susceptible agent. We assume that activities with low transmission probabilities also have low economic value.

The planner seeks to maximize the present discounted utility of consumption net of costs of treating infected agents and of death costs. In our model, absent any testing, the planner excludes agents who are known to be infected from any activity and allocates some of the agents whose types are not known to the activity with the lowest probability of transmission. Since the lowest probability of transmission activity has the lowest economic value, this policy tends to reduce output, but it saves lives. The quantitative version of our model generates output declines and death reductions broadly similar to those in Eichenbaum et al. (2020b) and Glover et al. (2020). We measure the welfare gains from optimal policy as the permanent percentage increase in consumption that would give the planner the same utility as under no policy. For reference, we note that the loss in welfare in the no-intervention economy relative to the no-pandemic economy is 6.66%. We show that the optimal policy yields a welfare gain of roughly .6% relative to no intervention. We then introduce a costly testing technology. The planner can choose to test a fraction of the population whose types are not known. We assume the test perfectly reveals whether an agent is infected. We show that optimal policy with this type of untargeted testing yields a welfare gain of .7% relative

to no intervention. That is, untargeted testing delivers gains of only .1% relative to welfare under optimal policy with no testing.

We allow for targeted testing by assuming that each agent whose type is not known is associated with a signal that he is infected. We think of this signal as combining information from a variety of sources. One example is contact tracing, which involves tracing people an infected person has come into contact with, persons whom these contacts contacted, and so on. This signal is informative in the sense that the probability of receiving the signal is higher for infected agents than for susceptible agents. We assume the signal is not perfectly revealing in that the probability an infected person receives the signal is strictly less than one. Out of these agents with the signal, the planner chooses the fraction to test. We choose the signal probabilities to be consistent with data from South Korea. That data suggests that 38% of infected people and 0.44% of susceptible people are associated with the signal. We show that the welfare gain from optimal policy with targeted testing relative to no intervention is roughly 3% of consumption forever. That is, targeted testing allows for a dramatic gain in welfare relative to a no-intervention policy.

In our model, targeted testing allows the planner to very precisely target some agents in order to isolate them. We separate out the effects of isolation from the informational gains to testing by considering a version of the model in which the planner cannot test after receiving the signal.[1] The planner simply chooses the fraction of individuals with a signal to isolate. We show that under optimal isolation, the welfare gains are 2% of consumption forever. That is, roughly 2/3 of the gains from optimal testing can be realized by forgoing testing and simply isolating agents suspected of being infected.

We conduct a variety of sensitivity exercises by varying the probability of receiving the signal. We show that if 60% of infected agents and 3% of susceptible agents are associated with the signal, the welfare gains relative to no intervention are about 5.5%. That is, the welfare loss from the pandemic is only about 1% of consumption. We also show that if a relatively small fraction of infected agents receives the signal, then the welfare gains are also smaller.

[1]Testing can, of course, be very valuable in learning about the current state of the system and the parameters governing its evolution. Our focus here is on testing to isolate infected individuals, rather than testing as a learning device.

These findings provide additional quantitative support for the policies advocated by Romer and Garber (2020), Romer (2020), and Holtemöller (2020).

Relation to other recent papers.

Here, we present a discrete-time version of the standard continuous-time SIR (Susceptible, Infected, Recovered) epidemiology model outlined (for economists) by Atkeson (2020b).[2] A long (and growing) list of papers emphasizes the trade-offs between the losses from restrictions on economic activity and the losses from allowing the virus to spread. See, among many others, Alvarez et al. (2020), Atkeson (2020b), Atkeson (2020a), Azzimonti et al. (2020), Baqaee et al. (2020), Bodenstein et al. (2020), Eichenbaum et al. (2020b), Farboodi et al. (2020), Garriga et al. (2020), Guerrieri et al. (2020), Hall et al. (2020), Jones et al. (2020), Kaplan et al. (2020), Krueger et al. (2020), Moser and Yared (2020), Rampini (2020), and Toxvaerd (2020). While none of these papers focuses on the role of testing, some other recent papers do. See, for example, Eichenbaum et al. (2020a), Berger et al. (2020), Acemoglu et al. (2020), and Piguillem et al. (2020). Our findings complement the results in this literature regarding the desirability of testing. In particular, Eichenbaum et al. (2020a) and Piguillem et al. (2020) also emphasize the critical role of isolation.

2. An Economic Model with SIR Contagion

Consider a discrete time infinite horizon model with a continuum of individuals on the unit interval. At any date, an individual is either *susceptible* S, *infected* I (and thus contagious), or *recovered* R, (where recovered can be either dead or alive). Of the infected, $I - \tilde{I}$ agents are known to be infected, and $\tilde{I}$ agents are not known to be infected (hereafter *unknown infected*). Let $(S_t, I_t, \tilde{I}_t, R_t)$ denote the fraction of agents who are susceptible, infected, unknown infected, and recovered (dead or alive) at date t. We assume that the planner knows which agents are recovered and which are known to be infected, but the planner cannot tell apart susceptible and unknown infected agents.

An infected person dies with probability $\gamma\delta$, stays alive with probability $\gamma(1-\delta)$, and stays infected with probability $(1-\gamma)$. We assume, as is conventional in much of the

[2] Atkeson (2020b), like other models in epidemiology, allows also for an *Exposed* state. Such models are referred to as SEIR models.

literature, that infection confers permanent immunity so that a recovered person always stays recovered—again, either dead or alive. If $S_0 + I_0 = 1$ (all individuals start as susceptible or infected), this assumption ensures that, for all t, $(1-\delta)R_t$ fraction of initially alive people are alive and δR_t fraction of initially alive people are dead.

In our model, economic activity is associated with meetings or interactions. Our economy has N types of intermediate goods labeled $i \in \{1, \ldots, N\}$. Good i is produced using activity i. Each activity of type i requires M_i meetings for each person engaged in that activity. The technology for producing good i is given by

$$(1) \quad y_{it} = b_i \bar{L}_{it},$$

where y_{it} is the amount of good i, b_i is a technology parameter that depends on activity i and the number of meetings, and $\bar{L}_{it}$ is the amount of effective labor allocated to good i. Type S and type R agents each supply one unit of effective labor, and infected agents supply ξ units of effective labor. The intermediate goods are combined into a final consumption good by a CES aggregator, so that the amount of the final good Y_t produced in period t is

$$(2) \quad Y_t = \sum\nolimits_i (y_{it}^{\frac{\sigma-1}{\sigma}})^{\frac{\sigma}{\sigma-1}}.$$

In the presence of a pandemic, meetings also lead to type-specific transmission of the virus. The purpose of indexing meetings by type i and allowing the transmission rate to depend on meeting type i is to allow the framework to consider multiple types of policy interventions, such as prohibiting or decreasing particular *types* of meetings. For instance, the probability of transmission while chatting on a sidewalk can depend on whether both people are wearing masks. Meeting while wearing masks can be considered a different type of meeting than meeting while not wearing masks. Meetings that occur only after each participant has had his temperature checked can be considered a different type of meeting from meetings where such temperature checks do not occur. Further, we later consider the *costs* of various policy interventions. While the rate of transmission for two workers standing next to each other might be the same regardless of what they are producing, the cost to society of reducing such meetings may very well depend on whether they are producing ventilators or academic

papers (since the latter can be more easily moved online). In such a case, these two activities would be considered different types of meetings.

Model with No Testing

To make the exposition easier, we begin by considering a version of the model with no testing and then introduce testing. In our model, economic activity induces infections and thereby the resulting laws of motion for the state variables. Let L_{it} denote the mass of people assigned to activity i, λ_{it} denote the fraction of agents assigned to activity i whose types are unknown (these consist of S and $\tilde{I}$ type agents), and μ_{it} the fraction of recovered agents who are assigned to activity i. We assume that agents who are known to be infected are assigned to activity N. Each activity i is characterized by the probability that a susceptible person who meets an infected person gets infected, p_i. Within an activity, meetings are independently drawn. Since the mass of people assigned to activity i is L_{it}, and the mass of infected people assigned to activity i is $\lambda_{it}\tilde{I}_t$, a susceptible person in a single meeting meets an infected person with probability $\lambda_{it}\tilde{I}_t/L_{it}$ and gets infected with probability $p_i\lambda_{it}\tilde{I}_t/L_{it}$. The probability of being infected in M_i meetings is then

$$1 - \left(1 - p_i \frac{\lambda_{it}\tilde{I}_t}{L_{it}}\right)^{M_i}, \tag{3}$$

where

$$L_{it} = \lambda_{it}(S_t + \tilde{I}_t) + \mu_{it}R_t. \tag{4}$$

Since the mass of susceptible people assigned to activity i is $\lambda_{it}S_t$, the law of motion for the mass of susceptible people in the population is

$$S_{t+1} = S_t - \sum_i \lambda_{it} S_t \left(1 - \left(1 - p_i \frac{\lambda_{it}\tilde{I}_t}{L_{it}}\right)^{M_i}\right). \tag{5}$$

Taking a Taylor series expansion of this law around $\tilde{I}_t = 0$, we obtain a law of motion similar to that in the SIR models, given by

$$(6) \quad S_{t+1} = \left[1 - \sum_i \left(\lambda_{it}\pi_i \frac{\lambda_{it}\tilde{I}_t}{L_{it}}\right)\right] S_t,$$

where $\pi_i = p_i M_i$. We use this approximation in our quantitative assessment of policies.

In terms of the law of motion for $\tilde{I}$, we assume that a person of type $\tilde{I}$ becomes known to be infected with exogenous probability $\tilde{\tau}$. Since an infected person stays infected with probability $1-\gamma$, the law of motion for $\tilde{I}$ is

$$(7) \quad \tilde{I}_{t+1} = (1-\gamma)(1-\tilde{\tau})\tilde{I}_t + S_t \sum_i \left(\lambda_{it}\pi_i \frac{\lambda_{it}\tilde{I}_t}{L_{it}}\right),$$

and the law of motion for R is

$$(8) \quad R_{t+1} = R_t + \gamma(1 - S_t - R_t).$$

Together with the adding up constraint that is $S_t + I_t + R_t = 1$, the system of equations, (5), (7), and (8) describe the dynamics of the system. This dynamical system has a continuum of steady states, indexed by S, the steady state fraction of susceptible individuals, with R, the steady state fraction of recovered individuals equal to $1-S$ (and thus $I = 0$). For expositional convenience, we describe the problem the planner solves below.

Given an initial state of the system $(\tilde{I}_0, R_0, S_0)$ and the policy variables λ_{it} and μ_{it}, the approximate dynamical system, with (5) replaced by (6), is identical to the familiar SIR models from epidemiology. A key variable in these models is $\mathcal{R}_{0,t}$ — the number of new infections per susceptible person per infected person, multiplied by the mean number of periods an infected person is infected. (The variable $\mathcal{R}_{0,t}$ is not to be confused with R_t, the fraction of recovered people in the population.) From equation (6), using the observation

that the mean number of periods infected is $1/\gamma$, we have

$$(9) \quad \mathcal{R}_{0t} \approx \frac{1}{\gamma} S_t \frac{\sum_i \left(\lambda_{it} \pi_i \frac{\lambda_{it} \tilde{I}_t}{L_{it}} \right)}{I_t S_t}.$$

Model with Testing

Next, we introduce testing into the model. At the beginning of each period, each person whose type is not known is associated with a public signal that he is infected. Let θ_X, $X \in \{S, \tilde{I}\}$ denote the probability this signal is received regarding an individual of type X. That is, the signal is useful information to the extent that $\theta_I > \theta_S$. With this formulation, the mass of agents who are associated with a signal of infection is $\theta_S S + \theta_I I$. The planner chooses to test the fraction τ of these individuals. We assume the test perfectly reveals whether an agent is infected. The planner then isolates all those who test positive. Thus, the law of motion for $\tilde{I}$ is now

$$(10) \quad \tilde{I}_{t+1} = (1 - \tau\theta_I) \left[(1 - \gamma)(1 - \tilde{\tau}) \tilde{I}_t + S_t \sum_i \left(\lambda_{it} \pi_i \frac{\lambda_{it} \tilde{I}_t}{L_{it}} \right) \right],$$

where L_{it} is given by

$$(11) \quad L_{it} = \lambda_{it} \left(S_t + (1 - \tau\theta_I) \tilde{I}_t \right) + \mu_{it} R_t,$$

and output in activity i is given by

$$(12) \quad y_{it} = b_i \bar{L}_{it} = b_i \left(\lambda_{it} (S_t + (1 - \tau\theta_I) \xi \tilde{I}_t) + \mu_{it} R_t \right).$$

The cost of testing is $C(\tau_t(\theta_S S_t + \theta_I I_t))$, where $C(\cdot)$ is the testing cost function. The role of testing is to remove some of the infected agents whose type is not known from current and future economic activity until they recover. This policy of removing some of the infected agents prevents them from infecting others, in both current and future periods. In addition to the testing cost, removing these agents is costly since they cannot engage in useful economic activity.

Aggregate consumption is given by $Y_t - C(\tau_t(\theta_S S_t + \theta_I I_t))$. The planner's preferences

over consumption are given by $\sum_{t=0}^{\infty} \beta^t U(Y_t - C(\tau_t(\theta_S S_t + \theta_I I_t))$. In addition, infection is associated with a utility cost ZI_t, where Z denotes the healthcare and related costs of being infected, and deaths are associated with a utility cost $D\gamma\delta I_t$, where we note that the mass of agents who die in period t is $\gamma\delta I_t$, and the parameter D measures the cost of a life.

The planning problem is then to choose a testing policy τ_t and labor allocation policies λ_{it} and μ_{it} to solve

$$\max \sum_{t=0}^{\infty} \beta^t \left[U\left(Y_t - C\left(\tau_t\left(\theta_S S_t + \theta_I I_t\right)\right)\right) - Z_t\left(I_t\right) - D_t\left(\gamma\delta I_t\right), \right]$$

subject to (1), (2), (4), (6), (8), (10), given the initial conditions $(S_0, I_0, \tilde{I}_0, R_0)$. We will refer to the version of the model with $\theta_I = \theta_S = 1$ as the model with *untargeted* testing and the model with $\theta_I > \theta_S$ as the model with *targeted* testing. Note that the programming problem for the model without testing is simply this programming problem with $\tau_t = 0$ for all t.

In order to understand how policy can be used to affect the course of the infection, consider a simple version of the model with two activities, *work* and *home*. Suppose that work produces higher output but is also associated with higher infection than the home activity, so that $b_{work} > b_{home} = 0$ and $\pi_{work} > \pi_{home} = 0$. Without any testing, it is optimal in general to assign some agents whose types are not known to stay home. This policy reduces economic activity but also reduces the rate of transmission of the virus. We refer to this policy as *indiscriminate* isolation, since the policy does not discriminate between infected and susceptible agents. Consider next the role of targeted testing in this simple model. Targeted testing allows the planner to isolate some of the infected agents by requiring them to stay at home. In this sense, targeted testing allows for a form of targeted isolation.

Model with Isolation and No Testing

As before, agents receive signals at the beginning of the period. In this version of the model, however, the planner does not test but simply isolates some fraction of the agents who have received signals in the current period. In particular, we assume that the planner does not use past signals in isolating individuals. This assumption implies that welfare under

the policy is a lower bound for a more elaborate policy that uses the entire history of past signals. The law of motion for $\tilde{I}$ is now

$$(13)\quad \tilde{I}_{t+1} = (1-\gamma)(1-\tilde{\tau})\tilde{I}_t + (1-\tau\theta_S) S_t \sum_i \left(\lambda_{it}\pi_i \frac{\lambda_{it}(1-\tau\theta_I)\tilde{I}_t}{L_{it}} \right),$$

where L_{it} is now given by

$$(14)\quad L_{it} = \lambda_{it}\left((1-\tau\theta_S) S_t + (1-\tau\theta_I)\tilde{I}_t \right) + \mu_{it} R_t,$$

and output in activity i is given by

$$(15)\quad y_{it} = b_i M_i \left(\lambda_{it}\left((1-\tau\theta_S) S_t + (1-\tau\theta_I)\xi\tilde{I}_t \right) + \mu_{it} R_t \right).$$

Note that as in the case with testing, the role of isolation is to remove some fraction of the infected people from economic activities where these people may infect others. One advantage of this policy is that it does not require the use of testing resources. A disadvantage of this policy is that some fraction of susceptible people is also removed from productive economic activities. Throughout our analysis of isolation policies we will assume that $\theta_I > \theta_S$.

Since it is always possible to set $\tau = 0$ with either testing or isolation, it immediately follows that welfare is higher than it is when these instruments are not available. In our next result, we show that isolation and testing are policies that are typically used in their entirety before the planner resorts to reducing labor allocation in productive economic activities with high rates of infection. In order to understand this result, it is useful to consider a special case with only two activities: work and home. Suppose that $b_{work} = 1, b_{home} = 0, \pi_{work} > 0, \pi_{home} = 0$. In this case, absent a pandemic, all agents would be assigned to the work activity. With a pandemic and no testing or isolation, it can be optimal to assign some of the workers of unknown type to stay at home. The trade-off of this policy is that assigning a larger number of agents to stay home reduces output but also reduces the rate at which the virus spreads. If only isolation policies are available, then it is possible to show that it is optimal to exhaust all isolation possibilities before assigning any workers of unknown type to stay home. We formalize this result in the following proposition.

Proposition 1. *Suppose that* $\pi_{home} = 0$, $b_{home} = 0$ *and* $\xi = 1$. *Then if* $S > \tilde{I}$, $\lambda_{work} < 1$ *only if* $\tau = 1$.

The proof of this result is in the appendix. This result also implies that if the cost of testing is sufficiently small, it is optimal to exhaust all testing possibilities. By continuity it follows also that if π_{home} and b_{home} are not too different from zero, the same result applies.

In summary, these results imply that in general, welfare under a regime with testing or isolation will be higher than welfare under a regime with no testing or no isolation.

Returns to Testing: A Simple Example

To illustrate the sense in which the gains from targeted testing are potentially large, consider the following simple example. Suppose there are two activities, home and work, and $b_{home} = 0$. Suppose also that the transmission rate of the virus at home is zero. For simplicity, assume that the utility function is linear. A planner who desires to reduce $\mathcal{R}_0$ from its unrestricted value of 2.5 to a value of 1 can do so by setting $\lambda_{work} = 0.4$. Note here $b_{work}\lambda_{work}$ is GDP per time period. If a time period is two weeks, the cost of this policy is $0.6 \times \$800B = \$480B$ per two-week period.

If one could identify 60% of the infected agents and assign them all to stay at home, then $\mathcal{R}_0$ drops from 2.5 to 1 without reducing GDP very much. One method to do this would be to test 60% of the population every week, if possible, and require those who test positive to stay home. It is straightforward to show that this *indiscriminate* method has a break-even cost of \$1200 per test. If we could instead achieve the same 60% reduction in the number of infected people in the workplace by testing $z \times 0.6$ fraction of population (contact tracing, testing targeted in areas of outbreaks), the break-even cost becomes $\$1200/z$.

3. Dynamics and the Effects of Social Distancing

In this section, in order to obtain intuition about the trade-offs that optimal policy must confront, we illustrate the behavior of our dynamical system for some simple cases. Suppose now that the fraction of agents who are known to be infected relative to the mass of infected agents is constant. Let this fraction be denoted by $q = 1 - \tilde{I}/I$. Suppose also that $\lambda_{it} = \mu_{it} = 1/N$ for all t. Then, it is easy to show that the law of motion for the mass of

infected agents is given by

$$(16)\quad I_{t+1} = I_t\,(1-\gamma) + S_t\left(1 - \frac{1}{N}\sum_i \left(1 - p_i \frac{(1-q)\,I_t}{S_t + (1-q)\,I_t + R_t}\right)^{M_i}\right),$$

and that for the mass of susceptible people is given by

$$(17)\quad S_{t+1} = S_t - S_t\left(1 - \frac{1}{N}\sum_i \left(1 - p_i \frac{(1-q)\,I_t}{S_t + (1-q)\,I_t + R_t}\right)^{M_i}\right).$$

In Figure 1, we display the evolution of this dynamical system in state space form, with I_t on the y-axis and S_t on the x-axis. Note that equation (17) implies that if $I_t > 0$, $S_{t+1} - S_t < 0$. That is, S moves to the left, or west, in Figure 1. To see how I_t evolves, we partition (S, I) space into those points to the right and left of the upsloping locus of (S, I) points such that I is constant. We derive this locus by setting $I_{t+1} = I_t = I$, $S_t = S$, delivering

$$(18)\quad \gamma I = \left(1 - \frac{1}{N}\sum_i \left(1 - p_i \frac{(1-q)\,I}{1-qI}\right)^{M_i}\right) S.$$

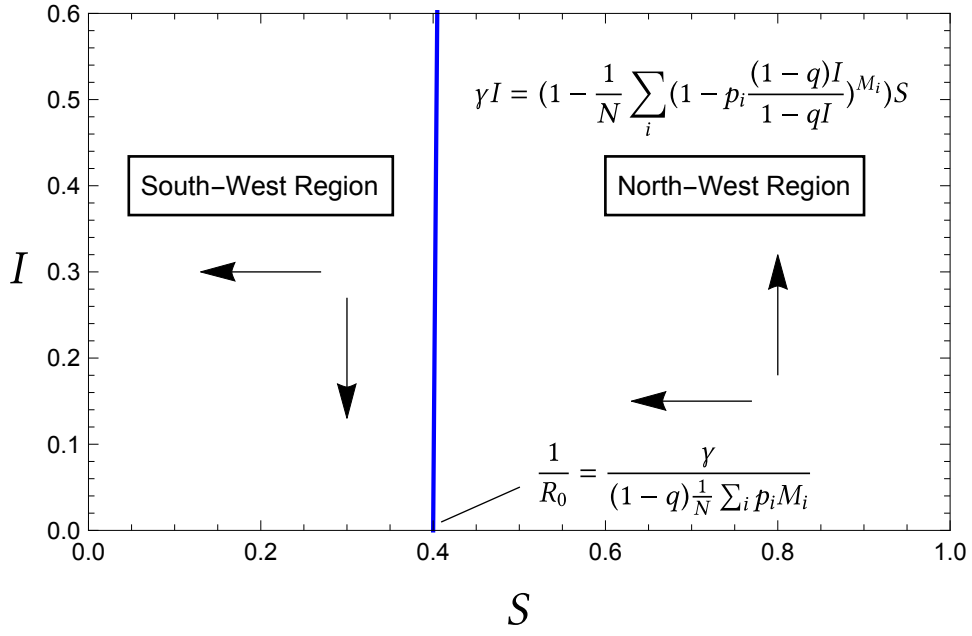
Note that this locus intersects the horizontal axis at

$$\lim_{I\to 0} \frac{\gamma I}{\left(1 - \frac{1}{N}\sum_i \left(1 - p_i \frac{(1-q)I}{1-qI}\right)^{M_i}\right)} = \frac{\gamma}{(1-q)\frac{1}{N}\sum_i p_i M_i} = \frac{1}{\mathcal{R}_0},$$

as indicated in the figure. To the right of this locus, the dynamics of the system are north-west. That is, $S_{t+1} < S_t$ from (17) and $I > 0$, and $I_{t+1} > I_t$ from S being greater than that associated with I being constant (and I_{t+1} being an increasing function of S_t in (16)). To the left of this locus, the dynamics of the system are south-west. Here, again, $S_{t+1} < S_t$ when $I_t > 0$, and $I_{t+1} < I_t$ from S being less than that associated with I being constant.

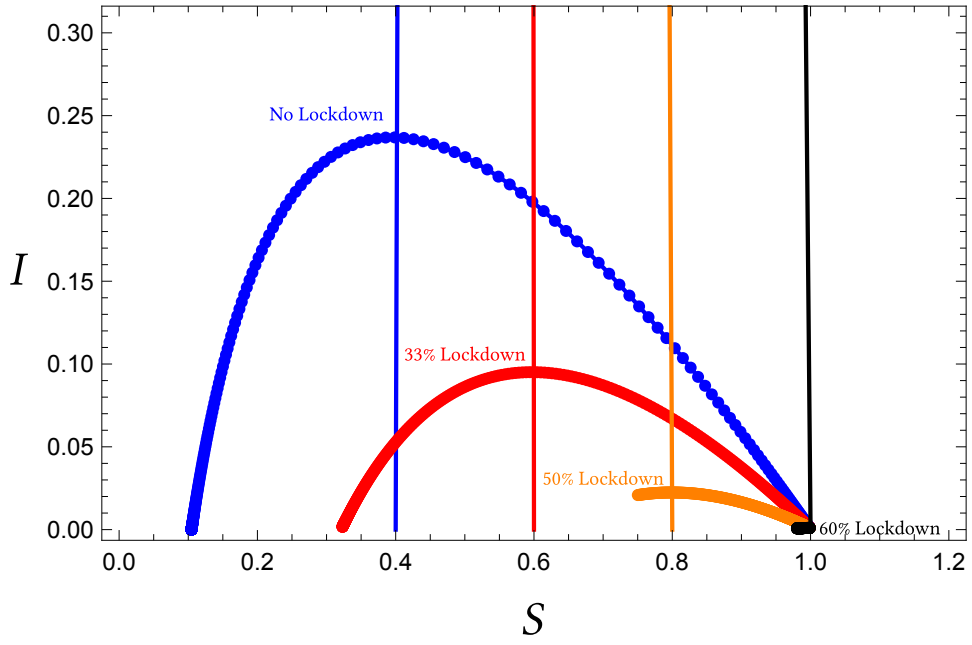
This implies that if the initial (S_0, I_0) is to the left of the locus, (S_t, I_t) converges to a steady state on the horizontal axis following a south-west path. If the initial (S_0, I_0) is to the right of the locus, (S_t, I_t) converges to a steady state on the horizontal following an arc-pattern, with I_t increasing as S_t decreases until (S_t, I_t) crosses the locus, converging again

Figure 1: (S, I) Phase Diagram



to a steady state on the horizontal axis. Note that this implies all steady states reachable from an initial state with $I_0 > 0$ have $S < 1/\mathcal{R}_0$. That is, assuming $I_0 > 0$, regardless of the initial state, the system converges to a steady state where, at most, $1/\mathcal{R}_0$ agents avoid infection. In particular, as $\mathcal{R}_0 \to \infty$, the fraction of individuals who never get infected goes to zero (and the fraction of individuals who eventually become infected goes to one). Figure 2 presents some computed examples of (S_t, I_t) paths starting from $(S_0, I_0) = (0.999, 0.001)$ with only two activities, home and work, and under the assumption that $p_{home} = 0$, $q = 0$, $\gamma = 1/18$, and $M \in \{25\gamma, 16.6667\gamma, 12.5\gamma, 10\gamma\}$, p_{work} is chosen so that the corresponding $\mathcal{R}_0 \in \{2.5, 1.67, 1.25, 1.0\}$. Here, we show the effect of reducing $\mathcal{R}_0$ by decreasing M from a high of $M = 25\gamma$ (implying $\mathcal{R}_0 = 2.5$, or approximately what epidemiologists consider $\mathcal{R}_0$ to be without social distancing) to a low of $M = 10\gamma$ (implying $\mathcal{R}_0 = 1$). For the high $M = 25\gamma$, I increases very quickly, and in a short number of periods, almost all people have recovered, or $(S, I) \approx (0.1, 0)$. For the low $M = 10\gamma$, I and S decrease slowly, and the system approaches a steady state with nearly all people having never been infected.

Figure 2: The Effects Differing Constant Economic Lockdowns For One Year



4. Lifting a Lockdown

The previous example gives rise to the sobering possibility that a lockdown must go on forever, otherwise the system returns to one where $\mathcal{R}_0$ is high and $I_0 > 0$, although $S_0 < 1$. The following example starts with $(S_0, I_0) = (0.999, 0.001)$ and has $M = 10\gamma$ (and thus $\mathcal{R}_0 = 1$) for 365 periods (or one year, assuming a one day period length), then permanently relaxes the lockdown to $M = 25\gamma$ (and $\mathcal{R}_0 = 2.5$). Here, the path after lifting the lockdown is essentially the same as if the lockdown had never been enforced. Figure 3 shows the result of this exercise in (S, I) space, and Figure 4 graphs the same exercise showing the infection rate, I_t, over time.

Figure 3: The Effect of a One Year Lockdown in (S, I) Space.

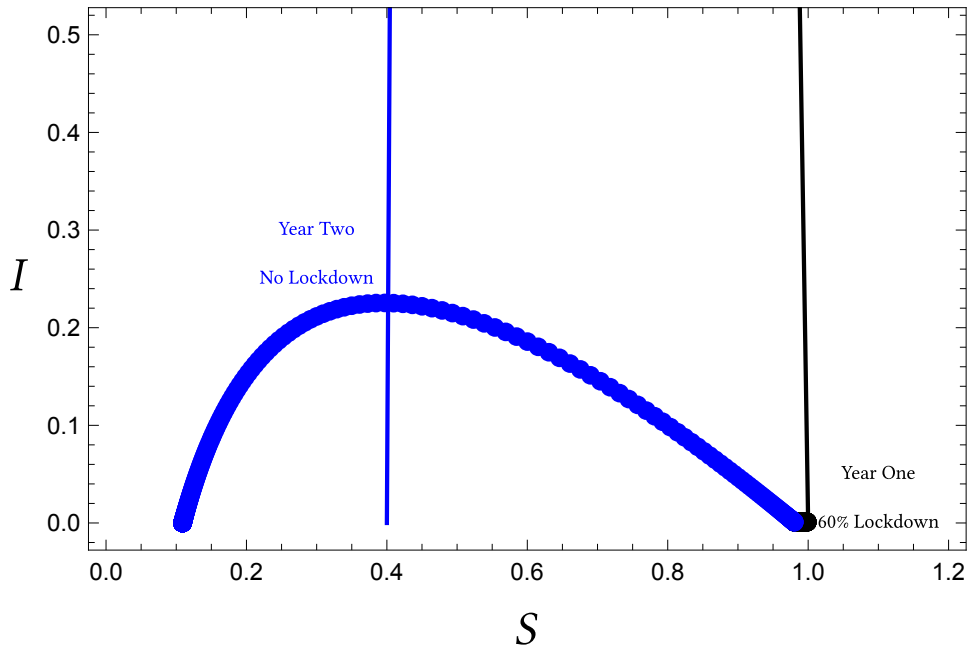
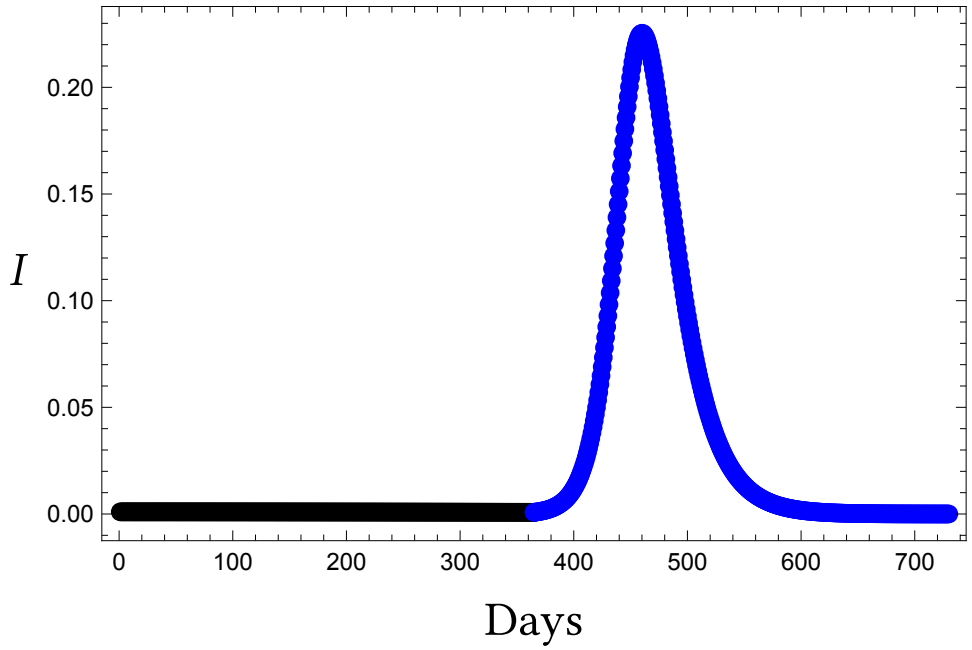


Figure 4: The Effect of a One Year Lockdown in (S, I) Space.



5. Calibration

We assume that the utility function U is the log function. We parameterize the testing cost function as $c(T)^{1+\nu}/(1+\nu)$. We assume that the economy has two activities: work and

home. This formulation allows us to relate our results to those in the literature, particularly Eichenbaum et al. (2020b) and Glover et al. (2020).

We set the parameter values to be very similar to equivalent parameter values in Eichenbaum et al. (2020b) and Glover et al. (2020). The parameter values are reported in Table 1. We set the time period to be one week and set the discount factor β assuming that the vaccine is expected to arrive in 18 months and the annualized discount rate for the planner is 4%. We set the exit rate γ assuming that the expected length of infection of recovery is 18 days. We set the exogenous rate at which unknown infected people become known, $\tilde{\tau}$, using the observation that infected agents are asymptomatic for the first five days of the infection and roughly half of all infected agents never display symptoms (see Glover et al. (2020)). We set the productivity of infected people ξ, following Eichenbaum et al. (2020b). The mortality rate δ is set at 0.5%; see Ferguson et al. (2020). We set the elasticity of substitution at 2 and normalize the productivity in the work sector to be 1 and that in the home sector be 0.1. We set the curvature parameter on the testing cost function, ν, at 1 and choose the parameter c so that the marginal cost of testing is \$50 if 1% of the population is tested. We follow Glover et al. (2020) in setting the cost of treating the infected, Z, so that the cost of treating an infected person is \$7500 over the course of infection. We set the cost of death parameter, D, so that the value of a life is equal to the present discounted value of 15 years of consumption. We set $\pi_{home} = 0.01$ and set π_{work} so that the reproduction rate $\mathcal{R}_0$ without any policy has an average value of approximately 3 in the first four weeks.

Since the main focus of our analysis is the role of testing and isolation, we experimented with a number of values for the signal probabilities, θ_S and θ_I. For our baseline calibration, we set $\theta_I = 0.38$ and $\theta_S = 0.0044$. To arrive at these numbers, we start with the view that South Korea was particularly effective at pursuing an aggressive test, trace, and isolate policy. In South Korea, about 1.8% of tests return a positive result, compared with a population infection rate of 0.021%. That is, conditional on having a *reason* to be tested (a signal in our model), a South Korean had a probability of testing 86 times higher than if tests were done randomly. Thus, the South Korean data suggest a value of $\theta_I/\theta_S = 86$, implying a very informative signal. In the South Korean data, 1.2% of the population has been tested. Since

Table 1: Model Parameters

β	Discount rate	Weekly model. Vaccine arrival 18 months	0.99
γ	Exit rate	18 day infection period	7/18
$\tilde{\tau}$	Prob. of becoming symptomatic	5 day incubation, 1/2 asymptomatic	$\frac{13}{18} \times \frac{1}{2}$
b_1	Productivity of work		1
b_2	Productivity of home		0.1
ξ	Infected productivity loss	Eichenbaum et al. (2020b)	0.8
δ	Death rate	Ferguson et al. (2020)	0.05%
σ	CES parameter		2
ν	Testing cost parameter		1
c	Testing cost parameter	MC of testing \$50 when 1% tested	4.17
Z	Treatment cost	Glover et al. (2020)	6.25
D	Death cost	Value of life 2.8 million dollars	1.84
π_1	Infection rate at home		0.01
π_2	Infection rate at work	$\mathcal{R}_0$ with no policy 2.5	1.5
θ_I	Signal prob. infected	$\theta_I/\theta_S = 86$ and 1.2% tested (SK)	0.38
θ_S	Signal prob. susceptible	$\theta_I/\theta_S = 86$ and 1.2% tested (SK)	0.0044

we initially set the proportion of infected at 2%, we find $\theta_I = 0.38$ and $\theta_S = 0.0044$ as the solution to

$$\theta_S S + \theta_I I = 0.012, \; I = 0.02, \; \frac{\theta_I}{\theta_S} = 86.$$

6. Findings

Here, we report on the findings from our quantitative model. Our measure of welfare is the standard compensating variation in consumption widely used in the macroeconomics literature. Specifically, we ask what permanent percentage increase in consumption relative to the no-intervention economy would give the planner the same utility as under our experiments. We report all welfare calculations relative to the no-intervention economy. For reference, we note that the loss in welfare in the no-intervention economy relative to the no-pandemic economy is 6.66%. The welfare changes in our experiments relative to the no-policy case arise from changes in the time paths of output, testing costs, infection costs, and death costs.

In Table 2, we report on the welfare measure in our experiments as well as a partial

decomposition of the change in welfare induced by changes in output and death costs. To measure the change in welfare induced by the changes in output, we compute the annuity value of the present discounted value of output in our experiments. To measure the changes in welfare induced by death, we report the cumulative fraction of the population that dies at the end of 52 weeks. In Figures 5, 6, and 7, we report on the time paths of the fraction of the population infected I_t, the fraction of population susceptible S_t, cumulative deaths, the reproduction rate $\mathcal{R}_{0t}$, the fraction of the population infected but not known to be so $\tilde{I}_t$, consumption, the mass tested, and the marginal cost of testing.

We begin by comparing outcomes in the no-policy case, optimal policy with no testing, and optimal policy with untargeted testing. We see that optimal policy with no testing yields a welfare gain of roughly 0.6%, and an optimal policy with untargeted testing yields a welfare gain of 0.7% relative to no intervention. That is, untargeted testing delivers very modest welfare gains. From Figure 5, we see that in all 3 cases, the economy goes through a severe recession that lasts about 3 months. We see that optimal policy reduces output at its trough by roughly 40% and by about 20% with no policy intervention. The primary gains to welfare relative to no policy come from a sharp reduction in the cumulative number of deaths. With no policy intervention, roughly 0.5% of the population dies, while with optimal policy, roughly 0.35% of the population dies. In all three experiments, the population eventually reaches herd immunity, though the steady state fractions are very different with and without optimal policy. The figure also shows that the main mechanism by which optimal policy reduces the cumulative death rate is by inducing a sharper recession, which in turn reduces the effective reproduction rate $\mathcal{R}_{0t}$ below 1 and thereby induces a reduction in the proportion of the population that is infected. The findings regarding optimal policy with no testing are broadly similar to the findings in Eichenbaum et al. (2020b) and Glover et al. (2020). Figure 5 also shows that with untargeted testing, as much as 15% of the population is tested. Note that at its peak, the marginal cost of testing is roughly \$800. In this sense, a relatively small fraction of aggregate resources is allocated to testing. The main reason is that untargeted testing is not very valuable.

Next, we compare outcomes in our benchmark targeted testing model with outcomes under optimal policy with no testing and untargeted testing. Table 2 shows that the welfare

gains to targeted testing are substantial. In particular, welfare rises by 2.5% relative to optimal policy with no testing and 3% relative to no policy. This table also shows that the cumulative deaths are about 0.15% lower once we allow for targeted testing and that the output loss is moderated by about 0.8% relative to no testing. Figure 6 shows that welfare rises dramatically mainly because a targeted testing policy ensures an initial decline in the effective reproduction rate $\mathcal{R}_{0t}$ and then keeps that rate at around 1. This way of controlling the reproduction rate ensures that the cumulative death rate is substantially lower. Figure 6 shows that even at the peak of targeted testing, only about 1.5% of the population is tested. The marginal cost of testing, even at its peak, is only about \$70. These results show that relative to untargeted testing, targeted testing is both inexpensive and immensely valuable.

Next, we compare outcomes under our benchmark targeted testing model with our benchmark targeted isolation model. We see that targeted isolation alone generates roughly two-thirds of the welfare gains that come from targeted testing. In this sense, targeted isolation is a very valuable tool if testing is not available.

We also conduct sensitivity analyses to the values of our signal probabilities. Table 3 shows that if $\theta_I = 0.6$, then welfare rises by about 5.7% relative to the no-policy case. Recalling that the pandemic with no policy delivers a welfare loss of 6.7%, we see that if the technology for tracking infected individuals is sufficiently effective, the welfare loss from the pandemic is only about 1%. For this parameter value we also see that the cumulative deaths are reduced very significantly to 0.04% from 0.35%. Figure 7 also shows that the recession that arises from the pandemic is mild. Table 3 also shows that if the technology for tracking infected individuals is much less effective than our benchmark case ($\theta_I = 0.15$), then the welfare, output, and death gains are modest relative to no targeted testing. The message of these findings is that the returns to improving the technology for tracking infected individuals can be exceptionally large.

Finally, we conduct sensitivity analyses (available upon request) on the initial fraction of infected agents I_0. We find the relative gains of testing and isolation policies continue to be substantial. We view this finding as suggesting that even if the pandemic is well under way and testing and isolation policies have not so far been conducted, it is not too late to implement such policies.

Table 2: Results on welfare, deaths, and output loss

Experiment	Welfare gain relative to no-intervention	Cumulative deaths	Output loss
No intervention	0	0.48%	1.3%
Opt policy: no testing	0.59%	0.35%	1.94%
Opt policy: untargeted testing	0.71%	0.3%	2.06%
Targ Test ($\theta_s = .0044, \theta_I = .38$)	3.07%	0.15%	1.28%
Targ Isolation ($\theta_s = .0044, \theta_I = .38$)	2.12%	0.26%	1.66%

Figure 5: Time paths for no policy, no testing, and untargeted testing

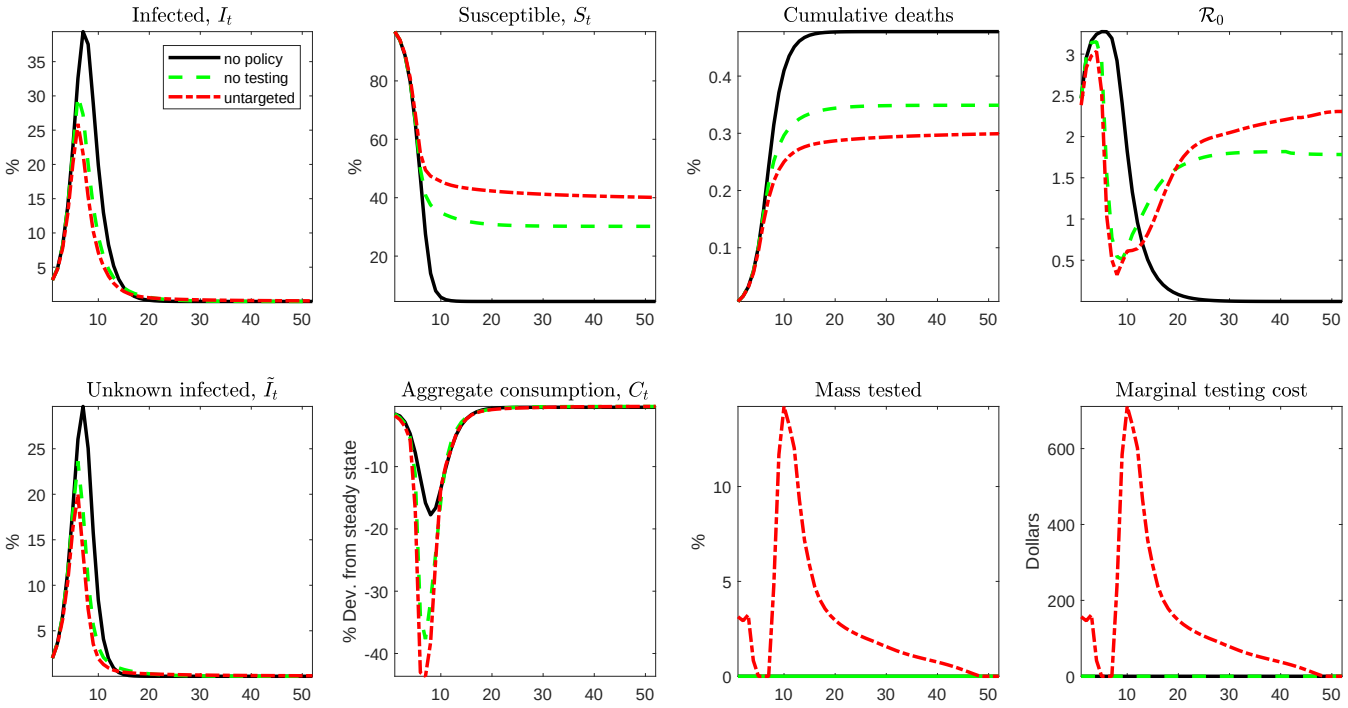


Table 3: Sensitivity analysis with respect to signal parameters

Experiment	Welfare gain relative to no-intervention	Cumulative deaths	Output loss
Targeted Test ($\theta_s = .03, \theta_I = .4$)	3.39%	0.14%	1.10%
Targeted Isolation ($\theta_s = .03, \theta_I = .4$)	1.75%	0.28%	1.79%
Targeted Test ($\theta_s = .03, \theta_I = .6$)	5.7%	0.04%	0.19%
Targeted Isolation ($\theta_s = .03, \theta_I = .6$)	3.99%	0.09%	1.56%
Targeted Test ($\theta_s = .03, \theta_I = .15$)	0.78%	0.3%	2.03%
Targeted Isolation ($\theta_s = .03, \theta_I = .15$)	0.71%	0.34%	1.99%

Figure 6: Time paths for no testing, untargeted testing, and targeted testing

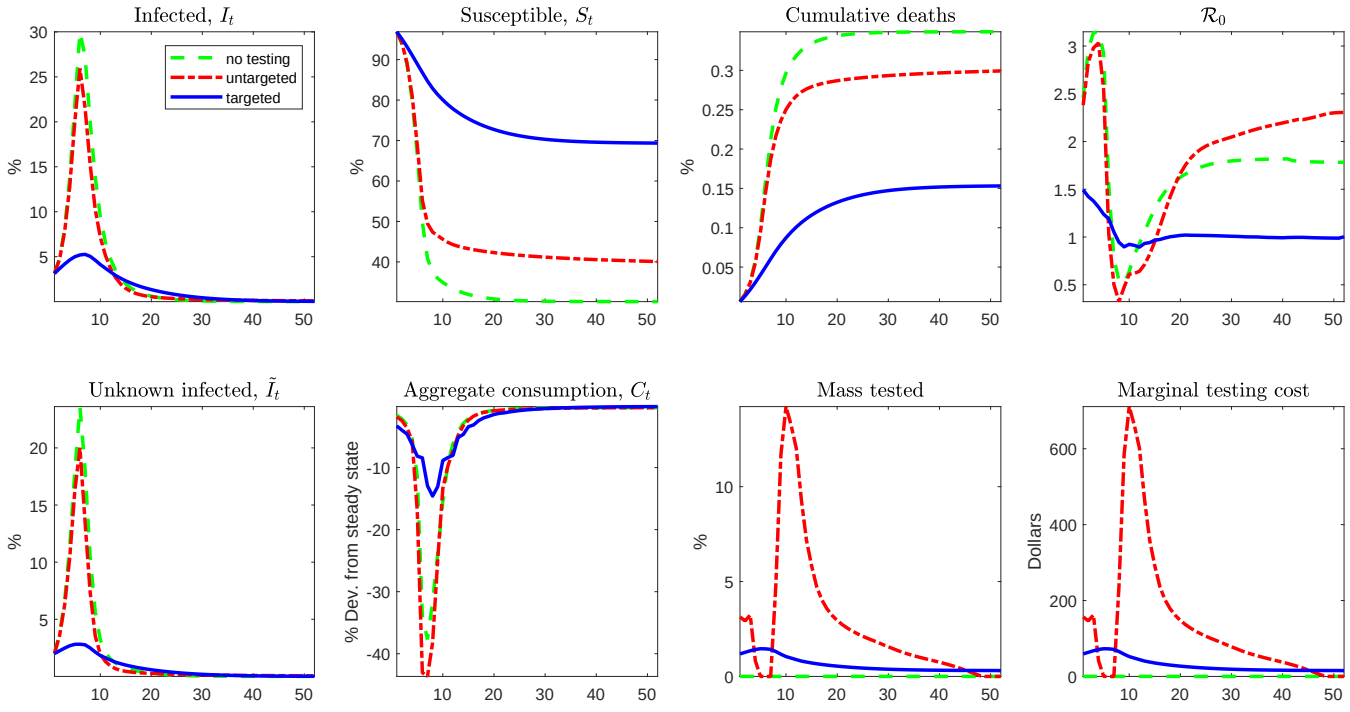
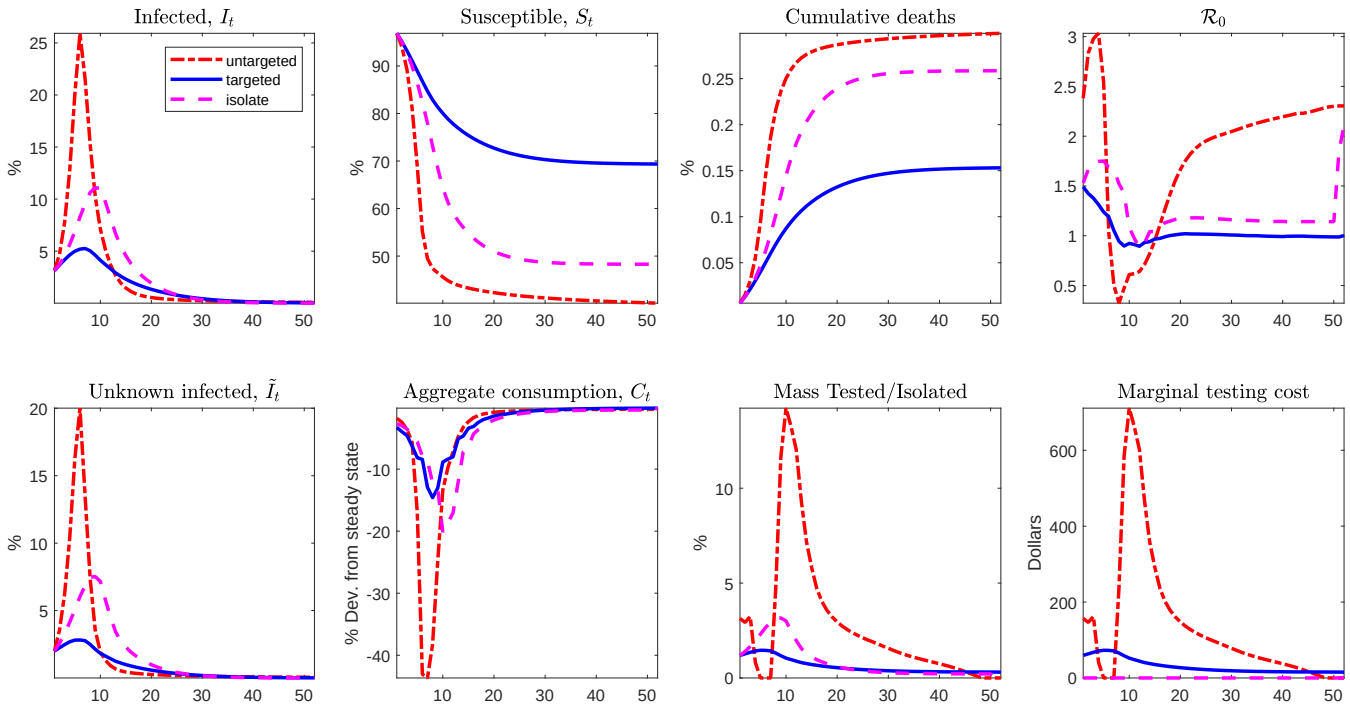


Figure 7: Time paths for untargeted testing, targeted testing, and isolation



7. Conclusion

We have argued that testing and isolation policies can deliver substantial welfare gains in the presence of pandemics. These welfare gains come from a reduced number of cumulative deaths and a shallower recession. Our model can readily be extended to allow for exogenous inflows of agents, some of whom may be infected. Such an extension is useful because we think of our model as one of a particular region or state, rather than of the world. In this context, inter-regional and international migration then introduces new sources of infections. In many situations, the new entering agents can be identified, and the testing and isolation of these agents is clearly valuable. Our findings also suggest that even if a pandemic is well under way, testing and isolation policies are very valuable.

References

Acemoglu, D., V. Chernozhukov, I. Werning, and M. D. Whinston (2020): "A Multi-Risk SIR Model with Optimally Targeted Lockdown," Working Paper 27102, National Bureau of Economic Research.

Alvarez, F. E., D. Argente, and F. Lippi (2020): "A simple planning problem for covid-19 lockdown," Tech. rep., National Bureau of Economic Research.

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

——— (2020b): "What will be the economic impact of COVID-19 in the US? Rough estimates of disease scenarios," Tech. rep., National Bureau of Economic Research.

Azzimonti, M., A. Fogli, F. Perri, and M. Ponder (2020): "Social Distance Policies in Network Cities," Tech. rep., National Bureau of Economic Research.

Baqaee, D., E. Farhi, M. Mina, and J. Stock (2020): "Evaluation the Economics and Epidemiology of Strategies for Reopening the Economy," Tech. rep.

Berger, D. W., K. F. Herkenhoff, and S. Mongey (2020): "An seir infectious disease model with testing and conditional quarantine," Tech. rep., National Bureau of Economic Research.

Bodenstein, M., G. Corsetti, and L. Guerrieri (2020): "Social Distancing and Supply Disruptions in a Pandemic," .

Eichenbaum, M., S. Rebelo, and M. Trabandt (2020a): "The Macroeconomics of Testing During Epidemics," .

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

Farboodi, M., G. Jarosch, and R. Shimer (2020): "Internal and external effects of social distancing in a pandemic," Tech. rep., National Bureau of Economic Research.

Ferguson, N., D. Laydon, G. Nedjati Gilani, N. Imai, K. Ainslie, M. Baguelin, S. Bhatia, A. Boonyasiri, Z. Cucunuba Perez, G. Cuomo-Dannenburg, et al. (2020): "Report 9: Impact of non-pharmaceutical interventions (NPIs) to reduce COVID19 mortality and healthcare demand," .

Garriga, C., R. Manuelli, and S. Sanghi (2020): "Optimal Management of an Epidemic: Lockdown, Vaccine and Value of Life," Working paper.

Glover, A., J. Heathcote, D. Krueger, and J.-V. Ríos-Rull (2020): "Health versus wealth: On the distributional effects of controlling a pandemic," Tech. rep., National Bureau of Economic Research.

Guerrieri, V., G. Lorenzoni, L. Straub, and I. Werning (2020): "Macroeconomic Implications of COVID-19: Can Negative Supply Shocks Cause Demand Shortages?" Tech. rep., National Bureau of Economic Research.

Hall, R. E., C. I. Jones, and P. J. Klenow (2020): "Trading off consumption and covid-19 deaths," Tech. rep., Working Paper.

Holtemöller, O. (2020): "Integrated assessment of epidemic and economic dynamics," Tech. rep., IWH Discussion Papers.

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

Kaplan, G., B. Moll, and G. Violante (2020): "Pandemics according to HANK," .

Krueger, D., H. Uhlig, and T. Xie (2020): "Macroeconomic dynamics and reallocation in an epidemic," Tech. rep., National Bureau of Economic Research.

Moser, C. A. and P. Yared (2020): "Pandemic Lockdown: The Role of Government Commitment," Tech. rep., National Bureau of Economic Research.

Piguillem, F., L. Shi, et al. (2020): "The optimal covid-19 quarantine and testing policies," Tech. rep., Einaudi Institute for Economics and Finance (EIEF).

Rampini, A. A. (2020): "Sequential Lifting of COVID-19 Interventions with Population Heterogeneity," Tech. rep., Federal Reserve Bank of Minneapolis.

Romer, P. (2020): "The road to responsibly reopen america," Tech. rep.

Romer, P. and A. Garber (2020): "Will Our Economy Die From Coronavirus," *The New York Times*.

Toxvaerd, F. (2020): "Equilibrium social distancing," *Cambridge-INET Working Paper Series*.

Appendix

Proof of Proposition 1

Consider the recursive formulation of the programming problem in the model with isolation:

$$V\left(\tilde{I},S,R\right)=\max_{\lambda_i\mu_i,\tau}\left\{\log Y-Z\left(I-\tilde{I}\right)-D\left(\gamma\delta I\right)+\beta V\left(\tilde{I}',S',R'\right)\right\}$$

subject to

$$(19)\quad \tilde{I}'=\left(1-\tilde{\tau}\right)\left(1-\gamma\right)\tilde{I}+\left(1-\tau\theta_S\right)S\sum_i\left(\lambda_i\pi_i\frac{\lambda_i\left(1-\tau\theta_I\right)\tilde{I}}{L_i}\right)$$

$$(20)\quad S'=\left[1-\left(1-\tau\theta_S\right)\sum_i\left(\lambda_i\pi_i\frac{\lambda_i\left(1-\tau\theta_I\right)\tilde{I}}{L_i}\right)\right]S,$$

where R' is given by (8), L_i is given by (14), Y is given by the CES aggregator over y_i given by (15), and $I=1-S-R$.

Clearly, the continuation value is decreasing in $\tilde{I}'$. We will use this result in the proof. Let $\lambda=\lambda_{work}$. Suppose, by way of contradiction, that $\lambda<1$ and $\tau<1$. We will construct a variation that increases λ and τ while keeping current output constant. We will show that this variation reduces $\tilde{I}'$ and thereby raises welfare. This contradiction establishes the proof. To this end, totally differentiate output with respect to τ and λ holding all other variables fixed. We have that

$$(21)\quad d\lambda=d\tau\frac{\lambda\left[S\theta_s+\theta_I\tilde{I}\right]}{\left[S\left(1-\tau\theta_s\right)+\left[1-\tau\theta_I\right]\tilde{I}\right]}.$$

Note that since $\xi=1$, output equals L. Since output is held constant, the rate of infections is now determined solely by

$$\left(1-\tau\theta_s\right)\lambda^2\left(1-\tau\theta_I\right).$$

Differentiating this expression, substituting from (21), and simplifying, we get that the sign

of the rate of infections is given by the sign of

$$\frac{\lambda^2 d\tau}{\left[S\left(1-\tau\theta_s\right)+\left[1-\tau\theta_I\right]\tilde{I}\right]}\left(1-\tau\theta_s\right)\left(1-\tau\theta_s\right)\left[\theta_I-\theta_s\right]\left[-S+\tilde{I}\right],$$

which is negative. Q.E.D.