Optimal Mitigation Policies in a Pandemic:
Social Distancing and Working from Home[*]

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Abstract

We study the response of an economy to an unexpected epidemic. The spread of the disease can be mitigated by reducing consumption and hours worked in the office. Working from home is subject to learning-by-doing. Private agents' rational incentives are relatively weak and fatalistic. The planner recognizes the infection and congestion externalities and implements front-loaded mitigation policies. In our calibrated model, private mitigation reduces the cumulative death rate by about 35% compared to more than 60% for the planner, albeit at the cost of a sharper drop in consumption.

Keywords: contagion, containment, covid 19, recession, R_0, social distancing, SIR model, learning-by-doing, mitigation, suppression, vaccine.

1   Introduction

The response to the Covid-19 crisis highlights the tension between health and economic outcomes. We propose a simple extension of the neoclassical model to quantify the trade-offs and guide policy. We are particularly interested in understanding the nature and timing of policy responses, as well as the tensions between private and public incentives. When will the private sector engineer the right response? When is there a need for policy intervention?

Our model has two building blocks: one for the dynamics of contagion, and one for consumption and production, including mitigation strategies (such as working from home). Our starting point is the standard SIR model of contagion used by public health specialists. Atkeson (2020b) provides a good summary of this framework. In a population of initial size N, the epidemiological state is given by the numbers of Susceptible (S), Infected (I), and Recovered (R) people. By definition, the cumulative number of deaths is $D = N - S - I - R$. Infected people transmit the virus to susceptible people at a rate that depends on the nature of the virus and on the frequency of economic and social interactions. Mitigation in the form of lockdowns and work-from-home reduce the latter and thus, curtail the spread of the disease. The rates of recovery (transitions from I to R), morbidity (I becoming severely or critically sick) and mortality (transition from I to D) depend on the nature of the virus and on the quality of health care services. The quality of health services depends in turn on the capacity of health care providers (number of ICU beds, ventilators) and the number of sick people.

The economic side of the model focuses on three key decisions: consumption, labor supply, and working from home. We use a canonical macroeconomic framework where these decisions are taken by a representative household. Both consumption and work increase the risk of contagion, which is the key link between the economic and epidemiological blocks of the model. We endow agents with the option of working from home, which reduces contagion risk but comes at a cost in the form of productivity losses. These losses decline as agents accumulate experience in working from home. This learning-by-doing aspect introduces additional dynamic considerations in the household's problem.

We use a calibrated version of the model to study the reaction of private agents in equilibrium to the announcement of an outbreak as well as that of a social planner. Upon learning of the risks posed by the virus, households cut spending and labor supply and increase time spent working from home. Broadly, their mitigation efforts are proportionate to the risk of infection, which – all else equal – is proportional to the fraction of infected agents I/N. Private responses differ from those of the planner because of two externalities. The first is the usual infection externality: households only take into account the risk that they become infected, not the risk that they infect others. The other is is a congestion

externality: agents do not internalize the fact that their actions can increase the fatality risk for others, through an overwhelmed healthcare system. Both these factors make private mitigation lower than the socially optimal level. We show that this wedge between private and social incentives can be particularly severe early on in the outbreak. When private agents become aware of the disease, the possibility of future infection reduces the value of being careful today. We term this the fatalism effect. The planner, on the other hand, internalizes the possibility of infecting others and a congested healthcare system and, as a result, her incentives to avoid infection increase sharply when she learns of the disease.

Our quantitative results show that this wedge has significant implications for the evolution of both macro and health outcomes. In equilibrium, the representative household's private response closely tracks the path of the infection rate. At the peak, labor supply drops by about 10% and 60% of workers work from home. The result is a time-path for infections that peaks at the same time as the zero-mitigation time-path albeit at a lower level. At the peak, the fatality rate is triple the baseline level, ultimately resulting in cumulative deaths of over 1% of the population. The planner, on the other hand, acts more quickly and aggressively to flatten the curve, cutting labor supply by 20% and prescribing a work-from-home ratio of 80% at the height of the disease. The infection rate peaks a few weeks later and at a much lower level than that of the equilibrium level. The peak fatality rate and the cumulative fatalities are also significantly reduced. The ability to work from home plays a significant role in ameliorating the economic impact of the mitigation strategies. While this is true both for equilibrium strategies and the planner's choices, the latter uses this option much more intensively. If we were to abstract from this option, the planner's optimal strategy has a peak drop in consumption of almost 45% (compared to 30% in the baseline model).

We then examine the robustness of our main results to assumptions about parameters (notably the fatality rate) and the arrival of news about the disease. We also consider a version with altruistic households and find that a high degree of altruism is necessary to get close to the planner's outcomes. Finally, we use the model to guide decision-making under uncertainty about key epidemiological parameters (the reproduction number and the fatality rate). The analysis reveals that, despite this uncertainty, the planner can get quite

close to her optimal strategy by actively suppressing the disease in its early stages while waiting for incoming data to resolve the uncertainty. Intuitively, this is possible because her optimal strategy in the first few weeks remains quite similar across a wide range of fatality and infection parameters.

Literature. There are a number of papers studying the trade-off between economic and health outcomes in the context of the Covid-19 pandemic. It is impossible to cite every paper from this large body of work, so we restrict ourselves to the most closely related early ones – Barro et al. (2020), Eichenbaum et al. (2020) and Alvarez et al. (2020). Barro et al. (2020) and Correia et al. (2020) draw lessons from the 1918 flu epidemic. Barro et al. (2020) find a high death rate (about 40 million people, 2% of the population at the time) and a large but not extreme impact on the economy (cumulative loss in GDP per capita of 6% over 3 years). The impact on the stock market was small. Correia et al. (2020) find that early interventions help protect health and economic outcomes. Our paper relates to an older literature on contagion dynamics, e.g. (Diekmann and Heesterbeek, 2000). We refer to the reader to Atkeson (2020b) for a recent discussion. Berger et al. (2020) show that testing can reduce the economic cost of mitigation policies as well as reduce the congestion in the health care system. Baker et al. (2020) document the early consumption response of households in the US.

Our model shares with Eichenbaum et al. (2020) the idea of embedding SIR dynamics in a simple macroeconomic framework. The SIR block is the same, but there are some notable differences in the economic model. Eichenbaum et al. (2020) consider hand-to-mouth agents who know their health status, while we use a representative household framework in the tradition of Lucas and Stokey (1987), where both consumption and health risks are pooled, and asymptomatic agents are unaware of their status. We also allow work-from-home as a distinct margin of mitigation, along with learning-by-doing, which adds an important dynamic element. The planner invests in the new technology to mitigate future disruptions. We also highlight the dynamic tension (the so-called fatalism bias) between the planner and the private sector's incentives.

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2 Benchmark Model

2.1 Households

There is a continuum of mass N of households. Each household is of size 1 and the utility of the household is

$$U = \sum_{t=0}^{\infty} \beta^t u \left(c_t, l_t; i_t, d_t \right)$$

where c_t and l_t are per-capita consumption and labor supply. The household starts with a continuum of mass 1 of family members, all of them susceptible to the disease. At any time $t > 0$ we denote by s_t, i_t and d_t the numbers of susceptible, infected and dead people. The size of the household at time t is therefore $1 - d_t$ and total household consumption is $(1 - d_t) c_t$. Among the i_t infected members, κi_t are too sick to work. The labor force at time t is therefore $1 - d_t - \kappa i_t$, and household labor supply is $(1 - d_t - \kappa i_t) l_t$. The number of household members who have recovered from the disease is $r_t = 1 - s_t - i_t - d_t$. In our quantitative analysis, we use the functional form

$$u \left(c_t, l_t; i_t, d_t \right) = \left(1 - d_t - \kappa i_t \right) \left(\log \left(c_t \right) - \frac{l_t^{1+\eta}}{1 + \eta} \right) + \kappa i_t \left(\log \left(c_t \right) - u_\kappa \right) - u_d d_t,$$

where u_κ is the disutility from being sick and u_d the disutility from death which includes lost consumption and the psychological cost on surviving members.[1] For simplicity, we assume that sickness does not change the marginal utility from consumption. This implies that the household will equate consumption for all alive members (i.e. independent of health status). The variables s_t, i_t and d_t evolve according to a standard SIR model described below.

At the beginning of time t, each household decides how much to consume c_t (per capita) and how much each able-bodied member should work l_t. We have normalized the disutility of labor so that $l = c = 1$ before the epidemic starts. Households become infected by shopping and by going to work. We compute infection in two steps. First, we define exposure levels for shoppers and for workers. Then, we aggregate these into a composite infection rate at

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[1]Formally $u_d = PsyCost - \log \left(c_d \right)$ where c_d is the consumption equivalent in death. Technically we cannot set $c_d = 0$ with log preferences but u_d is a large number.

the household level. We assume throughout the paper that asymptomatic individuals are unaware of their infection. Formally, $\{dead\}$ and $\{sick\}$ are the only observable states at the individual level. In particular, households cannot tell the difference between the s_t members who are susceptible and the $(1 - \kappa) i_t$ members who are infected but not sick. This modeling choice is the main difference between our model and the model of Eichenbaum et al. (2020) who make the polar opposite assumption. They assume that individuals know their types, do not share risks within a family, and behave in a hand-to-mouth fashion. We follow instead a Lucas and Stokey (1987) approach to model households' decisions and risk sharing, so our model stays close to a standard representative agent model.

We take into account the possibility of a cure/vaccine by adjusting the discount factor. Formally, this is equivalent to assuming that a cure/vaccine arrives stochastically at an exogenous arrival rate. Note that this combines both a treatment – which instantly cures sick individuals – and a vaccine - which eliminates the risk of future infections. Under this assumption, the economy jumps back to $l_t = c_t = 1$ when the cure/vaccine arrives. We can therefore focus on the path along which a cure has not yet been found, along with a suitable adjustment to the discount factor. Let $\tilde{\beta}$ be the pure time discount rate and ν the likelihood of a vaccine. We define the effective discount factor $\beta = \tilde{\beta} (1 - \nu)$ along the no-cure path.

2.2 Shopping

We define consumption exposure as

$$e^c c_t C_t,$$

where e^c measures the sensitivity of exposure to consumption and C_t is aggregate consumption.[2] The idea behind this specification is that household members engage in various activities related to consumption – such as shopping in a crowded mall, eating inside a restaurant, going to a hospital – which exposes them to infection risk. These are assumed to proportional to consumption expenditure c_t and for a given level of aggregate consumption, exposure is proportional to such activities. We study heterogeneity across sectors later

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[2]We use lower-case letters to denote household level variables and upper-case for aggregates.

in the paper.

2.3 Production and Working from Home

Production uses only labor, but a key feature of our model is the distinction between hours supplied by able bodied workers l_t and effective labor supply $\hat{l}_t$ per household. Effective labor supply is

$$\hat{l}_t = (1 - d_t - \kappa i_t) \left(l_t - \frac{\chi_t}{2} m_t^2 \right)$$

The first term captures the fact that the number of valid household member is decreased by death and sickness. The second term captures the cost of implementing mitigation strategies, denoted m_t (e.g., working from home at least some of the time). These strategies come at a cost – lost productivity, denoted by the term $\frac{\chi_t}{2} m_t^2$. The loss process χ_t is a decreasing function of the accumulated experience working from home and is given by:

$$\chi_t = \chi \left(\overline{m}_t \right)$$

where $\overline{m}_t$ is the stock of accumulated mitigation (with a depreciation rate of $1 - \rho_m$)

$$\overline{m}_{t+1} = \rho_m \overline{m}_t + m_t.$$

The function χ is positive, decreasing, and convex. In the quantitative analysis, we assume the following functional form

$$\chi_t = \bar{\chi} \left(1 - \Delta_\chi \left(1 - \exp \left(-\overline{m}_t \right) \right) \right)$$

The cost shifter initially (i.e. when $\overline{m}_t = 0$) is equal $\bar{\chi} > 0$ and then falls over time as people figure out how to work from home. The parameter Δ_χ indexes the maximum potential for learning by doing, since as $\overline{m}_t$ becomes large, the cost approaches $\bar{\chi} (1 - \Delta_\chi)$. The benefit of mitigation strategies is a reduction in the risk of infection. Specifically, exposure from work is given by

$$e^l \left(1 - m_t \right) l_t \left(1 - M_t \right) L_t$$

where, as before, upper-case letters denote aggregates. The aggregate resource constraint is

$$Y_t = C_t = \hat{L}_t = N\hat{l}_t$$

In our basic model, we ignore the issue of firm heterogeneity and market power. Therefore, price is equal to marginal cost

$$P_t = W_t = 1$$

where W_t is the wage per unit of effective labor, which we normalize to one.

2.4 Income and Contagion

At the end of each period, household members regroup and share income and consumption. Household labor income is $W_t\hat{l}_t = \hat{l}_t$ and the budget constraint is

$$(1 - d_t) c_t + \frac{b_{t+1}}{1 + r_t} \leq b_t + \hat{l}_t$$

Household exposure is

$$e_t = \bar{e} + (1 - d_t) e^c c_t C_t + (1 - d_t - \kappa i_t) e^l (1 - m_t) l_t (1 - M_t) L_t,$$

where $\bar{e}$ is baseline exposure, independent of market activities. Contagion dynamics follow a modified SIR model (the benchmark model is explained in the appendix):

$$s_{t+1} = s_t - \gamma e_t \frac{I_t}{N} s_t$$
$$i_{t+1} = \gamma e_t \frac{I_t}{N} s_t + (1 - \rho) i_t - \delta_t \kappa i_t$$
$$d_{t+1} = d_t + \delta_t \kappa i_t$$
$$r_{t+1} = r_t + \rho i_t$$

where γ is the infection rate per unit of exposure, ρ the recovery rate, κ the probability of being sick conditional on infection, and δ_t the mortality rate of sick patients. In the standard SIR model, exposure is assumed to be constant, whereas here it depends on consumption,

labor supply and mitigation strategies. Note that the number of new infected, $\gamma e_t \frac{I_t}{N} s_t$, is a function of the aggregate infection rate, taken as given by households. Finally, the mortality rate δ_t is described by an increasing function of the measure of sick agents:

$$\delta_t = \delta \left(\kappa I_t \right)$$

which captures the idea that an overloaded healthcare system can contribute to higher fatalities.

2.5 Market Clearing and Aggregate Dynamics

Infection dynamics for the the entire population are simply given by the SIR system above with aggregate variable $I_t = N i_t$, and so on. The aggregate labor force is $N \left(1 - \kappa i_t - d_t \right) l_t$ and total consumption is $N \left(1 - d_t \right) c_t$. The market clearing conditions are

$$\left(1 - d_t \right) c_t = \hat{l}_t,$$

and for the bond market

$$b_t = 0.$$

3 Decentralized equilibrium

3.1 Equilibrium Conditions

Since our model reduces to a representative household model and since $b = 0$ in equilibrium, we simply omit b from the value function. The household's optimal choices are the solution to the following recursive problem:

$$V \left(i_t, s_t, d_t, \overline{m}_t \right) = \max_{c_t, l_t, m_t} u \left(c_t, l_t; i_t, d_t \right) + \beta V \left(i_{t+1}, d_{t+1}, s_{t+1}, \overline{m}_{t+1} \right)$$

where $\bar{m}_{t+1} = \bar{m}_t + m_t$ and

$$u \left(c_t, l_t; i_t, d_t \right) = \left(1 - d_t \right) \log \left(c_t \right) - \left(1 - d_t - \kappa i_t \right) \frac{l_t^{1+\eta}}{1 + \eta} - u_\kappa \kappa i_t - u_d d_t \qquad ,$$

with effective labor given by

$$\hat{l}_t = (1 - d_t - \kappa i_t)\left(l_t - \frac{\chi_t}{2}m_t^2\right)$$

$$\chi_t = \bar{\chi}\left(1 - \Delta_\chi\left(1 - \exp\left(-\overline{m}_t\right)\right)\right)$$

The Lagrangian is then:

$$V_t = u\left(c_t, l_t; i_t, d_t\right) + \beta V_{t+1} + \lambda_t\left(\hat{l}_t + b_t - (1 - d_t)\,c_t - \frac{b_{t+1}}{1 + r_t}\right)$$

$$+ \lambda_{e,t}\left(e_t - \bar{e} - (1 - d_t)\,e^c c_t C_t - (1 - d_t - \kappa i_t)\,e^l\left(1 - m_t\right) l_t\left(1 - M_t\right) L_t\right)$$

$$+ \lambda_{i,t}\left(i_{t+1} - \gamma e_t \frac{I_t}{N} s_t - (1 - \rho)\,i_t + \delta_t \kappa i_t\right)$$

$$+ \lambda_{s,t}\left(s_{t+1} - s_t + \gamma e_t \frac{I_t}{N} s_t\right)$$

$$+ \lambda_{d,t}\left(d_{t+1} - d_t - \delta_t \kappa i_t\right)$$

The first order conditions for consumption and labor are then

$$c_t: \quad c_t^{-1} = \lambda_t + \lambda_{e,t} e^c C_t$$

$$l_t: \quad l_t^{\eta} = \lambda_t - \lambda_{e,t} e^l\left(1 - m_t\right)\left(1 - M_t\right) L_t$$

$$m_t: \quad \lambda_t \chi_t m_t = \frac{\beta V_{\bar{m},t+1}}{1 - d_t - \kappa i_t} + \lambda_{e,t} e^l l_t\left(1 - M_t\right) L_t$$

The remaining first order conditions are

$$e_t: \lambda_{e,t} = \left(\lambda_{i,t} - \lambda_{s,t}\right)\gamma \frac{I_t}{N} s_t$$

$$i_{t+1}: \lambda_{i,t} = -\beta V_{i,t+1}$$

$$s_{t+1}: \lambda_{s,t} = -\beta V_{s,t+1}$$

$$d_{t+1}: \lambda_{d,t} = -\beta V_{d,t+1}$$

The envelope conditions are

$$V_{i,t} = \kappa \frac{l_t^{1+\eta}}{1+\eta} - \kappa u_\kappa - \kappa \lambda_t \left(l_t - \frac{\chi_t m_t^2}{2} \right) + \lambda_{e,t} \kappa e^l (1 - m_t) l_t (1 - M_t) L_t - (1 - \rho) \lambda_{i,t} + \delta_t \kappa (\lambda_{i,t} - \lambda_{d,t})$$

$$V_{s,t} = (\lambda_{s,t} - \lambda_{i,t}) \gamma e_t \frac{I_t}{N} - \lambda_{s,t}$$

$$V_{d,t} = \frac{l_t^{1+\eta}}{1+\eta} - \log(c_t) - u_d - \lambda_t \left(l_t - \chi_t m_t^2 - c_t \right) + \lambda_{e,t} \left(e^c c_t C_t + e^l (1 - m_t) l_t (1 - M_t) L_t \right) - \lambda_{d,t}$$

$$V_{\overline{m},t} = \beta V_{\overline{m},t+1} + \lambda_t (1 - d_t - \kappa i_t) \frac{\overline{\chi}}{2} m_t^2 \Delta_\chi \exp(-\overline{m}_t)$$

3.2 Equilibrium with Exogenous Infections

To simplify the notation, we normalize $N = 1$, so we should think of our values as being per-capita pre-infection. When there is no risk of contagion, i.e. $i_t = 0$, $\lambda_{e,t} = 0$ and $V_{\overline{m},t+1} = 0$, we have $m_t = 0$ and from the optimal consumption and labor supply

$$c_t^{-1} = l_t^\eta$$

Since $m = 0$ we have $\hat{l}_t = l_t$ so market clearing is simply

$$c_t = l_t.$$

Combining these two conditions we get

$$c_t = l_t = 1$$

The pre-infection economy is always in steady state.

Consider now an economy with exogenous SIR dynamics: $e^c = e^l = 0$. This implies $m_t = 0$ and

$$c_t^{-1} = l_t^\eta.$$

Market clearing requires

$$(1 - d_t) c_t = (1 - d_t - \kappa i_t) l_t$$

therefore

$$l_t^{1+\eta} = 1 + \frac{\kappa i_t}{1 - d_t - \kappa i_t}$$

The labor supply of valid workers increases to compensate for the reduced productivity of the sick. Per capita consumption is

$$c_t = \left(\frac{1 - d_t}{1 - d_t - \kappa i_t} \right)^{-\frac{\eta}{1+\eta}}$$

As long as $\eta > 0$ consumption per capita decreases. Aggregate GDP decreases because of lost labor productivity and deaths.

The SIR system is independent from the economic equilibrium. As described in the Appendix, the share of infected agents I_t increases, reaches a maximum and converges to 0 in the long run. Assuming a constant δ, the long run solution solves

$$\log \left(\frac{S_\infty}{1 - I_0} \right) = -\frac{\gamma \bar{e}}{\rho + \delta \kappa} \left(\frac{1 - S_\infty}{N} \right),$$

and

$$D_\infty = \frac{\delta \kappa}{\delta \kappa + \rho} \left(1 - S_\infty \right).$$

When the congestion externality arises and δ_t increases, then we cannot obtain a closed-form solution for the long run death rate but the qualitative results are unchanged. The following proposition summarizes our results.

Proposition 1. *When contagion does not depend on economic activity $\left(e^c = e^l = 0 \right)$, the share of infected agents I_t increases, reaches a maximum and converges to 0 in the long run. The long run death rate is given by $D_\infty = \frac{\delta \kappa}{\delta \kappa + \rho} \left(1 - S_\infty \right)$ where the long run share of uninfected agents solves $\log \left(\frac{S_\infty}{1 - I_0} \right) = -\frac{\gamma \bar{e}}{\rho + \delta \kappa} \left(\frac{1 - S_\infty}{N} \right)$. Along the transition path, labor supply of able-bodied workers follows the infection rate while per-capita consumption moves in the opposite direction as $c_t = \left(1 - \frac{\kappa i_t}{1 - d_t} \right)^{\frac{\eta}{1+\eta}}$.*

3.3 Private Incentives for Mitigation

In this subsection, we characterize incentives of households to undertake mitigation. The incentives to mitigate depend on the shadow value of exposure $\lambda_{e,t} = (\lambda_{i,t} - \lambda_{s,t}) \gamma \frac{I_t}{N} s_t$ which is increasing in new infections, $\gamma \frac{I_t}{N} s_t$. So, for a given $\lambda_{i,t} - \lambda_{s,t}$, the private incentives to mitigate are proportional to the number of new cases. Now,

$$\lambda_{i,t} - \lambda_{s,t} = \beta \left(V_{s,t+1} - V_{i,t+1} \right)$$

with

$$V_{s,t} - V_{i,t} = u_\kappa \kappa + \kappa l_t \left(\lambda_t - \frac{l_t^\eta}{1+\eta} \right) - \rho \lambda_{i,t} + \left(1 - \gamma e_t \frac{I_t}{N} \right) \left(\lambda_{i,t} - \lambda_{s,t} \right) + \delta_t \kappa \left(\lambda_{d,t} - \lambda_{i,t} \right)$$

$$= u_\kappa \kappa + \kappa l_t \left(\lambda_t - \frac{l_t^\eta}{1+\eta} \right) - \rho \lambda_{i,t} + \left(1 - \gamma e_t \frac{I_t}{N} \right) \beta \left(V_{s,t+1} - V_{i,t+1} \right) + \delta_t \kappa \beta \left(V_{i,t+1} - V_{d,t+1} \right)$$

Fatalism Effect We now use an approximation to obtain a sharper characterization and gain more intuition. Specifically, we make the following assumptions (i) the non-monetary cost of death u_d is sufficiently large that it dominates the other terms in the expression for $V_{d,t}$ and $V_{i,t}$ (ii) there are no congestion effects on fatality, i.e. $\delta_t = \delta$. Then, the value of death and infection are approximately constant, i.e.

$$V_{d,t} \approx -\frac{u_d}{1-\beta}$$

$$V_{i,t} \approx \sum_{s=0}^{\infty} \beta^s (1 - \rho - \delta\kappa)^s \kappa \left(-u_\kappa + \delta \frac{\beta u_d}{1-\beta} \right) = \frac{\kappa u_\kappa + \delta\kappa \frac{\beta}{1-\beta} u_d}{1 - \beta (1 - \rho - \delta\kappa)} \equiv V_i$$

The value of avoiding an infection at time t is the discounted value of the disutility from sickness and death. The value of remaining susceptible is then

$$V_{s,t} \approx V_i \sum_{s=1}^{\infty} \beta^s \left(1 - \gamma e_{t+s-1} \frac{I_{t+s-1}}{N} \right)^{s-1} \gamma e_{t+s-1} \frac{I_{t+s-1}}{N}$$

Importantly, $V_{s,t}$ falls with the risk of infection in the near future. Formally,

$$\frac{\partial V_{s,t}}{\partial \frac{I_{t+s-1}}{N}} = V_i \beta^s \left[\left(1 - \gamma e_{t+s-1} \frac{I_{t+s-1}}{N} \right)^{s-1} \gamma e_{t+s-1} - (s-1) \left(1 - \gamma e_{t+s-1} \frac{I_{t+s-1}}{N} \right)^{s-2} (\gamma e_{t+s-1})^2 \frac{I_{t+s-1}}{N} \right]$$

$$= V_i \beta^s \left(1 - \gamma e_{t+s-1} \frac{I_{t+s-1}}{N} \right)^{s-1} \gamma e_{t+s-1} \left[1 - (s-1) \frac{\gamma e_{t+s-1} \frac{I_{t+s-1}}{N}}{1 - \gamma e_{t+s-1} \frac{I_{t+s-1}}{N}} \right]$$

The term inside the square bracket is positive (or equivalently, $\frac{\partial V_{s,t}}{\partial \frac{I_{t+s-1}}{N}}$ is negative) for low s. Since $V_{i,t}$ is approximately constant, this means that the difference $V_{s,t} - V_{i,t}$ *shrinks* when agents become aware of the disease, i.e. there is a perverse effect on incentives to mitigate. We term this channel *the fatalism effect*: early on, agents (correctly) anticipate that they are likely to become infected and so have weak incentives to avoid infection today. As we will see, the planner's solution considers other forces that offset this channel.

When δ is constant the fatalism effect rationally reduces privates incentives to mitigate the disease, i.e., $V_{s,t} - V_{i,t}$ is small, but it does not switch the sign, i.e., $V_{s,t} - V_{i,t}$ remains positive. This can change when δ is time varying as we discuss later.

4   Planner's Problem

Again, we normalize $N = 1$ for simplicity. The planner solves

$$\max U = \sum_{t=0}^{\infty} \beta^t u \left(C_t, L_t; I_t, D_t \right)$$

subject to

$$u \left(C_t, L_t; I_t, D_t \right) = (1 - D_t) \log (C_t) - (1 - D_t - \kappa I_t) \frac{L_t^{1+\eta}}{1+\eta} - u_\kappa \kappa I_t - u_d D_t$$

and

$$(1 - D_t) C_t = (1 - D_t - \kappa I_t) \left(L_t - \frac{\chi_t}{2} (M_t)^2 \right).$$

The first order conditions for consumption and labor are then (highlighted in red are the differences with the decentralized equilibrium)

$$C_t : \ C_t^{-1} = \lambda_t + \textcolor{red}{2\lambda_{e,t}e^c C_t}$$

$$L_t : \ L_t^{\eta} = \lambda_t - \textcolor{red}{2\lambda_{e,t}e^l (1 - M_t)^2 L_t}$$

$$M_t : \ \lambda_t \chi_t M_t = \frac{\beta V_{\bar{M},t+1}}{1 - D_t - \kappa I_t} + \textcolor{red}{2\lambda_{e,t}e^l (1 - M_t) L_t^2}$$

The marginal utilities of the planner with respect to exposure are twice as high as those of the private sector because of the contagion externalities: private agents only care about how their behavior affect their own infection risk. They do not care about how their behavior affects the infection risk of others.

The envelope condition that changes (relative to the equilibrium) is the one with respect to the number of infected people:

$$V_{I,t} = \kappa \frac{L_t^{1+\eta}}{1+\eta} - \kappa u_\kappa - \kappa \lambda_t \left(L_t - \chi_t M_t^2 \right) + \lambda_{e,t} \kappa e^l (1 - M_t)^2 L_t^2 - (1 - \rho) \lambda_{I,t}$$

$$-\gamma e_t S_t (\lambda_{I,t} - \lambda_{S,t}) - \left(\delta_t \kappa + \textcolor{red}{\delta_t' \kappa^2 I_t} \right)(\lambda_{D,t} - \lambda_{I,t})$$

where δ_t' denotes the derivative of the fatality rate with respect to the sick population. Let us now consider the planner's incentives to reduce consumption today in response to .

4.1   Incentives to Mitigate

Next, we characterize the planner's mitigation incentives and contrast them with the private incentives studied in section 3.3. The planner's incentives to avoid infection today depend on $V_{S,t+1} - V_{I,t+1}$. Under the same simplifying assumptions as before, $V_{D,t}$ is approximately constant (and the same as that of private agents):

$$V_D \approx -\frac{u_d}{1-\beta},$$

while the values of being infected and remaining susceptible are

$$V_{I,t} \approx \sum_{s=0}^{\infty} \beta^s \left(\prod_{\tau=0}^{s-1} (1 - \rho + \gamma e_{t+\tau} S_{t+\tau} - \delta \kappa) \right) \left(\kappa u_\kappa + \delta \kappa \frac{\beta}{1-\beta} u_d \right)$$

$$V_{S,t} = \sum_{s=1}^{\infty} \beta^s \left(1 - \gamma e_{t+s-1} \frac{I_{t+s-1}}{N} \right)^{s-1} V_{I,t+s} \gamma e_{t+s-1} \frac{I_{t+s-1}}{N}$$

The expression for $V_{S,t}$ shows that the planner also suffers from the fatalism effect: risk of future infection reduces the value of remaining safe today. But there is an additional effect which arises because of the $\gamma e_t S_t$ term in the first equation. This implies that the value of infection $V_{I,t}$ is amplified (i.e. becomes even more negative) when the planner becomes aware of the disease. Intuitively, the possibility of spreading makes the effective recovery rate from the planner's point of view equal to $\rho - \gamma e_t S_t$ is instead of ρ.

Thus, from the planner's point of view, both $V_{I,t}$ and $V_{S,t}$ become more negative when the disease arrives. The net effect, which drives her incentives to mitigate, depend on the relative strength of the two forces. Early on, the risk of an infected agent spreading the disease to others is quite high (since S_t is high) while the fatalism effect (which depends on $\frac{I_{t+\tau}}{N}$) is relatively muted, and therefore, $V_{S,t} - V_{I,t}$ rises, i.e. the planner finds it optimal to increase mitigation as soon as she becomes aware of the disease.

Finally, while we abstracted from congestion effects in this discussion for simplicity, re-introducing them creates additional incentives for the the planner to mitigate. To see why, note that with congestion effects, the planner's value of infection is given by (the terms that change relative to the case without congestion are highlighted in red):

$$V_{I,t} = \sum_{s=0}^{\infty} \beta^s \left(\prod_{\tau=0}^{s-1} (1 - \rho + \gamma e_{t+\tau} S_{t+\tau} - \delta_{t+\tau} \kappa) \right) \left(\kappa u_\kappa + \delta_{t+s} \kappa \frac{\beta}{1-\beta} u_d + \delta'_{t+s} \kappa^2 I_{t+s} \frac{\beta}{1-\beta} u_d \right)$$

Thus, the planner hates infection even more in the presence of congestion effects. This externality introduces another wedge between the private and social incentives to mitigate. This tension helps explain episodes like the Florida spring break controversy. In March 2020, as the arrival of the pandemic became broadly known in the US, many people chose to enjoy their spring break, arguing that if they were going to catch the virus, now would be as good a time as later, while public officials worried about the spread of the disease and

hospitals being overwhelmed.

5   Calibration

Before detailing our calibration strategy, we note that considerable uncertainty about key parameters remains even now. For an early discussion of the challenges, see Atkeson (2020a). We return to this issue later in the paper. We calibrate our model at the weekly frequency.

Contagion   The SIR block of the model is parameterized as follows. The recovery parameter is set to $\rho = 0.35$ following Atkeson (2020b). This value implies that the average duration for which an infected agent is contagious is about 20 days. The fraction of infected people who fall sick is $\kappa = 0.15$. For the exposure function, we normalize $\bar{e} + e^c + e^l = 1$ and set $e^c = e^l = \frac{1}{3}$, consistent with the estimates in Ferguson (2020). These parameters imply that total exposure $e = 1$ at the pre-pandemic levels of consumption and labor (the calibration of production and utility parameters will be described later). The parameter γ is then chosen to target the basic reproduction number (i.e. the average number of people infected by a single infected individual) of $\mathcal{R} = 3.3$ consistent with the estimate of Salje et al. (2020), yielding an estimated value of $\gamma = 1.2$. Finally, to parameterize the fatality rate and the congestion effects, we adopt the following functional form for the probability of death δ_t :

$$\delta_t = \bar{\delta} + \exp\left(\phi I_t\right) - 1$$

where the parameter ϕ indexes the strength of the congestion externality. We set $\bar{\delta}$ and ϕ to match two targets for the infection mortality rate:[3] a baseline value (the fraction of infected people who die even in the absence of congestion i.e. with $\delta_t = \bar{\delta}$) of 0.5% and an 'extreme' value of 1.5% when 3% of the population requires medical attention (i.e. the fraction of people who die when $\kappa I = 0.15 \times 0.2 = 0.03$). This procedure yields $\bar{\delta} = 0.012$ and $\phi = 0.8$. We examine the robustness of our results to these assumptions about fatality rates later in the paper.

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[3]The infection fatality rate is given by $\frac{\delta_t \kappa}{\delta_t \kappa + \rho}$.

Preferences and technology The utility parameter u_d is set to a value of 10, implying a flow disutility from death that is ten times per capita income. Such large non-monetary costs associated with loss of life are consistent with estimates in the literature and with values used by government entities like the EPA. For example, Greenstone and Nigam (2020) use an estimated value of a statistical life of \$11.5 million (in 2020 dollars) to the household from death. Assuming a rate of return of 5%, this translates into an annual flow value of \$575,000, or roughly 10 times per capita GDP. The flow disutility from sickness u_κ is set to a value of 0.5.

As we discussed earlier, our discount factor β is meant to captures both time discounting and the possibility of a cure/vaccine arriving at an constant, exogenous rate. The pure time discounting component corresponds to an annual discount factor of 0.95. For the cure/vaccine, we take a relatively pessimistic case as our baseline, with the annualized probability of 15%. The composite annual discount factor thus becomes 0.8, or equivalently, a weekly $\beta = (0.8)^{\frac{1}{52}} = 0.9957$.

Next, we calibrate the working-from-home technology, which involves picking values for two parameters – $\bar{\chi}$ and Δ_χ. For the former, we use estimates of the effect of the restrictions imposed by many countries in February and March 2020. A back-of-the-envelope calculation suggests that a policy which requires almost the entire labor force to work from home (i.e. $m \approx 1$) with no prior experience ($\overline{m} \approx 0$) causes GDP to fall by 25% below its normal level. Since this initial productivity loss is exactly equal to $\frac{\bar{\chi}}{2}$, we set $\bar{\chi} = 0.5$. Finally, for the long-run parameter, Δ_χ, we rely on Dingel and Neiman (2020), who estimate that roughly one-third of the jobs in the US can be done from home. Guided by this estimate, we set $\Delta_\chi = 0.34$. We also assume that accumulated learning depreciates very slowly, i.e. $\rho_m = 0.99$, which implies that the stock of knowledge evolves according to $\bar{m}_{t+1} = 0.99\bar{m}_t + m_t$.

6 Quantitative Results

Our benchmark exercise assumes an initial infection rate of $i_0 = 1\%$ for expositional purposes (it makes the figures easier to read in the early periods), but it is important to keep

in mind that this is a large shock. It seems likely that agents and policy makers become aware of the epidemic much earlier, so in our robustness analysis, we also report simulations starting at $i_0 = 0.1\%$.

Private Response We start with the decentralized solution. Figure 1 and Figure 2 show the behavior of the contagion and macro variables in the decentralized equilibrium, under three different assumptions about exposure and mitigation strategies. The blue line solid shows a situation where infection rates are exogenous, i.e. do not vary with the level of economic activity. Since infection is assumed to be exogenous, agents do not engage in mitigation, i.e., they ignore the pandemic. In fact, labor input rises (the solid line, top left panel in Figure 2), while per-capita consumption falls by about 2.5% (the dashed line), as able-bodied workers work harder to compensate for the workers who are sick. This is clearly not a realistic assumption, but it serves as a useful benchmark for the worst case scenario. In this scenario, almost the entire population is eventually infected and 1.75% of the population succumbs to the virus (bottom, left panel in Figure 1). The case mortality rate peaks at 2.5% roughly 15 weeks after the initial infection when about 6% ($\kappa \times I = 15\%$ of the peak infection rate of 40%) of the population is sick and the healthcare system is overwhelmed.

The red line describes the case where exposure is endogenous (i.e. varies with consumption and labor) but there is no work-from-home (WFH) technology, i.e, the only ways for the household to reduce exposure are to cut back on its consumption and labor supply. As we would expect, this leads to a sharp reduction in economic activity (top, left panel in Figure 2) by almost 30%. Importantly, the reduction is gradual, tracking the overall infection rate (consumption and labor hit their trough at 17 weeks for). Intuitively, when the fraction of infected people is low (as is the case in the early stages), a reduction in exposure has a small effect on future infection risk, relative to the resulting fall in consumption. And since each household does not internalize the effect it has on the future infection rate, it has little incentive to indulge in costly mitigation early on. This dynamic is reflected in the hump-shaped pattern in λ_e (the bottom, middle panel in Figure 2). As we will see, the planner's incentives change much more strongly at the beginning. The mitigation behavior

Figure 1: Decentralized Equilibrium: Contagion Dynamics
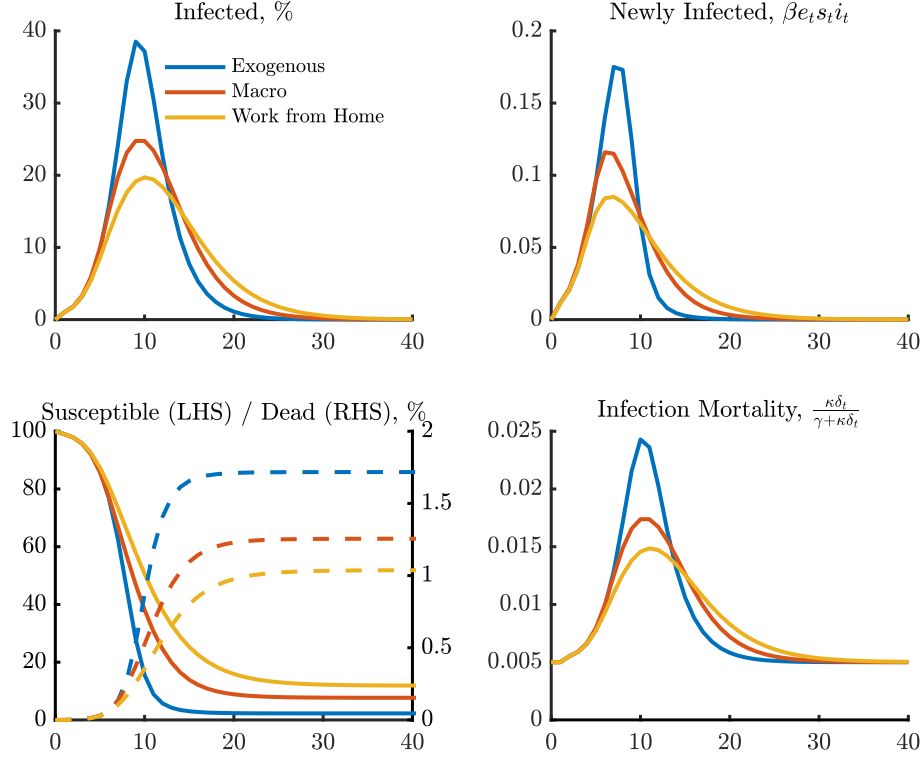
does lower the cumulative infection and death rates (relative to the exogenous infection case), by about 5% and 0.5% respectively.

Finally, the yellow line shows the effect of access to the WFH technology. This allows the household to reduce exposure without sacrificing consumption – now, the peak loss in consumption is 20% (compared to 30% without the WFH option), even as the exposure falls by more (0.55 compared to 0.65, bottom left panel in Figure 2). The fraction of time spent working from home (top, left panel in Figure 2) is hump-shaped, peaking at 60% at roughly the same time as the fraction of infected rate. This additional flexibility also lowers cumulative fatalities to 1%. However, the timing of mitigation strategies is mostly unchanged – households in the decentralized economy do not find it optimal to front-load their mitigation efforts.

Optimal Response   We now turn to the planner's solution, depicted in Figures 3 and 4. As before, the blue, red and yellow lines show the cases of exogenous infection, mitigation without WFH and mitigation with WFH respectively. As the yellow and red curves in

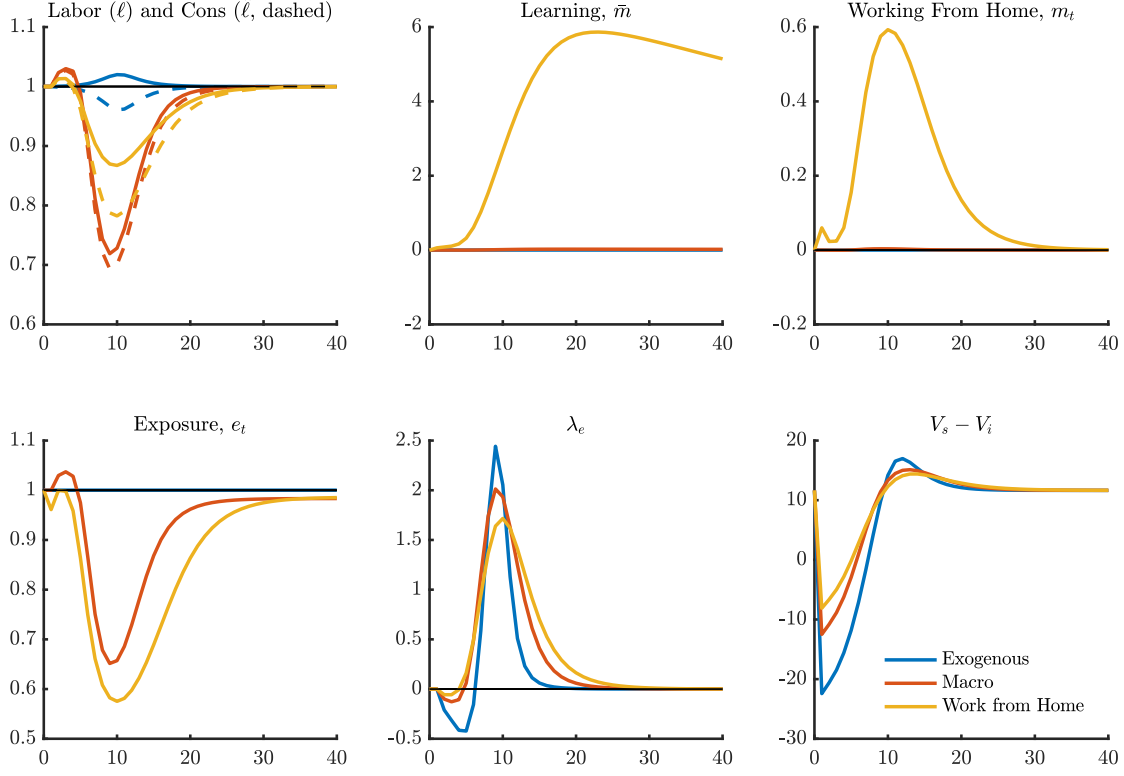Figure 2: Decentralized Equilibrium: Aggregates

Figure 3 show, the planner finds it optimal to "flatten the curve" more dramatically than agents in equilibrium. The peak infection rates are well below the decentralized equilibrium levels (10% versus 20%), leading to a lower peak fatality rate (1% versus 1.5%) as well as cumulative deaths (0.7%, compared to slightly over 1% in the decentralized equilibrium with WFH). To achieve this, the planner has to cut by more than 50%. Of course, this pushes the economy into a deeper recession with consumption falling by more than 30% even with WFH (top left panel in Figure4). The planner's response also displays a hump-shaped pattern, rising with the infection rate, but she does step on the brakes sooner (almost immediately) compared to private agents.

The availability of the WFH technology ameliorates the economic impact of the planner's suppression strategy, but does not significantly alter the contagion dynamics. Intuitively, fatalities are so costly that the planner aggressively suppresses the infection even in the absence of the WFH option: access to WFH simply allows the planner to achieve the same exposure outcomes at a lower cost. Notice also that the planner's incentives to use the WFH
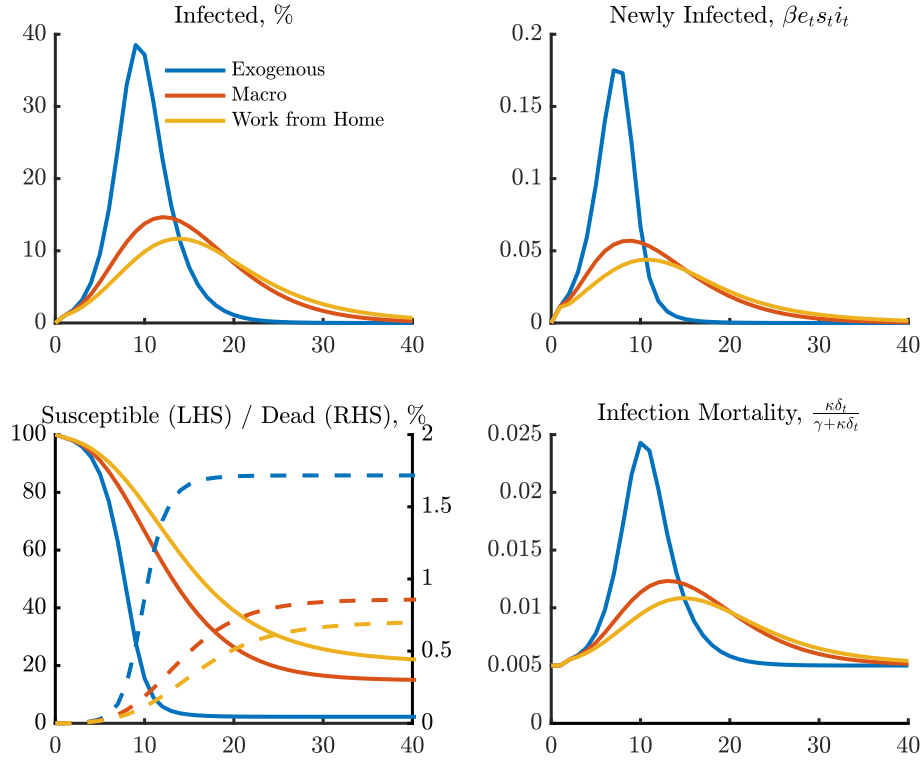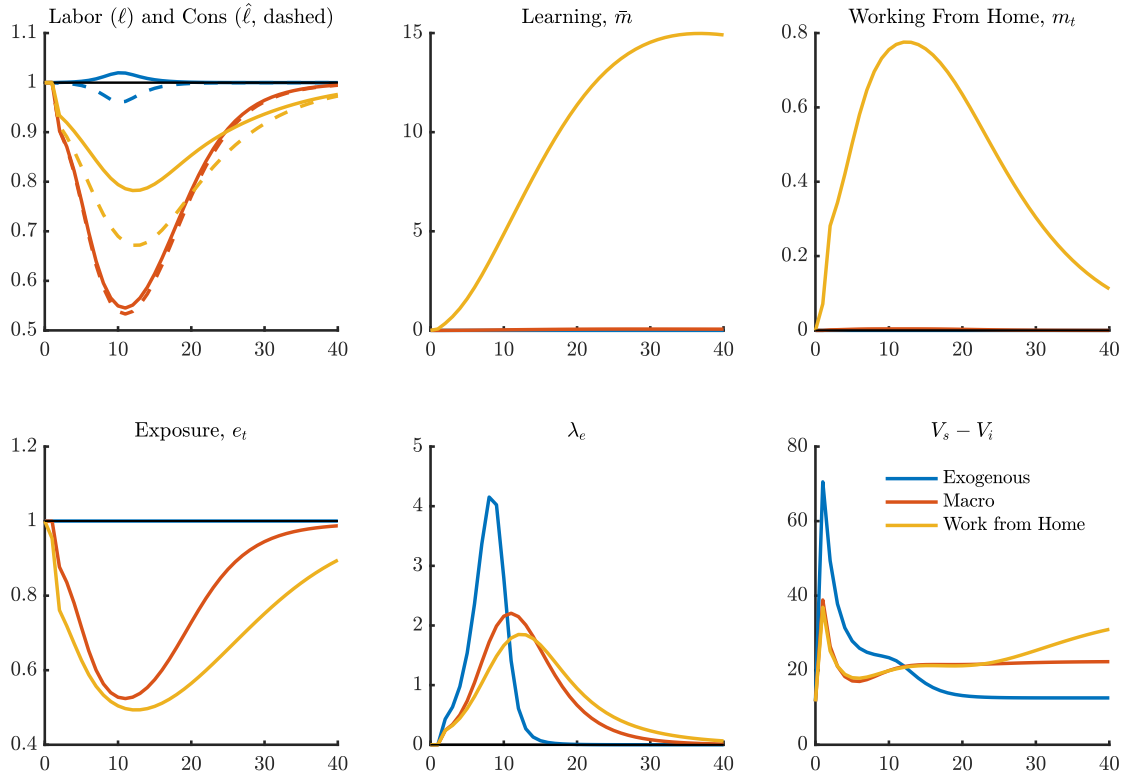
Figure 3: Planner Solution: Contagion Dynamics

Infected, %

Newly Infected, $\beta e_t s_t i_t$

Susceptible (LHS) / Dead (RHS), %

Infection Mortality, $\frac{\kappa \delta_t}{\gamma + \kappa \delta_t}$

Exogenous
Macro
Work from Home

Figure 4: Planner Solution: Aggregates

Labor (ℓ) and Cons ($\hat{\ell}$, dashed)

Learning, $\bar{m}$

Working From Home, m_t

Exposure, e_t

λ_e

$V_s - V_i$

Exogenous
Macro
Work from Home

technology are much stronger than that of private agents in a decentralized equilibrium: partly because her shadow value of exposure is higher, but also because she attaches a greater value to the future benefits of accumulated knowledge. As a result, m rises to as high as 0.8 relatively quickly. In other words, as long as a vaccine is not available, the planner has almost 80% of the population working from home at the peak of the disease.

Private versus Public Incentives Figure 5 illustrates the fatalism effect. The top graphs assume a constant risk of death δ. On the left we see the shrinking private incentives for safety early on, as $V_{s,t} - V_{i,t}$ decreases. On the right, we see that $V_{s,t} - V_{i,t}$ increases for the planner. The lower graphs illustrate another incentive problem that arises when δ is time-varying. If agents anticipate congestion in the future and if they think they are likely to become infected, they might prefer to increase their risk of infection today because it is better to be sick when δ is still relatively low. Not only does $V_{s,t} - V_{i,t}$ shrink, its sign can even flip, as we see in the bottom left panel. For the planner, by contrast, the risk of a higher δ increases the incentives to mitigate. We conclude that the misalignment of private incentives is larger during early stages of the epidemic, and is amplified by congestion externalities.

Smaller initial shock What is the value of an early warning? Suppose the planner becomes aware of the disease at $i_0 = 0.1\%$ (in the baseline, $i_0 = 1\%$). Figures 6 and 7 show her optimal response in this case. The changes relative to the $i_0 = 1\%$ case are relatively small – the extent of mitigation, work-from-home and the final death count are all quite similar across these cases (of course, as we would expect, the peaks occur slightly later with a smaller initial infection). These results suggest a modest value to detecting the disease at an earlier stage.

Lower fatality As we discussed earlier, there is little agreement in the literature on the mortality of Covid-19, with Atkeson (2020a) suggesting that the range of plausible values could be as wide as 0.1% to 1%. To explore the effect of this uncertainty, we repeat our analysis of the planner's problem with mortality rates that are a tenth of our baseline values, i.e. probability of death (conditional on being sick) is now given by $\frac{1}{10}\delta\left(\kappa I_t\right)$. The results

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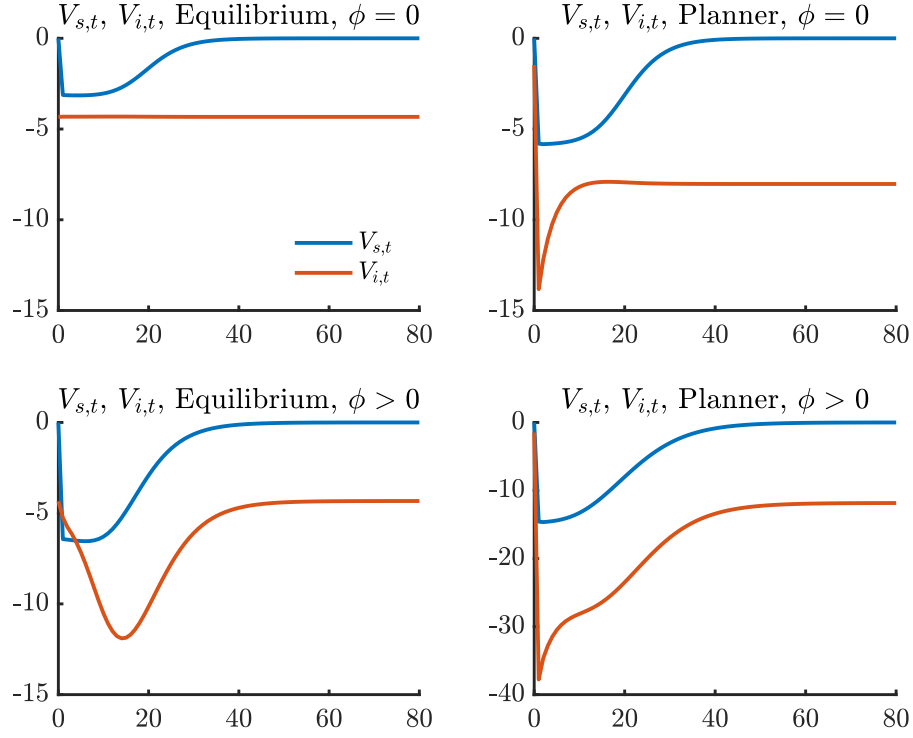Figure 5: Fatalism and Perverse Incentives

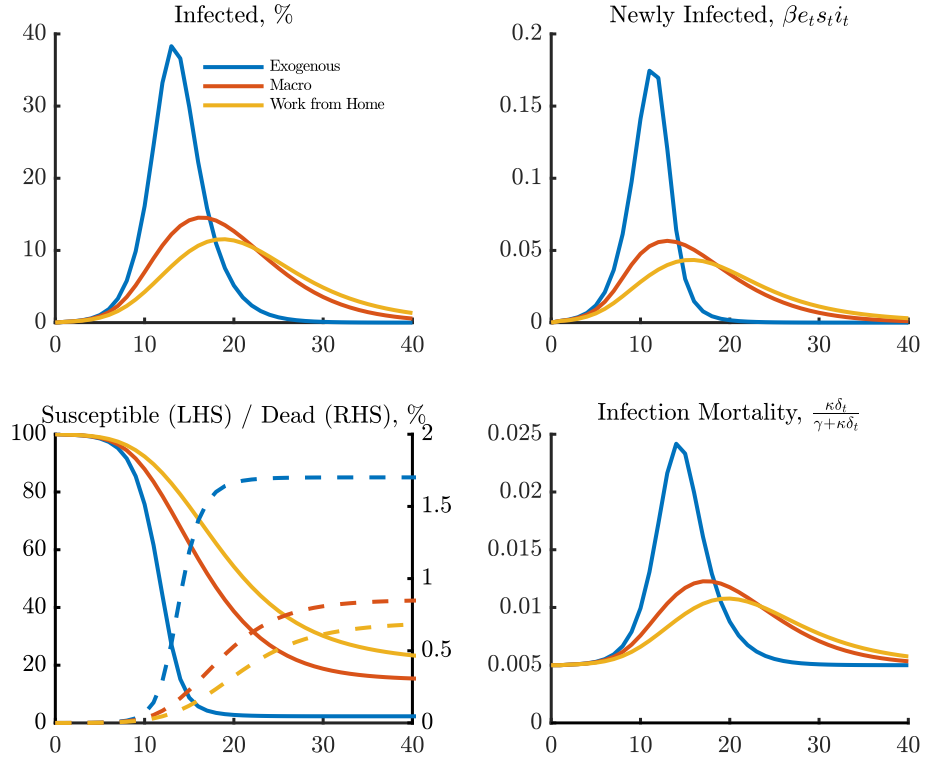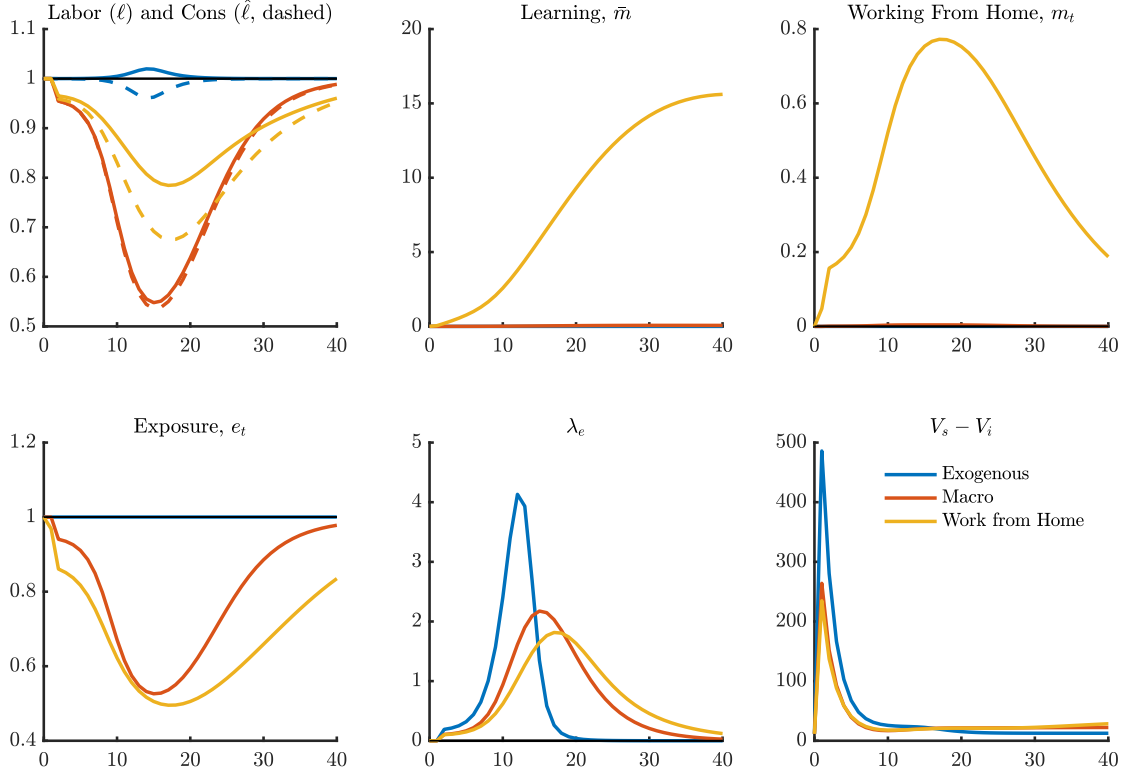Figure 6: Planner Solution: Contagion Dynamics with Smaller Infection Shock

Figure 7: Planner Solution: Aggregates with Smaller Infection Shock

are presented in Figures 10 and 11. As expected, the overall number of fatalities are much lower, as is the economic cost. The profile of the planner's solution is unchanged: she still finds it optimal to begin mitigation almost immediately.

Congestion externality Next, to isolate the role of the congestion externality, we repeat our analysis of the planner's solution with the parameter ϕ set to 0. The results, shown in Figures 10 and 11, display a similar pattern as Figures 3 and 4, but with a much less severe contraction. Intuitively, a healthcare system with sufficient slack capacity allows the planner to achieve similar outcomes in terms of fatalities with modestly higher infection rates or equivalently, with less mitigation (top, left panel in Figure 11). Accordingly, the recession is not as deep or persistent than in the baseline.

Altruistic households Finally, we consider the possibility that households partly internalize their effects on the aggregate outcome. Formally, we assume that households incorporate externalities with a weight τ while making their consumption, labor and miti-

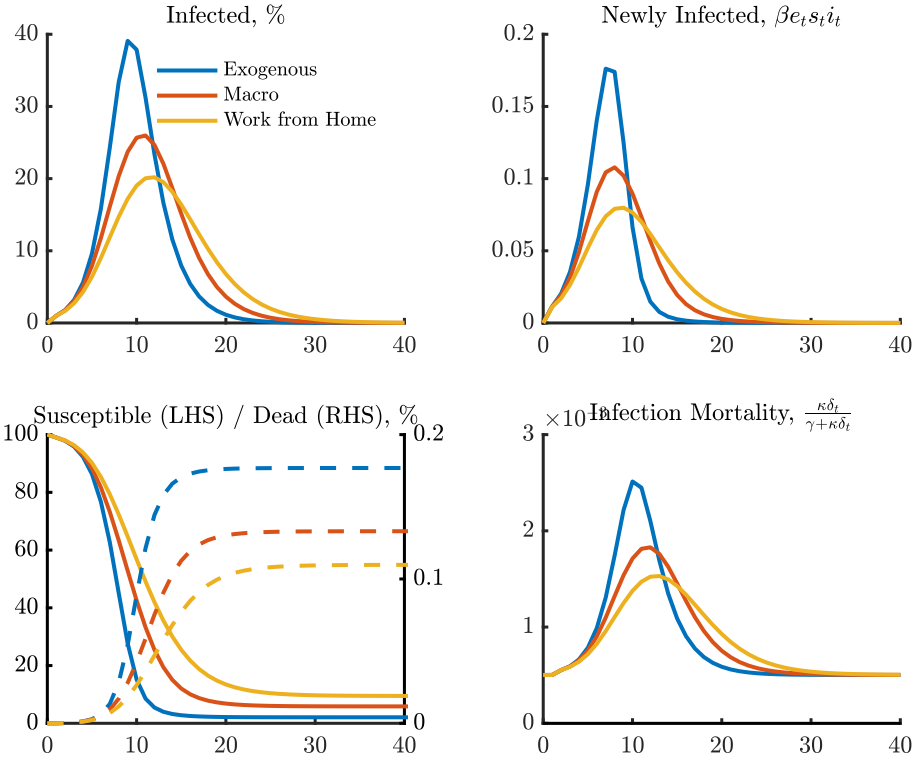## Figure 8: Planner Solution: Contagion Dynamics with Reduced δ_t

Infected, %

Newly Infected, $\beta e_t s_t i_t$

Susceptible (LHS) / Dead (RHS), %

Infection Mortality, $\frac{\kappa \delta_t}{\gamma + \kappa \delta_t}$

Figure 9: Planner Solution: Aggregates with Reduced δ_t

Labor (ℓ) and Cons ($\hat{\ell}$, dashed)

Learning, $\bar{m}$

Working From Home, m_t

Exposure, e_t

λ_e

$V_s - V_i$
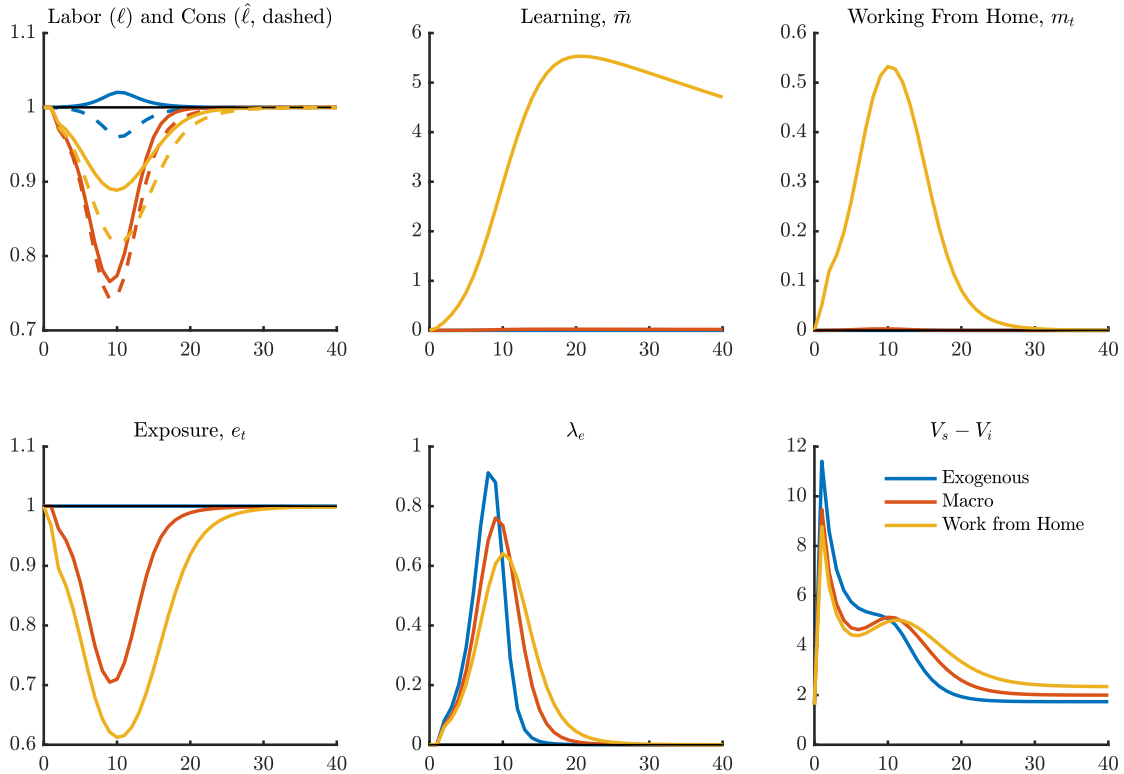
Figure 10: Planner Solution: Contagion Dynamics with $\phi = 0$

Infected, %

- Exogenous
- Macro
- Work from Home

Newly Infected, $\beta e_t s_t i_t$

Susceptible (LHS) / Dead (RHS), %

Infection Mortality, $\frac{\kappa \delta_t}{\gamma + \kappa \delta_t}$
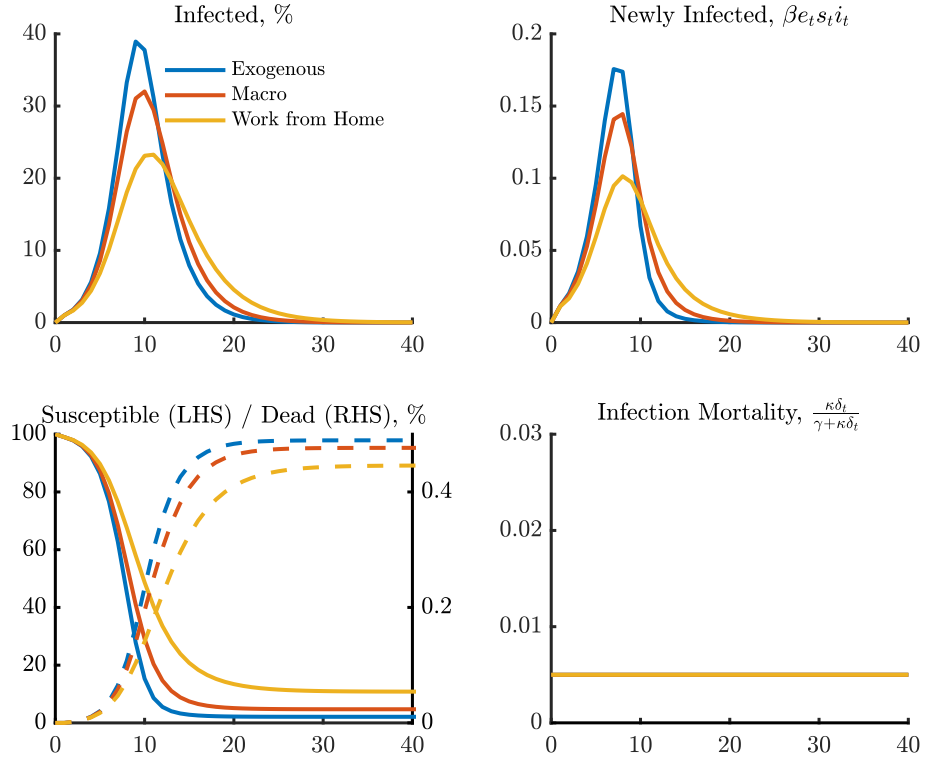
Figure 11: Planner Solution: Aggregates with $\phi = 0$

Labor (ℓ) and Cons ($\hat{\ell}$, dashed)

Learning, $\bar{m}$

Working From Home, m_t

Exposure, e_t

λ_e

$V_s - V_i$

- Exogenous
- Macro
- Work from Home

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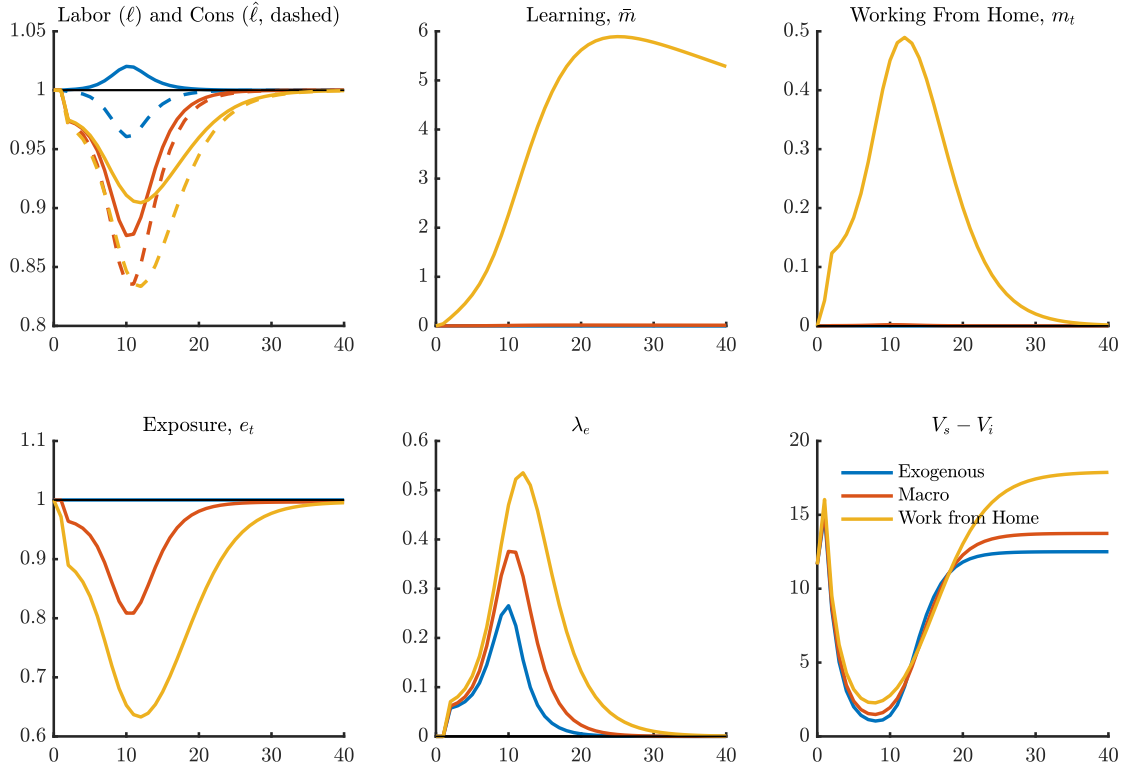
gation decisions. The *laissez-faire* equilibrium and the planner's optimum are obtained as special cases by setting $\tau = 0$ and $\tau = 1$ respectively. If $\tau = 0.33$, cumulative deaths are reduced to 0.9% (only slightly lower than the 1.0% under $\tau = 0$). If τ is assumed to be 0.5, i.e. households place equal weight on private and social incentives, the cumulative death rate falls to 0.85% (roughly half of the gap between the *laissez-faire* equilibrium and the planner's optimum). These results show that for private decisions to be close to that of the planner requires a very large degree of altruism.

Parameter uncertainty As we briefly alluded to earlier, there was (and in some respects, and continues to be) much uncertainty about the structural parameters of the disease. Here, we use our model to speak to the dilemma faced by policy-makers who have to make decisions with imperfect knowledge of key primitives. Atkeson (2020a) points out that, when one does not know the initial number of active cases, it is difficult "to distinguish whether the disease is deadly (1% fatality rate) or milder (0.1% fatality rate)." We illustrate this in Figure 12 where we show how very different combinations of primitives can have similar implications for the number of deaths, the variable that is arguably the best measured. The graph shows the evolution of the disease in the absence of any mitigation for two cases. The first case has a baseline reproduction number $\mathcal{R} = 3.3$ and an infection fatality rate of 0.5%, while the other has a lower reproduction number $\mathcal{R} = 2$ but a higher infection fatality rate of 1%. With an initial shock of $i_0 = 0.1\%$, over the first two months of the spread, the paths of the number of dead are almost identical, as shown in the first panel of the figure. Without accurate measures of other epidemiological variables, such as the number of newly infected or the fraction of cases recovered, it would be nearly impossible to distinguish the two diseases in the short-term. Over the longer-term, however, the two diseases have very different implications for the number of dead, as shown in the second panel.

To explore the role of this uncertainty, we look at the optimal policy of a planner under the two cases (starting from an initial value of $i_0 = 0.1\%$). The planner's choices for labor, consumption and exposure are shown in Figure 13. Interestingly, during the first few weeks, the planner's choices are quite similar under both cases – she cuts exposure by almost 10% immediately. The differences between the two cases become noticeable only

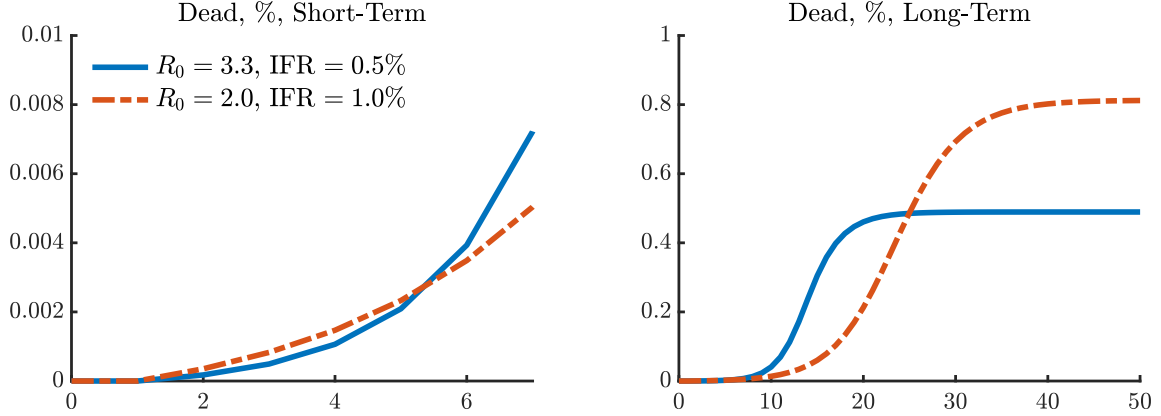Figure 12: Percentage of Population Dead for Two Diseases and Time Horizons
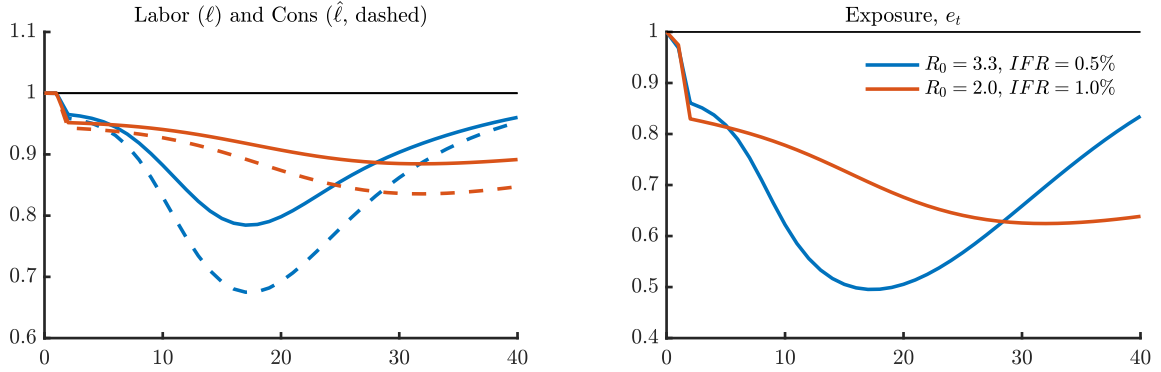

Figure 13: Percentage of Population Dead for Two Diseases and Time Horizons

after 8-10 weeks – the planner continues to ramp up mitigation aggressively for the more contagious (albeit less fatal) case while the less contagious version induces a more measured but persistent increase. This pattern suggests the following strategy for a planner faced with large uncertainty about the fatality rate: aggressively mitigate the spread of the disease for the first few weeks while waiting for more data. Assuming there is a substantial reduction in uncertainty within the first 10 weeks after the outbreak, this strategy will allow the planner to implement the optimal response, despite the initial uncertainty about a key parameter.

7   Conclusions

We propose an extension of the neoclassical model to include contagion dynamics, to study and quantify the trade-offs of policies that can mitigate the Covid-19 pandemic. Our model reveals two key insights. First, that relative to the incentives of private agents, a planner's

incentives to mitigate the spread of the disease are more front-loaded. Second, in our calibrated model, the prospect of mitigation together with the possibility of agents working from home gives quantitatively meaningful reductions in the spread of a disease and the economic costs.

Our modeling framework was kept simple to more clearly demonstrate the economic forces involved. Enriching it along various dimensions is an important step to sharper quantitative estimates as well as answers to other policy-relevant questions. For instance, the production-side of the economy could be enriched to multiple, heterogeneous sectors, which differ in their exposure risk as well as the ability to work from home. We have also abstracted from testing and the possibility of more targeted mitigation strategies.

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Appendix

A Planner's Problem

The planner understands that the dynamics follow

$$S_{t+1} = S_t - \gamma e_t I_t S_t$$

$$I_{t+1} = \gamma e_t I_t S_t + (1 - \rho) I_t - \delta (\kappa I_t) \kappa I_t$$

$$D_{t+1} = D_t + \delta (\kappa I_t) \kappa i_t$$

$$R_{t+1} = R_t + \rho I_t$$

We normalize $N = 1$ for simplicity.

$$\max U = \sum_{t=0}^{\infty} \beta^t u (C_t, L_t; I_t, D_t)$$

subject to

$$u (C_t, L_t; I_t, D_t) = (1 - D_t) \log (C_t) - (1 - D_t - \kappa I_t) \frac{L_t^{1+\eta}}{1 + \eta} - u_\kappa \kappa I_t - u_d D_t$$

and

$$(1 - D_t) C_t = (1 - D_t - \kappa I_t) \left(L_t - \frac{\chi (\bar{M}_t)}{2} (M_t)^2 \right)$$

The Lagrangian is

$$
\begin{aligned}
V_t \left(I_t, S_t, D_t, \bar{M}_t \right) =& u_t + \beta V_{t+1} + \lambda_t \left((1 - D_t - \kappa I_t) \left(L_t - \chi (\bar{M}_t) (M_t)^2 \right) - (1 - D_t) C_t \right) \\
&+ \lambda_{e,t} \left(e_t - \bar{e} - (1 - D_t) e^c C_t^2 - (1 - D_t - \kappa I_t) e^l (1 - M_t)^2 L_t^2 \right) \\
&+ \lambda_{i,t} \left(I_{t+1} - \gamma e_t I_t S_t - (1 - \rho) I_t + \delta (\kappa I_t) \kappa I_t \right) \\
&+ \lambda_{s,t} \left(S_{t+1} - S_t + \gamma e_t I_t S_t \right) \\
&+ \lambda_{d,t} \left(D_{t+1} - D_t - \delta (\kappa I_t) \kappa I_t \right)
\end{aligned}
$$

The first order conditions for consumption and labor are then (highlighted in red the difference with the decentralized equilibrium)

$$C_t: \ C_t^{-1} = \lambda_t + 2\lambda_{e,t}e^c C_t$$

$$L_t: \ L_t^\eta = \lambda_t - 2\lambda_{e,t}e^l \left(1 - M_t\right)^2 L_t$$

$$M_t: \ \lambda_t \chi_t M_t = \frac{\beta V_{\bar{M},t+1}}{1 - D_t - \kappa I_t} + 2\lambda_{e,t}e^l \left(1 - M_t\right) L_t^2$$

The marginal utilities of the planner with respect to exposure are twice as high as those of the private sector because of the contagion externalities: private agents only care about how their behavior affect their own infection risk. They do not care about how their behavior affects the infection risk of others.

The remaining first order conditions are the same as those of the private sector

$$e_t : \lambda_{e,t} = \left(\lambda_{i,t} - \lambda_{s,t}\right) \gamma I_t s_t$$

$$I_{t+1} : \lambda_{i,t} = -\beta V_{I,t+1}$$

$$S_{t+1} : \lambda_{s,t} = -\beta V_{S,t+1}$$

$$D_{t+1} : \lambda_{d,t} = -\beta V_{D,t+1}$$

The envelope conditions are

$$V_{I,t} = \kappa \frac{L_t^{1+\eta}}{1+\eta} - \kappa u_\kappa - \kappa \lambda_t \left(L_t - \frac{\chi_t}{2}M_t^2\right) + \lambda_{e,t}\kappa e^l \left(1 - M_t\right)^2 L_t^2 - \left(1 - \rho\right)\lambda_{i,t}$$

$$-\gamma e_t S_t \lambda_{i,t} - \left(\delta_t \kappa + \delta_t' \kappa^2 I_t\right)\left(\lambda_{d,t} - \lambda_{i,t}\right)$$

$$V_{S,t} = -\lambda_{s,t} - \gamma e_t I_t \left(\lambda_{i,t} - \lambda_{s,t}\right)$$

$$V_{D,t} = \frac{L_t^{1+\eta}}{1+\eta} - \log\left(C_t\right) - u_d - \lambda_t \left(L_t - \frac{\chi_t}{2}\left(M_t\right)^2 - C_t\right) + \lambda_{e,t}\left(e^c C_t^2 + e^l \left(1 - M_t\right)^2 L_t^2\right) - \lambda_{D,t}$$

$$V_{\bar{M},t} = \beta V_{\bar{M},t+1} + \lambda_t \left(1 - D_t - \kappa I_t\right)\frac{\bar{\chi}}{2}\Delta_\chi e^{\bar{M}_t}\left(M_t\right)^2$$

B Properties of Contagion Dynamics

B.1 Definitions

We start with the most basic concept in epidemiology, the basic reproduction number, which we denote by $\mathcal{R}$ because the usual notation "R_not" is terribly confusing. $\mathcal{R}$ is the expected number of cases directly generated by one case when everyone else is susceptible. The most basic model is to assume that when someone is infected there are three stages

1. a latency period T_1 when the individual is not yet infectious

2. infectious period $T_2 - T_1$

3. recovered period after T_2 when the individual is not infectious anymore

If the contact rate (exposure) is e and the probability of infection conditional on contact is γ, the expected number of secondary cases per primary case in a fully susceptible population is therefore

$$\mathcal{R} = \gamma e \left(T_2 - T_1 \right)$$

In our notations, e is the number of people that one individual meets per unit of time and γ is the probability of transmitting the disease conditional on a meeting between one infectious and one susceptible agent.

B.2 SIR Model

The SIR model builds on this idea. We define the length of one period so that $T_1 = 1$. If someone is infected in period t, then she will start spreading the disease in period $t + 1$. Let I_t be the number of infected individuals and S_t the number of susceptible individuals at the beginning of time t in a population of size N. Each infected agent meets e people. We assume that the meetings are random and that the population is always evenly mixed, therefore the probability of meeting a susceptible person is S/N. The number of meetings between infected and susceptible agents is therefore eIS/N and the total number of new infections is $\gamma e I_t \frac{S_t}{N}$. In our macro model e is an endogenous variable but we take it as a constant for now. We assume that recovery follows a Poisson process with intensity ρ. The

infection equation is then

$$I_{t+1} = \gamma e I_t \frac{S_t}{N} + (1 - \rho) I_t \tag{1}$$

Consider a population of size N (large) of initially susceptible individuals ($S_0 = N$). If one individual is infected, the total number of secondary infections from that individual is

$$\mathcal{R} = \sum_{\tau=0}^{\infty} \gamma e (1 - \rho)^\tau = \frac{\gamma e}{\rho} \tag{2}$$

Note that $\mathcal{R}$ is a number, not a rate per unit of time. The model has a steady state at $I = 0$ and $S = 1$ but it is unstable in the sense that if one individual gets infected the system converges to a different steady state. In general we can write

$$\frac{I_{t+1}}{I_t} = 1 + \gamma e \frac{S_t}{N} - \rho$$

When $S/N \approx 1$, the number infected people evolves exponentially as $\frac{I_{t+1}}{I_t} \approx 1 + \gamma e - \rho$. If $R_0 < 1$ then a small infection disappears exponentially. If $R_0 > 1$ then there is an epidemic where I initially grows over time. The growth continues as long as $\gamma e \frac{S_t}{N} > \rho$. Eventually the number of susceptible people decreases and growth slows down or reverses, depending on how we close the model.

There are two ways to close the model. The simpler one, called the SIS model, assumes that recovered agents ρI go back to the pool of susceptible agents. This is the model used to study the common cold. In that case $N = S_t + I_t$ and the equation becomes

$$I_{t+1} = \gamma e I_t \frac{N - I_t}{N} + (1 - \rho) I_t$$

and the steady state infection rate is $\frac{I}{N} = \max\left(0; 1 - \frac{\rho}{\gamma e}\right)$.

The other way is to introduce a population of recovered agents R who are not susceptible anymore. This model – called SIR – is used for flu epidemics, among others. In the simple

model, R is an absorbing state. The system becomes

$$S_{t+1} - S_t = -\gamma e I_t \frac{S_t}{N} \qquad (3)$$

$$R_{t+1} - R_t = \rho I_t \qquad (4)$$

and of course $N = S_t + I_t + R_t$. Note that S is (weakly) decreasing and R is (weakly) increasing, therefore their limits exist. Since $N = S_t + I_t + R_t$ so does the limit of I. For R and S to be constant it must be that I tends to zero. Therefore

$$\lim_{t\to\infty} I_t = 0$$

That is the first simple property of the solution. Second, since S is decreasing, I must be (at most) single-peaked. If I_0 is small and $R_o > 1$ then I_t must grow, reach a maximum, and then decrease towards zero. This is the typical shape of the curves found in the literature. Harko et al. (2014) provide an analytical solution to the differential equations (in continuous time).

Let us now study the long run behavior of S and R. Combining equations (3) and (4) we get

$$R_{t+1} - R_t = -N \frac{\rho}{\gamma e} \frac{S_{t+1} - S_t}{S_t}$$

This equation is simpler to write in continuous time

$$\frac{\dot{S}}{S} = -\frac{\gamma e}{\rho} \frac{\dot{R}}{N}$$

and to integrate the solution:

$$\log\left(\frac{S_t}{S_0}\right) = -\frac{\gamma e}{\rho}\left(\frac{R_t - R_0}{N}\right)$$

This equation holds along any transition path without exogenous shocks. In the limit, since

$S_\infty + R_\infty = 1$ we have the transcendental equation

$$\log\left(\frac{S_\infty}{S_0}\right) = -\frac{\gamma e}{\rho}\left(\frac{1 - S_\infty - R_0}{N}\right)$$

The long run steady state (S_∞, R_∞) depends on the initial conditions as well at the basic reproduction number $\mathcal{R}$. We can summarize our discussion in the following Lemma.

Lemma. *The SIR model is fully characterized by $\mathcal{R} = \frac{\gamma e}{\rho}$ and the initial conditions (S_0, R_0). If $\mathcal{R} < 1$, infections die out without epidemic. If $\mathcal{R} > 1$, a small infection I_0 creates an epidemic: I_t rises, reaches a maximum in finite time before declining towards zero: $I_\infty = 0$. The long run limits S_∞ and R_∞ exist and satisfy $S_\infty + R_\infty = 1$ and $\log\left(\frac{S_\infty}{S_0}\right) = -\mathcal{R}\left(\frac{1 - S_\infty - R_0}{N}\right)$.*

The complete model takes into account that some individuals will die from the disease. We assume that a fraction κ of infected agents become (severely) sick and a fraction δ of the sick patients die. Hence we have another absorbing state, D. The system of equation of the $SIRD$ model becomes

$$I_{t+1} = \gamma I_t \frac{S_t}{N} + (1 - \rho - \delta\kappa) I_t$$

$$S_{t+1} = S_t - \gamma I_t \frac{S_t}{N}$$

$$R_{t+1} = R_t + \rho I_t$$

$$D_{t+1} = D_t + \delta\kappa I_t$$

The number of sick people is κI_t and determines the pressure on the health care system. From the perspective of the epidemic we could aggregate D and R into one absorbing state: $\tilde{R} = D + R$ such that $\tilde{R}_{t+1} = \tilde{R}_t + (\rho + \delta\kappa) I_t$. The long run solution is

$$\log\left(\frac{S_\infty}{S_0}\right) = -\frac{\gamma e}{\rho + \delta\kappa}\left(\frac{1 - S_\infty - \tilde{R}_0}{N}\right)$$

and $\tilde{R}_\infty = 1 - S_\infty$ while $D_\infty = D_0 + \frac{\delta\kappa}{\delta\kappa + \rho}\left(\tilde{R}_\infty - \tilde{R}_0\right)$. From an economic and social perspective we need to keep track of D and R separately in any case.

B.3 SIR model with exogenous birth and death

The path dependence of the long run steady state is a somewhat artificial consequence of the lack of entry and exit. Suppose that ϵN people are both in state S each period, and also that there is a constant exogenous death rate ϵ. The system is

$$I_{t+1} = \gamma e I_t \frac{S_t}{N} + (1 - \rho - \epsilon) I_t$$

$$S_{t+1} = (1 - \epsilon) S_t - \gamma e I_t \frac{S_t}{N} + \epsilon N$$

$$R_{t+1} = (1 - \epsilon) R_t + \rho I_t$$

Note that population is constant: $S_{t+1} + I_{t+1} + R_{t+1} = N$. Now the steady state requires

$$\gamma e \frac{S}{N} I = I (\epsilon + \rho)$$

$$\gamma e I \frac{S}{N} = \epsilon (N - S)$$

$$\rho I = \epsilon R$$

Since $I > 0$ we can easily solve for the unique steady state

$$\frac{S}{N} = \frac{\epsilon + \rho}{\gamma e}$$

$$\frac{I}{N} = \epsilon \frac{1 - \frac{\epsilon + \rho}{\gamma e}}{\epsilon + \rho}$$

$$\frac{R}{N} = \rho \frac{1 - \frac{\epsilon + \rho}{\gamma e}}{\epsilon + \rho}$$

And now we can take the limit as $\epsilon \to 0$ to get $\frac{I}{N} = 0$, $\frac{S}{N} = \frac{\rho}{\gamma e} = \mathcal{R}^{-1}$ and $\frac{R}{N} = 1 - \frac{\rho}{\gamma e} = 1 - \mathcal{R}^{-1}$. Adding a small amount of exogenous birth and death would render the long run steady state independent of initial conditions.