

Endogenous Distancing

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Abstract: *We extend the standard SEIR model to include consumption and labour decisions of households to capture endogenous variations in the transmission rates of a viral infection in the presence of aggregate uncertainty about policy intervention. We explore and contrast the economic and epidemiological effects of various policy interventions: a baseline laissez-faire decentralised equilibrium with no policy intervention, severe restrictions, moderate restrictions, and a conditional lockdown based on the number of hospital admissions. In the baseline version of the model, the endogenous response of economic agents in a perfect foresight equilibrium with no policy uncertainty leads to a reduction of output of nearly 60%. This is associated with a very large decrease in welfare, which is reduced in all the scenarios in which policy can be implemented, even if the government never decides to do so. However, we find that these gains to welfare are small in magnitude compared to the welfare cost of the pandemic, and that the social welfare associated with no intervention increases relative to all of the intervention scenarios as the pandemic progresses. This suggests that while policy measures may be optimal ex ante, in the absence of a plausible vaccine or treatment, support for restrictions may wane considerably over time.*

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I. Introduction

The COVID-19 pandemic of 2020 has had an unprecedented impact on nearly every country in the world, whether through its impact on public health, with associated excess mortality and debilitating health consequences, or the economic impact instigated by both individual social distancing measures and public policies enacted by various governing bodies. There has been a significant debate over the desirability and effectiveness of latter measures, with many commentators suggesting that these imply unjustifiably large economic costs which do not have commensurate public health benefits. Despite that, much of the burgeoning and fast-growing economic literature on this topic and commentary by academic economists broadly suggest that there is little, if any, trade-off between measured economic activity and public health outcomes. The persistence of this debate over the most appropriate measures, and the degree to which governments have adapted their response to the evolving situation, have led to a large degree of uncertainty over the extent to which there are limits to various economic activities that necessitate social interaction.

Our approach proposes to provide a framework to answer questions over the desirability of various policy measures using a modified version of a standard SEIR model which includes five different epidemic states that agents might find themselves in and in which they have to make period-by-period decisions in terms of consumption and hours worked. We also introduce uncertainty to account for the public's inability to predict how governments might respond to various possible paths for the epidemic's progression. This allows us to consider the impact that uncertainty might have on the optimal decisions of individual agent types.

EPIDEMIOLOGICAL MODEL

The epidemiological component of our model is a simple discrete time extension of the classic SEIR model discussed in (Hethcote 2000), in which the β parameter that governs the rate at which interactions between the susceptible and the infected lead to new infections. We endogenise this parameter using a similar approach to that followed by (Eichenbaum et al. 2020), in which the rate of new infections are determined by the consumption and hours worked decisions of susceptible and infected agents. We also model a gradual progression of the epidemic, in which agents must first become exposed to the virus (infected but asymptomatic), before they become infected (infected and symptomatic), and subsequently hospitalised (infected and requiring intensive care). The introduction of the latter state introduces an additional cost to contracting the virus, as the low fatality rate does not capture the extent to which infections lead to debilitating consequences that nevertheless do not result in death.

ECONOMIC MODEL

The economic component of our model is very straightforward: agents supply labour that is paid at marginal productivity. They use wage income to spend on the final consumption

good and to pay a value-added tax on their consumption to the government, which is then rebated to them. This formulation allows us to increase the cost of engaging in economic activity while keeping agents’ income untouched. In addition to this, we model the government sector as having the ability to set taxes on consumption to achieve public health goals. We also allow for the possibility that the public faces significant uncertainty over which policies the government pursues.

FINDINGS

We uncover a positive relationship between the strength of the measures imposed by the government and the through of total output, which suggests that there is a trade-off between economic activity and public health measures. However, in this instance, maximising measured economic activity does not increase overall welfare; the possibility of the government implementing strict restrictions on consumption further reduces consumption and labour supply, but also increases welfare relative to a perfect foresight, no intervention scenario. We also find that while these measures are welfare-increasing *ex ante*, tracking the measure of aggregate well-being against the relevant counterfactual over time suggests that the preferences of individuals experiencing the pandemic ”switch”. In other words, if at the start of the epidemic agents are better off with restrictive measures even if they last for years, less than a year into the epidemic’s progression this is no longer the case, in our parametrisation. This is highly suggestive of the possibility of public health measures not being time-consistent.

II. Literature Review

In this section, we provide a brief overview of the growing macro-epidemiological literature adjacent to this paper, along with a review of the most up to date understanding of the relevant epidemiological facts about COVID-19.

A. *SEIR Models and Macro-S(E)IR Models*

The SEIR model outlined in this paper follows a long tradition in the literature on epidemiology, going back to (Kermack and Mckendrick 1927). We follow the work of (Eichenbaum et al. 2020), (G. Kaplan, Moll, and Violante 2020), (Toxvaerd 2020, (Rachel 2020), (Glover et al. 2020), (Krueger et al. 2020), among others, in outlining a model in which each individual agent type makes decisions over their consumption and labour supply in response to aggregate conditions. To our knowledge, none of these contributions allow for the role of aggregate uncertainty over prices or the policy response.

The only other paper in this literature to consider the possibility of time inconsistency in optimal policies is, to our knowledge, (Moser and Yared 2020), who analyse a three period model in which government commitment to a lockdown policy improves welfare by creating a more stable environment for investment. While we don’t provide a formal characterisation of this possible inconsistency, the mechanism is quite different to that

analysed here.

Finally, several other contributions such as (Alvarez et al. 2020) or (Acemoglu et al. 2020) are close to the modelling approaches listed above, but focus on the problem solved by the social planner, rather than the decentralised equilibrium, while (Hur 2020) is a partial equilibrium analysis where aggregate prices are fixed at their steady-state values, unlike in our paper.

B. Epidemiology of COVID-19

We searched the existing literature on COVID-19 for meta-analyses and relevant primary literature in order to establish appropriate parameter values for the model. These parameters are transition probabilities of a given state, estimated for a per-day value. They include: the probability that individuals recover given they have been exposed to the virus, the probability that individuals are infected given they have been exposed, the probability that individuals die given they have been infected, the probability that individuals exit the infectious state (based on the length of the infectious period), the probability that individuals recover given they have been infected, the probability that individuals are hospitalised given they have been infected, the probability that individuals recover given they have been hospitalised, and the probability that individuals die given they have been hospitalised.

Firstly, we established the probability that individuals entered the infected or recovery state given they had been exposed to the virus. Here, the infected state refers to experiencing symptoms that would likely reduce productivity; when individuals only develop a loss of smell (anosmia), for example, they would not be considered infected.

There have been many estimates of the proportion of people infected by COVID-19 who are asymptomatic. However, a major limitation of some estimates is that they are cross-sectional and are unable to distinguish between people who are pre-symptomatic (yet to develop symptoms) and people who are asymptomatic (do not develop symptoms throughout the course of the disease). Some meta-analyses have aggregated estimates which account for this distinction, by including prospective studies that have also followed up on patients to ascertain whether they have developed symptoms since their reports were first recorded, or retrospective studies that have measured symptoms over a longer period of time.

For example, a meta-analysis conducted by (He et al. 2020) estimates that around 48.9% of patients who test positive at a screening point are pre-symptomatic rather than asymptomatic, and only around 15.6% of confirmed COVID-19 patients are asymptomatic over a longer period of screening; they define asymptomatic patients as those who do not develop fever, cough or diarrhea.

Other meta-analyses, such as by (Buitrago-Garcia et al. 2020) and (Byambasuren et al. 2020), find estimates of 31% and 16% respectively when considering studies that screen cases over time. However, both (Buitrago-Garcia et al. 2020) and (Byambasuren et al. 2020) include studies where patients are recorded as asymptomatic when they do not report any symptoms, without a consistent definition of which symptoms are used (i.e. varying according to the definition of each study that has been included). They report that case

definitions for asymptomatic patients in the included studies usually included the absence of fever and cough. In another large population study by Menni et al. (2020), authors used data from a COVID-19 symptom tracking app to estimate that 8% of confirmed patients who reported any symptoms only experienced a loss of smell (anosmia).

We therefore used 15.6% as the proportion of people who enter the recovery state after exposure (i.e. are asymptomatic throughout the course of their infection), following the meta-analytic estimate by (He et al. 2020) who used a standard symptom criteria, meaning 84.4% was used as the proportion of people who enter the infected state after exposure (i.e. who develop symptoms).

To estimate a per-day probability of these values, we used estimates of the length of time when patients are infectious and the length of time from exposure to the development of symptoms (if they develop).

The length of time that COVID-19 patients are infectious (i.e. the infectious period) has not been measured directly (Walsh et al. 2020). This is because RT-PCR tests, which are used to detect the presence of SARS-CoV-2 virus (including only fragments of the virus) in potential cases, cannot determine whether those virus particles or fragments are infectious. However, several epidemiological and modelling studies have estimated the incubation period of COVID-19 (the length of time between exposure to the virus and the appearance of symptoms). A meta-analysis by McAloon et al. (2020) estimates the median incubation period to be 5.1 days.

Alongside this, epidemiological and modelling studies have also estimated the serial interval (the length of time between symptom onset in one infected individual and symptom onset in another individual they have infected); this is estimated to be 5.4 days and 5.19 days using fixed and random effects models respectively, in a meta-analysis by (Rai et al. 2020). Using data from household transmission, authors found that a maximum length of infectious period of 13 days showed the best fit to the data (medRxiv). (He et al. 2020) use data from viral load in pairs of infected individuals to estimate that the period of infectiousness begins a median of 12.3 days before symptom onset (95% confidence interval 5.9–17.0 days) and peaks at symptom onset.

We therefore used an estimate of 5.1 days for the incubation period, and an estimate of 13 days as the infectious period. This estimate of the incubation period, along with the total probability of entering the infected state given exposure, gives a 16.6% per-day probability of entering the infected state given exposure, assuming a uniform probability distribution.

Next, we searched for estimates of the infection fatality rate (IFR, the probability that individuals die given exposure to the virus). A systematic review and meta-analysis by (Meyerowitz-Katz and Merone 2020) gives a point estimate of the IFR 0.68% (95% confidence interval 0.53–0.82%), finding a high heterogeneity of estimates from published studies.

We searched for estimates of the duration of disease in COVID-19. A multicentre telephone survey of confirmed COVID-19 patients conducted by the CDC ((Tenforde et al. 2020), for example, estimates that the median duration of time patients took to return to

their usual state of health was around 16 days; but this may be an underestimate due to the nature of self-reported symptoms and that some patients were unavailable for follow-up, potentially due to hospitalisation or death. A prospective study of COVID-19 outpatients across multiple centres in Maryland, USA, estimated that patients returned to their usual health a median of 20 days after symptom onset and that the median time from symptom onset to hospitalisation was 11 days (Blair et al. 2020).

We therefore used an estimate of 20 days between symptom onset and recovery, and with an estimate of 5.1 days for the incubation period, this resulted in an estimate of 25.1 days for the duration of illness. Given the IFR of 0.68%, this results in a per-day estimate of 0.027% of death given exposure to the virus, assuming a uniform probability distribution.

In order to estimate the probability that individuals experienced severe or critical symptoms that required hospitalisation and the probability of death or recovery given individuals were hospitalised, we searched the available literature on hospital length of stay and case severity rate.

The proportion of infected patients who have serious or critical symptoms can also be challenging to estimate. This is because the proportion of people with severe symptoms has generally been reported only for confirmed cases, which means they depend on the testing regimen that was used to ascertain cases of the disease. Two meta-analyses therefore estimate the case severity rate (Hu et al. 2020) and conclude that the risk of severe disease in cases ranges from 12.6–23.5% and 17.4–34.9%, respectively. Notably, (Fu et al. 2020) assess the guidelines of each study in classifying patients with "severe" disease and compare them to the definition in the Guidelines of Diagnosis and Treatment Of COVID-19 (6th ed.) provided by the NHC.

The length of time for which COVID-19 patients are hospitalised appears to vary by age, country and hospitalisation procedure. For example, in a study of 5,700 hospitalised COVID-19 patients in the New York City area, researchers estimated the median length of stay was 4.1 days at the study end-point (Richardson et al. 2020). In contrast, in a nationwide study of 10,021 hospitalised COVID-19 patients in Germany, the median length of stay was 10.0 days (Karagiannidis et al. 2020). In the UK, the length of hospital stay for COVID-19 patients was 7 days (Docherty et al. 2020, and this length varies by patient age (Docherty et al. 2020). The ICNARC estimates the length of hospital stay for COVID-19 patients in critical care specifically is estimated to be 9 days in survivors and 8 days in non-survivors (ICNARC, 2020). And among patients who were hospitalised and recovered, the duration of hospital stay was a median of 13 days, according to a retrospective study conducted by Zhao et al. (2020). These differences may be in part due to differences in testing availability and hospital admissions criteria, as well as age demographics.

Similarly, the proportion of hospitalised patients who die from COVID-19 appears to vary by age, country and hospitalisation procedure. In a multicentre study in Wuhan, China, 21% of patients who were hospitalised with COVID-19 died from the disease. (Richardson et al. 2020) find that 533 out of 5700 patients, i.e. 9.7% of patients, who were hospitalised in the New York City area had died by the endpoint of the study (one month after enrollment), while (Karagiannidis et al. 2020) found that 22% of hospitalised patients in Germany, and

(Docherty et al. 2020) found that 26% of UK patients across 208 hospitals, had died of COVID-19. We therefore use an estimate of 26% of mortality for hospitalised patients. Using an estimate of 7 days for hospital length of stay and 26% mortality based on UK patient data, this results in a 3.71% per day probability of death given hospitalisation, assuming a uniform probability distribution.

III. Model

As is becoming standard in the current suite of Macro-SEIR models, we begin by separating our model into two different components: an epidemiological component that keeps track of the macro-level progression of the virus and population dynamics, along with a macroeconomic model that features decision-making at the level of each agent type. The behaviour of economic agents in the second component depends on, and in turns drives, the laws of motion for the macro-level population and economic aggregates. One way to conceptualise this is that individual agent types make decisions based exclusively on states that affect their own well-being, while the macroeconomic and population variables correspond to the cumulative effect of the decisions taken by each agent type.

A. Baseline Model

We begin our analysis by proposing a baseline Macro-SEIR model that shares several features with the model proposed by (Eichenbaum et al. 2020). We extend this baseline version to include two additional states: exposed and hospitalised in the epidemiological section of the model, and we introduce the possibility of lockdown uncertainty to account for the possibility that agents know a lockdown might be implemented but don't know when that would happen or the size of the tax.

EPIDEMIOLOGICAL MODEL

We set the population at 1 initially, with the initial number of exposed people set at a fraction E_0 . The remaining fraction of agents start off in the susceptible state S_0 . From the exposed state, some individuals recover at a rate ψ^r and transition to the recovered state. We assume that agents in this state cannot die before there is a sufficient worsening of their symptoms, but they can recover without ever developing any at a rate $\rho^{e,r}$. Once symptoms develop, the agent becomes infected and can subsequently transition to the recovery state with probability $\rho^{i,r}$, be hospitalised with probability ρ^h , or see their status worsen sufficiently to pass away with probability ρ^d . Once hospitalised, individuals can either recover with probability δ^r or pass away with probability δ^d . Recovered individuals are assumed to have immunity that lasts until the end of the current outbreak.

The baseline model features the following epidemiological states: Susceptible (S_t), Exposed (E_t), Infected (I_t), Hospitalised (H_t), Recovered (R_t), and Dead (D_t), while the laws

of motion for population dynamics are as follows:

$$\begin{aligned}
 S_{t+1} &= S_t - T_t \\
 E_{t+1} &= T_t + (1 - \rho^{e,r})(1 - \psi)E_t \\
 I_{t+1} &= (1 - \rho^{e,r})\psi E_t + (1 - \rho^h - \rho^{i,r} - \rho^d)I_t \\
 H_{t+1} &= \rho^h I_t + (1 - \delta^r - \delta^d)H_t \\
 R_{t+1} &= \rho^{e,r}E_t + \rho^{i,r}I_t + \delta^r H_t + R_t \\
 D_{t+1} &= \rho^d I_t + \delta^d H_t + D_t
 \end{aligned}
 \tag{1}$$

where T_t represents the total number of daily new cases. These are generated by mixing of the susceptible and the exposed and infected through consumption and work. The expression for new cases is given by:

$$T_t = \pi^p S_t(E_t + I_t) + \pi^c C_t^s(C_t^e + C_t^i) + \pi^n N_t^s(N_t^e + N_t^i) \tag{2}$$

We can construct a law of motion for the epidemiological system by defining the following variable:

$$\pi_t(\cdot) = \frac{T_t}{S_t}. \tag{3}$$

Using these, we can write the model as a Markov chain with endogenous transition probabilities:

$$\mathbf{X}_{t+1} = \mathbf{P}_t \mathbf{X}_t, \tag{4}$$

where:

$$\mathbf{X}_t = \begin{bmatrix} S_t \\ E_t \\ I_t \\ H_t \\ R_t \\ D_t \end{bmatrix}; \quad \mathbf{P}_t = \begin{bmatrix} 1 - \pi^s(\mathbf{X}_t) & 0 & 0 & 0 & 0 & 0 \\ \pi_t(\cdot) & (1 - \rho^{e,r})(1 - \psi) & 0 & 0 & 0 & 0 \\ 0 & (1 - \rho^{e,r})\psi & (1 - \rho^h - \rho^{i,r} - \rho^d) & 0 & 0 & 0 \\ 0 & 0 & \rho^h & (1 - \delta^r - \delta^d) & 0 & 0 \\ 0 & \rho^{e,r} & \rho^{i,r} & \delta^r & 1 & 0 \\ 0 & 0 & \rho^d & \delta^d & 0 & 1 \end{bmatrix},$$

The preceding matrix and law of motion fully describe the path of the epidemic, particularly as the transition matrix \mathbf{P}_t will change depending on the behaviour of economic agents in the model. In the next section we describe how economic agents behave.

ECONOMIC MODEL

All economic agents are assumed to be identical except for their epidemiological status, which varies according to the dynamics of the epidemic. Agents decide on how much to

consume and how many hours to work, decisions which are affected by the epidemiological state the agent is in.

The intra-temporal problem that agents face is one of maximising period utility which is given by:

$$u(c_t^i, l_t^i)$$

subject to the following constraints:

$$P_t c_t^i (1 + \mu_t) = (1 - \mu_t^w) W_t^i n_t^i + \Gamma_t,$$

and

$$1 = n_t^i + l_t^i$$

where P_t is the price of the consumption good, c_t^i is consumption and l_t^i leisure enjoyed by an agent in the epidemiological state i . On the income side, agents earn a wage W_t^i , which is taxed at a rate of μ_t^w , and they receive government transfers of an amount Γ_t . This last variable is simply a lump sum transfer from the government budget constraint to agents. The time constraint in equation ?? indicates that agents must allocate their time between leisure and work.

While the intra-temporal optimisation problems are identical across individuals, they each face different probabilities of transitioning into various different epidemiological states, as discussed at length in the previous section. We begin by discussing the decision of an agent who has recovered from the disease. In this state, agents maximise the following value function:

$$\begin{aligned} \max_{c_t^r, l_t^r} V^r(\mathbf{X}_t) &= u(c_t^r, l_t^r) + \beta V^r(\mathbf{X}_{t+1}) \\ \text{s.t. } P_t c_t^r (1 + \mu_t) &= (1 - \mu_t^w) W_t^r n_t^r + \Gamma_t^r, \\ 1 &= n_t^r + l_t^r \end{aligned} \tag{5}$$

Given the short time frame for the epidemic, we abstract from wealth accumulation and savings. This also implies no role for borrowing or banking services, and therefore government transfers are the only mechanism through which budget constraints can be alleviated.

Agents who do not recover from the illness pass away, and we set the value of this state according to:

$$V^d(\mathbf{X}_t) = u(0, 0) + \beta V^d(\mathbf{X}_{t+1}) \tag{6}$$

Hospitalised agents have temporarily reduced levels of consumption and do not supply

any labour as long as they remain in that state. Their value function is given by:

$$(7) \quad \begin{aligned} V^h(\mathbf{X}_t) &= u(c_t^h, 0) + \beta \left[\delta^d V^d(\mathbf{X}_{t+1}) + \delta^r V^r(\mathbf{X}_{t+1}) + (1 - \delta^d - \delta^r) V^h(\mathbf{X}_{t+1}) \right], \\ \text{s.t. } P_t c_t^h (1 + \mu_t) &= +\Gamma_t^h. \end{aligned}$$

Agents who are infected and exhibit symptoms face the following optimisation problem:

$$(8) \quad \begin{aligned} \max_{c_t^i, l_t^i} V^i(\mathbf{X}_t) &= u(c_t^i, l_t^i) + \beta \left[\rho^h V^h(\mathbf{X}_{t+1}) + \rho^{i,r} V^r(\mathbf{X}_{t+1}) + \right. \\ &\quad \left. + \rho^d V^d(\mathbf{X}_{t+1}) + (1 - \rho^h - \rho^d - \rho^{i,r}) V^i(\mathbf{X}_{t+1}) \right] \\ \text{s.t. } P_t c_t^i (1 + \mu_t) &= (1 - \mu_t^w) W_t^i n_t^i + \Gamma_t^i, \\ 1 &= n_t^i + l_t^i. \end{aligned}$$

This problem is very similar to that for the recovered households, with the exception that infected agents must eventually transition into one of three states: hospitalisation, recovery, or death; failure to transition will keep the agent in the infected state for another period.

As outlined above, when agents have been exposed to the virus, they are for all intents and purposes infected, but do not experience productivity-reducing symptoms. They optimise the following value function:

$$(9) \quad \begin{aligned} \max_{c_t^e, l_t^e} V^e(\mathbf{X}_t) &= u(c_t^e, l_t^e) + \beta \left[\psi V^i(\mathbf{X}_{t+1}) + \right. \\ &\quad \left. + (1 - \psi)(\rho^{e,r} V^r(\mathbf{X}_{t+1}) + (1 - \rho^{e,r}) V^e(\mathbf{X}_{t+1})) \right] \\ \text{s.t. } P_t c_t^e (1 + \mu_t) &= (1 - \mu_t^w) W_t^e n_t^e + \Gamma_t^e, \\ 1 &= n_t^e + l_t^e, \end{aligned}$$

where ψ captures the probability of developing symptoms once exposed to the virus. The final state to consider is that of susceptible individuals, who can become exposed through contact with both the exposed and the infected. They optimise the following value function:

$$(10) \quad \begin{aligned} \max_{c_t^s, l_t^s} V^s(\mathbf{X}_t) &= u(c_t^s, l_t^s) + \beta \left[\pi^s(\mathbf{X}_t) V^e(\mathbf{X}_{t+1}) + (1 - \pi^s(\mathbf{X}_t)) V^s(\mathbf{X}_{t+1}) \right] \\ \text{s.t. } P_t c_t^s (1 + \mu_t) &= (1 - \mu_t^w) W_t^s n_t^s + \Gamma_t^s, \\ 1 &= n_t^s + l_t^s, \\ \pi^s(\mathbf{X}_t) &= \pi^p(E_t + I_t) + \pi^c c_t^s (E_t c_t^e + I_t c_t^i) + \pi^n n_t^s (E_t n_t^e + I_t n_t^i). \end{aligned}$$

The probability of infection $\pi^s(\mathbf{X}_t)$ captures the various channels through which agents in the susceptible state might become infected: random contacts, through consumption, and in the workplace. Susceptible agents are also the only agents to see their behaviour

affected by the state of the disease; other agents are either infected or have become immune, so the progress of the virus has no impact on their well-being.

FIRMS

We assume that there is a continuum of identical firms who operate in a perfectly competitive environment. Given this assumption, we focus on the behaviour for a representative firm. Production is linear in labour and worker specific productivity:

$$(11) \quad Y_t = \sum_j \phi^j N_t^j$$

Where Y_t is output at time t , ϕ^j is productivity for state j and N_t^j is the total amount of labour of type j used in production. The total amount of labour employed is a simple sum of the hours worked by all the agent types:

$$N_t = N_t^s + N_t^e + N_t^i + N_t^r$$

The ϕ^j parameter reflects the labour productivity of each state, with the following constraint $\phi_s = \phi_r = \phi_e > \phi_i$ capturing the reduced productivity of infected agents.

Firms therefore maximise a profit function that takes the following standard form:

$$(12) \quad \Pi_t = P_t Y_t - W_t^s N_t^s - W_t^e N_t^e - W_t^i N_t^i - W_t^r N_t^r = 0.$$

Perfect competition in product, and labour markets ensures that all firms are price-takers, and free-entry ensures that they make zero profits.

GOVERNMENT

The government budget constraint is very straightforward: the fiscal authority collects taxes on consumption, and labour and then distributes these in lump-sum payments to all the agent types. In order to allow for a wider range of policy options, we allow for increases in government debt, which would be the different between revenue and the lump-sum transfers to all groups:

$$(13) \quad \mu_t P_t C_t + \mu_t^w W_t N_t + \Delta B_t = \Gamma_t^s S_t + \Gamma_t^e E_t + \Gamma_t^i I_t + \Gamma_t^h H_t + \Gamma_t^r R_t,$$

where $\Delta_t B_t$ is the increase in government debt in period t . We assume throughout that government has full control over how much to transfer to each group, along with the change in government debt. While changes in production are outside its direct control, the fiscal authority can affect the consumption and labour supply choices of the household through an income effect and through that affect output.

Therefore, the set of policy instruments at the disposal of the fiscal authority is given by: μ_t , a value added tax on consumption, μ_t^w , a tax on labour earnings, on the revenue side of the budget constraint; and Γ_t^s , the lump-sum transfer to susceptible households, Γ_t^e , the lump-sum transfer to exposed households, Γ_t^i , the lump-sum transfer to infected households, Γ_t^h , the lump-sum transfer to hospitalised households, and Γ_t^r the lump-sum transfer to recovered households, on the expenditure side of the budget constraint.

MARKET CLEARING

Equilibrium requires that all the markets discussed above are in equilibrium in every time period. In the labour market, equilibrium is guaranteed by ensuring that the supply of all forms of labour is equivalent to demand by forms, such that, in the aggregate:

$$(14) \quad N_t = S_t n_t^s + E_t n_t^e + I_t n_t^i + R_t n_t^r.$$

Finally, aggregate consumption of the final good is such that:

$$(15) \quad C_t = S_t c_t^s + E_t c_t^e + I_t c_t^i + H_t c_t^h + R_t c_t^r.$$

Having defined the market clearing conditions, we can discuss how agents form optimal plans in this economy.

EQUILIBRIUM CONDITIONS

As above, we begin by describing the optimal plans of recovered agents, as the decisions these agents make do not depend on the state of the epidemic nor can they transition into any of the other states. As a consequence, their optimal decisions do not depend on the actions of other agents or the state of the epidemic.

Therefore, their optimality conditions are given by:

$$(16) \quad \begin{aligned} \frac{(1 - \mu_t^w) W_t^r}{(1 + \mu_t) P_t} &= \frac{u_l(c_t^r, l_t^r)}{u_c(c_t^r, l_t^r)}; \\ P_t c_t^r (1 + \mu_t) &= (1 - \mu_t^w) W_t^r n_t^r + \Gamma_t^r; \\ V_t^r &= u(c_t^r, l_t^r) + \beta V_{t+1}^r \end{aligned}$$

Because the decisions made by these agents will depend on possibly stochastic variables such as the tax rates μ and governmental transfers, that means the value function for these agents will only be a function of purely stochastic variables, but not on the other value functions.

Intuitively, agents who have recovered no longer have to worry about the possibility of infection, nor do they need to worry about potentially transitioning to another state. Due

to the assumption that they cannot access capital markets, this implies they will consume all resources at their disposal in each time period, and supply labour according to the prevailing wage rate each day.

As discussed previously, the value functions for deceased agents is pinned down by:

$$V^d = \frac{u(0, 0)}{1 - \beta},$$

which is time-invariant and reflects the discounted value of zero consumption and enjoyment of leisure forever.

Hospitalised agents also don't make any decisions, but they can transition into either of the previously discussed states, while their consumption is pinned down by their rental income plus transfers from the government.

$$(17) \quad \begin{aligned} V_t^h &= u(c_t^h, 0) + \beta \left[\delta^d V_{t+1}^d + \delta^r V_{t+1}^r + (1 - \delta^d - \delta^r) V_{t+1}^h \right], \\ \text{with } c_t^h &= \frac{1}{P_t} \frac{\Gamma_t^h}{(1 + \mu_t)}. \end{aligned}$$

Infected agents who experience symptoms but not have yet been hospitalised will eventually transition to other epidemiological states, but these transition probabilities are exogenously determined by the disease parameters, so the behaviour of these agents has no impact on the likelihood of being in a different state in the next period. Their optimal plans are therefore defined very similarly to those of recovered agents:

$$(18) \quad \begin{aligned} \frac{(1 - \mu_t^w)}{(1 + \mu_t)} \frac{W_t^i}{P_t} &= \frac{u_l(c_t^i, l_t^i)}{u_c(c_t^i, l_t^i)}, \\ P_t c_t^i (1 + \mu_t) &= (1 - \mu_t^w) W_t^i n_t^i + \Gamma_t^i; \\ V_t^i &= u(c_t^i, l_t^i) + \beta \left[\rho^h V_{t+1}^h + \rho^{i,r} V_{t+1}^r + \rho^d V_{t+1}^d + (1 - \rho^h - \rho^d - \rho^{i,r}) V_{t+1}^i \right] \end{aligned}$$

These are nearly identical to the conditions for exposed agents, who are also infected and will therefore randomly transition to other states based on fixed probabilities. The following equations hold for this type of agent:

$$(19) \quad \begin{aligned} \frac{(1 - \mu_t^w)}{(1 + \mu_t)} \frac{W_t^e}{P_t} &= \frac{u_l(c_t^e, l_t^e)}{u_c(c_t^e, l_t^e)}, \\ P_t c_t^e (1 + \mu_t) &= (1 - \mu_t^w) W_t^e n_t^e + \Gamma_t^e; \\ V_t^e &= u(c_t^e, l_t^e) + \beta \left[\psi V_{t+1}^i + (1 - \psi)(\rho^{e,r} V_{t+1}^r + (1 - \rho^{e,r}) V_{t+1}^e) \right] \end{aligned}$$

Finally, the decisions that susceptible agents make have an impact on their probability

of infection, which in turn means that the probability of moving onto specific states is determined by these choices. First order conditions are then:

$$\begin{aligned}
 (20) \quad & \frac{(1 - \mu_t^w) W_t^s}{(1 + \mu_t) P_t} = \frac{u_l(c_t^s, l_t^s) - \beta \pi^n (E_t n_t^e + I_t n_t^i) (V_{t+1}^e - V_t^s)}{u_c(c_t^s, l_t^s) + \beta \pi^c (E_t c_t^e + I_t c_t^i) (V_{t+1}^e - V_t^s)}, \\
 & P_t c_t^s (1 + \mu_t) = (1 - \mu_t^w) W_t^s n_t^s + \Gamma_t^s; \\
 & V^s(\mathbf{X}_t) = u(c_t^s, l_t^s) + \beta [\pi^s(\mathbf{X}_t) V^e(\mathbf{X}_{t+1}) + (1 - \pi^s(\mathbf{X}_t)) V^s(\mathbf{X}_{t+1})]; \\
 & \pi^s(\mathbf{X}_t) = \pi^p(E_t + I_t) + \pi^c c_t^s (E_t c_t^e + I_t c_t^i) + \pi^n n_t^s (E_t n_t^e + I_t n_t^i).
 \end{aligned}$$

The intra-temporal equilibrium condition for this type of agent reflects the premium and penalty to leisure and consumption, respectively. A marginal decrease in labour market participation reduces the probability of transition to the exposed state, while an increase in consumption has the opposite effect. The probability of infection therefore affects the ratio of marginal utilities away from consumption and towards leisure whenever $V^e < V^s$ for any given wage rate.

Combining the constraints for every agent type, we get the following aggregate resource constraint:

$$(21) \quad Y_t = S_t c_t^s + E_t c_t^e + I_t c_t^i + H_t c_t^h + R_t c_t^r = C_t$$

IV. Quantitative Strategy

In this Section, we briefly describe the method used to solve the value functions and simulate the model, before going on to describe the chosen and calibrated parameter values.

SOLUTION METHOD

A. Solution Method

We develop a modified version of Reiter's backwards induction method that takes into account the fact that some combinations of the state space are not feasible. For example, although in the full n-dimensional set of combinations of proportions of the population with each type of health indicator there could be 55% of the population being Susceptible and 55% of the population being Infected, this is clearly not a feasible state space as the total proportion of the population across all the possible health indicators must sum to 100%.

Hence, we first find the set of feasible combinations of the population distribution over health indicators (essentially a diagonal matrix) and solve for value functions over that set of feasible possibilities. This necessitates taking into account potential forecasts outside the set of feasible states when obtaining expected values for a Susceptible individual's optimisation problem to ensure that the expected value remains an interpolation within the possible state vector rather than an extrapolation outside of it.

Once the value functions have been solved, we use them for the simulations in which each type of individual solves for their optimum consumption and labour choice every period. The specific solution algorithm is described in more detail in Appendix B

B. Parameter values

The model contains two distinct sets of parameters, namely those used in the SEIHR part of the model, and those used in the economic part of the model. We present the values we use for the parameters in each of the two parts of the model below.

We also include aggregate uncertainty in the form of the severity of lockdown μ . This can take three possible values: 0.1 representing no lockdown; 0.311 representing a medium lockdown; and 0.452 representing a severe lockdown (these latter two values are chosen to achieve a reduction in hours worked compared to a case of no lockdown of roughly 8% for a moderate lockdown and 15% for a severe lockdown, with the latter reflecting the reduction in hours worked observed in the USA). On average, no lockdown is expected to last 50 days on average; a medium lockdown is expected to last 120 days on average; and a severe lockdown is expected to last an average of 21 days. This gives the transition matrix between exogenous aggregate states below:

$$\begin{array}{lcl} \mu_{no} & & 1 - \frac{1}{50} \\ \mu_{med} & = & \frac{1}{150}(1 - p_2) \quad 1 - \frac{1}{150} \\ \mu_{severe} & & \frac{1}{56}(1 - p_3) \quad \frac{1}{56}p_3 \quad 1 - \frac{1}{21} \end{array} \quad \begin{array}{l} \frac{1}{50}p_1 \\ \frac{1}{150}p_2 \\ \frac{1}{56}p_3 \end{array} \quad \begin{array}{l} \frac{1}{50}(1 - p_1) \\ \frac{1}{150}p_2 \\ 1 - \frac{1}{21} \end{array}$$

Where we set $p_1 = 0.75$ to reflect the fact that governments are more likely to try a medium lockdown first rather than going straight from no lockdown to a severe one; $p_2 = 0.5$ to represent governments increasing or decreasing the lockdown with equal probability from a current medium severity (since a current medium lockdown could have been introduced after there being no lockdown previously, or as a relaxation of a previous severe lockdown); and $p_3 = 0.95$ to reflect the idea that a government is highly unlikely to go straight from a severe lockdown to having no lockdown.

SEIHR PARAMETERS

The parameters that form the SEIHR component of our model govern the exogenous daily transition probabilities between each health state and are estimated using information from a range of medical sources. The exact value for each parameter used is shown in Table 1. The transition from Exposed to Infected or Recovered is based on evidence suggesting that it takes at most 10 days for symptoms to show, such that we assume that every day there is a 10% chance of an individual leaving the Exposed state.¹ When they leave the Exposed state we assume that anosmia is the only symptom that does not reduce an individual's productivity, such that an individual leaving the Exposed state has a 92% chance of becoming Infected (i.e. developing productivity-diminishing symptoms) and an 8% chance of being

¹<https://www.medicalnewstoday.com/articles/how-long-does-it-take-for-COVID-19-symptoms-to-appear>

Recovered.² This gives a value of ψ^r , the daily probability of moving to Recovered given starting as Exposed, of 0.0079 and a value of ψ^i , the daily probability of moving to Infected given starting as Exposed, of 0.092.

An individual leaves the Infected state after 21 days on average.³ As 19% of people have “severe” or “critical” symptoms when they are infected, we assume that an individual leaving the Infected state has a 19% chance of being hospitalised, giving a daily probability of being Hospitalised given starting as Infected ρ^h of 0.009.⁴ We use the overall Infection Fatality Rate of 0.68% along with the average length of infection to obtain the value of ρ^d , the daily probability of death given starting as Infected, of 0.00032. This also gives an implied daily probability of Recovery given starting as Infected ρ^r of 0.047.

For individuals leaving the Hospitalised state, we use the fact that people who recover given that they start as Hospitalised take four days to do so on average, and that 79% of people who are Hospitalised end up recovering to obtain the value of δ^r , the daily probability of Recovery given starting as Hospitalised of 0.198.⁵ We also take the median length of hospital stay for those who are hospitalised of 8 days and combine that with the fact that 21% of hospitalised people die to obtain the value of δ^d , the daily probability of dying given starting as Hospitalised, of 0.026.⁶

Table 1—: Chosen Parameter Values for the SEIHR model

Parameter	Description	Value
$\rho^{e,r}$	Recover given Exposed	0.0079
ψ	Infected given Exposed	0.092
ρ^d	Death given Infected	0.00032
ρ^h	Hospitalised given Infected	0.009
$\rho^{i,r}$	Implied Recover given Infected	0.047
δ^r	Recover given Hospitalised	0.198
δ^d	Death given Hospitalised	0.026
χ	Denominator in endogenous transition from S to I	1.5

Finally, we choose the parameter χ , the denominator in the endogenous probability of moving from Susceptible to Exposed $\pi_t = \frac{T_t}{\chi}$ so as to match two aspects of the observed time-path of the spread and effect of infections. In particular, in most countries the repro-

²<https://twitter.com/ngehlenborg/status/1250307072861720583/photo/1>

³<https://covid.joinzoe.com/post/covid-long-term>

⁴<https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html>

⁵<https://jamanetwork.com/journals/jama/article-abstract/2765184>

⁶<https://twitter.com/ActuaryByDay/status/1264109917465649152>

duction rate absent any lockdown (R_0) was between 2.0 and 3.0 while the (first) peak of infections occurred at roughly 100-150 days after the start of the pandemic, and the trough of output reached roughly 20% - 30% below its level pre-pandemic.

The choice of the χ parameter has opposing effects in trying to match these two components of the effect of the pandemic. In particular, a higher value of χ reduces the size of the trough in output, but reduces the value of R_0 and increases the amount of time it takes for infections to peak. We choose a value of χ that places more weight on matching the value of R_0 and the timing of the peak of the pandemic rather than the exact value of the trough of output. This results in a value for χ of 1.5.

ECONOMIC PARAMETERS

The economic parameters of our model can be split into those related to production and those related to individual utility. For production, we set aggregate productivity A equal to 1, and the individual productivity of workers with health status Susceptible, Exposed, and Recovered are all set to 1 (i.e. $\phi^s = \phi^e = \phi^r = 1$). We set the productivity of Infected individuals ϕ^i to be 0.8, and the productivity of Hospitalised individuals ϕ^h to be zero. Note that this means Hospitalised individuals would otherwise not be able to consumer, so would have an undefined utility, which necessitates us setting the consumption of hospitalised individuals to a negligible value (specifically, $c_h = 10^{-6}$) so that they have a small amount of positive utility. Moreover, we set the value of being dead to have an arbitrarily large negative value.

The values we use for individual preferences are set out in Table 2. The values for the coefficient of relative risk aversion σ and labour elasticity ψ are taken from “standard” values used throughout the literature. The value for β is the daily value that results from an implied annual interest rate of 4%. We choose the value of the disutility of working φ so that individuals (i.e Susceptible and recovered people) work 1/3 of the time in steady-state.

Table 2—: Chosen Parameter Values for individual preferences

Parameter	Description	Value
σ	Coefficient of Relative Risk Aversion	2.0
φ	Disutility of working	221.0
ψ	Labour elasticity	3.0
β	Rate of Time Preference	$0.96^{\frac{1}{365}}$

V. Results

Our results are divided into two main sections. In section (V.A), we analyse the implications of adding aggregate uncertainty over the severity and duration of the restrictions to the model, and compare this with a *laissez-faire* scenario where all uncertainty is removed. There is no actual policy initiative in the scenario with uncertainty, but agents form expectations over the aggregate state knowing that it is possible for these measures to be implemented at any time. In section (V.B), we consider the implications of three types of intervention: a large tax on economic activity by setting the tax rate on consumption at 45.2% until the infection has run its course and reached the endogenous herd immunity threshold, a moderate tax on economic activity of 31.1% until the infection has run its course, and state-varying policy where the authorities introduce the higher rate when the hospitalisation threshold of 0.0084% of the population is reached⁷. We individually compare these outcomes to the *laissez-faire* baseline in which there is no policy intervention at all.

As discussed in a previous section, the tax levels for each of the interventions were chosen to match observed drops in hours worked of 8% and 15% relative to the baseline for both the moderate and severe interventions respectively. Each simulation of the model lasts 25,000 periods, to ensure that under any reasonable scenario the pandemic will be over in the final period. In each case, the pandemic starts with 10^{-6} fraction⁸ of the population becoming infected in period 5. Aside from these interventions, there is no specific fiscal support to any of the households; however, the government is assumed to balance the budget every period, with all tax revenue generated by each household being rebated back through a lump-sum payment.

OUTPUT & WELFARE

The linearity in the production technology, along with the assumption of only one production factor, ensures that measured output is simply the product of the labour supply of each type of agent with their respective idiosyncratic probability. We explore the welfare implications of the pandemic in using two different metrics. The first of these follows the standard approach of computing the consumption equivalent sacrifice that households would be willing to make in order to not have to experience the pandemic. In order to do so, we compute the perfect foresight value at time 0 associated with the consumption path for a susceptible individual in every scenario and calculate the equivalent of steady-state consumption they would be willing to forego in order to avoid experiencing the epidemic. This is equivalent to computing:

$$V_0^{s,m}(c_0^s, n_0^s) = V(\bar{c} - b^m, \bar{n})$$

⁷ According to the American Hospital Association, this corresponds to half of all Intensive Care Unit beds available throughout the country.

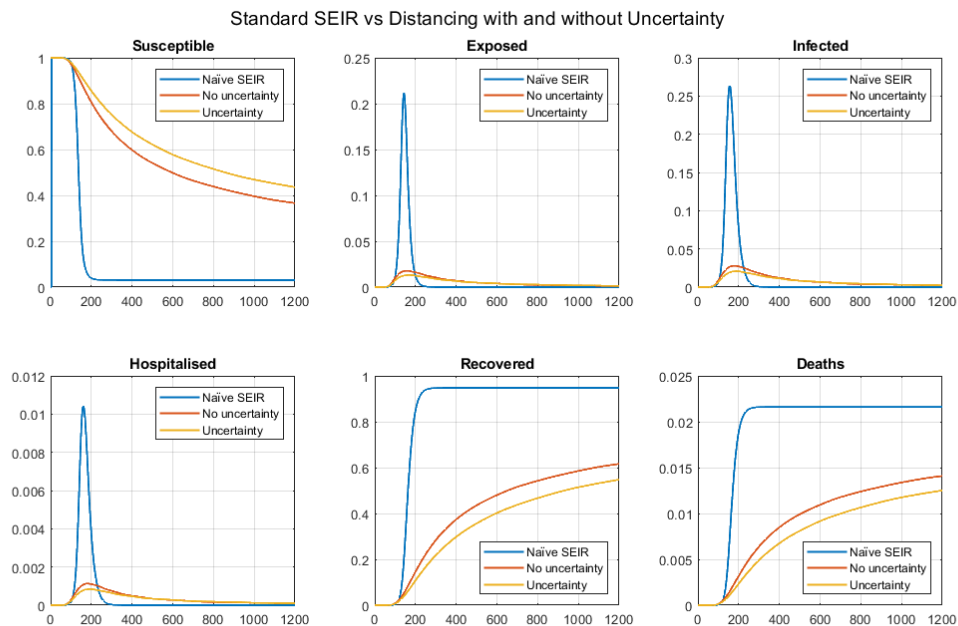
⁸ We assume that each outbreak starts with one case per million susceptible agents.

We also track total social welfare as the epidemic progresses. Doing so allows us to assess the viability of restrictive measures should the cost of their continuation in the future become so large as to generate public opposition and trigger concerns over time-consistency of any optimal plans outlined at time 0. This approach gives some insight into the possibility of "lockdown fatigue", whereby popular support for restrictive measures may wane over time as the pandemic progresses and individuals recover from the disease and the risk of infection for the remaining susceptible population falls.

A. Impact of Uncertainty

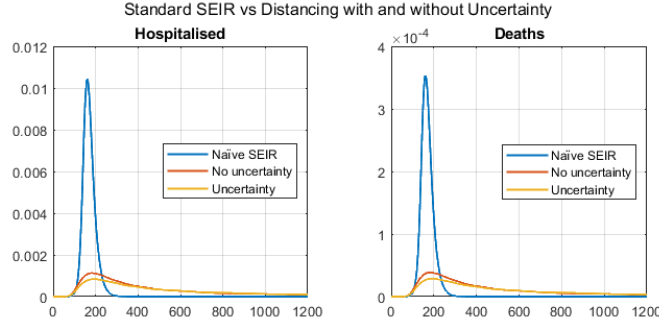
We begin by exploring the impact of the addition of uncertainty over policy outcomes on the progression of the epidemic. Figure 1 shows the progression of the epidemic under three different scenarios. The first of these is a standard or *naïve* SEIR model that omits any behavioural response by the agents in the model. The impact of considering an endogenous response by these agents is very large, with the peak of infections and hospitalisations an order of magnitude below what would be predicted by a standard epidemiological model. An example of this is illustrated by the daily death rates implied by each version of the

Figure 1. : Population Dynamics



model. In the *naïve* SEIR version without any behavioural responses by susceptible individuals, the daily death rate is predicted to reach 0.035% (figure 2 of the population, or just over 100 thousand individual cases a day, with the total number of ICU cases reaching 1% of the total population. In the version of the model with an endogenous response, the number of fatalities is reduced by an order of magnitude⁹. In addition to the stark impact of

Figure 2. : Deaths & Hospitalisations

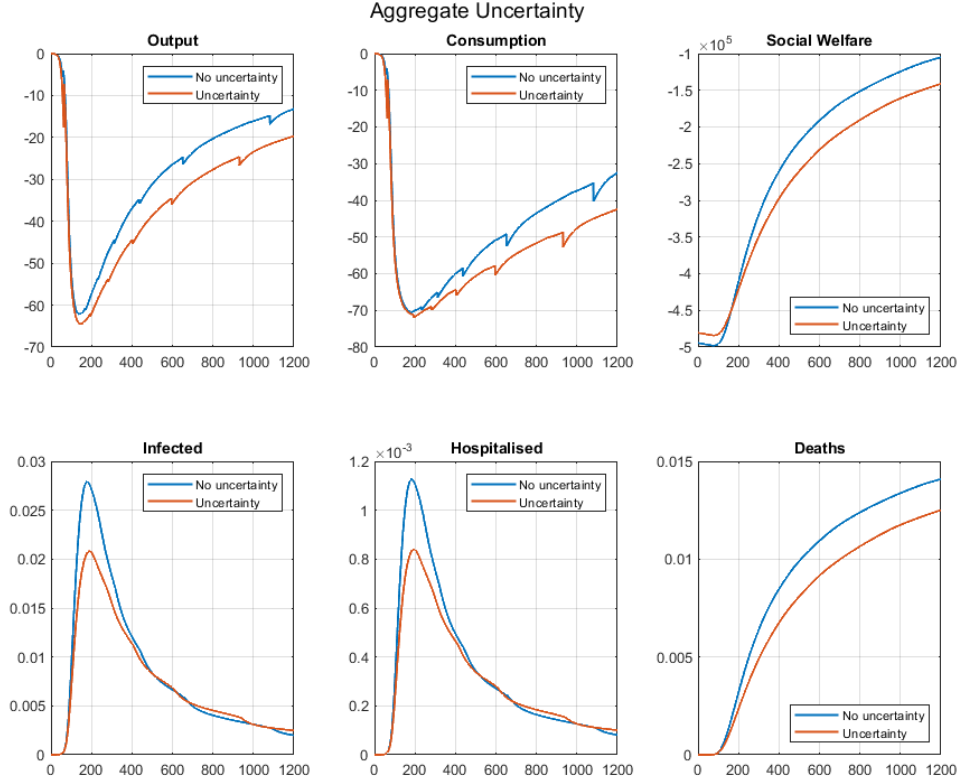


considering the possibility of agents changing their behaviour in response to the epidemic, the impact of policy uncertainty is also significant. In the model with perfect foresight and no policy response, the peak of the pandemic in terms of number of hospitalisations occurs two weeks earlier and is nearly 34% greater. While these differences are smaller in contrast with the impact of endogenous distancing measures, they occur without any policy intervention.

These changes in the progression of the epidemic are driven by the response of *susceptible* economic agents. Because there is no forward looking behaviour in the model, individuals' decisions are affected by aggregate conditions exclusively through the risk of infection. That means all agents who can no longer become infected respond to the *actual* policy rates, rather than forming expectations over what they might be. As we can see in figure 3, introducing the possibility of policy interventions reduces output relative to the counterfactual. This effect increases with the duration of the epidemic because the measures last until the total number of cases drops below a certain threshold. By slowing the spread, likelihood these measures will be in place at future dates also increases, which in turn reduces consumption and labour supply of the susceptible households. Performing the welfare analyses outlined above, introducing policy uncertainty reduces the cost of experiencing the pandemic as a susceptible agent; the value at time 0 for a susceptible individual is higher in that case. The welfare costs of the pandemic are quite large, however. Calculating the amount of steady-state consumption that susceptible agents would be willing to

⁹This is likely driven by an imperfect calibration based on outdated parameter values. A revised version of this paper will contain the updated version of these figures.

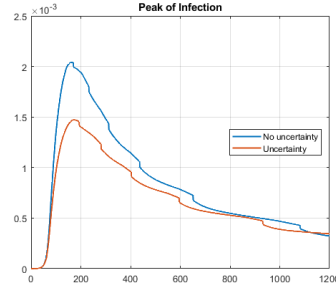
Figure 3. : Impact of Uncertainty



forego in order to not experience the epidemic suggests a very high cost even in the perfect foresight and no uncertainty scenario. Economic agents would be willing to sacrifice around 95% of steady-state consumption in order to avoid experiencing the epidemic altogether, an amount which rationalises the large effect of endogenous distancing we observe in the simulations. However, if we track the evolution of social welfare (computed as a simple weighed average of the values for each agent type), we see that this is reversed around 150 days into the pandemic in our preliminary parametrisation. While it remains optimal to maintain the possibility of imposing these measures *ex-ante*, support for these from a societal point of view might well wane as the pandemic progresses and policy makers are required to renew their commitment to optimal paths¹⁰. As we can see in figure 4, the

¹⁰The precise timing of this reversal, should it ever occur, is likely to change under a different parametrisation.

Figure 4. : Infection Risk



peak of the epidemic occurs roughly around this point, which suggests that the decreasing risk of infection makes susceptible households less willing to tolerate further measures. The results suggest that analysing the optimality of policies at time 0, while instructive to determine optimal policy paths, risks ignoring the possibility of waning popular adherence to these measures. It is outside the scope of this paper to fully explore the consequences of this possibility, but the possibility that policies aimed at mitigating the effects of the epidemic might suffer from some form of "time inconsistency" is one that future research should investigate more closely.

B. Policy Interventions

In this section, we analyse the impact of implementing three alternative policies on the path of the epidemic, as well as on economic activity and welfare.

SEVERE RESTRICTIONS

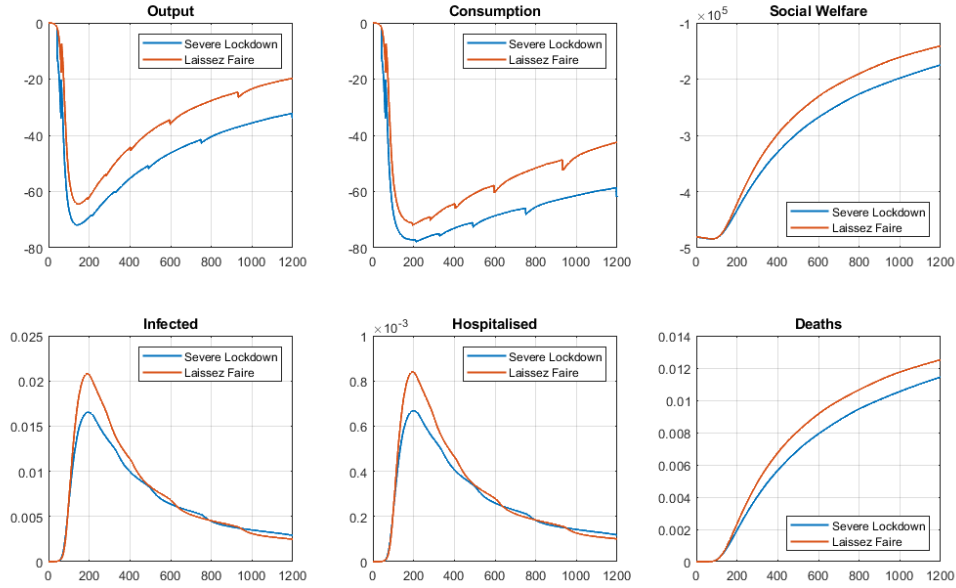
The first of these policies is a substantial increase in the consumption tax rate from 10% to 45.2% in period 43 days of the epidemic¹¹. The policy has the expected consequences of further reducing labour supply and consumption (and consequently economic output) relative to the scenario with policy uncertainty but no actual intervention, as can be seen in figure 5. As a consequence, the progression of the epidemic is further slowed down relative to the counterfactual with no intervention, with associated reductions in the numbers of hospitalised as well as the peak of deaths; in this scenario, at the peak of the number of fatalities, deaths are nearly 25% lower than in the case without any policy intervention.

Slowing down the progression of the pandemic further is associated with a marginally larger cost of the pandemic in terms of the welfare of an agent¹², but in this scenario any

This exercise simply suggests the possibility that welfare might well change with the progression of the epidemic.

¹¹The assumption here is that there is some lag between the first case and the introducing of restrictive measures. For example, between the first recorded case of COVID-19 on the 31st of January of 2020 and the government mandated lockdown on the 16th of March, 45 elapsed.

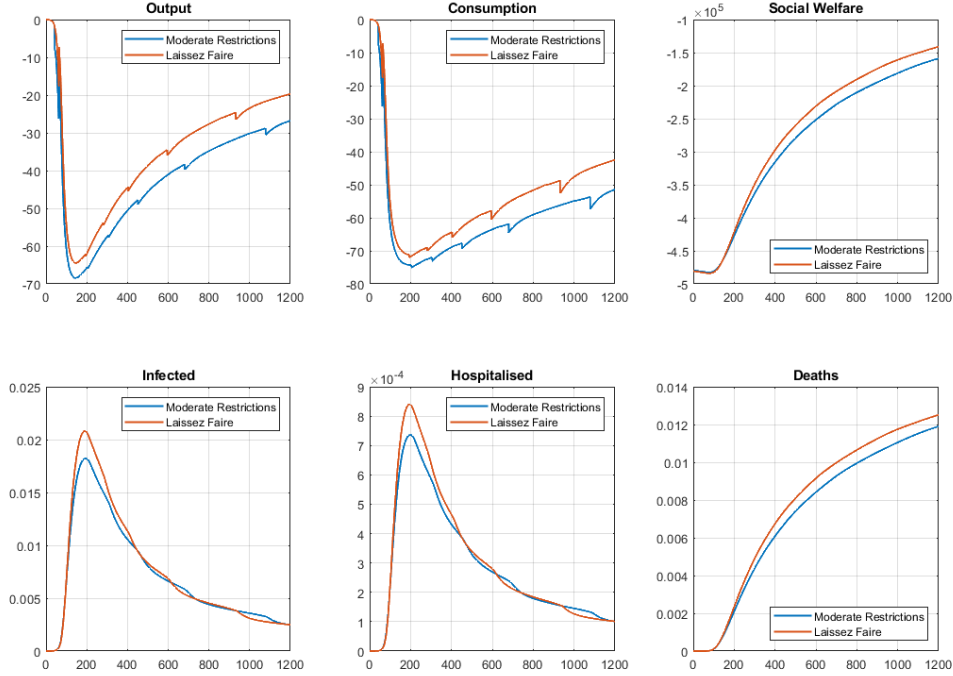
¹²Equivalent to 0.0059% of steady-state consumption.

Figure 5. : *Laissez-faire* vs Severe restrictions

potential fatigue around the measures being implemented would in all likelihood manifest itself at a much earlier stage. Another way of interpreting these results is that, according to our parametrisation, the additional costs imposed by the policy intervention above and beyond the *risk* that some policy will be implemented are almost entirely cancelled by the marginal slowing down of the progression of the epidemic that these entail.

MODERATE RESTRICTIONS

A second policy scenario we consider is similar to the one described above, but in which the tax rate is set at a lower value of 31.1% until the number of cases falls below a certain threshold. This has the expected effect of decreasing the number of hours worked along with consumption, as well as small reduction in the effective reproduction number. As is clear from a cursory analysis of figure 6, the effect is relatively minor in comparison to the effect of more restrictive measures. Despite this, moderate restrictions are preferred to uncertainty without an actual increase in containment measures, with an associated gain of 0.0151% in terms of steady-state consumption. While this scenario is marginally preferable to that of introducing uncertainty without any policy change, it is also the case that support for these policies could potentially suffer as the pandemic progresses; social welfare in the case with restrictions doesn't increase as rapidly as in the case only with

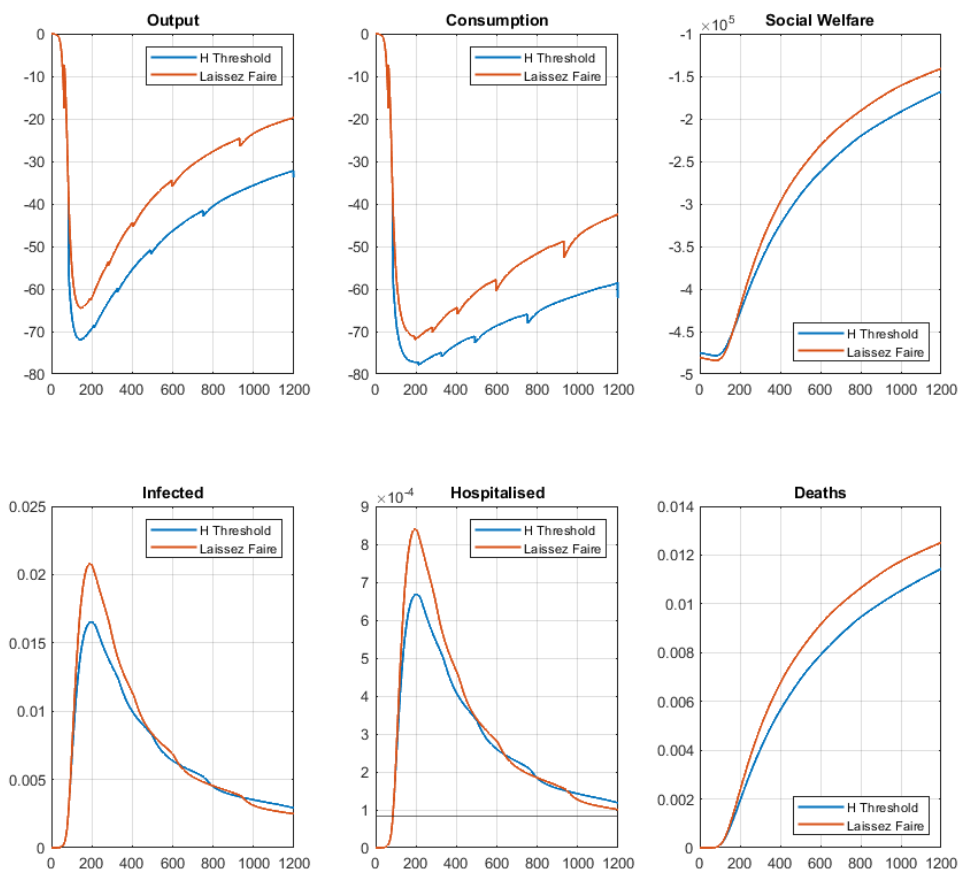
Figure 6. : *Laissez-faire* vs Severe restrictions

policy uncertainty.

HOSPITALISATION THRESHOLD

A final scenario we consider is one where policy restrictions are introduced when a threshold for the number of hospitalisations is reached. As discussed previously, we set this value to match 0.0084% of the total population. Because the restrictions do not immediately halt the pandemic, the number of hospitalisations rises rapidly, with total numbers remaining well above the threshold. When these numbers are sufficiently reduced, the authorities reduce the severity of the restrictions until the threshold is met again.

This results in a shorter duration for the restrictive measures, which is associated with an overall lower level of output relative to the no policy counterfactual, along with slightly lower peaks for the number of cases as well as hospitalisations. This scenario corresponds to the largest reduction in the overall cost of experiencing the pandemic by a susceptible individual out of all the scenarios analysed so far, with a reduction of around 0.07% of



steady-state consumption that would be sacrificed, and a reduction of 0.15% relative to the perfect foresight equilibrium when no policy is taken.

C. Discussion

This paper corroborates the findings in (G. Kaplan, Moll, Violante, and U. S. G. Kaplan 2020), given the parametrisation followed here and the simplified nature of economic activity, there is an extremely large amount of distancing that agents engage in to avoid the risk of contracting the disease. Output falls by nearly 70% relative to the steady-state and remains significantly lower for a large period of time; much of this is driven by behavioural changes by economic agents trying to minimise their risk of contracting the disease every

period.

Several factors contribute to this result. Production is determined exclusively by the amount of labour supplied, which means that any reduction in labour leads to equally sized reductions in output for all states except the infected and the hospitalised. Significant increases in these numbers would contribute to large decreases in disposable income that are not counteracted by any additional fiscal support. Agents are also locked out from financial markets and do not have access to any savings vehicle they might use to smooth consumption; a higher tax lowers the value of the susceptible state relative to the value of being exposed to the virus, which has the effect of dampening the fall in consumption that would arise from a higher consumption tax.

Furthermore, we don't consider the implications of standard extensions such as a vaccine or a higher death toll due to breaching ICU capacity, both of which contribute to making period of prolonged restrictions extremely costly to individuals as the epidemic will inevitably have to run its course.

The introduction of uncertainty is associated with an improvement in both aggregate welfare at the start of the epidemic, along with a lower steady-state consumption cost that individuals would be willing to bear to avoid it. This is a consequence of the negative externality associated with the behaviour of infected or exposed agents; they bear no cost for propagating the disease and would be unwilling to incur a cost to reduce their consumption or labour supply. The introduction of uncertainty over policy outcomes has a significant impact on the progression of the epidemic, but in our parametrisation, this does not translate into significant changes from an economic point of view. While implementing some measures does increase welfare, these changes are very small relative to the behavioural adjustments engendered by the effect of endogenous distancing.

In the simulations we ran, the welfare associated with *some* restrictions was higher at the beginning of the epidemic than the welfare for a susceptible individual in the *laissez-faire* counterfactual, with the latter preferring a rule that mandated higher restrictions whenever the hospitalisation rate increases above a certain value. However, these effects are very small on aggregate relative to behavioural changes induced by the risk of contagion.

VI. Conclusion

We developed an extended SEIR model that allows susceptible economic agents to adapt their behaviour in response to the risk of infection, with and without the implementation of public health containment measures. We also allow for aggregate uncertainty over the likelihood of different types of policies being implemented, which affects the expected pay-offs from transitioning and belonging to the various epidemiological states. We also provide a quantitative exercise where we explore the impact of the model in terms of the degree of endogenous social distancing that economic agents engage in, as well as the welfare implications of both policy uncertainty and actual containment plans.

The model generates a very large behavioural response of susceptible agents in this model, with economic activity dropping to nearly 70% of the steady-state value as agents reduce consumption and hours worked to avoid infection. Despite this large impact, we

also find that economic activity is inefficiently high as individuals, particularly those who have already been exposed to the virus, fail to sufficiently reduce economic activity in the absence of government interventions to contain the progression of the virus.

We also examine the implications of increased uncertainty regarding the possibility of measures being implemented, and find that the simple threat of intervention leads to susceptible agents reducing consumption and labour supply further, increasing the overall welfare of an individual at the beginning of the epidemic relative to the counterfactual. Given the large amount of time associated with each intervention, which can remain in place for years, carries a high economic cost, we try to address the question of how social welfare evolves as the pandemic progresses. We find that aggregate well-being is greater for moderate interventions relative to the *laissez-faire* outcome at the start of the pandemic, but that this is no longer true if it progresses past a certain threshold. While it may be justified, and indeed optimal, to commit to a course of action which susceptible individuals would nevertheless prefer to the *laissez-faire* scenario, as the pandemic progresses, individuals will be increasingly less likely to favour the continuation of such policies beyond a certain threshold. In the limit, it is plausible that retaining public support for these measures would prove impossible in that situation.

Our analysis is limited by a number of factors to be addressed in future research, such as the simplified nature of the productive process along with relaxing the constraint that individuals would be unable to access financial markets to smooth out fluctuations in consumption. Future research will expand the role that uncertainty plays by considering a richer set of policy interventions on the economic policy front, as well as the impact of vaccine development and the implications of testing programmes.

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MATHEMATICAL APPENDIX

Functional forms:

$$(A1) \quad u(c, l) = \frac{c^{1-\sigma}}{1-\sigma} + \varphi \frac{n^{1+\psi}}{1+\psi}$$

Equilibrium conditions:

Economic component:

$$(A2) \quad \frac{(1 - \mu_t^w)}{(1 + \mu_t)} \frac{W_t^r}{P_t} = \varphi(c_t^r)^\sigma (n_t^r)^\psi;$$

$$(A3) \quad c_t^h = \frac{1}{P_t} \frac{\Gamma_t^h}{(1 + \mu_t)};$$

$$(A4) \quad \frac{(1 - \mu_t^w)}{(1 + \mu_t)} \frac{W_t^i}{P_t} = \varphi(c_t^i)^\sigma (n_t^i)^\psi;$$

$$(A5) \quad \frac{(1 - \mu_t^w)}{(1 + \mu_t)} \frac{W_t^e}{P_t} = \varphi(c_t^e)^\sigma (n_t^e)^\psi;$$

$$(A6) \quad \frac{(1 - \mu_t^w)}{(1 + \mu_t)} \frac{W_t^s}{P_t} = \frac{u_l(c_t^s, l_t^s) - \beta \pi^n (E_t n_t^e + I_t n_t^i)(V_{t+1}^e - V_t^s)}{u_c(c_t^s, l_t^s) + \beta \pi^c (E_t c_t^e + I_t c_t^i)(V_{t+1}^e - V_t^s)};$$

$$(A7) \quad P_t c_t^r (1 + \mu_t) = (1 - \mu_t^w) W_t^r n_t^r + \Gamma_t^r;$$

$$(A8) \quad P_t c_t^i (1 + \mu_t) = (1 - \mu_t^w) W_t^i n_t^i + \Gamma_t^i;$$

$$(A9) \quad P_t c_t^e (1 + \mu_t) = (1 - \mu_t^w) W_t^e n_t^e + \Gamma_t^e;$$

$$(A10) \quad P_t c_t^s (1 + \mu_t) = (1 - \mu_t^w) W_t^s n_t^s + \Gamma_t^s;$$

$$(A11) \quad V_t^r = u(c_t^r, l_t^r) + \beta V_{t+1}^r;$$

$$(A12) \quad V^d \approx \frac{u(0, 0)}{1 - \beta};$$

$$(A13) \quad V_t^h = u(c_t^h, 0) + \beta \left[\delta^d V_{t+1}^d + \delta^r V_{t+1}^r + (1 - \delta^d - \delta^r) V_{t+1}^h \right];$$

$$(A14) \quad V_t^i = u(c_t^i, l_t^i) + \beta \left[\rho^h V_{t+1}^h + \rho^r V_{t+1}^r + \rho^d V_{t+1}^d + (1 - \rho^h - \rho^d - \rho^r) V_{t+1}^i \right];$$

$$(A15) \quad V_t^e = u(c_t^e, l_t^e) + \beta \left[\psi V_{t+1}^i + (1 - \psi)(\rho^r V_{t+1}^r + (1 - \rho^r) V_{t+1}^e) \right];$$

$$(A16) \quad V^s(\mathbf{X}_t) = u(c_t^s, l_t^s) + \beta \left[\pi^s(\mathbf{X}_t) V^e(\mathbf{X}_{t+1}) + (1 - \pi^s(\mathbf{X}_t)) V^s(\mathbf{X}_{t+1}) \right];$$

$$(A17) \quad \pi^s(\mathbf{X}_t) = \pi^p(E_t + I_t) + \pi^c c_t^s (E_t c_t^e + I_t c_t^i) + \pi^n n_t^s (E_t n_t^e + I_t n_t^i);$$

$$(A18) \quad Y_t = S_t c_t^s + E_t c_t^e + I_t c_t^i + H_t c_t^h + R_t c_t^r = C_t;$$

Epidemiological component:

$$(A19) \quad S_{t+1} = S_t - T_t$$

$$(A20) \quad E_{t+1} = T_t + (1 - \rho^r)(1 - \psi) E_t$$

$$(A21) \quad I_{t+1} = (1 - \rho^r)\psi E_t + (1 - \rho^h - \rho^r - \rho^d)I_t$$

$$(A22) \quad H_{t+1} = \rho^h I_t + (1 - \delta^r - \delta^d)H_t$$

$$(A23) \quad R_{t+1} = \rho^r E_t + \rho^r I_t + \delta^r H_t + R_t$$

$$(A24) \quad D_{t+1} = \rho^d I_t + \delta^d H_t + D_t$$

$$(A25) \quad T_t = \pi^p S_t(E_t + I_t) + \pi^c S_t c_t^s(E_t c_t^e + I_t c_t^i) + \pi^n S_t n_t^s(E_t n_t^e + I_t n_t^i)$$

SOLUTION ALGORITHM

The state variables of the model are the individual health state (whether the person is Susceptible, Exposed, Infected, Hospitalised, Recovered, or Dead); the endogenous aggregate state covering distribution of the population over each individual health indicators (the proportion of the population that are in each of Susceptible, Exposed, Infected, Hospitalised, Recovered, and Dead); and the exogenous severity of the policy measures). The value function is stored on a grid covering these states (note that we exclude Dead from the endogenous aggregate state and treat it as the remainder when the exiting population of Susceptible, Exposed, Infected, Hospitalised, and Recovered is subtracted from 1).

However, it is important to note that the distribution of the population over each individual health indicator is subject to a restriction. In particular, the total population must always sum to 1 (when expressed as proportion of the population at each state). This means that allowing the state space to cover the possibility that all people are susceptible or all people are recovered, means that some points in the state space defined over the full range of these health indicators are not feasible. For example, in our model it is not possible for the entire population to be susceptible and the entire population to be recovered at the same time.

Therefore, it is necessary to restrict the aggregate state of the distribution of the population over individual health indicators only to those combinations of Susceptible, Exposed, Infected, Hospitalised, Recovered, or Dead that are feasible. This complicates interpolation for the forecast endogenous aggregate state when obtaining expected values, but is resolved via the method we describe in more detail below.

Our solution algorithm is an adaptation of Reiter's backwards induction method. In short, we iterate on forecasts of the endogenous aggregate state given an initial guess of the value function until those forecasts converge in an inner loop, and then update the guess of the value function in an outer loop. The model is solved when the value functions in the outer loop converge.

More specifically, our solution algorithm proceeds as follows:

- 1) Make an initial guess of the value function and the forecasts of the endogenous aggregate state given the starting states
- 2) In the inner loop:
 - a) Given the starting endogenous state for Exposed, Infected, Hospitalised, Recovered, or Dead, calculate the forecasts of next period's Infected, Hospitalised,

Recovered, or Dead using the exogenous transition probabilities

- b) Calculate the expected future value of being Susceptible, Exposed, or Infected using the current guess of the value function and the transition probabilities for the exogenous severity of the restrictions on output.
 - i) This necessitates interpolating the value function stored on the set of feasible endogenous aggregate states. These are not stored on an n -dimensional grid, but by necessity are stored in a vector. Hence, for each forecast endogenous state, the value function for the nearest neighbours above and below each forecast endogenous aggregate state are identified and stored in a $5 \times 5 \times 2$ matrix, which is then interpolated linearly to obtain the forecast expected value.
 - ii) However, some forecasts might be outside the feasible combinations (e.g. the forecasts might imply 55% of the population are susceptible and 55% of the population are recovered) such that trying to find the expected value in such a case would imply extrapolating to an infeasible scenario rather than interpolating between feasible values. In order to avoid this, we iteratively shift in the forecasts to the lowest neighbour previously identified in that direction (e.g. first shift in the Recovered forecast to its lowest neighbour and check if the state is now feasible; if it is not feasible, then shift in the Hospitalised forecast to its lowest neighbour and check again; and so on) until the forecast state becomes a feasible one and then linearly interpolate over those values.
 - c) Find the optimum level of consumption and labour for the Susceptible types using Golden Section Search along with the implied endogenous transition probability to Exposed.
 - d) Use the resulting transition probability to Exposed to calculate the forecasts of the proportion of the population that is Susceptible or Exposed next period.
 - e) Iterate until the guess of the forecast endogenous aggregate state matches the actual endogenous aggregate state
- 3) In the outer loop:
- a) Update the value functions for the Exposed, Infected, Hospitalised, and Recovered type individuals using the period-optimisation for each type
 - b) Use the forecast endogenous aggregate state to obtain the expected value of being either Susceptible or Exposed next period.
 - c) Use these expected values, along with the implied endogenous transition probability to Exposed to find the consumption and labour decision of the Susceptible type using Golden Section Search along with the implied endogenous transition probability to Exposed.
- 4) Iterate until the value functions in the outer loop converge.

For the simulations, we use the value functions solved above along with the exogenous transition probabilities of the Exposed, Infected, Hospitalised, Recovered, or Dead to keep track of the proportion of the population that are each of those types over time. For Susceptible types, we repeat steps 3b and 3c above to find their optimum consumption and labour decisions, along with the endogenous transition probability to Exposed each period. We run this simulation three times to get one set of results - the first uses the forecast rules from the algorithm solved above; the second uses the observed population dynamics from this first simulation (so as to smooth out the “spiky” forecast paths used in that simulation); and the third uses the observed population dynamics from the second simulation (to fully incorporate the effect of any exogenous changes to the severity of the restrictions).