Economic Activity and COVID-19 Transmission: Evidence from an Estimated Economic-Epidemiological Model*

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May 29, 2020

Preliminary - Updated Regularly as New Data Becomes Available

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Abstract

We develop and estimate a spatial model of the joint evolution of economic variables and the spread of COVID-19 across U.S. counties. Agents in the model optimally trade-off engaging in market consumption with the risk of contracting the disease. To motivate the model, we use three novel county-level data sets to document key empirical relationships between non-pharmaceutical interventions (NPIs), health, mobility, and employment outcomes at a daily frequency. We investigate the relative importance of NPIs, such as stay-at-home orders, and endogenous social distancing. Finally, we use our estimated model to address key issues in battling the pandemic: How will the spread of the disease and economic activity evolve if current NPIs are relaxed? What are optimal implementable NPI policies, taking into account the trade-off between the spread of the virus and economic activity?

*The authors thank the IZA for generous research support through the Coronavirus Emergency Research Thrust. We thank SafeGraph for providing access to their data and the SafeGraph COVID-19 Data Consortium for helpful input. We thank Ray Sandza and Homebase for providing access to their payroll data. Support from the Ragnar Söderbergs stiftelse, and the European Research Council grant No. 759482 under the European Union's Horizon 2020 research and innovation programme is also gratefully acknowledged. Philipp Hochmuth and Fabian Sinn provided excellent research assistance.

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1 Introduction

The rapidly developing COVID-19 pandemic presents perhaps the most daunting challenge to economic policymakers since the Great Depression. From an economic point of view, the difficulty in addressing the problem is that the nature of the COVID-19 "shock" is unlike any other disturbance to the economy considered by mainstream economic paradigms. By engaging in market activity — be it going to work or buying goods or services — individuals subject themselves to infection risk and impose negative externalities on others by spreading the virus. Formulating an appropriate policy response to the pandemic requires accurate forecasts of *both* economic and epidemiological variables in response to alternative policies.

We begin by documenting empirical relationships between non-pharmaceutical interventions (NPIs) at the county level and epidemiological and economic outcomes. First, we show that newly documented cases and COVID-related deaths fall following the implementation of stay-at-home orders, but with a significant lag. Second, we show that economic behavior—namely, hours worked and visits to establishments—fall precipitously before the enactment of NPIs, such as stay-at-home orders. Following the implementation of the NPIs there is a subsequent significant further reduction in economic activity, suggesting the efficacy of NPIs at reducing interactions in the marketplace. We interpret these findings as evidence of a significant endogenous response of individuals to the spread of the virus in the absence of policy, a key feature missing in mainstream epidemiological models.

We fill this gap by developing a model that captures both the epidemiological mechanics of virus transmission as well as the endogenous response of individuals to changes in the epidemiological, economic, and policy environments. We call it an "economic SIR" model. To ensure that the model's implications are quantitatively relevant for real-world decision-making, we estimate the parameters by fitting the model to a panel of daily economic and epidemiological indicators for roughly 2800 U.S. counties. With the empirically disciplined model, we characterize the implications of number of policy options, including a notion of optimal implementable policy.

The estimated model accounts quantitatively for both epidemiological and economic dynamics as the pandemic unfolded. Agents significantly curtail economic activity in response to the rapid spread of the virus in the middle of March. Activity declines further as many locations implement NPIs. By the 23rd of March, we find that those NPIs are binding — constraining agents' economic choices — in more than 60% of counties. As the spread of the virus worsens, the NPIs become less binding — agents optimally choose to stay home even in the absence of policy.[1] By the end of April, NPIs are binding in only 20 percent

[1] Strictly speaking, the NPIs are always binding, in that some fraction of agents have their economic activ-

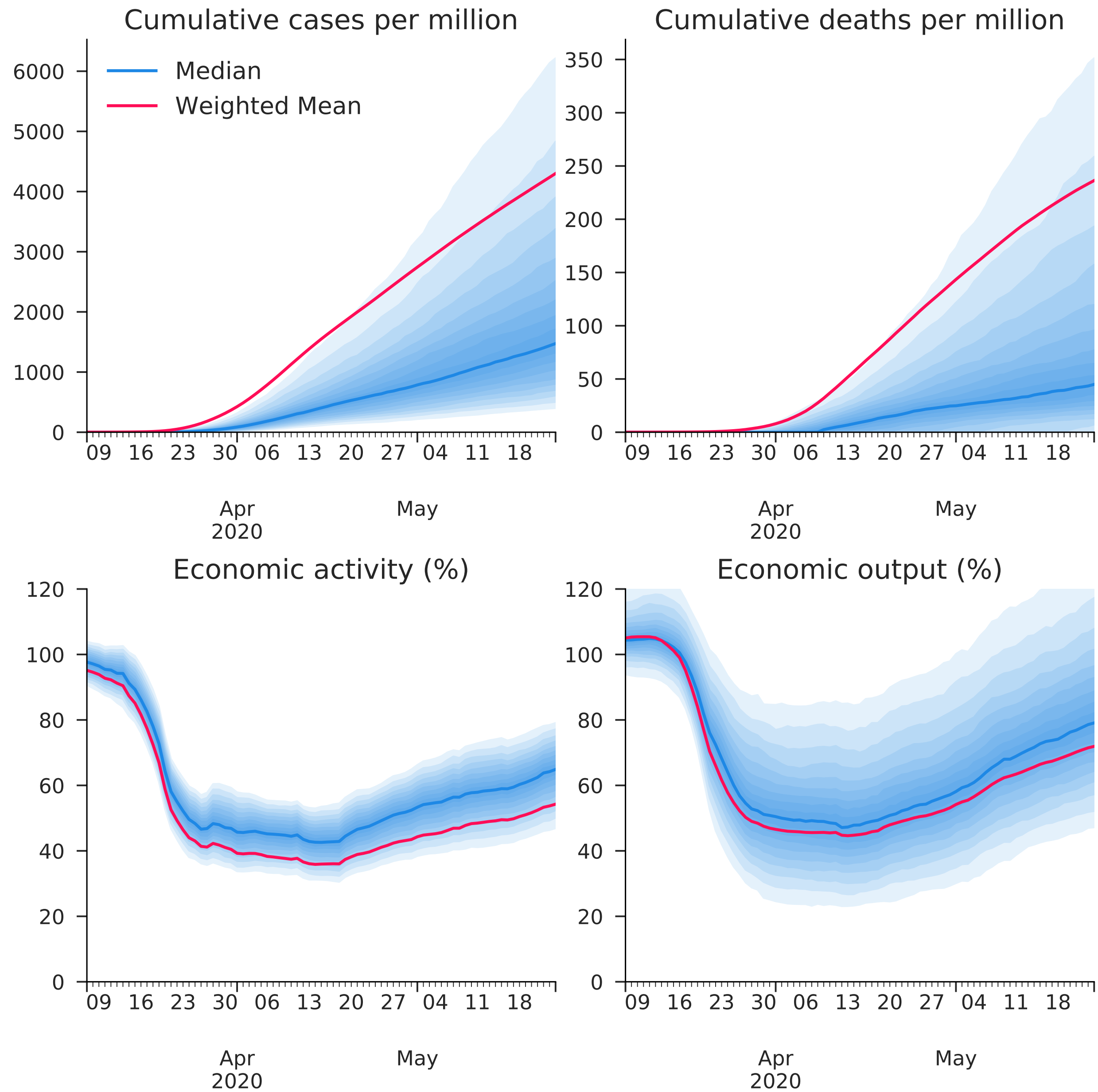


Figure 1: Clockwise from upper left: cumulative cases, cumulative deaths, hours worked and economic activity. The right lines are population-weighted means, the thick blue lines are for the median county, and the light blue lines trace out the distributions.

of counties. The large estimated share of "unconcerned" agents (22 percent) suggests that lockdowns have significantly decreased both economic activity and the spread of the virus. By the end of May, agents make roughly 40 percent fewer visits to establishments that they did before the onset of the pandemic, and hours worked are approximately 30 percent lower. The economic costs of the pandemic have been significant.

Confident in the success of our model to capture the salient economic and epidemiological features of the pandemic, we proceed to use the framework as a laboratory to conduct a number of counterfactual experiments. Our first set of experiments focuses on a short-run forecast of the economy through September 7, 2020 (Labor Day in the United States). We forecast the future path of the virus under two different policies: 1) continuation of status quo lockdowns; and 2) ending all lockdowns as of June 1, 2020. Under both policies, we predict a steady decline in death rates through Labor Day 2020, despite a relatively stable evolution in total cases. The stable linear increase in cases of about 20,000 per day masks significant heterogeneity across locations. Without a lockdown some counties will see daily cases continue to rise through the summer, whereas others will see declines, whereas continuing lockdowns will lead to a uniform decline in daily cases. The median county will see a decline in new daily cases of about percent over this period. At the same time, we see a nearly uniform decrease in daily deaths across locations. The discrepancy between the evolution of cases and deaths can be explained by continued improvements in testing capacity.[2] If lockdowns continue, we predict little to no improvement in economic activity or output through Labor Day. By ending the lockdowns we predict a roughly 10 percent increase in activity and 7 percent increase in output between June 1 and Labor Day. While these increases are significant, activity and output are still significantly below their pre-pandemic levels. Agents still fear infection and reduce activity even in the absense of formal lock downs. Our findings crystallize the notion that "reopening" is easier said than done. As long as people fear infection from the virus, they will continue to curtail their economic activity, leading to a decline in output.

Next, we investigate the longer-run properties of the model if no NPIs are enacted forward. Under such a scenario, we predict that sometime in 2021 the model reaches the "slow burn" regime, where the behavior of the model is roughly linear on the day to day basis. Essentially individuals reduce their economic activity until the reproductive factor (R_0) is approximately 1. The pandemic then plays out over the next decade, with a roughly constant decline in

ity constrained by the policy. That is because we assume that a constant fraction of agents is "unconcerned." As we explain in Section 4.2, those agents make economic decisions without regard to the risk of infection.

[2]We predict that throughout the summer the extent of under-reporting of true cases will continue to decline.

daily deaths and cases and an increase in economic activity that begins to accelerate in 2030 as the model approaches herd immunity. During this phase, about 100,000 people per year die in the U.S. as a result of the virus, and economic output recovers roughly linearly from about 80 percent of pre-pandemic levels. These protracted dynamics imply that ultimately 1 million people will die as a result of the virus over the next 10 years. We should caution the reader that the interpretation of these dynamics rely on several assumptions which may (and we hope) ultimately turn out to be incorrect: this assumes neither the development of a vaccine for COVID-19 nor the development of an effective treatment that could substantially reduce mortality, as well as no future policy response in terms of NPIs.

Next we investigate adaptive lockdown policies.

Next, investigate the cost in terms of lives lost of delayed action in responding to the pandemic. We ask how the number of deaths due to the virus would have been reduced if policy makers had implemented lockdown orders one week earlier. We find that through mid-May, approximately 28,000 fewer people would have died if lockdowns had been implemented earlier. These are comparable to those reported in the well known study Pei et al. (2020). Our model generates a significant reduction in economic activity (consistent with the data as we later demonstrate) prior to the implementation of most lockdown orders. As such the effects of implementing lockdowns one week earlier is likely smaller, as individuals had already begun to socially distance.

We next describe each of these elements of our contribution in more detail.

Our modeling framework extends the now-well-known Susceptible-Infected-Recovered (SIR) model from the epidemiology literature. Importantly, we include economic decision-makers that face a trade-off; they enjoy consumption but fear infection. As the virus spreads, individuals then rationally curtail their economic activities, causing an endogenous decline in aggregate economic activity even without the imposition of NPIs, such as stay-at-home orders. Allowing for this endogenous response is critical for accurately capturing the observed decline in economic activity preceding stay-at-home orders, and for forecasting the future path of the economy in the absence of a vaccine or an effective antiviral.

We show analytically that the economic SIR model predicts a pandemic trajectory with two distinct regimes for virus spread and economic activity. Furthermore, the model endogenously generates the switch from the first regime to the second. At the beginning of the outbreak, for a sufficiently low initial infection rate, agents' behavior is virtually the same as when the infection rate is zero. As a result, the initial regime exhibits exponential growth in the number of infections, just as in the standard SIR model, and a minimal change in economic activity. As the infected share grows, however, the model's optimizing agents begin to restrain their economic activity, and the pandemic shifts into a second regime we call the "slow burn." In the slow burn regime, agents' cautious behavior has the benefit of slowing new infections to an approximately constant growth rate—linear growth, as opposed to exponential–but slowing the spread of the virus comes at the cost of reducing aggregate economic activity. In the absence of any policy interventions, the slow burn regime persists until a sufficiently large share of the population has contracted the virus and recovered, i.e., until the population achieves herd immunity.

In an extension of the model, we rectify one of the unpleasant (and unrealistic) features of the standard SIR model—namely, we allow for the eradication of the virus. In the standard SIR model with Poisson transition rates between states, the share of infected agents never reaches zero in finite time (absent a cure). As such, there will still be some measure of infected agents alive even after curtailing economic and social activity for an extended period (e.g., 100 years). After reversing the lock-down, the system will immediately revert to exponential growth. We allow for the share of infected individuals to fall to zero in finite time if the stock of infected individuals falls below a pre-specified threshold (e.g., if the percentage of infected implies fewer than one infected person in a county). We refer to this as the "kill zone", and illustrate how changing this feature of the model modifies the optimal policy conclusions in battling the pandemic.

To generate quantitative predictions relevant to real-world decision-making, we give empirical discipline to the model's parameters in two ways. First, we estimate the structural model's parameters using a panel of 1340 U.S. counties accounting for 85% of the US population.[3] Second, we validate the model's key quantitative implications around the initial introduction of NPIs using a quasi-reduced-form approach.

To estimate the model we require data on the spread of the virus across time and space. Further, to discipline the connection between economic activity and the spread of the virus, we also need data on individuals' participation in the marketplace. Finally, to understand how market participation ultimately affects economic output, we need a measure of output from the data. We combine three novel data sources achieve these goals. First, we obtain daily, county-level data on confirmed COVID-19 cases and deaths from the *The New York Times*.[4] Second, we obtained hourly GPS location data from a large sample of smartphones. The data, collected by SafeGraph, record hourly visits to points of interest (POIs), including grocery stores, restaurants, hospitals, and an array of other public and private establishments. Third, we add in data on hours and employment based on worker-firm matched data from

[3] We limit our analysis to counties with population above 30 thousand that, as of April 30, 2020, have reached 1 confirmed COVID-19 case per 1 million inhabitants.

[4] Data from *The New York Times*, based on reports from state and local health agencies. See `https://www.nytimes.com/interactive/2020/us/coronavirus-us-cases.html`. Borough specific for New York City is sourced from the NYC Department of Health.

Homebase, a scheduling and time clock software provider.

Related Literature. Our paper relates to the mushrooming literature studying the epidemiological and economic response to the COVID-19 pandemic (e.g. Alvarez et al. (2020,?); Baker et al. (2020); Berger et al. (2020); Eichenbaum et al. (2020); Farboodi et al. (2020); Glover et al. (2020); Guerrieri et al. (2020); Jones et al. (2020); Kaplan et al. (2020); Keppo et al. (2019); Krueger et al. (2020); Toxvaerd (2020)).[5] Given the real-time and rapidly changing nature of those papers we think that instead of providing a discussion of each it is more instructive and expedient to summarize where we have innovated relative to that body of work and what our main contribution is.

Another strand of papers has focused on panel forecasting of the purely epidemiological side of the pandemic's evolution. See Liu et al. (2020) and Fernández-Villaverde and Jones (2020).

In addition to the now standard SIR model and its many variants, there is a large epidemiological literature on the notion of "behavioral change" by agents in response to their environment and new information. Most of these studies do not explicitly incorporate economic factors or optimizing behavior into their analysis, though there are exceptions such as Morin et al. (2013) and Fenichel et al. (2011). See Verelst et al. (2016) for an extensive review of this literature.

Relative to standard epidemiological models, we have added economic behavior — what is sometimes called a behavioral SIR model.[6] We have estimated the model at a granular level, using data on individual movements based on GPS and data on economic outcomes—namely, employment—to discipline the economic-epidemiological connection.

The rest of the paper is organized as follows. In Section two we describe some motivating empirical evidence on the relationships between virus spread, economic activity, and NPIs. In Section three we describe our economic SIR model. Next, in section four, we discuss the estimation strategy and how we bring the model to the data. We conduct a variety of policy experiments in section five, and in section six we conclude.

[5] We apologize for any papers we have missed. Due to the rapidly changing landscape, this likely is not an exhaustive literature list.

[6] We have opted for the term "economic SIR" model, as our agents are fully rational, optimizing agents.

2 Features of the Data

Before presenting our structural model, we first characterize some salient patterns of the key data series we use to inform our model.

2.1 County-Level Data at Daily Frequency

The key to informing the current policy-making environment is high-frequency granular data on the spread of the virus and economic activity. For the empirical analysis and the estimation of the model, we build a panel of four daily county-level variables on virus spread and economic activity, as well as a detailed panel on NPIs.

For epidemiological variables, we obtain daily county-level data on confirmed and probable COVID-19 cases and deaths from the *The New York Times*.[7] The *The New York Times* collects this data on a recurring basis from local public health authorities.[8]

For economic variables, we use detailed data on both foot traffic and hours worked. In particular, we obtain data on foot traffic to over 3.6 million consumer "Points-of-Interest" (POIs), including grocery stores, restaurants, hospitals, and an array of other public and private establishments. SafeGraph collects anonymized GPS location information from a large panel of more than 45 million smartphones in the U.S. This data corresponds to what we will refer to throughout the paper as "economic activity" or just "activity." The SafeGraph data, which is derived from a panel of smartphones, obviously does not represent a random sample of the U.S. population. However, Squire (2019) investigated potential sources of sampling bias in the SafeGraph data. The data are well representative at the county level (when compared to U.S. Census data from the American Community Survey) along a number of demographic dimensions, such as educational attainment and household income. However, very low-income individuals ($< \$10,000$ annual income) tend to be under-represented in the data. The SafeGraph analysis does not address bias in age sampling, but we suspect that given that the panel is based on smartphone users that very old households would tend to be under-represented.

We obtain information on hours worked from a large worker-firm matched dataset provided by Homebase. Homebase is a scheduling and time clock software provider. The data consist of daily time-card records at the establishment level. The time-cards include information on hours worked and wages. The data are anonymized but include information on

[7]Data from *The New York Times*, based on reports from state and local health agencies. See `https://www.nytimes.com/interactive/2020/us/coronavirus-us-cases.html`.

[8]Note that it is unclear in the mortality data if the date corresponds to when the death occurred or when the death was officially reported. In our estimation we account for this uncertainty through our modeling of measurement error in recorded mortality outcomes.

the zip code and industry of the establishment. The data are provided in real-time when the time cards are reported by the business that uses the software. As we detail in the appendix, small establishments and establishments in the food services and retail sectors tend to be overrepresented in the Homebase data. We refer the interested reader to Kurmann et al. (2020) for a more thorough discussion of the representativeness of the Homebase data and how it compares to the Quarterly Census of Employment and Wages conducted by the U.S. Census.

Finally, we assemble a county panel for NPIs. We assemble these data using various sources, including data made available by other researchers as well as our collection efforts using local and national news sources.

While the economic variables are available at the establishment or POI level, we aggregate these observations to the county level (and daily frequency) to match the level of granularity in the epidemiological data. We use data from January 1, 2019 through May 24, 2020.

2.2 Empirical Evidence

We begin by documenting the spread of the virus, both in terms of cumulative numbers of confirmed cases and deaths, and its economic impact across the United States. In Figure 1, we plot the population-weighted mean and distribution across counties for confirmed cases per 1,000,000 population, and we display the population-weighted change in visits to POIs and hours worked. We include the daily (as opposed to cumulative) epidemiological data in the Appendix.

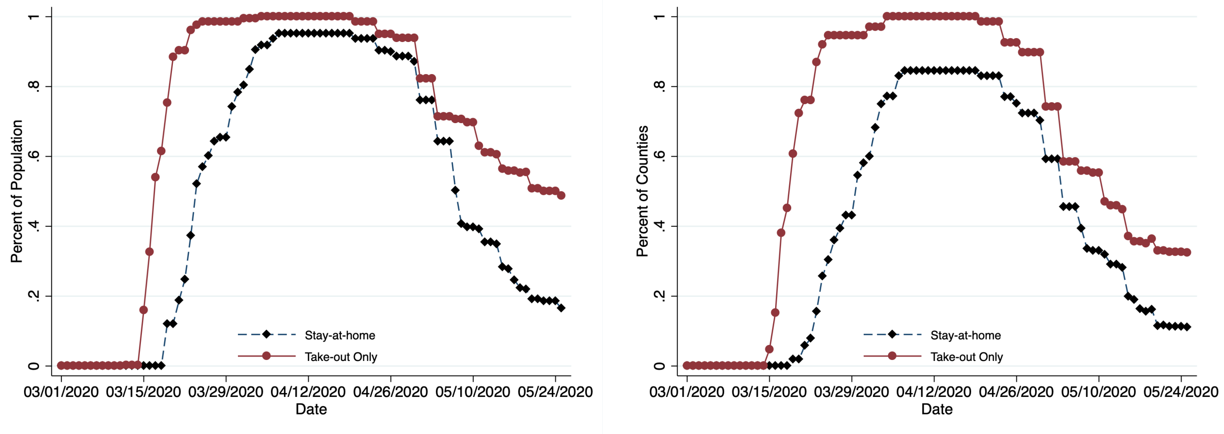
At the same time, as the virus was spreading, states, cities, and counties began implementing NPIs interventions. We focus our attention on stay-at-home orders and orders to make restaurants take-out only (i.e., suspend dine-in service). We plot the time series for the share of counties and population that is subject to those orders in Figures 2.

The fact that the roll-out of NPIs was not randomly assigned across time and space presents a challenge for making causal inferences of the effects of the policies. As such, our goal in this section is less ambitious. Namely, we document the relationship between epidemiological and economic variables in a window around the passage and expiration of NPIs. Later, we will take these as moments that we will use in the estimation of our model.

To that end, we run regressions of the following type:

$$x_{ct} = \alpha_c + \zeta_t + \sum_{\tau=-T,\tau\neq 0}^{\tau=T} \beta_\tau D_{c,\tau,t} + \varepsilon_{ct} \tag{1}$$

where $x_{c,t}$ is the county outcome of interest and $D_{c,\tau,t}$ is a dummy variable indicating if in

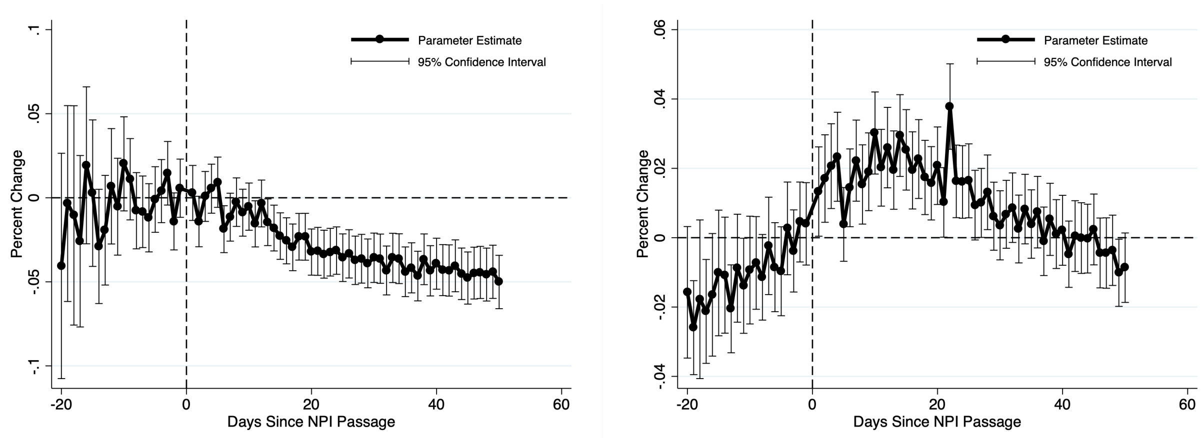


(a) Fraction of U.S. population subject to NPIs (b) Fraction of counties subject to NPIs

Figure 2: The time-series evolution of NPIs.

county c at time t is τ days have elapsed since the passage of the NPI, where negative values indicate days before the passage of the NPI (or expiration of the NPI for the reopening figure). For all regressions we weight the results by the county population in 2018 (obtained from the Census).

Our first results are for regressions on the epidemiological data. Here, we run the regression using the growth rate of cumulative cases and numbers of deaths. To account for the fact that the initially all counties have 0 confirmed cases and deaths, we perform the inverse hyperbolic sin transformation on those variables.[9]



(a) Reported cases (b) Deaths

Figure 3: The effect of non-pharmaceutical interventions on economic activity.

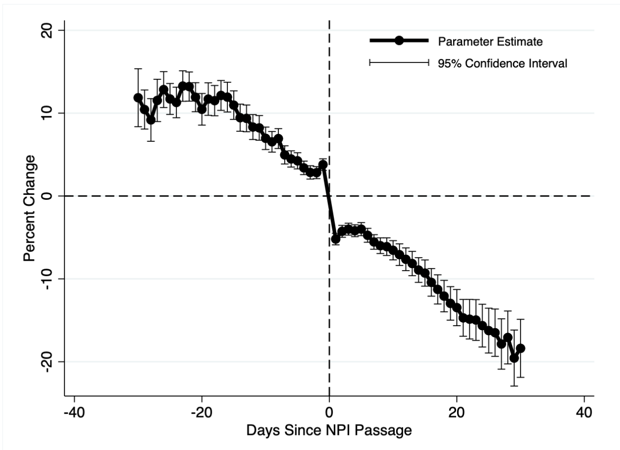
[9]This transform is commonly used when dealing with growth rates and "0" observations. The transform is give by $\tilde{y}_{ct} = \log\left(y_{ct} + \sqrt{y_{ct}^2 + 1)}\right)$.

Cases begin falling about 10 days after the passage of stay-at-home orders. Deaths initially rise after the passage of the stay-at-home order, but then begin falling at longer time horizons. This is not surprising, as deaths are a function of the existing stock of people infected, which only adjusts slowly after the passage of NPIs, even if the flow rates adjust more quickly. Further, this highlights potential endogeneity problems of the passage of NPIs, as areas that were faring worse in terms of infection rates were likely to implement NPIs.

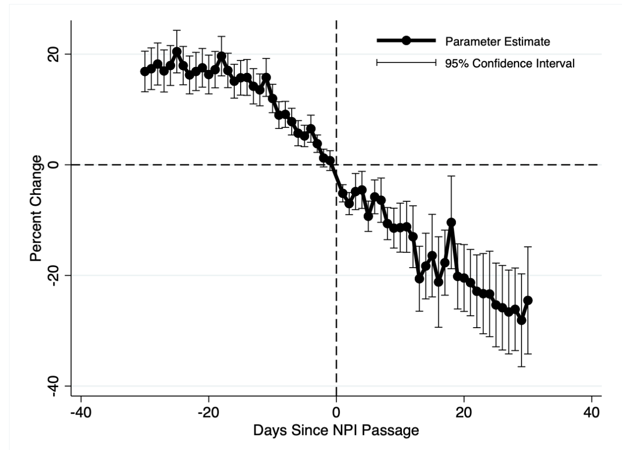
Next, look at the effect of NPIs on economic activity and hours worked. Here, we look at visits to POIs and hours worked. Visits and hours both respond strongly and immediately to the passage of stay-at-home orders and take-out only orders. Notably, however, both hours and visit begin to fall in advance of the pass of the NPIs. We interpret this as evidence of the endogenous response of individuals to the spread of the virus. As infection rates become more prevalent, individuals curtail their activity in the marketplace, and as a result labor demand falls.

Finally, we look at the effects of the expiration of NPIs on economic activity. Visits to POIs begin increasing as restaurant take-out only orders expire. Hours worked also increase in the period after the expiration relative to when the order was in place.

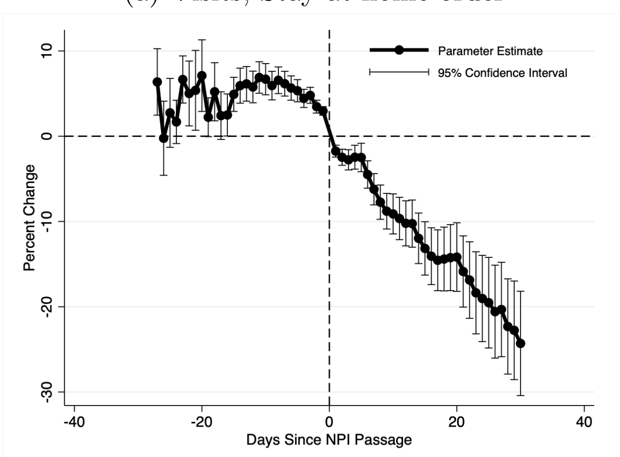
To summarize, individuals respond significantly in their economic behavior in response to the spread of the virus, even in the absence of policy interventions. Policy interventions further reduce economic activity. These reductions in economic activity also translate into lower rates of infection and lower excess deaths. We now will use these empirical findings to discipline our model.



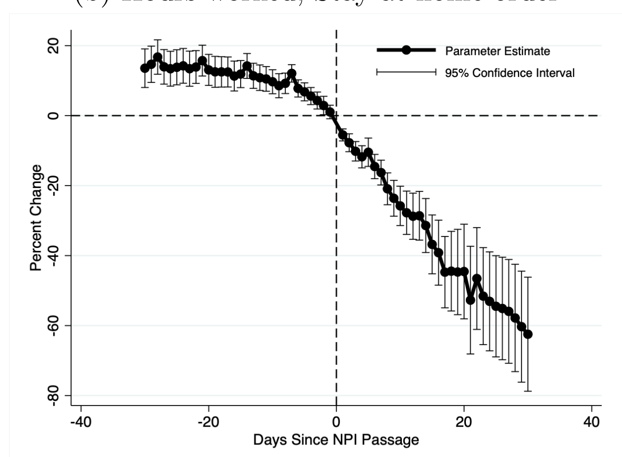
(a) Visits, Stay-at-home order



(b) Hours worked, Stay-at-home order

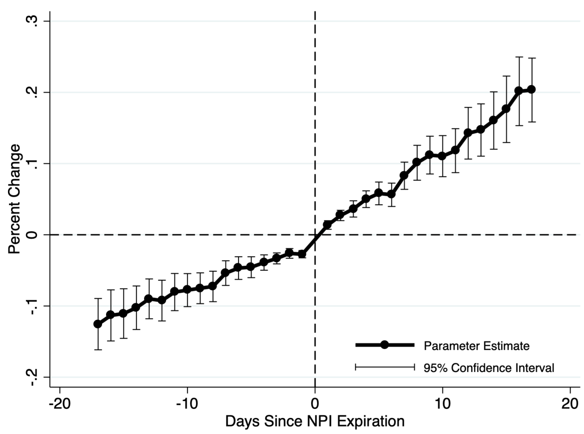


(c) Visits, Restaurant take-out only order

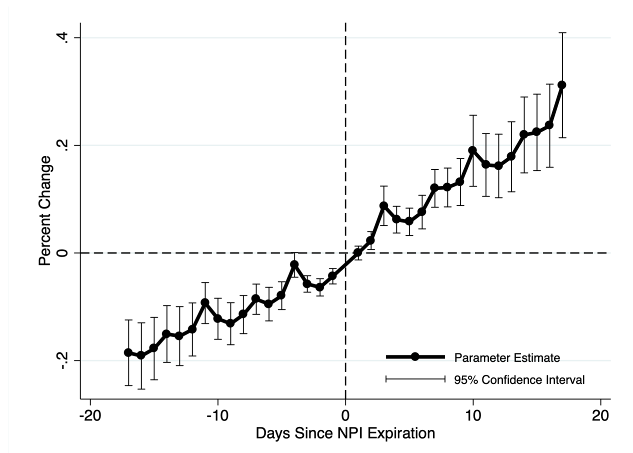


(d) Hours worked, Restaurant take-out only order

Figure 4: Non-pharmaceutical interventions on economic activity. The left column show the effects on visits to POIs in a 60 day window around the passage of NPIs. The right column show the same for hours worked. The top row is for the passage of stay-at-home orders and the bottom row is for orders that made restaurants take-out only.



(a) Visits, Restaurant take-out only order



(b) Hours worked, Restaurant take-out only order

Figure 5: Non-pharmaceutical interventions and economic activity. The left column show the effects on visits to POIs in a 40 day window around the expiration of NPIs. The right column show the same for hours worked. Estimates are weighted by county population.

3 Structural Model I: A Minimal Economic-SIR Model

We begin by describing the simplest version of our economic SIR model to focus on the principal mechanism by which economic behavior and virus transmission interact. We then present a more detailed model that includes two additional epidemiological states. The latter model is the one we ultimately take to the data. On the economic side of the model, susceptible agents can choose whether to participate in the economic marketplace, facing the potential risk of being exposed to infected individuals. They optimally trade off the gains from market participation, with the cost of potentially becoming infected with the virus. The more widespread the infection is in the population, the higher the risk that market participants will become infected. Further, as more infected agents that participate in the marketplace, more individuals become infected. The model thus features bi-directional interaction between economic activity and viral transmission.

The formal models are defined in continuous time and we adopt a few notational conventions when describing them. Constants and parameters are denoted with lowercase Greek letters, e.g., γ. "Stock" variables, namely the measures of individuals in each of the epidemiological states, are denoted with capital English letters, e.g., S. The flow-variable counterpart to each stock variable uses the same capital letter but adds a "$\cdot$" over the character, e.g., $\dot{S}$. Other endogenous variables are denoted with lowercase English letters, e.g., $n(t)$.

3.1 Basic SIR Model with Economic Agents

Here we explain how we extend the basic SIR model to incorporate economic decision-makers.

Epidemiological States. The model is populated with a time-invariant, unit measure of individuals. Individuals can be in one of three epidemiological states: Susceptible (S), Infected (I), or Recovered (R). Susceptible agents have never contracted the virus. Infected individuals have the virus and can spread it. Recovered individuals have previously had the virus and can no longer spread it and are immune from reinfection. The measures of individuals in each of the states at a given time t are denoted $S(t)$, $I(t)$, and $R(t)$.

Epidemiological state transitions. Over time agents move across epidemiological states. Here we specify the dynamics of those movements.

We assume S agents contract the virus at rate $n(t)$, at which point agents transition to the infected state $I(t)$. The S state has no source of inflows, and hence $S(t)$ declines according to

$$\dot{S} = -n(t)S(t). \tag{2}$$

It suffices to think of $n(t)$ as exogenous for the present purposes of describing the mechanics of state inflows and outflows. We describe below how the model's economic dimension interacts with the virus's prevalence and transmission rate.

The measure of agents in the infected state increases with the inflow of the $n(t)S(t)$ newly infected agents, while current $I(t)$ individuals recover at rate δ and transition to R. Hence, $I(t)$ changes according to

$$\dot{I} = n(t)S(t) - \delta I(t). \tag{3}$$

Lastly, since we assume that agents that recover from infection obtain immunity, the R state changes only via inflows of previously infected agents. The R state might thus be considered a terminal, or absorbing, state, and $R(t)$ increases over time according to

$$\dot{R} = \delta I(t). \tag{4}$$

If we were to stop here, and treat $n(t)$ as a constant parameter, say n instead of $n(t)$, then we would have the traditional SIR model.

Economic Decision: To z, or Not To z? We now introduce the novel economic dimension of the model. All agents receive stochastic shocks z that we interpret as economic needs. The shocks are drawn from a time-invariant distribution $F(z)$ with support $z \in [0, \infty)$. Regardless of the particular realization of z, satisfying the economic need requires agents to make an excursion, and the trip exposes the agent to the risk of infection. We assume that agents dislike being infected with the virus and incur a cost ψ from infection.

Agents in all epidemiological states face an identical economic environment. Still, the prospect of virus transmission has very different implications for the economic behavior of agents without the virus than for agents who have already had it. For I and R agents, there is no risk of infection, so they undertake an excursion for any value of z (in this simple version of the model we assume I agents do not quarantine. They are fully "economically active"). For Susceptibles, however, the risk of infection is real, and they are the model's critical economic decision-makers. We assume that conditional on taking an excursion, the probability of virus transmission to a Susceptible is increasing in, and proportional to, the infected share of the population: $\beta I(t)$. The parameter β is a time-invariant rate of virus transmission to a Susceptible during an excursion per unit of Infected. Susceptibles then optimally choose to satisfy a given need z only if the benefit exceeds the expected cost of taking an excursion, which gives rise to a decision by Susceptibles to satisfy z only if

$$z(t) > \beta I(t)\psi \equiv \bar{z}(t). \tag{5}$$

$\bar{z}(t)$ represents the reservation value of Susceptibles for undertaking an excursion, which varies endogenously over time through its dependence on $I(t)$. With each Susceptible optimally applying the decision rule in (5) to idiosyncratic economic needs distributed $F(z)$, the rate at which Susceptibles engage in economic activity ($a_S(t)$) is determined by the rate at which Susceptibles face economic needs z greater than $\bar{z}(t)$:

$$a_S(t) = 1 - F(\bar{z}(t)) = 1 - F(\beta I(t)\psi) \tag{6}$$

In equation (6), one can see that the epidemiological block of the model affects the economic side, as Susceptibles reduce their activity when infection risk becomes more pronounced.

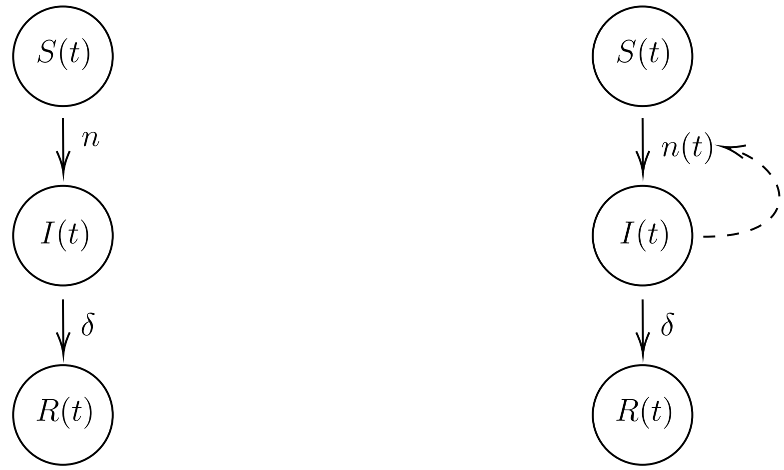
Endogenous transmission rate $n(t)$ from economic decisions. Our assumptions thus far imply that the virus transmission rate $n(t)$ to the Susceptible population is given by

$$n(t) = \beta I(t) a_S(t) = \beta I(t) \underbrace{\left[1 - F(\beta I(t)\psi)\right]}_{\substack{\text{effect of endogenous} \\ \text{activity response}}} \tag{7}$$

In equation (7), one can see a crucial difference between a traditional SIR model and our economic SIR model: the second term, $1 - F(\beta I(t)\psi)$ derived from the economic optimizing behavior of Susceptibles, introduces a nonlinear relationship between $I(t)$ and the transmission rate. Since $1 - F(\beta I(t)\psi)$ is bounded above by 1, so one can see that the endogenous reduction of activity by Susceptibles as $I(t)$ increases will tend to mitigate virus transmission. In other words, the economic dimension of the model affects the epidemiological.

3.1.1 Activity, Output, and the Relationship Between the Two

When we take our extended model to the data, we will use the model's implications for both economic activity ($a(t)$) and output ($y(t)$). It is helpful first to express these notions within the basic economic SIR model. We conceive of activity as the number of excursions taken, while the output is the value of the economic needs satisfied during those excursions. The model differentiates between activity and economic output because the Susceptibles



(a) SIR Model (b) Economic SIR Model

Figure 6: Structure of SIR and Economic SIR Models

restraining their economic activity optimally sacrifice their least valuable activities first. This leads to a potentially non-linear relationship between $a(t)$ and $y(t)$.

Activity. The activity rate is dictated by the active share of Susceptibles and the measures of infected and recovered agents. Since the infected and recovered agents have no risk from infection, they accept all z draws. Total activity (excursions) thus occurs at the rate

$$\begin{aligned} a(t) &= a_S(t)S(t) + I(t) + R(t) & (8) \\ &= 1 - \underbrace{F(\bar{z}(t))}_{\substack{\text{rate of foregone} \\ \text{excursions by } S(t)}} \cdot S(t) & (9) \end{aligned}$$

where the second equality comes from $S(t)+I(t)+R(t) = 1$ and substituting for $a_S(t)$. From equation (9), and the fact that $\bar{z}(t)$ is increasing in $I(t)$, one can see how higher infection risk reduces activity. If $I(t) = 0$ there is no risk and $\bar{z}(t) = 0$, so total activity occurs at its baseline rate of $a(t) = 1$. However, as $I(t)$ increases, the infection risk to Susceptibles increases, causing $\bar{z}(t)$ to increase and the total activity rate $a(t)$ to decline for a given $S(t)$. Equation (9) thus characterizes the effect of the epidemiological environment on the rate of total activity.

Output. The rate of economic output is given by the rate at which the different types of agents engage in activity multiplied by the value of economic needs being satisfied,

$$y(t) = \overbrace{(I(t)+R(t))}^{1-S(t)} \overbrace{\left(\int_0^\infty z(t)F'(z(t))dz(t)\right)}^{\mathbb{E}[z]} + a_S(t)S(t)\overbrace{\left(\int_{\bar{z}(t)}^\infty z(t)\frac{F'(z(t))}{1-F(\bar{z}(t))}dz(t)\right)}^{\mathbb{E}[z|z\geq\bar{z}]} \quad (10)$$

$$= \underbrace{\big(1-S(t)\big)\cdot\mathbb{E}[z(t)]}_{\text{agents consuming normally}} + \underbrace{S(t)\cdot\big[1-F(\bar{z}(t))\big]\cdot\mathbb{E}[z(t)|z(t)\geq\bar{z}(t)]}_{\text{agents restraining activity}} \quad (11)$$

Equation (11) makes clear the differing output rates induced by the differing behavior of agents who are concerned with infection and agents who are not. One can also express the output rate more expressly in terms of its shortfall from usual as[10]

$$y(t) = \underbrace{\mathbb{E}[z(t)]}_{\substack{\text{normal}\\\text{output rate}}} - S(t)\cdot\underbrace{F(\bar{z}(t))}_{\substack{\text{rate of foregone}\\\text{excursions by } S(t)}}\cdot\underbrace{\mathbb{E}[z(t)|z(t)\leq\bar{z}(t)]}_{\substack{\text{expected value of a}\\\text{foregone excursion}}} \quad (12)$$

If $I(t) = 0$, then $\bar{z}(t) = 0$ causing the second term to zero out and all S agents are economically active. and so the expression becomes $y(t) = \mathbb{E}[z(t)]$, which defines the baseline rate of output in the economy. The distribution $F(z)$ implies a certain relationship between economic output and activity $a(t)$.

In our estimated model, we assume that $\log(z)$ is normally distributed with mean $-\frac{\sigma^2}{2}$ (so $\mathbb{E}[z] = 1$) and variance σ^2. Given this assumption, the variance parameter σ^2 neatly summarizes the relationship between output and activity, as seen in Figure 7.

3.1.2 Two Regimes of Virus Spread:Exponential Growth and the "Slow Burn"

In this section we describe the nature of the two regimes of virus spread in the model. The first is an early phase of exponential growth and the second is a protracted period of approximately linear growth.

Exponential Infection Growth Regime. The baseline rate at which an Infected agent in the model reproduces the infection in the broader population, which we call the baseline reproductive rate, will be

$$R_0 = \frac{\beta}{\delta}. \quad (13)$$

[10]Equation (12) follows from substituting the equality $\mathbb{E}[z] = \mathbb{E}[z|z\leq\bar{z}]F(\bar{z}) + \mathbb{E}[z|z\geq\bar{z}](1-F(\bar{z}))$ into equation (11) and simplifying.

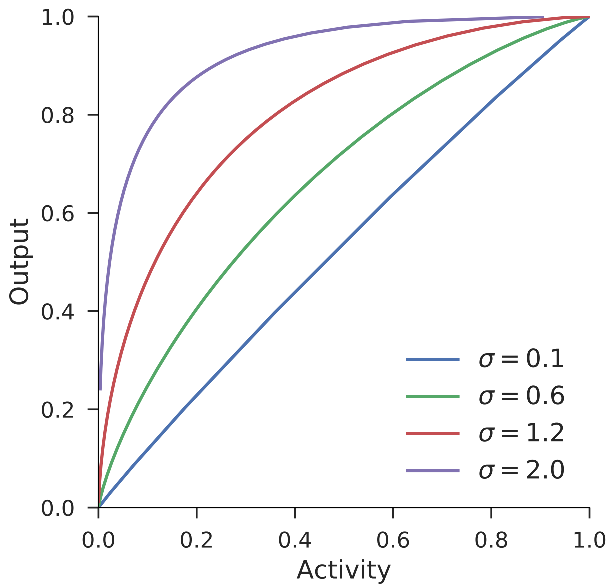


Figure 7: Relationship between economic output and activity given various values of σ, the standard deviation of $\log(z)$.

Namely, R_0 is the ratio of the rate at which Infecteds transmit the virus per unit of Susceptibles to the rate at which the Infecteds recover and stop transmitting the virus altogether. The baseline reproductive rate will obtain when $S(t)$ is near 1.

The effective reproductive rate, which accounts for the mitigating force of Susceptibles reducing their rate of economic activity when infections rise, will be

$$R_e = a_S(t)R_0 \,. \tag{14}$$

For $S(t)$ near one and very small values of $I(t)$, $a_S(t) \approx 1$ implying exponential growth of the infected share of the population with

$$\frac{\dot{I}(t)}{I(t)} = \beta - \delta. \tag{15}$$

The "Slow Burn" Regime. As the virus spreads, $a_S(t)$ will endogenously decline and become more important. The model then enters a pseudo-steady-state, in which I is approximately constant and R is still negligible, meaning

$$\beta \left[1 - F(\beta I(t)\psi)\right](1 - I(t)) = \beta a(t)S(t) = \delta \tag{16}$$

Writing this relationship in terms of $\bar{z}(t)$ gives

$$[1 - F(\bar{z})]\left(1 - \frac{\bar{z}(t)}{\beta\psi}\right) = \frac{\delta}{\beta} = \frac{1}{R_0} \tag{17}$$

For large $\beta\psi$, equation (17) has the approximate solution for the endogenous object $\bar{z}(t)$ of

$$\bar{z}(t) \approx F^{-1}\left(1 - \frac{1}{R_0}\right). \tag{18}$$

This immediately implies that the activity rate of Susceptibles satisfies

$$a_S(t) = \frac{1}{R_0} \quad \Rightarrow \quad R_e = 1 \tag{19}$$

Thus for sufficiently large $\beta\psi$ and while R is still relatively small, we should observe a constant fraction of infected people

$$I(t) = \frac{1}{\beta\psi} \cdot F^{-1}\left(\frac{R_0 - 1}{R_0}\right) \tag{20}$$

and constant levels of new infections

$$N(t) = \frac{1}{\psi R_0} \cdot F^{-1}\left(\frac{R_0 - 1}{R_0}\right) \tag{21}$$

Absent some other intervention, this "slow burn" regime will last until R becomes appreciably large, meaning "herd immunity" begins having an impact. As a simple example, if z is log-logistic distributed, the CDF will be $F(z) = \frac{z}{1+z}$ and the values of $I(t)$ and $N(t)$ are given by

$$I(t) = \frac{R_0 - 1}{\beta\psi} \quad \text{and} \quad N(t) = \frac{1}{\psi} \cdot \frac{R_0 - 1}{R_0}. \tag{22}$$

4 Structural Model II: Spatial Economic-SAIRD Model

The basic economic SIR model is too simplistic for a serious quantification. In this section, we develop a parsimonious spatial economic SAIRD-model that extends the economic SIR model along several dimensions. On the epidemiological side, we include two additional epidemiological states that are relevant to the analysis of COVID-19, as well a type of agent that is not concerned with contracting the virus. In addition, we allow for the possibility of eradicating the virus. On the economic side we now include multiple regions in the model (that we map to US counties) that will allow the model to capture the spread of the virus across space.

4.1 Epidemiological Environment

We begin by describing the expanded epidemiological environment. To ease with the exposition, we suppress the dependence of variables on region in this section.

Epidemiological States. The unit measure of individuals are partitioned across five epidemiological states: susceptible (S), asymptomatic (A), Infected (I), Recovered (R), or Dead (D), where we have added the A and D states. Asymptomatic agents carry and transmit the virus but show no symptoms, and indeed do not even know that they carry the virus. The other new state, Dead (D), is self explanatory. The inclusion of both the A and D states are consistent with, and are important aspects of, the COVID-19 experience. The other epidemiological states have the same interpretation as before. One might call the resulting model's epidemiological block SAIRD.

Epidemiological State Transitions. As in the simpler model, the S agents contract the virus at rate $n(t)$. The S category has no inflows from other states and hence the measure of Susceptibles decreases over time according to

$$\dot{S}(t) = -n(t)S(t). \tag{23}$$

We now assume that newly infected agents transition to the asymptomatic state. This feature of the model is consistent with reports that, when initially infected, agents are often asymptomatic but able to spread the virus for multiple days prior to developing symptoms. Furthermore, many infected individuals never exhibit symptoms at all (Russell et al., 2020), so we assume that agents in the A state can either develop symptoms (transitioning to I at rate λ) or recover without ever developing symptoms (transitioning to R at rate γ). The measure of $A(t)$ thus changes according to

$$\dot{A}(t) = n(t)S(t) - \lambda A(t) - \gamma A(t) \tag{24}$$

The measure of agents in the infected state increases with the inflow of $\lambda A(t)$ agents, while current $I(t)$ individuals recover at rate δ and die at rate κ.

$$\dot{I}(t) = \lambda A(t) - \delta I(t) - \kappa I(t) \tag{25}$$

Lastly, the R and D states are absorbing states, as they each have inflows but no outflows, with measures only increasing over time.[11] With asymptomatic and infected agents recovering at rates γ and δ respectively, $R(t)$ increases at rate

$$\dot{R}(t) = \gamma A(t) + \delta I(t). \tag{26}$$

With infected agents perishing at rate κ, $D(t)$ increases at rate

$$\dot{D}(t) = \kappa I(t). \tag{27}$$

Equations (23)–(27) characterize the epidemiological dynamics in the economic-SAIRD model, given the parameters governing epidemiological state transition rates, λ, δ, κ, and γ and the infection rate of Susceptibles, $n(t)$.

4.2 Economic Environment

We now assume that there are two types of economic agents. The first type behaves identically to the Susceptibles in the previous section's economic-SIR model—they are concerned about contracting the virus and optimally limit market activity in response to the infection' spread. Hence, we call these agents the "concerned." The second type of agent is "unconcerned" and does not adjust economic activity as the pandemic spreads. The second type of agent behaves identically to the standard SIR model. Thus, our model consists of a mix of cautious, optimizing agents, and automatons.

We assume that agents who are asymptomatic are unaware of the fact that they have contracted the virus and hence we model their economic behavior as identical to that of Susceptibles. Once agents become Infected, Recovered, or Dead, we assume that they know their status and there is no risk of infection, hence there is no meaningful distinction between these groups with respect to their economic behavior We denote the share of unconcerned agents as u, and the measures of each type if the S and A states as S_C and A_C for concerned and S_U and A_U for unconcerned.

[11] We are implicitly assuming that past infection to COVID-19 provides future immunity, at least over the time horizon relevant to the model estimation and usage.

Economic Decision: To z, or Not To z? Just as in the simpler economic SIR model, agents face economic needs of varying importance according to $F(z)$ and satisfying any need requires a risky excursion. The defining characteristic of the unconcerned types in the model is that they take an excursion to satisfy any economic need. This leaves only the S_C and A_C agents as the substantive economic decision makers.

Let $e(t)$ denote the share of the population that is both carrying the virus and economically active. Conditional on taking an excursion, we assume the probability of a Susceptible agent contracting the virus is proportional to $e(t)$, taking the form $\beta e(t)$. Agents choose to undertake an excursion to satisfy a given need z if the benefit exceeds the expected cost:

$$z > \beta \cdot e(t) \cdot \psi \equiv \bar{z}(t). \tag{28}$$

$\bar{z}(t)$ is the reservation value for undertaking an excursion, which varies endogenously over time through its dependence on $e(t)$.

The measure of economically active virus carriers, $e(t)$ is determined by the choices of A and I agents. With S_C and A_C agents following the decision rule in (28), but subject to idiosyncratic economic needs $F(z)$, the fraction of both S and A going out at time t, denoted by $a_S(t)$, is the fraction with draws of z greater than $\bar{z}(t)$ plus the share of unconcerned agents, u.

$$a_S(t) = u + (1 - u)[1 - F(\bar{z}(t))]. \tag{29}$$

We assume that infected individuals quarantine themselves by undertaking actions no less valuable than some level $\bar{z}_I$, which leads to an activity level among I agents of $a_I = 1 - F(\bar{z}_I)$. The non-quarantined I agents are economically active and thus contribute to the infectiousness of the economic environment. $e(t)$ then takes the form

$$e(t) = a_S(t)A(t) + a_I I(t). \tag{30}$$

Tracing through equations (28)–(30), one can see that $\bar{z}(t)$ and $e(t)$ ought to be simultaneously determined. For now we side-step this fixed-point problem by assuming that the concerned agents base their risk assessment on a moving average of recent activity, rather than current $e(t)$.

Endogenous Infection Rates from Economic Activity. The rate at which Susceptibles become infected, $n(t)$, is then given by the product of three terms: the base rate of virus transmission from virus carriers to Susceptibles, the rate at which virus carriers undertake activity, and the rate at which Susceptibles make excursions ($a_S(t)$):

$$n(t) = \beta \cdot e(t) \cdot a_S(t). \tag{31}$$

Economic Activity. The rate of activity is dictated by the recovered agents and the active shares of Susceptibles, Asymptomatics, and Infecteds. One can then characterize the rate of activity either constructively, as in equation (32), or as a deviation from the full activity rate, as in equation (33):

$$\begin{aligned} a(t) &= a_S(t)\big[S(t) + A(t)\big] + a_I I(t) + R(t) && (32) \\ &= 1 - \underbrace{F(\bar{z}(t))}_{\substack{\text{rate of foregone} \\ \text{excursions by } S \text{ \& } A}} \cdot \big[S(t) + A(t)\big] - \underbrace{F(\bar{z}_I) I(t) - D(t)}_{\substack{\text{missing excursions by} \\ \text{quarantined and deceased}}} && (33) \end{aligned}$$

Economic Output. Similarly, output can be equivalently characterized either constructively or in terms of shortfall from output when there are no infections:

$$\begin{aligned} y(t) \;=\; & \overbrace{\mathbb{E}[z(t)]}^{\substack{\text{“no virus”} \\ \text{output rate}}} - \big[S(t) + A(t)\big] \cdot \overbrace{F(\bar{z}(t))}^{\substack{S(t) \text{ foregone} \\ \text{excursion rate}}} \cdot \overbrace{\mathbb{E}[z(t) | z(t) \leq \bar{z}(t)]}^{\substack{\text{value of foregone} \\ \text{excursion}}} \\ & - \underbrace{F(\bar{z}_I) I(t) \mathbb{E}[z(t) | z(t) \leq \bar{z}_I]}_{\text{foregone value by quarantined } I} - D(t) \cdot \mathbb{E}[z(t)] \end{aligned} \tag{34}$$

4.3 Multiple Geographic Regions

The model presented to this point assumes that economic activity and virus transmission all occur within a single region. That model abstracts from some important features of the COVID-19 environment. In the extended, spatial version of the model all endogenous objects are implicitly indexed by their location i.

Distinct environments regions. In reality, different regions of the country may differ in a variety of ways that affect both the rate of disease transmission and the cost of infection. To reflect this possibility, we allow different regions to have distinct values of β and ψ, now more appropriately denoted β_i and ψ_i. We will describe this in greater detail in the estimation section.

Interaction between regions. Another important real-world challenge in managing the COVID-19 virus is the ability of agents in one region to spread the virus to agents in another region. To capture this aspect of the transmission environment, we extend the model to allow for inter-region activity and hence also risk of inter-region contamination. We introduce this mechanism into the model by replacing the term $\beta \cdot e(t)$ in equation (31) with $\beta_i^* e_i^*(t)$, where

$$\beta_i^* e_i^*(t) = (1-\alpha)\beta_i e_i(t) + \alpha \sum_i w_i \beta_i e_i(t). \tag{35}$$

where the weights w_i are proportional to the relative populations in the various regions and $\alpha \in [0,1]$ is a weight on the population weighted national infectiousness.

4.4 Policy Interventions

We consider two types of policy interventions for managing the spread of the virus.

Shutdown orders (extensive margin). One policy lever is the imposition of NPIs that restrict agents activity and mobility, such as lockdown orders. NPIs enter the model by imposing a minimum $\bar{z}^P(t)$ that agents internalize when deciding whether or not to undertake an excursion. Hence the *effective* reservation value of z for agents is given by

$$\bar{z}^E(t) = \max\left\{\bar{z}(t)\,, \bar{z}^P(t)\right\}. \tag{36}$$

Two aspects of this policy are worth pointing out. First, equation (28) (and equation (5) in the SIR model) describes how the optimizing behavior of agents takes the form of an endogenous $\bar{z}(t)$. From (36), one can see that if a policymaker imposes a $\bar{z}^P(t)$ below agents' endogenous $\bar{z}(t)$, then this has no effect on the evolution of any variables in the model. Second, the policy options are notably asymmetric, in the sense that policymakers can always raise the effective $\bar{z}^E$, but at no time can they lower the effective value below $\bar{z}$. Put more directly in real-world terms, policymakers can compel people to stay in, but cannot force people to go out.

Transmission mitigation per unit of activity (intensive margin). Another policy lever is requiring behaviors that mitigate transmission per unit of economic activity. In the real world, such behaviors take the form of wearing face masks, social distancing, improved sanitation practices, or allowing restaurant dining only outdoors.[12] Taking the face mask as the leading example, the idea here is that steps can be taken to lessen the risk of disease spread while conducting otherwise normal activity. We represent the evolution of this behavior in our model as a common factor that decreases the values of β over time. We denote

[12]Following a change in CDC guidance on April 3, 2020, nearly all states now require, or at least recommend, the wearing of face masks in public spaces.

Parameter	Interpretation within SAIRD model
Epidemiological State Transition Rates	
λ	infection rate for exposed
γ	recovery rate for exposed
δ	recovery rate for infected
κ	death rate for infected
Economic Environment	
β	infection rate per excursion
ψ	present cost of infection
$\bar{z}_i$	quarantine rate of infected
σ	variance of economic needs, $F(z)$
f_0	initial ratio of exposed to infected

Table 1: Model Parameters

these values $\widetilde{\beta}(t)$ and assume that they evolve according to an exponential decay process,

$$\widetilde{\beta}(t) = \widetilde{\beta}_0 + \left(\widetilde{\beta}_1 - \widetilde{\beta}_0\right)\Phi(\lambda_\beta t) \tag{37}$$

where $\widetilde{\beta}_0$ is normalized to 1, $\widetilde{\beta}_1$ represents the eventual reduction in β, λ_β governs the speed of this transition, and Φ is the smoothstep sigmoid function.

5 Estimating the Economic-SAIRD Model

To make useful quantitative statements with our Economic-SAIRD model, we infer parameter values based on ability to match the evolution of both economic and epidemiological variables. Letting $\boldsymbol{\theta}$ denote the model's free parameters, and $\{\bar{z}^P_{i,0:T}\}_i$ be the collection of time series of NPI orders for each location, the model predicts values for sequences of new infections ($\dot{I}_{i,1:T}$), new deaths ($\dot{D}_{i,1:T}$), economic activity ($a_{i,1:T}$), and economic output ($y_{i,1:T}$) at each date and location.

We estimate $\boldsymbol{\theta}$ from panel of daily, county-level observations over the period from January 1, 2020 to May 24, 2020 on NPIs, COVID-19 cases, COVID-19 deaths, point-of-interest visits, and hours worked, as described in Section 2.1. After removing counties with insufficient data, the estimation uses 1340 U.S. counties for inference, accounting for about 85 percent of the U.S. population.[13] The parameter vector $\boldsymbol{\theta}$ includes all of the location specific values of $\{\beta_i, \psi_i\}_i$. Thus we allow a small degree of heterogeneity across counties, which is meant to reflect variation in exogenous factors that affect the disease transmission environment,

[13]We use only counties with populations of at least 30,000. Some counties are excluded because of missing observations in the Homebase data.

such as population density and demographics. Conditional on these differences, however, the mechanics of virus transmission and economic decision-making are assumed to work the same way everywhere.

5.1 Likelihood

Given parameters, initial conditions, and NPIs, the model generates a deterministic trajectory for epidemiological and economic variables. To infer the parameters of the deterministic structural model from the noisy observed data, we introduce a notion of measurement error for each observed variable.[14] We then take the resulting probabilistic relationships between the data and model-predicted trajectories as a likelihood function and maximize it with respect to the model's structural parameters $\boldsymbol{\theta}$.

Let $x_{i,t,j}$ denote observable variable j in county i at time t and $\mathbf{X} = \{x_{i,t,j}\}_{i,t,j}$ denote all of the data used for estimation. Let $\ell_j(\cdot)$ be a series-specific likelihood function with $\ell_j(\boldsymbol{\theta}|x_{i,t,j}) \propto p_j(x_{i,t,j}|\boldsymbol{\theta})$ for some density $p_j(\cdot)$. The full likelihood function then takes the form

$$L(\boldsymbol{\theta}|\mathbf{X}) = \prod_{i,t,j} \ell_j(\boldsymbol{\theta}|x_{i,t,j}). \tag{38}$$

We estimate $\boldsymbol{\theta}$ by maximizing L.

Likelihood for Economic Variables. For the economic variables, we assume mean-zero additive Gaussian measurement errors around $\hat{x}_{i,t,j}$, as in

$$x_{i,t,j} = \hat{x}_{i,t,j}(\boldsymbol{\theta}) + \epsilon_{i,t,j} \ , \quad \epsilon_{i,t,j} \sim \text{Normal}(0, \sigma_j^2/w_i) \tag{39}$$

where σ_j^2 are estimated series-specific error variances and the w_i values are weights proportional to the relative county populations. The $\epsilon_{i,t,j}$ values are assumed independent across i, t, and j. For $j \in \{Activity, Output\}$, the likelihood for observation $x_{i,t,j}$ is then

$$\ell_j(\boldsymbol{\theta}|x_{i,t,j}) \propto \text{Normal}\left(x_{i,t,j} \,|\, \hat{x}_{i,t,j}(\boldsymbol{\theta}),\, \sigma_j^2/w_i\right). \tag{40}$$

Likelihood for Epidemiological Variables. For $j \in \{Cases, Deaths\}$, the observed values $x_{i,t,j}$ are discrete and bounded below by zero, while the model predicts population fractions $\hat{p}_{i,t,j}$ of new infections and deaths. Given a county's true population size n_i, we

[14]Since the structural model is deterministic, without measurement error the likelihood function is degenerate.

can map the model's predicted population share into an expected count value as $n_i \hat{p}_{i,t,j}$.[15] We then model the observed value $x_{i,t,j}$ as arising from a Poisson distribution with rate parameter informed by the model's predicted count, $n_i \hat{p}_{i,t,j}$. Denoting the model's prediction as $\hat{x}_{i,t,j}$, the likelihood for cases and deaths takes the form[16]

$$\ell_j(\boldsymbol{\theta}|x_{i,t,j}) \propto \text{Poisson}\left(x_{i,t,j} \,|\, \hat{x}_{i,t,j}(\boldsymbol{\theta})\right). \tag{41}$$

We construct the predicted values according to

$$\hat{x}_{i,t,j} = \begin{cases} r_t \cdot n_i \cdot \dot{I}_i(t) & \text{for } j = Cases \\ n_i \cdot \dot{D}_i(t) & \text{for } j = Deaths \end{cases} \tag{42}$$

where for $j = Cases$, we account for the probable under-reporting of cases at dates early in the sample by introducing the reporting factors r_t. The values $r_t \in [0, 1]$, and decay exponentially to 1. Hence, in the limit, we assume cases are correctly reported. For purposes of estimation, we additionally scale both the data and model predictions by factors of 0.1 and 10 for cases and deaths, respectively.

6 Properties and Implications of the Fitted Model

In this section we examine a number of quantitative implications of the estimated Economic-SAIRD model. We conclude the section with discussion of some additional points on the interpretation of our results.

6.1 Fit and Forecasts

Figures 8 and 9 compare the fitted model to the data.

Near-term forecasts. Generating forecasts from the model requires taking a stand on the future values of $\bar{z}_{i,t}$.[17] We consider a few different potential paths of $\bar{z}_{i,t}$, and generate forecasts from our last data point, May 24, 2020, through September 7, 2020 (Labor Day in the United States).

[15] Note that the observed value $x_{i,t,j}$ satisfies $x_{i,t} = n_i p_{i,t}$ for a true population fraction $p_{i,t}$.

[16] Recall that the expected value of a Poisson distribution is the value of the rate parameter.

[17] Since the shutdown orders are not endogenous in the model, there is no fully internally consistent method of handling this.

We forecast the future path of the virus under two different policies: 1) continuation of status quo lockdowns; and 2) ending all lockdowns as of June 1, 2020. Under both policies, we predict a steady decline in death rates through Labor Day 2020, despite a relatively stable evolution in total cases. The stable linear increase in cases of about 20,000 per day masks significant heterogeneity across locations. Without a lockdown some counties will see daily cases continue to rise through the summer, whereas others will see declines, whereas continuing lockdowns will lead to a uniform decline in daily cases. The median county will see a decline in new daily cases of about percent over this period. At the same time, we see a nearly uniform decrease in daily deaths across locations. The discrepancy between the evolution of cases and deaths can be explained by continued improvements in testing capacity.[18] If lockdowns continue, we predict little to no improvement in economic activity or output through Labor Day. By ending the lockdowns we predict a roughly 10 percent increase in activity and 7 percent increase in output between June 1 and Labor Day. While these increases are significant, activity and output are still significantly below their pre-pandemic levels. Agents still fear infection and reduce activity even in the absense of formal lock downs. Our findings crystallize the notion that "reopening" is easier said than done. As long as people fear infection from the virus, they will continue to curtail their economic activity, leading to a decline in output.

6.2 Counterfactuals and Policy Experiments

In this section we examine the fitted economic SAIRD model's implications for the trajectories of both the virus and the economy under a number of different policy scenarios. First, we examine the scenario in which policy-makers "do nothing." In the do nothing scenario, policymakers issue no lock-down orders, of any kind, anywhere, ever. Second, we consider optimal policy. Optimal policy entails maximizing the difference between the benefits of economic activity and the costs of infection. Third, we assess the costs and benefits of imposing lockdowns sufficiently stringent to eradicate the virus entirely.

6.2.1 Lockdown

In Figure 10, we show the model outcome given a fairly stringent lockdown lasting for three months. Here we can see that the lockdown is effective at reducing infection rates in the short run. However, after roughly two months, infections come surging back, albeit to relatively low

[18] We predict that throughout the summer the extent of under-reporting of true cases will continue to decline.

levels. On the economic side, there is a brief period of high output, followed by a subsequent collapse due to renewed avoidance.

6.2.2 Adaptive Lockdown Policy for Eradication

The next policy we consider is a readily implementable policy that enforces a lockdown in location i whenever the infected share of the population exceeds some threshold, $\bar{I}$.

$$\bar{z}_i^P(t) = \begin{cases} \bar{z}^* & \text{if } I_i(t) \geq \bar{I} \\ 0 & \text{otherwise} \end{cases} \tag{43}$$

We simulate the effects of this policy with an Infected threshold of $\bar{I} = 150/(1e6)$ (150 cases per million people) and lockdown level sufficient to reduce economic activity to 20 percent of its normal level, $\bar{z}^* = F^{-1}(0.2)$. Under this policy, of adaptive, location-specific lockdowns, output is initially reduced to the targeted 20 percent level in most locations, but is allowed to return to 100 percent of its normal level in roughly two months.

In this scenario we assume that if the measure of infected agents is driven below a certain threshold, 100 infections per million, then the virus is eradicated. To put this in perspective, the median county in the U.S. has a population of approximately 25,000, so we assume the virus is eradicated when on average 1/4 of a person becomes infected each day (in the median county).

Figure 11 shows simulations from a policy sufficient to eradicate the virus. While the model says that such an outcome is feasible, the economic implications of a sufficiently stringent policy are stark: to achieve eradication by means of lockdown orders would require that economic activity and output be driven below 20 percent of normal levels for roughly a month in most locations.

6.3 A Longer Run Simulation: Laissez-Faire Until Herd Immunity

The concept of herd immunity is defined as the point at which the inflow of new infections is just equal to the outflow rate. Once a population reaches herd immunity, new infections begin to fall quickly. In our model, the process of reaching herd immunity is, in principle, somewhat more nuanced than in simpler models without endogenous agent responses to the epidemiological environments. However, as herd immunity is approached in the model, economic activity increases and approaches its baseline level, yielding dynamics similar to those in model with a more simplistic infection mechanism. Achieving this outcome in the full cross-section takes about 55 percent of the population having, at some point, been infected and the process takes roughly 9 years to play out, after which infections steadily die out over the next 3 years. As this process plays out, cumulative mortality is about 0.3 percent of the population. Figure 12 shows the the trajectory of economic and epidemiological variables as this process plays out.

6.4 Discussion

On heterogeneous risk groups. Our model predicts trajectories of infections and deaths for the U.S. population and for geographically disaggregated sub-populations down to the county level. However, the model does not make predictions for other specific types of disaggregation, such as demographic. Although nationwide hard data remains somewhat lacking, there is substantial evidence that the burden of COVID-19 mortality is borne disproportionately by the elderly and infirm. For example, at time of writing, the detailed data on the breakdown of deaths in New York City show nearly half of COVID-19 deaths concentrated in the 75+ age range, and another quarter accounted for by the 65-74 age range.[19]

On the COVID-19 deaths data. While we build under-reporting of cases into our estimation, we do not do so for deaths. We estimate our model to best match the *reported* data for COVID-19 deaths. A number of journalistic outlets have documented evidence of "excess deaths" in a number of locations, which meaningfully exceed the number of reported COVID-19 deaths in those locations. To the extent that official death totals are meaningfully under-reported, the true mortality rate in our model (κ) should be higher, and readers of this mindset would want to accordingly adjust upwards our mortality predictions.

7 Conclusions

In this paper we developed and estimated a spatial model of the joint evolution of economic variables and the spread of COVID-19 across U.S. counties using high-frequency granular data. The model predicts a significant endogenous reduction in economic activity by agents in response to the spread of the virus. Policy interventions did succeed in lowering the spread of the virus, but by April, in the vast majority of counties they were no longer binding. This results highlights the challenges facing policy makers as they seek to "reopen" the economy. As long as the virus continues to spread, individuals will fear infection and optimal choose

[19] See `https://www1.nyc.gov/site/doh/covid/covid-19-data.page` and `https://github.com/nychealth/coronavirus-data`.

to socially distance. Even if all NPIs are eliminated, we only find about a 10% increase in activity over the next three months (compared to a 40% decline).

Our estimated model presents a sobering picture for the dynamics of the virus and economic activity going forward. Absent pharmaceutical advances to vaccinate against or treat the virus, we predict a protracted march towards herd immunity. Infections and deaths continue to increase roughly linearly for the next decade with an ultimate death toll of around 1,000,000.

Agents in the model optimally trade-off engaging in market consumption with the risk of contracting the disease. To motivate the model, we use three novel county-level data sets to document key empirical relationships between non-pharmaceutical interventions (NPIs), health, mobility, and employment outcomes at a daily frequency. We investigate the relative importance of NPIs, such as stay-at-home orders, and endogenous social distancing. Finally, we use our estimated model to address key issues in battling the pandemic: How will the spread of the disease and economic activity evolve if current NPIs are relaxed? What are optimal implementable NPI policies, taking into account the trade-off between the spread of the virus and economic activity?

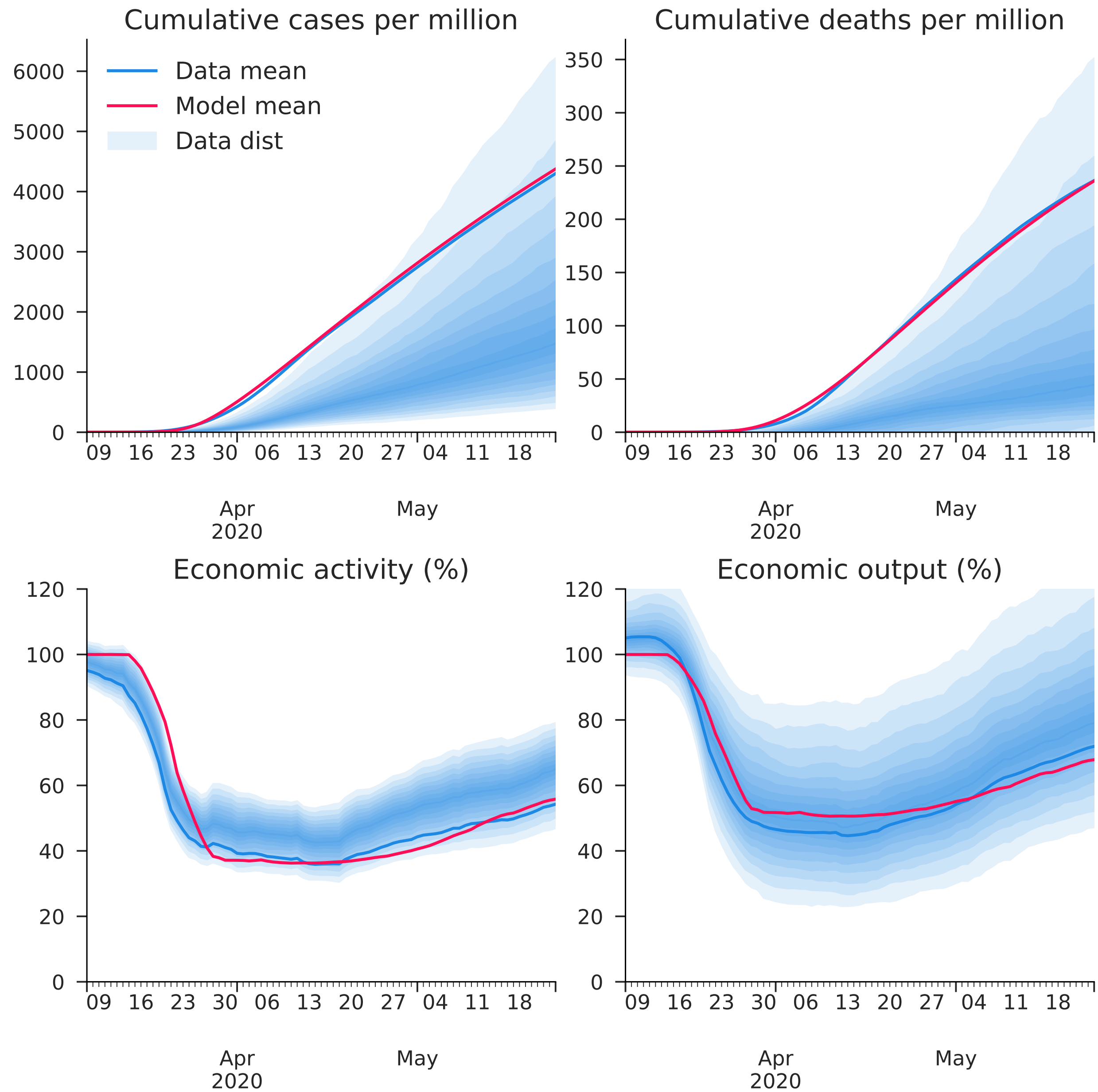


Figure 8: Model fit

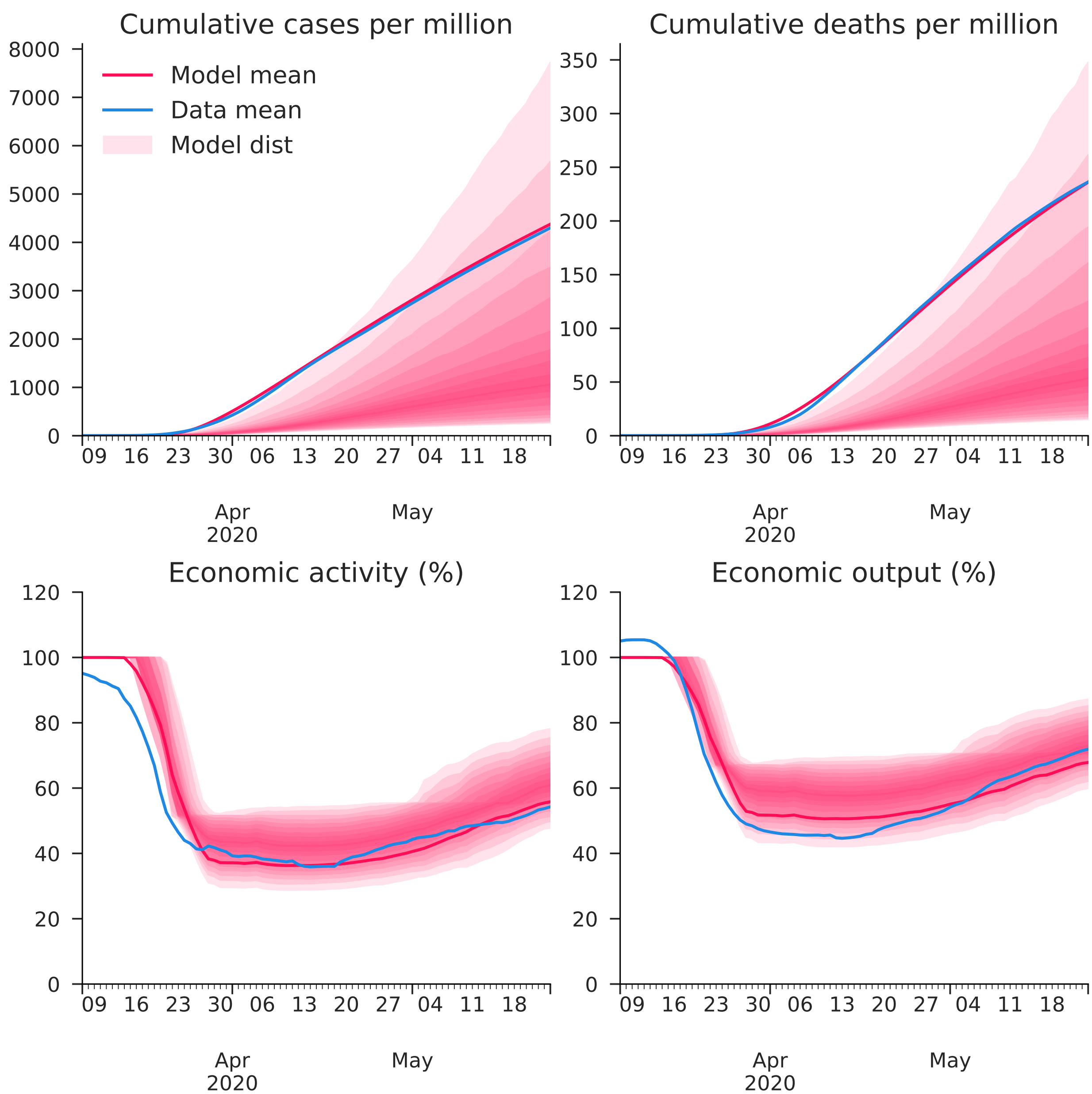


Figure 9: Model fit

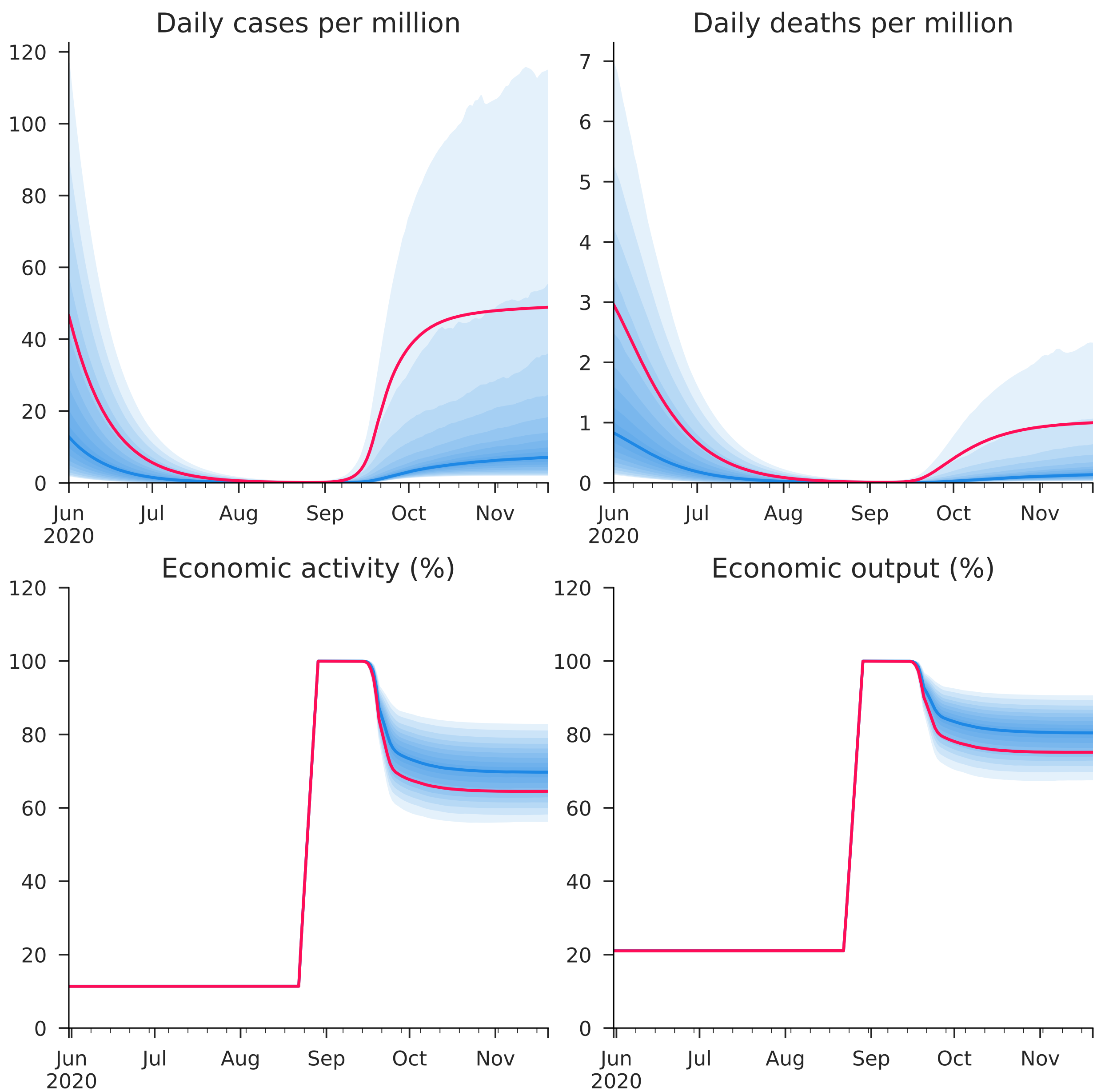


Figure 10: Stringent lockdown policy lasting for two months (weekly smoothing).

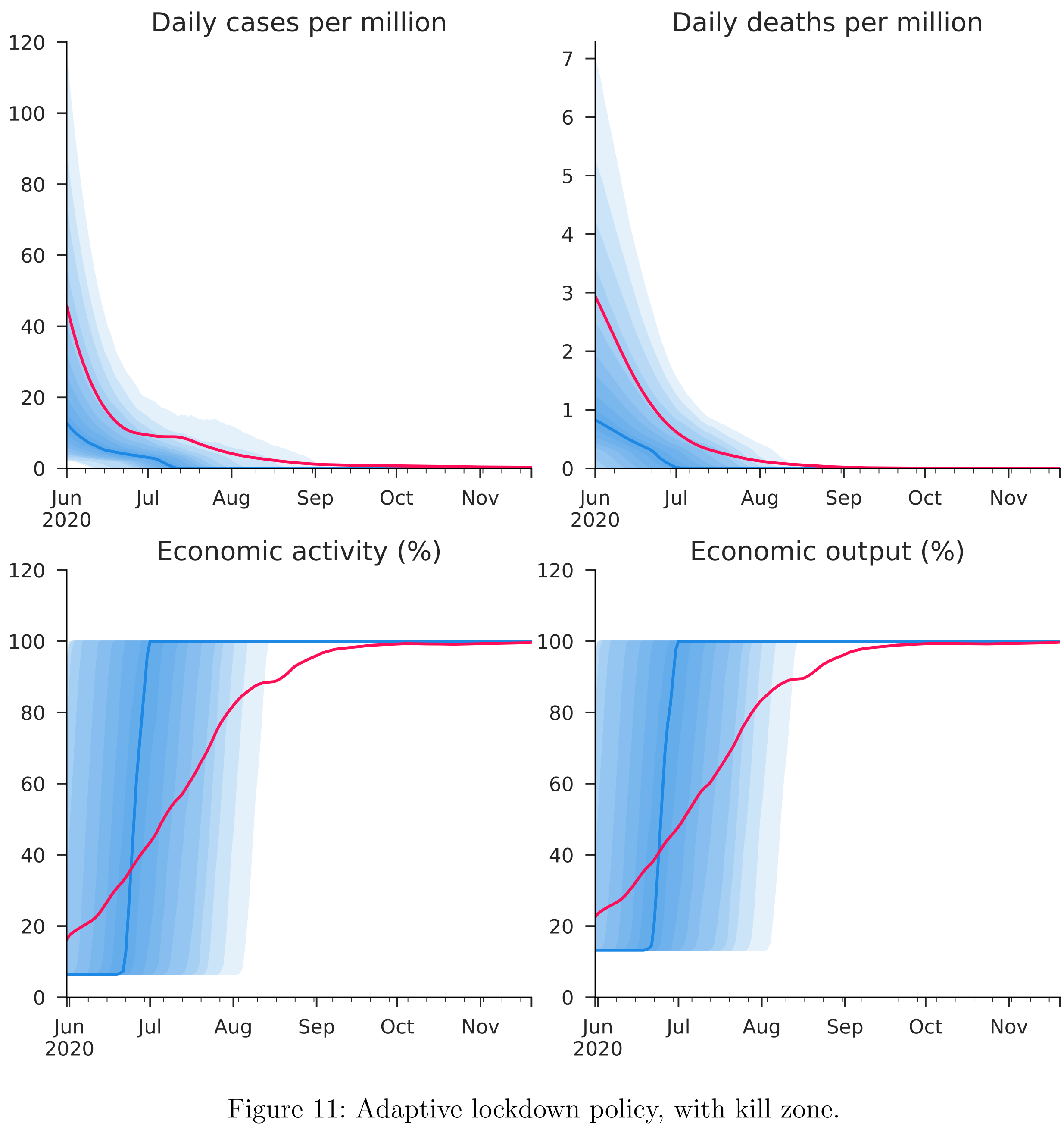


Figure 11: Adaptive lockdown policy, with kill zone.

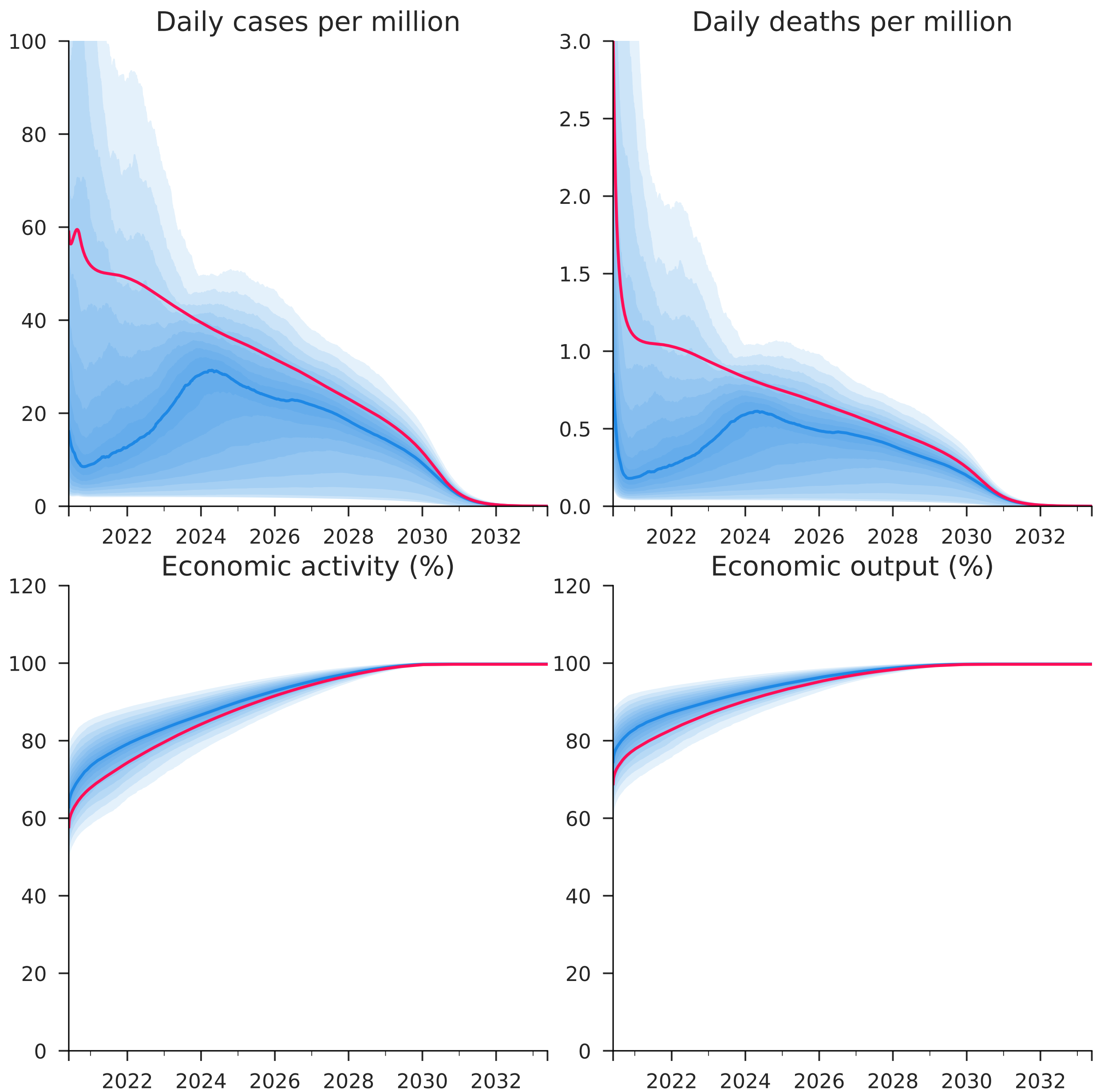


Figure 12: **Laissez-Faire until herd immunity.** Red lines are population-weighted means, thick blue lines are for the median county, and the light blue lines trace out bands of the full cross-sectional distributions.

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NAICS	Firms HB	Firms QC	Wages HB	Wages QC	Weekly Wage HB	Weekly Wage QC
21		0.00		0.01		2,406
22		0.00		0.01		2,641
23		0.08		0.06		1,193
31		0.04		0.12		1,419
42		0.06		0.06		1,612
44	0.14	0.11	0.15	0.07	331	636
48	0.01	0.03	0.01	0.04	363	1,086
51		0.02		0.05		2,506
52		0.05		0.12		2,867
53		0.04		0.02		1,256
54	0.05	0.13	0.04	0.12	336	1,949
55		0.01		0.05		3,001
56		0.06		0.05		834
61	0.04	0.01	0.02	0.02	158	973
62	0.08	0.17	0.06	0.13	282	963
71	0.02	0.02	0.02	0.01	181	712
72	0.37	0.07	0.46	0.04	205	420
81	0.04	0.09	0.02	0.02	313	758
99	0.25	0.02	0.22	0.00	242	1,070

Table 2: Comparison of Homebase and Quarterly Census of Employment and Wages

A Data Description

Here we provide the details on our data sources and construction.

A.1 COVID-19 Cases and Deaths

We use the county-level deaths information as collected and posted by the New York Times. This is with one exception: the NYT data only records deaths for NYC as a whole, although the Bronx and Queens are distinct counties. We collect these observations by hand from XX and add them to our data set.

A.2 Activity and Output

Activity. Our activity data comes from Safegraph.

Output. For output we use hours worked from Homebase.

Model Parameter	Value
$\mu[\beta]$	1.1628
$\sigma[\beta]$	0.6514
$\mu[\psi]$	749.1
$\sigma[\psi]$	98.3
λ	0.0182
γ	0.0641
δ	0.0771
κ	0.0017
σ	0.3894
f_0	0.0000
z_i	2.3900
$\bar{z}_1^L$	0.9090
$\bar{z}_2^L$	0.8735
σ_a	0.1266
σ_o	0.1848

Table 3: Point estimates of model parameters.

B Additional Estimation Output

C Figures

C.1 Policy

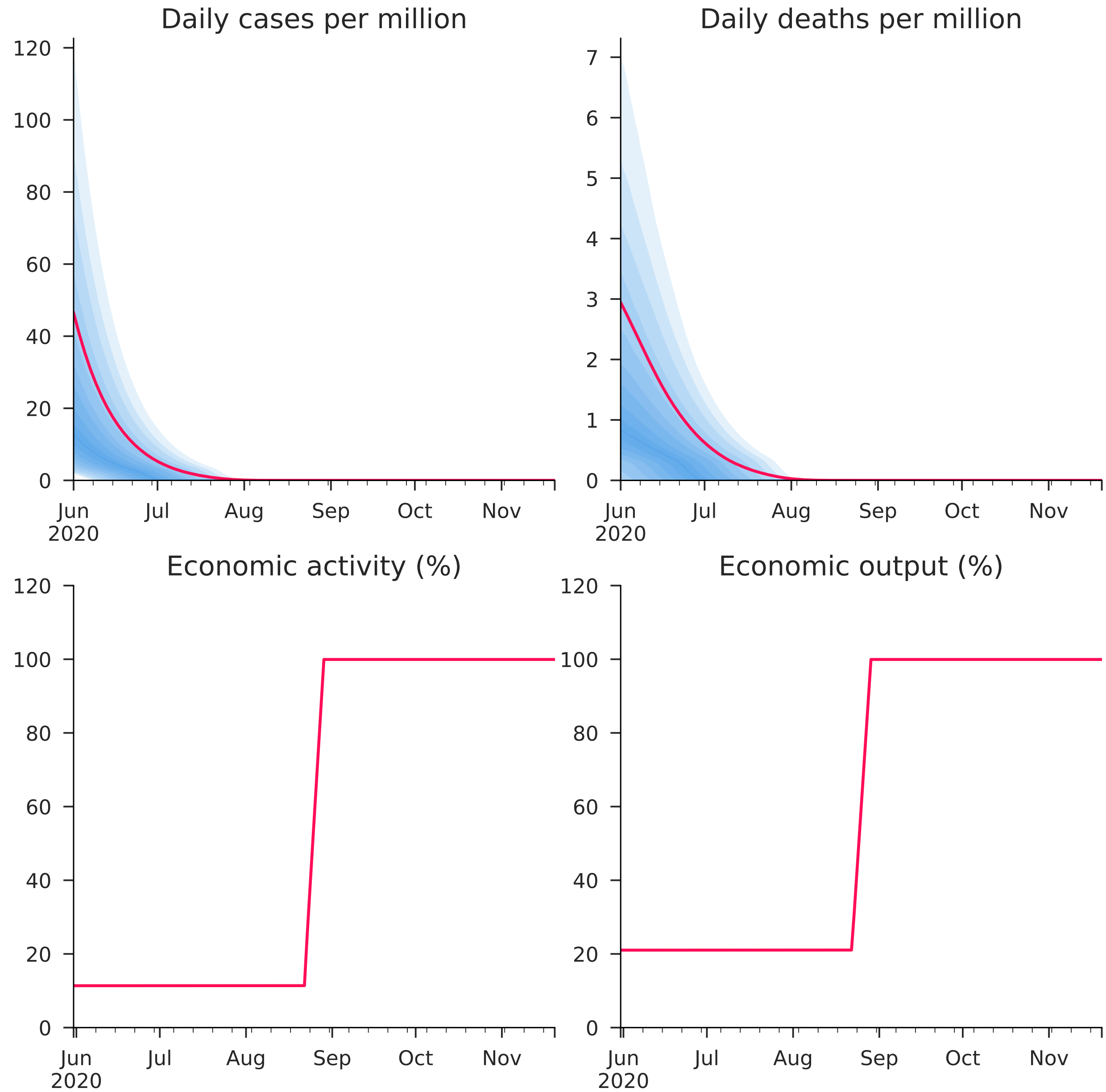

Figure 13: Stringent lockdown policy lasting for three months, with kill zone (weekly smoothing).

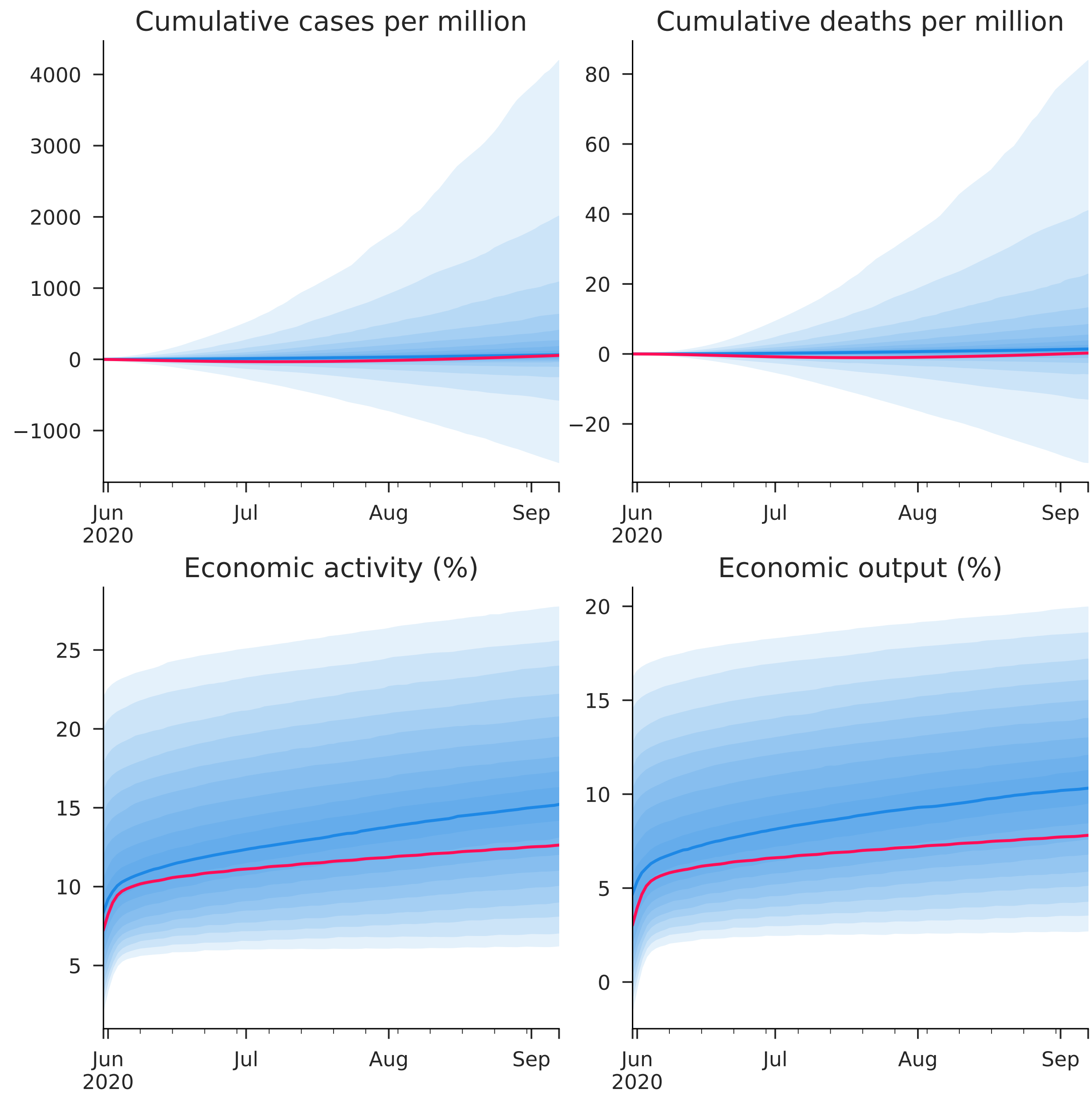

Figure 14: Difference in four main indicators between perpetual no lockdown and perpetual lockdown (weekly smoothing).

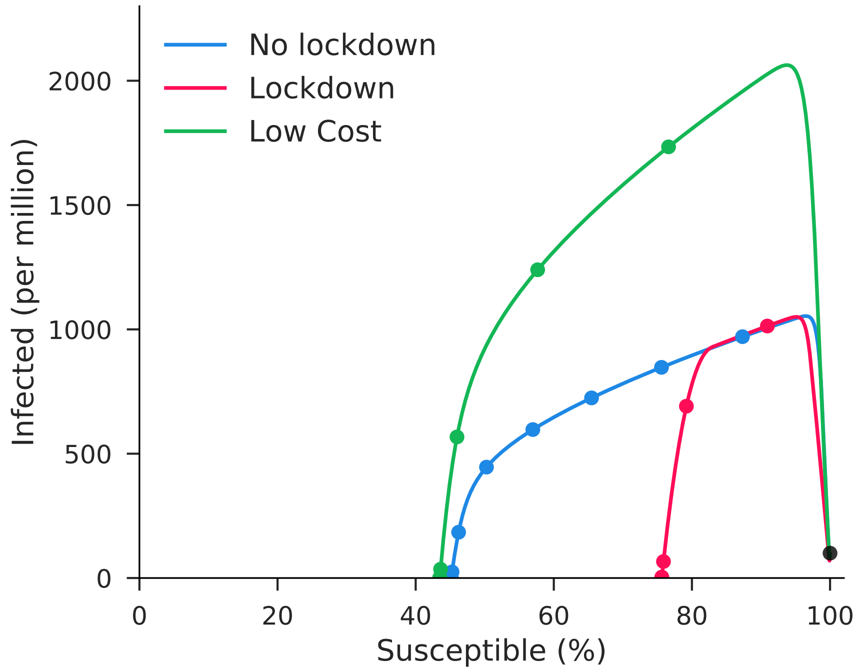

Figure 15: Phase diagram for model with endogenous activity for no lockdown, strict lockdown, and low cost (no lockdown) scenario. Indicator dots are spaced one year apart.

C.2 Phase Diagrams

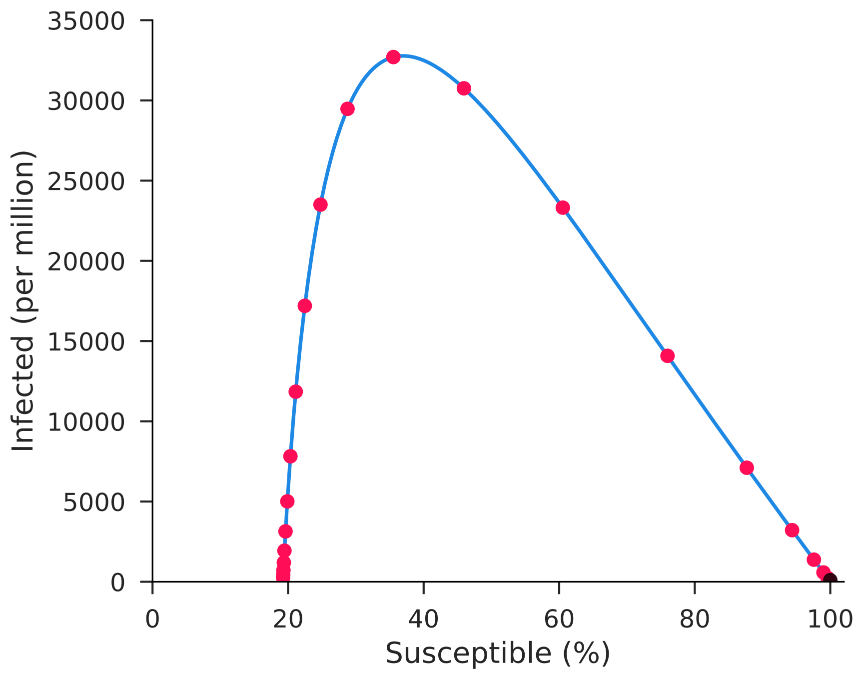

Figure 16: Phase diagram for model with low endogenous activity (ψ set at 1% of baseline value). Red dots are spaced one week apart.