Tracking $\mathcal{R}$ of COVID-19

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Abstract

We develop a new method for estimating the effective reproduction number of an infectious disease ($\mathcal{R}$) and apply it to track the dynamics of COVID-19. The method is based on the fact that in the SIR model, $\mathcal{R}$ is linearly related to the growth rate of the number of infected individuals. This time-varying growth rate is estimated using Kalman-filtering techniques from data on new cases. The method is very easy to apply in practice, and it performs well even when the number of infected individuals is imperfectly measured, or the infection does not follow the SIR model. The estimates of $\mathcal{R}$ for COVID-19 are provided in an online dashboard, and they are used to assess the effectiveness of non-pharmaceutical interventions in a sample of 14 European countries.

1 Introduction

The effective reproduction number ($\mathcal{R}$) plays a central role in the epidemiology of infectious diseases.[1] $\mathcal{R}$ is given by the average number of people infected by a single infectious individual. In standard models, the number of infected individuals increases as long as $\mathcal{R} > 1$. In an epidemic, real-time estimates of $\mathcal{R}$ are therefore essential for various public policy decisions (Atkeson, 2020; Leung, 2020). Such estimates can be used to study the effectiveness of non-pharmaceutical interventions (NPIs), or assess what fraction of the population needs to be vaccinated to reach herd immunity (Chinazzi et al., 2020; Kucharski et al., 2020; Wang et al., 2020). Some social scientists

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[1] For overviews of the benchmark models and the role of $\mathcal{R}$, see, among others, Chowell and Brauer (2009), Nishiura and Chowell (2009), Allen (2017), and Stock (2020).

have argued that $\mathcal{R} < 1$ should be viewed as a fundamental constraint on public policy during the current COVID-19 pandemic (Budish, 2020).

In this paper we develop a new way to estimate $\mathcal{R}$ in real time. The method is based on the fact that in the benchmark SIR model (Kermack and McKendrick, 1927), $\mathcal{R}$ is linearly related to the growth rate of the number of infected individuals. In the first step of the procedure, we use data on new cases to construct a time series of how many individuals are infected at a given point in time. Then, we estimate the growth rate of this time series by standard Kalman-filtering techniques. In the final step, we use a theoretical relationship implied by the SIR model to invert this growth rate for $\mathcal{R}$. The method is robust in the sense that the estimates remain fairly accurate even when new cases are imperfectly measured, or the true dynamics of the disease do not follow the SIR model.

We apply the new methodology to estimate $\mathcal{R}$ of COVID-19 in real time. As of April 29, The World Health Organization has confirmed more than three million cases of COVID-19 worldwide (World Health Organization, 2020). We use our estimates to calculate the basic reproduction number ($\mathcal{R}_0$) and evaluate the effectiveness of NPIs in a sample of 14 European countries. The estimates of $\mathcal{R}$ for COVID-19 are provided in an online dashboard and can be explored interactively (link to dashboard).

Relation to Existing Literature

There are two broad classes of existing methods that can be used to estimate $\mathcal{R}$ in real time (Chowell and Brauer, 2009; Nishiura and Chowell, 2009). First, one can estimate a fully-specified epidemiological model and then construct a model-implied time series for $\mathcal{R}$. Second, one may use approaches that leverage information on the serial interval of a disease (i.e., time between onset of symptoms in a case and onset of symptoms in his/her secondary cases). For example, imagine a disease with a fixed serial interval of, say, three days. In that case, we could estimate $\mathcal{R}$ by simply dividing the number of new cases today by the number of new cases three days ago. Cori et al. (2013) exploit this idea to develop a Bayesian estimator that accounts for the randomness in the onset of infections as well as variation in the serial interval; see also Thompson et al. (2019).[2] This method is implemented in a popular R package `EpiEstim`.[3]

[2] Wallinga and Teunis (2004) develop a related maximum-likelihood based method for estimating the so-called case reproduction number (Fraser, 2007). This approach has been implemented in an R software package `R0` by Obadia et al. (2012). However, as discussed by Cori et al. (2013), this approach cannot be used for real-time estimation of $\mathcal{R}$. The method proposed in the current paper estimates what is referred to as the instantaneous reproduction number in the literature.

[3] A team of researchers led by Timothy Churches and Nicholas Tierney has developed an online dashboard showing estimates of $\mathcal{R}$ for COVID-19 using `EpiEstim` for a number of countries (link).

The method proposed in this paper attempts to strike a balance between the two approaches mentioned above. Although our estimator is derived from standard epidemiological theory, we use the smallest amount of theoretical structure that is necessary to obtain our estimator. In particular, the theoretical relationship used to derive our estimator is exactly valid not only in the benchmark SIR model, but also in the SIS model and a generalized SIR model with time-varying parameters and stochastic shocks. Relative to the existing literature, our estimator does not need any tuning parameters, and it does not require parametric assumptions on the distribution of new cases (such as assuming that new cases are Poisson distributed).[4] Our approach and its mathematical derivation share some similarities with the estimator proposed by Bettencourt and Ribeiro (2008).[5]

A key advantage of using standard time-series filtering tools for estimating $\mathcal{R}$ is that valid confidence bounds are readily obtained. In addition, explicitly accounting for the dynamics in $\mathcal{R}$ is likely to produce better-behaved estimates in practice.[6] Finally, we note that in contrast to most existing methods, our method does not necessarily require Bayesian methods for estimation, and classical techniques can also be employed.

In independent contemporaneous work, Rondón-Moreno, Arroyo Marioli, and Bullano (2020) have developed a very similar estimator. These authors use the SIR model to develop an estimator based on Kalman-filtering techniques. Relative to their work, the current paper proposes an estimator based on growth rates, rather than changes in the number of infected individuals. The simplicity of our estimator allows us to analytically characterize the effects of measurement error, and show that the estimator remains exactly valid in the SIS and a generalized SIR model. We also document that our estimator is approximately valid in the SEIR model. Finally, we use the estimates to assess non-pharmaceutical interventions in a sample of European countries.

2 New Real-Time Estimator

We now derive our estimator for the baseline SIR model (Kermack and McKendrick, 1927). In the Appendix, we show that the same estimator obtains in the SIS model (Section A.2), and a generalized SIR model with time-varying parameters and stochastic

[4] For example, the method of Cori et al. (2013) assumes that $\mathcal{R}$ is constant over fixed windows of duration τ; the τ parameter effectively becomes a tuning parameter that needs to be chosen by the user.

[5] A team of analysts led by Kevin Systrom has developed an online dashboard tracking $\mathcal{R}$ for the US (both at the state and federal levels) using this estimator (link).

[6] For instance, the Cori et al. (2013) estimation procedure assumes that $\mathcal{R}$ remains constant over fixed windows of time (say, one week). As a result, confidence bounds obtained by their method quantify the uncertainty about the average value of $\mathcal{R}$ over one week, which is generally not the quantity of interest. In addition, their method does not impose any dynamic structure on the behavior of $\mathcal{R}$ over time, and hence the estimates can fluctuate significantly.

shocks (Section A.3).

2.1 Deriving the Estimator

The standard SIR model describes the evolution of susceptible (S_t), infected (I_t), and recovered (R_t) individuals by the following equations:

$$\begin{aligned} S_t &= S_{t-1} - \beta I_{t-1}\frac{S_{t-1}}{N} \\ I_t &= I_{t-1} + \beta I_{t-1}\frac{S_{t-1}}{N} - \gamma I_{t-1} \\ R_t &= R_{t-1} + \gamma I_{t-1} \end{aligned} \tag{1}$$

The model is stated at a daily frequency.[7] Here, N is population size, β is the daily transmission rate, and γ is the daily transition rate from infected to recovered. The recovered group consists of individuals who have either died or fully recovered.

The *basic reproduction number*, $\mathcal{R}_0$, is defined as $\mathcal{R}_0 \equiv \beta/\gamma$, and it gives the expected number of individuals infected by a single infective when everyone else is susceptible. The *effective reproduction number*, $\mathcal{R}_t$, is defined as $\mathcal{R}_t = \mathcal{R}_0 \times (S_{t-1}/N)$, and it equals the expected number of individuals infected by a single infective when a fraction (S_{t-1}/N) of individuals is susceptible.

From Eq. (1) the daily growth rate in the number of infected individuals is

$$\text{gr}(I_t) \equiv \frac{I_t - I_{t-1}}{I_{t-1}} = \gamma(\mathcal{R}_t - 1). \tag{2}$$

Denoting the estimated growth rate of infected individuals by $\hat{\text{gr}}(I_t)$, and given a value for the transition rate γ, the plug-in estimator for the effective reproduction number is

$$\hat{\mathcal{R}}_t = 1 + \frac{1}{\gamma}\hat{\text{gr}}(I_t). \tag{3}$$

For the estimator to be feasible, we need to (i) calibrate the transition rate from infectious to recovered, γ; and (ii) estimate the growth rate of I_t. There are two potential strategies for choosing γ. First, external medical evidence can be used, given that γ^{-1} is the average infectious period. Second, one can use information on the serial interval of the disease, given that the serial interval in the SIR model also equals γ^{-1} (see, e.g. Ma, 2020, pp. 133–134).

To estimate the growth rate of I_t empirically, we first construct a time series for I_t

[7] For other discrete-time epidemiological models, see Allen and Van Den Driessche (2008) or Stock (2020).

from data on new cases. The SIR model in Eq. (6) implies that

$$I_t = (1-\gamma)I_{t-1} + \text{new cases}_t. \tag{4}$$

We initialize I_t by $I_0 = C_0$ where C_0 is the number of total cases at some initial date, and then construct subsequent values of I_t recursively.[8] Finally, we use standard Kalman-filtering tools to smooth the observed growth rate of I_t by estimating a local-level model (see Section A.1 in the Appendix).

From Eq. (4), the growth rate $\text{gr}(I_t)$ is bounded below by $(-\gamma)$. Hence, for any estimator of $\text{gr}(I_t)$ that is some weighted average of the observed growth rates, the point estimate of $\mathcal{R}$ is automatically weakly positive. To ensure that lower confidence bounds are positive as well, we estimate the q-th quantile of $\mathcal{R}$ by $\max\{0, 1+\gamma^{-1}\hat{g}_q\}$, where $\hat{g}_q$ is an estimate of the q-th quantile of $\text{gr}(I_t)$. If an analyst wishes to avoid such ad-hoc truncation, non-linear filtering methods may be used (see, e.g., Creal, 2012).

2.2 Robustness to Misspecification and Data Problems

We have derived our estimator using the benchmark SIR model, assuming that the number of infected individuals is perfectly measured. A natural question is whether the estimator performs well when these assumptions are violated—as they are likely to be in practice.

In the Appendix, we show that the estimator remains exactly valid in the SIS model in which individual do not obtain immunity (Section A.2) and a generalized SIR model with time-varying parameters and stochastic shocks (Section A.3). In addition, we investigate the performance of the estimator when the disease follows the SEIR model using a Monte Carlo simulation (Section A.4). Provided that the average duration of infectiousness is correctly specified, we find that our estimator yields accurate results even when the true model is SEIR rather than SIR.

The simplicity of our estimator allows us to analytically characterize the effects of measurement error (Section A.5 in the Appendix). We use this characterization to investigate the performance of the estimator in a range of scenarios that are likely to be important in practice. The characterization can also be employed to obtain estimates of $\mathcal{R}$ that are adjusted for changes in testing practices. Overall, we conclude that the

[8] In the first version of this paper, we used data on new recoveries and deaths to construct a time series of infected individuals directly. However, for a disease such as COVID-19 with long average time-to-recovery, this alternative approach leads to estimates of $\mathcal{R}$ that are substantially delayed. In addition, data on new recoveries are often fairly noisy and/or exhibit lumpy reporting. Hence, we have opted for using only data on new cases.

method appears to provide reliable estimates in a number of situations with data problems that are of practical relevance.

3 Estimates for COVID-19

We now use data from the John Hopkins CSSE repository (Dong et al., 2020)[9] to obtain real-time estimates of $\mathcal{R}$ for COVID-19. For each country, we use data after the cumulative number of COVID-19 cases reaches 100. The estimation procedure is detailed in the Appendix (Section A.7).

For the baseline estimates, we assume that people are infectious for $\gamma^{-1} = 7$ days on average, similarly to Maier and Brockmann (2020) and Prem et al. (2020). This assumption is consistent with the evidence on the serial interval of COVID-19.[10] For example, Flaxman et al. (2020a) use an average serial interval of 6.5 days. The recent meta-analysis of Park et al. (2020) finds that estimates of the serial interval for COVID-19 generally range between 4 and 8 days. In addition, we document that $\gamma^{-1} = 7$ leads to estimates of the basic reproduction number ($\mathcal{R}_0$) that are in line with the current best estimates (Liu et al., 2020). However, we also investigate the effects of different choices for γ on our results.

In the Appendix (Section A.9), we perform two empirical validation exercises of our estimates. First, we document that our estimates of $\mathcal{R}$ are predictive of future deaths. Given that deaths are arguably more accurately measured, this finding helps assuage concerns that our estimates are contaminated by data problems with new cases. Second, we find that past mobility data (Google, 2020)[11] is predictive of future values of $\mathcal{R}$. Taken together, the two validation exercises suggest that the estimates contain valuable information on the dynamics of COVID-19.

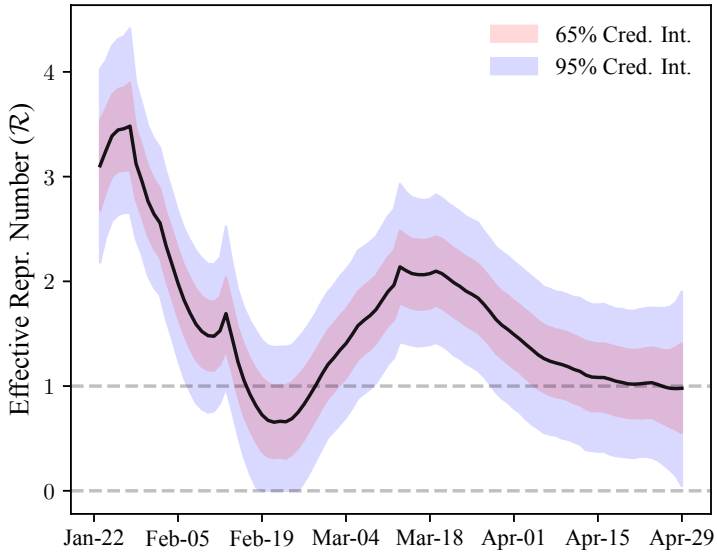
3.1 Main Results

Our estimates of $\mathcal{R}$ for COVID-19 for the world as a whole are provided in Figure 1. The graph highlights two distinct phases of the pandemic, first in China and later in Europe and the US. At the beginning of the sample in late January, the estimates of $\mathcal{R}$ are above 3; the estimates fall below one by February 19, consistent with a containment of the epidemic in China. Note that there is a moderate upwards jump in the estimated $\mathcal{R}$ around the second week of February. This jump was caused by a temporary change in

[9] The data is publicly accessible online (link).
[10] In the SIR model, the serial interval is also equal to γ^{-1} (see, e.g. Ma, 2020, pp. 133–134).
[11] The data is publicly accessible online (link).

Figure 1
$\mathcal{R}$ of COVID-19: Estimates for the World

Notes: Estimates of the effective reproduction rate ($\mathcal{R}$) of COVID-19 for the world as whole. The sample consists of all dates after the total number of reported cases worldwide has reached 100. 65% and 95% credible bounds shown by the shaded areas.



COVID-19 case definitions in the Hubei province in China; the new definition included clinically-diagnosed COVID-19 cases (Tsang et al., 2020). The estimates of $\mathcal{R}$ start increasing around February 19, coinciding with the spread of the pandemic to Europe and the US. Our estimates indicate that $\mathcal{R}$ has been trending down since mid-March globally. However, the rate of decline is substantially lower than what was observed in late January and early February. The current point estimate of $\mathcal{R}$ for the world as a whole is very close to one.

Figure 2 plots the estimated for $\mathcal{R}$ for China, Italy, and the US.[12] For all three countries, the estimated $\mathcal{R}$ is initially above 3. For China, the estimated $\mathcal{R}$ falls below one around the third week of February. However, the estimated $\mathcal{R}$ in China drifted up towards one during late March and early April, potentially caused by a wave of imported cases; the current point estimate for China is substantially below one. In Italy, the estimated $\mathcal{R}$ has been steadily falling since March but at a slower rate than previously observed in China, with the point estimate for Italy falling below one in early April. In the US, a striking difference is that the point estimates of $\mathcal{R}$ are, in fact, increasing in the first two weeks of the epidemic, rising from around 2.9 to almost 3.6. A likely explanation is that the fraction of non-detected cases went down substantially in this

[12] In the Appendix, we also provide a graph of the raw data for $\text{gr}(I_t)$ as well its smoothed version that is used for estimating $\mathcal{R}$ (Figure A.5).

Figure 2
$\mathcal{R}$ of COVID-19: Selected Countries

Notes: Estimates of the effective reproduction rate ($\mathcal{R}$) of COVID-19 for selected countries. The sample consists of all dates after the total number of reported cases in the country has reached 100. 65% credible bounds shown by the shaded areas.

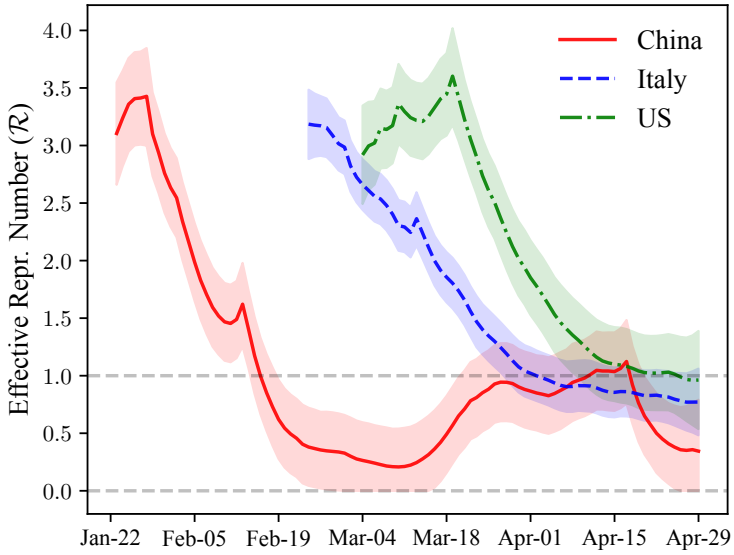


Table 1
Estimates of the Basic Reproduction Number ($\mathcal{R}_0$)

Notes: Estimates of the basic reproduction number ($\mathcal{R}_0$) for a sample of 14 European countries. The countries included in the sample are Austria, Belgium, Denmark, France, Germany, Greece, Italy, Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and United Kingdom. The basic reproduction number is calculated by averaging our estimates of the effective reproduction number in the first 7 days of the epidemic, where the start of the epidemic is defined as the day when the cumulative number of cases reaches 100.

Number of Days Infectious:	5	6	7	8	9	10
$\hat{\mathcal{R}}_0$	2.08	2.37	2.67	2.97	3.28	3.58
CI Lower Bound (95%)	1.47	1.70	1.92	2.14	2.38	2.60
CI Upper Bound (95%)	2.74	3.11	3.48	3.86	4.24	4.62

period, inflating the estimates of $\mathcal{R}$ upward (see Section A.5 in the Appendix).[13] The confidence bounds, however, are consistent with $\mathcal{R}$ in the US being stable over the first two weeks of the epidemic. The current point estimate of $\mathcal{R}$ in the US is very close to one.

[13] The number of tests conducted in the US went up substantially during this period; see, for example, the data provided by Our World in Data (2020).

3.2 Basic Reproduction Number

Our estimates can be used to measure the basic reproduction number ($\mathcal{R}_0$), i.e., the average number of individuals infected by a single infectious individual when the population is fully susceptible. We estimate $\mathcal{R}_0$ by the average value of $\mathcal{R}_t$ in the first week of the epidemic. The results for a sample of 14 European counries are shown in Table 1; the set of countries is the same as analyzed by Flaxman et al. (2020a).[14]

Under our baseline assumption that the individuals are on average infectious for 7 days ($\gamma = 1/7$), we obtain an estimate of $\mathcal{R}_0 = 2.67$ (95% CI: 1.92–3.48). For COVID-19, a recent meta-study has estimated a median $\mathcal{R}_0$ of 2.79 (Liu et al., 2020) which is very close to our baseline point estimate. As is to be expected, the estimate of $\mathcal{R}_0$ is quite sensitive to the choice of γ, with an additional day of infectiousness estimated to increase $\mathcal{R}_0$ by around 0.3.

3.3 Assessing Non-Pharmaceutical Interventions

Finally, we use our estimates to assess the effects of non-pharmaceutical interventions (NPIs) in a sample of 14 European countries studied by Flaxman et al. (2020a). The definitions of NPIs and their introduction dates are provided by Flaxman et al. (2020a).[15]

We first perform an event-study analysis. Figure 3 plots the estimated values of $\mathcal{R}$ around the introduction of a lockdown. $\mathcal{R}$ declines substantially after a lockdown is introduced, going from around 2.18 on the day of the intervention to around 1.32 two weeks later. However, $\mathcal{R}$ is decreasing before the lockdown as well. In particular, there is no visually detectible break in the slope of $\mathcal{R}$ after the lockdown (i.e., no "kink"). In the Appendix, we show that a similar pattern is observed for the remaining NPIs studied by Flaxman et al. (2020a). In particular, we document the behavior of $\mathcal{R}$ around the introduction of public-event bans (Figure A.7), case-based measures (such as self-isolation whenever feeling ill and experiencing fever; Figure A.8), school closures (Figure A.9), and social-distancing measures (Figure A.10). Except for school closures and public-event bans, there is no visually apparent break in the trend of $\mathcal{R}$ around the date of the policy intervention.

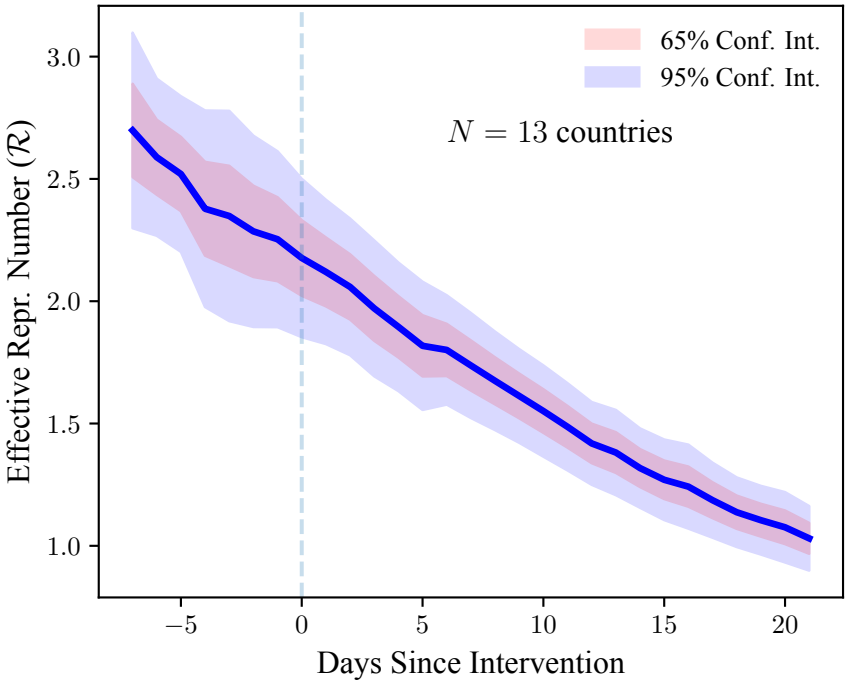
A potential concern with the evidence in Figure 3 is that our estimates of $\mathcal{R}$ use information from the full sample. Hence, estimates of $\mathcal{R}$ *after* the lockdown implicitly depend on the estimates of $\mathcal{R}$ *before* the lockdown. This feature of the estimation procedure may result in low statistical power to detect any effects of NPIs. To investigate this

[14] The updated results of that study are available online (link). See also Flaxman et al. (2020b). The countries included in the sample are Austria, Belgium, Denmark, France, Germany, Greece, Italy, Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, and United Kingdom.

[15] The data are publicly available in an online repository (link).

Figure 3
$\mathcal{R}$ and Policy Interventions: Lockdowns

Notes: The graph plots the estimated effective reproduction number ($\mathcal{R}$) one week before and three weeks after a lockdown is introduced in a country. The original sample consists of 14 European countries studied by Flaxman et al. (2020a). Heteroskedasticity-robust confidence bounds are shown by the shaded areas.



possibility, we conduct a power analysis (Section A.8 in the Appendix). Given our empirical estimates of signal-to-noise ratios, we find that the statistical procedure appears sufficiently powerful to detect moderate changes in $\mathcal{R}$.

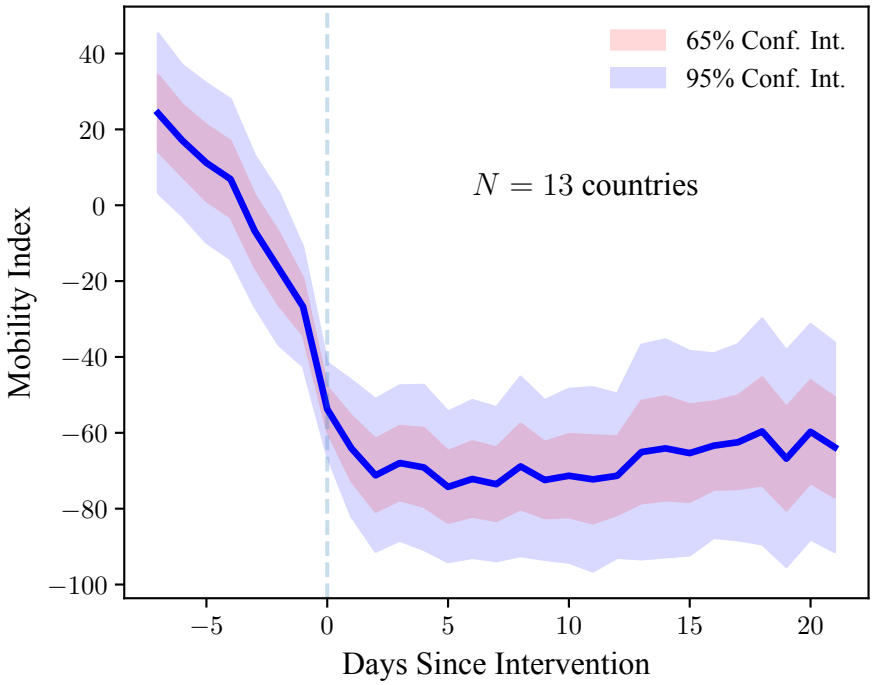
To investigate why the behavior of $\mathcal{R}$ appears to be unaffected by lockdowns, we use mobility data from Google's "COVID-19 Community Mobility Reports" (Google, 2020). The results are provided in Figure 4. The graph shows that essentially all of the decline in mobility occurs *before* the imposition of the lockdown. Mobility is low and appears to be very stable after the lockdown is introduced. The finding is consistent with the recent work of Farboodi, Jarosch, and Shimer (2020) who document the same pattern for the US using data from *SafeGraph*, as well as anecdotal reports from Sweden (e.g., The New York Times, 2020).

To assess the effects of NPIs more formally, we employ the following fixed-effect regressions (Table 2). Specifically, we regress $\mathcal{R}$ on a set of indicator variables capturing

Figure 4

Mobility Around Introduction of Lockdowns

Notes: The graph plots the mobility index (constructed from "COVID-19 Community Mobility Reports" of Google (2020)) one week before and three weeks after a lockdown is introduced in a country. See Section A.9 for details on the construction of the mobility index. The original sample consists of 14 European countries studied by Flaxman et al. (2020a). Heteroskedasticity-robust confidence bounds are shown by the shaded areas.



interventions and different types of fixed effects:

$$\log(R_{i,t}) = (\text{fixed effects}) + \sum_{j=1}^{5} \beta_i \text{NPI}_{i,t}^{(j)} + u_{i,t}.$$

Here, $\text{NPI}_{i,t}^{(j)}$ is an indicator variable that equals 1 after the j-th NPI is introduced, and zero before its introduction. The index i denotes countries, and t stands for the number of days since the outbreak of the epidemic.

Column (1) of Table 2 provides estimated effects of NPIs when only country fixed effects are included. We observe a strong negative effect of lockdowns, social distancing, and measures of self isolation. Taken at face value, the estimates suggest that lockdowns reduce $\mathcal{R}$ by almost 60%. Bans of public events and school closures are not statistically significant in this specification. These regressions (as well the point estimates) are fairly similar to the statistical analysis performed by Flaxman et al. (2020a).

The regression with country fixed effects only, however, is likely misspecified. Im-

Table 2

Effective Reproduction Number After Introduction of NPIs

Notes: Results of panel-data regressions of the (log of) effective reproduction number ($\mathcal{R}$) on indicator variables that are equal to 1 after the introduction of a non-pharmaceutical intervention (NPI) and 0 before the introduction. The sample consists of 14 European countries studied by Flaxman et al. (2020a). Regressions always include country fixed effects; regressions in columns (2)–(4) also include days-since-outbreak fixed effects. Outbreak is defined as the date on which 100 cases of COVID-19 are reached. The regression with mobility controls in (3) includes the one- and two-week lags of the mobility index, constructed from Google (2020); see Section A.9 for details. The regression with testing controls in (4) controls for the change in the number of daily tests per capita conducted in the country. To allow for reasonably precise estimation of days-since-outbreak fixed effects, we only consider days after the outbreak for which we have data for at least 5 countries. Heteroskedasticity-robust standard errors in parentheses.

	(1)	(2)	(3)	(4)
Lockdown	-0.57***	-0.09*	-0.05	-0.01
	(0.04)	(0.05)	(0.04)	(0.06)
Public Events	-0.06	0.22***	0.17***	0.32***
	(0.04)	(0.04)	(0.04)	(0.06)
School Closure	0.0	-0.15***	-0.1**	-0.06
	(0.06)	(0.05)	(0.04)	(0.05)
Self Isolation	-0.13**	-0.1**	-0.08**	-0.22***
	(0.06)	(0.04)	(0.04)	(0.04)
Social Distancing	-0.18***	-0.09*	-0.09*	0.05
	(0.06)	(0.05)	(0.05)	(0.05)
N	742	742	586	327
R^2	0.55	0.87	0.86	0.93
Country FE	✓	✓	✓	✓
Days-Since-Outbreak FE		✓	✓	✓
Mobility Controls			✓	✓
Testing Controls				✓

$^{*}p<0.1$; $^{**}p<0.05$; $^{***}p<0.01$

plicitly, such a specification assumes that the only reason why $\mathcal{R}$ can fall is because of introduction of NPIs. However, $\mathcal{R}$ would likely trend downwards even in the absence of any public policy interventions. First, $\mathcal{R}$ tends to fall during an epidemic as the number of susceptibles is depleted. Second, people may adjust their behavior even in the absence of any policy measures. Failing to control for the dynamics of $\mathcal{R}$ in the absence of NPIs therefore likely leads to an over-estimation of the effects of NPIs.

We acknowledge that obtaining credible counterfactuals in the present empirical context is extremely challenging. However, we can exploit the panel structure of the dataset to reduce the potential endogeneity problems in the previous specification. We do so by including days-since-outbreak fixed effects. Intuitively, with such fixed effects we are comparing $\mathcal{R}$'s in two countries (e.g., country A and country B) that are both five days from the outbreak (say), with a school closure in country A but not in country B.

The results from the regression with days-since-outbreak fixed effects are shown in column (2). The coefficient for lockdowns becomes substantially smaller in absolute value and only marginally statistically significant. The coefficients for self-isolation and social-distancing measures are also reduced and lose some of their statistical significance. The coefficient for public events is highly statistically significant but positive rather than negative. A naïve interpretation would suggest that banning public events has a positive effect on $\mathcal{R}$. More likely, however, is that the positive coefficient is due to countries where $\mathcal{R}$ is declining more slowly being faster to ban public events. In the Appendix (Table A.3), we show that the results remain similar when the NPIs are included separately, reducing concerns about potential multicollinearity problems between the different NPI variables.

In column (3), we also include lagged mobility variables as additional controls. With mobility controls, the coefficient on lockdowns loses statistical significance altogether. School closures, self isolation, and social-distancing measures are estimated to have a statistically-significant negative effect on $\mathcal{R}$, with each of these NPIs estimated to reduce $\mathcal{R}$ by around 10%.

A potential concern is that countries may introduce NPIs and simultaneously increase the number of tests for COVID-19 that they perform. To help alleviate this concern, in column (4) we add the change in the daily number of tests per capita as an additional explanatory variable.[16] While the sample size is reduced significantly as we do not have testing data for all countries in the sample, the results are largely unchanged.

We caution the readers against over-interpreting the results of this section. Obtaining good estimates of the true causal impact of NPIs is extremely challenging. As a result, even our best estimates still suffer from important potential endogeneity issues. In particular, the timing of NPIs is not random. Countries that introduced NPIs earlier likely did so because they had previously observed a stubbornly high $\mathcal{R}$. Finally, we find that people had largely adjusted their mobility patterns *before* lockdowns were introduced. We also show that mobility measures are highly correlated with $\mathcal{R}$ (see Section A.9). Hence, social distancing does appear to be effective at reducing $\mathcal{R}$.

[16] The data on daily tests per capita comes from *Our World in Data* (Our World in Data, 2020).

4 Conclusions and Limitations

In this paper we develop a new way to estimate the effective reproduction number of an infectious disease ($\mathcal{R}$). The new methodology is straightforward to apply in practice, and it appears to yield reliable results. We use the new method to track $\mathcal{R}$ of COVID-19 around the world, and assess the effectiveness of public policy interventions in a sample of European countries.

The current paper faces several limitations. First, a local-level specification for the growth rate implicitly assumes that the growth rate of the number of infected individuals remains forever in flux. However, in the long-run, this growth rate must converge to zero. Since our model does not capture this feature, it seems likely that our estimated confidence bounds are overly conservative in the late stages of an epidemic. Second, when applying the model to cross-country data, one may achieve important gains in statistical efficiency if the model is estimated jointly for all countries (for example, by estimating a multivariate local-level model). Finally, for assessing the effects of NPIs more accurately, it would be desirable to collect data for a larger sample of countries.

In our empirical application, we find fairly limited effects of lockdowns in the sample of European countries that we consider. The reason is likely that people had already changed their mobility behavior before lockdowns were introduced. Given that even our best estimates still suffer from potential endogeneity problems, it is important to interpret these results cautiously. However, from an economic perspective, these findings suggest that there may be no steep trade-off between public health and the economy. In other words, even if NPIs that are currently in place are lifted, it is not clear whether people would voluntarily return to their pre-pandemic mobility and consumption patterns. Our real-time estimator may be used to track the dynamics of COVID-19 as the current restrictions are relaxed.

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Appendix A Supplementary Methods and Materials

A.1 Local-Level Model

We specify a local-level model for the growth rate of the number of infected individuals:

$$\begin{aligned} \text{gr}(I_t) &= \mu_t + \varepsilon_t, \qquad \varepsilon_t \sim \text{i.i.d. } \mathcal{N}(0, \sigma_\varepsilon^2) \\ \mu_t &= \mu_{t-1} + \eta_t, \quad \eta_t \sim \text{i.i.d. } \mathcal{N}(0, \sigma_\eta^2) \end{aligned} \tag{5}$$

We estimate $\text{gr}(I_t)$ by the value for $\hat{\mu}_t$ given by the Kalman smoother (see Durbin and Koopman, 2012, Chapter 2). To estimate the unknown parameters (σ_ε^2 and σ_η^2), in principle either classical or Bayesian methods can be used. However, in practice, sample sizes for estimating $\text{gr}(I_t)$ are usually limited, especially early on in the epidemic. Hence, incorporating prior knowledge generally leads to better-behaved estimates. The amount of smoothing is informed by the data via the estimation of σ_ε^2 and σ_η^2. The local-level model can also be thought as a model-based version of exponentially-weighted moving-average smoothing (Muth, 1960).

The local-level model can be viewed as a reduced-form specification for $\text{gr}(I_t)$. The local-level model is sufficiently flexible to capture rich dynamic patterns in the data. In addition, in Section A.2 of the Appendix, we provide a theoretical rationale for the local-level specification. In particular, Eq. (5) arises naturally in a generalized SIR model (in the early stages of an epidemic) when the transmission rate follows a random walk.

A.2 SIS Model

We now show that the estimator in Eq. (3) also obtains when the dynamics of the disease follow the SIS model. The SIS model, again in discrete time, is given by

$$\begin{aligned} S_t &= S_{t-1} - \beta I_{t-1} \frac{S_{t-1}}{N} + \gamma I_{t-1} \\ I_t &= I_{t-1} + \beta I_{t-1} \frac{S_{t-1}}{N} - \gamma I_{t-1} \end{aligned}$$

The only difference from the SIR model in Eq. (1) is that formerly infected individuals do not obtain immunity after recovery and instead again join the pool of susceptibles. As is well known, the basic reproduction number $\mathcal{R}_0$ in the SIS model is the same as in the SIR model (e.g., Chowell and Brauer, 2009) and given by $\mathcal{R}_0 = \beta/\gamma$. Since the law of motion for I_t in the SIS model is the same as in the SIR model, we can repeat the same steps as in the benchmark analysis to arrive at Eq. (3).

A.3 Generalized SIR Model

In this section, we show that the estimator in Eq. (3) also obtains in a much more general version of the SIR model. Specifically, we consider the following generalized SIR model:

$$\begin{aligned} S_t &= S_{t-1} - \beta_t I_{t-1} \frac{S_{t-1}}{N} - v_{1,t} \\ I_t &= I_{t-1} + \beta_t I_{t-1} \frac{S_{t-1}}{N} - \gamma I_{t-1} + v_{1,t} - v_{2,t} \\ R_t &= R_{t-1} + \gamma I_{t-1} + v_{2,t} \end{aligned}$$

Differently from the baseline model in Eq. (1), we now allow β_t to vary arbitrarily over time. In addition, we introduce random shocks $v_{1,t}$ and $v_{2,t}$. The shocks are i.i.d., and the time-varying support of $v_{1,t}$ is $[0, S_{t-1} - \beta_t I_{t-1}/N]$, while the support of $v_{2,t}$ is $[0, I_{t-1} + \beta_t I_{t-1} S_{t-1}/N]$. We also assume that $\mathbb{E}_{t-1}[v_{1,t} - v_{2,t}] = 0$, so that the conditional expectation $\mathbb{E}_{t-1}[I_t]$ coincides with the value for I_t given by the noiseless SIR model. With these modifications, the model can capture rich patterns of infectious disease dynamics. For example, "super spreader events" can be modeled either as $v_{1,t}$ shocks or as a spike in β_t. The model can also capture richer forms of population structures than the baseline SIR model. For example, if individuals who are more infectious (e.g., those with more connections in a network model) are more likely to become infected first, that can be captured by assuming that β_t becomes lower over time.

Defining the (time-varying) basic reproduction number as $\mathcal{R}_0^{(t)} = \beta_t/\gamma$, and $\mathcal{R}_t \equiv \mathcal{R}_0^{(t)} S_{t-1}/N$, we obtain that

$$\text{gr}(I_t) = \gamma(\mathcal{R}_t - 1) + v_t,$$

where $v_t \equiv (v_{1,t} - v_{2,t})/I_{t-1}$. Taking expectations on both sides of the equation, we arrive at

$$\mathbb{E}[\mathcal{R}_t] = 1 + \frac{1}{\gamma}\,\mathbb{E}[\text{gr}(I_t)].$$

Hence, the generalized SIR model of the present section leads to the same estimator as the baseline SIR model in Eq. (1).

Finally, we note that if γ varies deterministically over time, the equation above remains essentially unchanged, the only difference being that γ is replaced by γ_t. If γ_t follows a non-degenerate stochastic process, then the estimator for $\mathbb{E}[\mathcal{R}_t]$ would need to correct for the covariance between γ_t and $\mathcal{R}_t$.

A.4 SEIR Model: Monte Carlo Simulation

Our estimation method uses a structural mapping between $\mathcal{R}_t$ and $\text{gr}(I_t)$ derived from the basic SIR model. While we can generalize the basic SIR model in several directions (Section A.3), and the estimator remains valid in an SIS model (Section A.2), the model is nevertheless restrictive. In particular, it ignores incubation periods as well as transmission during the incubation period. These features are likely especially important when modeling COVID-19.

We now perform a simulation exercise to see how our estimator of $\mathcal{R}_t$ performs in a richer model that accounts for these additional features. Specifically, we consider an SEIR model in which the exposed are infectious:

$$\begin{aligned} S_t &= S_{t-1} - \beta I_{t-1}\frac{S_{t-1}}{N} - \beta\epsilon E_{t-1}\frac{S_{t-1}}{N} \\ E_t &= E_{t-1} + \beta I_{t-1}\frac{S_{t-1}}{N} + \beta\epsilon E_{t-1}\frac{S_{t-1}}{N} - \kappa E_{t-1} \\ I_t &= I_{t-1} + \kappa E_{t-1} - \gamma I_{t-1} \\ R_t &= R_{t-1} + \gamma I_{t-1} \end{aligned} \tag{6}$$

Here, E_t denotes the number of individuals that are exposed at day t, κ is the daily transition rate from exposed to infected, and $\epsilon \in [0, 1]$ measures the degree to which the exposed are less infectious than the infected. If $\epsilon = 0$, the exposed are not infectious at all, and we obtain the benchmark SEIR model. If $\epsilon = 1$, the exposed are as infectious as the infected, and the model is isomorphic to the standard SIR model.

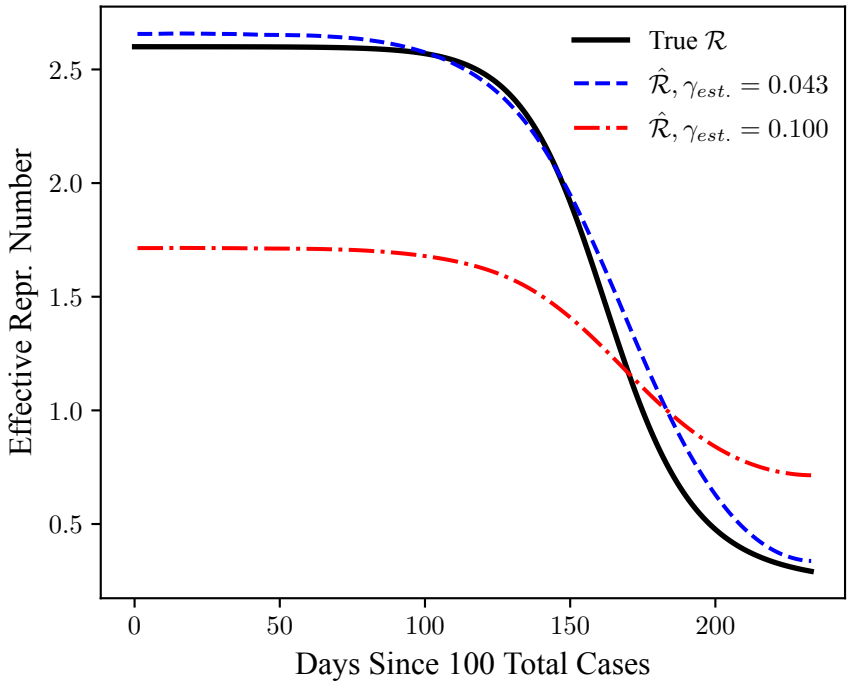
We calibrate the parameters following Wang et al. (2020) who apply the benchmark SEIR model (with $\epsilon = 0$) to study the dynamics of COVID-19 in Wuhan. In particular, we use $\kappa = 1/5.2$ and $\gamma = 1/18$ as in Wang et al. (2020). Then, we set $\epsilon = 2/3$, following Ferguson et al. (2020) who assume that symptomatic individuals are 50% more infectious than the asymptomatic (that is, $\epsilon^{-1} = 1.5$). Finally, we choose β by targeting a basic reproduction number of $\mathcal{R}_0 = 2.6$, again as in Wang et al. (2020). In the model above, $\mathcal{R}_0$ is given by $\mathcal{R}_0 = \beta/\gamma + \beta\epsilon/\kappa$, implying $\beta = \mathcal{R}_0\gamma\kappa/(\gamma\epsilon + \kappa)$. The formula yields $\beta \approx 0.12$. Finally, we set $S_0 = 11 \times 10^6$ (approximating the population size of Wuhan), $E_0 = R_0 = 0$, and $I_0 = 1$.

The Monte Carlo design is as follows. First, we simulate the deterministic system in Eq. (6) using the parameters above. Then, we calculate the growth rate in the true number of infected individuals, i.e., $\text{gr}(I_t) = I_t/I_{t-1} - 1$. However, instead of knowing the true growth rate, the statistician is assumed to observe a noisy version of it given by $\widetilde{\text{gr}}(I_t) = \text{gr}(I_t) + \varepsilon_t$. Here, ε is an i.i.d. normal disturbance with mean zero and standard deviation of 0.10. The standard deviation of the disturbances is roughly equal to the

Figure A.1

Monte Carlo Simulation: Effects of Misspecification

Notes: Estimates of the effective reproduction rate ($\mathcal{R}_t$) when the true dynamics of the disease follow an SEIR model. We investigate two values for $\gamma_{\text{est.}}$, the transition rate from infected to recovered, that are used when estimating $\mathcal{R}_t$. First, we use the correct value of $\gamma_t extest. = (\gamma^{-1}+\kappa^{-1})^{-1} \approx 0.043$. Second, we use a misspecified values of $\gamma_{\text{est.}} = 1/10$. Average values from 10,000 Monte Carlo replications are shown. See text for more details.



range of the true growth rates. Hence, the amount of noise used in the simulation is fairly large. For each realization of the disturbances, we estimate $\mathcal{R}_t$ using our method. As in our empirical application, only data after 100 total cases have been reached is used.

We investigate two values for $\gamma_{\text{est.}}$ that are used when estimating $\mathcal{R}_t$ via Eq. (3). First, we consider a situation in which the statistician uses the correct time that individuals are infected, given by $\gamma_{\text{est.}} = (\gamma^{-1} + \kappa^{-1})^{-1}$ where γ and κ are the true parameter values of the SEIR model. Second, we investigate a case in which the statistician incorrectly things that individuals are infectious only for ten days ($\gamma_{\text{est.}} = 1/10$). We repeat the process for 10,000 Monte Carlo replications.

The results of the Monte Carlo simulation are shown in Figure A.1. When the statistician uses the correct number of days that an individual is infectious (that is, taking into account the incubation time), the estimates of $\mathcal{R}_t$ from our method are very close to their true theoretical values. That is in spite of the fact that our estimator for $\mathcal{R}_t$ is derived

assuming that the dynamics of the disease are described by an SIR model. However, we also show that if the statistician misspecifies the number of days than an individual is infectious (assuming 10 days instead of the true number of 23.2 days), the estimates of $\mathcal{R}_t$ are substantially biased, especially in the early stages of the epidemic. As is to be expected from Eq. (3), underestimating the number of days that an individual is infectious leads to a downwards bias in the estimates of $\mathcal{R}_t$ early on in the epidemic (when $\mathcal{R}_t > 1$), and upwards bias when the true $\mathcal{R}_t$ falls below one. Overall, the results imply that the new method performs well when estimating $\mathcal{R}_t$ even when the true dynamics of the disease do not follow the SIR model, provided that the duration of infectiousness used in the estimation is sufficiently accurate.

A.5 Effects of Potential Data Issues

We now discuss the effects of various data issues on the performance of our estimator.

Reporting delays. In practice, data may be subject to significant reporting delays. For example, suppose that due to testing constraints there is a lag of ℓ days between the date that an individual becomes infected and the date on which the case is registered. In this case, the estimates of $\mathcal{R}$ would also be subject to delay of ℓ days. If there are significant reporting delays, one may first obtain, say, one-week-ahead forecasts of new cases, and then use these forecasts to construct a time series for I_t.

Imperfect detection. A natural worry with any estimator of $\mathcal{R}$ is that it may be substantially biased if not all of infected individuals are detected. Given the simplicity of our estimator, we can analytically assess the effects of imperfect detection.

Suppose that the true numbers of susceptible, infected, and recovered individuals are given by S_t^*, I_t^*, and $\mathcal{R}_t$, respectively. Their evolution is the same as in Eq. (1). However, we only observe $I_t = \alpha_t I_t^*$, where $\alpha_t \equiv I_t/I_t^*$ is the *detection rate*. In practice, α is typically less than one, although the mathematical calculation below does not require this.

With this notation, we have that

$$\text{gr}(I_t) = \text{gr}(\alpha_t)[1 + \text{gr}(I_t^*)] + \text{gr}(I_t^*) \approx \text{gr}(\alpha_t) + \text{gr}(I_t^*),$$

since $\text{gr}(\alpha_t) \times \text{gr}(I_t^*) \approx 0$ at a daily frequency; the approximation is exact in continuous time. Using the approximation above and Eq. (2), we therefore obtain that the bias of the estimator under imperfect detection is given by

$$\hat{\mathcal{R}}_t - \mathcal{R}_t \approx \frac{1}{\gamma}\text{gr}(\alpha_t).$$

We now discuss several cases of practical importance:

- Constant detection rate ($\alpha_t = \alpha$). If the detection rate is constant over time, then our estimator is unbiased, and $\hat{\mathcal{R}}_t = \mathcal{R}_t$. Hence, for example, even if we only detect 10% of the infectives (but the fraction detected remains constant over time), the estimator remains unbiased. Note that if the number of tests increases over time, that is *not* inconsistent with $\alpha_t = \alpha$ given that the number of infected individuals is likely to be growing at the same time.
- Constant growth in the detection rate ($\text{gr}(\alpha_t) = g_\alpha$). If the growth rate of α_t is constant over time, then our estimate of $\mathcal{R}_t$ is biased upwards if $g_\alpha > 0$ and downwards if $g_\alpha < 0$. Note, however, that we are often mostly interested in the *trend* of $\mathcal{R}$ over time and whether the trend is affected by various policy interventions. The trend in $\mathcal{R}$ is estimated accurately even if $g_\alpha \neq 0$. Intuitively, constant growth in the detection rate leads to a level bias, but the slope is still estimated correctly.
- Detection rate converges over time ($\alpha_t \to \alpha$). The final case of interest occurs when the detection rate converges to a constant over time. For example, if everyone is detected towards the end of the epidemic, we would have $\alpha_t \to 1$. Since our method uses Kalman-filtering techniques to estimate the growth rate of I_t, transient fluctuations in α_t would have a limited effect on the estimates of $\mathcal{R}$ later on in the sample. Given that we are often precisely interested in the behavior of $\mathcal{R}$ in the later stages of the epidemic (when the detection rate is likely fairly constant), our method would still yield reliable estimates.

Imported cases. Our estimates may be biased if the fraction of cases that is imported changes over time (the previous results on imperfect detection apply to misclassification because of imported cases, too). If the source of infections is known, it is possible to correct for the issue by simply not including imported cases when constructing the time series for I_t.

A.6 Foundation for the Local-Level Model

When estimating $\mathcal{R}$, we use a local-level specification for the growth rate of the number of infectives. In this section, we show that the local-level model arises naturally in an SIR model in the early stages of an epidemic when the transmission rate follows a random walk.

Specifically, consider the generalized SIR model of Section A.3. We now specialize

Table A.1
Priors

Notes: Priors used in the Bayesian estimation of $\mathcal{R}$. See text for description on how the priors for the precision of the irregular component ($1/\sigma_\varepsilon^2$) and the signal-to-noise ratio ($q \equiv \sigma_\eta^2/\sigma_\varepsilon^2$) are calibrated based on cross-country frequentist estimates.

Parameter	Prior
Precision of irregular component ($1/\sigma_\varepsilon^2$)	Gamma$(0.17725, 0.00111)$
Signal-to-noise ratio ($q \equiv \sigma_\eta^2/\sigma_\varepsilon^2$)	Gamma$(0.06886, 0.50411)$
Initial value (μ_0)	$\mathcal{N}(0.35, 0.5^2)$

the process for the transmission rate β_t to be a random walk:

$$\beta_t = \beta_{t-1} + \eta_t, \eta_t \sim \text{i.i.d. } \mathcal{N}(0, \sigma_\eta^2),$$

with a given initial value $\beta_0 > 0$. Using the law of motion for β_t, we calculate that

$$\text{gr}(I_t) = \left(\frac{S_{t-1}}{N}\right)\beta_t - \gamma + v_t \approx \beta_t - \gamma + v_t$$

in the early stages of the epidemic when $S_t \approx N$. Now define $\tilde{\beta}_t$ recursively by $\tilde{\beta}_t = \tilde{\beta}_{t-1} + \eta_t$ with the initial condition $\tilde{\beta}_0 = \beta_0 - \gamma$. Then, the growth rate of I_t follows a local-level model with

$$\begin{aligned} \text{gr}(I_t) &= \tilde{\beta}_t + v_t \\ \tilde{\beta}_t &= \tilde{\beta}_{t-1} + \eta_t \end{aligned}$$

Provided that the distribution of v_t can be approximated with a normal distribution, we directly obtain the specification in Eq. (5). Alternatively, to obtain an exact normal local-level model, we could assume that $v_{1,t} = v_{2,t} = 0$ (no shocks in the original model, just as in Eq. (1)) but that instead of observing the true growth rate $\text{gr}(I_t)$, we only observe $\text{gr}(I_t) + \varepsilon_t$ where ε_t is i.i.d. normally distributed mean-zero measurement error.

A.7 Estimation Details

To estimate $\mathcal{R}$ of COVID-19, we use Bayesian filtering methods. We employ the following strategy to calibrate the prior distributions. First, we estimate a local-level model for $\text{gr}(I_t)$ using a frequentist Kalman filter with diffuse initial conditions. The procedure yields maximum likelihood estimates of σ_ε^2 (variance of the irregular component) and

Table A.2
Power Analysis: Parameter Values

Notes: Parameter values used in the power analysis. The parameters values for q (signal-to-noise ratio) and σ_ε^2 (variance of the irregular component) are given by the median estimates from the 14 countries considered in the empirical analysis of Section 3.3. The mean duration of infectiousness is assumed to be $\gamma^{-1} = 7$, and the Kalman gain ω is calculated from Eq. (7).

γ	q	σ_ε^2	ω
0.143	0.051	0.022	0.201

the signal-to-noise ratio $q \equiv \sigma_\eta^2/\sigma_\varepsilon^2$ for each country in the sample (with σ_η^2 denoting the variance of the level component). We then use the distribution of $\hat{\sigma}_\varepsilon^2$ and $\hat{q}$ across countries to calibrate the priors for the precision of the irregular component ($1/\sigma_\varepsilon^2$) and the signal-to-noise ratio (q). To ensure that the priors are not too "dogmatic," we inflate the variance of the estimates by a factor of 3 when calibrating the prior distributions. We use a gamma prior for both the signal-to-noise ratio and the precision of the irregular component, and we calibrate the parameters of the gamma distribution by matching the expected value and variance of the gamma-distributed random variables to their sample counterparts. Finally, we use a fairly uninformative normal prior for the initial value of the smoothed growth rate. The resulting priors are given in Table A.1.

Intuitively, these priors shrink the estimates of the precision and signal-to-noise ratio for each country towards their grand mean (average across countries). Such Bayesian shrinkage ensures that the parameter estimates are well behaved even though the sample size for many countries is fairly small, and the data are often noisy. We use the Stan programming language (Gelman, Lee, and Guo, 2015) to specify and estimate the Bayesian model.[17]

A.8 Power Analysis

In this section, we study the statistical power of the empirical analysis in Section 3.3 using a Monte Carlo simulation.

We now describe the design of the power study. Intuitively, we simulate data using a stochastic process that is calibrated to match the properties of the observed data. We then simulate a sharp drop in the effective reproduction number—say, because of a lockdown. We apply our estimator to the simulated data and ask how often this abrupt change is detected by the estimation procedure.

Optimal nowcasts from the local-level model in the steady state can be written as

[17] In particular, we use the `pystan` interface to call Stan from Python (link).

(Muth, 1960; Shephard, 2015, Section 3.4):

$$m_t = \omega y_t + (1-\omega) m_{t-1}, \omega = \frac{q + \sqrt{q^2+4q}}{2+q+\sqrt{q^2+4q}}, \tag{7}$$

where we denote $y_t \equiv \text{gr}(I_t)$, $m_t \equiv \mathbb{E}_t[\mu_t]$, $q \equiv \sigma_\eta^2/\sigma_\varepsilon^2$ is the signal-to-noise ratio, and ω is the steady-state Kalman gain. Hence, nowcast errors, $m_t - \mu_t$, follow an AR(1) process with

$$m_t - \mu_t = (1-\omega)(m_{t-1} - \mu_{t-1}) + \{\omega\varepsilon_t - (1-\omega)\eta_t\}.$$

Given that the shocks ε_t and η_t are uncorrelated, the variance of nowcast errors is

$$\mathbb{V}\text{ar}(m_t - \mu_t) = \frac{\omega^2\sigma_\varepsilon^2 + (1-\omega)^2 q\sigma_\varepsilon^2}{1-(1-\omega)^2}. \tag{8}$$

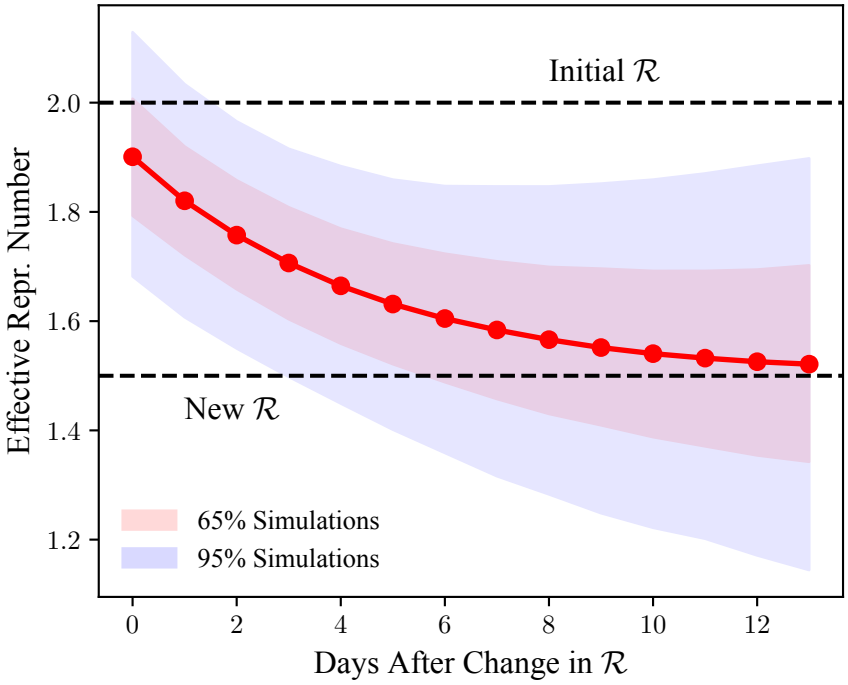
The design of the power analysis is as follows:

1. We set $\gamma = 1/7$, and calibrate the remaining parameters of the data-generating process (q and σ_ε^2) using the median values of the empirical estimates from Section 3.3. The resulting parameter values are given in Table A.2.
2. We simulate the true growth rate of the number of infected individuals, μ_t. We initially set the true growth rate of the number of infected individuals to $\mu_0 = 1/7$, implying an effective reproduction number of 2. At time 1, we simulate an abrupt decline in $\mathcal{R}_t$ by setting $\mu_1 = 1/14$, yielding a new effective reproduction number of 1.5, or a decline of 25%. For $2 \leq t \leq 14$, we simulate μ_t as a random walk, as in Eq. (5).
3. We simulate the observed growth rate of the number of infected individuals, $y_t = \text{gr}(I_t)$, as $y_t = \mu_t + \varepsilon_t$ where ε_t is an i.i.d. normal random variable with mean zero and variance σ_ε^2.
4. We simulate the nowcasts m_t of the growth rate of the number of infected individuals. We draw the initial nowcast m_0 from a normal distribution with mean $1/7$ and variance given in Eq. (8), and simulate further values of m_t by the recursion in Eq. (7). The estimated value of $\mathcal{R}$ is then given by the estimator in Eq. (3).
5. We repeat steps 2–4 for 14 times, to simulate data for 14 "countries", as in the empirical application, and obtain estimates of the effective reproduction number by averaging across the 14 "countries".
6. We repeat steps 2–5 for 10,000 Monte Carlo replications.

The results of the power analysis are shown in Figure A.2. We observe that in 95%

Figure A.2
Power Analysis: Monte Carlo Results

Notes: Power study of the statistical analysis of Section 3.3 (effects of non-pharmaceutical interventions on $\mathcal{R}$, the effective reproduction rate). We simulate an abrupt change in $\mathcal{R}$ from 2.0 to 1.5 using a data-generating process that is calibrated to match our empirical estimates in Section 3.3. We then apply the estimator of Eq. (3) to the simulated data and ask how often the change is detected by the estimation procedure. The solid line gives the average estimate of $\mathcal{R}$, while the shaded lines denote 65% and and 95% of simulations (in particular, the shaded area for 65% of simulations is given by the 17.5 and 82.5 percentiles of the estimated $\mathcal{R}$ across simulations, and the shaded area for 95% of simulations is given by the 2.5 and 97.5 percentiles of the estimated $\mathcal{R}$'s). 10,000 Monte Carlo replications used.

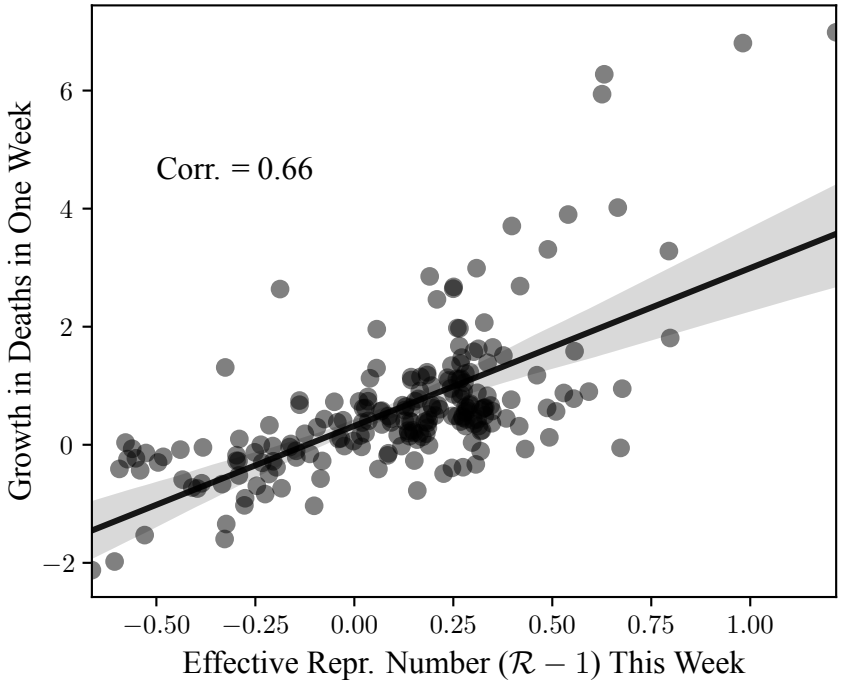


of the simulations, the change in $\mathcal{R}$ is detected as soon as two days after the drop in $\mathcal{R}$. Hence, the analysis in Section 3.3 appears sufficiently powerful to detect moderate changes in $\mathcal{R}$. The key reason why the analysis has high statistical power, even though the signal-to-noise ratio is quite low (see Table A.2) is that data from multiple countries are used to obtain cross-country averages. This feature of the estimation procedure reduces estimation error substantially. While the signal-to-noise ratio is fairly low, we also note that the weight placed on data is that are more than one-week old is only $(1-\omega)^7 \approx 20.8\%$. Hence, one week after the change in $\mathcal{R}$, the estimates of $\mathcal{R}$ are based primarily on data received after the change in $\mathcal{R}$.

The power analysis in the current section is arguably somewhat conservative. Specifically, we assume that after the abrupt decline, $\mathcal{R}$ follows a random walk rather than staying fixed at the new level. As a result, as time goes on, the estimates of $\mathcal{R}$ become

Figure A.3
$\mathcal{R}$ and Future Deaths

Notes: Relationship between current estimates of the effective reproduction number ($\mathcal{R}$) and the growth rate of the number of new deaths in one week. The data is aggregated to a weekly frequency. Both variables are residualized to subtract country fixed effects by performing the within transformation. Only data after the cumulative number of deaths reaches 50 is included in the scatter plot. We include all countries in the John Hopkins database for which we have at least 20 observations after the outbreak. We remove data for the week of 2020-04-13–2020-04-19 in China that contain a large number of deaths that were previously unrecognized.



more "spread out" across simulations, as is visible towards the end of Figure A.2.

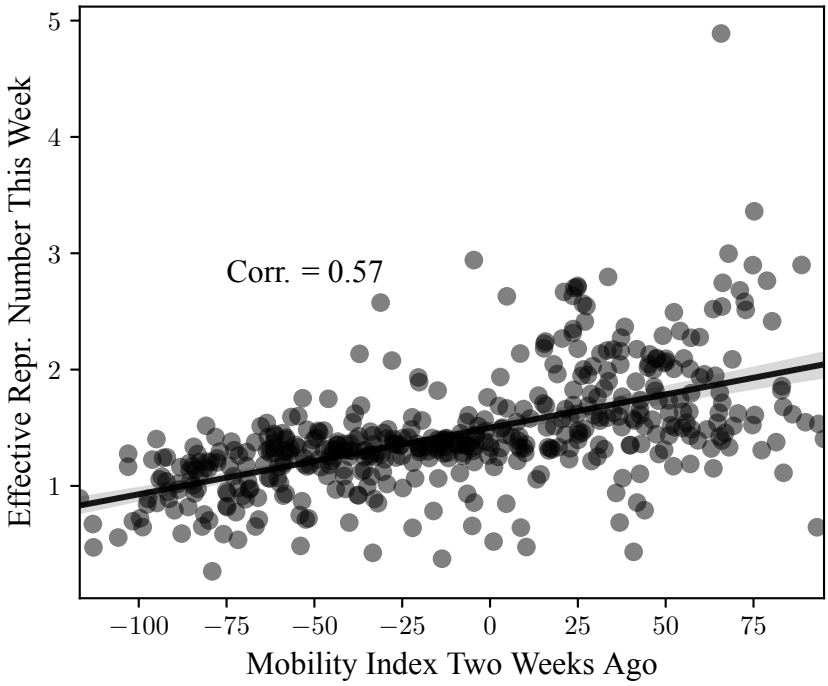
A.9 Empirical Validation

In this section, we perform two empirical validation exercises to check the performance of our estimates in practice.

Since our estimates are based on data on new cases, they may be misleading if new cases are subject to significant measurement problems. To help assuage this concern, we now perform the following exercise. We ask whether *current* values of $\mathcal{R}$ help predict *future* growth in deaths. Since deaths are likely to be measured more accurately, this exercise provides a test of whether our estimates contain meaningful information and are not contaminated by data problems.

Figure A.4
$\mathcal{R}$ and Past Mobility

Notes: Relationship between current estimates of the effective reproduction number ($\mathcal{R}$) and value of the movement index two weeks ago (first principal component of the six movement categories in Google (2020)). The data is aggregated to a weekly frequency. Both variables are residualized to subtract country fixed effects by performing the within transformation. We include all countries in the John Hopkins database for which we have at least 20 observations after the outbreak.



Formally, we consider the following regression:

$$\text{gr}(d_{i,t+1}) = \alpha_i + \beta(\hat{\mathcal{R}}_{i,t} - 1) + u_{i,t},$$

where i denotes a particular country, and t indexes calendar weeks. Although our original data is daily, we aggregate to a weekly frequency; otherwise, measures of the growth rate of new deaths are too noisy. In addition, we only include weeks after the cumulative number of COVID-19 deaths has reached 50. Given that we have panel data, we can include country fixed effects α_i to account for time-invariant unobserved heterogeneity (such as differences in average age—a key correlate of COVID-19 mortality (Verity et al., 2020)—or family structures). The relationship given above is predicted by the baseline SIR model.[18]

[18] Specifically, consider Eq. (2). Letting $\text{CFR} = d_t/I_{t-\ell}$ denote the case fatality rate (assumed to be constant over time), with ℓ standing for the average time between becoming infected and death, we have that $\text{gr}(d_t) = \text{gr}(I_{t-\ell})$, yielding the regression equation above.

The relationship is shown in Figure A.3. In the scatter plot, both variables are residualized to remove country fixed effects. We observe a strong positive relationship between the value of $\mathcal{R}$ this week and the growth in deaths one week later (corr. = 0.66). In Supplementary Figure (Figure A.6), we demonstrate that there is also positive correlation (corr. = 0.40) between $\mathcal{R}$ and deaths two weeks later. We note that while the average medical duration from the onset of symptoms to death for COVID-19 is longer than two weeks (around 18 days, see Verity et al., 2020), the duration from *reported* cases to deaths is likely to substantially shorter because of reporting delays. For example, Hortaçsu, Liu, and Schwieg (2020) assume that new cases of COVID-19 are reported with a lag of 8 days in their baseline calculations (5 days for symptoms to appear, consistent with the evidence from Lauer et al. (2020) and Park et al. (2020), as people are unlikely to be tested without exhibiting symptoms, and an additional 3 days to capture delays in obtaining test results, based on andecdotal reports from the US). Since deaths are likely reported in a timely manner, if new cases are reported with a lag of 8 days, we would expect an average duration of around 10 days ($\approx$1.43 weeks) between reported cases and reported deaths.

As a second validation check, we ask whether our estimates of $\mathcal{R}$ are correlated with past movement data, as it should be if the estimates are meaningful. For information on movement, we use aggregated smartphone location data collected by Google and published in their "COVID-19 Community Mobility Reports" (Google, 2020). Google provides data on percentage changes in movement for six types of places: (i) groceries and pharmacies; (ii) parks; (iii) transit stations; (iv) retail and recreation; (v) residential; and (vi) workplaces. Since the six categories are strongly correlated, we take the first principal component of the six categories (the first principal component explains around 85% of the total variance in the data). We refer to the first principal component as the "Mobility Index." As shown in Figure A.4, current estimates of $\mathcal{R}$ are strongly correlated with the value of the mobility index two weeks ago (corr. = 0.57).

For both validation exercises performed in the present section, we include all countries for which we have at least 20 observations after the onset of the epidemic (100 cumulative cases of COVID-19 reached). If we narrow the sample down to countries with more and higher-quality data—such as the sample of European countries analyzed in Section 3.3—the correlations generally become substantially stronger. Hence, we consider the tests of the present section to be conservative.

Appendix B Supplementary Figures and Tables

Figure A.5
Growth Rate of the Number of Infected Individuals

Notes: Raw data for the growth rate of the number of infected individuals (solid lines) and our estimate of its time-varying average (dashed lines) for China, Italy, and the US.

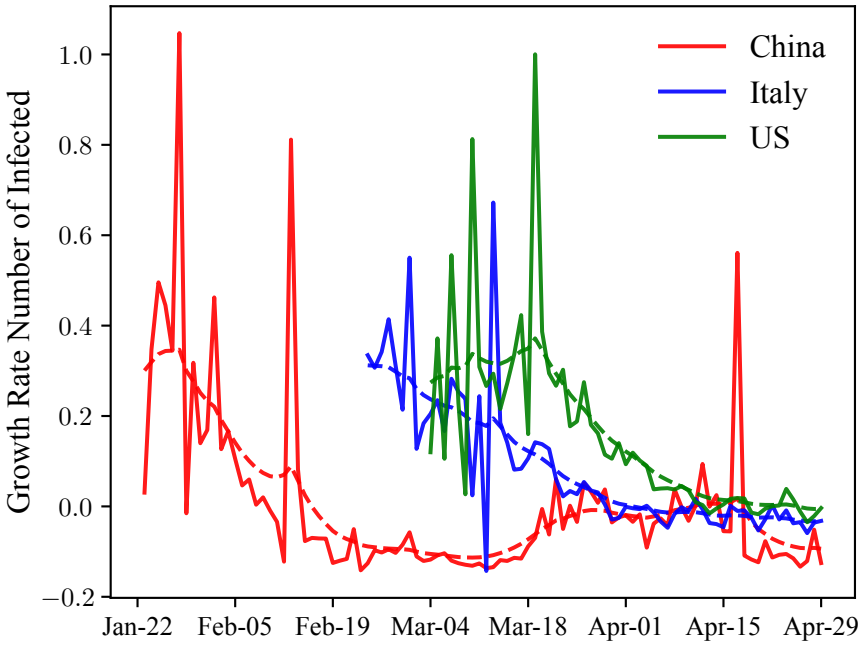


Figure A.6

$\mathcal{R}$ and Deaths in Two Weeks

Notes: Relationship between current estimates of the effective reproduction number ($\mathcal{R}$) and the growth rate of the number of new deaths in two weeks. The data is aggregated to a weekly frequency. Both variables are residualized to subtract country fixed effects by performing the within transformation. See Figure A.3 for more details.

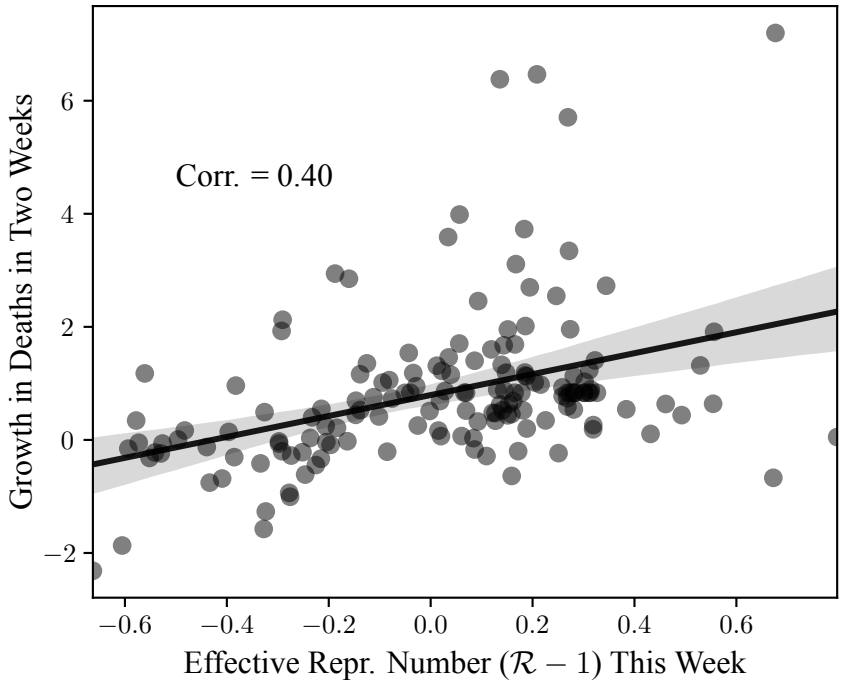


Figure A.7

$\mathcal{R}$ and Policy Interventions: Bans of Public Events

Notes: The graph plots the estimated effective reproduction number ($\mathcal{R}$) one week before and three weeks after public events are banned in a country. The original sample consists of 14 European countries studied by Flaxman et al. (2020a). For the event-study graph, we restrict the sample to countries for which data on $\mathcal{R}$ is available for the whole event window. Heteroskedasticity-robust confidence bounds are shown by the shaded areas.

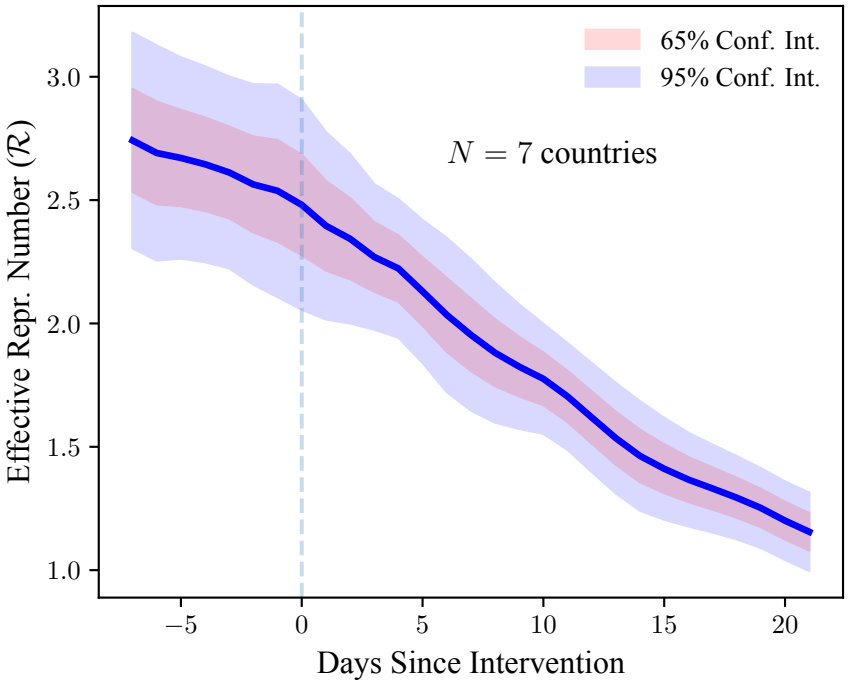


Figure A.8
$\mathcal{R}$ and Policy Interventions: Case-Based Measures

Notes: The graph plots the estimated effective reproduction number ($\mathcal{R}$) one week before and three weeks after case-based measures are introduced in a country. The original sample consists of 14 European countries studied by Flaxman et al. (2020a). For the event-study graph, we restrict the sample to countries for which data on $\mathcal{R}$ is available for the whole event window. Heteroskedasticity-robust confidence bounds are shown by the shaded areas.

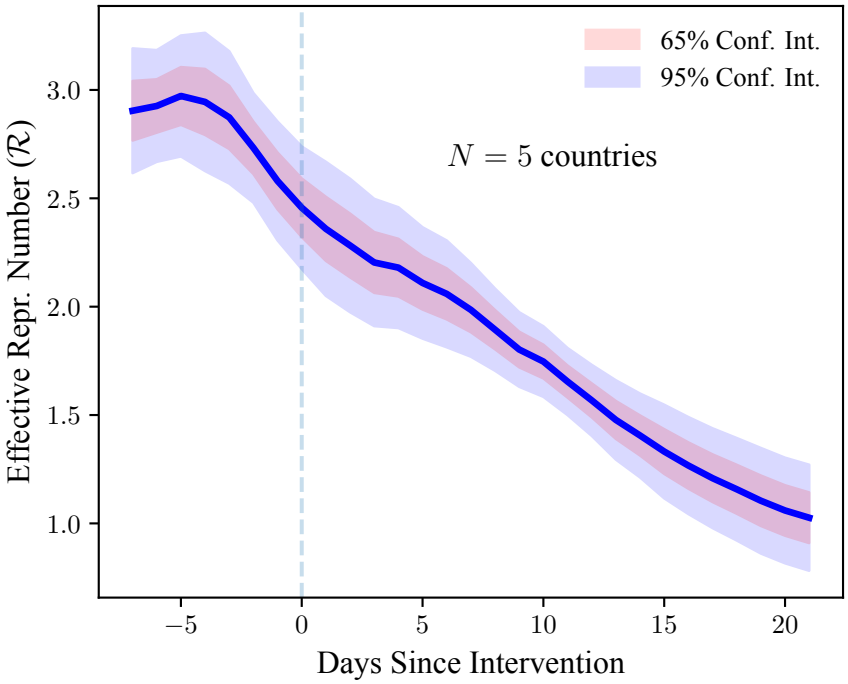


Figure A.9

$\mathcal{R}$ and Policy Interventions: School Closures

Notes: The graph plots the estimated effective reproduction number ($\mathcal{R}$) one week before and three weeks after school closures are ordered in a country. The original sample consists of 14 European countries studied by Flaxman et al. (2020a). For the event-study graph, we restrict the sample to countries for which data on $\mathcal{R}$ is available for the whole event window. Heteroskedasticity-robust confidence bounds are shown by the shaded areas.

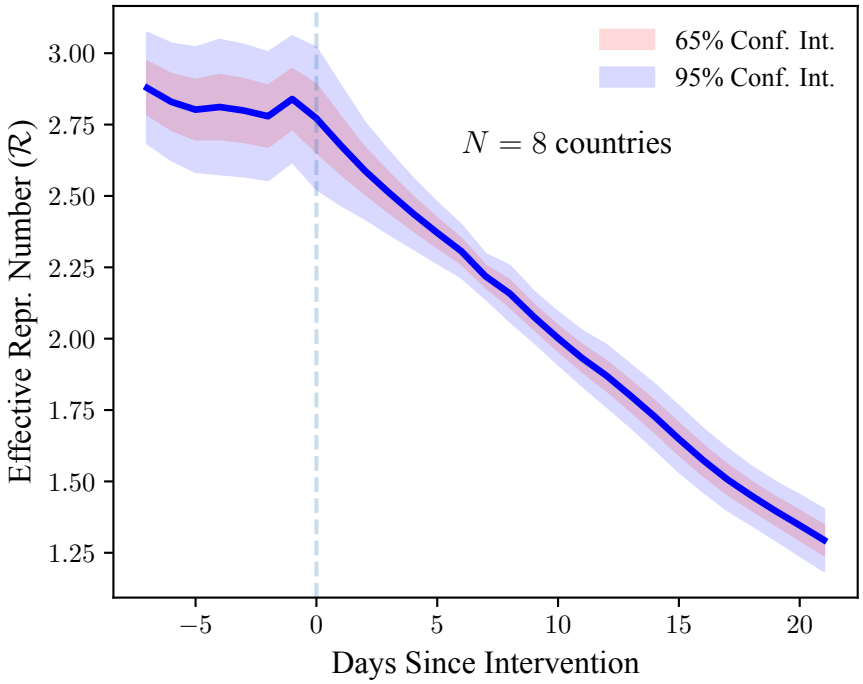


Figure A.10

$\mathcal{R}$ and Policy Interventions: Social Distancing

Notes: The graph plots the estimated effective reproduction number ($\mathcal{R}$) one week before and three weeks after social distancing is encouraged in a country. The original sample consists of 14 European countries studied by Flaxman et al. (2020a). For the event-study graph, we restrict the sample to countries for which data on $\mathcal{R}$ is available for the whole event window. Heteroskedasticity-robust confidence bounds are shown by the shaded areas.

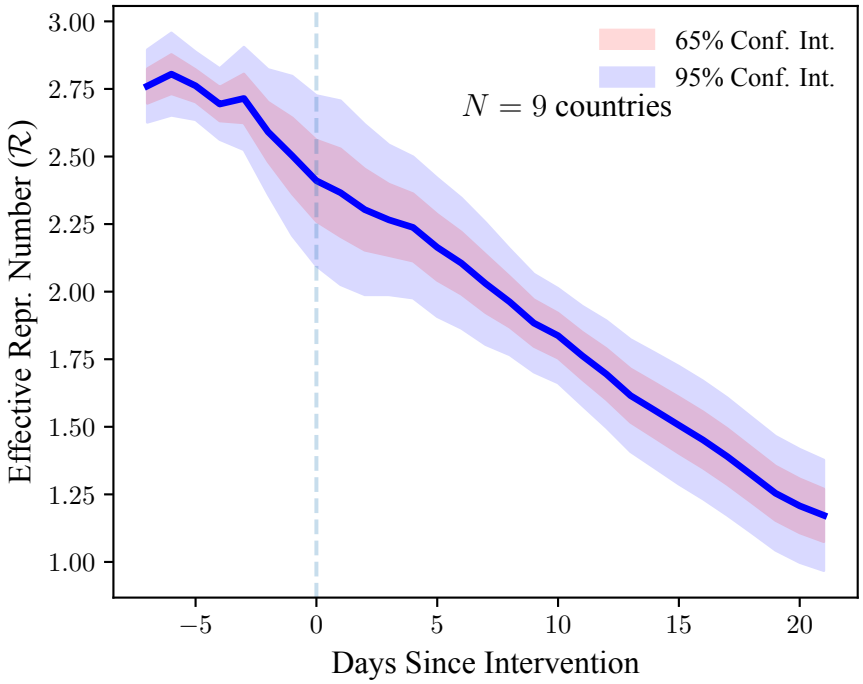


Table A.3

Effective Reproduction Number and NPIs

Notes: Results of panel-data regressions of the (log of) effective reproduction number ($\mathcal{R}$) on indicator variables that are equal to 1 after the introduction of a non-pharmaceutical intervention (NPI) and 0 before the introduction. The regressions are similar to those in Table 2 except that the intervention variables are included separately (one-at-a-time). See Table 2 for more details.

	(1)	(2)	(3)	(4)	(5)
Lockdown	-0.09**				
	(0.04)				
Public Events		0.09**			
		(0.04)			
School Closure			-0.12**		
			(0.05)		
Self Isolation				-0.14***	
				(0.05)	
Social Distancing					-0.12**
					(0.06)
N	742	742	742	742	742
R^2	0.86	0.86	0.86	0.86	0.86
Country FE	✓	✓	✓	✓	✓
Days-Since-Outbreak FE	✓	✓	✓	✓	✓
Mobility Controls					
Testing Controls					

$^{*}p<0.1$; $^{**}p<0.05$; $^{***}p<0.01$