

**Understanding the  
effectiveness of government  
interventions in Europe's  
second wave of COVID-19**

# **Abstract**

# Aim

Update estimates of the relative effectiveness of NPIs based on data from Europe's second wave (a joint collaboration between the Brauner and Flaxman teams).

# Methods

- Collect a large dataset of NPI implementations (114 subnational areas in 7 countries)
- Estimate the effectiveness of 17 NPIs from case and deaths data using a hierarchical regression model with the renewal equation and convolutions from infection to report as novel link functions.
- Address limitations in modeling from previous studies

# Results

- Similar to previous findings.
- Targeted closure of business worked well.
- Reduced impact from school closures

# Summary

- A well conducted study that draws on a large team to conduct novel work in a robust framework.
- Major improvement is in the quality of the data rather than the method.
- The nature of the observational data is highly complex and many of these issues may have been insufficiently dealt with.

# Introduction

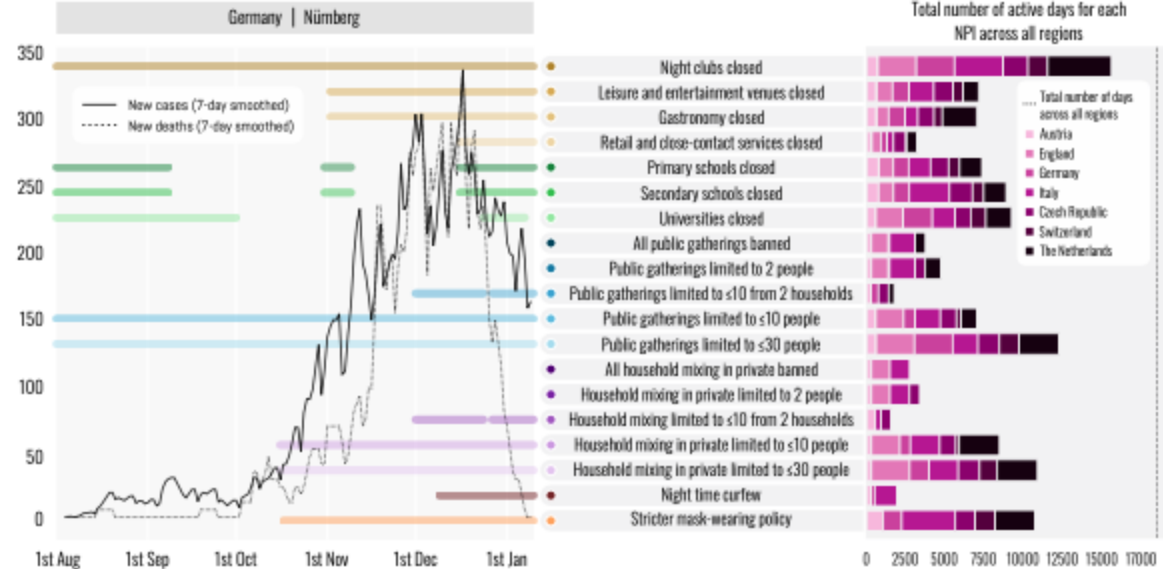
# Previous work

- Earlier studies used data based on NPI introductions in a very short time window in March 2020.
- Nearly all countries and regions imposed the same NPIs in the same order.
- Numerous data quality issues and varying testing/reporting regimes in the first wave.
- Lack of subnational data may have induced ecological fallacies.

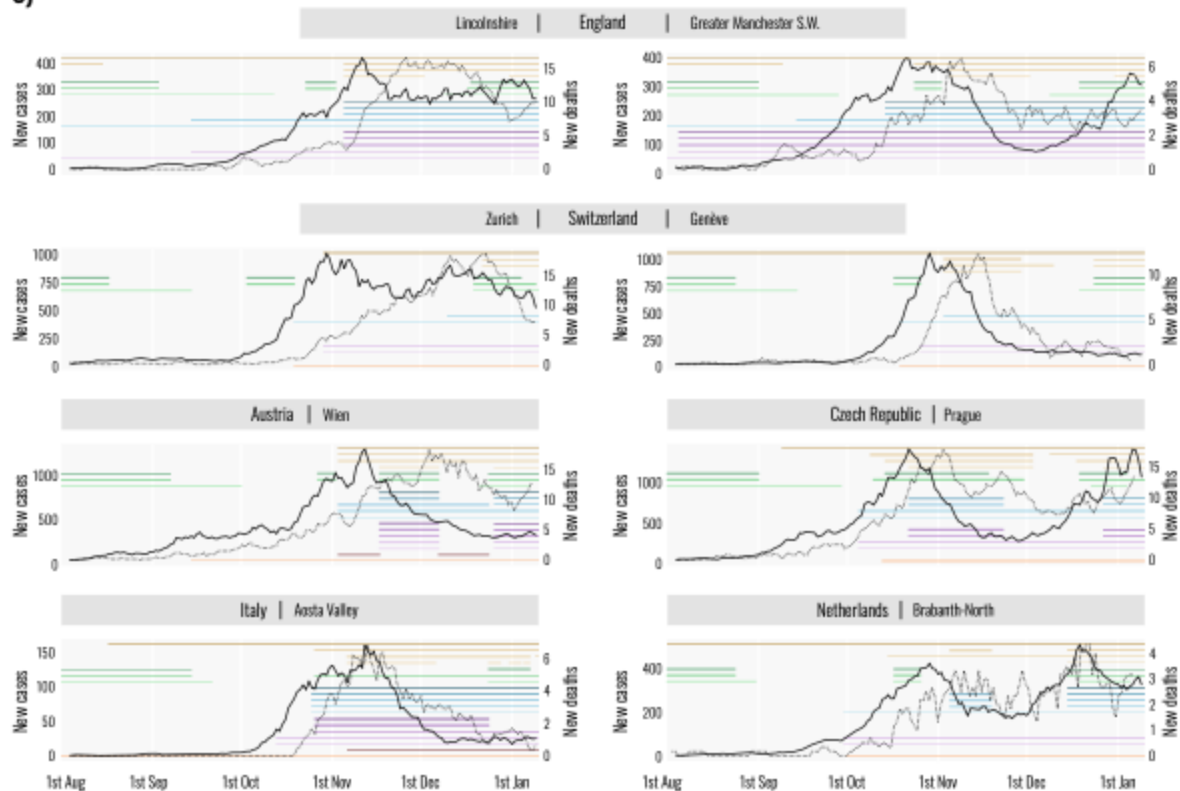


# **This work**

- Bespoke data categorisation
- Latent stochastic infection model (fancy regression)
- Robust evaluation of assumptions
- 1st August to 9th January 2021
- 7 countries with 114 regions of analysis



**C)**



# Results

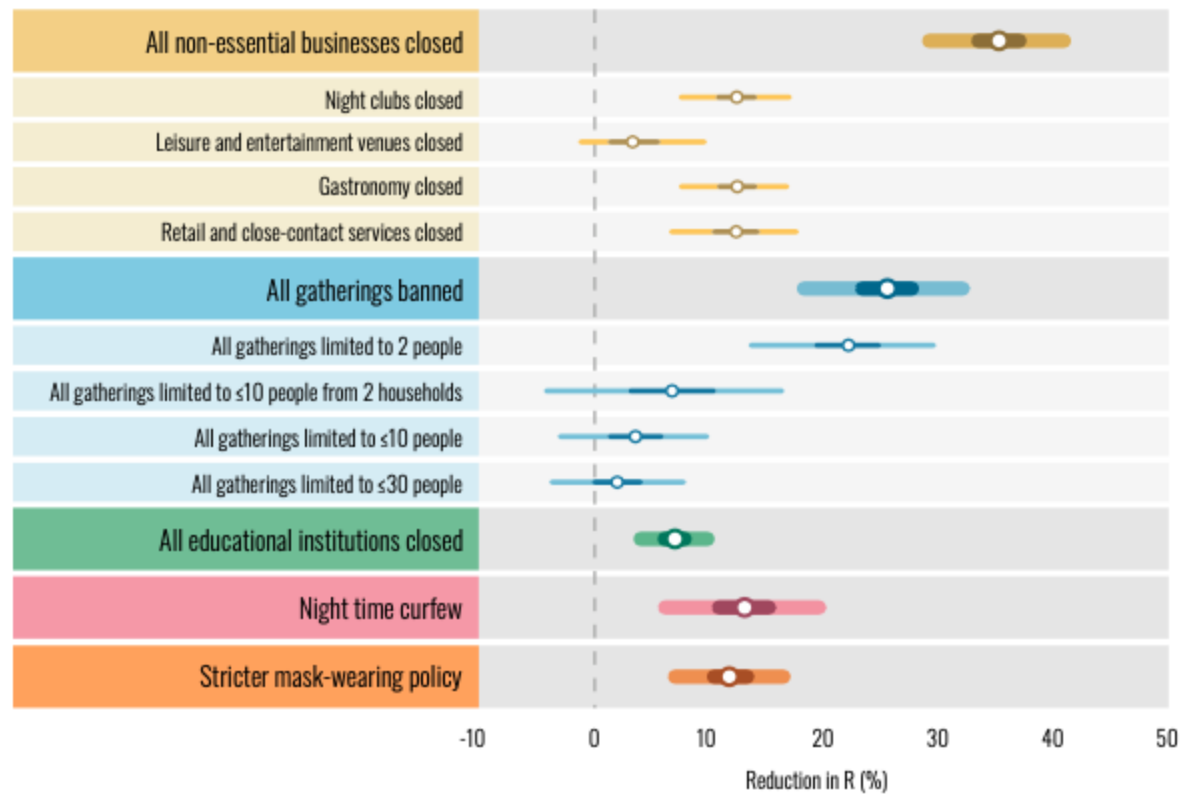
## First vs second wave

- Pairwise 6969 days on average with on NPI and not the other with minimum of 635 region days
- NPIs reduced Rt by 66% [95% CI: 61%-69%] vs 77%-82%
- The most stringent set reduced Rt by 56% [95% CI: 40%-64%] vs 76%-82%

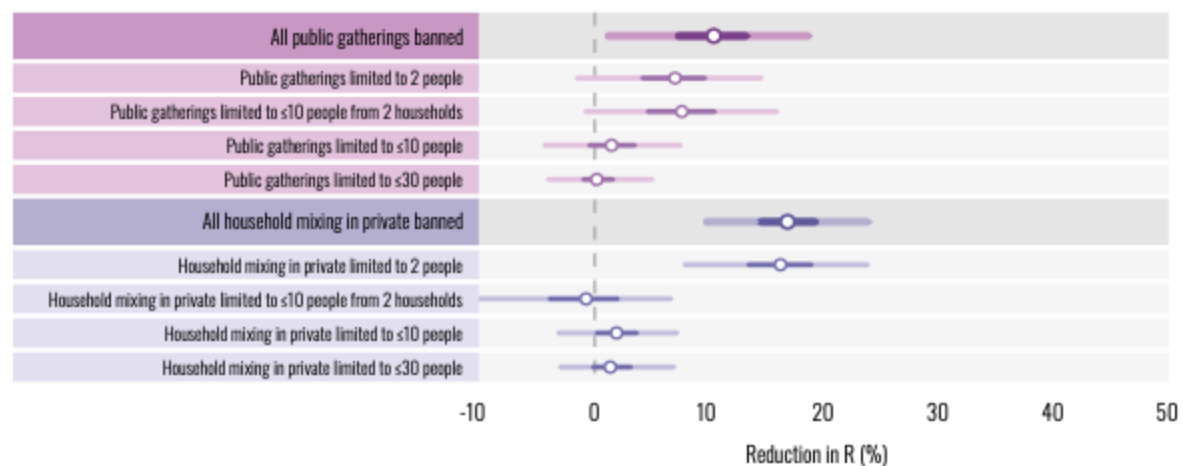
## Effect estimates

- Lockdown effect: 52% [95% CI: 47-56%]
- Stricter mask-wearing policy (mandatory in most or all shared/public spaces) and a night time curfew had moderate, but statistically significant effects [12%, 95% CI: 7-17%] and [13%, 6-20%])

A)

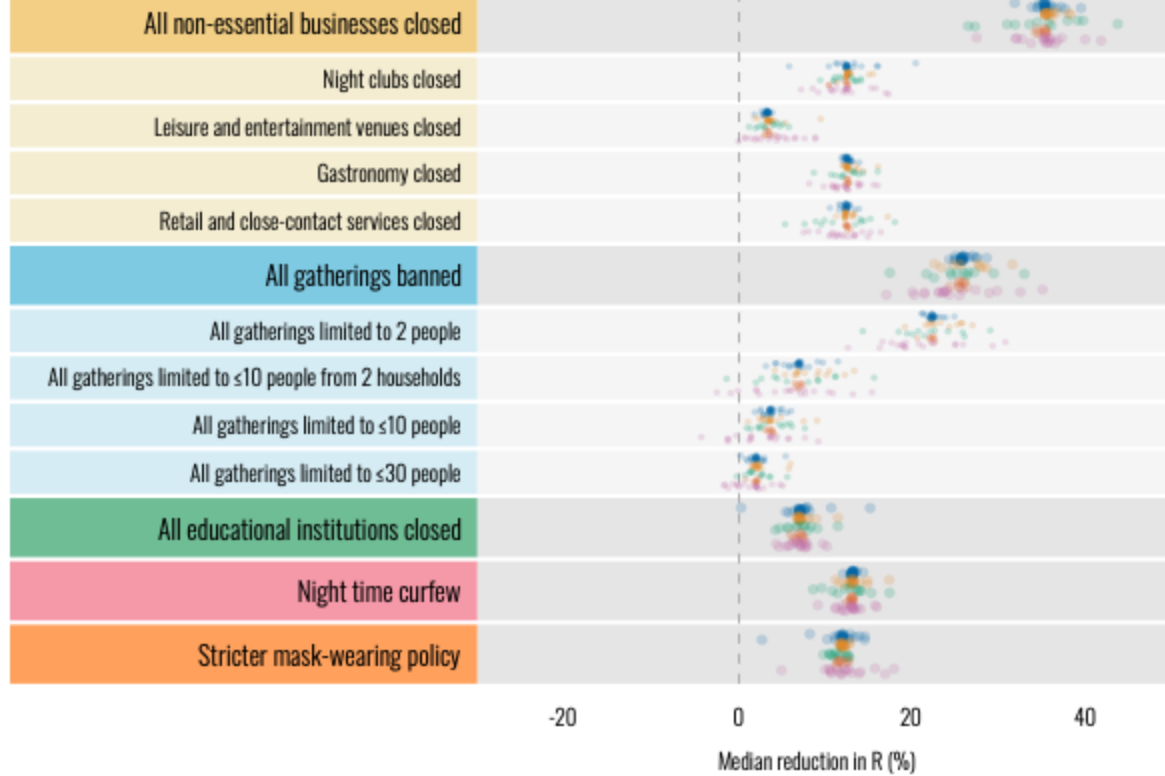


B)

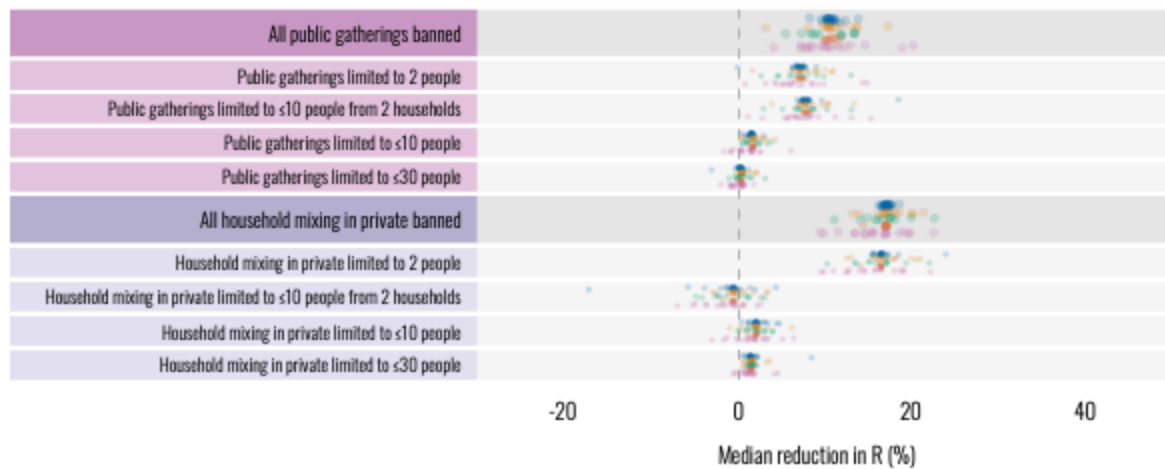


# Estimate of robustness

- 17 sensitivity analyses
- Stability of unobserved effects (unrecorded NPIs, changes to ascertainment and fatality rates)
- Sensitivity analysis in SI is made up of lots of graphs, investigate in your own time.



**B)**





# Conclusions

# Summary

- Estimates can be used to inform reopening
- First such estimates

# Limitations

- We don't know the impact of voluntary safety measures or variants
- No effectiveness estimate can apply to all regions.

# Methods

# Summary

- Lots of details about NPI collection - looks robust.
- Model: Nothing, all in supplement.

# Code

<https://github.com/MrinankSharma/COVID19NPISecondWave>

*TLDR: it is pretty nice python*

**Model**

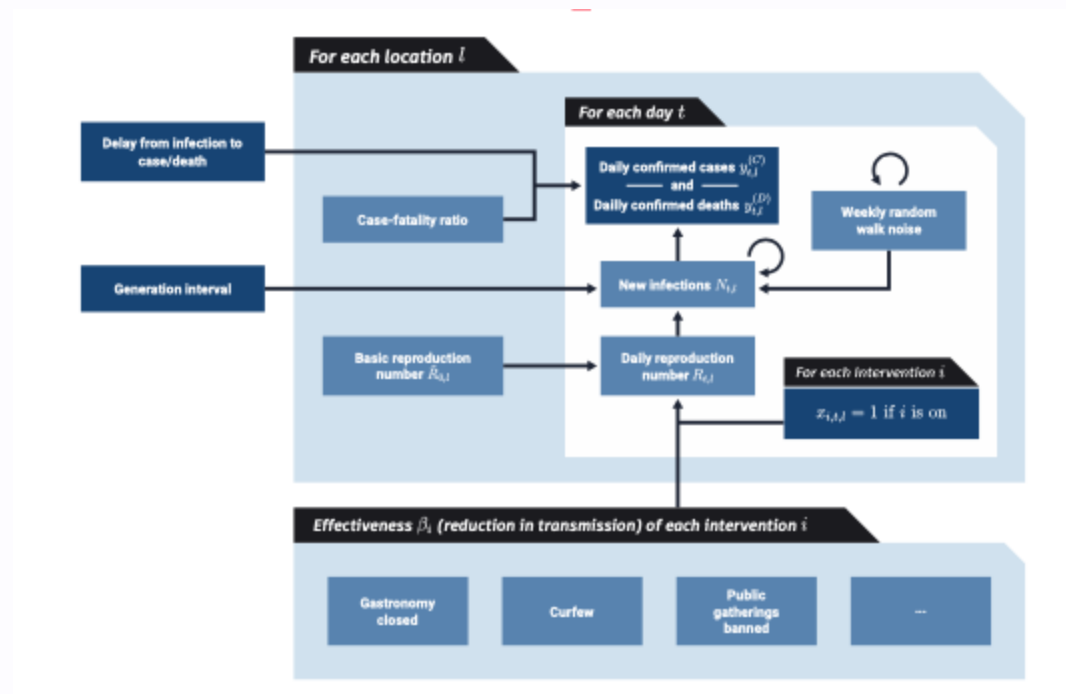
# Summary

- Fixed effects NPI regression model with a 7 day random walk.
- Fixed generation time renewal equation based model to estimate infections
- Fixed delays to death and report
- IFR fit by location but fixed in time (explored UK varying IRFs taken from the imperial dashboard as a sensitivity)



# Summary

- Country specific overdispersion in the negative binomial observation model.
- Implemented in NumPyro using NUTS MCMC.
- McAloon incubation period
- Linelist data from Austria, Germany, and UK for report delay.



(weekly) random walk. The random walk term allows  $R_{t,l}$  to change from one week to the next. Precisely,  $R_{t,l}$  follows:

$$R_{t,l} = \underbrace{\tilde{R}_{0,l}}_{\substack{R \text{ at } t=0 \text{ if no} \\ \text{NPIs active}}} \overbrace{\left( \prod_{i=1}^I \exp(-\beta_i x_{i,t,l}) \right)}^{\text{effect due to active NPIs}} \underbrace{\exp(z_{t,l})}_{\text{latent random walk}}, \quad (1)$$

where  $x_{i,t,l} = 1$  means NPI  $i$  is active in location  $l$  on day  $t$  ( $x_{i,t,l} = 0$  otherwise), and  $I$  is the number of NPIs. We now explain each of these terms in more detail.

We place a prior distribution over  $\tilde{R}_{0,l}$ , the reproduction number (in the absence of NPIs) on August 1st 2020. In fact, many locations had some recorded interventions active at  $t = 0$ . Therefore, we chose the mean of the prior on  $\tilde{R}_{0,l}$  carefully. We ensured the prior on  $R_{0,l}$  matched published estimates<sup>a</sup> of  $R_t$  for the first week of August from (3) and (4). For clarity,  $\tilde{R}_{0,l}$  is the reproduction number that *would* have been observed in location  $l$  at  $t = 0$  had no NPIs been active. The prior over  $\tilde{R}_{0,l}$  follows:

$$\tilde{R}_{0,l} \sim \text{Truncated Normal}(1.35, 0.3^2), \quad (2)$$

where truncation prevents values of  $\tilde{R}_{0,l}$  less than 0.1.

We parameterise the effect of NPI  $i$  with the effect parameter  $\beta_i$ . This parameter is independent of time and shared across all locations, i.e., the effectiveness of a particular NPI is assumed to be identical across regions (though the random walk described below can account for differences). We place an Asymmetric Laplace prior over the effect parameter  $\beta_i$ , with scale parameter 30, asymmetry parameter 0.5, and location parameter 0. This prior has mean 0.05 and standard deviation 0.07. The prior allows for (unbounded) positive and

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<sup>a</sup>The prior over  $R_{0,l}$  depends on  $\tilde{R}_{0,l}$ , the interventions active at  $t = 0$  in location  $l$  and the prior on the effectiveness of NPIs. We fixed the intervention prior first and then chose the prior on  $\tilde{R}_{0,l}$

negative effects as we cannot exclude the possibility that an NPI increases transmission. However, our prior places 80% of its mass on positive effects, reflecting a belief that NPIs are more likely to reduce transmission than to increase it. Furthermore, this is a shrinkage prior—it places more than 80% of its mass on “small” effectiveness (less than 10% change in  $R_{t,l}$ ).

noise terms follow:

$$z_{t,l} = \begin{cases} 0 & t \leq 13 \\ z_{t-1,l} + \varepsilon_{\lfloor (t-14)/7 \rfloor, l} & \text{if } t \bmod 7 = 0, \\ z_{t-1,l} & \text{otherwise} \end{cases} \quad (3)$$

where  $\lfloor \cdot \rfloor$  denotes the floor operation and  $\varepsilon_{i,l} \sim \text{Normal}(0, \sigma_R^2)$ . In words,  $z_{t,l}$  is set to 0 for the first two weeks, meaning that  $R_{t,l}$  depends only on  $\tilde{R}_{0,l}$  and the active interventions for the first two weeks. Then, every week, the value of  $z_{t,l}$  may increase or decrease depending on the noise variable  $\varepsilon_{i,l}$ . If we observe that transmission increased in a particular week, then we may infer  $\varepsilon_{i,l} > 0$  and vice versa.

The random walk addresses an important limitation—we cannot include all possible factors that affect transmission. We can attempt to attribute effect sizes to NPIs at a time  $t$ , but we need to agnostically account for other unobserved factors that could have changed transmission (e.g. behaviour, adherence). By using a random walk, we include a latent stochastic process that agnostically models unobserved trends and residual structural correlations.

*walk prior appears to be shared in time and space*

$$\bar{N}_{t,l} = R_{t,l} \sum_{\tau=1}^{32} (\bar{N}_{t-\tau,l} \cdot \pi_{GI}[\tau]). \quad (4)$$

Renewal processes have a strong relationship to Hawkes processes and arise naturally from a Bellman Harris branching process (6,5). The renewal equation has also been shown to be equivalent to a susceptible-exposed-infected-recovered Erlang model. The renewal equation therefore specifies an epidemiologically motivated function class. One issue with the renewal equation is that it specifies a deterministic expectation for the number of new infections. This is generally suitable as infections become large, but in low incidence settings, estimation of  $R_{t,l}$  can be sensitive to random fluctuations and noise. Therefore, we include an additive noise term, reflecting a belief that changes in the number of infections at low infection counts provide limited evidence to ascertain  $R_{t,l}$ , and must be treated with caution. Thus, the actual number of infections follows:

$$N_{t,l} = \text{softplus}(\bar{N}_{t,l} + \epsilon_{t,l}), \quad (5)$$

where  $\epsilon_{t,l}^{(N)} \sim \text{Normal}(0, 5^2)$  (sensitivity analysis in Fig. S9). We use the  $\text{softplus}(\cdot)$  rectifier to ensure that  $N_{t,l} \geq 0$ .

We seed the model with one week of unobserved initial infections<sup>b</sup>

$$N_{-t,l} = \text{Lognormal}(\tilde{\mu} = 0, \tilde{\sigma} = 3), \quad \text{for } 1 \leq t \leq 7. \quad (6)$$

**Table 1: Table of epidemiological parameters, their distributional forms and their source.**

<b>Delay</b>	<b>Distributional form of delay</b>	<b>Source</b>
Generation interval	Gamma(mean=4.83, sd = 1.73)	Meta-analysis (14)
Incubation period	Gamma(mean=5.53, sd = 4.73)	Meta-analysis (14)
Onset to reported death	Gamma(mean=18.61, sd = 13.62)	Linelist (Sec. 1.2)
Onset to case confirmation	Gamma(mean=5.28, sd = 3.75)	Linelist (Sec. 1.2)