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# **On the complexity of Modelling the phases of SARS-CoV2 transmission**

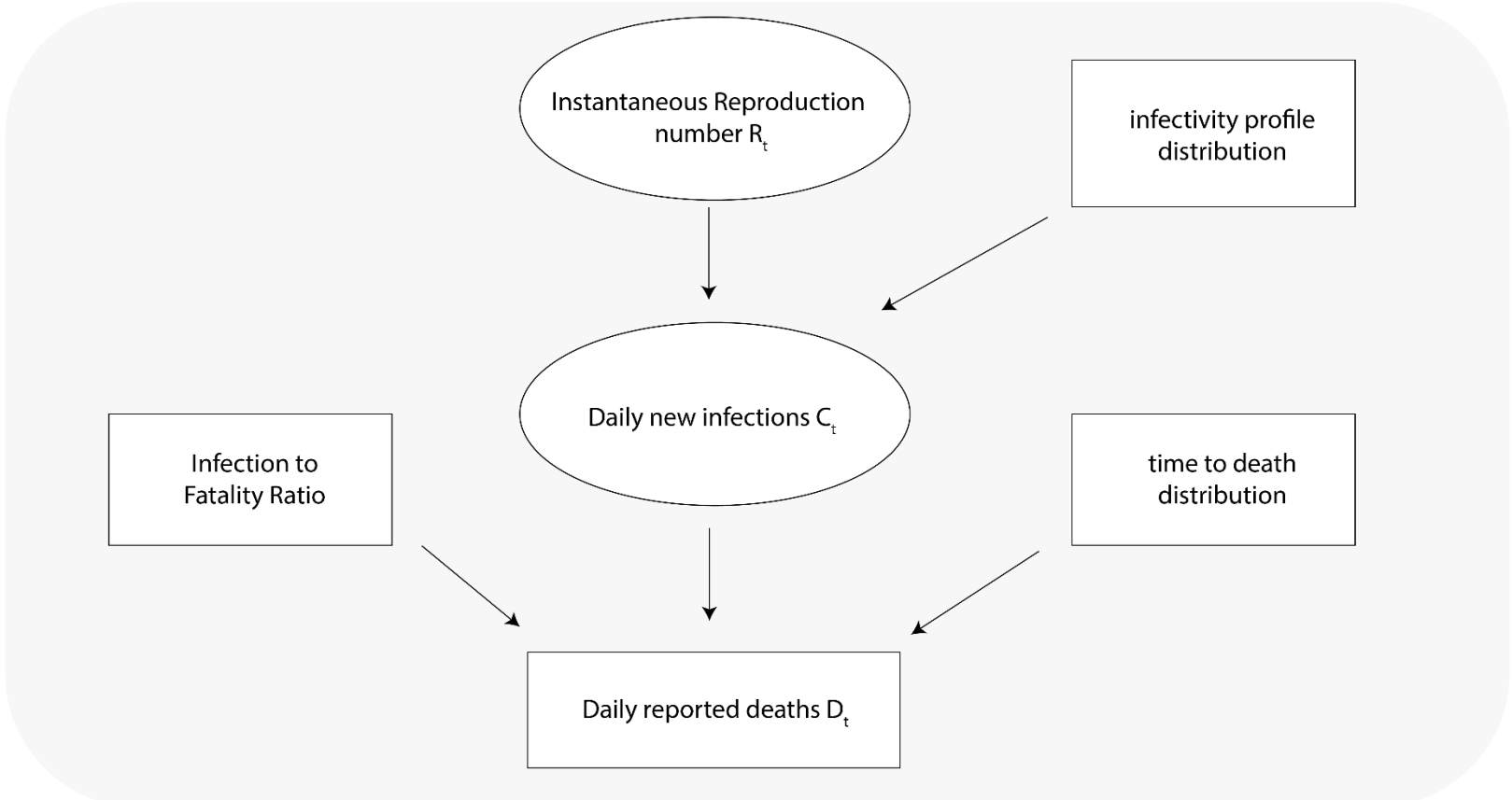
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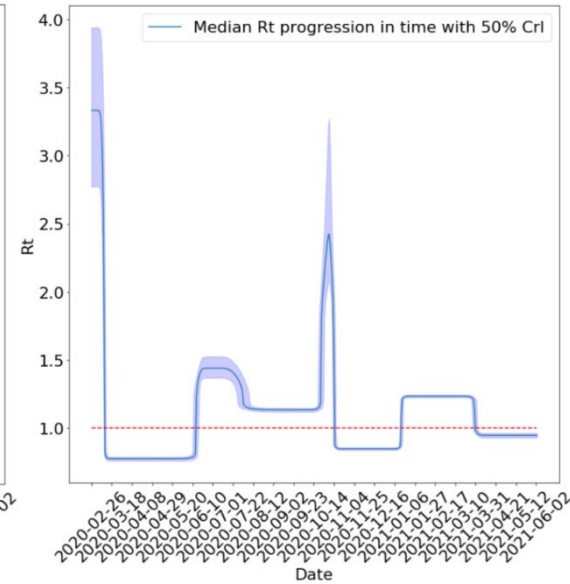
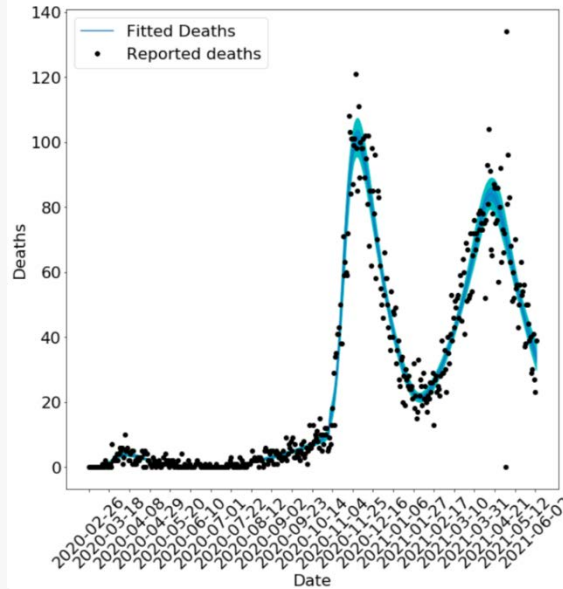
# A graphical representation of the stochastic epidemic model



## Some considerations about this model

- We consider that the true infection dynamics can be described by a hierarchical stochastic epidemic model with piece-wise constant reproduction number building on the work of Flaxman et al.
- The Flaxman et al. model was published in *Nature* on June 2020.
- The main source of criticism for this model was that the time of the changes of the  $R_t$  was a-priori defined.
- Also, it was assumed that all the NPIs had a positive effect on the transmissibility.
- We amend the transmission mechanism of the Flaxman model by inferring the location and magnitude of  $R_t$  changes using stochastic changepoints.

## Greece model fit until 2021-06-03



## Introduction of Bayesian non-parametrics into our framework (work in progress)

- In the previous framework the number of stochastic changepoints was a-priori selected and not learned from the data.
- We use Bayesian non-parametric methods in order to determine an appropriate model complexity directly from data in a fully Bayesian manner.
- Specifically, the stick-breaking construction of the Dirichlet process.
- The non-parametric nature of this model makes it an ideal candidate for modelling the phases of an epidemic where the distinct number of phases is unknown beforehand.

## Some early promising results from simulations

- We simulate data from an epidemic for  $t = 1, \dots, 150$ .
- At time points  $T_1 = 60$  and  $T_2 = 100$  we have a change in transmissibility.

$$R_t = \begin{cases} 2.5 & t < T_1 \\ 0.95 & T_1 \leq t < T_2 \\ 0.7 & T_2 \leq t \end{cases}$$

variable	mean	sd	95% Cr.I.
$R_1$	2.50	0.0	(2.50, 2.50)
$R_2$	0.95	0.0	(0.95, 0.95)
$R_3$	0.70	0.0	(0.70, 0.70)
$T_1$	60.39	0.24	(60.10, 60.96)
$T_2$	100.59	0.21	(100.11, 100.90)

Table I: Results were obtained using Hamiltonian Monte Carlo.

# Thank you for your attention