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RESEARCH REPORT

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SEIR Filter – Stochastic Model of Pandemics

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1 Introduction

There are lots of epidemiological models at hand, most of them working well, simply because is not difficult to fit (nearly) exponential growth/decline and it is not difficult to make the growth factor dependent on various factors. What is difficult, however, is to distinguish the impact of the factors from random fluctuations. Moreover, as many counter-measures as well as their release come hand in hand, it is hard to distinguish their impact. In statistics, the former phenomenon is called insignificance, the latter either non-idetifiability (if the data do not allow to distinguish two parameters) or co-linearity (if the parameters cannot be distinguished with sufficient certainty). Last but not least, the epidemics is observed only indirectly through noisy and/or delayed data, which obstacle cannot be handled only by adding noise to the observations. Needless to say that, once these phenomenons are not taken into account or are handled insufficiently, wrong policy recommendations stem from the models.

Mathematical statistics disposes of tools to handle all significance, co-linearity and partial observability; however, to our best knowledge, there is no work is systematically doing so for compartment epidemiological models. The goal of the present paper is to start filling this gap by proposing a general stochastic epidemiological model, which we call SEIR Filter.

Our model is a discrete time discrete space one. Next we argue that this is the best choice for practical use.

There is a large literature on continuous time diffusion models, see e.g. [2] and the references therein. Yet these models are able to capture global properties of an epidemics and handle noise, their practical usage is limited as it is difficult to incorporate heterogeneity, caused by non-pharmaceutical intervention, into these models. Moreover, given discrete time data, discretization of these models is needed which does not inherit their favorable properties.

Another wide class of models are those with continuous time and discrete state space. In [3], for instance, even an estimation procedure is developed for such a model. Yet these models are realistic, respecting integer sizes of the compartments, again there is a problem with their application in practice. When schools are closed, for instance, which time exactly should we change the transition matrix?

As we have premised, we regard discrete-time discrete-space stochastic models as the most practical. One of the models closest to our one is that by [7] (or [12]). When estimating its parameters, the authors use Monte Carlo simulation to evaluate likelihood functions. We argue and demonstrate that parameters of these models can be estimated more straightforward way, as the likelihood (or other estimating) function can be computed exactly, which allows for quicker and more reliable estimation.

In addition to the tractability of the estimating functions, our model allows for closed form formulas for expected future compartment sizes and for reproduction number. We also give simple criteria for vanishing and explosion of the epidemics, as well as bounds of limiting expected sizes given stationary imports. All this allows various applications of the model, e.g. in optimal control of the pandemics.

After a rigorous probabilistic formulation of the model (Section 2), we discuss its basic probabilistic properties (Section 3), its autonomous sub-models and reproduction number (Section 4) and asymptotic properties (Section 5). Next, we discuss estimation of the model (Section 6) and demonstrate its usage by its application to the COVID pandemics in Czech Republic 2020 (Section 7). Finally, we conclude the paper (Section 8).

2 Model Definition

Assume an infinite population, each individual of which is either susceptible, or finds himself in one of the compartments $S_1, \dots, S_k$. Let $I_t \in \mathbb{N}_0^k$, $t \in \mathbb{N}_0^+$, be an observable external inflow (import) of individuals into the compartments and let $Z_t \in \mathbb{R}^n$, $t \in \mathbb{N}_0^+$, be an observed exogenous process.

For any $t \in \mathbb{N}_0$, let $X_t = (X_t^1, \dots, X_t^k) \in \mathbb{N}_0^k, t \in \mathbb{N}_0$, be a possibly hidden stochastic process of the sizes of the compartments $(1, \dots, k)$ which we define later. Let

$$Y_t \in \mathbb{R}^n, \qquad Y_t = FX_t + \epsilon_t, \qquad t \in \mathbb{N}_0,$$

be a process of observations where F is a deterministic $n \times k$ matrix with rank n, and ϵ_t is a random vector. Denote $(\mathcal{F}_t)_{t\geq 0}$ and $(\mathcal{G}_t)_{t\geq 0}$ the filtrations induced by (X, Y, I, Z), by (Y, I, Z), respectively. The filtrations may be seen as information flows; in particular, $(\mathcal{F}_t)$ represents all the information while $(\mathcal{G}_t)$ the observable one. We assume that

$$\mathbb{E}(\epsilon_{t+1}|\mathcal{F}_t) = 0, \qquad \mathrm{var}(\epsilon_{t+1}|\mathcal{F}_t) = \mathrm{diag}(\Gamma_t X_t), \qquad t \in \mathbb{N}_0,$$

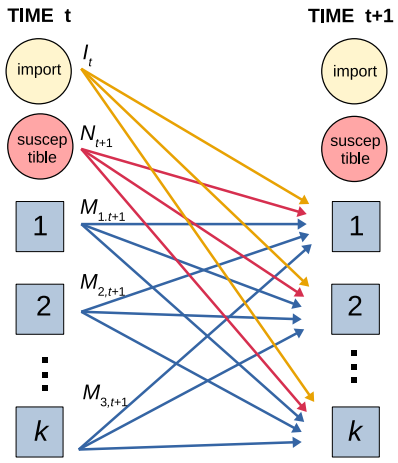
where $\Gamma_t \in \mathcal{G}_t$ is a random matrix.

We define X recursively: We let X_0 to be a possibly random vector and, for any $t \in \mathbb{N}$, we put

$$X_{t+1} = I_t + N_{t+1} + M_{1,t+1} + \cdots + M_{k,t+1}.$$

Here, $N_{t+1} \in \mathbb{N}_0^k$ is the inflow of domestically infected indivuduals (different from I_t) such that $N_{t+1}|\mathcal{F}_t \sim \mathrm{Po}(B_t X_t)$ where $B_t = (\beta_t^{ij})_{1\leq i,j\leq k}$, $B_t \in \mathcal{G}_t$ (meaning that B is a $(\mathcal{G}_t)$-adapted process) is a random matrix and, for any vector x, $\mathrm{Po}(x)$ stands for a vector of independent Poisson variables with the intensities given by x. Further, for any i, $M_{i,t+1} \in \mathbb{N}_0^k$ is the distribution (not a probability one) of those, who found themselves in compartment i at t, between the compartments at $t+1$. In particular, $M_{i,t+1}^j$ (the j-th component of $M_{i,t+1}$) is the number of the individuals who transited from compartment j to compartment i from t to $t+1$. Assuming the individuals to change their state according to a common transition matrix $P_t = (p_t^{ij})_{1\leq i,j\leq k} \in \mathcal{G}_t$ with the transitions being conditionally independent given $\mathcal{F}_t$, we get that $M_{i,t+1}$ has a multinomial conditional probability distribution with parameters X_t^i and $P_t^{(i)}$ where, for any matrix A, $A^{(i)}$ is the i-th column of A.

The assumed flows of individuals are illustrated by the following chart.



Finally, we assume $N_{t+1}, M_{1,t+1}, \dots, M_{k,t+1}, \epsilon_{t+1}$ to be mutually conditionally independent given $\mathcal{F}_t$ (which, in words, means that all the dependence between the inflows, the transitions and the observation can be explained by the state of the system at t). Consequently,

$$X_{t+1}|\,\mathcal{F}_t \sim \bigcirc_{1\le i\le m}\text{Multinomial}(X_t^i, P_t^{(i)}) \circ \text{Po}(B_t X_t) \circ \delta(\bar{I}_t),$$

where $\bigcirc$ and $\circ$ stand for the summation of (mutually) independent random vectors.

3 Model Properties

By probability calculus, we get that

$$\mathbb{E}\left[\begin{array}{c} X_{t+1} \\ Y_{t+1} \end{array}\middle|\, \mathcal{F}_t\right] = \begin{bmatrix} E \\ F \end{bmatrix} (T_t X_t + I_t), \tag{1}$$

$$\text{var}\left(\begin{array}{c} X_{t+1} \\ Y_{t+1} \end{array}\middle|\, \mathcal{F}_t\right) = \begin{bmatrix} E \\ F \end{bmatrix} \Lambda_t(X_t) \begin{bmatrix} E \\ F \end{bmatrix}^T + \text{diag}\begin{pmatrix} 0_{\text{k}} \\ \Gamma_{\text{t}} \text{X}_{\text{t}} \end{pmatrix}, \qquad t \ge 0,$$

where E is the identity matrix and

$$T_t \stackrel{\text{def}}{=} P_t + B_t, \qquad \Lambda_t(X_t) \stackrel{\text{def}}{=} \sum_{i=1}^{k} [\text{diag}(P_t^{(i)}) - P_t^{(i)}(P_t^{(i)})^T] X_t^i + \text{diag}(B_t X_t), \qquad t \ge 0.$$

Note that

$$\Lambda_t(x) = \sum_{i=1}^{k} \Phi_{t,i} x^i, \qquad \Phi_{t,i} \stackrel{\text{def}}{=} \text{diag}(B_t^{(i)} + P_t^{(i)}) - P_t^{(i)}(P_t^{(i)})^T, \qquad t \ge 0, x \in \mathbb{R}_+^k,$$

i.e. Λ_t is linear in x.

Consequently, for any $t, s \in \mathbb{N}_0, t > s$,

$$\mathbb{E}(X_t|\mathcal{F}_s) \;=\; \mathbb{E}(T_{s,t-1}X_s + \sum_{\theta=s}^{t-1} T_{\theta+1,t-1} I_\theta|\mathcal{F}_s) \;=\; \mathbb{E}(T_{s,t-1}|\mathcal{F}_s)X_s + \sum_{\theta=s}^{t-1} \mathbb{E}(T_{\theta+1,t-1} I_\theta|\mathcal{F}_s),$$

where, for any matrix process A, $A_{s,t} \stackrel{\text{def}}{=} A_t \times \cdots \times A_s$ with $A_{s,s-1} \stackrel{\text{def}}{=} E$.

In the special case that

$$B_\tau \equiv B_s, \qquad P_\tau \equiv P_s, \qquad s \le \tau \le t, \tag{2}$$

we have

$$\mathbb{E}(X_t|\mathcal{F}_s) = T_s^{t-s} X_s + \sum_{\tau=s}^{t-1} T_s^{t-\tau-1} \mathbb{E}(I_\tau|\mathcal{F}_s),$$

and

$$\mathbb{E}(X_t|\mathcal{G}_s) = T_s^{t-s} \mathbb{E}(X_s|\mathcal{G}_s) + \sum_{\tau=s}^{t-1} T_s^{t-\tau-1} \mathbb{E}(I_\theta|\mathcal{G}_s)$$

If, in addition, $\mathbb{E}(I_\theta|\mathcal{G}_s) \equiv \mu$ for some $\mu \in \mathcal{G}_s$ and $(E - T_s)$ is invertible, the latter formula simplifies to

$$\mathbb{E}(X_t|\mathcal{G}_s) = T_s^{t-s} \mathbb{E}(X_s|\mathcal{G}_s) + (E - T_s)^{-1}(E - T_s^{t-s})\mu.$$

4 Sub-epidemics and Reproduction Number

We say that the subset of compartments $D = \{s_1, \dots, s_m\}$ is *subepidemic* if, for any t and any $i \in D$ and $j \notin D$, $\beta_t^{ij} \equiv \beta^{ji} \equiv 0$, and $p_t^{ij} \equiv 0$. In words this means that, for any $i \in D$, the i-th compartment does not increase through direct infection, the infection does not depend on the compartment, and it is impossible to get to the state i once being outside D.

Let, after a possible re-ordering, $m \in \mathbb{N}$ be such that $\{1, \dots, m\}$ is subepidemic (such m always exists because it can be always put to k). For any vector $x \in \mathbb{R}^k$, denote $\overline{x}$ its restriction to $(1, \dots, m)$ and, for any matrix $A \in \mathbb{R}^{k \times k}$, denote $\overline{A}$ its restriction to $(1, \dots, m) \times (1, \dots, m)$.

Observe that $\overline{X}$ follows its own version of our model, namely that

$$\overline{X}_{t+1} \big| \, \mathcal{F}_t \sim \bigcirc_{1 \le i \le m} \text{Multinomial}(\overline{X}_t^i, \overline{P}_t^{(i)}) \circ \text{Po}(\overline{B}_t \overline{X}_t) \circ \delta(\overline{I}_t).$$

For any t, we define the reproduction number r_t (of a subepidemic $\{1, \dots, m\}$) as

$$r_t \stackrel{\text{def}}{=} \sum_{\tau=t}^{\infty} \mathbf{1}^T \mathbb{E}(B_\tau \overline{P}_{t,\tau-1} | \mathcal{F}_{t-1}) \pi_t, \qquad \pi_t = \mathbb{E}\left\{ \nu\left(\overline{N}_t + \overline{I}_{t-1}\right) \big| \, \mathcal{F}_{t-1} \right\}.$$

where ν is unit normalization of a vector. Observe that r_t complies with the usual definition of reproduction number as it equals to the conditional expectation (w.r.t. $\mathcal{F}_{t-1}$) of the infections caused by an individual having arrived at t. To see it, note that π_t is the conditional distribution of the state in which a randomly chosen newcomer (the one brought by the import or by the infection) finds himself at t, and observe that, for each newcomer at t, the expected number of those infected by him at $t+1$ is given by the sum of the components of $\overline{B}_t \pi_t$, the expected number infected at $t+1$ is given by the sum of components of $\overline{B}_{t+1} \overline{P}_t \pi_t$ etc.

If $\mathcal{F}_t \neq \mathcal{G}_t$ (i.e. X is not fully observed), then the reproduction number has to be estimated, most naturally by its conditional expectation with respect to the known information:

$$\tilde{r}_t \stackrel{\text{def}}{=} \mathbb{E}(r_t | \mathcal{G}_t) = \sum_{\tau=t}^{\infty} \mathbf{1}^T \mathbb{E}(\overline{B}_\tau \overline{P}_{t,\tau-1} \pi_t | \mathcal{G}_{t-1}).$$

In the special case of $\overline{B}_\tau \equiv \overline{B}_{t-1}, \overline{P}_\tau \equiv \overline{P}_{t-1}$, $\tau \ge t$, with $\rho(\overline{P}_{t-1}) < 1$ where ρ is the spectral radius, the formula simplifies to

$$\tilde{r}_t = \mathbf{1}^T \overline{B}_{t-1} \left(\sum_{i=0}^{\infty} \overline{P}_{t-1}^i \right) \mathbb{E}(\pi_t | \mathcal{G}_{t-1}) = \mathbf{1}^T \overline{B}_{t-1} (E - \overline{P}_{t-1})^{-1} \mathbb{E}(\pi_t | \mathcal{G}_{t-1}).$$

Note that, once $\overline{N}_t$ is not observed, there could be difficulties computing $\mathbb{E}(\pi_t | \mathcal{G}_{t-1})$ – yet the estimate $\mathbb{E}(\pi_t | \mathcal{G}_{t-1}) \doteq \nu(\overline{B}_{t-1} \mathbb{E}(\overline{X}_{t-1} | \mathcal{G}_{t-1}) + \overline{I}_{t-1})$ seems a straightforward choice, it is generally not unbiased due to the normalization. This problem, however, vanishes if the imports and new infections all fall into a single state (typically called exposed), in which case $\pi_t \equiv (1, 0, \dots, 0)^T$.

5 Asymptotic Behavior

Keep assuming that $\{1, \dots, m\}$ is subepidemics. The next Proposition states conditions for vanishing, explosion and "stationary" behavior of the subepidemic compartments sizes.

Proposition 1. *(i) If $\overline{T}_t \le S$ component-wise, where S is deterministic with $\sigma \stackrel{def}{=} \rho(S) < 1$, and if $\mathbb{E}\overline{I}_t = o(t^{-\alpha})$ for some $\alpha > 0$, then $\overline{X}_t \to 0$ almost sure. Here, ρ denotes the spectral radius of a matrix.*

(ii) If $\overline{T}_t \geq R$ where R is deterministic irreducible with $\varrho \overset{def}{=} \rho(R) > 1$ and either $\mathbb{E}\overline{X}_0 \neq 0$ or $\mathbb{E}\overline{I}_\tau \neq 0$ for some τ, then $\|\mathbb{E}\overline{X}_t\| \to \infty$.
(iii) If $\mathbb{E}\overline{I}_t \equiv \mu$ for some μ and $R \leq \overline{T}_t \leq S$ such that $\sigma \overset{def}{=} \rho(S) < 1$, then

$$\liminf_t \mathbb{E}\overline{I}_t \geq (E-R)^{-1}\mu, \qquad \limsup_t \mathbb{E}\overline{I}_t \leq (E-S)^{-1}\mu$$

Proof. (i) We have

$$\mathbb{E}\overline{X}_t = \mathbb{E}(\mathbb{E}(\overline{X}_t|\mathcal{G}_0)) = \mathbb{E}(\overline{T}_{0,\tau-1}X_0 + \sum_{\theta=0}^{t-1}\overline{T}_{\theta+1,t-1}\mathbb{E}(\overline{I}_\theta|\mathcal{G}_0))$$
$$\leq \mathbb{E}(S^tX_0 + \sum_{\theta=0}^{t-1}S^{t-\theta-1}\mathbb{E}(\overline{I}_\theta|\mathcal{G}_0)) \leq a_t + b_t, \qquad a_t = S^t\mathbb{E}\overline{X}_0, \qquad b_t = \sum_{\theta=0}^{t-1}S^{t-\theta-1}\mathbb{E}\overline{I}_\theta$$

Thanks to the sub-unit spectral radius of S, we have $a_t \to 0$. Further, by the non-negativity of H and the properties of convergence, there exists $c \in \mathbb{R}^m_+$ such that $\mathbb{E}\overline{I}_t \leq c(t+1)^{-1}$. Thus, for any ς fulfilling $\sigma < \varsigma < 1$, we get, after re-indexing the sum,

$$b_t = \sum_{\tau=0}^{t-1}S^\tau\mathbb{E}\overline{I}_{t-\tau-1} \leq \sum_{\tau=0}^{t-1}S^\tau c\frac{1}{(t-\tau)^\alpha} = \underbrace{\frac{1}{t^\alpha}}_{\to 0} \times \underbrace{\sum_{\tau=0}^{t-1}(\varsigma^{-1}S)^\tau c}_{\to(E-\varsigma^{-1}S)^{-1}c} \underbrace{\left(\frac{\varsigma^{\tau/\alpha}}{t-\tau}\right)^\alpha}_{\leq d} \to 0;$$

the second convergence holding because $\rho(\varsigma^{-1}S) = \frac{\sigma}{\varsigma_t} < 1$, the upper bound d existing as $f(\tau) \overset{\text{def}}{=} \frac{t\varsigma^{\tau/\alpha}}{t-\tau}$ increases in $\tau = t-1$ and its derivative has only a single root, so we have $f(\tau) \leq \max(f(0), f(t-1)) = \max(1, \varsigma^{\frac{t-1}{\alpha}}t) \leq d \overset{\text{def}}{=} \max(1, \frac{1}{e\varsigma^{1/\alpha}|\alpha^{-1}\ln\varsigma|})$ on $[0, t-1]$. Finally, thanks to the non-negativity of $\overline{X}$, convergence of $\mathbb{E}\overline{X}_t$ suffices for a.s. convergence of $\overline{X}_t$.

(ii) Let $\mathbb{E}\overline{X}_0 \neq 0$ and $\varrho > 1$. As R is irreducible non-negative ϱ is its eigenvalue and the corresponding eigenvector x is positive by the Perron-Frobenius Theorem. Further, by the irreducibility of T, there exists n such that $y \overset{\text{def}}{=} R^n\mathbb{E}\overline{X}_0 > 0$ component-wise, so there exist $e > 0$ such that $y \geq ex$. Thus

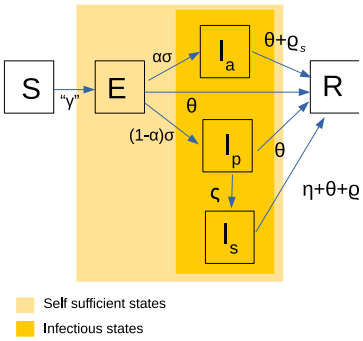
$$\mathbb{E}\overline{X}_t \geq R^t\mathbb{E}\overline{X}_0 \geq R^{t-n}y \geq eR^{t-n}x$$

norm of which converges to infinity. The proof for $\mathbb{E}\overline{I}_\tau \neq 0$ is analogous.

(iii)

$\mathbb{E}\overline{X}_t = \sum_{\tau=0}^{t-1}\overline{T}_{\tau,t-2}\mu + \overline{T}_{0,t-1}\mathbb{E}\overline{X}_0 \leq \sum_{\tau=0}^{t-1}S^\tau\mu + S^{t-1}\mathbb{E}\overline{X}_0 \to (\sum_{\tau=0}^{\infty}S^\tau)\mu = (E-S)^{-1}\mu$ and similarly for R. □

Example 2. Say there are five states E – exposed, I_a – infectious asymptomatic, who will never show symptoms, I_p – infectious pre-symptomatic, who will later show symptoms, I_s – infectious symptomatic, and R – removed, including recovered, dead, and infectious isolated. All the $I_\bullet$ states are equally infectious, i.e. $\beta_t^{Ex} = \beta_t$, $x \in \{I_a, I_p, I_s\}$, where β is a $\mathcal{G}_t$-adapted process. The probability that the exposed transits to $\{I_a, I_p\}$ is σ, the probability of completely asymptomatic course is α, the probability of transition from I_p to I_s is ς. Further, the probability of ending I_a or I_s, by natural causes (recovery, end of infectiousness, death in case of I_s) is ϱ_a, ϱ_s, respectively. Finally, the probability that a symptomatic individual isolates himself is η and the probability that the individual is isolated regardless of his state is θ_t for some $\mathcal{G}_t$-adapted process θ. The situation is illustrated on the following Figure:



If we neglect (small) joint probabilities of natural exits from the infectious states and the isolations, we get

$$P_t = \begin{bmatrix} 1-\sigma-\theta_t & 0 & 0 & 0 & 0 \\ \alpha\sigma & 1-\varrho_a-\theta_t & 0 & 0 & 0 \\ (1-\alpha)\sigma & 0 & 1-\varsigma-\theta_t & 0 & 0 \\ 0 & 0 & \varsigma & 1-\varrho_s-\eta-\theta_t & 0 \\ \theta_t & \theta_t+\varrho_a & \theta_t & \theta_t+\eta+\varrho_s & 1 \end{bmatrix}, \qquad B_t = \begin{bmatrix} 0 & \beta_t & \beta_t & \beta_t & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

Clearly, we can put $m = 4$ (the first four states are subepidemic), getting

$$\overline{T}_t = C(\beta_t) - \theta_t E, \qquad C(\beta) = \begin{bmatrix} 1-\sigma & \beta & \beta & \beta \\ \alpha\sigma & 1-\varrho_a & 0 & 0 \\ (1-\alpha)\sigma & 0 & 1-\varsigma & 0 \\ 0 & 0 & \varsigma & 1-\varrho_s-\eta \end{bmatrix}.$$

By the well known rule, we have $\rho(\overline{T}_t) = \rho(C(\gamma_t)) - \theta_t$. We consider two ways of decreasing the spectral radius: decreasing the infection rate β_t (typically by some counter-epidemic measures) and increasing the isolation rate θ_t (e.g. by strengthening the tracing capacity).

Once there is a "target" spectral radius ρ_0, all the combinations of β and θ yielding $\rho(\overline{T}_t) = \rho_0$ fulfill $\rho_0 + \theta - \rho(C(\beta)) = 0$ giving a "marginal rate of substitiution" $\theta(\beta)' = -\frac{\partial}{\partial\beta}\rho(C(\beta))$ of the infectiousness by the isolation, i.e. how much we have to increase the isolation speed when we release the restrictions.

Example 3. Assume the fraction ν of the population is non-compliant, which means that, once a restriction on social contacts is imposed, they apply it only partially. Assume that, without restrictions, the population is mixed which means that each individual, compliant or not, has, up to a constant, $(1-\nu)$ contacts with the compliant individuals and ν contacts with the non-compliant ones. Once there is a measure under which the compliant individuals restrict their opportunities to contacts by ϕ, the non-compliant ones do so only to $f(\phi) > \phi$. As a result, the compliant ones will have, up to a constant, $\phi^2(1-\nu)$ contacts with the compliant ones, $\phi f(\phi)\nu$ contacts with the non-compliant ones, while the non-compliant will have $\phi f(\phi)(1-\nu)$ and $f(\phi)^2\nu$ contacts with the compliant, non-compliant, respectively.

Assuming a simple epidemic model with compartments I_c - infected compliant, I_n - infected non-compliant, and R - removed, with the course of infection being the same for both the compartments such that $\beta_t^{1i} = \beta c_i$ where β is a constant and c_i is the number of contacts of the i-th sub-population, this gives

$$P_t = \begin{bmatrix} 1-\varrho & 0 & 0 \\ 0 & 1-\varrho & 0 \\ \varrho & \varrho & 1 \end{bmatrix}, \qquad B_t = \begin{bmatrix} \beta\phi^2(1-\nu) & \beta\phi f(\phi)\nu & 0 \\ \beta\phi f(\phi)(1-\nu) & \beta f(\phi)^2\nu & 0 \\ 0 & 0 & 1 \end{bmatrix}$$

where ϱ is a removal rate (perhaps consisting of an artificial and a natural part). This gives

$$\overline{T}_t = \beta C + (1-\varrho)E, \qquad C = \begin{bmatrix} \phi^2(1-\nu) & \phi f(\phi)\nu \\ \phi f(\phi)(1-\nu) & f(\phi)^2\nu \end{bmatrix}$$

with

$$\varrho(\overline{T}_t) = \beta\rho(C) + (1-\varrho).$$

As the characteristic polynomial of C is

$$\lambda^2 - \lambda g, \qquad g = g(\phi,\nu) = \phi^2(1-\nu) + f(\phi)^2\nu$$

we clearly have $\rho(C) = g$.

Now say that our goal is to decrease $\rho(\overline{T}_t)$ to a predetermined value r by finding appropriate $\phi = \phi(\nu)$. In order to do so, we have to solve

$$\beta g(\phi(\nu),\nu) + (1-\varrho) = r.$$

Clearly, $\phi(0) = \phi_0 \stackrel{\text{def}}{=} \sqrt{\frac{r-1+\varrho}{\beta}}$. For $\nu > 0$ we get, by the Implicit function theorem,

$$\frac{\partial}{\partial\nu}\phi = \frac{f(\phi(\nu))^2 - \phi(\nu)^2}{2\phi(\nu)(1-\nu) + 2f(\phi(\nu))f'(\phi(\nu))\nu}.$$

Note that the derivative depends neither on r nor on ϱ . Thus we can easily compute how the non-compliance influences strictness of the necessary restrictions. For instance, we can get by the first-order Taylor expansion at $\nu = 0$:

$$\phi(\nu) \doteq \phi_0 + \nu\frac{f(\phi_0)^2 - \phi_0^2}{2\phi_0} = \phi_0\left(1 - \frac{\nu}{2}\right) + \nu\frac{f(\phi_0)^2}{2\phi_0}$$

roughly holding for ν close to zero.

6 Estimation

For any stochastic process A and integers $s > t$, denote $\hat{A}_{s|t} = \mathbb{E}(A_s|\mathcal{G}_t)$. When $T_\tau \in \mathcal{G}_t, t < \tau \leq s-1$ (which is trivially true if $s = t+1$), we get that

$$\begin{bmatrix} \hat{X}_{s|t} \\ \hat{Y}_{s|t} \end{bmatrix} = \mathbb{E}\left(\mathbb{E}\left(\begin{bmatrix} \hat{X}_s \\ \hat{Y}_s \end{bmatrix}\middle|\mathcal{F}_{s-1}\right)\middle|\mathcal{G}_t\right) = \begin{bmatrix} E \\ F \end{bmatrix}\left(T_{s-1}\hat{X}_{s-1|t} + \hat{I}_{s-1|t}\right)$$
$$= \begin{bmatrix} E \\ F \end{bmatrix}\left(T_{t,s-1}X_t + \sum_{\theta=t}^{s-1} T_{\theta+1,s-1}\hat{I}_{\theta|t}\right),$$

$$\begin{aligned} W_{s|t} &\stackrel{\text{def}}{=} \operatorname{var}(X_s|\mathcal{G}_t) = \operatorname{var}(\mathbb{E}(X_s|\mathcal{F}_{s-1})|\mathcal{G}_t) + \mathbb{E}(\operatorname{var}(X_s|\mathcal{F}_{s-1})|\mathcal{G}_t) \\ &= \operatorname{var}(T_{s-1}X_{s-1} + I_{s-1}|\mathcal{G}_t) + \mathbb{E}(\Lambda_{s-1}(X_{s-1})|\mathcal{G}_t) \\ &= T_{s-1}W_{s-1|t}T_{s-1}^T + 2T_{s-1}\operatorname{cov}(X_{s-1}, I_{s-1}|\mathcal{G}_t) + \operatorname{var}(I_{s-1}|\mathcal{G}_t) + \Lambda_{s-1}(\hat{X}_{s-1|t}) \end{aligned}$$

and

$$V_{s|t} \stackrel{\text{def}}{=} \operatorname{var}\left(\begin{matrix} X_s \\ Y_s \end{matrix}\middle|\mathcal{G}_t\right) = \operatorname{var}\left(\begin{matrix} X_s \\ FX_s + \epsilon_s \end{matrix}\middle|\mathcal{G}_t\right) = \begin{bmatrix} E \\ F \end{bmatrix} W_{s|t} \begin{bmatrix} E \\ F \end{bmatrix}^T + \operatorname{diag}\begin{pmatrix} 0_{\mathrm{k}} \\ \Gamma_{\mathrm{s-1}}\hat{\mathrm{X}}_{\mathrm{s-1|t}} \end{pmatrix}$$

Unfortunately, due to the non-Gaussianity, we have analytical formulas neither for $X_{t|t}$ nor for $\mathrm{W_{t|t}}$, so we can formulate neither the likelihood function nor a least square estimate. Two, from the computational point of view equivalent, ways to cope with this are using estimates of the conditional expectation and variance, or normally approximating the residuals. We go the latter way: in the present Section, we assume that $\left[\begin{smallmatrix} X_{t+1} \\ Y_{t+1} \end{smallmatrix}\right] \Big| \mathcal{F}_t$ is normal with mean given by (1) and

$$\mathrm{var}\left(\begin{matrix} X_{t+1} \\ Y_{t+1} \end{matrix} \middle| \mathcal{F}_t \right) = \begin{bmatrix} E \\ F \end{bmatrix} \Lambda_t(X_t \vee 0) \begin{bmatrix} E \\ F \end{bmatrix}^T + \mathrm{diag}\begin{pmatrix} 0_{\mathrm{k}} \\ \Gamma_{\mathrm{t}}(\mathrm{X_t} \vee 0) \end{pmatrix}$$

Given this assumption, we have, by the well known formula (see e.g. [1]),

$$\hat{X}_{t|t} = I_{t-1} + \hat{X}_{t|t-1} + K_t\left(Y_t - \hat{Y}_{t|t-1}\right)$$
$$K_t \stackrel{\mathrm{def}}{=} V^{XY}_{t|t-1}(V^{YY}_{t|-1})^{-1} = W_{t|t-1}F^T D_t^{-1}, \qquad D_t = FW_{t|t-1}F^T + \mathrm{diag}(\Gamma_t \hat{X}_{t|t-1})$$

$$W_{t|t} \quad = \quad V^{XX}_{t|t-1} \quad - \quad V^{XY}_{t|t-1}(V^{YY}_{t|t-1})^{-1}V^{YX}_{t|t-1} \quad = \quad W_{t|t-1} \quad - \quad W_{t|t-1}F^T D_t^{-1} F W_{t|t-1}$$

Note that K_t may be seen as a conditional version of a Kalman gain matrix.

Assume that $F = F(\Theta_0)$,$P_t = P_t(\Theta_0)$, $B_t = B_t(\Theta_0)$, $\Gamma_t = \Gamma_t(\Theta_0)$ and $I_t = I_t(\Theta_0)$ where $\Theta_0 \in \mathbb{R}^r$ is an unknown parameter. For its estimation, it is possible to to use either nonlinear least squares, i.e.

$$\hat{\Theta} = \arg\min \sum_t (Y_t - \hat{Y}_{t|t}(\Theta))^T U_t (Y_t - \hat{Y}_{t|t}(\Theta))$$

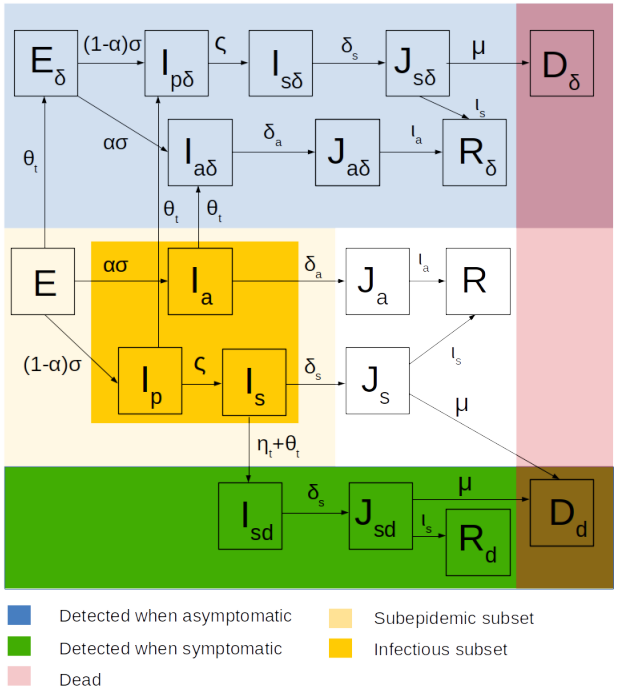
where $U_t \in \mathcal{G}_{t-1}$ is a suitable weighting matrix, or

$$\tilde{\Theta} = \arg\min \sum_t \varphi(Y_t - \hat{Y}_{t|t}(\Theta), D_t(\Theta)), \qquad \varphi(x,v) = -\frac{k\ln 2\pi + \ln\det(v) + x^T v^{-1} x}{2}$$

Both these estimators are consistent and asymptotically normal under some conditions, see [5], [6], respectively. Verifying these conditions for our model is, however, beyond the scope of this short technical report and remains as topic of a future research. Note also that our proof of Proposition 1 is not valid for the approximate model, as $\overline{X}$ is not necessarily positive here.

7 Application to The COVID Pandemics in Czech Republic

We applied our model to the data from the first and second wave of the pandemics in the Czech Republic between February and November of 2020. We considered a generalized version of the model from Example 2, compartments of which are shown in the following Picture:



To the compartments from Example 2 we added their "detected" versions, distinguishing detection when being asymptomatic (subscript δ) and when being symptomatic (subscript d). Moreover, for both the symptomatic and the asymptomatic course, we added the states in which the individuals are RNA positive, but not infectious (denoted by J). We also distinguish two "removed" states: recovered (R) and dead (D). All the J and R states have three versions: undetected, detected when asymptomatic and detected when symptomatic. Assuming that once the undetected course ends by death, the detection takes place before the time of death, we have only two death states: detected when asymptomatic and detected when symptomatic.

We use the following parameters of the disease, obtained from the literature:

Par.	description	Source
$m_E = 5.08$	mean of incubation period	[4]
$\alpha = 0.179$	probability of asymptomatic course	[8]
$m_a = 4$	expected duration of presymptomatic period	[9]
$m_i = 8$	expected duration of infectiousness	[13]
$m_y = 22.6$	expected duration of RNA positivity for asymptomatic	[10]
$m_s = 25.2$	expected duration of RNA positivity for asymptomatic	[10]

Assuming the compartment occupation times to be exponential, we get the following transition probabilities:

Par.
$\sigma = 1 - \exp\{-1/m_E\}$
$\varsigma = 1 - \exp\{-\frac{1}{m_q}\}$
$\delta_s = 1 - \exp\{-\frac{1}{m_s}\}$
$\delta_a = 1 - \exp\{-\frac{1}{m_i+m_a}\}$
$\iota_a = 1 - \exp\{-\frac{1}{m_y-m_i-m_a}\}$
$\iota_s = (1-r)(1 - \exp\{\frac{1}{m_s-m_i-m_a}\})$
μ - estimated

Similarly as in the Example, we assumed equal infectiousness β_t for all the infectious states I_a, I_p, I_s, varying in time:

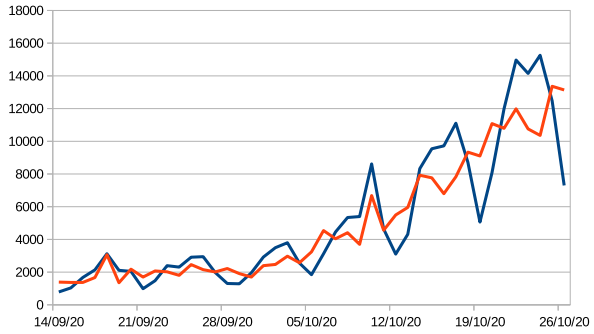
$$\beta_t = \beta c_t p_t \tag{3}$$

where β is an (estimated) constant, c_t is the contact reduction (with $c_0 = 1$) and p_t is the reduction caused by personal protection.

The "removal" are η and θ, which both are also time-varying. Reflecting the weekly pattern of reporting, we assume

$$\theta_t = \phi_{t+1}\theta, \qquad \eta_t = \phi_{t+1}\eta$$

where ϕ_t is the adjustment for the day of the week, having different values for different weekdays and being computed in a standard way from $\ln R_t$ where R_t is the total number of positive cases.[1] The following graph illustrates de-seasoning we made:



We use three data series as observations: the daily numbers of detected cases, distinguished between symptomatic (S), asymptomatic (A) and daily numbers of dead (D), i.e.

$$Y_t = \begin{bmatrix} A_t \\ S_t \\ D_t \end{bmatrix}.$$

As for the transformation matrix F, we put $F = (f^{ij})$ where $f^{1i}$is one/zero if the state i is/is not *detected asymptomatic on* the Figure above, and similarly with f^{2i} – *detected symptomatic* and f^{3i} – *dead*.

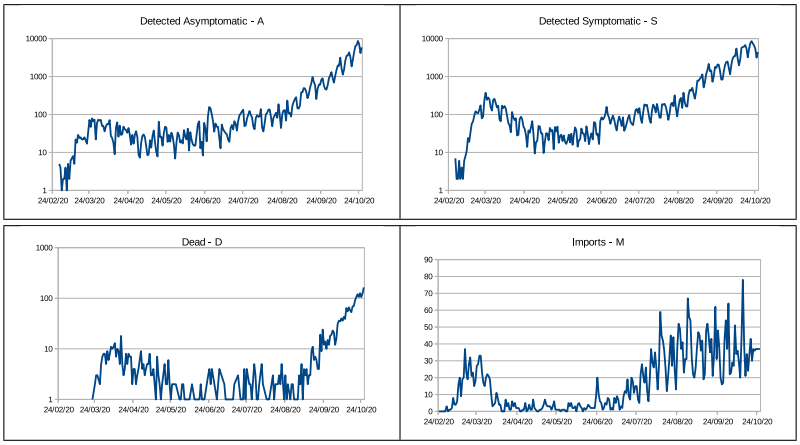
Assuming the imports only to the state E, we take

$$I_t = r_t M_{t+8}$$

[1] In particular we assume $\Delta R_t = \phi_t \Delta r_t$ where r is a "trend" without oscillations and $\prod_{i=1}^{7} \phi_i = 1$, which gives $\ln(\Delta R_t) = \ln(\phi_t) + \ln(\Delta r_t)$ and $\sum_{i=1}^{7} \ln \phi_i = 0$. Assuming r to be locally linear, we estimate $\ln \phi_t = \frac{1}{4}\sum_{i=1}^{4}\left(\ln(\Delta R_{t-7i}) - \frac{1}{7}\sum_{j=-3}^{3}\ln(\Delta R_{t-7i+j})\right) - \frac{1}{18}\sum_{i=1}^{28}\ln(\Delta R_{t-i})$

where M_t is the number of detected with the indicated infection abroad and r_t is a multiplication factor. We took $r_t = \iota$ for $t < 30$ (the first month), where ι is an unknown parameter; this allowed us to reflect the excess numbers of dead in comparison with the detected, which suggests that many of the cases remained unnoticed in the beginning of the pandemics. For the next months of the pandemics, we took $r_t = \frac{1}{1-\alpha}$ to reflect the fact that the fraction α of imports will remain asymptomatic.

The following graphs show the time series we mentioned.



To compute c_t and p_t we used [11] which is a longitudinal study, inquiring a panel of 3000 respondents about their (weekly) intense contacts, observance of several personal protection measures (see the Table below), and some other variables. Using this, we compute $c_t = \frac{C_{t-\Delta}}{C_0}$ where C_t is the average reported number of contacts people had at a given time (the weekly reponses values are linearly interpolated assuming the responded values on Wednesdays) and Δ is an unknown parameter, reflecting our lack of knowledge concerning a delay.

With determining the level of personal protection p_t, the situation is more complicated, as the study monitors observance of several protective measures. However, if we assume that the i-th measure reduces the probability of infection by λ_i, we get that, denoting π_t^i the average observance of the i-th measure among the respondents at t, that the average reduction brought by the the measure will be $(1 - \pi_t^i) \times 1 + \pi_t^i(1 - \lambda^i) = 1 - \pi_t^i \lambda^i$. This gives total reduction

$$p_t \stackrel{\text{def}}{=} \prod_{i=1}^{q} (1 - \pi_t^i \lambda^i).$$

where q is the number of measures. Unfortunately, λ_i are unknown and their estimation would bring a serious danger of over-fitting and/or co-linearity (series $\pi_t^1, \ldots, \pi_t^q$ are almost perfectly co-related). To overcome this difficulty, we applied factor analysis to $(C_t, \pi_t^1, \ldots, \pi_t^q)$ on the respondent level, treating the responses in different times as separate observations. As a result, we extracted two main factors as shown in the following Table

f	g	Value
0.563	0.352	Avoiding caughing people (yes or no)
0.315	0.669	Avoiding crowded places
0.269	0.454	Wearing a mask or a respirator
0.394	0.610	Restricting physical contact with people
0.539	0.095	Using desinfection
0.526	0.295	Avoiding people being in contact with an infected
0.081	0.734	Avoiding public transport
0.417	0.100	Taking vitamines
0.640	0.201	Avoiding touching nose and eyes
0.565	0.261	Extra hygiene
0.661	0.121	Washing hands after coughing
0.670	−0.244	Washing hands after using public transport.
0.084	−0.517	C_t

It can be seen that, while the first factor speaks more about contacts, the second one concerns personal protection, lacking connection with C. Being interested in the protection, we approximate

$$\pi_t^i \doteq \overline{\pi}_t^i + \nu_t f_t$$

where $\overline{\pi}_t^i$ is the average of π_t^i over time and respondents, ν_t is a constant and f_t is average of the second factor over respondents at t. Having that. we could approximate

$$p_t \doteq \prod_{i=1}^{q}(1-\lambda^i(\overline{\pi}_t^i+\nu_t f_t)) = \exp\left\{\sum_{i=1}^{q}\ln(1-\lambda^i(\overline{\pi}_t^i+\nu_t f_t))\right\}$$
$$\doteq \exp\left\{-\sum_{i=1}^{q}\lambda^i(\overline{\pi}_t^i+\nu_t f_t)\right\} = \omega_0\exp\{-\omega_1 f_t\}$$

where $\omega_0, \omega_1 \geq 0$. Consequently, we take

$$\beta_t = bc_t\exp\{-\omega_1 f_t\}, \qquad b = \beta\omega_0.$$

Not assuming additional errors of observations, we $\Gamma_t \equiv 0$.

For estimation, we used Weighted least squares with weights

$$U_t = \operatorname{diag}\left(\frac{1}{\max(\overline{r}_t, 20)}, \frac{1}{\max(\overline{r}_t, 20)}, \frac{1}{\max(\overline{d}_t, 5)}\right)$$

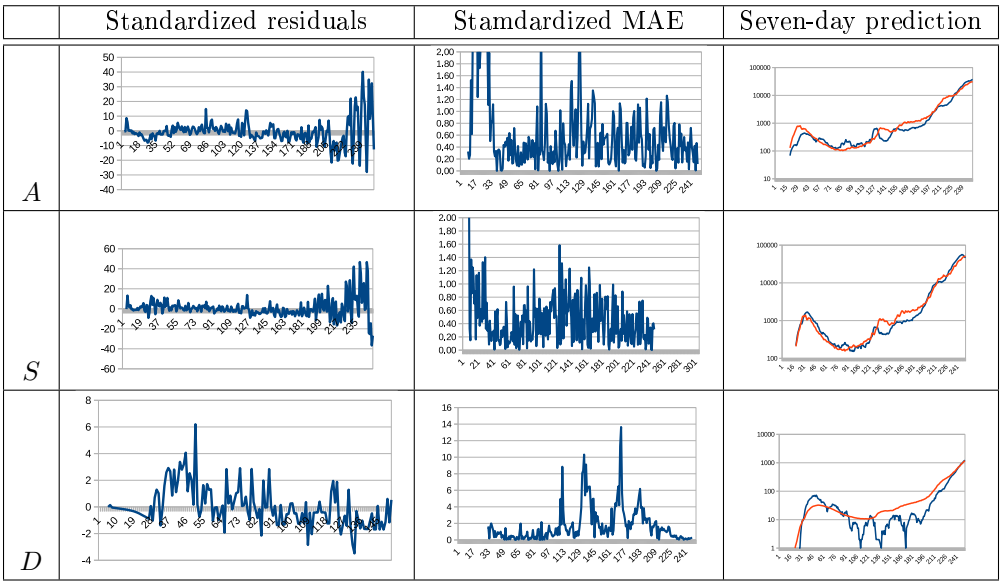
where $\overline{r}_t$ is the weekly average of reported positive over past week and $\overline{d}_t$ is the weekly average of dead.

The results of the estimation, based on data from February 24th to November 5th, 2020, can be seen in the following Table:

	Estimate	Std. Error	z	Significance
ι	0	0.204	0	1^{***}
b_0	0	0.302	0	1^{***}
ω_1	2.30022	1.004	2.291	0.022^{*}
Δ	5.14679	0.314	16.39	0^{***}
μ	0.00198165	0	5.722	0^{***}
θ	0.0347199	0	257.228	0^{***}
η	0.644159	0.005	142.484	0^{***}

Yet the standard errors and significance are only indicative here (we did note verify regularity conditions), the result suggest a good fit.

The following graphs show standardized residuals, standardized mean absolute errors of a one day ahead prediction and the predictions of weekly amounts. The red lines stand for predictions, the blue ones for actual data.



The residuals are standardized in the sense that the actual residual is divided by the standard deviation of the one-day ahead prediction (its square is found on the diagonal of the $V_{t|t-1}$). The MAEs are standardized divided by the weekly averages, i.e. they estimate in fact the MAEs of the relative increments. The predictions are in-sample in the sense that they use parameters originated from the estimation over the whole period; however, the predictions are always based only on the data available at the moment of prediction.

These results clearly show limitations of the present simple parametrization of the model. First of all, the "standardized" residuals are far from having unit variance which means that the actual variance of the observations is much bigger than that predicted by the model, the problem being more severe with the reported infections and less severe with deaths. It is clear that assuming a non-zero Γ_t (i.e. observation errors), can make the variances right; then, however, the prediction errors increase; resolving this puzzle is a topic for the future research.

It is also clear from the graphs that, the model tends to over-estlimate reported infections between the individual waves. This can be naturally attributed to better tracing when numbers are low. In addition, asymptomatic numbers are over-estimated even during the first wave. A natural solution here is to assume a variable rates θ, which was proved to increase the prediction power,[2] but it can bring a danger of over-fitting.

8 Conclusion

We presented a stochastic epidemic model, formulated its basic properties and suggested way of its estimation, all demonstrated by a real-life example. They are, however, things to be done,

[2] In the Situation report 44 by BISOP , for instance, θ_t was approximated by a piece-wise linear function with kinks at 24/02/20, 16/03/20, 26/04/20. 04/07/20, 06/08/20, 16/09/20 and 27/10/20 and taking values $\theta_0, \dots, \theta_6$ at these points. We got the following parameters by ML estimation:

in the first place formulation of regularity conditions which would assure desirable. asymptotic properties. Another issue to deal with is the underestimation of the variances of X and or Y; the most probable reason for this is that the actual infection is not Poisson but rather the arrival of new cases is stochastically dependent. However, even as it is, it can be immediately used for statistically correct modeling of an epidemics.

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	Estimate	Std. Error	z	Significance
ι	9.96795	0.801	12.441	0***
b	0.618509	0.035	17.539	0***
α	0.549047	0.026	21.402	0***
ω_1	0.550946	0.061	8.986	0***
θ_0	0.0194497	0.015	1.317	0.0939*
θ_1	0.00402584	0.003	1.318	0.0937*
θ_2	0.0204434	0.006	3.346	0.0004***
θ_3	0.0260418	0.004	5.789	0***
θ_4	0.0248783	0.004	6.784	0***
θ_5	0.00966628	0.001	16.701	0***
θ_6	0.0221601	0.001	14.897	0***
η	0.624971	0.007	89.31	0***

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