SEIR Filter: A Stochastic Model of Epidemics

Martin Šmíd,[1][2] Luděk Berec,[3] Aleš Antonín Kuběna,[1] René Levínský,[4]
Jan Trnka,[5] Vít Tuček,[6] Milan Zajíček[1]

Abstract

There are many epidemiological models at hand to cope with the present pandemic; it is, however, difficult to calibrate these models when data are noisy, partial or observed only indirectly. It is also difficult to distinguish relevant data from noise, and to distinguish the impact of individual determinants of the epidemic.

In mathematical statistics, the tools to handle all of these phenomena exist; however, they are seldom used for epidemiological models. The goal of this paper is to start filling this gap by proposing a general stochastic epidemiological model, which we call SEIR Filter.

Technically our model is a heterogeneous partially observable vector auto-regression model, in which we are able to express closed form formulas for the distribution of compartments and observations, so both maximum likelihood and least square estimators are analytically tractable. We give conditions for vanishing, explosion and stationary behaviour of the epidemic and we are able to express a closed form formula for reproduction number.

Finally, we present several examples of the model's application. We construct an estimate age-cohort model of the COVID-19 pandemic in the Czech Republic. To demonstrate the strengths of the model, we employ it to analyse and compare three vaccination scenarios.

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[1]Czech Academy of Sciences, Institute of Information Theory and Automation, Pod Vodárenskou věží 4, 18200 Praha 8, Czech Republic

[2]Corresponding author, email: smid@utia.cas.cz

[3]Centre for Mathematical Biology, Institute of Mathematics, Faculty of Science, University of South Bohemia, Branišovská 1760, 37005 České Budějovice, Czech Republic

[4]CERGE-EI, Politických vězňů 7, 11121 Praha 1, Czech Republic

[5]Department of Biochemistry, Cell and Molecular Biology, Third Faculty of Medicine, Charles University, Ruská 87, 100 00 Praha 10, Czech Republic

[6]Department of Mathematics, University of Zagreb, Bijenička 30, 10000 Zagreb, Croatia

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1. Introduction

Whereas fundamental rationale behind mainstream epidemic models, dating back to the works of Ross [25] and Kermack and McKendrick [14] about a century ago, is relatively easy to comprehend, problems arise when we aim to parameterize such models to data for specific epidemics. The source of troubles is not only absence of clear and consistent protocols for data collection, but also factors that can never be fully intercepted, such as degrees of compliance in many adopted interventions. This is also the case for COVID-19, despite the somewhat paradoxical observation, that we now arguably have the best data on any epidemic in the history of mankind.

In regards to COVID-19, many issues arise requiring careful modelling, such as quantifying the effects of various non-pharmaceutical interventions [10, 4, 16]. Two questions are particularly interesting in this context: how effective testing and tracing has to be to counteract an intervention relaxation [19] and how stringent the interventions should be in order to compensate for partial non-compliance of the population [1]. To answer these questions, models for correctly predicting uncertainty are of vital importance.

Upon construction of realistic models, various data issues have to be coped with, starting from noise (caused both by the epidemic process itself and the data collection), insignificance (difficulty to distinguish impacts of factors from random fluctuations) or co-linearity (difficulty to distinguish two parameters with sufficient certainty). The most severe difficulty however, is that relevant data (e.g. the numbers of infectious individuals) are hidden and observed only indirectly (through the numbers of confirmed cases, for instance). Obviously, once any of these phenomena are handled insufficiently, models can provide incorrect policy recommendations.

Mathematical statistics has developed tools to handle these issues. However, to our best knowledge, there is no work systematically doing so for compartmental epidemic models. The goal of this paper is to make some progress in this area by proposing a general stochastic epidemic modelling framework.

Stochastic epidemic models do obviously exist [3]. Apart from agent-based models [2], they are commonly formulated as Markov chains [9] or stochastic differential equations [7]. However, many of these studies are

theoretical, examining behavior models they construct without providing any link to real data and hence an estimation procedure. On the other hand, many other studies use elaborate filtering methods to estimate parameters of their epidemic models, but these models are largely deterministic. Their statistical models serve just as tool on the way to understand specific epidemics [24, 27]. Here, we bridge these two approaches, precisely formulating and analyzing a general stochastic epidemic model, discussing its highly practical implications, as well as providing an estimation procedure exemplified on the COVID-19 epidemic in the Czech Republic. Due to practical reasons, we develop our model as discrete in both time and state space. Although applicable to a wide class of compartment settings, we call our framework the SEIR Filter.

Models close to our model were developed by [15, 23]. To estimate their parameters, the authors used Monte Carlo simulations to evaluate likelihood functions. On contrary to these (and many other) studies, our framework enables model parameters to be estimated in a straightforward way, since the exact likelihood (or other estimating) function can be derived. This approach makes faster and more reliable parameter estimation possible. In addition to the tractability of the estimating function, our model allows for closed form formulas for expected future compartment sizes and the reproduction number. In addition, we provide simple criteria for vanishing and explosion of the epidemic, as well as for bounds that limit expected epidemic sizes given stationary imports. As we demonstrate by stylized examples, many applications of the model are possible, including derivation of implicit formulas regarding compensation measures to intervention relaxation or non-compliance, and of optimal control strategies; in this way, we touch, yet not solve, the questions mentioned above.

From the mathematical statistics perspective, we model the epidemic by a partially observed inhomogeneous heteroskedastic vector autoregression process. In line with the usual practice [8], we assume over-dispersed probability distributions both for the infections and the between-compartmental transitions; thanks to this, we are able to handle realistically not only point estimators, but also the uncertainty associated with them. Moreover, having a standard statistical output of the parameters estimation, we are able to answer questions concerning significance (via P-values[7]), possible co-linearity

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[7]It should be, however, noted that we do not prove asymptotic properties of the

(via the estimator's correlations) and the hidden compartments states (via the estimate of state-space distribution).

To demonstrate usefulness of our framework, we apply it to the actual COVID-19 epidemic in the Czech Republic. In particular, we consider four age cohorts, for each of which we model incidence, admission to– and release from hospitals and deaths. To estimate the model's parameters, we use several partially overlapping datasets; the ability to create statistically correct estimates based on such heterogeneous datasets is, in our opinion, one of the greatest contributions of our approach. We demonstrate both in-sample and out-of-sample prediction ability of our model and, as an example of its possible use, we compare three strategies of vaccination: no vaccination, vaccination without preferences and the old-first strategy.

The paper is organized as follows. After a rigorous probabilistic formulation of the model (Section 2), we discuss its basic probabilistic properties (Section 3), its autonomous sub-models and the reproduction number (Section 4), and its asymptotic properties (Section 5). Next we introduce an age-structured version of the model (Section 6) and suggest a way to optimally control the epidemic (Section 7). Further, we discuss estimation of model parameters (Section 8). Finally, we apply our model to the COVID-19 epidemic in the Czech Republic (Section 9) and demonstrate possible model applications by comparing three possible vaccination scenarios (Section 10). Section 11 then concludes the paper.

2. Model Definition

Assume a population of size $s \in \mathbb{N}$, where s is large. Each individual of the population is either susceptible, or finds himself in one of the compartments $S_1, \ldots, S_k$. Let $I_t \in \mathbb{N}_0^k$, $t \in \mathbb{N}_0^+$, be a possibly hidden external inflow (import) of individuals into the compartments and let $Z_t \in \mathbb{R}^p$, $t \in \mathbb{N}_0$, be an observed exogenous process.

For any $t \in \mathbb{N}_0$, let $X_t = (X_t^1, \ldots, X_t^k) \in \mathbb{N}_0^k, t \in \mathbb{N}_0$, be a possibly hidden stochastic process of the compartment sizes 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 errors vector.

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estimators.

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Denote $(\mathcal{F}_t)_{t\geq 0}$ and $(\mathcal{G}_t)_{t\geq 0}$ the filtrations induced by (X, Y, I, Z) and (Y, Z), respectively. These filtrations may be seen as information flows, $(\mathcal{F}_t)_{t\geq 0}$ representing time evolution of all the information and $(\mathcal{G}_t)_{t\geq 0}$ representing the evolution of the observable observable. Recall that a random variable ξ is measurable with respect to σ-algebra $\sigma(\Xi)$ generated by a random element Ξ if and only if there exists a measurable function Φ such that $\xi = \Phi(\Xi)$. Thus, saying that ξ is $\mathcal{G}_t$-measurable (abbreviated as $\xi \in \mathcal{G}_t$) means, that ξ may be devised from the values of (Y, Z) up to t.

We assume that

$$\mathbb{E}(\epsilon_{t+1}|\mathcal{F}_t) = 0, \qquad \mathrm{var}(\epsilon_{t+1}|\mathcal{F}_t) = \mathrm{diag}(\Gamma_t(X_t, X_t^2)), \qquad t \in \mathbb{N}_0,$$

where Γ_t is a $\mathcal{G}_t$-measurable affine linear function (i.e. $\Gamma_t(x, y) = \gamma_{t,0} + \gamma_{t,1}x + \gamma_{t,2}y$ for some $\gamma_{t,0} \in \mathbb{R}^k$ and $\gamma_{t,1}, \gamma_{t,2} \in \mathbb{R}^{k\times k}$ where all $\gamma_{t,0}, \gamma_{t,1}$ and $\gamma_{t,2}$ are $\mathcal{G}_t$-measurable).

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 individuals such that $N_{t+1}|\mathcal{F}_t \sim \mathrm{CPo}(A_t X_t, L)$ where $A_t = (\alpha_t^{ij})_{1\leq i,j\leq k} \in \mathcal{G}_t$ is a random matrix and, for any vector x, $\mathrm{CPo}(x, L)$ stands for a vector of independent Compound Poisson variables with the intensities given by x and the embedded distribution L. Observe that, by basic properties of Compound Poisson distribution,

$$\frac{\mathrm{var}(N_{t+1}^i|\mathcal{F}_t)}{\mathbb{E}(N_{t+1}^i|\mathcal{F}_t)} = v \stackrel{\mathrm{def}}{=} \frac{\mathbb{E}L^2}{\mathbb{E}L}$$

(with $\frac{0}{0} \stackrel{\mathrm{def}}{=} \frac{\mathbb{E}L^2}{\mathbb{E}L}$), $t \geq 0, 1 \leq i \leq k$. Further, for any i, $M_{i,t+1} \in \mathbb{N}_0^k$ such that $M_{i,t+1}^j$ (the j-th component of $M_{i,t+1}$) is the number of individuals who transited from the compartment j to the compartment i between t to $t+1$. Further we assume $M_{i,t+1}$, for any i, to follow Dirichlet Multinomial (DM) conditional distribution with parameters $\left(X_t^i, \frac{p_t^{1,i}}{c_i}, \ldots, \frac{p_t^{k,i}}{c_i}\right)$ where $P_t = (p_t^{ij})_{1\leq i,j\leq k} \in \mathcal{G}_t$ is a "mean" transition matrix and $(c_1, \ldots, c_k)$ are deterministic dispersion parameters.

Remark 1. If we assumed the individuals to change their state according to P_t with the transitions being conditionally independent given $\mathcal{F}_t$, then we

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would get $M_{i,t+1}|\mathcal{F}_t$ as Multinomial with parameters X_t^i and $P_t^{(i)}$ where, for any matrix Σ, $\Sigma^{(i)}$ is its i-th column. In practice, however, the assumption of the conditional independence is unrealistic, as the course of infection differs between individuals and tends to cluster. The standard way of coping with this situation is using DM distribution.

The flow of individuals between states is illustrated by the following chart.
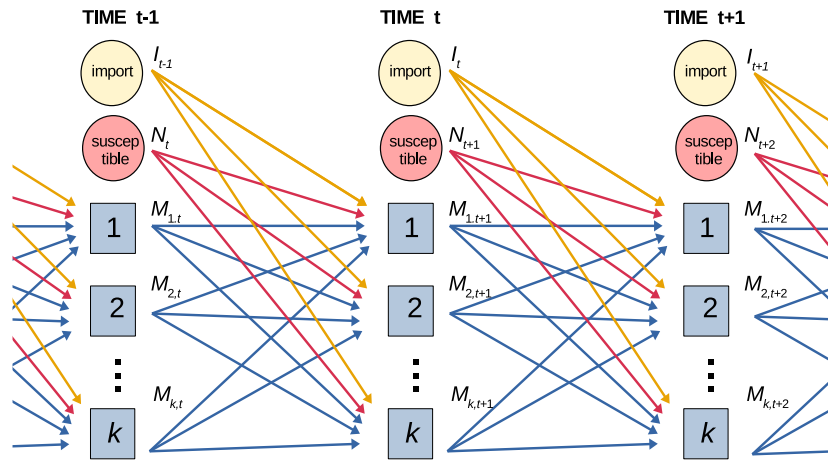
Finally, we assume $N_{t+1}, M_{1,t+1}, \ldots, M_{k,t+1}, \epsilon_{t+1}$ to be mutually conditionally independent given $\mathcal{F}_t$ (which, in words, means that all dependence between the inflows, the transitions and the observations can be explained by the state of the system at t). Consequently,

$$X_{t+1}|\mathcal{F}_t \sim \bigcirc_{1\leq i\leq k}\mathrm{DM}\left(X_t^i, \frac{1}{c_i}P_t^{(i)}\right) \circ \mathrm{CPo}(A_tX_t, L) \circ \delta(I_t),$$

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

Remark 2. The variability of the individual infectiousness may be naturally reflected by the choice of L. To demonstrate it, assume that only a single compartment (labeled I) is infectious, that all new infections fall to a single compartment (labeled E), and that the number of risk contacts of each infectious individual is Poisson. Let $t \geq 0$ and denote $N_{t+1,i}$ the number of the infections, caused by the i-th individual at t.

If the intensity of the contact distribution and the contagion probability were the same for all individuals, and equal to c and p, respectively, then it

would be $N_{t+1,i} \sim \text{Po}(cq)$, $i \in \mathbb{N}$; consequently, $N_{t+1}^E \sim \text{Po}(\lambda X_t^I)$, $\lambda = cq$, so we may put $\alpha_t^{EA} = \lambda$ and $L = \delta_1$, having $\mathbb{E}(N_{t+1}^E|\mathcal{F}_t) = \text{var}(N_{t+1}^E|\mathcal{F}_t) = \lambda X_t$.

Now consider a more realistic situation in which the infectiousness randomly varies between individuals. A standard way of modeling this situation is assuming, for each i, that the intensity λ_i of $N_{t+1,i}$ is chosen from Gamma distribution, implying that $N_{t+1,i}$ is negative binomial (see [28]). In particular, once $\lambda_i \sim \Gamma(k, \theta)$, and $N_{t+1,i}|\lambda_i = \text{Po}(\lambda_i)$, we are getting that $N_{t+1,i} \sim \text{NB}(k, p)$, $p = \frac{\theta}{1+\theta}$, with

$$\mathbb{E}N_{t+1,i} = \kappa \stackrel{\text{def}}{=} \theta k, \qquad \text{var}(N_{t+1,i}) = \kappa v, \qquad v = 1 + \frac{\kappa}{k}. \qquad (1)$$

As the Negative Binomial distribution can be represented by a Compound Poisson one and as the sum of independent Compound Poisson distributions is Compound Poisson, we have that N_{t+1}^E is Compound Poisson[8] and it follows from (1) that

$$\mathbb{E}(N_{t+1}^E|\mathcal{F}_t) = \kappa X_t, \qquad \text{var}(N_{t+1}^E|\mathcal{F}_t) = \kappa X_t v$$

Remark 3. The authors of [6] claim that the total number T of individuals infected by a single COVID-infectious individual is $T \sim \text{NB}(K, P)$ where $K \doteq 0.1$ and P is such that $\mathbb{E}T = R_0$ where R_0 is the basic reproduction number. As R_0 of COVID-19 is generally assumed to be around 2.5 and $\mathbb{E}T = K\frac{P}{1-P}$, it follows that $P \doteq \frac{25}{26}$. Consequently, the variance-to-mean ratio is $\tilde{v} \stackrel{\text{def}}{=} \frac{1}{1-P} \doteq 26$. Assuming that an individual is infectious for f days and that his contacts are restricted by a factor β, we may, in light of the Compound Poisson reformulation of T, conclude that the number $N_{t+1,i}$ of daily infected individuals is Compound Poisson with $\mathbb{E}N = \frac{\beta}{f}\mathbb{E}T$, $\text{var}(N) = \frac{\beta}{f}\text{var}(T)$, i.e. $N_{t+1,i}$ and consequently N_{t+1}^E has the same variance-to-mean ratio as T, i.e. $v = \tilde{v} \doteq 26$.

Remark 4. For any $x \in \mathbb{N}_0, c \geq 0$ and $p \in [0,1]^k$ such that $\sum_{i=1}^k p^i = 1$, we have

$$\mathbb{E}\left(\text{DM}(x, \frac{p}{c})\right) = px = \mathbb{E}\left(\text{Multinomial}(x, p)\right)$$

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[8]In particular, $N_{t+1,i} = \text{CPo}(k\ln(1+\theta), \text{Log}(p))$, where Log is the Logarithmic distribution ([28]). Thus, $N_{t+1}^E \sim \text{CPo}(X_t^I k\ln(1+\theta), \text{Log}(p))$ so we may put $\alpha_t^{EA} = k\ln(1+\theta)$, $L = \text{Log}(p)$.

and

$$\mathrm{var}\left(\mathrm{DM}(x, \tfrac{p}{c})\right) = [\mathrm{diag}(p) - pp^T]\frac{x+c}{1+c}x = \mathrm{var}(\mathrm{Multinomial}(x, p))\frac{x+c}{1+c},$$

from which it is clear that $\mathrm{DM}(x, \tfrac{p}{c}) \to \mathrm{Multinomial}(x, p)$ as $c \to \infty$ and that the deviation of the Multinomial variance matrix grows with decreasing c.

3. Model Properties

By probability calculus, we get that

$$\mathbb{E}(X_{t+1}|\mathcal{F}_t) = \mathbb{E}(T_t X_t + I_t|\mathcal{F}_t) = \mathbb{E}\left(\sum_{i=1}^{k} M_{t+1,i} + N_{t+1} + I_t \,\middle|\, \mathcal{F}_t\right)$$

$$\sum_{i=1}^{k} P_t^{(i)} X_t^i + (\mathbb{E}L)A_t X_t + I_t = T_t X_t + I_t,$$

where, for any matrix Σ, $\Sigma^{(i)}$ denotes its i-th column, and

$$T_t \stackrel{\mathrm{def}}{=} P_t + B_t, \qquad B_t = (\beta_t^{i,j})_{1 \le i,j \le k} \stackrel{\mathrm{def}}{=} (\mathbb{E}L)A_t, \qquad t \ge 0. \qquad (2)$$

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 Σ, $\Sigma_{s,t} \stackrel{\mathrm{def}}{=} \Sigma_t \times \cdots \times \Sigma_s$ with $\Sigma_{s,s-1} \stackrel{\mathrm{def}}{=} E$ where E is the identity matrix.

In the special case that

$$B_\tau \equiv B_s, \qquad P_\tau \equiv P_s, \qquad s \le \tau \le t, \qquad (3)$$

we have

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

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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

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

As for variance, we have

$$\mathrm{var}(X_{t+1}|\mathcal{F}_t) = \sum_{i=1}^{k} \mathrm{var}(M_{t+1,i}|\mathcal{F}_t) + \mathrm{var}(N_{t+1}|\mathcal{F}_t) + \mathrm{var}(I_t|\mathcal{F}_t)$$

$$= \sum_{1 \le i \le m} \mathrm{var}\left(\mathrm{DM}\left(X_t^i, \frac{1}{c_i}P_t^{(i)}\right)\right) + \mathrm{var}(\mathrm{CPo}(A_t X_t, L)) + 0$$

$$= \sum_{i \le t \le m} \frac{X_t^i + c_i}{1 + c_i} X_t^i [\mathrm{diag}(P_t^{(i)}) - P_t^{(i)}(P_t^{(i)})^T] + \mathrm{diag}(v B_t X)$$

$$= \Lambda_t(X_t, X_t^2)$$

where

$$\Lambda_t(x, y) \stackrel{\mathrm{def}}{=} \sum_{i=1}^{k} \frac{y^i + x^i c_i}{1 + c_i} [\mathrm{diag}(P_t^{(i)}) - P_t^{(i)}(P_t^{(i)})^T] + \mathrm{diag}(v B_t x)$$

(note that Λ_t is linear in x, y). Consequently,

$$\mathbb{E}\left[\begin{matrix} X_{t+1} \\ Y_{t+1} \end{matrix}\middle| \mathcal{F}_t\right] = \begin{bmatrix} E \\ F \end{bmatrix}(T_t X_t + I_t), \tag{4}$$

$$\mathrm{var}\left(\begin{matrix} X_{t+1} \\ Y_{t+1} \end{matrix}\middle| \mathcal{F}_t\right) = \begin{bmatrix} E \\ F \end{bmatrix}\Lambda_t(X_t, X_t^2)\begin{bmatrix} E \\ F \end{bmatrix}^T + \mathrm{diag}\left(\begin{matrix} 0_k \\ \Gamma_t(X_t, X_t^2) \end{matrix}\right), \qquad t \ge 0.$$

4. Sub-epidemics and Reproduction Number

We say that the subset of compartments $D = \{s_1, \ldots, s_m\}$ 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

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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 $\{1, \ldots, m\}$ 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 $(1, \ldots, m)$ and, for any matrix $A \in \mathbb{R}^{k \times k}$, denote $\overline{A}$ its restriction to $(1, \ldots, m) \times (1, \ldots, m)$.

Observe that $\overline{X}$ follows a slightly modified version of our model, namely

$$\overline{X}_{t+1} \big| \mathcal{F}_t \sim \bigcirc_{1 \leq i \leq m} \mathrm{DM}^-\big(\overline{X}_t^i, \frac{1}{c}\overline{P}_t^{(i)}, c\big) \circ \mathrm{CPo}(\overline{B}_t \overline{X}_t, L) \circ \delta(\overline{I}_t).$$

where, for any $x \in \mathbb{N}_0, \alpha \in \mathbb{R}_+^m$ and $c > 0$, $\mathrm{DM}^-(x, \alpha, c)$ is the marginal distribution of the first m components of $\mathrm{DM}\left(x, \left[\frac{\alpha}{c - \sum_{i=1}^m \alpha^i}\right]\right)$ (by the aggregation property of DM).

For any t, we define the reproduction number r_t (of a subepidemic $\{1, \ldots, m\}$) 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 (recall that $\overline{P}_{t,s} = \overline{P}_t \times \cdots \times \overline{P}_s$). 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 $\overline{B}_\tau \equiv \overline{B}_{t-1}, \overline{P}_\tau \equiv \overline{P}_{t-1}, \tau \geq t$, with $\rho(\overline{P}_{t-1}) < 1$ where ρ is the spectral radius, the formula simplifies to

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

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Note that, once $\overline{N}$ and/or $\overline{I}$ are possibly not observed, there could be difficulties computing $\mathbb{E}(\pi_t | \mathcal{G}_{t-1})$. Although the estimate $\mathbb{E}(\pi_t | \mathcal{G}_{t-1}) \doteq \nu(\overline{B}_{t-1}\mathbb{E}(\overline{X}_{t-1}|\mathcal{G}_{t-1}) + \mathbb{E}(\overline{I}_{t-1}|\mathcal{G}_{t-1}))$ 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 and labeled E), in which case $\pi_t \equiv (1, 0, \ldots, 0)^T$.

5. Asymptotic Behavior

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

Proposition 5. *(i) If $\overline{T}_t \leq S$ component-wise, where S is deterministic with $\sigma \overset{def}{=} \rho(S) < 1$, and if $\mathbb{E}\overline{I}_t = o(t^{-\alpha})$ for some $\alpha > 0$, then $\overline{X}_t \to 0$ almost surely. 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

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

Proof. (i) We have

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

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

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,

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

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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.

$\square$

Example 6. 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, which includes the recovered, the dead, and the infectious isolated. We index the states by e, a, p, s, r. For simplicity we assume $v = 1$ which means that the new infections follow a Poisson distribution rather than a Compound Poisson one. All the infectious states are equally infectious, i.e. $\beta_t^{ex} = \beta_t$, $x \in \{a, p, s\}$, where β is a $\mathcal{G}_t$-adapted process. The probability that the exposed transits to $\{a, p\}$ 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 finding herself in state E, I_a, I_p or I_s is isolated is θ_t for some $\mathcal{G}_t$-adapted process θ^i. The situation is illustrated on the following Figure:
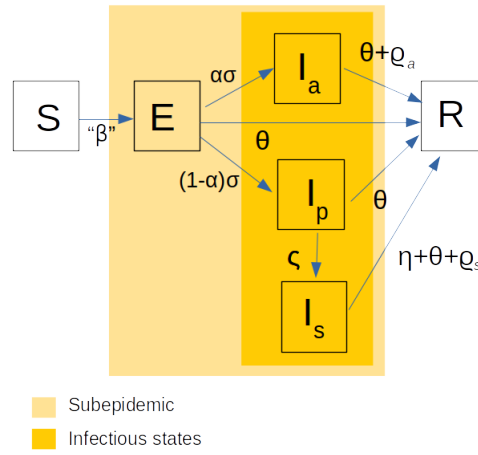
If we neglect (small) joint probabilities of natural exits from the infectious states and the isolations, we get

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

$$
B_t = \begin{bmatrix}
0 & \beta_t & \beta_t & \beta_t & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{bmatrix}
$$

Clearly, we can put $m = 4$ (the first four states form a sub-epidemic), getting

$$
\overline{T}_t = Q + \beta_t C - \operatorname{diag}(\theta_t, \theta_t, \theta_t, \theta_t),
$$

$$
Q = \begin{bmatrix}
1 - \sigma & 0 & 0 & 0 \\
\alpha\sigma & 1 - \varrho_a & 0 & 0 \\
(1 - \alpha)\sigma & 0 & 1 - \varsigma & 0 \\
0 & 0 & \varsigma & 1 - \varrho_s - \eta
\end{bmatrix}, \qquad
C = \begin{bmatrix}
0 & 1 & 1 & 1 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0
\end{bmatrix}.
$$

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

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

Example 7. Assume that a fraction ν of the population is non-compliant, which means that, once a restriction on social contacts is imposed, they obey 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 imposed, the compliant individuals restrict their opportunities to contacts by ϕ, while 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 $\beta_t^{1i} = \beta c_i$, $i \in \{1, 2\}$, 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 $\rho(C) = g$.

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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, $\phi(0) = \phi_0 \overset{\text{def}}{=} \sqrt{\frac{r-1+\varrho}{\beta}}$. 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, by the first-order Taylor expansion at $\nu = 0$, we get

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

roughly holding for ν close to zero.

6. Cohort Model

In the present Section, we assume the population is split into r (age) cohorts of sizes $s_1, \ldots, s_r$, $s_1 + \cdots + s_r = s$. The members of each cohort may be either susceptible or belong to one of κ analogous compartments. Naturally assuming that individuals do not migrate between cohorts, we get the overall transition matrix as

$$P_t = \begin{bmatrix} P_t^1 & 0 & \cdots & 0 \\ 0 & P_t^2 & \cdots & 0 \\ \vdots & \vdots & \ddots & 0 \\ 0 & 0 & \cdots & P_t^r \end{bmatrix}$$

where P_t^i are $\kappa \times \kappa$ cohort transition matrices, $1 \leq i \leq \kappa$, $t \geq 0$. Note that once there are dispersion parameters $c_1^i, \ldots, c_\kappa^i$ associated with each matrix P_t^i (meaning that, once the j-th compartment of the i-th cohort is of size x, the transfers from the cohort to the cohort's compartments follow $\mathrm{DM}(x, \frac{(P^i)^{(j)}}{c_i^j}))$, the dispersion parameters of the overall model are $(c_1^1, \ldots, c_\kappa^1, c_1^2, \ldots c_\kappa^2, c_1^3, \ldots \ldots, c_\kappa^r)$.

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We assume that the contagions can happen across cohorts and that the probability of contagion, i.e. the transfer of a susceptible individual to the i-th compartment of the p-th cohort, upon a risk contact with a member of the j-th compartment of the q-th cohort does not depend on p or q, being equal to ϖ_t^{ij}. Further we assume that, on average, a member of the p-th cohort has ν^{pq} risk contacts with the q-th cohort, assuming that the number of contacts with the infectious compartments (those with non-zero $\varpi_t^{i\bullet}$) is equal. Under these assumptions, the probability of a transfer of a susceptible individual from cohort p into the i-th compartment is roughly $e_t^{pi} \overset{\text{def}}{=} b_p \sum_{q=1}^r \sum_{j=1}^k \nu^{pq} \varpi_t^{ij} \frac{X_{tq}^i}{s_q}$, where X_{tq}^i is the size of the i-th compartment of cohort q and b is a constant. Consequently, the total number of the infections in the i-th compartment of the p-th cohort will be $s_p e_t^{pi}$, which gives

$$B_t = Q \otimes C_t$$

where

$$Q = \begin{bmatrix} b_1 \nu^{11} & b_1 \nu^{12} \frac{s_1}{s_2} & \cdots & b_1 \nu^{1r} \frac{s_1}{s_r} \\ b_2 \nu^{21} \frac{s_2}{s_1} & b_2 \nu^{22} & \cdots & b_2 \nu^{2r} \frac{s_2}{s_r} \\ \vdots & \vdots & \ddots & \vdots \\ b_r \nu^{r1} \frac{s_r}{s_1} & b_r \nu^{r2} \frac{s_r}{s_2} & \cdots & b_r \nu^{rr} \end{bmatrix}, \qquad C_t = \begin{bmatrix} \varpi_t^{11} & \varpi_t^{12} & \cdots & \varpi_t^{1k} \\ \varpi_t^{21} & \varpi_t^{22} & \cdots & \varpi_t^{2k} \\ \vdots & \vdots & \ddots & \vdots \\ \varpi_t^{k1} & \varpi_t^{k2} & \cdots & \varpi_t^{kk} \end{bmatrix}.$$

It may be convenient to re-parametrize the model by multiplying C by a constant and adjusting constants b; in this case, however, the components of C cease to be interpretable as probabilities.

7. Optimal Control of the Epidemic

Assume the settings of Example 6 and assume that X_0 is known. Our aim is to minimize the size of the epidemic at time t given that we are ready to pay a given price c_0. We assume that, to achieve infection rate β, a cost $\gamma(\beta)$ has to be paid where γ is a strictly decreasing convex positive function defined on $(0, \beta_0]$ with $\gamma(\beta_0) = 0$ and $\gamma(0-) = \infty$. Further, to achieve the isolation rate θ_t^i in the i-th compartment, the price $\delta(\theta_t^i, X_t^i) \overset{\text{def}}{=} d\theta_t^i X_t^i$ has to be paid where d is a constant. This reflects the real-life situation in which the cost of global restrictions does not depend on the infection size while the cost of isolation does, for instance through the number of call-center workers involved in tracing.

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Our problem is to find

$$
V_0(X_0, c_0) \stackrel{\text{def}}{=} \inf_{\sum_{\tau=0}^{t-1}[\gamma(\beta_\tau)+\delta(\theta_t^k, X_t^k)+\cdots+\delta(\theta_t^k, X_t^k)] \leq c_0, \beta, 0 \leq \theta \leq q, \theta_\tau \in \mathcal{F}_\tau, \beta_\tau \in \mathcal{F}_\tau, 1 \leq \tau < t} \mathbb{E}(\mathbf{1}'X_t)
$$

where q is the diagonal of Q. The problem may be rewritten by means of Bellman equations

$$
V_\tau(x, c) = \inf_{\gamma(\beta)+d\theta^k x^k+\cdots+d\theta^k x^k+y \leq c, \beta \geq 0, \theta \geq 0, y \geq 0} \mathbb{E}(V_{\tau+1}(J, y)), \qquad 0 \leq \tau < t,
$$

$$
V_t(x, c) = \mathbf{1}'x,
$$

$$
J \sim \mathcal{L}(x, \beta, \theta) \stackrel{\text{def}}{=} \bigcirc_{1 \leq i \leq m} \text{Multinomial}^-(x^i, Q^{(i)} - \Delta_i \theta^i) \circ \text{Po}(\beta Cx)
$$

where Δ_i is the vector with the unit component on the i-th place and zeros otherwise.

Though the final problem is convex,

$$
V_{t-1}(x, c) = \inf_{\gamma(\beta)+d\theta^1 x^1+\cdots+d\theta^m x^m+y \leq c, 0 \leq \theta \leq q, \beta, y \geq 0} \mathbf{1}'(Qx - \text{diag}(\theta)x + \Delta_e \beta \sum_{i \neq e} x^i),
$$

its objective function is not jointly convex in (u, v, x, c), so the convexity of V_{t-1} is not guaranteed. Moreover, as neither the optimal solution nor the expectation of the objective functions of all but the last problem are analytically tractable, it is necessary to resort to approximations. To this end, we can use the sample mean approximation

$$
V_\tau(x, c) \doteq \tilde{V}_\tau(x, c)
$$

where $\tilde{V}_t = V_t$ and, recursively,

$$
V_\tau(x, c) \doteq \tilde{V}_\tau(x, c) \stackrel{\text{def}}{=} \inf_{\gamma(\beta)+d\theta^k x^k+\cdots+d\theta^k x^k+y \leq c, \beta, \theta, y \geq 0} \frac{1}{r} \sum_{i=1}^{r} \tilde{V}_{\tau+1}(J_i, y)
$$

where $J_1, \ldots, J_r$ is an i.i.d. sample from $\mathcal{L}(x, \beta, \theta)$.

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8. Estimation

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

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

$$
W_{s|t} \overset{\text{def}}{=} \text{var}\left(X_s|\mathcal{G}_t\right) = \text{var}\left(\mathbb{E}\left(X_s|\mathcal{F}_{s-1}\right)|\mathcal{G}_t\right) + \mathbb{E}\left(\text{var}\left(X_s|\mathcal{F}_{s-1}\right)|\mathcal{G}_t\right)
$$
$$
= \text{var}\left(T_{s-1}X_{s-1} + I_{s-1}|\mathcal{G}_t\right) + \mathbb{E}\left(\Lambda_{s-1}(X_{s-1}, X_{s-1}^2)|\mathcal{G}_t\right)
$$
$$
= T_{s-1}W_{s-1|t}T_{s-1}^T + 2T_{s-1}\text{cov}(X_{s-1}, I_{s-1}|\mathcal{G}_t) + \text{var}\left(I_{s-1}|\mathcal{G}_t\right)
$$
$$
+ \Lambda_{s-1}(\hat{X}_{s-1|t}, \text{diag}(W_{s-1|t}) + \hat{X}_{s-1|t}^2)
$$

(we have used linearity of Λ_{s-1}, thanks to which $\mathbb{E}(\Lambda_{s-1}(\hat{X}_{s-1|t}, X_{s-1}^2)|\mathcal{G}_t) = \Lambda_{s-1}(\mathbb{E}(X_{s-1}|\mathcal{G}_t), \mathbb{E}(X_{s-1}^2|\mathcal{G}_t))$, and the well known formula $\text{var}(X) = \mathbb{E}X^2 - (\mathbb{E}X)^2$). Consequently,

$$
V_{s|t} \overset{\text{def}}{=} \text{var}\left(\begin{matrix} X_s \\ Y_s \end{matrix}\middle|\mathcal{G}_t\right) = \text{var}\left(\begin{matrix} X_s \\ FX_s + \epsilon_s \end{matrix}\middle|\mathcal{G}_t\right)
$$
$$
= \begin{bmatrix} E \\ F \end{bmatrix} W_{s|t}\begin{bmatrix} E \\ F \end{bmatrix}^T + \text{diag}\left(\begin{matrix} 0_k \\ \Gamma_{s-1}(\hat{X}_{s-1|t}, \text{diag}(W_{s-1|t}) + \hat{X}_{s-1|t}^2) \end{matrix}\right).
$$

Unfortunately, due to the non-Gaussianity, we have analytical formulas for
none of $X_{t|t}$, $I_{t|t}$ and $W_{t|t}$, so we can formulate neither the likelihood function
nor a least square estimate. From the computational point of view, two
equivalent ways to cope with this are using estimates of the conditional
expectation and variance, or normally approximating the residuals. We
choose the latter way here and assume that $\begin{bmatrix} X_{t+1} \\ Y_{t+1} \end{bmatrix}\middle|\mathcal{F}_t$ is normal, with mean
given by (4) and

$$
\text{var}\left(\begin{matrix} X_{t+1} \\ Y_{t+1} \end{matrix}\middle|\mathcal{F}_t\right) = \begin{bmatrix} E \\ F \end{bmatrix}\Lambda_t(X_t \vee 0, X_t^2)\begin{bmatrix} E \\ F \end{bmatrix}^T + \text{diag}\left(\begin{matrix} 0_k \\ \Gamma_t(X_t \vee 0, X_t^2) \end{matrix}\right).
$$

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Moreover, we assume that $I_t \in \mathcal{G}_t, t \geq 0$ (i.e. the import is observable). Given these assumptions, we have, by the well-known formula (see e.g. [5]),

$$\hat{X}_{t|t} = I_{t-1} + \hat{X}_{t|t-1} + K_t \left(Y_t - \hat{Y}_{t|t-1} \right), \qquad K_t \stackrel{\text{def}}{=} \tilde{V}_{t|t-1}^{XY} (\tilde{V}_{t|-1}^{YY})^{-1}$$

$$\text{var}(X_t | \mathcal{G}_t) = \tilde{V}_{t|t-1}^{XX} - \tilde{V}_{t|t-1}^{XY} (\tilde{V}_{t|t-1}^{YY})^{-1} \tilde{V}_{t|t-1}^{YX}$$

where $\tilde{V}_{s|t} \stackrel{\text{def}}{=} \text{var}(\begin{bmatrix} X_s \\ Y_s \end{bmatrix} | \mathcal{G}_t)$ given the normal approximation. Note that K_t may be seen as a conditional version of the Kalman gain matrix.

If $\mathbb{P}[\hat{X}_t < 0]$ is negligible (which is typically true when modeling large epidemics), then we can neglect truncation in the formula for the variance approximate $\tilde{V}_{t|s} \doteq V_{t|s}$. This further gives

$$K_t \doteq W_{t|t-1} F^T D_t^{-1}, \qquad D_t = F W_{t|t-1} F^T + \text{diag}(\Gamma_t \hat{X}_{t|t-1}),$$

$$W_{t|t} \doteq W_{t|t-1} - W_{t|t-1} F^T D_t^{-1} F W_{t|t-1}.$$

Assume that $F = F(\Theta_0)$, $P_t = P_t(\Theta_0)$, $B_t = B_t(\Theta_0)$, $\Gamma_t = \Gamma_t(\Theta_0)$, $I_t = I_t(\Theta_0)$, and $c = c(\Theta_0)$, where $\Theta_0 \in \mathbb{R}^r$ is an unknown parameter. For its estimation, it is possible 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 [12] and [13], respectively. Verifying these conditions for our model is, however, beyond the scope of this introductory study and remains the topic of a future research.

It should be noted that our proof of Proposition 5 is not valid for the approximate model, as $\overline{X}$ is not necessarily positive given the Normal approximation.

9. Application to The COVID Pandemics in the Czech Republic

We applied our model to the data from the Czech Republic epidemic between February 2020 and January 2021. We considered a widely generalized
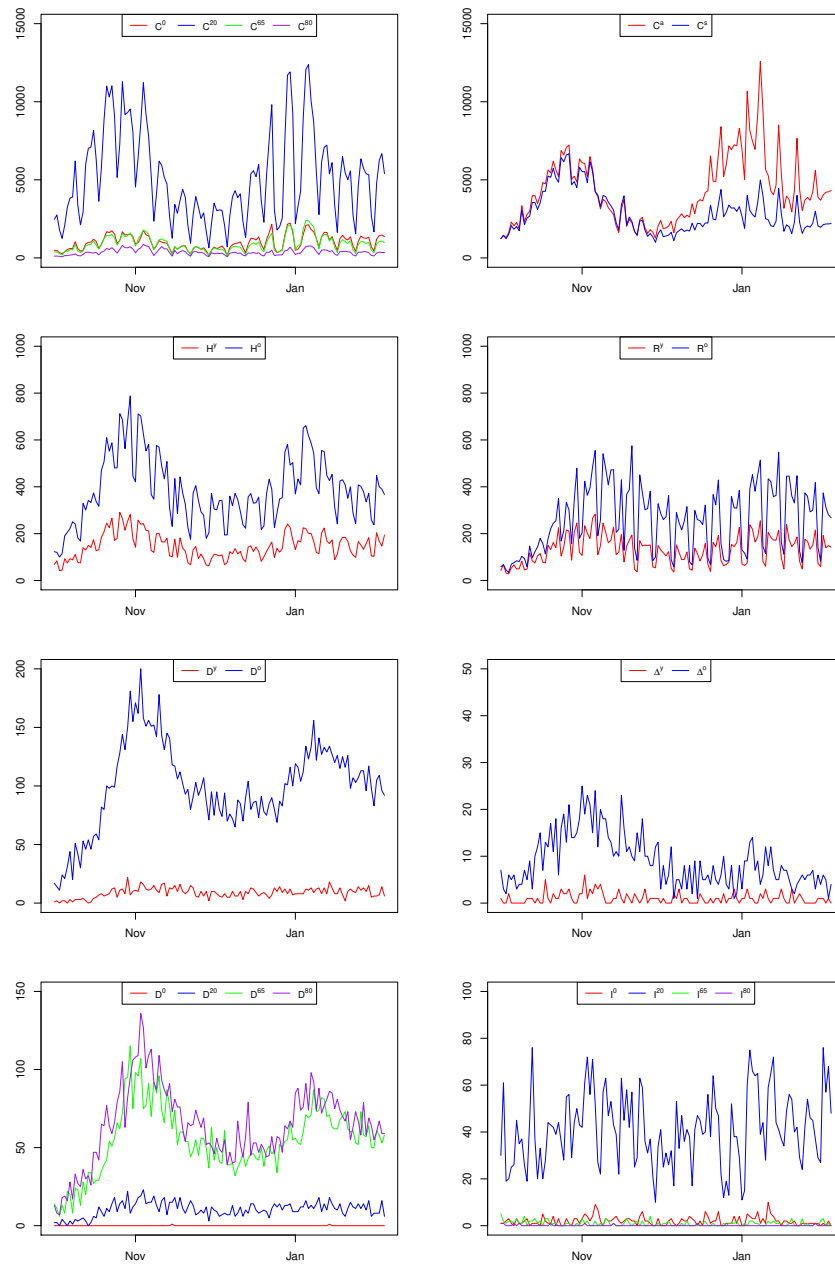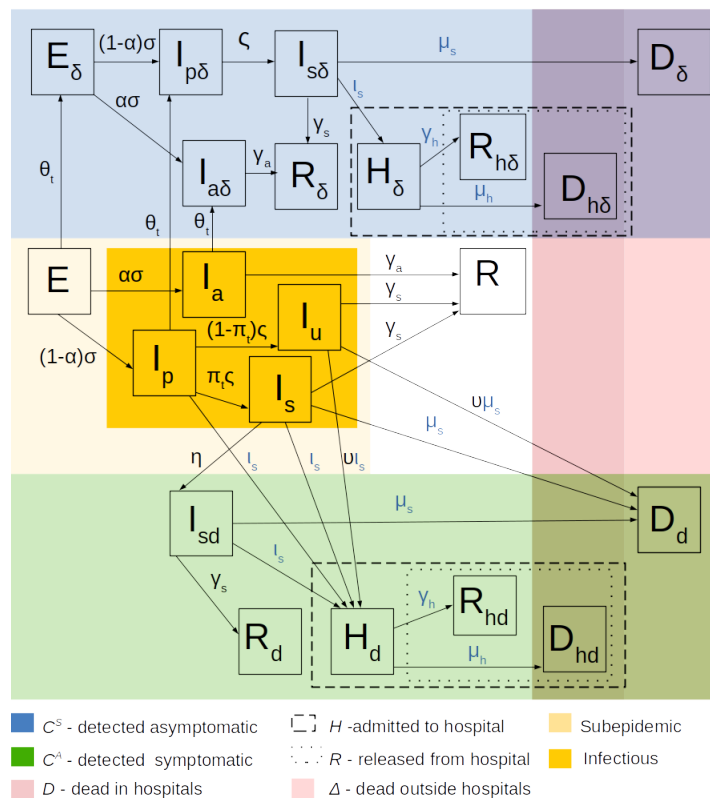
Figure 1: Inputs of the Czech COVID-19 model

Figure 2: Structure of the Czech COVID-19 model.

version of the model from Example 6, compartments of which are shown in Figure 2.

To reflect the fact that many cases are (intentionally) undetected, we added the compartment I_u. Members of I_u can be detected only if they are hospitalized or die, otherwise they never undergo testing. Further, to all the compartments from Example 6, we added their "detected" versions, distinguishing detection when being asymptomatic (subscript δ) or symptomatic (subscript d). We distinguish three "removed" states: recovered (R), hospitalized (H) and dead (D). Assuming that each individual who gets to the hospital is detected upon entry, we have two versions of H states: detected when symptomatic (H_d) and detected when asymptomatic (H_δ). Similarly, we assume that each dead is detected and we distinguish whether the individual dies in- or outside of hospital; consequently, we have four "dead" states: $D_{hd}, D_d, D_{h\delta}$ and D_δ. The case of R is analogous with the exception that recovery can happen without detection, so we have five versions: R, R_{dh}, R_d, $R_{\delta h}$, and R_δ.

We distinguish four age cohorts: 0 to 19 years, 20 to 64 years, 65 to 79 years and more than 80 years; consequently, having four compartments for each state, we have $4 \times 21 = 84$ compartments in total.

Some transition parameters (components of P_t) are common for all compartments (black symbols in Figure 2), the others are specific for each compartment (blue symbols). Some parameters are constant in time, some (these with index t) are time varying. The values of some parameters were externally determined, some were estimated. The detailed description of parameters, way of their determination, computation or estimation, and their estimated values can be found in Appendix, Table 2 and below. Here we only mention that we take

$$\theta_t = \vartheta_0 + \vartheta_1 q_t, \tag{5}$$

where q_t is the observed probability that an infectious individual is accepted to hospital without being previously detected, which, by the Bayes rule, is, up to a constant, equal to the overall detection probability. The rate π_t is set so that the detection probability holds in the model (see the Appendix). The value of q_t is publicly available as it is, as of February 2021, one of the input of the national counter-epidemic system PES.

The normalized "mixing matrix" Q has been computed from the estimate of the Czech overall contact matrix by [22], and is listed in Appendix, Table 3.

In line with [21], reporting $3 - 25$ times lower secondary attack rate of asymptomatic individuals, we assume that the probability of infection by I_a is four-times less in comparison with I_p, I_s and I_u. Further, we assume the dependence of infectiousness on the contact restriction and personal protection, and we reflect the natural immunization. Namely, we have

$$\varpi_t^{EI_s} = \varpi_t^{EI_{\acute{u}}} = \varpi_t^{EI_s} = \varpi_t, \qquad \varpi_t^{EI_a} = \frac{1}{4}\varpi_t, \qquad \varpi_t^{\bullet} = 0 \text{ otherwise,}$$

where

$$\varpi_t = g_t h_t v_{t-7} p_{t-7}. \tag{6}$$

Here, v_t is the average number of weekly risk contacts of an individual, p_t is the reduction caused by personal protection, $h_t \overset{\text{def}}{=} 1 - \frac{\sum_{i=1}^{k} \hat{X}_{t|t}^i}{s}$ is the adjustment for immunization (we assume that once infected individuals cannot be infected again), and g_t is the adjustment for new mutations, see Appendix for details.

The values of v_t and p_t were taken from [20] which is a longitudinal study, inquiring a panel of 3000 respondents about their (weekly) risk contacts, observance of several personal protection measures (see Appendix), and some other variables. While the value of v_t is being directly questioned by the survey, we compute the level p_t of personal protection as

$$p_t = \omega_0 \exp\{-\omega_e e_{t-7} - \omega_f f_{t-7}\} \tag{7}$$

where e_t is a certain linear combination of observation rates of various personal protective measures, f_t is the reported level of fear caused by the pandemic and ω_0, ω_e and ω_f are constants (see Appendix for further explanation).

For better comparison with other models, we use normalized version of the personal contacts level $w_t \overset{\text{def}}{=} \frac{v_t}{v_0}$ where v_0 is the pre-pandemic value of v, so we have, up to constant multiplication,

$$\varpi_t = g_t h_t w_{t-7} \exp\{-\omega_e e_{t-7} - \omega_f f_{t-7}\}; \tag{8}$$

with the corresponding constant becoming part of estimated parameters b_0, b_{20}, b_{65} and b_{80}.

As for the dispersion parameters, we assume $v = 26$ (see Remark 3) and, for each cohort i, we put $c_x^i \equiv c_h$, $x \in \{H_d^i, H_\delta^i\}$ (all "hospital" states) and $c_x^i \equiv c_a$ otherwise. This restriction of the parameter space was done in order to avoid over-parametrization, but to reflect the larger variability of

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incidences and hospitalizations in comparison with the mortality data (note
that dead mostly come from hospitals). Note also that, for terminal states,
the dispersion parameters have no effect, as their "target" distribution is
Dirac.

We used the following daily data series as inputs (see Figure 1):

$\tilde{I}^0, \tilde{I}^{20}, \tilde{I}^{65}, \tilde{I}^{80}$ - numbers of positively tested with infection abroad of individual
cohorts,

serving as imports (see below) and the following series which we consider as
observations (variables Y) in our model:

C^A, C^S - incidences of asymptomatic and symptomatic

$C^0, C^{20}, C^{65}, C^{80}$ - incidences in individual cohorts

$D^0, D^{20}, D^{65}, D^{80}$ - deaths in individual cohorts

H^y, H^o - admissions to hospitals of individuals from the first two cohorts
and second two cohorts

R^y, R^o - numbers of released from hospitals of the first and second two
cohorts

D^y, D^o - dead in hospitals of the first and second two cohorts

Δ^y, Δ^o - dead outside hospitals of the first and second two cohorts

The data come from the public repository of the Ministry of Health of
the Czech Republic and from the The Institute of Health Information and
Statistics of the Czech Republic, where the former is publicly available while
the latter is available only to research institutions. See Figure 1 for overview.

The data had to be pre-processed. First of all, series C, H and R, showing
strong weekly pattern, have been de-seasoned. Second, as the total reported
incidence exceeds (by about 2 percent) the personal level data (probably
because details are not always known), we took the former as decisive. Next,
as the reports of symptomatic an asymptomatic results of tests give slightly
less totals then the incidence, we took only ratios from the former multiplied
by the total incidences as C^a and C^s. Finally, as the total reported number
of actually hospitalized, which we denote by L, exceeds (by units of per-
cents) the value $H - R$ coming from the anonymized hospitalization data, we

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adjusted series H and R so that $L \doteq H - R$ (doing it exactly is impossible, as plain multiplication could cause unrealistic values of H and R).

Only three series take part as exogenous variables (values Z) in our model:

w - normalized contact restriction

e - personal protection index

f - level of reported fear

q - probability of detection in hospital

(see explanations above).

The matrix F, transferring compartment sizes X to observations Y, is zero-one. Which components are equal to one can be partially devised from Figure 2, the exact definition of F is given in Appendix, Figure 6. The lack of a row of F corresponding to C^a is intentional as C^a is redundant (equal to $C^0 + C^{20} + C^{65} + C^{80} - C^S$) and would cause linear dependence in F if included.

Assuming the imports only to the state E, we take,

$$I_t^{E^i} = \tilde{I}_{t+8}^i$$

for each cohort i (the time-shift reflects the delay in reporting).

Reflecting the notorious unreliability of incidence data, we put

$$\Gamma_t(x, y) = \mathrm{diag}(\beth_s, \beth_c, \beth_c, \beth_c, \beth_c, 0, \ldots, 0)Fx$$

where $\beth_s$ and $\beth_c$ are estimated parameters (note that the non-zero rows of Γ_t correspond to $C^S, C^0, C^{20}, C^{65}, C^{80}$ respectively).

For estimation, we used Weighted Least Squares applied to the seven day forecasts with weekly average increments as weights:

$$U_t = \mathrm{diag}\left(\left(\frac{Y_t^i - Y_{(t-7)\vee 0}^i}{7 \wedge t} \vee 5\right)^n_{i=1}\right), \qquad t > 0.$$

Due to the possibly different nature of the first wave (until May) and irregular behavior of the pandemic in summer (caused by small incidence with local outbursts), we used only data from October onward for the (final) estimation. To get "reasonable" initial state for our estimate, we roughly estimated the

model for the whole period of the pandemic and took its state from the end of August as the initial state for the final estimation; to minimize the influence of the initial value, however, the WLS criterion was computed only for the period from October 1, 2020 till January 21, 2121 .

Unfortunately, the estimation did not directly lead to quality results. The reason was that minimization with free c_u, c_d, γ_s and γ_c (dispersion) parameters tends to underestimate forecast variances, which leads to unrealistically narrow confidence bounds. In order to get more realistic variances, we adjusted the risk parameters of the estimate so that the variance of standardized (one day forecast) residuals were close to one; we use grid search here. Then we re-estimated the model by WLS with the adjusted risk parameters fixed. Yet the value of the new WLS minimum is about 25 percent higher than the original one, the variances in the new model are realistic. Finding a fully automatic estimation procedure getting realistic variances, perhaps by regularization with respect to variance of residuals, is a challenging topic of future research. The C++ code for the WLS estimation, employing NLopt library for minimization, is available on `https://github.com/cyberklezmer/seir/tree/ejor`.

The standardized residuals (i.e. the residuals divided by the standard error of the estimate) of the seven-day predictions for the individual observables, may be seen in Figure 3. Figure 4, on the other hand, shows the predictions fit of the total incidence $C_t \stackrel{\text{def}}{=} C_t^0 + C_t^{20} + C_t^{65} + C_t^{80}$, the total number of deaths $D_t \stackrel{\text{def}}{=} D_t^0 + D_t^{20} + D_t^{65} + D_t^{80}$ and the actual occupancy of hospitals $L_t \stackrel{\text{def}}{=} H_t^y + H_t^o - R_t^y - R_t^o$. For each variable, the left-hand graph shows the fit of the (in-sample) seven day predictions, the right-hand graph depicts the (out of sample) predictions for 14 days following Jan 21.

10. Vaccination Experiment

Finally, as a demonstration of applicability of our model, we evaluated outcomes of three vaccination scenarios

novac The infectiousness ϖ_t increases by 50% on January 22 (by emergence of a new mutation and/or release of the counter-epidemic measures) and keeps being constant until the end of May

elderly The infectiousness rises as above, but 30000 shots of vaccine a day will start to be applied on February 1st. Vaccines are distributed to the
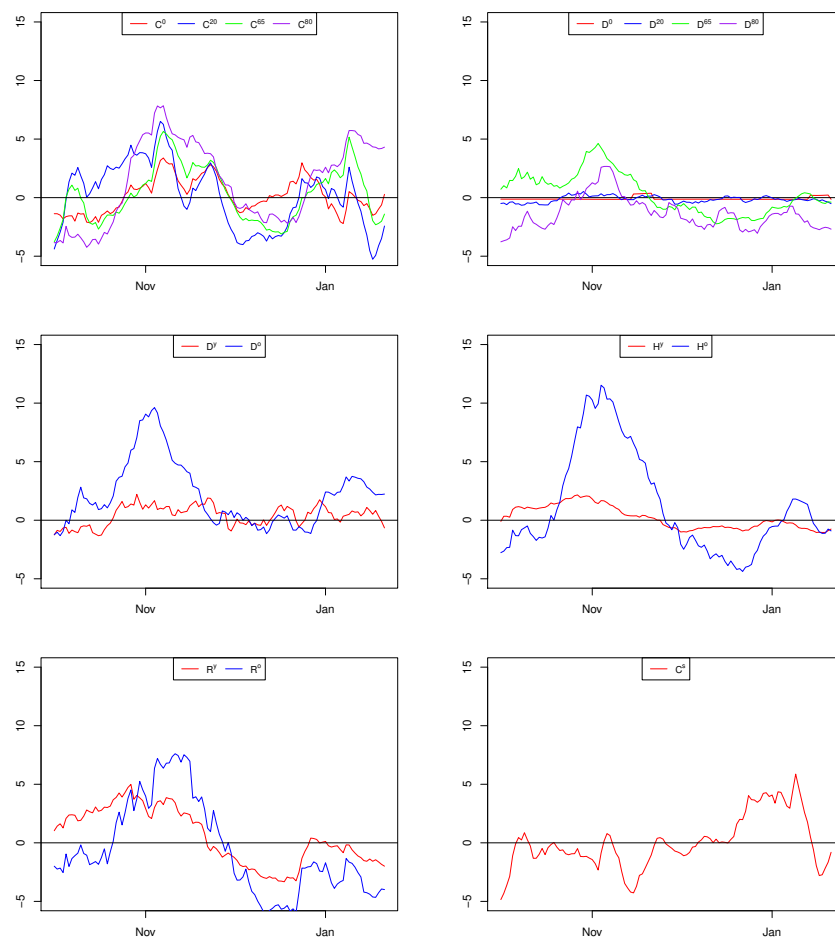
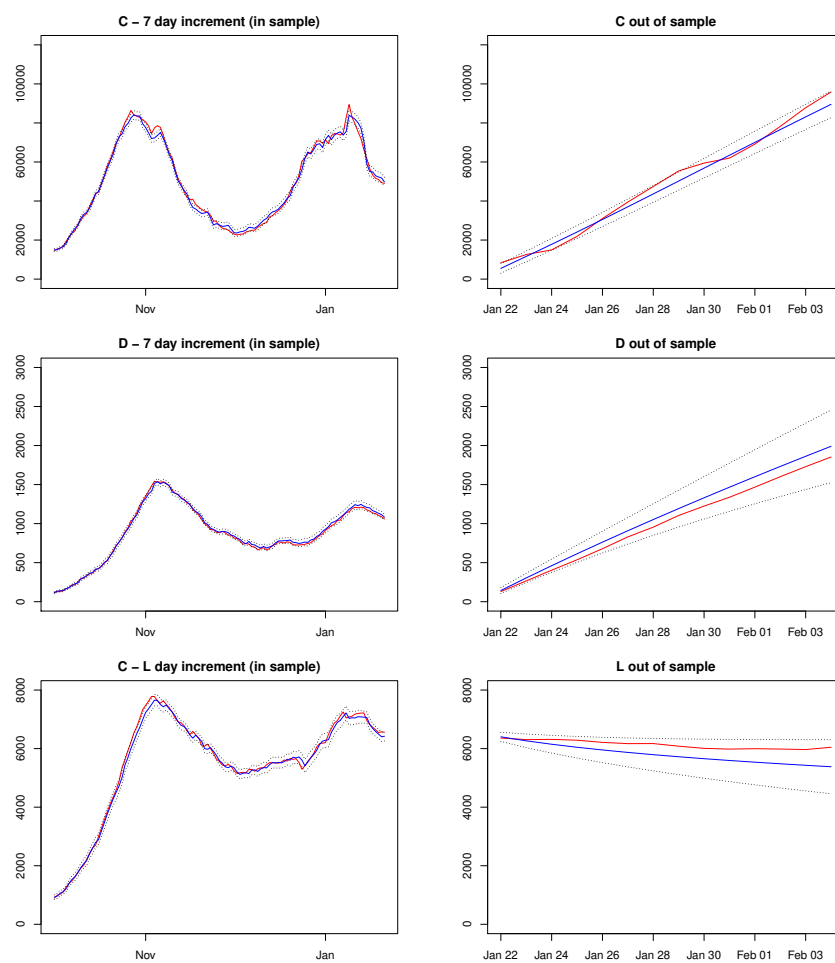Figure 3: Residuals of the Czech COVID-19 model.

Figure 4: Predictions of the Czech COVID-19 model. Red – actual, Blue – predicted, dotted – 95 % confidential bound.

	novac	middle	elderly	novac -middle	novac -elderly	middle -elderly
Total incidence	872050	814270	843350	57780	28700	-29080
Total deaths	15712	12413	11710	3298	4001	703
Average hospitalization	7067	5906	5753	1160	1314	154

Table 1: Outcomes of Vaccination Scenarios

cohort 80+ first and, as late as the whole cohort is vaccinated, vaccines start to be given to the cohort 65-79. After 35 days, each vaccinated individual gets a second shot (these shots do not count to the daily number 30000).

middle The situation is the same as in "elderly" scenario with the difference that only 15000 vaccines a day are given to the oldest, the rest are given to cohort 20-64 (perhaps infrastructural workers).

As for the vaccine effect, we (conservatively) assume that vaccinated individuals can be infected the same way as if they were not vaccinated; however, the probability of the asymptomatic course rises: 14 days after the first shot to 0.9 for the first two cohorts (0-64), or to 0.7 for those who are older than 65. After 14 days from the second shot, the probability further rises to 0.95 and 0.8, respectively. We insert these effects into the model by adjusting the overall probability α of asymptomatic course in individual cohorts according to the number of vaccinated therein.

The results of simulation are shown in Figure 5 and Table 1. Not surprisingly, vaccination saves all the infections, deaths and hospital beds. Prioritizing the older saves lives and beds, however, adds infections (which partially due to greater efficiency of the vaccine for the younger cohorts, maybe also due to greater number of contacts of the young). The most important message, however, is that vaccination is not a "silver bullet" solving everything – there are still large numbers of dead even with vaccination, which could be saved if the infectiousness was reduced, too, perhaps by some unpopular measures.

11. Conclusion

We presented a stochastic epidemic model, formulated its basic properties and suggested way of its estimation, all demonstrated by a real-life examples. We demonstrated that our model has good predictive power. They are,
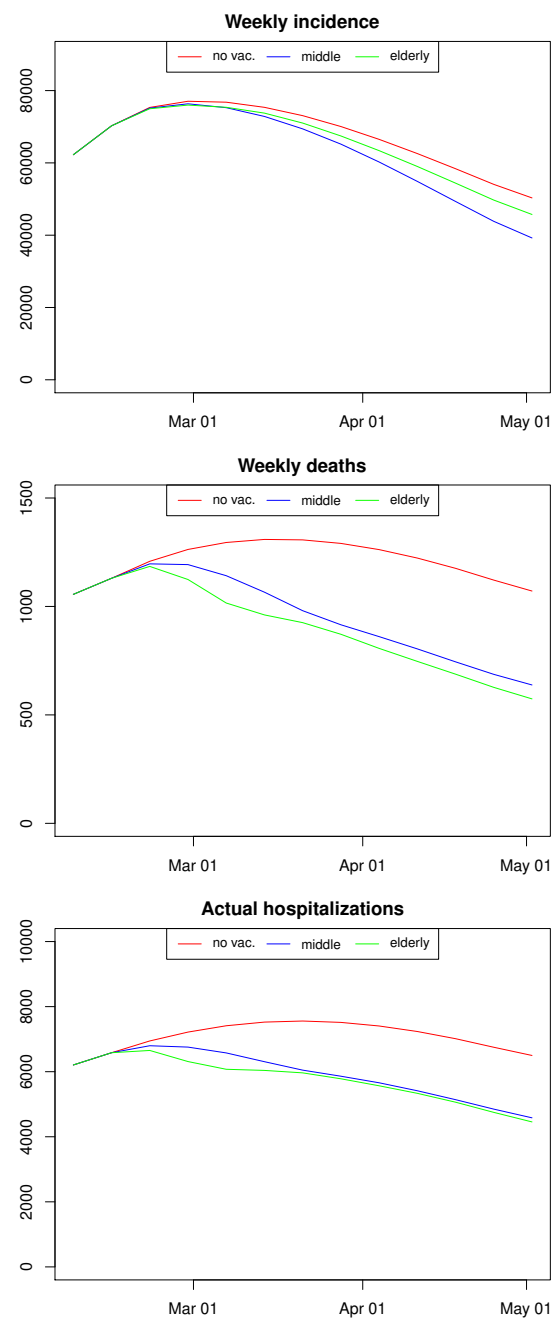
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Figure 5: Outcomes of Vaccination Scenarios

however, many things to be done. Most importantly, a reliable automatic estimation procedure should be suggested, guaranteeing good fit of both means and variances. Second, regularity conditions which would ensure asymptotic properties should be formulated, yet it can be a difficult task. However, even as it is, it can be immediately used for statistically correct modeling of the present and perhaps also another epidemics.

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Appendix: Parameters of the Czech COVID Model

Table 2 lists the majority of parameters, some of them being estimated, the rest being determined. The following Table lists values, which we surveyed from literature in order to compute selected parameters.

	Description	Source
$m_E = 5.08$	mean of incubation period	[11]
$\alpha = 0.179$	probability of asymptomatic course	[17]
$m_a = 4$	expected duration of presymptomatic period	[18]
$m_i = 8$	expected duration of infectiousness	[26]

The parameters, not included in Table 2 are θ_t, which is defined by 5, π_t, which we define later, and ϖ_t which is defined by (8) but with owing explanation how p_t is computed.

The quantity π_t is set so that the overall detection probability, which we estimate by δq_t where q_t is discussed in the main text and δ is an estimated parameter. If we assume that, over time, the states of an individual follows a *continuous-time* Markov chain with transition matrix given by the parameters listed in Figure 2, then the probability of detection (excluding the cases when an undetected arrives to hospital or dies) is

$$d_t \overset{\text{def}}{=} \mathbb{P}[E \to E_\delta \vee E \to I_a \to I_{a\delta} \vee E \to I_p \to I_{p\delta} \vee E \to I_p \to I_s \to I_{sd}]$$
$$= \frac{\theta_t}{\theta_t + \sigma} + \frac{\alpha\sigma}{\theta_t + \sigma}\frac{\theta_t}{\theta_t + \gamma_a} + \frac{(1-\alpha)\sigma}{\theta_t + \sigma}\left(\frac{\theta_t}{\theta_t + \varsigma + \iota_s} + \frac{\pi_t\varsigma}{\theta_t + \varsigma + \iota_s}\cdot\frac{\eta}{\eta + \gamma_s + \mu_s + \iota_s}\right);$$

by putting $d_t = \delta q_t$, we get π_t (trimming when π_1 falls outside $[0,1]$).

To evaluate the level of personal protection p_t is difficult task, as the study [20] 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

	Value	Estimated	Remark
υ	26 (0.6023)	N	see Remark 3
c_a	400 (10.2154)	Y	
c_h	1000 (53.4429)	Y	
$\beth_c$	1 (0.0244)	Y	
$\beth_s$	6 (0.1732)	Y	
ν	1.37717 (0.0008)	Y	see (**??**)
ω_e	1.0134 (0.0135)	Y	
ω_f	5.10832 (0.0092)	Y	
δ	1.29932 (0.0041)	Y	see (**??**)
ϑ_1	0.02668 (0.0001)	Y	see (5)
η	0.10584 (0.0003)	Y	
ϑ_0	0.01298 (0)	Y	see (5)
σ	0.1787 (0.0004)	N	$\sigma = 1 - \exp\{-1/m_E\}$
ς	0.2212 (0.0009)	N	$\varsigma = 1 - \exp\{-\frac{1}{m_a}\}$
u	0.35298 (0.0062)	Y	
γ_s	0.079 (0.0002)	N	$\gamma_s = 1 - \exp\{-\frac{1}{m_s}\}$
γ_a	0.12 (0.0028)	N	$\gamma_a = 1 - \exp\{-\frac{1}{m_i+m_a}\}$
b^0	3.26536 (0.0303)	Y	
ι_s^0	0.00135 (0.0001)	Y	
μ_s^0	0 (0.0003)	Y	
γ_h^0	0.99974 (0.0597)	Y	
μ_h^0	0 (0.0298)	Y	
b^{20}	3.71562 (0.0356)	Y	
ι_s^{20}	0.00092 (0)	Y	
μ_s^{20}	0.00003 (0)	Y	
γ_h^{20}	0.07866 (0.0015)	Y	
μ_h^{20}	0.00724 (0.0007)	Y	
b^{65}	4.6724 (0.0456)	Y	
ι_s^{65}	0.0026 (0)	Y	
μ_s^{65}	0.00165 (0.0001)	Y	
γ_h^{65}	0.00521 (0.0002)	Y	
μ_h^{65}	0.01421 (0.0002)	Y	
b^{80}	5.2102 (0.0616)	Y	
ι_s^{80}	0.56583 (0.0061)	Y	
μ_s^{80}	0.0358 (0.0015)	Y	
γ_h^{80}	0.1237 (0.0008)	Y	
μ_h^{80}	0.03104 (0.0002)	Y	

Table 2: Parameters of the Czech COVID Model

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 corelated). To overcome this difficulty, we applied factor analysis to $(v_t, \pi_t^1, \ldots, \pi_t^q)$ on the respondent level, treating the responses in different times as separate observations. As a result, we extracted two following main factors:

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	w_t

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

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

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

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Cohort	0	20	65	80
Population	2188232	6374077	1690530	441100
Q	0	20	65	80
0	$b^0 \times 0.54$	$b^0 \times 0.11$	$b^0 \times 0.02$	$b^0 \times 0.02$
0	$b^0 \times 0.6$	$b^0 \times 0.92$	$b^0 \times 0.12$	$b^0 \times 0.12$
65	$b^{65} \times 0.09$	$b^{65} \times 0.1$	$b^{65} \times 0.14$	$b^{65} \times 0.14$
80	$b^{80} \times 0.02$	$b^{80} \times 0.02$	$b^{80} \times 0.02$	$b^{80} \times 0.06$

Table 3: Popluation of Cohorts and Mixing Matrix Q

could approximate

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

$$\doteq \exp\left\{-\sum_{i=1}^{q}\lambda^i(\overline{\pi}_t^i + \nu_t e_t)\right\} = \omega_0 \exp\left\{-\omega_e e_t\right\}$$

where $\omega_0, \omega_e \geq 0$. Similarly we can evaluate (assumed) reduction caused by the average reported fear f_t of infection, giving (7).

As for the adjustment g_t for the new virus variants, we assume $g_t = 1$ up to November 20, 2020, $g_t = \nu$, where ν is an (estimated) parameter, since January 1, 2021, and that g_t is linearly interpolated between the dates. Yet the dates do not correspond to the common knowledge about mixing of the British variant, they come from a regression estimate of the reproduction number by υ.

Finally, we list the population of individual cohorts together with the "mixing matrix" Q (Table 3) and the definition of transformation matrix F (Figure 6).
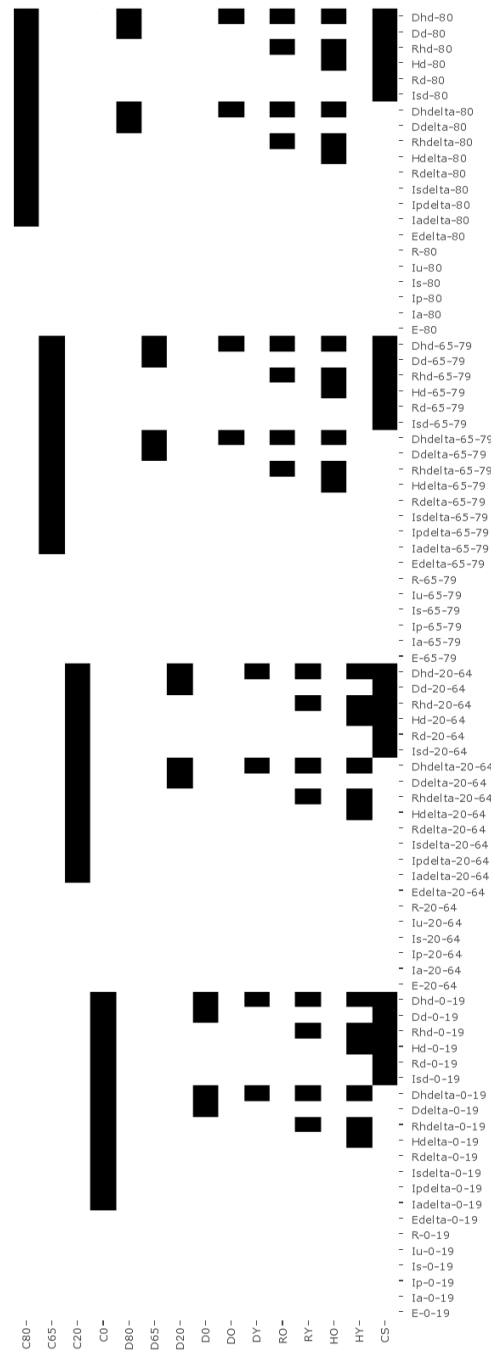
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Figure 6: Definition of Transformation Matrix F