Monitoring and prediction of an epidemic outbreak using syndromic observations

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Abstract

The paper presents an algorithm for syndromic surveillance of an epidemic outbreak formulated in the context of stochastic nonlinear filtering. The dynamics of the epidemic is modeled using a generalized compartmental epidemiological model with inhomogeneous mixing. The syndromic (typically non-medical) observations of the number of infected people (e.g. visits to pharmacies, sale of certain products, absenteeism from work/study etc.) are used for estimation. The state of the epidemic, including the number of infected people and the unknown parameters of the model, are estimated via a particle filter. The numerical results indicate that the proposed framework can provide useful early prediction of the epidemic peak if the uncertainty in prior knowledge of model parameters is not excessive.

Keywords: Emerging epidemics, bio-terrorism, inhomogeneous mixing, social network, mathematical biology, nonlinear filtering, particle filter, imprecise likelihood.

1. Introduction

Epidemics can impose significant challenges on modern societies, not only by affecting the health of the general population, but also by causing negative trends in the economy (medical treatments, absenteeism from work, missed business opportunities, etc). The ongoing epidemics of AIDS, tuberculosis and the recent outbreaks of SARS and H1N1 (swine flu) provide some revealing examples. In the absence of an effective cure against many diseases, the best approach to mitigate an epidemic outbreak (malicious or natural) resides in the development of capability for its early detection and for prediction of its
further development \cite{38, 40, 22, 5, 19, 4}. Such a capability would allow making any countermeasures (quarantine, vaccination, medical treatment) much more effective and less costly \cite{39, 40}.

Syndromic surveillance is referred to as systematic collection, analysis, and interpretation of public health data for the purpose of early (and often cost effective) detection of an epidemic outbreak, its continuous monitoring, and timely response by public health agencies \cite{38, 40, ?}. The rationale for this method rests on a resalable assumption that a spread of an infectious disease is usually associated with the measurable changes in the social behaviour.

It is important to highlight that although the measurements (data streams) for syndromic surveillance often rely on the medical observations (the visits to medical practitioners, occurrence of particular symptoms, the number of diagnosed cases, etc) they are not restricted to the pure medical data. Recent studies \cite{13, 31, 7}, have demonstrated that “non-medical” sources of syndromic data streams, such as the absenteeism from work/school, the pharmaceutical sales, Internet queries, Twitter messages, and alike, can enable one to draw important conclusions regarding the epidemic state in the community. The ‘Google Flu’ project \cite{14} (flu-related searches in Google), is a well publicised example of this approach.

The algorithms for syndromic surveillance and its practical implementation have recently attracted significant attention by scientists and practitioners; there is a vast amount of literature devoted to this topic (for more comprehensive review see \cite{38, 40} and refs therein). In general, all algorithms applied in this area can be divided into two main groups, the data mining methods and the information fusion (also known as data assimilation) methods. Data mining is primarily concerned with the extraction of patterns from massive amounts of raw data without using dynamic models of the underlying process (i.e. epidemic spread) \cite{14, 7}. Information fusion algorithms, on the contrary, strongly rely of mathematical models: in this case, the dynamic model of an epidemic outbreak and the measurement model of a particular syndromic data stream \cite{11, 6, 21}. Evidently the accuracy of the information fusion algorithms is significantly determined by the fidelity of the underlying models.

This paper presents a study of a recursive information fusion algorithm for syndromic
surveillance. The problem is formulated in the Bayesian context of stochastic nonlinear filtering and solved using a particle filter. While this problem formulation and the particle filter implementation have been considered earlier, see [13], [29], [25], [35], this paper introduces several novelties. In order to overcome the limitations of the standard “compartment” model of epidemic (the “well-mixed” approximation) we employ a more flexible alternative, initially proposed in [36] (and later extended in [24], [30]). The adopted epidemic model has the explicit parameter of “mixing efficiency” (or level of social interaction) and is therefore more appropriate to represent the variety of social interactions in a small community (self-isolation and panic). A significant advantage of the adopted epidemiological model is a rigorous estimation of its noise component (scaling law of noise level with the population size of a community, see below) resulting in more accurate parameter estimations. Furthermore, a more flexible model of syndromic measurements, validated with a limited data set available in the literature [14], [13], is adopted in the paper. This measurement model is derived from [14], which has been extensively validated by observations and is allowed to have imprecisely known parameters. The optimal sequential estimator (filter) and predictor are then formulated in the Bayesian framework and solved using a particle filter. The paper includes numerical results that verify the theoretical framework and analyses the sensitivity to partially known epidemic parameters. From this perspective, the present study is a significant extension of our earlier work [29], [35].

2. Problem specification

This section describes the models to be used in estimation. To describe the dynamics of an epidemic outbreak we employ the generalized SIR epidemic model proposed in [10], [9], [1], [8] with stochastic fluctuations. According to this model the population of the community can be subdivided into three interacting groups: susceptible, infectious and recovered individuals. Let the number of susceptible, infectious and recovered be denoted by $S$, $I$ and $R$, respectively, so that $S + I + R = P$, where $P$ is the total population size. The dynamic model of epidemic progression in time can be be expressed by two stochastic differential equations [2], [10], [9] and the “conservation” law for the
population as follows:

\[
\frac{ds}{dt} = -\alpha i s \nu + \sigma_q \xi, \tag{1}
\]

\[
\frac{di}{dt} = \alpha i s \nu - \beta i - \sigma_q \xi + \sigma_\beta \zeta, \tag{2}
\]

\[
r = 1 - s - i. \tag{3}
\]

The explanation of notation: \( s = S/P, i = I/P, r = R/P \) and \( \xi, \zeta \) are two uncorrelated white Gaussian noise processes, both with zero mean and unity variance. The terms \( \sigma_q \equiv \sigma_q(s, i) \) and \( \sigma_\beta \equiv \sigma_\beta(s, i) \) are introduced to capture the demographic noise (random variations in the contact rate \( \alpha \) and in the recovery time \( \beta \) between individuals), for details see [37], [10], [9].

The parameters of this model are described next: \( \beta \) is the recovery time, that is a reciprocal of the average infectious period for the disease; \( \alpha = \rho \cdot \beta \), where \( \rho \) is a well known parameter in epidemiology, referred to as the basic reproductive number, which represents the average number of people infected by a direct contact with a sick person. Finally \( \nu \) is the population mixing parameter, which for a homogeneous population equals 1. In the presence of an epidemic, \( \nu \) may vary as people may behave unpredictably (panic) or avoid the risk of infection (self-isolation). In general, the epidemic model parameters can be assumed to be partially known as interval values, that is \( \alpha \in [\alpha, \bar{\alpha}], \beta \in [\beta, \bar{\beta}] \) and \( \nu \in [\underline{\nu}, \bar{\nu}] \).

The amplitude of the noise terms \( \sigma_q, \sigma_\beta \) in (1) and (2) can be established from a scaling law of Gaussian fluctuations generated by the random contact rate \( q = \alpha i s \nu \) and recovery time \( \beta \). Thus for a dynamical system (1) - (3) consisting of a large number of individuals \( P \) we can write (for details see [10], [9], [37])

\[
\sigma_q(s, i) \approx \sqrt{\frac{\alpha i s \nu}{P}}, \quad \sigma_\beta(s, i) \approx \sqrt{\frac{\beta i}{P}}. \tag{4}
\]

With further reasonable assumptions \( s \approx s_0 \approx 1, r \approx r_0 \approx 0, i \approx i_0 \approx 1/P \), that holds for the initial stage of epidemic (where \( s_0, i_0, r_0 \) are initial values of \( s, i, r \)) expressions (4) can be reduced to the following scaling for the noise components of the model

\[
\sigma_q(s, i) \approx \sqrt{\frac{\alpha}{P}}, \quad \sigma_\beta(s, i) \approx \sqrt{\frac{\beta}{P}}. \tag{5}
\]

The measurement model is introduced next. Following [14], [7] we can assume that
a power law relationship holds for odds-ratios of syndromic observations and number of infected people:

\[ \frac{z_j}{1 - z_j} \propto \left( \frac{i}{1 - i} \right)^{\varsigma_j}, \tag{6} \]

where \( z_j \) is the observable syndrome with index \( j = 1, \ldots, N \) (normalized by population size \( P \)), \( \varsigma_j \) is the power-law exponent (in general different for different syndromes). Since at the initial stage of epidemic \( i \ll 1 \), \( z_j \ll 1 \), eq. (6) can be reduced to a simple power-law model

\[ z_j = b_j i^{\varsigma_j} + \tau_j, \tag{7} \]

where \( b_j = \text{const} \). The noise term \( \tau_j \) is added to simulate random nature of measurement (measurements noise) which is assumed to be uncorrelated to other syndromes and noises \( \xi \) and \( \zeta \). Since \( z_j \geq 0 \) the noise term \( \tau_j \) associated with syndrome \( j \) should be modelled with a random variable that provides strictly non-negative realisations (e.g. Poissonian \[21\] or truncated Gaussian \[10\]). In the current study we use a random variable that obeys the log-normal distribution: this means that we set \( \tau_j = \sigma_j \eta_j \), where \( \eta_j \) is the standard log-normal noise \( \eta_j \sim \ln \mathcal{N}(0, 1) \), see below.

Parameters \( b_j, \sigma_j, \varsigma_j \) typically are not known, but with a representative dataset of observations the model (7) can be easily calibrated (see results of the linear regression fits in \[14\]). The data fit reported in \[13\] suggests that \( \varsigma_j \) may be close to unity (but it is not precisely known because of significant scattering of data points). To cater for this uncertainty we assume that \( \varsigma_j \) can take any value in an interval, \( \varsigma \in [\varsigma, \bar{\varsigma}] \) around \( \varsigma = 1 \) (for the sake of simplicity we also assume that all \( \varsigma_j = \varsigma = \text{const} \)). Unfortunately \[14\] do not report any specific values of fitting parameters, so we have to use some heuristic values for \( b_j, \sigma_j \) in our simulations.

The scaling law for the noise parameter \( \sigma_j \) in (7) can be deduced by employing the arguments leading to (5) and thus we arrive at the different scaling \( \sigma_j \propto (1/P)^{\epsilon} \), \( \epsilon = \min\{(\varsigma + 1)/2, 1/2\} \). The later scaling law in conjunction with the scaling laws (5) provides a consistent way to compare predictive skills of our algorithm for communities with different population size and to draw conclusive decisions about its performance before any operational deployment.

Being formulated in the Bayesian framework, our problem is to estimate the (normalised) number of infected \( i \) and susceptible \( s \) at time \( t \), using syndromic observations
collected up to time $t$. Let $\mathbf{x}$ denote the state vector to be estimated; it includes $i$ and $s$, but also the imprecisely known parameters $\alpha$, $\beta$ and $\nu$. The formal Bayesian solution is given in the form of the posterior probability density function (PDF) $p(\mathbf{x}_t|z_{1:t})$, where $\mathbf{x}_t$ is the state vector at time $t$ and $z_{1:t}$ denotes all observations up to time $t$. Using the posterior $p(\mathbf{x}_t|z_{1:t})$ one can predict the progress of the epidemic using the dynamic model (1)-(3).

3. Optimal Bayesian solution for imprecise likelihood

3.1. Formulation

The model (1)-(3) is given in continuous time. For the purpose of computer implementation, we require a discrete-time approximation of this model. The state vector is adopted as

$$
\mathbf{x} = \begin{bmatrix} i & s & \alpha & \beta & \nu \end{bmatrix}^T
$$

(8)

where $^T$ denotes matrix transpose. Neglecting for the moment the process noise terms, the evolution of the epidemic state can be written according to (1)-(2) as

$$
\dot{\mathbf{x}} = \mathbf{g}(\mathbf{x})
$$

where

$$
\mathbf{g}(\mathbf{x}) = \begin{bmatrix} (\alpha s' - \beta) i & -\alpha i s' & 0 & 0 & 0 \end{bmatrix}^T.
$$

The nonlinear differential equation governing the evolution of the state cannot be solved in the closed-form. The Euler method provides a simple approximation valid for small integration interval $\tau > 0$: $\mathbf{x}(t + \tau) \approx \mathbf{x}(t) + \tau \mathbf{g}(\mathbf{x}(t))$. The state-evolution in discrete-time $t_k$ can then be expressed as:

$$
\mathbf{x}_{k+1} \approx \mathbf{f}_k(\mathbf{x}_k) + \mathbf{w}_k
$$

(9)

where $k = t_k/\tau$ is the discrete-time index and $\mathbf{f}_k(\mathbf{x}_k)$ is the transition function given by

$$
\mathbf{f}_k(\mathbf{x}_k) = \begin{bmatrix}
\mathbf{x}_k[1] + \tau \mathbf{x}_k[1] (\mathbf{x}_k[3]\mathbf{x}_k[2]^{[5]} - \mathbf{x}_k[4]) \\
\mathbf{x}_k[2] - \tau \mathbf{x}_k[3]\mathbf{x}_k[1]\mathbf{x}_k[2]^{[5]} \\
\mathbf{x}_k[3] \\
\mathbf{x}_k[4] \\
\mathbf{x}_k[5]
\end{bmatrix}
$$

(10)

In this notation $\mathbf{x}_k[i]$ represents the $i$th component of vector $\mathbf{x}_k$. Process noise $\mathbf{w}_k$ in (9) is assumed to be zero-mean white Gaussian with a diagonal covariance matrix $\mathbf{Q}$, which according to (5) can be expressed as $\mathbf{Q} \approx \text{diag}[(\alpha + \beta)\tau^2/P^2, \alpha \tau^2/P^2]$. 


The optimal Bayes filter is typically presented in two steps, prediction and update. Suppose the posterior PDF at time $t_k$ is given by $p(x_k | z_{1:k})$. Then the prediction step computes the PDF predicted to time $t_m = t_k + \tau$ as 

$$p(x_m | z_{1:k}) = \int \pi(x_m | x_k) p(x_k | z_{1:k}) \, dx_k$$

(11)

where $\pi(x|\mathbf{x}')$ is the transitional density. Let $\mathcal{N}(\mathbf{y}; \mathbf{\mu}, \mathbf{P})$ denote a Gaussian PDF with mean $\mathbf{\mu}$ and covariance $\mathbf{P}$. Then according to (9) the transitional density is given by

$$\pi(x | x'=) = \mathcal{N}(x; f_k(x'), \mathbf{Q}).$$

(12)

The prediction step is carried out many times with tiny sampling intervals $\tau$ until an observation $z_{j,k+1}$ becomes available about syndrome $j$ at time $t_{k+1}$.

The predicted PDF at $t_{k+1}$ is $p(x_{k+1} | z_{1:k})$. In the standard Bayesian estimation framework, this PDF is updated using the measurement $z_{j,k+1}$ by multiplication with the measurement likelihood function [16]. According to (17), the likelihood function in this case is $\ell(z_{j,k+1} | x_{k+1}) = \ln\mathcal{N}(z; h(x_{k+1}; \varsigma), \sigma^2_j)$, where $h(x_{k}; \varsigma) = b_j \cdot x_{k}[1]^\varsigma$. Now, the problem is that $h(x_{k}; \varsigma)$ defined this way is not a function because $\varsigma \in [\varsigma, \varsigma]$.

An elegant solution to the imprecise measurement transformation is available in the framework of random set theory [20]. In this approach $h(x; \varsigma)$ defines a closed set $\Sigma_x$ on the measurement space $\mathcal{Z}$. The closed set $\Sigma_x$ is random because the state $x$ is random. In fact, a random closed set (RCS) $\Sigma_x$ can be seen as a composite mapping. The first is the measurable mapping which defines the random variable $x : \Omega \to \mathcal{X}$, where $\Omega$ is the sample space and $\mathcal{X}$ is the state space. The second mapping is $h(x; \varsigma) : \mathcal{X} \to \mathcal{I}\mathcal{Z}$, where $\mathcal{I}\mathcal{Z}$ is the set of closed sets of $\mathcal{Z}$. The RCS $\Sigma_x$ is therefore a random variable that takes values as closed intervals of $\mathcal{Z}$.

The Bayes updated step using measurement $z_{j,k+1}$ is now defined as [20]:

$$p(x_{k+1} | z_{1:k+1}) = \frac{\tilde{\ell}(z_{j,k+1} | x_{k+1}) \cdot p(x_{k+1} | z_{1:k})}{\int \tilde{\ell}(z_{j,k+1} | x_{k+1}) \cdot p(x_{k+1} | z_{1:k}) \, dx_{k+1}}$$

(13)

where $\tilde{\ell}(z_{j,k} | x_k)$ is referred to as the generalised likelihood function. For the measurement model (7), $\tilde{\ell}(z_{j,k} | x_k)$ is defined as [27]:

$$\tilde{\ell}(z_{j,k} | x_k) = \phi(z_{j,k}; \Sigma_{x_k}, \sigma^2_j) - \phi(z_{j,k}; \Sigma_{x_k}, \sigma^2_j)$$

(14)
where

\[ \Sigma_x = \min \{ h(x; \xi), h(x; \tau) \} \] (15)

\[ \bar{\Sigma}_x = \max \{ h(x; \xi), h(x; \tau) \}. \] (16)

and \( \phi(u; \mu, P) = \int_{-\infty}^{u} \ln \mathcal{N}(y; \mu, P) \, dy \) is the cumulative log-normal distribution.

The recursions of the Bayes filter start with the initial PDF (at time \( t_k = 0 \)), denoted \( p(x_0) \), which is assumed known.

### 3.2. Particle filter implementation

The proposed Bayesian estimator cannot be solved in the closed form. Instead we develop an approximate solution based on the particle filter (PF) [3], [28]. The PF approximates the posterior PDF \( p(x_k | z_{1:k}) \) by a weighted random sample, that is:

\[ p(x_k | z_{1:k}) \approx \sum_{i=1}^{S} w_k^{(i)} \delta_{x_k^{(i)}}(x). \] (17)

Here \( \delta_x(x) \) is the Dirac delta function focused at \( y \), \( x_k^{(i)} \) is a sample (particle), \( w_k^{(i)} \) is its weight and \( S \) is the particle count. As \( S \) is increased, approximation (17) becomes more accurate. The weights are normalised so that:

\[ \int p(x_k | z_{1:k}) \, dx_k \approx \sum_{i=1}^{S} w_k^{(i)} = 1. \] (18)

The adopted PF implementation is known as the bootstrap PF [15]. The algorithm starts by forming a random sample from the initial PDF \( p(x_0) \): \( x_0^{(i)} \sim p(x_0) \), for \( i = 1, \ldots, S \). The initial weights are equal, that is \( w_0^{(i)} = 1/S \). The prediction steps consist of propagating particles using the dynamic model (9). This is implemented by drawing samples from the transitional density (12). The prediction steps are carried out until a measurement \( z_{j,k} \) becomes available, that is until time \( t_k \). Suppose the set of predicted particles at time \( t_k \) is \( \{ x_k^{(i)}_{k-1}; i = 1, \ldots, S \} \).

The update step is implemented in two stages. First the unnormalised importance weights of predicted particles are computed as [28]:

\[ \tilde{w}_k^{(i)} = \ell(z_{j,k} | x_k^{(i)}_{k-1}) \] (19)
with generalised likelihood $\tilde{\ell}$ specified in (14). This is followed by the weight normalisation, that is

$$w^{(i)}_k = \tilde{w}^{(i)}_k / \sum_{n=1}^{S} \tilde{w}^{(n)}_k$$  \hspace{1cm} (20)$$
for $i = 1, \ldots, S$.

The second stage is the resampling step. The role of resampling is to eliminate (in the probabilistic manner) the particles with low importance weights and to clone the samples with high importance weights. This is carried out by sampling with replacement, with the probability of sampling each $x^{(i)}_{k|k-1}$ equal to the normalised importance weight $w^{(i)}_k$. The result is a mapping of a random measure $\{w^{(i)}_k, x^{(i)}_{k|k-1}\}_{i=1}^{S}$ into a new random measure with uniform weights: $\{1/S, x^{(i)}_k\}_{i=1}^{S}$. Several computationally efficient resampling schemes have been reported; we have implemented the systematic resampling algorithm [18], following the pseudo-code given in Table 3.2 of [28].

4. Numerical Results

4.1. Experimental data set

The estimation and prediction of an epidemic will be carried out using an experimental data set obtained using a large-scale agent based simulated population model [32], [33] of a virtual town of $P = 5000$ inhabitants, created in accordance with the Australian Census Bureau data. The agent based model is rather complex and takes a long time to run. It includes typical age/gender breakdown and family-household-workplace habits with the realistic day-to-day people contacts for a disease spread. The blue line in Fig. 1 shows the number of people of this town infected by a fictitious disease, reported once per day during a period of 154 days (only first 120 days shown). The dashed red line represents the deterministic SIR model fit (using the entire batch of 154 data points, and setting $w_k = 0$ in (10)), with estimated parameters $\hat{\alpha} = 0.2399$, $\hat{\beta} = 0.1066$, $\hat{\nu} = 1.2042$. The parameter estimates were obtained using importance sampling via the progressive correction algorithm [26]. Fig. 1 serves to verify that the generalized SIR model, although very simple and fast to run, is remarkably accurate in explaining the data obtained from a very complex simulation system (for further details see [36]).
Figure 1: The solid line, representing the number of infected people over time, was obtained from the agent based simulation population model. The dashed line shows the generalised SIR model fit.

4.2. Estimation and prediction results

The true number of infected people in simulations was chosen to be the output of the agent based simulated population model, shown by the solid line in Fig. 1. The measurements are generated synthetically in accordance with (7), using the following parameters: \( \varsigma = 1.05 \) and \( b_j = 0.25, \sigma_j = 0.01, \) for all \( j = 1, 2, 3, 4 \) monitored syndromes. Independent measurements concerning all \( N_z = 4 \) syndromes were available on a daily basis during the first 25 days. The problem was to perform estimation sequentially as the measurements become available until the day number 25, and after that to predict the epidemic size.

The initial PDF for the state vector was chosen as \( p(x_0) = p(i_0)p(s_0)p(\alpha_0)p(\beta_0)p(\nu_0) \) with \( p(i_0) = \mathcal{N}_{[0,1]}(i_0; z_j, 1/b_j, \sigma_j^2) \), \( p(s_0) = \mathcal{N}_{[0,1]}(s_0; 1-z_j, 1/b_j, \sigma_j^2) \), \( p(\alpha_0) = \mathcal{U}_{[0.18, 0.50]}(\alpha_0) \), \( p(\beta_0) = \mathcal{U}_{[0.1, 1.0]}(\beta_0) \), \( p(\nu_0) = \mathcal{U}_{[0.95, 1.3]}(\nu_0) \), where \( \mathcal{N}_{[a,b]}(x, \mu, P) \) and \( \mathcal{U}_{[a,b]}(x) \) denote a truncated Gaussian distribution restricted at support \([a, b]\) and a uniform distribution with limits \([a, b]\), respectively. The number of particles was set to \( S = 10000 \).

The first run of the proposed simulation setup was carried out assuming that \( \varsigma \in [1.03, 1.07] \). This corresponds to the case of fairly precise knowledge of \( \varsigma \) (recall the true value was 1.05). Fig. 2(a) shows the histograms of particle filter estimated values of \( \alpha, \beta \) and \( \nu \), after processing all 25 days of measurements (i.e. in total 100 measurements,
since $N_z = 4$). This figure reveals that the uncertainty in parameters $\alpha$ and $\beta$ has been substantially reduced, compared with initial $p(\alpha_0) = U_{[0.18, 0.50]}(\alpha_0)$ and $p(\beta_0) = U_{[0.1, 0.143]}(\beta_0)$. The uncertainty in $\nu$, on the other hand, has not been reduced, indicating that this parameter cannot be estimated. While this is unfortunate, it does not appear to be a serious problem since the prior on $\nu$ in practice is tight ($\nu \approx 1$). This is confirmed in Fig. 2(b) which shows a sample of 100 overlayed predicted epidemic curves (gray lines) based on the state estimate after 25 days. Fig. 2(b) indicates the prediction performance; the timing of the peak of the epidemic appears to be fairly accurate, while the size of the peak is more uncertain. Most importantly, however, the true epidemic curve (solid red line) appears to be always enveloped by prediction curves.

The second run of the proposed simulation setup corresponds to the case with fairly imprecise knowledge of $\zeta$, that is $\zeta \in [0.85, 1.15]$. The results are shown in Fig. 3. Comparing Figs. 2 and 3 one can make two observations: first, in accordance with our expectations, the estimation and prediction results are more uncertain when knowledge of $\zeta$ is imprecise; second, even when knowledge of $\zeta$ is imprecise, the curve corresponding to the true number of infected people (solid red line) is always enveloped by predictions.

5. Conclusions

We have developed an algorithm for syndromic surveillance of an epidemic outbreak formulated in the context of stochastic nonlinear filtering. The dynamics of the epidemic is modeled using a compartmental epidemiological model with inhomogeneous mixing which explicitly includes a parameter responsible for the level of social interactions. This model enables simulation of a rich variety of social behavior that may be observed in a small community in response to epidemic (i.e. fear, self-isolations, panic).

The syndromic (typically non-medical) observations are used as an algorithm input (e.g. web queries, Twitter messages, sales of certain products, absenteeism from work/study etc). The algorithm (a particle filter) provides continuous estimation of the state of the epidemic, including the number of infected people and the unknown parameters of the model. The numerical results indicate that the proposed framework can provide useful early prediction of the epidemic peak if the uncertainty in prior knowledge of model parameters (including social behavior) is not excessive.
Figure 2: Estimation/prediction results from the particle filter after processing measurements collected over 25 days of surveillance assuming that $\varsigma \in [1.03, 1.07]$ (true value $\varsigma = 1.05$): (a) the histograms of estimated parameters $\alpha$, $\beta$ and $\nu$ (true values indicated by vertical red lines); (b) Prediction results for a random sample of 100 particles (gray lines); the red line is the experimental curve from Fig.1.
Figure 3: Estimation/prediction results from the particle filter after processing measurements collected over 25 days of surveillance assuming that $\zeta \in [0.85, 1.15]$ (true value $\zeta = 1.05$): (a) the histograms of estimated parameters $\alpha$, $\beta$ and $\nu$ (true values indicated by vertical red lines); (b) Prediction results for a random sample of 100 particles (gray lines); the red line is the experimental curve from Fig.1.
Our future work would encompass a generalization of the proposed framework to include detection and prediction of other social and behavioral processes that can be described by similar population models (computer worm attacks, propaganda campaigns, complex social engagements, sensor networks, quorum sensing, etc, see [23], [12], [34]).

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References

[1] R. M. Anderson, C. Fraser, and A. C. Ghani. Epidemiology, transmission dynamics and control of SARS: the 2002-2003 epidemic. *Philos. Trans. R. Soc. B Biol. Sci.*, 359:1091–1105, 2004.

[2] R. M. Anderson and R. M. May. Population biology of infectious diseases: Part 1. *Nature*, 280:361–367, 1979.

[3] M. S. Arulampalam, S. Maskell, N. Gordon, and T. Clapp. A tutorial on particle filters for non-linear/non-Gaussian Bayesian tracking. *IEEE Trans. Signal Processing*, 50(2):174–188, Feb. 2002.

[4] C. Fraser at al. Pandemic potential of a strain of influenza A (H1N1): Early findings. *Science*, 324(5934):1557 – 1561, 2009.

[5] S. Cauchemez and N. M. Ferguson. Likelihood-based estimation of continuous-time epidemic models from time-series data: Application to measles transmission in London. *J. R. Soc. Interface.*, 5(25):885–897, 2008.

[6] B. Cazelles and N. P. Chau. Using the kalman filter and dynamic models to assess the changing HIV/AIDS epidemic. *Mathematical Biosciences*, 140(2):131–154, 1997.

[7] A. Culotta. Detecting influenza outbreaks by analyzing twitter messages. In *Proc. 2010 Conf. on Knowledge Discovery and Data Mining*, Washington, D.C., 2010.

[8] D.J. Daley and J. Gani. *Epidemic Modelling*. Cambridge Univ. Press, 1996.

[9] C. E. Dangerfield, J. V. Ross, and M. J. Keeling. Integrating stochasticity and network structure into an epidemic model. *J. R. Soc. Interface*, 6(38):761–774, 2009.

[10] C. Dargatz. A diffusion approximation for an epidemic model. Technical report, Ludwig-Maximilian Universitat Munchen, 2007.

[11] C. Egat, F.C. Arrat, C. L. Ajaunie, and H. Wackernagel. Early detection and assessment of epidemics by particle filtering. In *Proc. 6th Europ. Conf. Geostatistics for Environmental Applications (GeoENV VI)*, pages 23–35, Rhodes, Greece, 2008. Springer.

[12] S. Eubank, V. S. A. Kumar, and M. Marathe. *Epidemiology and wireless communication: Tight analogy or loose metaphor?* Springer, 2008.
[13] G. Eysenbach. Infodemiology: Tracking flu-related searches on the web for syndromic surveillance. In Proc. 2006 Symp of American College Med. Inf. Ass. (AMIA 2006), pages 244–248, 2006.

[14] J. Ginsberg, M.H. Mohebbi, R. S. Patel1, L. Brammer, M. S. Smolinski, and L. Brillant. Detecting influenza epidemics using search engine query data. Nature, 457:1012–1015, 2009.

[15] N. J. Gordon, D. J. Salmond, and A. F. M. Smith. Novel approach to nonlinear/non-Gaussian Bayesian state estimation. IEE Proc.-F, 140(2):107–113, 1993.

[16] A. H. Jazwinski. Stochastic Processes and Filtering Theory. Academic Press, 1970.

[17] C. Jégat, F. Carrat, C. Lajaunie, and H. Wackernagel. Early detection and assessment of epidemics by particle filtering. In A. Soares, M. J. Pereira, and R. Dimitrakopoulos, editors, geoENV VI Geostatistics for Environmental Applications, volume 15, pages 23–35. Springer, 2008.

[18] G. Kitagawa. Monte Carlo filter and smoother for non-Gaussian non-linear state space models. Journal Of Computational and Graphical Statistics, 5(1):1–25, 1996.

[19] L. Dailey, R. E. Watkins, and A. J. Plant. Timeliness of data sources used for influenza surveillance. J. Am. Medical Inf Ass., 14(5):177185, 2007.

[20] R. Mahler. Statistical Multisource Multitarget Information Fusion. Artech House, 2007.

[21] J. Mandela, J. D. Beezleya, L. Cobba, and A. Krishnamurthy. Data driven computing by the morphing fast Fourier transform ensemble Kalman filter in epidemic spread simulations. Procedia Computer Science, 1(1):1221–1229, 2010.

[22] Z. R. Mnatsakanyan, H. S. Burkom, M. R. Hashemian, and M. A. Coletta. Distributed information fusion models for regional public health surveillance. Information Fusion, 12(2):doi:10.1016/j.inffus.2010.12.002, 2011.

[23] Y. Moreno, M. Nekovee, and A. Vespignani. Efficiency and reliability of epidemic data dissemination in complex networks. Phys. Rev. E, 69(5):055101–1–5, 2004.

[24] A. S. Novozhilov. On the spread of epidemics in a closed heterogeneous population. Mathematical Biosciences, 215(2):177185, 2008.

[25] J. B. S. Ong, M. -C. Chen, A. R. Cook, H. C. Lee, V. J. Lee, R. T. P. Lin, P. A. Tambyah, and L. G. Goh. Real-time epidemic monitoring and forecasting of H1N1-2009 using influenza-like illness from general practice and family doctor clinics in singapore. PLoS ONE, 5(4):e10036, 2010.

[26] N. Oudjane and C. Musso. Progressive correction for regularized particle filters. In Proc. 3rd Int. Conf. Information Fusion, Paris, France, 2000.

[27] B. Ristic. Bayesian estimation with imprecise likelihoods: Random set approach. IEEE Signal Processing Letters, 2011. In review.

[28] B. Ristic, S. Arulampalam, and N. Gordon. Beyond the Kalman filter. Artech House, 2004.

[29] B. Ristic, A. Skvortsov, and M. Morelande. Predicting the progress and the peak of an epidemic. In Proc. IEEE Int. Conf. Acoustics, Speech and Signal Processing (ICASSP 2009), pages 513–516, Taipei, Taiwan, April 2009.

[30] M. Roy and M. Pascual. On representing network heterogeneities in the incidence rate of simple epidemic models. J. Ecological Complexity, 3(1):80–90, 2006.

[31] N. M. Schuster, M.A. Rogers, and L.F. Jr. Using search engine query data to track pharmaceutical
utilization: A study of statin. *The American Journal of Managed Care*, 16(8):e215–e219, 2010.

[32] A. Skvortsov, R. Connell, P. Dawson, and R. Gailis. Epidemic modelling: Validation of agent-based simulation by using simple mathematical models. In *International Congress on Modelling and Simulation (MODSIM 2007)*, pages 657–662, Christchurch, New Zealand, December 2007.

[33] A. Skvortsov, R. Connell, P. Dawson, and R. Gailis. Epidemic spread modeling: Alignment of agent-based simulation with a simple mathematical model. In *Proc. Int. Conf. Bioinformatics & Comput. Biology*, pages 487–890, Las Vegas, USA, June 2007. CSREA Press.

[34] A. Skvortsov, B. Ristic, and M. Morelande. Networks of chemical sensors: A simple mathematical model for optimisation study. In *Proc. 5th International Conference on Intelligent Sensors, Sensor Networks and Information Processing (ISSNIP 2009)*, pages 385–390, Melbourne, Australia, 2009.

[35] A Skvortsov, B Ristic, and C Woodruff. Predicting an epidemic based on syndromic surveillance. In *Proc. 13th Int. Conf. Information Fusion*, Edinburgh, UK, July 2010.

[36] P. D. Stroud, S. J. Sydoriak, J. M. Riese, J. P. Smith, S. M. Mniszewski, and P. R. Romero. Semi-empirical power-law scaling of new infection rate to model epidemic dynamics with inhomogeneous mixing. *Mathematical Biosciences*, 203:301–318, 2006.

[37] O. A. van Herwaarden and J. Grasman. Stochastic epidemics: Major outbreaks and the duration of the endemic period. *J. Math. Biology*, 33(4):581–601, 1995.

[38] M. Wagner, A. Moore, and R. Aryel. *Handbook of Biosurveillance*. Elsevier, 2006.

[39] J. Walden and E. H. Kaplan. Estimating time and size of bioterror attack. *Emerging Infectious Diseases*, 10(7):1202–1205, July 2004.

[40] A.G. Wilson, G.D. Wilson, and D.H. Olwell. *Statistical Methods in Counterterrorism: Game Theory, Modeling, Syndromic Surveillance, and Biometric Authentication*. Springer, 2006.