Detectability limits of epidemic sources in networks

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Abstract

The detection of an epidemic source or the patient zero is an important practical problem that can help in developing the epidemic control strategies. In this paper, we study the statistical inference problem of detecting the source of epidemics from a snapshot of a contagion spreading process at some time on an arbitrary network structure. By using exact analytic calculations and Monte Carlo simulations, we demonstrate the detectability limits for the SIR model, which primarily depend on the spreading process characteristics. We introduce an efficient Bayesian Monte Carlo source probability estimator and compare its performance against state-of-the-art approaches. Finally, we demonstrate the applicability of the approach in a realistic setting of an epidemic spreading over an empirical temporal network of sexual interactions.

Introduction

The majority of biological, technological, social and information systems structures can be represented as a complex network \cite{1,2,3}. The most prevalent type of dynamic processes of public interest characteristic for the real-life complex networks are contagion processes \cite{4}. Different mathematical methods have been used to study the epidemic spreading on complex networks including the bond percolation method \cite{7,8}, the mean-field approach \cite{9,10}, the reaction-diffusion processes \cite{11,12}, pair and master equation approximations \cite{13} as well as models with the complex compartmental structure along with population mobility dynamics \cite{14}.

Epidemiologists detect the epidemic source or the patient-zero either by analysing the temporal genetic evolution of virus strains \cite{5} or try to do a contact backtracking \cite{6} from the available observed data. However, in cases where the information on the times of contact is unknown, or incomplete, the backtracking method is no longer adequate. This
becomes especially hard if the only data available is a static snapshot of an epidemic process at some time. Even in cases when we do have some information on the times of contact, the longer the recovery time and subtler the symptoms the harder it becomes to establish the proper ordering of the transmissions that have occurred. Due to its practical aspects and theoretical importance, the epidemic source detection problem on contact networks has recently gained a lot of attention in complex network science community. This has led to the development of many different source detection estimators for static networks, which vary in their assumptions on the network structure and the spreading process models [15, 16, 17, 18, 19, 20, 21, 22, 23].

For the source detection with the SI model the following interesting results have been obtained. Zaman et. al. developed a rumor centrality measure, which is the maximum likelihood estimator for regular trees under the SI model [15]. Dong et. al. also studied the problem of rooting the rumor source with the SI model and demonstrated the asymptotic source detection probability on regular tree-type networks [16]. Comin et. al. compared the different centrality measures e.g. the degree, the betweenness, the closeness and the eigenvector centrality as the source detection estimators [22]. Wang et. al. addressed the problem of source estimation from multiple observations under the SI model [17]. Pinto et. al. used the SI model and assumed that the direction and the times of the infection are known exactly, and solved diffusion tree problem using breadth first search from sparsely placed observers [19].

In the case of the SIR model there are two different approaches. Zhu et. al. adopted the SIR model and proposed a sample path counting approach for the source detection [18]. They proved that the source node on infinite trees minimizes the maximum distance (Jordan centrality) to the infected nodes. Lokhov et. al. used a dynamic message-passing algorithm (DMP) for the SIR model to estimate the probability that a given node produces the observed snapshot. They use a mean-field-like approximation (independence approximation) and an assumption of a tree-like contact network to compute the marginal probabilities [20].

The main contributions of the paper are the following:

(i) given the non-uniqueness of finding a single epidemic source of the SIR realization on general networks, we turn the problem to finding a source probability distribution, which is a well-posed problem;

(ii) we develop the analytic combinatoric and the direct Monte-Carlo approaches for determining theoretical source probability distribution and produce the benchmark solutions on the 4-connected lattice;

(iii) we measure the source detectability by using the normalized Shannon entropy of the estimated source probability distribution for each of the source detection problems and observe the existence of the highly detectable and the highly undetectable regimes;

(iv) using the above insights, we construct the Soft Margin epidemic source detection estimator for the arbitrary networks (static and temporal) and show that it is robust and more accurate than the state-of-the-art approaches and much faster than the analytic combinatoric or the direct Monte-Carlo approach;

(v) by using the simulations of the sexually transmitted disease (STD) model on a realistic time interval of 200 days on an empirical temporal network of sexual contacts (see the network visualization in Figure 4, plot C) we demonstrate the robustness to the uncertainty in the epidemic starting time, the network interaction orderings and in incompleteness of observations.

Although we use the SIR model of epidemic spreading, our algorithms are easily applicable to other compartmental models, e.g. SI and SEIR and all other compartmental models where the states cannot be recurrent.

1 Detectability limits

The main goals of this work are to better understand the nature of the epidemic source detection problem in networks, characterize its complexity, and develop efficient algorithms for estimating source probability distribution. Next, we introduce the terminology and formalize the problem. In a general case, the contact-network during an epidemic process can be temporal
and weighted, but first we will concentrate our analysis on a static undirected and non-weighted network $G(V,E)$, where $V$ denotes the set of nodes or vertices and $E$ denotes the set of links or edges. The random vector $\vec{R} = (R(1), R(2), \ldots, R(N))$ indicates which nodes got infected up to a certain time $T$. The random variable $R(i)$ is a Bernoulli random variable, which has the value of 1 if the node $i$ got infected before the time $T$ and the value of 0 otherwise. We will use the SIR model as the main contagion model, which is parametrized by the probability $p$ that an infected node infects a susceptible neighbour node and by the probability $q$ that an infected node recovers in one discrete time step. Let us assume that we have observed one epidemic realization $\vec{r}^*_{\vec{G}}$ of $\vec{R}$ at a time $T$ of the SIR process $(p,q,T)$ on a network $G$, and we want to infer which nodes are the most likely to be the source of realization $\vec{r}^*_{\vec{G}}$ for the given SIR process and network. The set of possible source node candidates $S = \{\theta_1, \theta_2, \ldots, \theta_m : \vec{r}^*(\theta_i) = 1\}$ is determined by the union of the infected and the recovered nodes at the time $T$. The node with the highest posterior probability for being the source of the epidemic spread for a given realization $\vec{r}^*$ is $\Theta_{MAP} = \arg \max_{\theta_i \in S} P(\Theta = \theta_i | \vec{R} = \vec{r}^*_{\vec{G}})$. By applying the Bayes theorem, we get the following expression:

$$P(\Theta = \theta_i | \vec{R} = \vec{r}^*) = \frac{P(\vec{R} = \vec{r}^*) P(\Theta = \theta_i)}{\sum_{\theta_k \in S} P(\vec{R} = \vec{r}^*) P(\Theta = \theta_k)}$$

(1)

Unless stated otherwise, due to the simplicity of the notation in the rest of the manuscript we do not put the following variables: $p,q,T,G$ to the condition $P(\Theta = \theta_i | \vec{R} = \vec{r}^*, T, p, q, G)$. Thus, the core of the source detection problem is the determination of the source probability distribution over nodes that have been infected/recovered in a given observed epidemic realization. For simplicity, we will assume that all nodes have the same prior probability and therefore the maximum posterior probability node is equal to the maximum likelihood node, but the methodology is also applicable in cases when the source prior probabilities are not uniform. Next, we proceed with construction of the ideal algorithm, i.e. a procedure for the calculation of the exact probability distribution over the nodes in the realization $\vec{r}^*_{\vec{G}}$. The development of this kind of algorithm is thus necessary both with respect to obtaining main insights about detectability limits and for proper assessment of other algorithms. Here we describe two complementary approaches to such an algorithm: direct Monte-Carlo simulations and analytical calculations.

With the direct Monte-Carlo simulation approach, for each node $i$ from the set $S$ of the realization $\vec{r}^*$, a large number $n$ of epidemic spreading simulations with duration $T$ is performed with $i$ as an epidemic source. The number of simulations $n_i$ which coincides with the realization $\vec{r}^*_{\vec{G}}$ is recorded. After the simulations for all potential nodes in the realization $\vec{r}^*$ are finished, the probability of the node $i$ being the source of the epidemic is calculated as

\[ P(\vec{R} = \vec{r}^*, \Theta = \theta_i) = \frac{\text{number of simulations where node } i \text{ is the source}}{\text{total number of simulations}} \]
If the size of the realization $\bar{r}_s$ is big, the number of simulations required to obtain reliable frequencies can be very large. As the estimation for different nodes is independent, the computations are done in parallel by using a high performance Message Passing Library with the C++ language and Igraph library [28]. Furthermore, we have employed a pruning mechanism for the SIR model, where the Monte Carlo simulation is stopped at time step $t < T$ if the current simulated realization has infected a node which has not been infected in $\bar{r}_s$. This pruning mechanism provides a substantial acceleration and, what is more important, does not induce any errors to our estimation (for more information, see supplementary material section 1). The statistical significance of direct Monte-Carlo simulation results are controlled with independent simulations, where $n$ is usually in the range $10^6 - 10^9$. Then, we choose the ML node as the node with the highest probability in $(P^n_i)$. The PDFs which satisfy the two following conditions:

$$|P_{ML}^{2n} - P^n_i|/P_{ML}^{2n} \leq c, |P^n_i - P^{2n}_i| \leq c,$$

(2)

are said to converge with the direct Monte Carlo method. The relative error convergence with the value $c$ on all the nodes is a too strict computational condition for practical purposes as we are interested in finding the high probability source nodes.

The alternative approach, the analytical combinatoric approach assigns to each node of degree $n$ a generating function which is maximally $(n+1)$-dimensional, which captures the events of node first infection and infection spreading through its edges at specific times. Then, by combining the generating functions of all the infected nodes from a realization, we are able to merge all contributions together and get the source probability distribution $P(\Theta = \theta_i|\bar{R} = \bar{r}_s)$. The detailed description of analytical combinatoric method can be found in supplementary material section 1.

Now, we proceed with a synthetic example (see Figure 1) where we demonstrate ill-posedness of finding a single true source. By ill-posedness of the problem in this case we mean possible non-uniqueness of the solution, i.e. it is possible that the same epidemic realization has been generated from several or even many nodes (in this case: $\{v_7, v_{10}, v_{11}, v_{12}, v_{15}, v_{19}\}$). On the contrary, the problem of finding the source probability distribution $P(\Theta = \theta_i|\bar{R} = \bar{r}_s)$ is a well-posed problem. In this example, we show that the direct Monte-Carlo simulations and analytical calculations are in excellent agreement (maximal absolute difference $< 10^{-10}$). The synthetic realization $\bar{r}_s$ belongs to the SIR contagion process ($p = 0.2, q = 0.3, T = 5$) on a small 4-connected lattice. The source probability distribution is calculated by analytical combinatoric method with generating functions and verified with direct Monte Carlo estimator with $n = 10^9$ simulations per node. A serious disadvantage of the analytical method is that for configurations which are not tree-like, the analytical calculations become prohibitively intricate. Therefore, after validating the agreement between two approaches, we continue with the direct Monte-Carlo approach as an ideal algorithm, which can easily be evaluated on non-tree like realizations.

Next, we introduce the normalized Shannon entropy $H$ of the source probability distribution, as a measure of source detectability for a particular realization:

$$\frac{-1}{H_m(\bar{r}_s)} \sum_{\theta_i \in S} P(\Theta = \theta_i|\bar{R} = \bar{r}_s) \ln(P(\Theta = \theta_i|\bar{R} = \bar{r}_s)).$$

(3)

In this expression, the Shannon entropy of the source probability distribution is normalized with the maximum possible entropy of particular realization $H_m(\bar{r}_s)$, which is uniform distribution over all potential candidates in $S$. We have estimated the average $H$ over different epidemic realizations in four distinct $(p, q)$ regions on the 4-connected lattice ($N = 30 \times 30$ nodes), to demonstrate the existence of highly detectable (Figure 2 plot C and D) and highly undetectable (Figure 2 plot A and B) regimes in different parts of the SIR model parameters’ space.

## 2 Soft Margin estimator

In this section we proceed to construct an estimator which would be able to sufficiently resemble the
characteristics of the ideal algorithm, i.e. to find an approximate source probability distribution, but with far less computational power than the direct Monte-Carlo method or the analytical combinatorics method. Next, we introduce some useful definitions. The $i$-th sample of the random variable $R_\theta$ is denoted with $r_{\theta,i}$ and it is obtained with the Monte Carlo simulation of the contagion process. In general, the similarity function $\varphi : (R^N \times R^N) \rightarrow [0,1]$ compares two realizations of dimension $N$ and outputs a real number in interval $[0,1]$. We will use the Jaccard similarity function $\varphi(r^*_i, r^*_j)$, which is the size of the intersection of sets of infected nodes in $r^*_i$ and $r^*_j$ normalized by the size of the union of sets of infected nodes in $r^*_i$ and $r^*_j$. The random variable $\varphi(r^*_i, R_\theta)$ measures the similarity between a fixed realization $r^*_i$ and a random realization that comes from $SIR$ process with the source $\theta$. The $i$-th sample of the random variable $\varphi(r^*_i, R_\theta)$ is denoted with $\varphi(r^*_i, r_{\theta,i})$ or sometimes simply with $\varphi_i$. The cumulative distribution function of the random variable $\varphi(r^*_i, R_\theta)$ is denoted with $F_\theta(x)$, where $x$ denotes the value the a similarity variable. The unbiased estimator of $F_\theta(x)$ from $n$ samples $\varphi(r^*_i, r_{\theta,i})$ of the random variable $\varphi(r^*_i, R_\theta)$ is the following empirical distribution function:

$$
\hat{F}_\theta(x) = \hat{P}(\varphi(r^*_i, R_\theta) \leq x) = \frac{1}{n} \sum_{i=1}^{n} 1_{[0,x)}(\varphi(r^*_i, r_{\theta,i}))
$$

(4)

where $1_{[0,x)}(y)$ is a function defined to be 1 if $y \in [0, x)$ and 0 otherwise. The probability density function is the derivative of cumulative distribution function:

$$
f_\theta(x) = \frac{d}{dx} \hat{F}_\theta(x) = \frac{1}{n} \sum_{i=1}^{n} \delta(x - \varphi(r^*_i, r_{\theta,i}))
$$

(5)

where $\delta(x)$ denotes the Dirac delta distribution. Now, we define the **Soft Margin estimator** with the following formula:

$$
\hat{P}(R = r^*_i | \Theta = \theta) = \int_{0}^{1} w_\theta(x) f_\theta(x)dx,
$$

(6)

where $w_\theta(x)$ is a weighting function and $f_\theta(x)$ is the PDF function of the random variable $\varphi(r^*_i, R_\theta)$. We
use the following Gaussian weighting form: \( w_a(x) = e^{-(x-1)^2/a^2} \). In the limit where the parameter \( a \to 0 \), we obtain the unbiased estimate (direct Monte Carlo method) of the likelihood \( P(\vec{R} = \vec{r}^*_n | \Theta = \theta) \). For cases when the parameter \( a > 0 \), we obtain an estimator which estimates the likelihood by using the Soft Margin function \( w_a(x) \) to accept a contribution of a specific realization \( \vec{r}_{\theta,i} \), contrary to the unbiased estimate (=0) which rejects the contribution of all realizations that are not the same as the observed realization \( \vec{r}^*_n \). The time complexity analysis for the Soft Margin estimator is given in the supplementary materials section 6 and the Soft Margin implementation details are given in the supplementary section 5.

The motivation for the Soft Margin was the following: we turn the problem of choosing the realization with the similarity \( \varphi = 1 \) to the problem of choosing realizations with the similarity in the interval where the contributions drops with the Gaussian function \( w_a(x) \) from the point \( \varphi = 1 \). Using the property of delta distribution, we simplify the expression for the Soft Margin estimator to:

\[
\hat{P}(\vec{R} = \vec{r}_n | \Theta = \theta) = \frac{1}{n} \sum_{i=1}^{n} e^{-\frac{(\varphi(\vec{r}_n, \vec{r}_{\theta,i}) - 1)^2}{a^2}}. \tag{7}
\]

Note that the similarity samples \( \varphi(\vec{r}^*_n, \vec{r}_{\theta,i}) \) are in the interval \([0, 1]\), e.g. if the \( i \)-th simulated realization \( \vec{r}^*_n \) is identical to observed realization \( \vec{r}^*_n \) then their similarity is 1.

We can relax the assumption on parameters we need by using marginalization of conditional probabilities. By marginalizing the temporal duration parameter \( T \), we get the following expression for \( \hat{P}(\vec{R} = \vec{r}_n | \Theta = \theta_i) \)

\[
\hat{P}(\vec{R} = \vec{r}_n | \Theta = \theta_i) = \sum_{T_j} P(\vec{R} = \vec{r}_n | \Theta = \theta_i, T = T_j) P(T = T_j | \Theta = \theta_i), \tag{8}
\]

and if we have a prior knowledge on the unknown variable \( T \), we can use it in our estimation procedure as well. We will use this method to find the source node when the duration of epidemic is not known in advance.

Finally, we do not need to set the Soft Margin width parameter \( a \) in advance. After we calculate the estimated PDF for every potential source \( \vec{r}_\theta(x) \), we can choose the parameter \( a \) as the infimum of the set of parameters for which the PDFs have converged. In experiments on temporal networks, for each parameter \( a \) in range \((1/2, (1/2)^2, (1/2)^3, ..., (1/2)^{15})\) we estimate the probability of ML node: \( P_{ML}^{n_1} \) and \( P_{ML}^{n_2} \) with \( n_1 = 15000 \) and \( n_2 = 20000 \) simulations and set the convergence condition to be: \( |P_{ML}^{n_1} - P_{ML}^{n_2}| / P_{ML}^{n_2} \leq 0.05 \). The time complexity analysis for the Soft Margin estimator is given in the supplementary materials section 6 and the Soft Margin implementation details are given in the supplementary section 5.

3 Results

In this section we: (i) make a comparison of the source detection estimators on controlled benchmark cases on a static network and (ii) demonstrate the applicability of the Soft Margin estimator on an empirical temporal network of sexual contacts.

3.1 Comparison of estimators on a static benchmark network

In order to do a proper comparison of different source detection estimators, there has to exist a proper measure of quality of solution. Because of the non-uniqueness of a single source node we will not compare the estimators by their ability to detect the true source, but instead by comparing their estimated source probabilities to the source probability distribution of the ideal algorithm. We have generated a series of benchmark cases for which we have calculated the probability distributions over the potential source candidates using the direct Monte Carlo estimator. Note that the direct Monte Carlo estimator has been validated by comparison with the analytical solution (see Figure 1 and supplementary materials section 1). In order to be sure that the direct Monte Carlo estimator outputs valid results on realizations with cycles, we set its convergence condition (see formula 2) to \( c = 0.05 \).

We have used a small 4-connected lattice \((N = 30 \times 30)\) and SIR processes with different parameters \((p, q, T)\) with the direct Monte Carlo estimator.
with \([10^6 - 10^8]\) simulations per source, depending on the convergence condition, to obtain the source PDFs for our benchmark. In the supplementary materials section 4, we provide a detailed explanation of the benchmark dataset with 160 benchmark examples and the public URL for download. Next, we compare the representatives of three different classes of source detection estimators: network centrality estimators, belief propagation estimators and Monte Carlo estimators. For the network centrality estimation, we use the Jordan estimator [18], which assigns a weight to each potential node candidate which is equal to the maximal topological distance from the node candidate to all other infected nodes in a realization. Although the Jordan estimator uses a very simple rule, it outperforms most of other network centrality measures. The representative of the belief propagation estimators is the Dynamic Message Passing Algorithm (DMP) [20], which uses a mean-field-like approximation (independence approximation) about the node states along with a recursive analytical formula for the tree-like networks to estimate the source likelihoods. The authors of [20] compare performance of the DMP algorithm to other approaches like rumor [15], distance and Jordan centrality [18] and demonstrate superior performance for the problems on tree-like contact networks. More details about our implementations of the Jordan and DMP estimators are given in the supplementary material section 3. Finally, we use our Soft Margin estimator which falls into the general class of the Monte Carlo estimators. Note that, when comparing different Monte Carlo estimators, we evaluate them with a few orders of simulations less than the number of simulations used to generate the benchmark standard solution. For each estimator, we measure the ML accuracy performance and ML probability estimate error. ML accuracy measures the expected number of times in which the estimators rank the ML node as rank 1 and relative ML error measures the ability to reconstruct the ML node probability. In Figure 3, we can see the mean relative errors and the accuracy of the ML node for different estimators. From this analysis, we observe that the most estimators are trying to produce a valid ranking (ML accuracy) without estimating the true probability (ML relative error). The Soft Margin estimator is trying to estimate both the valid ranking and a valid probability at the same time. The source probability distribution for the observed realization contains more information about the initial conditions than just the ranking of potential candidates, especially for cases where the detectability limits are more pronounced. The detailed performance analysis of this and other estimators in specific \((p, q)\) regimes as well as the influence of the Soft Margin weights \(a\) can be found in supplementary material section 3.

3.2 Empirical temporal network case study

Now, we demonstrate the applicability of our inference framework to detect the source of the simulated STD epidemic spreading in an empirical temporal network of sexual contacts in Brazil (see Figure 4, plot C). We would like to note that this publicly available dataset [26] was obtained from Brazilian Internet community and it is used as an approximation of temporal sexual contacts (see the Disclaimer section). The dataset consists out of the triplets \((v_i, v_j, t)\), which represents the event that the nodes \(v_i\) and \(v_j\) had a sexual interaction at a time \(t\). We took a sample of this dataset between days \([1000 - 1800]\) \((\approx 3.5k\) nodes), very similar to the authors [26] in the original study. In this case we use a temporal network with the SIR model of the STD \((p = 0.3, q = 0.01)\). The upper limit of the transmission probability for the STD disease that was previously used on this contact network is \(p = 0.3\) [26]. The recovery parameter \(q = 0.01\) represents a disease with the mean recovery of 100 days.

Note that here the calculation of exact source probability distributions is computationally too demanding with the direct Monte Carlo or Analytical Combinatorics method. Therefore, we use the Soft Margin estimator with the smallest width \(a\) for which the ML node probability estimate converged and measure how well we can detect the true source (as we do not have an exact solution). Our experiments consist of two parts: (i) simulation of STD spreading through a temporal network of sexual contacts and (ii) detection of the patient zero from the observed process. Realizations in (i) are generated by using the STD
Figure 3: Comparison of different estimators (Network centrality (Jordan), Belief Propagation (DMP) and Monte Carlo (Soft Margin)) performance with the Maximum Likelihood relative probability estimation error (plot A) and Maximum Likelihood accuracy (plot B) with respect to the 160 different benchmark cases. Benchmark cases were calculated on a small 4-connected lattice with \( N = 30 \times 30 \) and SIR process with different parameters \( (p, q, T) \) with the direct Monte Carlo estimator with \( 10^8 \) simulations per source depending on convergence condition \( (2) \) with \( c = 0.05 \).

model on the exact temporal contact network, where the patient zero is a randomly selected active node at time point \( t_0 \) and epidemic observed at time \( t \). In detection process (ii), we assume that we know the STD model parameters, the realization vector \( r^*_s \) at time \( t \), but we relax the assumption on knowing the duration of the epidemic \( T = t - t_0 \) or the epidemic start moment \( t_0 \) and exact ordering times of temporal contacts in the network.

The relaxation of knowing the starting point of the epidemic \( t_0 \) is done by using the marginalization formula \( (8) \), where we sample over all possible starting points \( t_0 \) from a uniform probability distribution over \( [t_0 - \epsilon, t_0 + \epsilon] \), \( 2\epsilon = \{0, 50, 100\} \) days. In Figure 4 plot A, we show the results when the starting time \( t_0 \) was chosen from interval \( [100 - 200] \) days, the end of the epidemic was set to the day \( t = 300 \) and we have used different uniform priors \( (\epsilon) \) for the moment \( t_0 \). When the \( \epsilon = 50 \) days of uniform uncertainty, we can still detect the source within its first neighbourhood with probability of approximately 0.6 and within first 10 ranked nodes with probability of approximately 0.8. These results are of great practical importance, since in reality we do not know the exact starting times, but rather only an upper and a lower bound on starting point and we are still able to detect the source.

Next, we argue that the temporal ordering of interactions in this network plays an important role in estimating the source node and its detectability. Furthermore, it is very unrealistic to have exact temporal information about contact interactions in real life. Therefore, we have made a series of experiments where, for a given realization from a real temporal network, we try to estimate the source node in a case of uncertainty of the orderings of the contact times within a time window of the length \( \Delta \). We use a randomization algorithm which permutes time stamps inside of a bin of \( \Delta \) days from the start to the end of the contact interaction network in a non-overlapping way. Therefore, this randomization permutes all time stamps that fall to the following temporal windows: \( [0, \Delta - 1], [\Delta, 2\Delta - 1], ..., [k\Delta, t] \) of length \( \Delta \). The intuition behind this randomization is that, in reality, usually the data gathering procedure cannot guarantee ordering of temporal interactions smaller than some granularity of \( \Delta \) days so that all orderings inside
Figure 4: Source detection of simulated sexually transmitted infections spreading in an empirical temporal network of sexual contacts in Brazil. The experiment consists out of 500 experiments where the number of infected nodes is least 1% of network sample (≈ 3.5k nodes) and the initial moment $t_0$ was chosen in period between [100 – 200] days, the initial source was randomly selected from the set of active nodes at moment $t_0$ with the SIR STD model ($p = 0.3, q = 0.01$) and realization $r^*_s$ was observed at time $t = 300$ day. Mean number of infected nodes in 500 experiments is 77.3 and mean diameter of infected subgraph was 7.4. Figure A: The influence of prior knowledge about initial outbreak moment $[t_0 - \epsilon, t_0 + \epsilon]$ with $\Delta = 0$ (no randomization on network time stamps). Average execution time (per realization) to calculate source probability distribution over all potential candidates was around 12 seconds (on 50 cpu cores). Figure B: The influence of detecting the source node from temporal networks with randomized temporal ordering of interactions within $\Delta$ days, with $\epsilon = 0$ (we know the staring time $t_0$). Figure C: Visualization (using Gephi [27]) of aggregated empirical temporal network of sexual contacts in Brazil [26], on which the case study has been performed.
\( \Delta \) days become equally likely. From Figure 4, plot B, we observe that higher uncertainty in orderings (higher \( \Delta \)) reduces the detectability of the source of infection. But it is also important to notice, that our estimations are robust to the perturbation of small sizes. Results for source detection for different values of \((p, q)\) parameters and for the case when only a random subset of the node states is observed can be found in supplementary materials section 7.

4 Discussion and conclusion

Recently, different solutions have been proposed for the problem of epidemic source detection in the network setting. These solutions are based on a number of assumptions on the contact network structures and spreading models, but there is still a big gap from applying these methods to realistic case studies. In contrast to these approaches, our methodology is applicable to a more general and realistic setting, and is limited solely by the ability to produce sample realizations of the particular contagion process by simulations. Besides this methodological advancement, there are more specific contributions which include an analytic and Monte Carlo approaches for calculating the proper source probabilities. Moreover, we identified different source detectability regimes for the SIR model and produced datasets with near exact source probability distributions, that can serve as benchmark problems for future algorithmic developments. We have also shown that our Soft Margin algorithm estimates source probabilities with much higher precision than other estimators on static network by comparing the results against near exact source probability distributions on benchmark cases.

For the first time, we have demonstrated the ability to detect the source node of the simulated STD epidemics on a realistic time scale of approximately 200 days on an empirical temporal network of sexual contacts - even with the uncertainty in the epidemic starting time, the network interaction orderings and incompleteness of observations. A high detectability of the first neighbourhood can give insights and directions to the epidemiologists about the possible location where to start a field investigation. Furthermore, the execution times for detecting the source node from a single realization on this empirical temporal network are relatively low, under one minute on the machine with 50 cpu cores. Since most of the real social networks exhibit temporal behaviour and since our methodology is not dedicated to a particular spreading process, the potential applicability varies from epidemics to rumor and information propagations, where the detectability or undetectability of the initial source can have serious implications to privacy issues in future. Applications of this methodology along with the use of the effective distance measure \([23]\) to the complex meta-population spreading models is not within the scope of this research and is left for future work. We would also like to motivate the research community into studying the phase transitions of detectability for different spreading models or with different network topologies.

Disclaimer

The authors of this study used the published existing dataset of sexual contacts in high-end prostitution because it contains valuable and rarely available information on temporal network of contacts serving as pathways of STD spreading. It is important to note that the use of this dataset does not reflect the authors’ views, opinions and attitudes on prostitution and it does not in any way imply that the authors support the activities documented in the dataset or the way the data were gathered.

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References

[1] Newman MEJ (2003) The Structure and Function of Complex Networks. SIAM Review 45: 167–256.

[2] Dorogovtsev SN, Goltsev AV, Mendes JFF (2008) Critical phenomena in complex networks. Reviews of Modern Physics 80: 1275–1335.

[3] Boccaletti S, Latora V, Moreno Y, Chavez M, Hwang DU (2006) Complex networks: Structure and dynamics. Phys Rep 424: 175-308.

[4] Vespignani A (2012) Modelling dynamical processes in complex socio-technical systems. Nat Phys 8: 32–39.

[5] Worobey, M., Han, G.-Z. and Rambaut, A. (2014) Genesis and pathogenesis of the 1918 pandemic H1N1 influenza A virus. Proc. Natl. Acad. Sci. USA: 201324197+

[6] Auerbach D M and Darrow W W and Jaffe H W and Curran J W (1984) Cluster of cases of the acquired immune deficiency syndrome: Patients linked by sexual contact. The American Journal of Medicine 76, 3 (1984) .

[7] Mollison D (1977) Spatial contact models for ecological and epidemic spread. Journal of the Royal Statistical Society Series B (Methodological) 39: 283–326.

[8] Kenah E, Robins JM (2007) Second look at the spread of epidemics on networks. Phys Rev E 76: 036113.

[9] Castellano C, Pastor-Satorras R (2010) Thresholds for epidemic spreading in networks. Phys Rev Lett 105: 218701.

[10] Pastor-Satorras R, Vespignani A (2001) Epidemic spreading in scale-free networks. Phys Rev Lett 86: 3200–3203.

[11] Colizza V, Pastor-Satorras R, Vespignani A (2007) Reaction–diffusion processes and metapopulation models in heterogeneous networks. Nat Phys 3: 276–282.

[12] Colizza, V., Barrat, A., Barthlomew, M., Vespignani, A. (2006) The role of the airline transportation network in the prediction and predictability of global epidemics. Proc. Natl. Acad. Sci. USA 103: 2015.

[13] Gleeson JP (2013) Binary-state dynamics on complex networks: Pair approximation and beyond. Phys Rev X 3: 021004.

[14] Broeck W, Gioannini C, Goncalves B, Quaggiotto M, Colizza V, et al. (2011) The gleamviz computational tool, a publicly available software to explore realistic epidemic spreading scenarios at the global scale. BMC Infectious Diseases 11, 37 (2011) .

[15] Shah D, Zaman T (2010) Detecting sources of computer viruses in networks: theory and experiment. In: Proc. of the ACM SIGMETRICS 2010.

[16] Dong W, Zhang W, Tan CW (2013) Rooting out the rumor culprit from suspects. Proc. of IEEE Intl. Symp. on Information Theory 2013 .

[17] Wang Z, Dong W, Zhang W and Tan C W (2014) Rumor Source Detection with Multiple Observations: Fundamental Limits and Algorithms. Proc. of ACM SIGMETRICS 2014.

[18] Zhu K, Ying L (2013) Information source detection in the sir model: A sample path based approach. Information Theory and Applications 2013: San Diego, CA, USA.

[19] Pinto PC, Thiran P, Vetterli M (2012) Locating the Source of Diffusion in Large-Scale Networks. Physical Review Letters 109: 068702+.

[20] Lokhov AY, Mezard M, Ohta H, Zdeborova L (2013) Inferring the origin of an epidemic with dynamic message-passing algorithm. Unpublished, http://arxiv.org/abs/13035315
[21] Antulov-Fantulin N, Lancic A, Stefancic H, Sikic M, Smuc T (2013) Statistical inference framework for source detection of contagion processes on arbitrary network structures. Unpublished, http://arxiv.org/abs/13040018.

[22] Comin CH, da Fontoura Costa L (2011) Identifying the starting point of a spreading process in complex networks. Phys Rev E 84: 056105.

[23] Brockmann D, Helbing D (2013) The Hidden Geometry of Complex, Network-Driven Contagion Phenomena. Science 342: 1337–1342.

[24] Antulov-Fantulin N, Lancic A, Stefancic H, Sikic M (2013) Fastsir algorithm: A fast algorithm for the simulation of the epidemic spread in large networks by using the susceptible–infected–recovered compartment model. Information Sciences 239: 226 - 240.

[25] Hadamard J (1902) Sur les problèmes aux drives partielles et leur signification physique. Princeton University Bulletin: 4952.

[26] Rocha LEC, Liljeros F, Holme P (2011) Simulated epidemics in an empirical spatiotemporal network of 50,185 sexual contacts. PloS Computational Biology 7: e1001109–.

[27] Bastian M, Heymann S, Jacomy M (2009) Gephi: an open source software for exploring and manipulating networks. International AAAI Conference on Weblogs and Social Media.

[28] Csardi G, Nepusz T (2006) The igraph software package for complex network research. InterJournal, Complex Systems 1695.
Supplementary material:
Detectability limits of epidemic sources in networks

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1 Analytical combinatorics approach

In this section, we describe the general procedure for calculating exact source probability distributions by using the formalism of generating functions. We start, by introducing all needed definitions and suitable notations for this approach. Let $G = (V, E)$ be a given graph and $v_i, v_j \in V, i \neq j$. We define the discrete sample space $\Omega$ of elementary events for stochastic epidemic spreading on $G$ as the Cartesian product of elementary events on each edge $(v_i, v_j)$: (i) disease propagated from node $v_i$ to node $v_j$, (ii) disease propagated from node $v_j$ to node $v_i$ and (iii) disease did not propagate through the edge.

Let $S\{i,j\} = \{w_{i,j,k} : 1 \leq k \leq m_{i,j}\}$ be the set of all simple paths $w_{i,j,k}$ (with cardinality $m_{i,j}$) in $G$ beginning at $v_i$ and ending at $v_j$. Let $W_{i,j,k}$ denote the event in which the epidemic process has passed through each edge consequently for all vertices in $w_{i,j,k}$ in the appropriate direction, while we don’t put any restrictions on the spreading of the epidemic for other edges in the graph. We will use the SIR model as the main contagion model, which is parametrized by the probability $p$ that an infected node infects a susceptible neighbour node and by the probability $q$ that an infected node recovers in one discrete time step.

Now, let’s define the event $i \rightarrow j$ as $\bigcup_{k=1}^{m_{i,j}} W_{i,j,k}$; intuitively, that is an event in which the vertex $v_i$ has become infected during the epidemic process (possibly as the initial source of the epidemic), after which the epidemic has spread to the vertex $v_j$.

We define a realization $r$ as a function $r : V \rightarrow \{0, 1\}$ such that $r(v_k) = 1$ if $v_k$ was infected during the epidemic process and $r(v_k) = 0$ otherwise. Now, let’s define the random vector $R$ which describes the state of all vertices at the end of the epidemic, and the random variable $I$ which describes a choice of the initial source of the epidemic. Note, that in the main manuscript we have used the following notation: $r^\ast$ for realization, $\tilde{R}$ for random vector and $\Theta$ for the source nodes, but only in this section due to the simplicity of notation in analytical formulas we use the equivalent notation $r$, $R$ and $I$. We have

$$P(R = r | I = i) = \mathbb{P}(\bigcap_{v_j \in r^{-1}(1)} i \rightarrow j \bigcap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C | I = i) =$$

$$\mathbb{P}(\bigcap_{v_j \in r^{-1}(1)} \bigcup_{k=1}^{m_{i,j}} W_{i,j,k} \bigcap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C | I = i)_{S_{r,i} = \{g \in \mathbb{N}^{r^{-1}(1)} : g(v_j) \in \{1, 2, \ldots, m_{i,j}\}\}} =$$

where $S_{r,i}$ is a set of functions defined on $r^{-1}(1)$ (nodes infected in $r$) such that each function maps a vertex in $r^{-1}(1)$ to an index of a path from the source $i$ to that vertex. The cardinality $|S_{r,i}|$ of the set $S_{r,i}$ is equal to the product of the number of simple paths $m_{i,j}$ from node $i$ to all other nodes $j$ in the set $r^{-1}(1)$. In order to get a better intuition about the set $S_{r,i}$ see the Example 2 from this section.

$$\mathbb{P}(\bigcup_{\psi \in S_{r,i}} (\bigcap_{v_k \in r^{-1}(1)} W_{i,\psi(v_k)} \bigcap (i \rightarrow k)^C | I = i)_{\text{inclusion - exclusion principle}}$$

$$= \sum_{\ell=1}^{|S_{r,i}|} \sum_{J \subseteq S_{r,i}, |J| = \ell} (-1)^{\ell-1} \mathbb{P}(\bigcap_{\psi \in J} (\bigcap_{v_k \in r^{-1}(1)} W_{i,\psi(v_k)} \bigcap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C | I = i))$$

For practical purposes, it is useful to note that $(\bigcap_{\psi \in J} \bigcap_{v_k \in r^{-1}(1)} W_{i,\psi(v_k)} \bigcap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C) = \bigcap_{\psi \in J} (\bigcap_{v_k \in r^{-1}(1)} (W_{i,k,\psi(v_k)} \bigcap (i \rightarrow k)^C)).$ In conclusion, to be able to evaluate $P(R = r | I = i)$, it suffices to be able to evaluate terms of the form $P(\bigcap_{\psi \in J} (\bigcap_{v_k \in r^{-1}(1)} (W_{i,\psi(v_k)} \bigcap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C)) | I = i)$.

We will now proceed to describe how to obtain their values using generating functions.
For a given vertex \( v \), where \( n - k \) is the number of edges incident to \( v \) through which infection may be spread (edges which eventually transmit the infection to \( v \) are not eligible) but is not spread, we observe the probability of the event that, for the first time after the moment of its infection, through predetermined \( k \) (ordered) edges the infection spreads successively after \( \ell_1, \ell_2, \ldots, \ell_k \) time steps (in the same order), while it does not spread through the remaining \( n - k \) edges, in case that until the end of the simulation from the step of initial infection of the vertex \( v \) there remains \( l \) steps. Note that any number of edges on which no restrictions are placed are also allowed, since they do not affect the calculation in any way. We denote that event as \( (Y_1, Y_2, \ldots, Y_k, t_R) = (i_1, \ldots, i_k, l) \). The corresponding generating function of a multivariate sequence of the form \( \mathbb{P} ((Y_1, Y_2, \ldots, Y_k, t_R) = (i_1, \ldots, i_k, l)) \) is

\[
    f_{X_n=k} (x_1, \ldots, x_k, r) = \sum_{i_1, \ldots, i_k} \mathbb{P} ((Y_1, Y_2, \ldots, Y_k, t_R) = (i_1, \ldots, i_k, l)) r^l \prod_{j=1}^{k} x_{j}^{i_j} =
\]

\[
    = \sum_{l=0}^{\infty} \left( \sum_{i_1, \ldots, i_k} \mathbb{P} ((Y_1, Y_2, \ldots, Y_k) = (i_1, \ldots, i_k) | t_R = l) \prod_{j=1}^{k} x_{j}^{i_j} \right) \mathbb{P} (t_R = l) r^l
\]

If \( T_v \) is a number of steps from the initial infection of the vertex \( v \) until its recovery, we have

\[
    \sum_{i_1, \ldots, i_k} \mathbb{P} ((Y_1, \ldots, Y_k) = (i_1, \ldots, i_k) | t_R = l) \prod_{j=1}^{k} x_{j}^{i_j} =
\]

\[
    = \sum_{i_1, \ldots, i_k} \left( \sum_{m=1}^{l} \prod_{j=1}^{k} \mathbb{P} (T_v = m | Y_j = i_j, T_v = m, t_R = l) \prod_{j=1}^{k} x_{j}^{i_j} + \mathbb{P} (T_v > l) (1 - p)^{(n-k)} \sum_{1 \leq l_1, \ldots, l_k \leq m} \prod_{j=1}^{k} \mathbb{P} (Y_j = i_j | T_v > l, t_R = l) x_{j}^{i_j} \right)
\]

\[
    q \sum_{m=1}^{l} \left( \sum_{1 \leq l_1, \ldots, l_k \leq m} \prod_{j=1}^{k} p(1 - p)^{(n-k)} \prod_{i_j=1}^{m} (1 - q)^{(n-k)} \prod_{j=1}^{k} x_{j}^{i_j} + (1 - q)^{(n-k)} \prod_{j=1}^{k} p(1 - p)^{(n-k)} \prod_{i_j=1}^{m} (1 - q)^{(n-k)} \prod_{j=1}^{k} x_{j}^{i_j} \right)
\]

\[
    = \sum_{l=0}^{\infty} \left( q \sum_{m=1}^{l} \left( \prod_{j=1}^{k} p(1 - p)^{(n-k)} \prod_{i_j=1}^{m} (1 - q)^{(n-k)} \prod_{j=1}^{k} x_{j}^{i_j} + (1 - q)^{(n-k)} \prod_{j=1}^{k} p(1 - p)^{(n-k)} \prod_{i_j=1}^{m} (1 - q)^{(n-k)} \prod_{j=1}^{k} x_{j}^{i_j} \right) + \mathbb{P} (t_R = l) r^l \right)
\]

The equality above motivates the following definition:
\[ F_{X_n = k}(x_1, \ldots, x_k, r) := \sum_{l=0}^{\infty} \left( q \sum_{m=1}^{l} (1-q)^{m-1}(1-p)^{(n-k)m} \prod_{j=1}^{k} p \sum_{i_j=1}^{m} (1-p)^{i_j-1} x_j^{i_j} + (1-q)^l(1-p)^{(n-k)l} \prod_{j=1}^{k} p \sum_{i_j=1}^{l} (1-p)^{i_j-1} x_j^{i_j} \right)^r. \]

Note that \( F \) is the generating function corresponding to a multivariate sequence of the form \( P((Y_1, Y_2, \ldots, Y_k) = (i_1, \ldots, i_k)|t_R = l) \). The factor \( P(t_R = l) \) in \( f \) follows from the information on the spreading process along the paths from the initially infected node to the observed node and it is calculated from the corresponding products of \( F \) functions for nodes on these paths (see Example 1).

In further calculations, it’s important to know the values of particular coefficients denoted as \( \langle x_1^{i_1} \cdots x_k^{i_k} r^l \rangle \) of the generating function defined as above. From (1) we obtain the following coefficients:

\[
\langle x_1^{i_1} \cdots x_k^{i_k} r^l \rangle F_{X_n = k}(x_1, \ldots, x_k, r) =
\begin{align*}
&k = 0, l = 0 \Rightarrow \langle x_1^{i_1} \cdots x_k^{i_k} r^l \rangle F_{X_n = k}(x_1, \ldots, x_k, r) = 1 \\
&k > 0, l = 0 \Rightarrow \langle x_1^{i_1} \cdots x_k^{i_k} r^l \rangle F_{X_n = k}(x_1, \ldots, x_k, r) = 0
\end{align*}
\]

\[
k > 0, l > 0 \Rightarrow \langle x_1^{i_1} \cdots x_k^{i_k} r^l \rangle F_{X_n = k}(x_1, \ldots, x_k, r) = (1-q)^l(1-p)^{nl} + q \frac{(1-p)^n - (1-q)^l(1-p)^{(l+1)n}}{1 - (1-q)(1-p)^n} =
\]

\[
p^k(1-q)^l(1-p)^{l(n-k)}(1-p)^{i_1+\ldots+i_k-k} + q p^k(1-p)^{i_1+\ldots+i_k-k} \sum_{m=\max\{i_1, \ldots, i_k\}+1}^{l} (1-q)^{m-1}(1-p)^{m(n-k)} =
\]

\[
p^k(1-p)^{i_1+\ldots+i_k-k}[(1-q)^l(1-p)^{l(n-k)} + q \sum_{m=\max\{i_1, \ldots, i_k\}}^{l} (1-q)^{m-1}(1-p)^{m(n-k)}] =
\]

\[
p^k(1-p)^{i_1+\ldots+i_k-k}[(1-q)^l(1-p)^{l(n-k)} + q \frac{(1-q)^{\max\{i_1, \ldots, i_k\}-1}(1-p)^{(n-k)\max\{i_1, \ldots, i_k\}} - (1-q)^l(1-p)^{(l+1)(n-k)}}{1 - (1-q)(1-p)^{n-k}}] =
\]

When we calculate \( P(\bigcap_{\psi \in J} \bigcap_{v_\kappa \in r^{-1}(1)} (W_{(i,\kappa)}, \psi(v_\kappa) \cap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C) | I = i \) \), each vertex \( v \in r^{-1}(1) \setminus \{v_i\} \) has at least one adjacent vertex which transmits the infection to it; let \( v_1, \ldots, v_\eta \) be all such vertices, \( l_1, \ldots, l_\eta \) be the times left from the time of their initial infection until the end of the simulation and \( j_1, \ldots, j_\eta \) be the times when they first transmitted the infection to \( v \) counting from time of their initial infection. Then, we easily get the time \( l \) remaining from the moment of initial infection of the vertex \( v \) until the end of the simulation as \( \min_{1 \leq k \leq \eta} (l_k - j_k) \). Since the product of generating functions corresponds to convolution of their sequences, it’s easy to see that, in its coefficients, a product of generating functions defined as above contains all information we need to calculate \( P(\bigcap_{\psi \in J} \bigcap_{v_\kappa \in r^{-1}(1)} (W_{(i,\kappa)}, \psi(v_\kappa) \cap \bigcap_{v_k \in r^{-1}(0)} (i \rightarrow k)^C) | I = i \) \).

**Example 1:**
For a specific realization from Figure 1 from the main manuscript, we can calculate the probability of each vertex being the source of the epidemic. We assign to each node \( i \) "dummy" variables \( r_i \), and to each edge a "dummy" variable \( x_k \), the restriction being that the first time of vertex’ infection plus the time remaining to the end of the simulation always equals \( T \) and that first time of infection transmission
through any edge (counting from the first time of infection of the spreading vertex) can not be greater than the time remaining until the end of the simulation (also counting from the first time of infection of the spreading vertex). For $t = 5, p = 0.2, q = 0.3$ we get:

$$P(R = r | I = 7, T = t) =$$

$$\sum_{n_1=1}^{t} \sum_{n_2=1}^{t-n_1} \sum_{n_3=1}^{t-n_1-n_2} \sum_{n_4=1}^{t-n_1-n_2-n_3} \sum_{n_5=1}^{t-n_1-n_2-n_3-n_4} \langle r_{11}^{r_{11}} f_{11}^{r_{11}} f_{n_1}^{r_{11}} f_{n_2}^{r_{11}} f_{n_3}^{r_{11}} f_{n_4}^{r_{11}} f_{n_5}^{r_{11}} \rangle$$

$$F_{x_1}=1(x_1, r_7) F_{x_3}=3(x_2, x_3, r_11) F_{X_3}=0(r_10) F_{X_2}=0(r_12) F_{X_3}=1(x_5, r_15) F_{X_2}=0(r_19) = 0.0001368350$$

$$P(R = r | I = 11, T = t) =$$

$$\sum_{n_2=1}^{t} \sum_{n_3=1}^{t-n_2} \sum_{n_4=1}^{t-n_2-n_3} \sum_{n_5=1}^{t-n_2-n_3-n_4} \langle r_{12}^{r_{12}} f_{12}^{r_{12}} f_{n_2}^{r_{12}} f_{n_3}^{r_{12}} f_{n_4}^{r_{12}} f_{n_5}^{r_{12}} \rangle$$

$$F_{x_4}=4(x_1, x_2, x_3, r_11) F_{X_3}=0(r_7) F_{X_2}=0(r_12) F_{X_3}=0(r_10) F_{X_2}=1(x_5, r_15) F_{X_2}=0(r_19) = 0.0003924644$$

$$P(R = r | I = 10, T = t) =$$

$$\sum_{n_3=1}^{t} \sum_{n_4=1}^{t-n_3} \sum_{n_5=1}^{t-n_3-n_4} \langle r_{15}^{r_{15}} f_{15}^{r_{15}} f_{n_3}^{r_{15}} f_{n_4}^{r_{15}} f_{n_5}^{r_{15}} \rangle$$

$$F_{x_4}=5(x_1, x_2, x_3, r_11) F_{X_3}=0(r_7) F_{X_2}=0(r_12) F_{X_3}=0(r_10) F_{X_2}=1(x_5, r_15) F_{X_2}=0(r_19) = 0.0001368350$$

$$P(R = r | I = 12, T = t) =$$

$$\sum_{n_4=1}^{t} \sum_{n_5=1}^{t-n_4} \sum_{n_5=1}^{t-n_4-n_5} \langle r_{19}^{r_{19}} f_{19}^{r_{19}} f_{n_4}^{r_{19}} f_{n_5}^{r_{19}} f_{n_5}^{r_{19}} \rangle$$

$$F_{x_4}=6(x_1, x_2, x_3, r_11) F_{X_3}=0(r_7) F_{X_2}=0(r_12) F_{X_3}=0(r_10) F_{X_2}=1(x_5, r_15) F_{X_2}=0(r_19) = 0.0001772788$$

$$P(R = r | I = 15, T = t) =$$

$$\sum_{n_5=1}^{t} \sum_{n_5=1}^{t-n_5} \sum_{n_5=1}^{t-n_5-n_5} \langle r_{19}^{r_{19}} f_{19}^{r_{19}} f_{n_5}^{r_{19}} f_{n_5}^{r_{19}} f_{n_5}^{r_{19}} \rangle$$

$$F_{X_4}=2(x_4, x_5, r_15) F_{X_2}=0(r_19) F_{X_3}=3(x_1, x_2, x_3, r_11) F_{X_3}=0(r_7) F_{X_3}=0(r_10) F_{X_2}=0(r_12) = 0.0002219489$$

Figure 1: Realization on grid network, where the infected nodes are coloured with red.
\begin{align*}
P(R = r | I = 19, T = t) &= \\
&= \sum_{n_5=1}^{t} \sum_{n_4=1}^{t-n_5} \sum_{n_3=1}^{t-n_4-n_5} \sum_{n_2=1}^{t-n_4-n_5-n_3} \sum_{n_1=1}^{t-n_4-n_5-n_2-n_3} \langle r_1^{19} x_5^{n_5} r_4^{n_4} x_4^{n_4} r_3^{n_3} x_3^{n_3} r_2^{n_2} x_2^{n_2} r_1^{n_1} x_1^{n_1} \rangle \\
&= F_{X_3=1} (x_5, r_{19}) F_{X_3=1} (x_4, r_{15}) F_{X_3=3} (x_1, x_2, x_3, r_{11}) F_{X_3=0} (r_{10}) F_{X_3=0} (r_7) F_{X_2=0} (r_{12}) = 0.0001553419
\end{align*}

The script in Wolfram Mathematica that calculates source likelihoods for this toy example is downloadable here\(^1\). The probability distribution over sources we get via Bayes formula:

\[
P(I = i | \mathbf{R} = \mathbf{r}^* ) = \frac{P(\mathbf{R} = \mathbf{r}^* | I = i) P(I = i)}{\sum_j P(\mathbf{R} = \mathbf{r}^* | I = j) P(I = j)},
\]

and since our prior source probabilities are equal we get:

\[
P(I = i | \mathbf{R} = \mathbf{r}^* ) = \frac{P(\mathbf{R} = \mathbf{r}^* | I = i)}{\sum_j P(\mathbf{R} = \mathbf{r}^* | I = j)}.
\]

**Example 2:**

Now, let us consider a simple network with the source node \(v_1\) and the following infected nodes \(r^{-1}(1) = \{v_1, v_2, v_3, v_4\}\).

\[
\begin{array}{c|c|c|c}
 x & v_2 & v_3 & v_4 \\
\hline
 v_1(x) & 1 & 1 & 1 \\
 v_2(x) & 1 & 2 & 1 \\
 v_3(x) & 1 & 1 & 2 \\
 v_4(x) & 1 & 2 & 2 \\
\end{array}
\]

Now we enumerate simple paths from the source node \(v_1\) to all other infected nodes. From node \(v_1\) there exist only one simple path \(W_{(1,2),1}\) to node \(v_2\). From node \(v_1\) there exist only two simple paths: \(W_{(1,3),1}\) and \(W_{(1,3),2}\) to node \(v_3\). From node \(v_1\) there exists only two simple paths: \(W_{(1,4),1}\) and \(W_{(1,4),2}\) to node \(v_4\). The set \(S_{r=1} = \{v_1(x), v_2(x), v_3(x), v_4(x)\}\) consists of all the functions defined on \(r^{-1}(1)\) (nodes infected in \(r\)) such that each function maps a vertex in \(r^{-1}(1)\) to an index of a path from the source 1 to that vertex, as denoted in the table above.

Let us recall the formula which is used to calculate the source probability:

\[
P(R = r | I = i) = P(\bigcap_{v_j \in r^{-1}(1)} i \to j) \cap \bigcap_{v_k \in r^{-1}(0)} (i \to k)^C | I = i).
\]

We will now just expand the first term in this formula for our specific example for node \(i = 1\).

\[
\bigcap_{v_j \in r^{-1}(1)} i \to j = \bigcap_{v_j \in r^{-1}(1)} \bigcup_{\ell=1}^{m_{i,j}} W_{(i,j),\ell} = (W_{(1,2),1}) \cap (W_{(1,3),1} \cup W_{(1,3),2}) \cap (W_{(1,4),1} \cup W_{(1,4),2})
\]

\[
= ((W_{(1,2),1} \cap W_{(1,3),1}) \cup (W_{(1,2),1} \cap W_{(1,3),2})) \cap (W_{(1,4),1} \cup W_{(1,4),2}) = (A \cap W_{(1,4),1}) \cup (A \cap W_{(1,4),2})
\]

\(^1\)http://lis.irb.hr/epidemic/
Now, we will explicitly write down a few events on this graph. The event is the Cartesian product of elementary events on each edge $(v_i, v_j)$: (i) "→" disease propagated from node $v_i$ to node $v_j$, (ii) "←" disease propagated from node $v_j$ to node $v_i$ and (iii) "||" disease did not propagate through the edge. The events are written down as a Cartesian product on edges: $(v_1, v_2), (v_1, v_3), (v_1, v_4), (v_3, v_4)$. For example the event:

$W_{(3,2),1} = \{\rightarrow\} \times \{\leftarrow\} \times \{\rightarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\}$,

describes the event of disease passing from node $v_1$ to $v_2$ and from $v_3$ to $v_1$ and no restrictions on other edges. We will now write few more events on this example:

$W_{(1,2),1} = \{\rightarrow\} \times \{\leftarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\}$,

$W_{(1,2),1} \cap W_{(3,2),1} = \{\rightarrow\} \times \{\leftarrow\} \times \{\rightarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\}$,

$W^{C}_{(3,2),1} = X \cup Y \cup Z$

$X = \{\leftarrow, ||\} \times \{\rightarrow, ||\} \times \{\rightarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\}$

$Y = \{\rightarrow\} \times \{\rightarrow, ||\} \times \{\rightarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\}$

$Z = \{\leftarrow, ||\} \times \{\leftarrow\} \times \{\rightarrow, \leftarrow, ||\} \times \{\rightarrow, \leftarrow, ||\}$. 

7
2 Direct Monte Carlo estimator and pruning

With the direct Monte Carlo simulation approach, for each node $i$ from the realization $\vec{r}_s$, a large number $n$ of epidemic spreading simulations with duration $T$ is performed with $i$ as an epidemic source. The number of simulations $n_i$ which coincide with the realization $\vec{r}_s$ is recorded. After the simulations for all potential source nodes in the realization $\vec{r}_s$ are finished, the probability of the node $i$ being the source of the epidemic is calculated as $n_i/\sum_j n_j$. Here we devise the rule for pruning realizations at some temporal point $t < T$ whose contribution is zero. Let us also define the error term for every simulated realization $\vec{r}_t$ at some point in time $t < T$ w.r.t observed realization $\vec{r}_T^*$ at time $T$:

$$\text{error}_t(\vec{r}_t^*, \vec{r}_T^*) = \frac{1}{N} \sum_{k \in V} ((r_t^*(k) == 0) \land (r_t^*(k) == 1)).$$ (2)

The error term calculates the number of corresponding nodes which are non-infected in the observed realization $\vec{r}_T^*$ at time $T$ and infected in the simulated realization $\vec{r}_t^*$ at time $t$.

**Proposition:** Monte Carlo SIR realization simulation $\vec{r}_t^*$ at time $t < T$ can be terminated if the error term $\text{error}_t(\vec{r}_t^*, \vec{r}_T^*) > 0$ and it will have no effect on the final estimation.

Proof: If at time $t$ the error term $\text{error}_t(\vec{r}_t^*, \vec{r}_T^*) > 0$, then at time $T$ the error can only increase: $\text{error}_T(\vec{r}_T^*, \vec{r}_T^*) \geq \text{error}_t(\vec{r}_t^*, \vec{r}_T^*)$ because the error term $\text{error}_t(\vec{r}_t^*, \vec{r}_T^*)$ is monotonic increasing function w.r.t. time $t + 1, t + 2, ..., T$ and direct Monte Carlo estimator rejects any realization with positive error term: $\text{error}_T(\vec{r}_T^*, \vec{r}_T^*) > 0$. The infected state $(r_t^*(k) == 1)$ is absorbing state w.r.t time $t$. Once the node leaves the susceptible state it can not come back to it in the SIR model.

The pruning mechanism provides a substantial acceleration (see Figure 2) without inducing any errors to our estimation.

![Figure 2: The speed up of direct Monte Carlo estimation with the pruning rule for experiment with $n$ simulations per source node. Comparison of run-time per source detection experiment on 30 cpu cores(4 x AMD Opteron Processor 6134, 2.3 GHz with 8 cores each) averaged over 10 experiments.](image-url)
3 Benchmark analysis

Here, we provide the comparison of different estimators for the SIR model w.r.t. ML probability errors (see Figure 4) and ML accuracy (see Figure 5) on the benchmark dataset. The correct solutions were calculated with the direct Monte Carlo estimator with $[10^6 - 10^8]$ simulations per source depending on convergence condition. The convergence condition for direct Monte Carlo solution at $2x$ number of simulations was set to be: ML node relative error is: $|P^{ML}_{2x} - P^{ML}_{x}| / P^{ML}_{2x} \leq 0.05$ and the maximal absolute error for all other nodes is: $|P^{x}_i - P^{2x}_i| \leq 0.05$. We have compared the centrality-like estimators: Distance [6] and Jordan [5] centrality, Belief propagation estimator: DMP [1], different Monte Carlo estimators: AUCDF, AvgTopK and NaiveBayes [4] and two baseline solutions: Rnd (source likelihood is random number from $[0,1]$) and Const (all sources are equal).

Most of the aforementioned estimators do not output explicit source probability distribution function, but rather a ranking list with appropriate weights $w_i$, from which we calculate source pdf by re-normalization with factor $\sum_j w_j$ to get a PDF. We have used our implementation of distance [6], Jordan [5] centrality and Belief propagation estimator DMP [1].

In this manuscript, we use a conservative information about the node state at observed moment $t$, we only observe whether node is susceptible or not (realization $R$ is a binary vector). This implies that we do not need additional information to distinguish whether node state is recovered or it is still infective. The original DMP [1] estimator additionally assumes that one can distinguish the recovered from infective state. Therefore, in order to apply the DMP [1] algorithm to our scenario, we had to adopt the estimation formula so that probability of node being infected is merged with probability of node being recovered in order to estimate the probability of being in either Infective or Recovered compartment. All other calculations were implemented according to the original algorithm [1]. In order to verify our implementation of the DMP algorithm, we have compared our DMP implementation on tree network, where the node state probability estimation should be correct. We have measured the difference between probability estimate that the node is susceptible after $T$ steps with the DMP and the SIR Monte Carlo simulation algorithm and we observe that less than 1 % of nodes have the relative error greater than 0.001, which means that the SIR Monte Carlo simulation algorithm estimates are very close to DMP on tree networks (see Figure 3).

Figure 3: Comparing Dynamic Message Passing (DMP) estimates of the node state probability for $p = 0.3, q = 0.5, T = 10$ with the SIR simulation estimates (NaiveSIR) on the Albert-Barabashi tree network ($N = 5000, m_0 = 2, m = 1$). Distribution of relative errors of node being susceptible with SIR simulation ($n = 10^4$) w.r.t. DMP on tree network.
In a limit where the parameter $a \to 0$, for the Soft Margin estimator we obtain the unbiased estimate of the likelihood $P(R = \vec{r}^*_0 | \Theta = \theta)$. For cases when the parameter $a > 0$ we obtain an estimator which is estimating the likelihood by using the tail of pdf function $f(x)$ in a way that it uses the values of slightly different realizations to get estimate for observed realization $\vec{r}^*$.

In Figure 6 plot A and B, we can see the effect of different Soft Margin widths $a$ on the convergence. As the soft margin width parameter $a$ decreases, it becomes more similar to the unbiased estimator, but the convergence becomes slower.

Figure 4: The comparison of maximum likelihood probability errors with box-plots for different estimators and soft margin with $a = 0.031$. The error is relative error of maximum likelihood estimation w.r.t. gold standard ML probability obtained with direct Monte Carlo method for different parameters: $A = (p = 0.3, q = 0.3, T = 5)$, $B = (p = 0.3, q = 0.7, T = 5)$, $C = (p = 0.7, q = 0.3, T = 5)$ and $D = (p = 0.7, q = 0.7, T = 5)$.

Figure 5: The comparison of accuracy of detecting the maximum likelihood node with mean accuracy and standard deviation for different estimators and soft margin with $a = 0.031$. ML accuracy is the ratio of how many times the estimator ranks the ML node on rank 1 and total number of trials (ranking measure). plot A-D correspond to different parameters: $A = (p = 0.3, q = 0.3, T = 5)$, $B = (p = 0.3, q = 0.7, T = 5)$, $C = (p = 0.7, q = 0.3, T = 5)$ and $D = (p = 0.7, q = 0.7, T = 5)$. 
Figure 6: Comparison of Soft Margin estimators with different weights $a$ with respect to the Maximum Likelihood relative probability estimation error (plot A) and Maximum Likelihood accuracy (plot B) using the average over 160 different benchmark cases. Benchmark cases were calculated on a small regular network (4-connected grid $N = 30 \times 30$) and SIR process with different parameters $(p,q,T)$ with the direct Monte Carlo estimator with $[10^6 - 10^8]$ simulations per source depending on convergence condition.
4 Benchmark dataset

Together with this manuscript, we provide a set of SIR realizations along with their inverse solutions represented as probability over potential candidate nodes. We provide 160 benchmark realizations (downloadable here\footnote{http://lis.irb.hr/epidemic/}) on a small regular network (4-connected grid $N = 30 \times 30$) for different SIR parameters: $A = (p = 0.3, q = 0.3, T = 5)$, $B = (p = 0.3, q = 0.7, T = 5)$, $C = (p = 0.7, q = 0.3, T = 5)$ and $D = (p = 0.7, q = 0.7, T = 5)$. The realizations are in the textual form with the following format:

file: realization_2.txt

#source: 159
#p: 0.700000
#q: 0.700000
#T: 5
#node states:
0
1
0
1

Each realization contains the inverse solution $P(\Theta = \theta_i | \vec{R} = \vec{r}_* )$ calculated with the direct Monte Carlo estimator with $[10^6 - 10^8]$ simulations per source depending on the convergence condition. The convergence condition is relative ML node error: $|P^{2x}_{ML} - P^x_{ML}|/P^{2x}_{ML} \leq 0.05$ and the maximal absolute error for all other nodes: $|P^x_i - P^{2x}_i| \leq 0.05$. The solutions are provided in textual form with the following format:

file: inverse_solution_2.txt

#Number of simulation: 10000000
#Node source probabilities:
0.000000
0.240000
0.000000
0.110000
0.000000

The network is given in the gml network format.
5 Soft Margin implementation details

In this section, we explain in more details: (i) how to set the Soft Margin width parameter \( a \) and (ii) how we relax the assumption on epidemic starting moment \( t_0 \) by marginalization of conditional probabilities. Let us recall how we calculate source probability distribution via Bayes formula:

\[
P(\Theta = \theta_i | \mathbf{R} = \mathbf{r}_s) = \frac{P(\mathbf{R} = \mathbf{r}_s | \Theta = \theta_i)P(\Theta = \theta_i)}{\sum_{\theta_j} P(\mathbf{R} = \mathbf{r}_s | \Theta = \theta_j)P(\Theta = \theta_j)}
\]

(3)

and since our prior source probabilities are equal we get:

\[
P(\Theta = \theta_i | \mathbf{R} = \mathbf{r}_s) = \frac{P(\mathbf{R} = \mathbf{r}_s | \Theta = \theta_i)}{\sum_{\theta_j} P(\mathbf{R} = \mathbf{r}_s | \Theta = \theta_j)}.
\]

(4)

Now, the Soft Margin estimates the likelihood via the following formula:

\[
\hat{P}(\mathbf{R} = \mathbf{r}_s | \Theta = \theta) = \int_{0}^{1} w_a(x) \hat{f}_\theta(x) dx = \int_{0}^{1} w_a(x) \frac{1}{n} \sum_{i=1}^{n} \delta(x - \varphi(\mathbf{r}_s^*, \mathbf{r}_{\theta,i})) dx,
\]

(5)

which can be simplified by using the property of delta distribution: \( \int_{-\infty}^{\infty} f(x) \delta(x - b) dx = f(b) \),

\[
\hat{P}(\mathbf{R} = \mathbf{r}_s | \Theta = \theta) = \frac{1}{n} \sum_{i=1}^{n} \int_{0}^{1} w_a(x) \delta(x - \varphi(\mathbf{r}_s^*, \mathbf{r}_{\theta,i})) dx = \frac{1}{n} \sum_{i=1}^{n} w_a(\varphi(\mathbf{r}_s^*, \mathbf{r}_{\theta,i})).
\]

(6)

We use the following weighting form: \( w_a(x) = e^{-(x-1)^2/a^2} \) and thus the likelihood estimation is equal to:

\[
\hat{P}(\mathbf{R} = \mathbf{r}_s | \Theta = \theta) = \frac{1}{n} \sum_{i=1}^{n} e^{-((\varphi(\mathbf{r}_s^*, \mathbf{r}_{\theta,i}) - 1)^2/a^2}.
\]

(7)

Simplified, for each potential candidate \( \theta \), by simulations we obtain \( n \) samples of variable \( \varphi(\mathbf{r}_s^*, \mathbf{R}_\theta) \); \( \{ \varphi_{\theta,1}, \varphi_{\theta,2}, \ldots \varphi_{\theta,n} \} \) from which we calculate the source likelihood: \( \hat{P}(\mathbf{R} = \mathbf{r}_s | \Theta = \theta) = \frac{1}{n} \sum_{i=1}^{n} e^{-((\varphi_{\theta,i} - 1)^2/a^2} \).

Now, after we calculate \( \hat{f}_\theta(x) \) for every potential source, we recalculate the source probability distribution for different values of parameter \( a \) in range: \( \{1/2, (1/2)^2, (1/2)^3, \ldots, (1/2)^{15}\} \). Then, we measure the convergence property of estimated PDFs: \( \hat{P}_a^n(\Theta = \theta_i | \mathbf{R} = \mathbf{r}_s) \) for different values of Soft Margin weight \( a \) and different number of simulations \( n \). Then we choose the parameter \( a \) as the minimum of the set of parameters for which the PDFs have converged. We use the following convergence condition for the source PDFs: \( |\hat{P}_a^n(\Theta = \theta_{MAP} | \mathbf{R} = \mathbf{r}_s) - \hat{P}_a^n(\Theta = \theta_{MAP} | \mathbf{R} = \mathbf{r}_s)| \leq 0.05 \), where \( \theta_{MAP} \) is the node \( i \) with the maximum estimated source probability in \( \hat{P}_a^n(\Theta = \theta_i | \mathbf{R} = \mathbf{r}_s) \). The smaller the parameter \( a \), the estimations becomes more similar to the direct Monte Carlo estimator if the PDFs have converged. Note, that the maximum likelihood (ML) node is the same as the maximum posteriori (MAP) node if our prior source probabilities are equal. As the computational complexity of calculating \( \hat{f}_\theta(x) \) is a few orders of magnitude higher than complexity of recalculating source PDF for different parameters \( a \), one does not need to set parameter \( a \) in advance but rather choose the near-optimal value of \( a \) for specific number of simulations \( n \).
Up to this point we have assumed that the epidemic duration $T$ or starting point $t_0$ were given in advance. Strictly speaking we should have written the parameter $T$ in all the conditional probabilities $P(\tilde{R} = r_s | \Theta = \theta, T = t - t_0)$ instead of just $P(\tilde{R} = r_s | \Theta = \theta)$, but this would just complicate the notation since we could also put other given parameters like $G$, $p$ and $q$ in the condition. Instead, unless otherwise stated, we assume that parameters: $T$, $G$, $p$ and $q$ are given. Now we explain how to relax the assumption on specific epidemic parameters. For example, if we know the time $t$ when the realization was observed, but the epidemic starting moment $t_0$ is not known in advance, by marginalization over all possible $t_0$ outcomes we get:

$$P(\tilde{R} = r^*_s | \Theta = \theta) = \sum_{t_0=0}^{t} P(\tilde{R} = r^*_s, T = t - t_0 | \Theta = \theta),$$

where the variable $T$ denotes the epidemic duration. This expression can be further transformed into:

$$P(\tilde{R} = r^*_s | \Theta = \theta) = \sum_{t_0=0}^{t} P(\tilde{R} = r^*_s | \Theta = \theta, T = t - t_0) P(T = t - t_0 | \Theta = \theta).$$

Now, the term $P(\tilde{R} = r^*_s | \Theta = \theta, T = t - t_0)$ can be calculated with the Soft Margin estimator like before, and the term $P(T = t - t_0 | \Theta = \theta)$ denotes the prior distribution of epidemic duration or epidemic start. But, we do not estimate $\hat{P}(\tilde{R} = r^*_s | \Theta = \theta)$ by definition due to its computational cost, but rather by another sample estimation. First, we sample a $T_i$ from the prior probability distribution $P(T = t - t_0 | \Theta = \theta)$ and then obtain the sample realization $\tilde{r}_{\theta,i}$ for a given $T_i$. We repeat the procedure $n$ times, obtain $n$ temporal samples $\{T_1, ..., T_n\}$ and obtain $n$ corresponding realizations $\{\tilde{r}_1, ..., \tilde{r}_n\}$. Then, we estimate $\hat{P}(\tilde{R} = r_s | \Theta = \theta)$ with the Soft Margin estimator from $n$ realizations: $\{\tilde{r}_1, ..., \tilde{r}_n\}$ from their similarities to the observed realization: $\{\varphi(\tilde{r}_1^*, \tilde{r}_{\theta,1}), ..., \varphi(\tilde{r}_n^*, \tilde{r}_{\theta,n})\}$.

$$\hat{P}(\tilde{R} = r_s | \Theta = \theta) = \sum_{t_0=0}^{t} \hat{P}(\tilde{R} = r_s | \Theta = \theta, T = t - t_0) \hat{P}(T = t - t_0 | \Theta = \theta) \sum_{k_i} \hat{P}(\tilde{R} = r_s | \Theta = \theta, T = t - t_0) \hat{P}(T = t - t_0 | \Theta = \theta)$$

If we regroup $k_i$ realizations with the same $T_i$ we get:

$$\hat{P}(\tilde{R} = r_s | \Theta = \theta) = \sum_{t_0=0}^{t} \frac{1}{k_i} \sum_{i=1}^{n} \frac{1}{n} \sum_{i=1}^{n} e^{-\frac{-(\varphi(\tilde{r}_i^*, r_{\theta,i}))^2}{\sigma^2}} \frac{k_i}{n} = \frac{1}{n} \sum_{i=1}^{n} e^{-\frac{-(\varphi(\tilde{r}_i^*, r_{\theta,i}))^2}{\sigma^2}}.$$

Together with this manuscript, we provide the C++ implementation code of the Soft Margin and other estimators (downloadable here 3).

3http://lis.irb.hr/epidemic/
6 Soft Margin time complexity

The average run-time complexity $\overline{RT}$ of Soft Margin estimator is:

$$\overline{RT} \propto |\vec{r}^*| \times n \times \overline{RT}_M,$$

where the term $|\vec{r}^*|$ denotes the number of potential sources in the observed realization, the term $n$ denotes number of samples of the random variable $R_\theta$ or alternatively the number of simulations of a contagion process and $\overline{RT}_M$ denotes the average run-time complexity of sampling one realization from contagion process $M$. Sampling the realizations from a contagion process in our case is equal to one Monte Carlo simulation of stochastic contagion model and returning one realization vector $\vec{r}_{\theta,j}$. Note that in the worst-case scenario the number of potential sources is proportional to the network size $|\vec{r}^*| \propto N$, but in reality we are mostly interested in source detection problem when the number of potential sources is much smaller than the network size.

Note, that the calculations of likelihood for different sources $\theta$ in $\vec{r}^*$ are computed in scalable parallel way with the MapReduce paradigm. The "Map" step distributes the source independent problems to worker nodes and "Reduce" step collects likelihood estimators and provides source probability distribution.

In the case when contagion process is the SIR model on an arbitrary static network, the average run-time complexity for single SIR discrete simulation (NaiveSIR algorithm [2]) is:

$$\overline{RT}_{M1} \propto E(X_T) \times k \times T,$$

where the term $E(X_T)$ denotes the expected number of infected nodes up to temporal threshold $T$ and $k$ is the average node degree.

In the case when the contagion process is the SIR model on temporal network, the run-time complexity for single SIR discrete simulation is:

$$\overline{RT}_{M2} \propto L_T,$$

where $L_T$ denotes the number of interactions during epidemic process with duration $T$.

Note that after we have calculated the estimated PDF for each potential candidate node $\hat{f}_\theta(x)$, we can estimate source probabilities for different weight parameters $a$ since this step is far less demanding than the previous steps.

Table 1: The source detection execution times on an empirical temporal network [3]. Execution times of source detection are measured with parallel computation on 50 CPU cores on the AMD Opteron(tm) Processor 6380, 2.5 GHz each. The computations are done in parallel by using a high performance Message Passing Library with the C++ language. Averaging was done over 50 independent experiments where the initial moment $t_0$ was chosen in period between $[100 – 200]$ days, the initial source was randomly selected from the set of active nodes in $t_0$ moment with the SIR STD model ($p = 0.3, q = 0.01$) and realization $\vec{r}_{\theta}$ was observed at time $t = 300$ days. The run times are averaged over 50 independent experiments with the mean realization size $|\vec{r}^*|$ equal to 86. We have used the Soft Margin estimator, where the width parameter $a$ was chosen as a minimum of parameters from set: $\{1/2, (1/2)^2, (1/2)^3, ..., (1/2)^{15}\}$ for which the ML estimate converged up to 0.05 of relative change between consecutive simulations.

| Number of simulations | n = 5000 | n = 10000 | n = 15000 | n = 20000 |
|-----------------------|----------|-----------|-----------|-----------|
| Mean time [s]         | 2.9      | 5.8       | 8.7       | 11.6      |
In this section, we show the results for source detection on temporal networks for contagion with the SIR model with high transmission probability $p = 0.8$ with recovery parameter $q = 0.05$ (expected recovery is 20 days). In Figure 7 plot A, we show the results when starting $t_0$ was uniformly chosen from the interval $[100 - 200]$ day, the end of epidemic was set to the day $t = 300$ and we used different uniform priors ($\epsilon$) on $t_0$ moment. Plot B in the Figure 7 demonstrates the effect of detecting the source node from network with randomized temporal ordering with parameter $\Delta$.

Figure 7: The Source detection of simulated sexually transmitted infections spreading in an empirical spatio-temporal network of sexual contacts in Brazil. The experiment consists of 500 experiments where the initial moment $t_0$ was uniformly chosen in period between $[100 - 200]$ days, the initial source was randomly selected from the set of active nodes in the moment $t_0$ with the SIR model ($p = 0.8, q = 0.05$) and realization $r^*_t$ was observed at time $t = 300$ day. Figure A: The influence of prior knowledge about initial outbreak moment $[t_0 - \epsilon, t_0 + \epsilon]$. Figure B: The influence of detecting the source node from temporal networks with randomized temporal ordering of interactions within $\Delta$ days.

In all the cases so far, we have assumed that we know the states of all the nodes in the network at the temporal snapshot $t$. Now, we will show that we can relax that assumption. We will assume that we can only observe the states of a random subset $O \subseteq V$ of all the nodes in the network. In Figure 8, we show the performance results for the source detection of STD disease when we know the states of 100%, 50% and 20% of all the nodes in the network chosen randomly. Realization vectors $r^*_t$ now can have the following values: $\{0, 1, ?\}$, where the "?" denotes the unknown state. In order to apply our methodology, we only need to adopt the similarity function in a way that it can handle the unknown
states and determine the set of potential candidate sources $S$. We use the same similarity function like in the main manuscript (Jaccard similarity), but we neglect the comparison with the missing state "?". The set of potential candidates is the union of all the nodes with state "1" and all the nodes with state "?" which are not surrounded with neighbours with "0" state only (they cannot be the initial source).

Figure 8: The Source detection of simulated sexually transmitted infections spreading in an empirical spatio-temporal network of sexual contacts in Brazil when we know the states of 100%, 50% and 20% of all the nodes in the network chosen randomly. The experiment consists of 100 experiments where the initial moment $t_0$ was uniformly chosen in period between $[100 - 200]$ days, the initial source was randomly selected from the set of active nodes in the moment $t_0$ with the SIR model ($p = 0.3$, $q = 0.01$) and realization $r^*_t$ was observed at time $t = 300$ day.
References

[1] Lokhov AY, Mezard M, Ohta H, Zdeborova L (2013) Inferring the origin of an epidemic with dynamic message-passing algorithm. Unpublished, http://arxiv.org/abs/13035315

[2] Antulov-Fantulin N, Lancic A, Stefancic H, Sikic M (2013) Fastsim algorithm: A fast algorithm for the simulation of the epidemic spread in large networks by using the susceptible–infected–recovered compartment model. Information Sciences 239: 226 - 240.

[3] Rocha LEC, Liljeros F, Holme P (2011) Simulated epidemics in an empirical spatiotemporal network of 50,185 sexual contacts. PloS Computational Biology 7: e1001109–.

[4] Antulov-Fantulin N, Lancic A, Stefanic H, Sikic M, Smuc T (2013) Statistical inference framework for source detection of contagion processes on arbitrary network structures. Unpublished, http://arxiv.org/abs/13040018 .

[5] Dong W, Zhang W, Tan CW (2013) Rooting out the rumor culprit from suspects. Proc. of IEEE Intl. Symp. on Information Theory 2013 .

[6] Comin CH, da Fontoura Costa L (2011) Identifying the starting point of a spreading process in complex networks. Phys Rev E 84: 056105.