BAYESIAN INFERENCE OF DIFFUSION NETWORKS WITH UNKNOWN INFECTION TIMES

Shohreh Shaghaghian and Mark Coates

Department of Electrical and Computer Engineering, McGill University

ABSTRACT

The analysis of diffusion processes in real-world propagation scenarios often involves estimating variables that are not directly observed. These hidden variables include parental relationships, the strengths of connections between nodes, and the moments of time that infection occurs. In this paper, we propose a framework in which all three sets of parameters are assumed to be hidden and we develop a Bayesian approach to infer them. After justifying the model assumptions, we evaluate the performance efficiency of our proposed approach through numerical simulations on synthetic datasets and real-world diffusion processes.

***Index Terms*—** Information Diffusion, MCMC Inference, Gibbs Sampling

1. INTRODUCTION

Propagation of information has been widely studied in various domains. Despite different applications [1], all of the diffusion models have three main components in common: *Nodes*, i.e., the set of separate agents; *Infection*, i.e., the change in the state of a node that can be transferred from one node to the other; and *Causality*, i.e., the underlying structure based on which the infection is transferred between nodes. The term *cascade* is usually used to refer to the temporal traces left by a diffusion process. One of the main goals of studying the diffusion process is to infer the causality component using observations related to the cascades. While the majority of studies (e.g. [2–5]) assume that the cascades are perfectly observed, some studies ([6–10]) investigate scenarios in which the cascade trace is not directly observable or is at least partially missing. Two important examples of such scenarios are the outbreak of a contagious disease (with nodes being geographical regions) and the impact of external events on the stock returns of different assets. These studies either assume that some portion of the cascade data is observable and try to infer the causality structure from this portion (e.g. [8–10]) or infer the structure using some other observable property of the cascade without inferring the cascade trace itself (e.g. [6]). In this paper, we assume that the cascade or more precisely the infection times cannot be directly observed. Instead, we observe time series with statistics that change as a function of the true infection times. As opposed to the previous works, we intend to not only infer the causality structure but also estimate the unobserved infection times.

Another related body of literature addresses time series segmentation and investigates the techniques of detecting single ([11]) and multiple ([12]) changepoints in univariate ([13, 14]) and multivariate ([15, 16]) time series. Some of these methods involve an underlying graphical model that captures the correlation structure between the time series, but there is no notion of an infection network. In this paper, we develop a framework in which the infection times, parental relationships and link strengths can be estimated by simultaneously modeling the network structure and performing time series segmentation.

The paper is organized as follows. In the rest of this section, we briefly review related work. In Section 2, we describe our system model and formulate the diffusion problem. We present our proposed inference approach and discuss its modeling assumptions. We evaluate the performance of our suggested approach using both synthetic and real world datasets and present the simulation results in Section 3. The concluding remarks are made in Section 4.

Related Work: Most of the earlier work exploring techniques for inferring the structure of an infection or diffusion network assumed that cascades were perfectly observed. [2] proposes a generative probabilistic model of diffusion that aims to realistically describe how infections occur over time in a static network. The infection network and transmission rates are inferred by maximizing the likelihood of an observed set of infection times. [3] investigates the diffusion problem in an unobserved dynamic network for the case when the dynamic process spreading over the edges of the network is observed. Stochastic convex optimization is employed to infer the dynamic network. [4] proposes a Bayesian framework to estimate the posterior distribution of connection strengths and information diffusion paths given the observed infection times. [5] studies the creation of new links in the diffusion network and proposes a joint continuous-time model of information diffusion and network evolution.

In contrast to the work described above, some studies focus on the problem where cascades are not perfectly observed. In [6], it is assumed that the partially observed probabilistic information about the state of each node is provided, but the

978-1-4673-7802-4/16/$31.00 ©2016 IEEE

exact state transition times are not observed. These transition times are related to the observed trace via the noise dynamics function. The underlying network is inferred by minimizing the expected loss over all realizations of the observable trace. [8] studies the theoretical learnability of tree-like graphs in a setting where only the initial and final states are observed. The goal in [9] is to reconstruct the so called node couplings using dynamic message-passing equations when the cascade observations are only partially available. [10] develops a two-stage framework to identify the infection source when the node infections are only partially observed and the diffusion trace is incomplete. This paper is categorized in this second group of studies by proposing an approach to simultaneously infer the structure and casacade trace of a diffusion process.

2. SYSTEM MODEL AND INFERENCE PROCEDURE

We consider a set of N nodes $\mathcal{N} = \{1, \dots, N\}$ and assume that node $s \in \mathcal{N}$ is the source of a contagion C which is transmissible to other nodes of the network. When C is transferred from node j to node i ($i, j \in \mathcal{N}$), we say node i is infected by node j. In this case, we refer to node j as the parent of node i, and denote it by z_i. We model this infection process by a directed, weighted graph $G = (\mathcal{N}, \mathcal{E}, \boldsymbol{\alpha}_{N\times N})$ where $\mathcal{E}$ is the set of weighted edges, and α is a $N \times N$ link strength matrix. Component α_{ij} of this matrix denotes the strength of the link between two nodes i and j. A directed edge $j \to i$ exists if and only if $z_i = j$. The set of potential parents for node i is denoted by π_i (i.e. $z_i \in \pi_i$). The definitions of parents and candidate parents simply implies that $\forall j \in \pi_i : t_j < t_i$ and $\forall j \notin \pi_i : \alpha_{ij} = 0$.

As mentioned in Section 1, we focus on the scenarios where none of the main infection parameters (link strengths, parents, and infection times) are directly observed. We assume that the only observation we get from an arbitrary node $i \in \mathcal{N}$ is a discrete time signal of length T denoted by $\mathbf{d}_i = \{d_i^n\}_{n=1:T}$. We denote the set of all observed time signals by $\mathbf{d} = (\mathbf{d}_1, \dots, \mathbf{d}_N)$. The goal is to infer the infection parameters $(\mathbf{z}, \mathbf{t}, \boldsymbol{\alpha})$ that best explain the received signal vector $\mathbf{d}$ where $\mathbf{z} = (z_1, \dots, z_N)$ and $\mathbf{t} = (t_1, \dots, t_N)$. More precisely, we aim to find the most probable set of parameters $(\mathbf{z}^*, \mathbf{t}^*, \boldsymbol{\alpha}^*)$ conditioned on the received signals $\mathbf{d}$, i.e.

$$(\mathbf{z}^*, \mathbf{t}^*, \boldsymbol{\alpha}^*) = \underset{(\mathbf{z},\mathbf{t},\boldsymbol{\alpha})}{\arg\max} \quad f(\mathbf{z}, \mathbf{t}, \boldsymbol{\alpha}|\mathbf{d}) \tag{1}$$

In order to solve (1), we need to first derive the joint conditional distribution $f(\mathbf{z}, \mathbf{t}, \boldsymbol{\alpha}|\mathbf{d})$. Using Bayes' rule we have,

$$f(\mathbf{z}, \mathbf{t}, \boldsymbol{\alpha}|\mathbf{d}) = \frac{f(\mathbf{d}|\mathbf{t}, \mathbf{z}, \boldsymbol{\alpha}) f(\mathbf{t}|\mathbf{z}, \boldsymbol{\alpha}) f(\mathbf{z}|\boldsymbol{\alpha}) f(\boldsymbol{\alpha})}{f(\mathbf{d})} \tag{2}$$

We consider proper prior distributions for components of equation (2). As justified in [4], we assume that link strengths α_{ij} are independent and model their probability distribution by a Gamma distribution with parameters a_{ij} and b_{ij} i.e. $\alpha_{ij} \sim \Gamma(a_{ij}, b_{ij})$. Therefore,

$$f(\boldsymbol{\alpha}) = \prod_{i\in\mathcal{N}, j\in\pi_i} f(\alpha_{ij}) = \prod_{i\in\mathcal{N}, j\in\pi_i} \frac{x^{a_{ij}-1} e^{-\frac{x}{b_{ij}}}}{\Gamma(a_{ij}) b_{ij}^{a_{ij}}} \tag{3}$$

We also assume that conditioned on the link strengths, the nodes' parents are independent and follow multinomial distributions i.e.

$$f(\mathbf{z}|\boldsymbol{\alpha}) = \prod_{i\in\mathcal{N}} f(z_i|\alpha_{ij_{j\in\pi_i}}) = \prod_{i\in\mathcal{N}} \frac{\alpha_{iz_i}}{\sum_{j\in\pi_i} \alpha_{ij}} \tag{4}$$

The next step is to consider a proper prior conditional distribution for infection times. As proposed in [17], we assume t_i follows an exponential distribution with parameter α_{iz_i}. Without loss of generality, we can assume that $t_1 \geq t_2 \geq \cdots \geq t_N$. Therefore,

$$f(\mathbf{t}|\mathbf{z}, \boldsymbol{\alpha}) = \prod_{i\in\mathcal{N}} f(t_i|\mathbf{z}, \boldsymbol{\alpha}, t_{i+1:N}) = \prod_{i\in\mathcal{N}} \alpha_{iz_i} e^{-\alpha_{iz_i}(t_i - t_{z_i})} \tag{5}$$

Finally, we assume that node i's observed data, $\mathbf{d}_i$, is independent of the observations from other nodes and that it follows two different distributions before and after being infected at t_i. Hence,

$$f(\mathbf{d}|\mathbf{z}, \mathbf{t}, \boldsymbol{\alpha}) = \prod_{i\in\mathcal{N}} f(\mathbf{d}_i|t_i) \tag{6}$$

With the proposed distributions in (3)-(6), we can calculate the probability of any arbitrary set $(\mathbf{z}_0, \mathbf{t}_0, \boldsymbol{\alpha}_0)$ up to a constant $\frac{1}{f(\mathbf{d})}$ using (2). Since the optimization problem of (1) cannot be easily solved, we use MCMC methods to sample from a probability distribution $f(\mathbf{z}, \mathbf{t}, \boldsymbol{\alpha}|\mathbf{d})$. We use Gibbs sampling to generate samples of this posterior distribution. In other words, we use full conditional distributions for each of the infection parameters t_i, z_i, α_{ij} ($i, j \in \mathcal{N}$) to generate samples. We denote the parents and infection times of all the nodes in the network except node i respectively by $\mathbf{z}_{\bar{i}}$, $\mathbf{t}_{\bar{i}}$. Also, the link strength of all the possible links except the link between nodes i and j is denoted by $\boldsymbol{\alpha}_{\overline{ij}}$. Using Bayes' rule, the full conditional probablities for Gibbs sampling are:
(a) For parent of an node i,

$$f(z_i|\mathbf{d}, \mathbf{z}_{\bar{i}}, \mathbf{t}, \boldsymbol{\alpha}) \propto f(t_i|z_i, \alpha_{iz_i}, t_{z_i}) f(z_i|\alpha_{ij_{j\in\pi_i}}) \tag{7}$$

(b) For infection time of an node i,

$$f(t_i|\mathbf{d}, \mathbf{z}, \mathbf{t}_{\bar{i}}, \boldsymbol{\alpha}) \propto f(d_i|t_i) f(t_i|z_i, \alpha_{iz_i}, t_{z_i}) \prod_{k\in C_i} f(t_k|\alpha_{ki}, t_i) \tag{8}$$

(c) For link strength between nodes i and $j \in \pi_i$,

$$f(\alpha_{ij_{j\in\pi_i}}|\mathbf{d}, \mathbf{z}, \mathbf{t}, \boldsymbol{\alpha}_{\overline{ij}}) \propto f(t_i|z_i, \alpha_{iz_i}, t_{z_i}) f(z_i|\alpha_{ij_{j\in\pi_i}}) f(\alpha_{ij}) \tag{9}$$

We evaluate the proficiency of the proposed inference approach in Section 3.

3. SIMULATION RESULTS

3.1. Synthetic Data

We generate a dataset based on the model (3)-(6). We first randomly choose π_is (for all $i \in \mathcal{N}$) and an underlying directed tree $\mathcal{T}$ with adjacency matrix $\mathbf{A} = [A_{ij}]$, where $A_{ij} = 1$ if and only if there is a directed edge from i to j. The link strength value α_{ij} ($j \in \pi_i$) is generated using the gamma distribution $\Gamma(a_1, b_1)$ if $A_{i,j} = 1$ and $\Gamma(a_2, b_2)$ if $A_{i,j} = 0$. We refer to these α values as *true alphas* and denote them by $\boldsymbol{\alpha}^R = [\alpha_{ij}^R]_{N\times N}$. Then, we choose the parent of node i i.e. z_i from all the nodes $j \in \pi_i$ based on a random sampling with weights α_{ij}. These parents are called *true parents* and are denoted by $\mathbf{z}^R = (z_1^R, \ldots, z_N^R)$. Knowing the values of z_i and α_{iz_i}, we then generate the *true infection times* $\mathbf{t}^R = (t_1^R, \ldots, t_N^R)$ based on the exponential distributions described in (5). Finally, we generate the data d_i based on two different Gaussian distributions with parameters (μ_{1i}, σ_{1i}) and (μ_{2i}, σ_{2i}) for all nodes $i \in \mathcal{N}$, i.e.

$$f(d_i|t_i) = \frac{e^{-[\frac{\sum_{n=1}^{t_i-1}(d_i^n-\mu_{1i})^2}{2\sigma_{1i}^2} + \frac{\sum_{n=t_i}^{T}(d_i^n-\mu_{2i})^2}{2\sigma_{2i}^2}]}}{\sqrt{2\pi}^T \sigma_{1i}^{t_i}\sigma_{2i}^{T-t_i}} \quad (10)$$

We generate M samples using full conditional distributions of equations (7)-(9) to infer the network parameters $(\mathbf{z}^R, \mathbf{t}^R, \boldsymbol{\alpha}^R)$. We denote the set of all generated samples by $\mathcal{M}$ and refer to the mth sample as S^m. The parent vector, infection time vector, and strength matrix of the mth sample are respectively denoted by $S_{\mathbf{z}}^m$, $S_{\mathbf{t}}^m$, and $S_{\boldsymbol{\alpha}}^m$. Denoting the most observed parent-infection time pair (i.e. the pair that has been repeated the most among the M generated samples) by $(\hat{\mathbf{z}}, \hat{\mathbf{t}})$, we estimate the components of the link strength $\hat{\boldsymbol{\alpha}} = [\hat{\alpha}_{ij}]_{N\times N}$ by $\hat{\alpha}_{ij} = \frac{1}{|\mathcal{S}|}\sum_{k\in\mathcal{S}}[S_{\boldsymbol{\alpha}}^k]_{ij}$ where $\mathcal{S} = \{m \in \mathcal{M} | S_{\mathbf{z}}^m = \hat{\mathbf{z}}, S_{\mathbf{t}}^m = \hat{\mathbf{t}}\}$. In order to evaluate the performance of our proposed inference approach, two main questions should be answered: (1) Does the network structure improve detection of infection times? (2) How much accuracy is lost in terms of detecting the parents and estimating link strengths when time series are observed instead of the actual infection times?

The first question can be answered by comparing the accuracy of infection time estimates for two cases. In the first case, we detect the infection time of each node independently (i.e. $\hat{t_i}' = \arg\max_{t_i} f(t_i|d_i)$), while in the second case we exploit the network structure to find the infection times as explained in Section 2. We denote the vector of all $\hat{t_i}'$s by $\hat{\mathbf{t}}'$ and define the infection time deviation function $D_t(\mathbf{t}_x^1, \mathbf{t}_x^2)$ as the average number of samples that are different in the arbitrary infection time vectors $\mathbf{t}_x^1 = (t_{x1}^1, \ldots t_{xN}^1)$ and $\mathbf{t}_x^2 = (t_{x1}^2, \ldots t_{xN}^2)$ i.e. $\forall \mathbf{t}_x^1, \mathbf{t}_x^2 \in \mathcal{R}^{1\times N}: \quad D_t(\mathbf{t}_x^1, \mathbf{t}_x^2) \triangleq \frac{1}{N}\sum_{i=1}^{N} |t_{x_i}^1 - t_{x_i}^2|$. Figure 1 shows the average and 95% confidence intervals of deviation values for $\mathbf{t}_x^1 = \hat{\mathbf{t}}, \hat{\mathbf{t}}'$ and $\mathbf{t}_x^2 = \mathbf{t}^R$ in 100 networks

Scenario	μ_2	b_2
A	100	0.9
B	100	0.6
C	11	0.9
D	11	0.6

Table 1: Test Scenarios

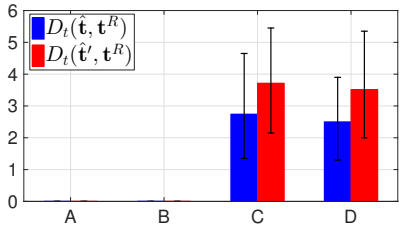


Fig. 1: Infection Time Deviations

of $N = 20$ nodes using four extreme sets of parameters described in Table 1. In all these scenarios, $\mu_{i1} = 10$, $\mu_{i2} = \mu_2$, $\sigma_{i1} = \sigma_{i2} = 1$ for all $i \in \mathcal{N}$ and $a_1 = 9$, $b_1 = 0.5$, $a_2 = 10$. $M = 10^5$ samples are generated and the first 10^3 generated samples are discarded. As we see in Figure 1, in scenarios *A* and *B*, infection times can be detected with high likelihood thus both performance metrics are zero. However, in scenarios *C* and *D*, we see that exploitation of the network structure results in smaller deviation from the true values. The infection time estimates are in average more accurate.

We now compare our proposed framework with a more idealized situation in which the $\mathbf{t}_i$ values are known. We denote the parents and link strengths estimated with knowledge of the infection times by $\hat{\mathbf{z}}'$ and $\hat{\boldsymbol{\alpha}}'$ and define deviation functions $D_z(\mathbf{z}_x^1, \mathbf{z}_x^2)$ and $D_\alpha(\boldsymbol{\alpha}_x^1, \boldsymbol{\alpha}_x^2)$. The parent deviation function $D_z(\mathbf{z}_x^1, \mathbf{z}_x^2)$ is defined as the number of nodes whose parents are different in $\mathbf{z}_x^1 = (z_{x1}^1, \ldots, z_{xN}^1)$ and $\mathbf{z}_x^2 = (z_{x1}^2, \ldots, z_{xN}^2)$ i.e. $\forall \mathbf{z}_x^1, \mathbf{z}_x^2 \in \mathcal{R}^{1\times N}: \quad D_z(\mathbf{z}_x^1, \mathbf{z}_x^2) \triangleq \sum_{i=1}^{N} I(z_{xi}^1 - z_{xi}^2)$, where $I(x) = 1$ if $x \neq 0$ and $I(x) = 0$ otherwise. Finally, for the deviation of link strengths we have, $\forall \boldsymbol{\alpha}_x^1, \boldsymbol{\alpha}_x^2 \in \mathcal{R}^{N\times N}: D_\alpha(\boldsymbol{\alpha}_x^1, \boldsymbol{\alpha}_x^2) \triangleq \frac{1}{N}\sum_{\hat{\alpha}_{ij}>0} |\alpha_{xij}^1 - \alpha_{xij}^2|$.

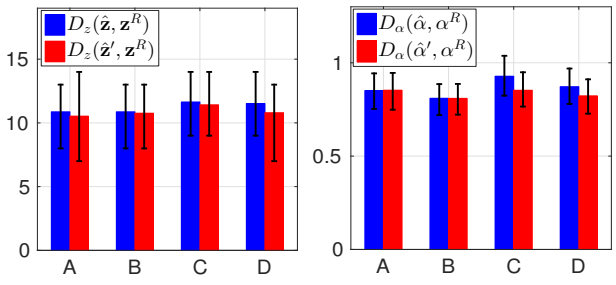


Fig. 2: Deviations in Detection of $\mathbf{z}$ and $\boldsymbol{\alpha}$

Figure 2 shows the values of the defined performance metrics for $\mathbf{z}_x^1 = \hat{\mathbf{z}}, \hat{\mathbf{z}}'$ and $\mathbf{z}_x^2 = \mathbf{z}^R$. We see that in scenarios *C* and *D* (where the noise is greater and infection times are more difficult to estimate), not knowing the exact infection times results in larger deviations in estimating the network parameters. Overall, however, the deterioration in estimation accuracy is not dramatic.

3.2. Real Data

We study the outbreak of Avian Influenza (H5N1 HPAI) [18]. Figure 3 shows the observed locations of reported infections for both domestic and wild bird species for the period of January 2004 to February 2016. We divide the observation points to eight main regions using K-means clustering and generate a time series $\mathbf{d}_i$ to the ith region. The value of this time series at day n (d_i^n) denotes the number of separate locations within the region i in which the disease was reported on that day. We

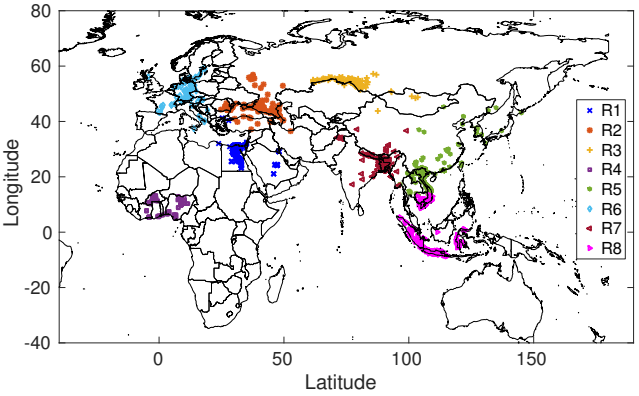


Fig. 3: H5N1 HPAI outbreak in 2004-2016

model the number of observations in each region by a Poisson distribution:

$$f(\mathbf{d}_i|t_i) = \prod_{n=1}^{t_i-1} \frac{\lambda_{1i}{}^{d_i^n} e^{-\lambda_{1i}}}{d_i^n!} \prod_{n=t_i}^{T} \frac{\lambda_{2i}{}^{d_i^n} e^{-\lambda_{2i}}}{d_i^n!} \qquad (11)$$

where $\lambda_{1i} = \frac{\sum_{n=t_1}^{t_i} d_i^n}{t_i-1}$ and $\lambda_{2i} = \frac{\sum_{n=t_i+1}^{T} d_i^n}{T-t_i+1}$. The link strength parameters a_{ij} and b_{ij} of equation (3) are derived by fitting a gamma distribution to the inverse of distances between observation points of regions i and j. Figure 4 shows the time series for the eight regions. Regions R5 and R8 are the first regions in which the disease is observed. The first infections for these regions were reported on the same day, so we assume that they were both sources of the infection. We infer the infection parameters for the period 2004-2007 by generating $M = 10^6$ samples and discarding the first 10^4 ones. The green line in Figure 4 shows the end of the study period. Region R4 has almost no reported infections for this period so we exclude it when estimating the underlying infection graph. The detected infection times are shown in Figure 4 by red vertical lines. Figure 5 shows the four most probable configurations of the infection network and their percentages among generated samples. The edge weights in these graphs are estimated link strengths.

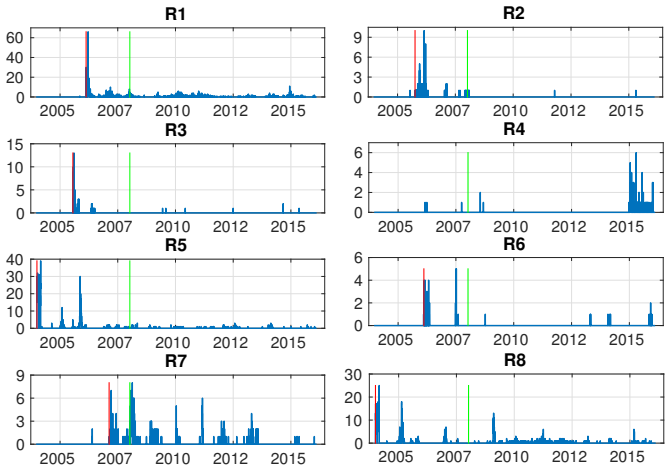
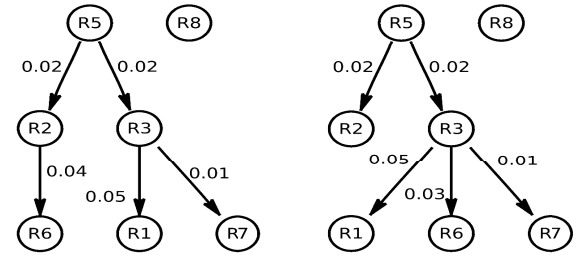
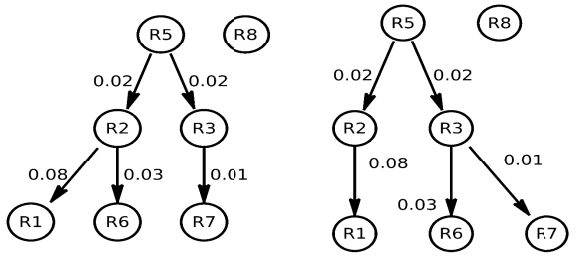


Fig. 4: Observed Time Series in the Impacted Regions



(a) Configuration 1, Weight= 48% (b) Configuration 2, Weight= 23%



(c) Configuration 3, Weight= 17% (d) Configuration 4, Weight= 10%

Fig. 5: Most Possible Network Configurations

4. CONCLUSION

In this paper, we have proposed a framework for inferring the underlying graph based on which an infection is diffused in a network structure. We designed the model to address scenarios where the infection times are unknown. We evaluated the performance using synthetic datasets, demonstrating that (i) the incorporation of the model could improve the estimation of infection times compared to univariate changepoint estimation when the data match the model; and (ii) the absence of exact knowledge of infection times does not lead to significant deterioration in performance. We illustrated how the model and inference methodology could be applied to analyze the outbreak of a virus. Incorporating multiple changepoint detection approaches can be studied as a future work.

5. REFERENCES

[1] A. Guille, H. Hacid, C. Favre, and D. A. Zighed, "Information diffusion in online social networks: A survey," *ACM SIGMOD Record*, vol. 42, no. 2, pp. 17–28, 2013.

[2] M. Gomez-Rodriguez, D. Balduzzi, B. Schölkopf, G. T. Scheffer *et al.*, "Uncovering the temporal dynamics of diffusion networks," in *Int. Conf. on Mach. Learn. (ICML)*, 2011, pp. 561–568.

[3] M. Gomez-Rodriguez, J. Leskovec, and B. Schölkopf, "Structure and dynamics of information pathways in online media," in *Proc. of ACM Int. Conf. on Web Search and Data Min.*, 2013, pp. 23–32.

[4] V. R. Embar, R. K. Pasumarthi, and I. Bhattacharya, "A bayesian framework for estimating properties of network diffusions," in *Proc. of ACM SIGKDD Int. Conf. on Knowl. Discov. and Data Min.*, 2014, pp. 1216–1225.

[5] M. Farajtabar, Y. Wang, M. Gomez-Rodriguez, S. Li, H. Zha, and L. Song, "Coevolve: A joint point process model for information diffusion and network coevolution," in *Advances in Neural Info. Process. Syst.*, 2015, pp. 1945–1953.

[6] E. Sefer and C. Kingsford, "Convex risk minimization to infer networks from probabilistic diffusion data at multiple scales," in *Int. Conf. on Data Eng. (ICDE)*, 2015, pp. 663–674.

[7] E. Sadikov, M. Medina, J. Leskovec, and H. Garcia-Molina, "Correcting for missing data in information cascades," in *Proc. of ACM Int. Conf. on Web Search and Data Min.* ACM, 2011, pp. 55–64.

[8] K. Amin, H. Heidari, and M. Kearns, "Learning from contagion (without timestamps)," in *Int. Conf. on Mach. Learn. (ICML)*, 2014, pp. 1845–1853.

[9] A. Y. Lokhov and T. Misiakiewicz, "Efficient reconstruction of transmission probabilities in a spreading process from partial observations," *arXiv preprint arXiv:1509.06893*, 2015.

[10] M. Farajtabar, M. Gomez-Rodriguez, N. Du, M. Zamani, H. Zha, and L. Song, "Back to the past: Source identification in diffusion networks from partially observed cascades," in *Int. Conf. on Artif. Intell. and Stat.*, 2015.

[11] I. A. Eckley, P. Fearnhead, and R. Killick, "Analysis of changepoint models," *Bayesian Time Series Model.*, pp. 205–224, 2011.

[12] R. Killick, P. Fearnhead, and I. Eckley, "Optimal detection of changepoints with a linear computational cost," *J. of the American Stat. Assoc.*, vol. 107, no. 500, pp. 1590–1598, 2012.

[13] P. Fearnhead, "Exact bayesian curve fitting and signal segmentation," *IEEE Trans. on Signal Process.*, vol. 53, no. 6, pp. 2160–2166, 2005.

[14] ——, "Exact and efficient bayesian inference for multiple changepoint problems," *Stat. and Comput.*, vol. 16, no. 2, pp. 203–213, 2006.

[15] X. Xuan and K. Murphy, "Modeling changing dependency structure in multivariate time series," in *Proc. of Int. Conf. on Mach. learn.*, 2007, pp. 1055–1062.

[16] D. S. Matteson and N. A. James, "A nonparametric approach for multiple change point analysis of multivariate data," *J. of the American Stat. Assoc.*, vol. 109, no. 505, pp. 334–345, 2014.

[17] M. Gomez-Rodriguez, J. Leskovec, and A. Krause, "Inferring networks of diffusion and influence," *ACM Trans. on Knowl. Discov. from Data (TKDD)*, vol. 5, no. 4, p. 21, 2012.

[18] "EMPRES-i global animal disease information system," Downloaded from . http://empres-i.fao.org., Jan. 2016.