NETWORK INFECTION SOURCE IDENTIFICATION UNDER THE SIRI MODEL

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ABSTRACT

We study the problem of identifying a single infection source in a network under the susceptible-infected-recovered-infected (SIRI) model. We describe the infection model via a state-space model, and utilizing a state propagation approach, we derive an algorithm based on dynamic message passing (DMP), which we call DMP+, to infer the infection source. The DMP+ algorithm uses the partial or complete observations of node states at a particular time, where the elapsed time from the start of the infection is unknown. It is able to incorporate side information (if any) of the observed states of a subset of nodes at different times, and of the prior probability of each infected or recovered node to be the infection source. Simulation results suggest that the DMP+ estimator outperforms the DMP and Jordan center estimators over a wide range of infection and re-infection rates.

Index Terms—Infection source identification, SIRI model, side information, regular tree, Facebook network

1. INTRODUCTION

Consider an infection, which can be a computer virus, disease or rumor, spreading in a network of nodes. A node is said to be in infected state if it “possesses” that infection [1][2]. For example, in the case of a rumor spreading in an online social network like Facebook, an infected node is a user who has posted the rumor on his social page in the recent past. A node is in susceptible state if it is not infected and has not been exposed to the infection. A recovered node corresponds to a user who has removed the rumor post or the post is not within a predefined number of most recent postings of the user. An infection follows a susceptible-infected (SI) model if a susceptible node may become infected if it has infected neighbors, and an infected node stays infected [3]. The infection has a susceptible-infected-recovered (SIR) model if an infected node may recover from an infection but then stays uninfected forever [3], and a susceptible-infected-recovered-infected (SIRI) model if a recovered node may again relapse into an infected state, i.e., it does not require any infected neighbors to reinfect it [4][5][6]. In the Facebook example, a user may repost a rumor after he has removed it due to influences external to the Facebook network [6]. The SIRI model reduces to an SI or SIR model if the probability of recovery or reinfection is equal to zero, respectively.

Suppose that after an unknown elapsed amount of time since the start of an infection spreading, we have a snapshot of the states of a subset of the network, and we want to identify the infection source based on this snapshot and the network topology. This is known as the network source identification problem, and has been extensively studied under the SI and SIR models. Various source estimators such as the distance (or rumor) center [1][2][7], Jordan center [8][9][10], dynamic message passing (DMP) estimator [11][12], belief propagation (BP) estimator [12][13], have been proposed and studied. Each of these estimators seeks to find an approximate maximum likelihood (ML), maximum a posteriori (MAP), or most likely infection path estimator of the true infection source, and may require different levels of prior information about the spreading process. For example, the distance and Jordan center estimators do not require any knowledge of the infection rates, which are utilized by DMP and BP estimators.

In this paper, we consider identifying a single infection source in a network under the SIRI model, which is more general than the SI and SIR models. The SIRI model is frequently used to describe the transmission of a contagious disease with relapse, such as bovine tuberculosis or human herpes virus, in which recovered individuals may revert back to the infectious class due to reactivation of the latent infection or incomplete treatment [1][2][4][14]. The model is also used as a simplified version of general multi-strain models, where after an initial infection, immunity against one strain only gives partial immunity against a genetically close mutant strain [15]. A further example of SIRI type of infection spreading is rumor spreading in an online social network, as alluded to earlier in the Facebook example. It is thus of both practical and theoretical interest to consider infection spreading and source identification in a network under the SIRI model. However, the problem is more challenging than the one under the SI and SIR models because a node may become infected and recovered multiple times. Therefore, it is unclear if the infection source estimators currently proposed in the literature can be applied directly to the SIRI model, and if this will lead to significant performance deterioration.

In this paper, we aim to find an approximate MAP estimator for inferring the infection source under the SIRI model. Our estimator is derived as a non-trivial extension of the DMP estimator, which cannot be applied directly to the SIRI model because it violates the assumption of unidirectional state transitions [16]. Our estimator is also related to the revised version of DMP called DMPr in [13], which is also applicable only to the SIR model. Furthermore, our new estimator is able to incorporate side information such as the prior probability of each candidate node being the infection source, and additional observations on subsets of nodes in periods other than the snapshot time. We call our new estimator the DMP+ estimator. Simulations are performed on random regular tree networks and a subset of Facebook network to evaluate the proposed estimator and compare its performance with those of the Jordan center estimator, and the DMP estimator. Our simulation results suggest that the new DMP+ estimator outperforms both the Jordan center and DMP estimator over a wide range of infection and reinfection rates.

2. INFECTION MODEL AND ASSUMPTIONS

In this section, we characterize the SIRI infection spreading using a state-space approach [17]. Throughout this paper, we assume a common underlying probability space with probability measure \( P \). We also use \( 0 : n \) to denote the integer set \( \{0,1,\ldots,n\} \).
Let the network over which the infection spreads be described by a directed graph, \( \mathcal{N}, \mathcal{E} \), where \( \mathcal{N} \triangleq 1 : N \) is the node set and \( \mathcal{E} \) is the edge set. A directed edge \((l,k)\) exists from node \( l \) to node \( k \) if node \( l \) can infect node \( k \), in which case node \( l \) is said to be an in-neighbor of node \( k \), and conversely node \( k \) is said to be an out-neighbor of node \( l \). We denote the set of in-neighbors of a node \( k \) as \( \mathcal{N}_k \).

Suppose that there is a single node \( s^* \) in the network that starts the infection at time 0, and suppose that we observe the node states of a partial or complete set of nodes in the network at a particular time \( T_j \), which we call the snapshot time. We assume that \( T_j \) is unknown, and that time is discretized into \( 0 : T_j \). Let \( S, I, \) and \( R \) denote the susceptible, infected, and recovered states, respectively. Let \( \alpha_{lk} \) be the probability of an infected node \( l \) infecting an out-neighbor \( k \) within a time slot, where \( \alpha_{lk} = 0 \) if \( l \notin \mathcal{N}_k \). Let \( \beta_k \) be the probability of an infected node \( k \) reducing to within a time slot, and \( \gamma_k \) be the probability of a recovered node \( k \) being infected again within a time slot, where \( \gamma_k = 0 \) if \( \beta_k = 0 \). These probabilities are assumed to be given or have been inferred prior. The possible state transitions of a node are depicted in Fig. 1. It is evident that the infection process at node \( k \) reduces to the SIR model if \( \gamma_k = 0 \), and the SI model if we further have \( \beta_k = 0 \). This admits a heterogeneous spreading model containing different infection processes of SI, SIR and SIRI at different nodes. Our model thus subsumes those studied in \([1,2,8,13,16]\).

We assume that conditioned on the node states in time slot \( t-1 \), all node transitions in time slot \( t \) are independent of each other.

We use \( P^S_k(t) \), \( P^I_k(t) \) and \( P^R_k(t) \) to denote the probabilities of a node \( k \) in \( \mathcal{N} \) to be in states \( S, I, \) and \( R \) at time \( t \), respectively. Adopting a state-space modeling approach, we define these three probabilities as state variables. The infection model is then obtained by describing the evolution of the three state variables in time using difference equations. Using the state transitions for the SIRI model, we have for all \( t \in 1 : T_j \) and \( k \in \mathcal{N} \),

\[
P^S_k(t) = \mathbb{P}(\hat{U}_k(t-1) \mid S_k(t-1)) \cdot P^S_k(t-1),
\]

\[
P^I_k(t) = \beta_k P^S_k(t-1) + (1 - \gamma_k) P^R_k(t-1),
\]

\[
P^R_k(t) = 1 - P^S_k(t) - P^R_k(t),
\]

where \( \hat{U}_k(t-1) \) is the event that no in-neighbor passes the infection to node \( k \) in during time \( t-1 \) to \( t \), and \( S_k(t-1) \) is the event that node \( k \) is in state \( S \) at time \( t-1 \). By assuming that in-neighbors pass infection to a node independently, we can express the probability \( \mathbb{P}(\hat{U}_k(t-1) \mid S_k(t-1)) \) as,

\[
\mathbb{P}(\hat{U}_k(t-1) \mid S_k(t-1)) = \prod_{l \in \mathcal{N}_k} \left(1 - \alpha_{lk} P^I_k(S_k(t-1)=1) \right),
\]

where \( P^I_k(S_k(t-1)=1) \triangleq \mathbb{P}(I_k(t) \mid S_k(t-1)=1) \), for all \( t_1, t_2 \in 0 : T_j \), which is the probability that node \( l \) is in state \( I \) at time \( t_2 \) given that node \( k \) is in state \( S \) at time \( t_1 \). (Note that equation (4) is exact if the graph \( (\mathcal{N}, \mathcal{E}) \) is acyclic.) To complete the model, we need to have an explicit expression for \( P^I_k(S_k(t-1) \mid t=1) \).

To that end, we introduce two classes of auxiliary state variables. For all \( t \in 0 : T_j \), \( l \in \mathcal{N}_k \), \( k \in \mathcal{N} \), let \( \theta_{lk}(t) \) be the the probability of a node \( l \) not infecting its out-neighbor \( k \) up to time \( t \), given that node \( k \) is in state \( S \) at time \( t \); and let \( \phi_{lk}(t) \) be the probability of a node \( l \) to be in state \( I \) at time \( t \) and not infecting its out-neighbor \( k \) up to time \( t \), given that node \( k \) is in state \( S \) at time \( t \). Then it can be shown that

\[
P^I_k(S_k(t-1) \mid t=1) = \frac{\theta_{lk}(t-1)}{\theta_{lk}(t-1)},
\]

where \( \theta_{lk}(t-1) \) and \( \phi_{lk}(t-1) \) are updated by

\[
\theta_{lk}(t) = \theta_{lk}(t-1) - \alpha_{lk} \phi_{lk}(t-1),
\]

\[
\phi_{lk}(t) = (1 - \alpha_{lk})(1 - \beta_k) \phi_{lk}(t-1) + \gamma_k P^I_k(S_k(t-1) \mid t),
\]

and the auxiliary conditional probabilities are computed from

\[
P^I_k(S_k(t-1) \mid t=1) = \frac{\gamma_k P^I_k(S_k(t-1) \mid t) + (1 - \alpha_{lk})(1 - \beta_k) \phi_{lk}(t-1)}{\theta_{lk}(t-1)},
\]

and

\[
P^I_k(S_k(t-1) \mid t=1) = 1 - P^I_k(S_k(t-1) \mid t) - P^I_k(S_k(t-1) \mid t),
\]

In comparison to the DMP equations \([11]\), the new term \( \gamma_k P^I_k(S_k(t-1) \mid t) \) arises from the reinfection of a recovered node, which is unique to the SIRI infection model. This term is then computed by \([9]\), which further depends on the new equation \((5)\) and equation \((6)\).

The key equation \((5)\) follows from an important observation:

\[
P^S_k(t) = P^S_k(0) \prod_{l \in \mathcal{N}_k} \theta_{lk}(t-1) \prod_{l \in \mathcal{N}_k} \theta_{lk}(t-1) = P^S_k(t-1) \prod_{l \in \mathcal{N}_k} \theta_{lk}(t-1)
\]

\[
= P^S_k(t-1) \prod_{l \in \mathcal{N}_k} \left(1 - \alpha_{lk} \phi_{lk}(t-1) \right)
\]

\[
= P^S_k(t-1) \prod_{l \in \mathcal{N}_k} \left(1 - \alpha_{lk} P^I_k(S_k(t-1) \mid t-1) \right),
\]

where the last equality follows from \([1] \) and \([4]\). We note that for a directed acyclic graph, the above equations are exact, while we will treat these as approximations for general network graphs.

In summary, the SIRI infection model consists of the basic state dynamics described by \((1)\) \((4)\) and the auxiliary state dynamics described by \((5)\) \((7)\). The initial conditions of these equations will be given in the next section.

We proceed to associate virtual observation equations with the state dynamics. Let \( \mathcal{N}^{\mathcal{S}}_r, \mathcal{N}^{\mathcal{I}}_r, \) and \( \mathcal{N}^{\mathcal{R}}_r \) denote the sets of nodes observed to be in states \( S, I, \) and \( R \) at time \( t \), respectively; and \( \mathcal{N}^{\mathcal{S}^R}_r \) denote the set of nodes observed to be in an uninfected state but are indistinguishable to be in state \( S, I, \) or \( R \) at time \( t \). The rest of the nodes whose states are unknown or not observed are collected into a set denoted by \( N^{\mathcal{S}^I}_r \), i.e., \( \mathcal{N}^{\mathcal{S}^I}_r = \mathcal{N} \setminus (\mathcal{N}^{\mathcal{S}}_r \cup \mathcal{N}^{\mathcal{I}}_r \cup \mathcal{N}^{\mathcal{R}}_r \cup \mathcal{N}^{\mathcal{S}^R}_r) \). Stack the three states into a single vector as \( P_k(t) \triangleq [P^S_k(t) P^I_k(t) P^R_k(t)]^T \). We define the virtual observations as

\[
y_k(t) = C_k(t) P_k(t),
\]

where the observation vector \( C_k(t) \in \mathbb{R}^{1 \times 3} \) takes one of the feasible values:
3. IDENTIFYING THE INFECTION SOURCE

Other than observing the partial or complete node states at an observation snapshot, we may also have some side information. For example, we may have information regarding the states of some nodes in time slots other than the snapshot time. Alternatively, we may have a priori knowledge of the infection process to allow us to assign prior probabilities of each candidate node to be the infection source. We introduce notations to represent this information before formulating the source identification problem.

For each node $k$, let $W_k$ be the set of time slots during which the state of node $k$ is observed, which includes the snapshot time. We represent the time slots in $W_k$ relative to the unknown snapshot time $T_j$, which is to be inferred. For each $t \in W_k$, let $J_{k,t} \in \{S, I, R, SR\}$ be the observed state of node $k$ at time $t$, where $SR$ represents an observation state in which we are unable to distinguish an uninfected state as either $S$ or $R$ state. We lump all observation times into a set $T^O = \bigcup_{k \in N} W_k$. The nodes observed at time $t \in T^O$ are grouped into $N^S_k, N^I_k, N^R_k$ and $N^SR_k$. Here the set $N^O_k$ excludes observed susceptible nodes whose in-neighbors are also observed to be susceptible, because these nodes do not contribute to the infection realization. We use $N^O$ to denote the full node set $N$ with such uninformative nodes removed, and $N^O_k$ to denote the neighbors of node $k$ in the reduced graph for all $k \in N^O$. We further define $N^O_0 = N^{SR}_0 \cup N^I_0 \cup N^R_0 \cup N^SR_0$, for all $t \in T^O$, which lumps all nodes observed at time $t$, and $N^O = \bigcup_{t \in T^O} N^O_0$, which lumps all nodes with at least one observation up to the snapshot time, and $N^C = \bigcup_{t \in T^O} \{N^I_0 \cup N^R_0 \cup N^SR_0 \}$, which lumps all nodes possible to be the infection source (i.e., the candidate sources). Moreover, the prior probability of a candidate to be the infection source is defined by $P^O_k$, for all $k \in N^O$.

We formulate the problem as an approximate MAP estimation of the infection source. Ideally, the true infection source and the true snapshot time should be estimated as

$$\hat{s}, \hat{T}_j \in \arg\max_{s \in N^C,T_j \in T^C} P(s^* = s, s, T_j),$$

where $T^C$ is the set of candidate snapshot times, and $P(s^* = s, s, T_j)$ is the joint probability of node $s$ being the infection source $s^*$, and $N^C$ being the given observations. By Bayes’ rule, the ideal estimator is equivalent to maximizing $P(N^O | s^* = s) P(s^* = s)$. For tractability, we approximate the joint probability $P(N^O | s^* = s)$ by a mean-field probability $\prod_{k \in N^O} \prod_{t \in W_k} P^O_{k,t}(s_{I,k,t}(0))$. As the probabilities $P^O_{k,t}(s_{I,k,t}(0))$ can be computed by iterating the infection model (1)-(10) for a given infection source $s$, this mean-field probability can be computed and then used to compare different candidate infection sources. Together with an approximate prior $P(s^* = s)$, the estimator gives an approximate MAP estimation of the infection source.

Therefore the inference problem can be written in the following optimization form:

$$(P0) \max_{\{T_j \} \in N^C} \sum_{s \in N^C} P^I(s_0) \hat{P}(s = s^*, N^O)$$

subject to $P^I(s_0) = 1 - P^I(s, \forall k \in N^C), (12)$

$P^I(s, \forall k \in N^O, N^C), (13)$

$P^I(s, \forall k \in N^C), (14)$

$P^I(s, \forall k \in N^O, N^C), (15)$

$P^I(s, \forall k \in N^O, N^C), (16)$

$P^I(s, \forall k \in N^O, N^C), (17)$

$T_j \in T^C, P^I(s, \forall k \in N^O, N^C), (18)$

where the infection model (1)-(10) is applied to a reduced graph which excludes the aforementioned uninformative susceptible nodes observed, and (12)-(17) specify initial conditions of the infection model. In the objective function, we let

$$\hat{P}(s = s^*, N^O) = P^I(s_0) \prod_{k \in N^O} \prod_{t \in W_k} y_k(t),$$

which is a mean-field approximation of the ideal joint probability defined in (11). Note that the observation variables $y_k(t)$ in the objective function are expressed by state variables $P_k(t)$ via (10), and that the basic state variables $P_k(t)$ and the auxiliary state variables $\theta_k(t)$, for all $t \geq 0$, $l \in N^O$, and $k \in N^O$, are determined by the decision variables $P^I(s_0)$ for all $k \in N^O$ via (1)-(10). Therefore, the optimization essentially depends on the decision variables $\{P^I(s_0)\}_{t \in N^O}$ and $T_j$. We also note that $T_j$ is larger than the elapsed time between the time when the first side information is observed and the snapshot time.

Because of the fractional and product nonlinearity involved in the infection model (1)-(10), it is difficult to solve (P0) by a standard optimization solver. Nonetheless, it is straightforward to get a solution by enumerating and comparing all feasible solutions of (P0). Given a candidate source and a value of $T_j$, the algorithmic complexity of computing the objective function is $O(dT_j|N^C|)$, where $d$ is the average in-degree of the reduced graph. Since we need to enumerate all candidate values of $T_j$ for all candidate sources, the overall algorithmic complexity is then $O(dT_j|N^C|)^{|T^C|}$. The inference process inherits the merit of the DMP method, except that it is applicable to the SIRI infection model and is able to incorporate side information. For this reason, we call this new estimator the DMP+ estimator.

Although (P0) is derived by assuming that the in-neighbors pass infections to a node independently (cf. (4)), we can still apply (P0) heuristically to a network where this assumption is violated. We expect that the impact on the performance of DMP+ to be small if the network is sparse or has weak correlations between infections passed by neighboring nodes. We note that a similar argument was used to explain the success of well-known BP methods in general network while the methods are based on a similar independence assumption [18].
4. PERFORMANCE EVALUATION VIA SIMULATIONS

We apply the proposed DMP+ estimator to infer single infection sources in random regular tree networks and a subset of the Facebook network under the SIRI model, and then compare the inference results with those obtained with the DMP and Jordan centre (JC) estimators. In applying the DMP estimator to an SIRI model, we ignore the reinfection probability of a recovered node and hence naively treat the model as an SIR model. Like DMP+, we also implement DMP on a reduced graph by eliminating susceptible nodes whose neighbors are all susceptible. During the inference, we assume that the snapshot time falls in the range \([\tau + 1, T_f + \tau - 1]\), where \(T_f\) is the true elapsed time (which may be replaced with a conservative value in practice) and \(\tau\) is the elapsed time since the first side information is observed till the snapshot observation time. In simulations we set \(\tau\) to \(T_f/2\) if \(T_f\) is even and \((T_f - 1)/2\) if \(T_f\) is odd. We assume uniform infection, recovery and reinfection rates at all nodes in the network.

In the case of regular tree networks, we randomly generate a tree network of 1000 nodes with each node’s degree equal to 4, and randomly choose an infection source. We then simulate the infection spreading until either 10 time slots or no less than 20% of the nodes are infected or recovered. We observe the states of all nodes at the last simulation time slot. To simulate the availability of side information, we also collect the states of a random selection of 1% of the nodes at time \(T_f - \tau\). Then we evaluate the three estimators under two sets of parameter settings: (i) the node infection rate increases from 0.05 to 0.95, while the recovery and the reinfection rates are fixed at 0.5; and (ii) the reinfection rate increases from 0.05 to 0.95, while the infection and the recovery rates are fixed at 0.5. To compare the estimators, we adopt the metric of normalized rank of the true infection source [11][12]. We first rank the candidate sources in descending order according to the objective function values (in (P0)); we retrieve the rank \((\geq 1)\) of the true infection source, subtract it by the ideal rank value of 1 (yielding the rank error), and then divide this difference by the total number of candidate sources, which results in the desired normalized rank.

The inference results are shown in Fig. 2 where each point on the curves is averaged over 1000 random instances. Under the parameter setting (i), we observe that DMP+ outperforms DMP and JC (i.e., results in a lower normalized rank of the true source) for most infection rates, except when the infection rate is less than 0.2. This is because in that case, there are too few number of infected or recovered nodes that can be used in the inference process. We also observe that incorporating the small amount of additional observations (side information) improves the performance of both DMP and DMP+, while the JC estimator is unable to use that information as it uses only the network topology. On the other hand, under the parameter setting (ii), we observe that DMP+ almost always outperforms DMP and JC. The advantage becomes more obvious as the reinfection rate increases. When the additional side information is available, we again see improvements in DMP’s and DMP+’s performances.

In the case of the Facebook network subset with 500 nodes, we randomly select a node and fix it as the infection source, and then perform simulations under the same parameter settings as the regular trees. The results are shown in Fig. 3 where each point on the curves is averaged over 300 spreading instances. Under the parameter setting (i), we observe that DMP+ outperforms DMP in the whole range of infection rates, and is worse than JC only if the infection rate is small (roughly less than 0.12). Under the parameter setting (ii), it turns out that DMP+ always outperforms DMP and JC. The performance gain increases and then saturates as the reinfection rate becomes larger. Again, we see that incorporating additional observations improves the performances of both DMP and DMP+ under either of the two parameter settings.

5. CONCLUSION

We have introduced a state-space description of an SIRI infection model, and using the state propagation equations, we have derived an approximate MAP estimator for the infection source, given the observations of a partial or complete set of node states at a snapshot time. Our proposed estimator is able to incorporate side information like observations of node states at intermediate times during the infection spreading and prior beliefs of potential candidate infection sources, into the inference procedure. Simulations on random regular tree networks and a subset of the Facebook network suggest that the DMP+ estimator outperforms the Jordan center and DMP estimator for large ranges of the infection and reinfection rates.

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