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# Causal Effects Under Interference

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## Abstract

In randomized experiments, it is often assumed that the response of a given unit only depends on its own characteristics and the treatment to which it is assigned. However, there are numerous situations of interest to researchers where this may not be a plausible assumption, particularly when there is social interaction between individuals in an experiment, and the ability to separately identify the effects of treatment assignment and peer influence have important implications. This paper presents a general framework for reliably identifying and estimating causal effects in experimental settings where there is potential interference between units. The framework consists of (i) learning the potential for interference between connected units, (ii) a probabilistic graphical model for estimating individuals' exposure to treated units, and (iii) methods that make use of estimated exposures to compute causal effects of interest. We then evaluate the performance of the proposed framework on by simulating synthetic experimental data on a variety of real world social networks.

## 1 Introduction

Reliable causal inference is central to a variety of disciplines in the natural and social sciences. While the Randomized Control Trial (RCT) has long been the gold standard in terms of causal inference, central to it is an assumption in the economic literature first coined by Rubin [1990] known as SUTVA, or the stable unit treatment value assumption, which states that there is no interference between units, and no hidden variations of treatment. Essentially, the outcome of each individual does not depend on the treatment assignment of others. When this assumption is violated, there are said to be spillover effects. And it often is. In an RCT on 61 million Facebook Users in the buildup to the 2020 United States Congressional elections, the treatment—sending a message that encouraged users to vote—was observed to “spillover” to the treated individual’s closest Facebook friends Robert M. Bond [2012].

A number of recent works have sought to address estimation of both treatment and spillover effects in the presence of SUTVA violations, particularly in the context of an observed social network between individuals. Toulis and Kao [2013] seek to estimate spillover effects directly, developing both an experimental design using sequential randomization, and a Bayesian procedure that assumes a linear response. Other approaches consist of the researcher specifying an “exposure mapping” to categorize individuals, e.g. treated, neighbor of treated, etc. and then use inverse probability weighting based on the probability of assignment for each individual Aronow and Samii [2017]. Due to the inherent graphical nature of the problem, this paper seeks to examine how a probabilistic graphical model may be well suited to capture interference and spillover effects in a social network. We develop a flexible framework that defines a probabilistic graphical model over the social network, and uses this model to estimate unit level exposure to treated units, allowing us to compute causal effects of interest.

## 2 Setting and Assumptions

We consider the setting of a randomized experiment on a social network of  $N$  nodes. The network,  $G = (V, E)$ , is observed, and may or may not contain node-level covariates or edge strengths. Let  $\mathbf{Z} = (Z_1, Z_2, \dots, Z_N)$  be a treatment assignment vector over  $N$  units, where  $Z_i \in \{0, 1\}$  specifies which of the possible treatments unit  $i$  receives. We assume that  $P(Z_i = 1)$  is the same for all  $i \in \{N\}$ , so treatment assignment is unconfounded. We define  $\mathbf{C} = (C_1, C_2, \dots, C_N)$  to be a latent exposure vector, where  $C_i \in \{0, 1\}$  specifies if unit  $i$  is exposed to the treatment via social interaction. Here, we assume that all treated units are exposed, such that  $Z_i \times C_i = 1$ , but that treatment units do not experience further spillover effects. If we could observe  $\mathbf{C}$ , then our work would be done, since we could partition the units into treatment, exposure, and control groups, and employ classic techniques to infer their differences in outcome.

Instead, we define  $\pi_i = P(C_i = 1)$  to be the exposure probability for unit  $i$  given some instantiation of the experiment  $\mathbf{Z}, \mathbf{C}$ . Since an individual's social exposure to the treatment depends on the random assignment in  $G$ , so does  $\pi_i$ . For simplicity, we assume that the effect of treatment assignment is homogeneous. Furthermore, we assume that social interaction, and thus social exposure to the treatment, only occurs through the edges  $E$  of  $G$ . However, we do not assume that spillover effects are homogeneous, and examine both this and the heterogeneous case in later sections.

## 3 Estimating Exposures and Causal Effects

We propose modeling the spread of influence in the social network as a Pairwise Markov Random Field, defined as  $B = (P, H)$ , where  $H = (V, E)$  is an isomorphism of the social network  $G$ , given by the bijection  $f : G(V) \mapsto H(V)$  where  $f(v_i) = X_i \sim \text{Bernoulli}(p)$ , i.e.  $H$  is an undirected graph with the skeleton of  $G$  where each node in  $H(V)$  is a Bernoulli random variable.  $P$ , then, is defined as a Gibbs distribution that factorizes over  $H$ :

$$P(X_1, X_2, \dots, X_n) = \frac{1}{Z} \prod_{i \in V} \phi_i(X_i) \prod_{(i,j) \in E} \phi_{i,j}(X_i, X_j)$$

By having each individual represented as a bernoulli random variable in  $H$ , we can reformulate the problem of estimating the exposure probability for node  $i$ ,  $\pi_i(\mathbf{Z})$ , into an inference problem, where we seek to infer marginal probability distributions for each node. Observe that for any treated unit, they are assumed to be already exposed, so  $X_i = 1$  for all individuals  $i$  where  $Z_i = 1$ . This observation acts as our evidence in  $P$  when performing inference on the unknown marginal distributions of the other nodes in  $H$ . Formally, we infer the posterior distribution  $P(X_i | e)$ , where  $e = \{X_i = 1 \mid Z_i = 1\}$ . This reformulation allows us to use a host of existing inference algorithms to learn the posterior marginals.

### 3.1 Unary and Edge Potentials

Having reformulated estimating  $\pi_i(\mathbf{Z})$  as inference of marginals in a Pairwise MRF, properly defining the edge and unary potentials in  $H$  becomes an important specification. In this section, we discuss several potential approaches. In the setting considered by this paper, treatment assignment is unconfounded, so the unary potentials  $\phi_i(X_i)$ , are uniform across the nodes such that  $\phi_i(X_i = 1) = \phi_i(X_i = 0) \forall i$ . However, in the case that treatment assignment is, for instance, confounded by covariates, it may make sense to use the unary potentials as a prior on similarity with the treatment group, since influence may flow to “similar” nodes more easily.

The edge potentials  $\phi_i(X_i, X_j)$  have an even more intuitive interpretation, representing the potential for influence, or “strength” of a social connection between two nodes in  $H$ . Here, we propose two possible approaches for specifying the edge potentials. Both involve first specifying the probability of each edge in the social network. The first is that, given domain knowledge within the experiment, the researcher may have a prior on the distribution of edge probabilities, and can make use of this prior belief. For instance, let  $p_{i,j}$  be the probability of an edge between node  $i$  and node  $j$ . A reasonable prior could be  $p_{i,j} \sim \text{Beta}(5, 5)$ . This belief could be incorporated by initializing the edge potentials according to this distribution. The next is a latent variable model introduced by Handcock et al. [2007]. Where we model  $p_{i,j}$  using a logistic regression where the probability of an edge depends on

euclidean distance in latent space:

$$\log\text{-odds}(p_{i,j}) = \beta X_{i,j} - \|z_i - z_j\|^2 \quad (1)$$

Where  $z_i$  and  $z_j$  are node  $i$  and node  $j$ 's respective positions in latent space, and  $X_{i,j}$  is some vector valued edge covariates. Here, it is assumed that the existence of an edge is independent of other edges. Handcock jointly estimates the parameter  $\beta$  and the latent  $z_i$ 's using MCMC, with prior:

$$z_i \sim \sum_{g=1}^G \lambda_g \text{MVN}_d(\mu_g, \sigma_g I_g)$$

Where  $G$  is the possible number of latent social clusters. Since we can take  $G = 1$ , the latent variable model provides a flexible and well studied approach to estimating the probability of edges between nodes in a variety of experimental settings. We test both possible approaches to estimating edge probabilities in the following section.

Given the edge probabilities  $p_{i,j}$ , we can specify the the edge potentials in  $H$ . In doing so, we make the assumption that a higher edge probability leads to greater social influence, and thus likelihood that two adjacent nodes have the same value, i.e.  $P(X_i = X_j)$  is monotonically increasing in  $p_{i,j}$ . To acheive this, we define the edge potentials as:

$$\phi_{i,j}(X_i, X_j) = 1 - p_{i,j} \mathbf{1}\{X_i \neq X_j\}$$

We found that this specification resulted in a plausible empirical results across a number of different social networks, but this is another place where a researcher may choose to incorporate prior beliefs in specifying how social influence propagates in a specific network.

### 3.2 Estimating Causal Effects

With a distribution over the exposure probabilities for each node, we can turn to the problem of estimating the causal effects of interest. First, we are interested in estimating the average treatment effect, ATE. A naïve estimate of ATE in our setting would be a simple difference of means between the treatment and control groups. However, as discussed, in the presence of spillover effects, this estimate is that of both ATE, and whatever spillover effects may be present in the experiment. Instead, we propose the OLS regression:

$$y_i = \alpha + \rho Z_i + \gamma(1 - Z_i)\pi_i + \varepsilon_i \quad (2)$$

Where  $\rho$  is the parameter of interest. Since  $\pi_i = 1$  if  $Z_i = 1$ , we add the interaction term  $(1 - Z_i)$ , so that the causal effect of treatment assignment is only contained in  $\rho$ . Unfortunatley, the coefficient  $\gamma$  does not have much causal interpretation, since  $\pi_i$  is merely the probability that unit  $i$  is socially exposed to the treatment. In theory, the above regression model, and our approach in general, is better suited to the heterogeneous spillover effects case, since potential outcomes are linear in  $\pi_i$ . In this case, we could interperent  $\gamma \times \pi_i$  to be the partial effect of exposure for unit  $i$ .

In the case of homogeneous spillover effects, what we really desire is to be able to classify each non-treated unit into either the socially exposed ( $C_i = 1$ ), or control group ( $C_i = 0$ ), as doing so would allow for perfect identification of average spillover effects. There are a number of possible approaches. One might be assigning units with exposure probability sufficiently close to zero to the control group, and having a similar condition for assigment to the expsoure group. Another may be to explicitly learn a classifier. If treatment assigment were confounded by latent or observed covariates, this may be possible without taking the outcome of interest into account, since the treatment and exposure groups would have greater similarity than the controls. However, the setting of this paper assumes that treatment assignment is unconfounded,

## 4 Simulations

In order to test the performance of our proposed methods, we simulate random experiments on two real world social networks. The first is a group of 55 eigh-graders, with edges between students who were surveyed on which other students they would like to sit next to in class. The second data set is a social network between 61 employees of the Aarhus Computer Science Department, where edges

represent colleagues who ate lunch together in a given week. We simulate the outcome of interest drawing from a normal distribution  $y_i \sim \mathcal{N}(\mu, \sigma^2)$ , and then consider a dilated effects scenario, where spillover is half of the average treatment effect. For each graph, we consider three possible data generating processes for the spillover effects. In the first, all neighbors of treated units are socially influenced. In the second, social influence is propagated across each edge  $(i, j)$  with probability  $p_{i,j}$  according to the latent variable model (1), i.e. socially influenced neighbors of treated units can influence their neighbors etc. until all possible influence has propagated across the network. The third is the same as the second process except  $p_{i,j} = 0.5$  for all edges in the network. We also test two different specifications for the edge potentials in each scenario. In the first, the edge potentials accord with the latent variable model (1), and in the second we assume that  $p_{i,j} \sim \text{Beta}(5, 5)$ . The following table report the point estimates of the OLS regression (2) and corresponding standard errors following 1,000 simulated treatment assignments on each of the possible combinations of data generating processes, edge potentials, and social networks. In the setting of this paper, since treatment assignment is unconfounded

Table 1: Simulation Results (True parameter:  $\rho = 3$ )

DGP	$\phi_{i,j}$	Eighth Graders		Aarhus CS	
		$\bar{y}^1 - \bar{y}^0$	$\rho$	$\bar{y}^1 - \bar{y}^0$	$\rho$
Neighbors	Latent	2.67	3.03 (0.30)	2.31	3.03 (0.43)
	Beta	2.66	3.14 (0.38)	2.32	3.63 (0.63)
Latent	Latent	2.73	2.93 (0.33)	2.55	2.93 (0.43)
	Beta	2.72	2.95 (0.35)	2.54	3.08 (0.61)
$p_{i,j} = 0.5$	Latent	2.55	2.67 (0.38)	2.31	2.48 (0.46)
	Beta	2.56	2.81 (0.40)	2.32	2.64 (0.67)

The estimates for  $\rho$

## 5 Discussion

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